



Third Edition

Understanding the Essentials of
**CRITICAL CARE
NURSING**

Kathleen Ouimet Perrin • Carrie Edgerly MacLeod



Understanding the Essentials of Critical Care Nursing

Third Edition

Kathleen Ouimet Perrin, PhD, RN, CCRN

Carrie Edgerly MacLeod PhD, APRN-BC



330 Hudson Street, NY, NY 10013

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About the Authors

KATHLEEN OUIMET PERRIN, PhD, RN, CCRN, is professor emerita and adjunct professor of nursing at Saint Anselm College in Manchester, New Hampshire, where she has taught critical care nursing, professional nursing, ethics, health assessment and understanding suffering. While teaching at the college, she received the AAUP award for Excellence in Teaching. She received her bachelor's degree from the University of Massachusetts, Amherst, her master's degree from Boston College, and her PhD from Union Institute and University in Cincinnati, Ohio. She has been a practicing critical care nurse for more than 40 years, and has been a member of the American Association of Critical Care (AACN) Nurses for nearly as long. Kathleen has served on the board of the Southern New Hampshire AACN and is a past president of the chapter. She has been on numerous review panels for the national AACN. She was a member of the board of directors and President of the Epsilon Tau chapter of Sigma Theta Tau International. She has published and presented in the areas of critical care nursing, nursing ethics, nursing history, suffering experienced by patients and health care providers, and conflict among members of the health care team. She has written two other nursing texts: *Nursing Concepts: Ethics and Conflict*, and *Palliative Care Nursing: Caring for Suffering Patients*, which won an AJN Book of the Year Award in 2011.

CARRIE EDGERLY MACLEOD PhD, APRN-BC currently works as an assistant professor at Saint Anselm College in Manchester, New Hampshire where she teaches critical care nursing. She also works as a nurse practitioner in Cardiac Surgery in Massachusetts. She has worked in critical care settings at major teaching institutions in New Hampshire and New York. She received her bachelor's degree from Saint Anselm College and both her master's degree and PhD from the William F. Connell School of Nursing, Boston College. She has served as a faculty member at both at Saint Anselm and Boston College where she taught pharmacology, pathophysiology, and critical care nursing. She has published in the areas of patients' and family caregivers' experiences after cardiac surgery. Dr. MacLeod has lectured on management of the critically ill client at many symposiums across the United States. She has received both academic and clinical awards for her contributions to critical care nursing and client care.

Dedication

This book is dedicated to my husband, Robin. He insisted that I should write the first edition of this book, and he has continued to support me as I developed each subsequent edition.

It is also dedicated to critical care nurses, specifically to those critical care nurses whom I have seen develop from novice nurses into expert clinicians. It has been an absolute joy to watch former students as they evolved from fledgling nurses into expert practitioners, capable of caring for the very sickest of patients, educating future nurses, and advancing the profession of nursing. One of the most fulfilling experiences in my life has been watching my former students and seeing them develop into nurses far better than I could ever hope to be. I hope this book will serve as a foundation for nurses in the future as they make that transition.

—*Kathleen Ouimet Perrin PhD, RN, CCRN*

I would like to dedicate this book to my husband, David, and my daughters, Annie and Kate. Like most things in my life, I could not have taken this journey without the three of you and the support you give me every day. I also want to thank Kathleen Perrin for her guidance and mentoring over these many years. She inspired me as my professor and continues to do so as my friend. I would not be the nurse I am today if not for her. Lastly, I would like also to dedicate this book to my parents, James and Jean Edgerly, who are the reasons I became a nurse. Every time a nurse helps to save a life, I think of them. I am so proud of our profession and what we do as nurses each and every day.

—*Carrie Edgerly MacLeod PhD, APRN-BC*

Thank You

Our heartfelt thanks go out to our colleagues from schools of nursing across the country who have given their time generously to help us create this exciting new edition of our book. We have reaped

the benefit of your collective experience as nurses and teachers, and we have made many improvements due to your efforts. Among those who contributed to this edition are:

Textbook Contributors

Allanah M. Bachman, MSN, ACNP, GNP
Department of Cardiac Surgery North Shore Medical Center
Salem, Massachusetts

Sue Barnard MS, APRN, CCRN
Trauma/Stroke Program Coordinator
Saint Joseph Hospital
Nashua, New Hampshire

Michele Bettinelli, BS, RN, CCRN
Lahey Hospital and Medical Center
Burlington, Massachusetts

Destiny Brady, MSN, RN, CCRN
Clinical Instructor
Critical Care Nursing
Saint Anselm College
Manchester, New Hampshire

Tricia Charise MS, ACNP
Boston Medical Center, Department of Trauma Surgery
Boston, Massachusetts

Linda Edelman, PhD, BSN, RN
Assistant Professor
University of Utah College of Nursing
Salt Lake City, Utah

Ernest Grant PhD, RN
University of North Carolina at Greensboro
Greensboro, North Carolina

Shirley Jackson, MS, RN, CCRN
Critical Care Nurse Specialist
Elliot Hospital
Manchester, New Hampshire

June Kasper, MS RN
Clinical Educator, Endoscopy
Lahey Clinical Medical Center
Burlington, Massachusetts

Mary Kazanowski PhD, APRN, ACHPN
APRN, Palliative Care Team,
Elliot Hospital
Manchester, New Hampshire

Erin McDonough, DNP, AGACNP-BC
Lead Critical Care Nurse Practitioner
Catholic Medical Center
Manchester, New Hampshire

Betsy Swinny, MSN, RN, CCRN
Faculty III & Critical Care Educator
Baptist Health System, School of Health Professions
San Antonio, Texas

Reviewers

Katrina Allen-Thomas, RN, MSN, CCRN
Faulkner State Community College
Bay Minette, Alabama

Marylee Bressie, DNP, RN, CCNS, CCRN, CEN
Capella University
Minneapolis, Minnesota

Annie Grant, RN, BSN, MSN, CNS
Florence-Darlington Technical College
Florence, South Carolina

Laura Logan, MSN, RN, CCRN
Stephen F. Austin State University
Nacogdoches, Texas

Predrag Miskin, DrHS, MScN, RN, PHN
Samuel Merritt University
San Mateo, California

Bridget Nichols, RN, BAN, MSN, CCRN
University of South Dakota
Sioux Falls, South Dakota

Jill Price, PhD, MSN, RN
Chamberlain College of Nursing
Kapaa, Hawaii

Preface

This book is an introduction to critical care. It focuses on elements that are essential for the novice critical care nurse to understand—whether the novice is a student or a new graduate. When critical care nursing was introduced as a specialty more than 50 years ago, the focus of care was on patient observation and prevention of complications of the disease or treatment. Over the past 50 years, critical care has become curative care. Now, most patients have favorable outcomes, surviving to return home following complex treatments that often include life support. However, patient survival and well-being do not just depend on the development of new and ever more complex treatments. Rather, the presence of well-educated, expert nurses has been shown to have a significant impact on patient outcomes. This book focuses on the essentials for beginning critical care nurses so that they may deliver the safe, effective care that *optimizes* patients' outcomes.

We are fortunate that critical care practice has changed from the early years when health care providers learned as they went along, experimented with new interventions on their patients, and often relied on intuition to choose those interventions. Intuition could not be trusted as a basis for practice, and the experiences provided too small a sample to draw inferences. Whenever possible, this book relies on evidence-based recommendations for collaborative and nursing practice. It cites individual research studies, but more often cites meta-analyses and evidence-based practice recommendations made by respected professional organizations. When the foundation for practice is based on evidence, it is far more likely to be safe and effective.

Since the last edition of the text in 2013, much has changed in the provision of care to critically ill patients. Evidence supports significant changes in the provision of sedation and pain medication as well as the management of ventilation, heart failure, stroke, blood or volume resuscitation in trauma, palliative care and sepsis. All of these new recommendations for practice are incorporated in the third edition of this text.

Critical care nursing is an evolving specialty. *Understanding the Essentials of Critical Care Nursing* is intended to provide novice critical care nurses with a firm foundation so that they are able to understand the complexities of care, deliver safe, effective care, and begin their transition to expert critical care nurses.

Organization and Key Themes of This Book

The topics for these chapters were chosen after reviewing suggestions for foundational critical care content from a variety of nursing organizations, including the American Association of Critical Care Nurses and the National Council of State Boards of Nursing. The first chapter addresses what is unique about critical care and critical care nursing, including legal and ethical issues nurses encounter. The second chapter focuses on the needs and concerns that are common to critically ill patients or their families, and it explores ways nurses might meet those needs. The remaining chapters describe the essentials of providing care to patients with disorders that are commonly seen in critical care settings. There is no attempt to cover all possible content. Rather, the text concentrates on problems that the new critical care nurse is most likely to encounter. Because many patients die in critical care units, or shortly after being transferred out of critical care units, the final chapter discusses care of the dying patient.

A recurrent theme in this book is safe practice. As critical care has become more complex, the potential for error has increased. Chapter 1 includes a discussion of some of the reasons why errors are common in critical care units. Fortunately, there are documented ways in which nurses can prevent or limit health care errors. One of the most effective ways to prevent errors is to improve communication and collaboration among members of the health care team, as described in Chapter 1. In each subsequent chapter, a Safety Initiative feature describes specific recommendations by the Institute for Health Care Improvement and other national groups that, when implemented, can limit errors and enhance patient safety.

As we have gained expertise in critical care, we have learned that not all adult patients with a particular diagnosis are the same. Specifically, we have begun to realize that older and overweight adults have unique needs. With the increasing numbers of people in these cohorts, knowledge of how to care for them must be part of the foundation of critical care practice. We have included information on gerontological and bariatric patients as separate **Gerontologic Considerations** and **Bariatric Considerations** in each chapter.

In this text, **Nursing Actions** are a component of **Collaborative Management**. The content in the Nursing

Action sections emphasize nursing interventions required for safe, effective medical and surgical management of the patient—for example, what are the nursing actions when administering amiodarone, or what nursing assessments are essential after a patient has a cardiac catheterization. In contrast, the **Nursing Care** sections highlight interventions that focus on providing care to a patient and creating a healing environment. Nursing Care sections focus on promoting patient comfort, providing adequate nutrition, and assisting the patient and family to cope with the critical illness or impending death.

Nursing management of critical care patients includes using some of the latest technology developed for the health field. **Building Technology Skills** text sections focus on specific technology that the nurse is most likely to encounter when caring for patients experiencing the conditions discussed in the chapter, and the related skills required to use that technology.

A critically ill patient is a dynamic system of interrelated factors. In order to help visual learners understand the relationships between and among these factors, each chapter includes at least one **Visual Map** to illustrate the relationships among the disease states, collaborative interventions, and outcomes.

Commonly Used Medications, those that are most often prescribed for the conditions addressed in the chapter are highlighted in these boxes. For each medication, information is provided on dosing information, desired effects, nursing responsibilities, and potential side effects.

Safety is an essential focus in critical care settings. **Safety Initiative** boxes highlight specific issues related to the content in each chapter. Included are the purpose, the rationale, and highlighted recommendations.

In each chapter, a **Case Study** of a real-life patient scenario illustrates the chapter content and provides an example of collaborative and nursing management. Critical thinking questions allow the reader to solve the posed problems. The case studies continue on the Com-

panion Website, offering learners the opportunity to extend the textbook learning and submit responses to their instructors.

Critical Thinking Questions are also located at the end of the chapter; these are designed to help students develop a deeper understanding of the content and explore relationships among concepts discussed in the section.

Essentials identify evidence-based practices, communication strategies, safety measures, or system-based practices that the novice nurse must know to practice safely. The areas chosen to be highlighted as essentials are derived from the Robert Wood Johnson Nurse of the Future initiative. The goal of this feature is very similar to the overall goal of QSEN (Quality and Safety Education for Nurses), which is “to meet the challenge of preparing future and new nurses who will have the knowledge, skills and attitudes necessary to continuously improve the quality and safety of the healthcare system within which they work.”

Reflect On is a feature that promotes reflection and journaling on some of the difficult issues that nurses encounter in their practice. This feature was added to the second edition because reflection on the difficult issues and times in personal practice has been shown to facilitate a novice nurse’s progression to expert nurse.

In addition to the features that were retained from previous editions, a new feature added to this third edition.

Why/Why not? This feature asks students to critically analyze WHY they should be implementing a specific collaborative management strategy or nursing action for a patient and why it might NOT be appropriate to implement the strategy or action for that particular patient. The Why/Why not feature addresses questions concerning medications, therapies, diagnostic testing, patient and family interactions, collaborative communication, and more. The feature can be used for in-class discussion or individual student journaling concerning the most appropriate collaborative management or nursing care to provide to critically ill patients in complex situations.

Acknowledgments

We appreciate the energy, time, and thought that the authors of all the chapters put into this edition, giving up weekends and holidays, and persisting despite personal and family difficulties. They brought their expertise in critical care nursing to each of their chapters and their knowledge is one of the foundations of this book.

We appreciate the hard work of the reviewers who made certain that all of our content was absolutely accurate and up to date. We also benefitted from suggestions from our students who used the previous editions of this book. Their thoughtful comments were the basis for revisions in this edition.

We could not have completed this task without the assistance and advice of our editors at Pearson who have remained with us from the previous editions. From the time Pamela Fuller developed the idea of this book, she has been incredibly supportive. Barbara Price has been our constant e-mail companion, keeping us on track, helping us understand the process of electronic publication, and easing all the chapter authors through the rough spots.

Kathleen Ouimet Perrin *Ph. D., RN, CCRN*
Carrie Edgerly MacLeod *PhD, APRN-BC*

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Chapter 1

What Is Critical Care?

Kathleen Perrin, PhD, RN, CCRN

Abbreviations

AACN	American Association of Critical-Care Nurses	IHI	Institute for Healthcare Improvement
AHRQ	Agency for Healthcare Research and Quality	IOM	Institute of Medicine
ANA	American Nurses Association	QSEN	Quality and Safety Education for Nurses
ICU	Intensive Care Unit	SCCM	Society of Critical Care Medicine



Learning Outcomes

Upon completion of this chapter, the learner will be able to:

1. Analyze the key components of safe, effective care in the critical care environment.
2. Explain the essential attributes of the role of critical care nurse.
3. Examine the multidisciplinary nature of care within the critical care environment.
4. Explain the ethical and legal issues in critical care.
5. Differentiate among the major factors that affect the well-being of critical care nurses.

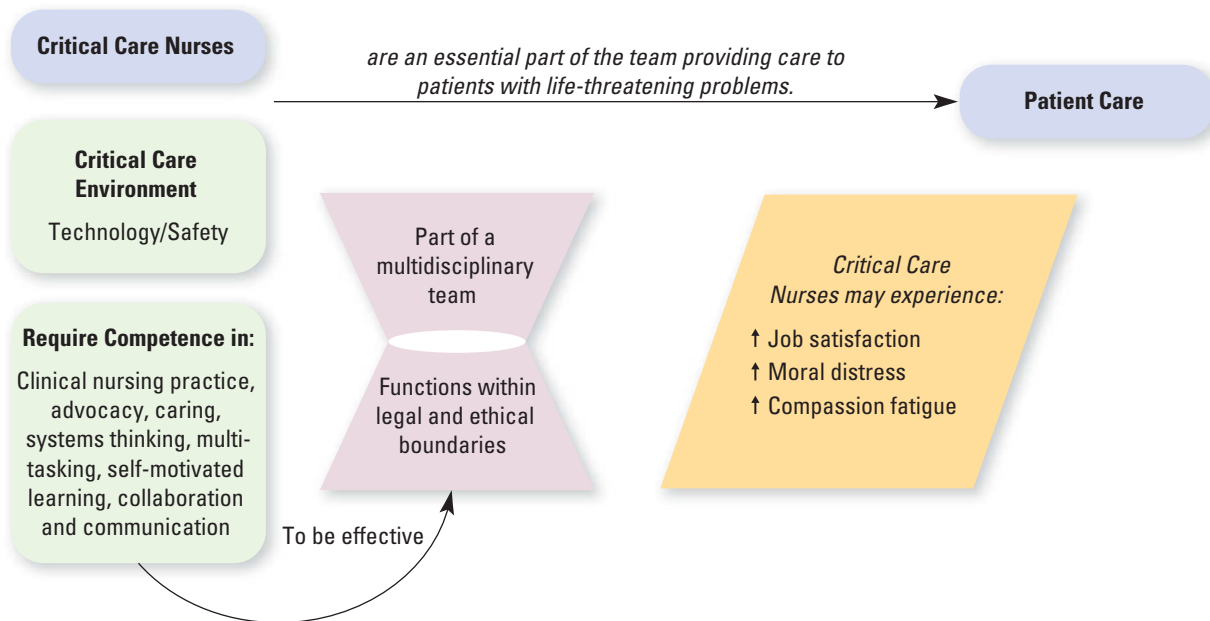
The Critical Care Environment

Critical care is defined by the Department of Health and Human Services (2008) as the direct delivery of medical care for a critically ill or injured patient. To be considered critical, an illness or injury must acutely impair one or more vital organ systems to such a degree that there is a high probability of life threatening deterioration. Critical care involves highly complex decision-making and is usually, but not always, provided in a critical care area such as a coronary care unit, an intensive care unit, or an emergency department. Visual Map 1-1 displays the role of the nurse and the multidisciplinary team in the delivery of critical care to a patient.

Trends in Critical Care Units

Although seriously ill patients had historically been grouped together and cared for by a designated nurse, usually near the nurses' station, they were not separated from other patients and placed in critical care units until the early 1950s. At that time, the use of mechanical ventilation and cardiopulmonary resuscitation began, and it became more efficient to provide care to gravely ill patients with specially trained nurses in one location in the hospital. By 1958, approximately 25% of community hospitals had an intensive care unit (ICU), and by the late 1960s, nearly every hospital in the United States had an ICU.

The number of critical care beds in hospitals has been increasing since 1985, and the number of noncritical care



Visual Map 1-1 Critical Care Overview

beds has been decreasing. These changes are a result of technological advances that have allowed critical care to become a cost-effective way to treat many patients. The use of noninvasive monitoring and targeted pharmacological therapy has resulted in fewer complications, and, therefore, the cost of caring for some critically ill patients has decreased. This has produced not only cost savings but shortened hospital stays as well, especially for patients with specific organ system failures such as severe sepsis and acute respiratory failure (Society of Critical Care Medicine [SCCM], n.d.).

According to the SCCM, there are currently nearly 6,000 ICUs across the United States with every acute care hospital having at least one ICU. However, there are many differences among the units that are called ICUs. Kirchhoff and Dahl (2006) determined that “unit findings often varied depending on the size of the unit, or size or location of the hospital the unit was in (e.g., urban, suburban, or rural hospitals)” (p. 18). In their study, the median number of beds in an ICU was 16, and the average number of admissions was about 2,000 per year. In most critical care units, the length of patient stay was between two and five days.

Critical care units need to differ because not all hospitals are intended to meet the needs of all types of patients and severity of illness. In 2003, the SCCM endorsed guidelines for critical care services based on three levels of care (Haupt et al., 2003). These guidelines suggested that each hospital provide a level of care appropriate to its mission and the regional needs for critical care services. The recommended levels of care are:

- **Level I:** Comprehensive care for a wide variety of disorders. Sophisticated equipment, specialized nurses, and physicians with specialized preparation (intensivists) are continuously available. Comprehensive support services from pharmacy, nutrition, respiratory, pastoral care, and social work are nearby. Most of these units are located in teaching hospitals.
- **Level II:** Comprehensive critical care for most disorders but the unit may not be able to care for specific types of patients (e.g., cardiothoracic surgical patients). Transfer arrangements to Level I facilities must be in place for patients with the specific disorders for which the unit does not provide care.
- **Level III:** Initial stabilization of critically ill patients provided but limited ability to provide comprehensive critical care. A limited number of patients who require routine care may remain in the facility, but written policies should be in place determining which patients require transfer and where they ought to be transferred.

Critical care units also differ in whether they are open or closed. In an open ICU, nurses, pharmacists, and respiratory therapists are ICU-based, but the physicians directing patient care may have other obligations. These physicians may or may not choose to consult an intensivist to assist with the management of their ICU patients. In a **closed ICU**, patient care is provided by a dedicated ICU team that includes a critical care physician. The Leapfrog Group (2014) recommends that ICUs should be closed units because mortality rates are 40% and morbidity rates

are 30% lower in closed ICUs. If all ICUs were closed, 55,000 deaths could be prevented each year. Unfortunately, there are not a sufficient number of intensivists to ensure that every ICU in the country can be closed, and only about 30% of ICUs meet Leapfrog's standards. In a report to Congress, the Department of Health and Human Resources (n.d.) stated that vulnerable populations, especially the uninsured and those living in rural areas, receive less than optimal care because smaller hospitals are unable to have intensivists consistently available.

Characteristics of the Critical Care Environment

Clearly, the specific nature of the critical care unit and the type of care delivered vary depending on the size and level of the unit. However, over the past 15 years across the level and size of critical care units, there have been more patients receiving care. In addition, those patients have been more acutely ill. Moreno, Rhodes, and Donchin (2009) state that there is a current pandemic of critical illness in part because the population is older and sicker. It is anticipated that the demand for critical care services will continue to grow over the next 20 years as the baby boom generation ages because Americans over the age of 65 utilize the majority of ICU services.

There are other commonalities among ICUs. The SCCM (n.d.) states that critical care is provided by multi-professional teams of highly experienced and professional physicians, nurses, and others. These healthcare professionals use their expertise to interpret information and provide care utilizing technologically advanced equipment that leads to the best outcomes for their patients. The qualities of specialized nursing are discussed later in this chapter, but some of the issues associated with specialized equipment and intensified, comprehensive care are discussed next.

Critically ill patients require complex, carefully coordinated care. When a care pattern is complex, failure in one part of the system can unexpectedly affect another. In addition, the care provided to critically ill patients is often coupled, meaning there is little or no buffer between events. Thus, if anything goes wrong, everything can unravel quickly. In addition, when things are tightly coupled, even when an error is identified, it can be difficult to prevent the situation from deteriorating. In part, this is because of the complexity and high degree of coupling of care in critical care areas, specifically emergency departments (EDs), ICUs, and operating rooms (ORs), where healthcare errors most commonly occur.

However, not only do the critically ill patients receive highly complex care, but the care they receive is also highly technological. In a foundational study, Leape and Brennan (1991) found that 44% of healthcare errors were

related to technology, and that all errors were more likely to occur in technologically advanced fields such as vascular, cardiac, and neurosurgery. The Institute of Medicine (Kohn, Corrigan, & Donaldson, 2000) postulates that technology increases errors for several reasons, including the following:

- Technology changes the tasks people do by shifting the workload and eliminating human decision-making.
- Although technology may decrease human workload during nonpeak hours, it often increases the workload during peak hours or when the system fails or is inadequate (e.g., when medication-scanning devices fail without warning and nurses are required to utilize paper systems to dispense medications then must back-track and re-document when the scanner is working).
- When the system becomes opaque, users no longer know how to perform a function without it (e.g., when intravenous [IV] pumps are constantly used to calculate doses of continuous medication infusions, nurses can no longer calculate the rate to infuse a drug at a specific dose of mcg/kg/min by hand). Therefore, errors may occur when the system fails.
- When devices are not standardized and demand precision to use (e.g., an ICU uses multiple brands of IV pumps or ventilators), problems can result.

Sandelowski (1997) expressed concern about how nurses interact with technology, believing that when nurses focus on interpreting machine-generated texts (such as rhythms on an electrocardiogram [ECG] monitor), they may fail to touch patients enough or in the right way. She warned that technology could change the way nurses obtain information from patients and the information they obtain. Thus, the use of technology, although essential to the delivery of critical care, can also predispose to errors in the delivery of care.

After reviewing the Institute of Medicine (IOM) concerns about patient safety in all healthcare environments, the Robert Wood Johnson Foundation established and funded the Quality and Safety Education for Nurses (QSEN) project in 2005. QSEN targeted six competencies for improving the quality and safety of healthcare systems and nursing practice. The competencies include patient-centered care, teamwork and collaboration, evidence-based practice, quality improvement, safety, and informatics. This text focuses on describing the ways to deliver the safest, most effective collaborative care for specific patients according to the most recent evidence.

Safety

The safety of all patients is a concern. However, safety for vulnerable, unstable patients receiving critical care is

paramount. Mattox (2010) confirmed that the most vulnerable of unstable ICU patients are at highest risk for medical error (e.g., patients in isolation, patients with limited English proficiency or health literacy, and patients at end of life). Valentin et al. (2006) examined errors that occurred in 205 ICUs worldwide during one 24-hour period. Only about a quarter of the ICUs reported no errors. The remaining units reported the following types of errors:

- Dislodgment of lines, catheters, and drains
- Medication errors (such as wrong dose, wrong drug, or wrong route)
- Failure of infusion devices
- Failure or dysfunction of a ventilator
- Unplanned extubation while ventilator alarms were turned off

From these data, Valentin et al. (2006) concluded, “Sentinel events related to medication, indwelling lines, airway, and equipment failure in ICUs occur with considerable frequency. Although patient safety is recognized as a serious issue in many ICUs, there is an urgent need for development and implementation of strategies for prevention and early detection of errors” (p. 1591).

This concern about the frequency of errors and the need to develop preventive strategies is also apparent in a study by Garrouste-Orgeas et al. (2010), who measured the incidence and rates of **adverse events** in critical care. Twenty-six percent of the patients they followed experienced at least one adverse event. Garrouste-Orgeas et al. (2010) concluded that serious errors were common in critical care settings and translated to a rate of 2.1/1,000 patient days. These preventable errors were often associated with a combination of human factors and system-wide problems that caused errors or near misses (Garrouste-Orgeas et al., 2012). They concluded that it is important to find ways to develop work conditions (systems) that engineer out slips and lapses so that treatment is delivered as intended. In a system-based approach, the focus is not on who committed the error but rather determination of how the error occurred.

Since the release of the Institute of Medicine’s (IOM) report, *To Err Is Human* (Kohn et al., 2000), there has been a focus on uncovering system-wide problems and diminishing the potential for errors in hospitals in the United States. To decrease the potential for errors, the report recommends the following:

- **Utilizing constraints:** An example of a constraint is when the height, weight, and allergies of the patient must be on file to obtain medication for the patient.
- **Installing forcing functions or system-level firewalls:** An example of a forcing function is that concentrated

(undiluted) potassium chloride (KCl) is no longer available on hospital units.

- **Avoiding reliance on vigilance:** Because humans cannot remain vigilant for a protracted amount of time, checklists, protocols, and rechecking with another professional should be required before major procedures and before potentially dangerous medication administration. Examples are timeouts before surgery or double-checking doses on intensive insulin protocols.
- **Simplifying key processes.**
- **Standardizing key processes.**

Essential for Safety

To limit errors, critical care nurses need to consistently utilize existing checklists and standardized procedures rather than rely on memory.

Landrigan et al. (2010) undertook a study to determine whether the effort to reduce errors following publication of the IOM report had translated into significant improvements in the safety of patients. Unfortunately, despite studying institutions that had shown a high level of engagement in efforts to improve patient safety, they found that “harms remained common with little evidence of widespread improvement” (p. 2124). Most chapters of this text include safety initiatives that have been shown to enhance the safe care of patients when correctly and consistently implemented.

Reflect On

What might explain why Landrigan found so little improvement in patient safety?

There are documented ways in which healthcare providers can enhance safe, effective care and limit risks to critically ill patients. These include developing a multidisciplinary approach to patient care, encouraging a culture of safety, providing adequate staffing, and limiting work hours.

Multidisciplinary Approach to Care

Since the 1986 study by Knaus, Draper, Wagner, and Zimmerman, it has been apparent that when members of various disciplines collaborate in the care of critically ill patients, the patients have better outcomes. Evidence suggests that care should be delivered by a multidisciplinary team headed by a full-time critical care-trained physician and consisting of at least an ICU nurse, a respiratory therapist, and a pharmacist (Kim, Barnato, Angus, Fleisher,

& Kahn, 2010). Daily rounding by such a multidisciplinary team has been associated with fewer adverse drug effects, reduced duration of mechanical ventilation, and shorter ICU stay. Strategies that encourage teamwork and communication among staff members caring for critically ill patients can further improve patient outcomes (Whelan, Burchill, & Tilin, 2003).

Instituting a Culture of Safety

Benner (2001) recommends building a moral community and a culture of safety among team members. She defines a culture of safety as the “practice responsibility of all health-care team members working together in the moment to provide good healthcare” (p. 282). Sammer, Lykens, Singh, Mains, and Lackan (2010) considered how healthcare leaders might be able to determine if a “culture of safety” exists within their institutions. They determined that there were seven essential properties of a culture of safety. These included many of the elements that QSEN emphasizes: teamwork, evidenced-based practice, communication, and patient-centered care as well as a few additional elements—leadership, learning, and justice.

In a critical care unit that has embraced a culture of safety, practitioners have a responsibility to their patients to make their errors known, have them corrected, and share them with the patient, the patient’s family, and other practitioners. This sharing of information benefits the patient but ultimately benefits team members and future patients as well. When providers realize that multiple factors contribute to errors in the complex ICU environment, the focus shifts from one of “shame and blame” for errors to one of practice improvement. With practice improvement as the goal rather than punishment of the healthcare provider who committed the error, the reporting of errors results in the examination of the factors that contributed to the error and changes in practice patterns.

Henneman (2007) described a series of errors that occurred one day while she was caring for two critically ill patients. She noted that only one of the errors was a medication-dispensing error; it was the only error that was easy to identify and was reported in the traditional pattern. The remainder of the errors resulted from failures of communication or collaboration and breakdowns in the system. These errors were equally harmful to the patient as the medication-dispensing error, yet they were not readily identified as errors and were not reported. She believes that she did not report them because “I had become so accustomed to the system failures that I stopped recognizing them as such” (p. 33). If a culture of safety had been established, the breakdowns in communication and collaboration might have been identified as errors and reported. When a culture of safety is established, Henneman believes

that nurses will no longer have to “work around” system failures, and patient safety will not be jeopardized. A study by Huang et al. (2010) supports her view, finding that decreases in perception of the safety climate by ICU personnel were associated with poorer patient outcomes.

Ensuring Adequate Staffing Even the best teamwork and most competent staff will not consistently overcome inadequate staffing. Tarnow-Mordi, Hau, Warden, and Shearer (2000) demonstrated that “patients exposed to high ICU workload were more likely to die than those exposed to lower ICU workload” (p. 188). The three measures of ICU workload most closely tied to mortality in their study were peak occupancy of the ICU, average nursing requirement/occupied bed per shift, and the ratio of occupied to appropriately staffed beds. This study remains significant because it is the only published study that has related total nursing requirement, not just nurse/patient ratio, to patient outcome (Kiekkas et al., 2008). The American Association of Critical-Care Nurses (AACN) agrees that adequate staffing should not be defined as a specific nurse/patient ratio. In its report, *Standards for Establishing and Sustaining Healthy Work Environments: A Journey to Excellence* (2005), the AACN states that the basis for effective staffing is the realization that the needs of critically ill patients fluctuate repeatedly throughout their illness. Instead of mandating a fixed nurse/patient ratio, the AACN recommends instituting the following measures to ensure adequate staffing:

- The healthcare organization should have staffing policies grounded in ethical principles and support the obligation of nurses to provide quality care.
- Nurses ought to participate in all phases of the staffing process, from education to planning to assigning nurses with the appropriate competencies, to meet the needs of the patients.
- The healthcare organization should develop a plan to evaluate the effectiveness of staffing decisions and to use the data to develop more effective staffing models.
- The healthcare organization should provide support and technological services that increase the effectiveness of nursing care delivery and allow nurses to spend their time meeting the needs of the patients and those of the patients’ families.

Limiting Hours of Work The IOM recommended that nurses work no more than 60 hours each week or 12 hours in a 24-hour period (Page, 2004). In 2006, Scott, Rogers, Hwang, and Shang determined that when critical care nurses worked longer than 12 hours, the likelihood of errors and near errors increased and the nurses’ vigilance decreased. Unfortunately, in their study of 502 nurses, only

one critical care nurse left work on time every day. Most nurses rarely left work on time, even those who were working 12-hour shifts. These extended work hours increased the nurses' potential for errors. In addition, Scott et al. found that two-thirds of the nurses struggled to stay awake at least once during the 28-day study period and that 20% fell asleep. Allen et al. (2014) determined that this pattern of sleepiness and sleep deprivation was especially apparent in nurses who worked consecutive 12 hour shifts, especially consecutive night shifts. Such sleep and fatigue leads to faulty decisions and decision regret (Scott, Arslanian-Engoren and Engoren, 2014). Some states have addressed this issue by limiting the number of hours that nurses can work. Bae and Yoon (2014) found that state policies limiting mandatory overtime and consecutive work hours resulted in an 11.5% decrease in the likelihood of nurses working more than 40 hours in a week.

Essential for Safety

Nurses are aware of the number of hours they have worked in a day or week. They need to limit their work hours to 12 hours per shift and 60 hours per week to enhance patient safety.

The Role of the Critical Care Nurse

According to the American Association of Critical Care Nurses (AACN, n.d.a), "**critical care nursing** is that specialty that deals specifically with human responses to life-threatening problems. A critical care nurse is a licensed professional nurse who is responsible for ensuring that acutely and critically ill patients and their families receive optimal care." In 2013, 57% of nurses stated their primary employment position was a hospital, and 17% identified their primary nursing practice position as acute care/critical care (Budden, Zhong, Mouton, and Cimiotti, 2013). However, critical care nurses work wherever patients with potentially life-threatening problems may be found, and that includes EDs, outpatient surgery centers, and even schools.

The AACN believes that critical care nursing should be defined more by the needs of the patients and those of their families than by the environment in which care is delivered or the diagnoses of the patients. Therefore, the organization developed the Synergy Model for Patient Care based on the patient's characteristics, the nurses' competencies, and three levels of outcomes derived from the patient, the nurse, and the healthcare system. An underlying assumption of the synergy model is that optimal patient outcomes occur when the needs of the patient and his or her family align with the competencies of the nurse.

Competencies of Critical Care Nurses as Defined by the AACN in the Synergy Model

The AACN Synergy Model for Patient Care (AACN, n.d.b) describes each of the competencies of the critical care nurse on a continuum of expertise from 1 to 5, ranging from competent to expert.

Clinical Inquiry

According to the AACN's Synergy Model for Patient Care, the critical care nurse should be engaged in the "ongoing process of questioning and evaluating practice and providing informed practice." Although worded slightly differently, this competency is similar to the QSEN competencies of evidence-based practice and quality improvement. One way that critical care nurses might demonstrate clinical inquiry would be to provide care based on the best available evidence rather than on tradition. An expert critical care nurse might be able to evaluate research and develop evidence-based protocols for nursing practice in her agency, whereas a competent nurse might follow evidence based agency policies and protocols. Critical care nurses (both novice and expert) can develop the mindset that questioning practice is an issue of safety. A safe practitioner is one who wonders, "Why do we do things this way?" or "Why am I being asked to provide this specific type of care to this patient at this moment?"

Clinical Judgment

The Synergy Model (AACN, n.d.b) states that the critical care nurse should engage in "clinical reasoning which includes clinical decision-making, critical thinking, and a global grasp of the situation, coupled with nursing skills acquired through a process of integrating formal and experiential knowledge." A competent critical care nurse is able to collect and interpret basic data and then follow pathways and algorithms when providing care. She might focus on some specific aspect of care, which a more experienced nurse might recognize as less important. This nurse, when unsure about how to respond, often defers to the expertise of other nurses. An expert nurse is able to use past experience, recognize patterns of patient problems, and "see the big picture." Her previous experience coupled with the ability to see the "big picture" often allows her to anticipate possible untoward events and develop interventions to prevent them.

For example, an ED nurse received a report that a patient with stable vital signs who had a chest injury from a falling brick wall would be arriving in the ED in approximately 5 minutes. On arrival, the patient was extremely pale with new-onset chest pain. The expert ED nurse

requested the new graduate get the physician immediately while she prepared for chest tube insertion. By the time the physician arrived, the patient was displaying clear signs of a tension pneumothorax. However, the expert nurse had everything prepared for immediate chest tube insertion and decompression, and the patient recovered quickly.

Caring Practices

In its descriptions of nursing competencies, AACN defines caring behaviors as “nursing activities that create a compassionate, supportive, and therapeutic environment for patients and staff, with the aim of promoting comfort and preventing unnecessary suffering.” A caring critical care nurse can make an enormous difference in the critical care experience for a frightened patient and family. Whereas a competent nurse might focus on the basic and routine needs of the patient, an expert nurse is able to anticipate patient/family changes and needs, varying caring approach to meet their needs. For example, a son was frightened and kept leaving the bedside of his dying mother. The expert critical care nurse placed a chair at the mother’s bedside and stayed with the son, showing him how to stroke her brow gently and speak to her softly.

Advocacy and Moral Agency

The American Nurses Association (ANA) in its *Code of Ethics for Nurses* (2015) states, “The nurse promotes, advocates for, and protects the rights, health, and safety of the patient” (p.14). On its website (AACN, n.d.a), AACN states that “Foremost, the critical care nurse is a patient advocate and defines advocacy as ‘respecting and supporting the basic rights and beliefs of the critically ill patient.’” The National Council of State Boards of Nursing lists eight elements for the standard of nurse advocacy for patients. Clearly, nursing professional organizations and the nursing licensure body expect nurses to recognize that their patients may be vulnerable and may require assistance to obtain what they need from the healthcare system. However, it is sometimes difficult for nurses to advocate for their patients in the current system. Before the nurse can be an effective advocate, she needs to examine some of her own values and beliefs.

A nurse might want to consider the following:

- What types of issues (including end-of-life issues) might arise in the clinical setting for which the patient may need an advocate?
- What is owed to the patient, and what are the duties of the nurse in those circumstances?
- If she encountered one of those situations, how would the nurse be able to determine what the patient or family desires or what would be in the patient’s best interests?
- Would the nurse be able to differentiate her needs and desires from those of the patient? How certain could she be?
- How would the nurse act for her patient or empower her patient and his family to communicate their needs and desires to the rest of the healthcare team?
- How would the nurse respond if she thought that the quality of a patient’s care was being jeopardized?
- How would the nurse ensure that the discussion was a mutual exploration of concerns and not a confrontation?

According to the AACN (n.d.a), a competent nurse assesses her personal values and patient rights, represents the patient if the patient’s needs and desires are consistent with her framework, and acknowledges death as a possible outcome. However, an expert nurse advocates from the family/patient perspective, whether it is similar to or different from her own; empowers the patient and family to speak for or represent themselves; and achieves mutuality in relationships. For example, a patient and his wife wanted to withdraw interventions because the patient was clearly deteriorating and dying. However, their children, who were scattered about the country and had not seen their father during the hospitalization, were unwilling to support the decision. The nurse caring for the patient helped the wife gather the family at the patient’s bedside. Then the nurse stayed with the patient and his wife as they explained the patient’s condition and their decision to the children.

Systems Thinking

The AACN (n.d.b) in its Synergy Model defines systems thinking as managing the existing environmental and system resources for the benefit of patients and their families. For a vulnerable patient and family, being in an unfamiliar and overwhelming healthcare system can be intimidating, even frightening. Having a nurse who knows how the system works and explains it to the patient and family, or who helps the patient and family obtain what they need, can make the difference between an experience that is overpowering for the family and one that the patient and family believe they can endure. A competent nurse might see himself as a resource for the patient on the specific unit where the patient is receiving care, whereas an expert nurse might know how to negotiate and navigate for the patient throughout the healthcare system to obtain the necessary or desired care. For example, a patient with ALS requested extubation and discharge home for palliative care. His ICU nurse worked for several days with the hospice and palliative care nurses to prepare his home environment and family for his transition to care at home.

Facilitator of Learning

In the Synergy Model, AACN (n.d.b) states that nurses should be able to facilitate both informal and formal learning for patients, families, and members of the healthcare team. A competent nurse might follow planned educational programs using standardized materials or see the patient and family as passive recipients of educational materials. An expert nurse would be able to “creatively modify or develop patient/family educational programs and integrate family/patient education throughout the delivery of care.” For example, a nurse providing heart failure education realized that many of her patients who could not read would not admit that to her. She discovered that if she showed her patients clearly legible print and asked if the print was okay for them to read, the patients who could not read would readily say that the print was a problem and was too difficult to read. The nurse could then plan appropriate ways to teach her patients.

Response to Diversity

AACN (n.d.b) defines response to diversity as “sensitivity to recognize, appreciate, and incorporate diversity into the provision of care.” A similar QSEN competency is patient-centered care. A competent nurse might recognize the values of the patient but still provide care based on a standardized format. An expert nurse would anticipate the needs of the patient and family based on their cultural, spiritual, or personal values, and would tailor the delivery of care to incorporate these values.

For example, a terrified patient was being rushed to a medical center several hours from his home for an emergent mitral valve replacement. Despite the need for haste, the expert nurse realized the importance of faith to this patient and thus arranged for him to receive the Sacrament of the Sick prior to his transfer.

Collaboration

AACN (n.d.b) defines collaboration in its Synergy Model as “working with others in a way that promotes each person’s contributions toward achieving optimal and realistic patient/family goals.” The corresponding QSEN competency is teamwork and collaboration. A competent nurse might participate in multidisciplinary meetings and listen to the opinions of various team members. On the other hand, an expert nurse might facilitate the active involvement and contributions of others in meetings and role model leadership and accountability during the meetings. For example, a preceptor encouraged and assisted his orientee to present information on a complex patient with placenta accreta during multidisciplinary rounds and later during nursing grand rounds.

The Interdisciplinary Nature of Delivery of Care in Critical Care Environments

For optimum patient outcomes, critical care is delivered by a multidisciplinary team whose members trust each other and communicate and collaborate well.

Communication

In 2005, the AACN declared, “Nurses must be as proficient in communication as they are in clinical skills” (p. 190). Optimal patient care is not possible without skilled communication, and errors are frequent in situations where communication between healthcare providers and patients and their families is impaired. Rothschild et al. (2005) found that 13.7% of errors in critical care were related to problems communicating clinical information. Meanwhile, The Joint Commission (2006) determined that a breakdown in communication was the leading root factor in sentinel events between 1995 and 2004 and again in 2005. More recently, impaired verbal or written communication was identified as the cause of approximately 24% of errors in administration of parenteral drugs in ICUs (Valentin et al., 2009). Skilled communication has at least two essential components: the determination of appropriate content for the message, and the way in which the message is conveyed.

Situation Background Assessment Recommendation

The Institute for Healthcare Improvement (IHI; n.d.) currently advocates a technique borrowed from the military that it believes will improve communication among healthcare professionals. This technique, called **Situation Background Assessment Recommendation (SBAR)**, provides a process for determining what information is appropriate and delivering it in a specific manner. The IHI anticipates that using SBAR will prevent what it describes as “multiple calls to the physician when the record makes clear that the patient is deteriorating but the physician is unaware or does not understand the nurse’s statements.” On its website, the IHI provides a document titled “SBAR Report to Physician about a Critical Situation” to guide nurses’ communication. The format is:

S: Situation

- I am calling about (patient, name, location).
- The problem I am calling about is (the nurse states specifics).
- I have assessed the patient personally.

- Vital signs are:
- I am concerned about (the nurse states what the specific concern is).

B: Background

- The patient's immediate history is:
- The patient's other physical findings are (e.g., mental status).
- The patient's treatments are (e.g., oxygen therapy).

A: Assessment

- This is what I think the problem is _____.
- *Or*, I'm not sure what the problem is but the patient is deteriorating.

R: Recommendation

- I suggest (or request) that you (the nurse states the desired course of action).

Two-Challenge Rule

There are times when members of the healthcare team do not listen and respond to each other even when essential information has been communicated in an appropriate format. Sachs (2005) reported on a 38-year-old woman who experienced a critical illness and fetal demise following numerous errors in communication and planning as well as errors in judgment at Beth Israel Deaconess Medical Center. One of the strategies that the hospital adopted to prevent similar problems in the future was to train everyone to challenge other healthcare providers, even those senior to them, if they disagreed with the proposed course of action.

The two-challenge rule is a method adapted from the airline industry's crew resource management. When following the two-challenge rule, a nurse who disagrees with another healthcare provider's proposed intervention would respectfully state his concerns about the intervention twice and then would seek a superior as soon as possible and explain his concerns.

Essential for Communication

Logical, clear reporting of information and respectful presentation of opinion or disagreement enhances patient safety.

Assertive Communication

The second element of the two-challenge rule is that the nurse should present his concerns respectfully while stating his disagreement. There is mounting evidence that disrespectful and intimidating communication contributes significantly to healthcare errors. Connie Barden, past president of the AACN, believes that nurses should speak

with a bold voice. According to her, a bold voice is one that does not blame or whine, it does not argue, and it looks past complaints to work with others on solutions (Barden, 2003).

Lindeke and Sieckert (2005) stated that when a nurse needs to make a case for or against a specific action, she should be assertive but not aggressive. To do this, the nurse should question the decision calmly and directly rather than hinting. For example, "Dr. Jones, I don't understand why dobutamine is the appropriate medication for Mrs. Green. Would you explain?" The nurse also avoids using disclaimers such as "you might think differently" because that can undermine her position.

Collaboration

Evidence supports a model of critical care delivery where dedicated critical care personnel work as a team (Kim et al., 2010). Since 1986 when Knaus et al. found that outcomes in ICUs were "more related to interaction and coordination of each hospital's intensive care unit staff than to the unit's administrative structure, amount of specialized treatment use, or the hospitals' teaching status," the link between teamwork and patient outcomes in ICUs has been repeatedly reaffirmed. For example, Boev and Xia (2015) demonstrated good nurse-physician collaboration resulted in a decrease in infections acquired during critical care hospitalization. Whelan et al. (2003) concluded that the "evidence is sufficient to warrant the implementation of policies designed to improve the level of communication and collaboration among staff members in intensive care units" (p. 527).

Collaboration is a process, not a single event, and it requires that members of the healthcare team develop a pattern of sharing knowledge and responsibility for patient care. A number of characteristics influence the degree of collaboration that occurs among members of a healthcare team. These characteristics are discussed in the sections that follow.

Development of Emotional Maturity. According to Lindeke and Sieckert (2005), emotional maturity is foundational to collaboration because of the positive individual attributes of people who are emotionally mature. These include being lifelong learners, actively identifying best practices, and keeping their skills current. Emotionally mature people are positive, humble, and willing to take responsibility for their failures and try again.

Essential for Collaboration

Having the emotional maturity to pause and reflect on one's own motivations as well as those of others can lead to improved communication and collaboration (Rushton, 2009).

Understanding the Perspective of Others. There is evidence that members of the differing health professions have different perspectives on what ought to be communicated as well as what ought to be the goals of patient care. For example, critical care nurses and physicians see the subsets and phases of the illnesses of the same groups of patients differently. The nurse may see only the sickest of a group of patients, whereby only half of the patients survive to hospital discharge. These patients may be severely debilitated at the time of discharge, and the nurse may worry about their ability to survive. The physician or home health nurse, in contrast, may see the same subset of patients on a longer term basis and realize how infrequently they need hospitalization and how well they do afterward. Thus, the physician may realize that the actual outcomes of care are better than the hospital nurse envies them to be (Shannon, 1997).

When the prognosis is unclear, there is some indication that nurses and physicians use different types of cues to determine what they believe will be the outcome. According to Anspach (1987), physicians are more likely to rely on technological cues, such as lab tests or findings from a physical examination, to determine prognosis, whereas nurses tend to attach more significance to interactive cues, such as when a patient appears depressed, cannot be comforted, or appears to be suffering. Anspach suggests that nurses' reliance on the interactive cues is related to the time they spend caring for and interacting with their patients. There is currently no indication that one type of cue is more likely to predict a patient's outcome than the other is.

These differences in viewpoints can make collaboration more difficult. However, if healthcare providers are willing to pause and listen to each other, as Rushton (2009) suggests, a holistic view of the patient may be developed.

Building the Team

Important to team functioning is the identification of a common goal. In healthcare that goal is usually patient well-being. Unfortunately, when a patient is critically ill, team members may disagree on what constitutes patient well-being and thus what is in the patient's best interests. Critical care units that have daily multidisciplinary rounds and dialogue about patient prognosis, and establishment of patient goals have been demonstrated to have better team functioning and patient outcomes (Rothen et al., 2007). Nurses have an important role on the multidisciplinary team in helping the team to recognize that the overall goal is related to the patient's values and quality-of-life preferences as well as assisting in the establishment of the daily patient goals and the treatment plan (Martin & Koesel, 2010). Daily goals tools to enhance communication, collaboration, and patient safety are readily available (Siegle, 2009).

An approach advocated by Agency for Healthcare Research and Quality (AHRQ; n.d.) to enhance communication and collaboration is TeamSTEPPS, which includes the brief, huddle, and debrief.

- Briefs are established ways to engage in planning. They are usually conducted at the beginning of a shift and include all who will be on the unit during the shift. During the brief, the team is formed, roles are designated, and goals are set. Topics during a brief might also include patient transfers, discharges, and admissions; patients who are unstable, are dying, or have specific needs; and families with specific issues.
- Huddles are for problem solving and may be called by anyone whenever needed. They are used so that a team member may regain situation awareness or to discuss critical issues, assign resources, and express concerns.
- Debriefs are informal discussions that occur after an event or a shift designed to improve team functioning and patient outcomes. They include a discussion of the event, an analysis of why it occurred, and a discussion of what the team might do differently the next time.

Negotiating Respectfully

Nurses encounter a variety of barriers when they attempt to negotiate. The most important one may be that power is often unbalanced in healthcare negotiations. Lindeke and Sieckert (2005) stated that nurses should therefore contribute to teams from positions of strength by being innovative and by demonstrating integrity in collaboration. In contrast, Gardner (2004) stated that to achieve collaboration there must be some form of power sharing. She identified the informal power bases of information, expertise, and goodwill and recommended that nurses ask for opinions from the quiet, less-verbal members of a group as a means of demonstrating goodwill and sharing power.

Managing Conflict Wisely

Encountering conflict can be stressful, but acknowledging it and managing it well is the cornerstone to successful collaboration (Gardner, 2004). Furthermore, when nurses embrace conflict and engage in it assertively yet respectfully, there is an opportunity for growth and innovation in clinical practice. According to Gardner, when encountering conflict the nurse must distinguish between emotional conflict, which arises from relationships, and task conflict, which centers on judgments concerning the best way to achieve an agreed-upon goal. Task conflict, which is easier to manage and is often healthy, can usually be resolved with a discussion of the risks and benefits of each approach. When emotional conflict develops, the nurse manager may need to redirect concerns away from the issue and encourage the disputants to resolve the issue in private.

Ethical and Legal Issues in the Delivery of Critical Care

Nurses who take care of critically ill patients often encounter situations in which there is disagreement among the healthcare team, the patient, and the patient's family about how to proceed. In such circumstances, it is important that the nurse know the legal issues involved as well as how to engage in ethical discourse and decision-making.

Ethical Dilemmas

Ethical decision-making in critical care takes time, an understanding of the language and concepts of ethics, and an ability to make appropriate distinctions (Perrin and McGhee, 2007). It requires an appreciation of what it is to be human and of the successes and limits of medicine. In addition, decision-making in nursing ethics takes an ability to communicate with people who are in distress, an awareness of cultural and religious values, and the ability to compromise. Because nurses are intimately involved with patients and families, they may have strong feelings or beliefs about what should be done. Perrin and McGhee believe it is important that nurses learn to translate these feelings and beliefs into ethical discourse so that they can participate in the discussion of what ought to be done for their patients. Rushton (2009) states that nurses should utilize questions to help first themselves and then all the participants to see the situation in a common light and envision new possibilities. Nurses are often the people who carry out the chosen interventions, so they ought to believe that morally justifiable resolutions to the dilemmas have been identified.

When Is a Situation an Ethical Dilemma?

When a nurse encounters a situation that makes her wonder, "Is this what I ought to be doing to provide care for this patient?" she should explore whether it is an **ethical dilemma**. Ethical dilemmas may exist when there is a conflict between the duties, rights, or values of the people involved in the situation. They may occur when those involved believe that different principles ought to motivate their behavior, or when they believe that considerations of the consequences of their actions should drive their decision-making. An ethical dilemma might be defined as a situation that gives rise to conflicting moral claims, resulting in disagreements about choices for action. A cue that a nurse is dealing with an ethical dilemma is the language used to describe the situation. Ethical dilemmas are usually described in terms of right or wrong, duty or obligation, rights or responsibilities, and good or bad. Ethical dilemmas are commonly identified by the question, "What should be done?" Critical care nurses identify some patient situations

as particularly likely to be high risk for ethical dilemmas, such as when patients with poor prognosis are receiving aggressive treatments, when patients' advance directives are violated, and when distressed family members are in disagreement with the plan of care recommended by the healthcare team (Pavlish, Hellyer, Brown-Saltzman, Miers, & Squire, 2015).

What Information is Necessary to Make an Informed Decision?

Once it seems apparent that an ethical dilemma exists, the first essential step is the identification of significant information. Without a clear understanding of the particulars of the situation, the nurse will not be able to fully understand the dilemma or choose an action wisely.

It is important that the nurse understand the patient's medical condition. In order to limit potential confusion, it is helpful if the person, family, and all healthcare providers share an understanding of both the patient's disease state and the goals for treatment. Disagreement about a patient's prognosis, disease progression, and likely outcome is frequently the reason that healthcare providers, the patient, and family members are unable to agree on a treatment plan. Rushton (2009) recommends such simple questions as "What are the facts?" and "What's the big picture?"

Ethical Frameworks That Help Nurses to Understand and Resolve Dilemmas

Utilizing ethical frameworks or perspectives is rather like using a filter: it helps the nurse sort the material and identify what information is important when making her decision. It also assists in identifying appropriate alternatives for action. It is important that the nurse recognize what ethical reasoning system she is using and identify the ethical viewpoints of those involved in the dilemma (Perrin & McGhee, 2007).

All Things Considered, Determining What Ought to Be Done

How should one make a decision and choose a course of action? Most people will state that making any decision should involve clear thinking and consideration of the implications of each of the alternatives. When preparing to make a decision, the nurse might ask herself the following questions:

- What ethically justified goals can be identified?
- What are the ethically justified alternatives for action or what are the choices?
- Are there practical constraints to following any of them?

- What arguments can be constructed in favor of these alternatives (this includes considering the probable consequences)?
- How can these arguments be evaluated?
- What ought to be done?

When the nurse has made a decision she feels prepared to defend, presenting the decision to healthcare colleagues will allow them to evaluate the decision. This publicity and scrutiny will help to assure all those involved of the soundness of the decision. Besides, most healthcare decision-making is a group process.

Nurses may be represented in the group decision-making process in several ways. First, they may represent their ethical perspective on the situation to a multidisciplinary meeting. For example, many nurses bring complex situations to the ethics committees of the hospitals where they practice. More commonly, nurses serve as the intermediary between patients, families, and healthcare providers, helping each group to understand the concerns of the others. Nurses often translate the ethical perspectives of their patients for other healthcare providers and clarify what the providers are saying to patients and families. Less frequently, nurses may act independently on their own moral decisions.

Issues with Both Legal and Ethical Aspects

Critical care nurses regularly encounter a variety of issues that have both ethical and legal implications. Some of the issues are discussed briefly in this chapter, and others, such as those concerning end of life, are discussed later in the text. In either case, the content in this text serves merely as an introduction to these complex issues.

Informed Consent

Obtaining **informed consent** has both legal and ethical ramifications. When a patient gives consent, he agrees to the suggested treatment or procedure. Legally, if a nurse treats or touches a patient without consent, it is considered battery, even if the treatment is appropriate and has no negative effects. Consent is usually implied rather than written for “routine” procedures like turning, dressing changes, or most medication administration. However, even if the nurse does not ask the patient for consent, the nurse should explain the procedure. Consent is also implied when a person goes to an ED acutely ill and unresponsive. However, written informed consent should always be obtained before complex procedures such as invasive and surgical procedures, blood administration, and chemotherapy, which might have serious side effects or consequences for the patient.

Informed consent has three principal components. The decision to permit the treatment or procedure:

- must be made voluntarily,
- must be made by a competent adult, and
- the patient must understand his condition and the possible treatments.

In other words, the patient’s decision must be an autonomous choice, not coerced or manipulated by healthcare providers or family members. In addition, the patient must be capable of rational thought and be able to recognize what the prospective treatment involves.

It is common for healthcare providers and family members to question the decision-making ability of critically ill patients. Many are intubated and unable to communicate even their most basic needs clearly. Other patients may be experiencing pain or are depressed and not capable of thinking clearly. Whenever possible, patients should be allowed to participate in the consent process, and it has been shown that they remember and value the participation (Sims, 2008). However, many critically ill patients lack the capacity to give informed consent. A determination of incapacity does not require a legal proceeding; it is a clinical judgment that can be assessed during conversation with the patient. To determine capacity, the nurse might ask the following questions (Chell, 1998):

- Does the patient understand her medical condition?
- Does the patient understand the options and the consequences of her decision?
- In addition, if the patient is refusing to consent to recommended medical treatment, is the refusal based on rational reasons?

A loss of capacity may be temporary—for example, when a patient has been experiencing pain or is heavily medicated. During the period of incapacity, a surrogate healthcare decision maker might be requested to consent to treatment for the patient. Some states allow next of kin to make decisions when a patient is incapacitated. Others require that a decision maker, known as a healthcare proxy or durable power of attorney for healthcare purposes, be designated by the patient in an advance directive or appointed by the courts. Often the patient may be critically ill with sepsis and organ failure so the care that the surrogate decision maker is asked to consent to may determine if the patient lives or dies.

If a surrogate decision maker is having difficulty determining what ought to be done, the nurse might explain that there are two common ethically accepted modes for making surrogate decisions. The first is the best interest standard in which the decision maker decides what he believes is in the best interests of the patient. For example, following his wife’s stroke a husband might decide that she would

consent to administration of rTPA because it would be in her best interests to have minimal neurological impairment. The second method is substituted judgment in which the surrogate decides what he thinks the patient would have decided had she been able to make the decision. For example, a husband might realize that his wife had been in atrial fibrillation and heart failure prior to her stroke and had been saying for months that she could not go on any more. He might decide that she would not have consented to the treatment, so he would make that decision for her.

Whether it is the patient or a surrogate who is making the decision, it is essential that the decision maker have adequate information about the possible treatment(s) to make a decision. Although the nurse does not usually provide the initial information for the informed consent, it is the nurse who consistently reinforces the teaching and ensures that the decision maker understands

- the nature and purpose of the proposed treatment or procedure,
- the expected outcome and likelihood of success,
- the likely risks involved,
- the alternatives to treatment, and
- the risks if no treatment is selected.

Assisting a critically ill patient or the patient's surrogate to make a decision is not easy. Although the previous list makes it appear that precisely what information ought to be provided is clear, that is often not the case. As Rosenbaum (2015) notes, it is possible to provide too much information to the decision maker leading to cognitive overload and a poor decision. Patients/surrogates may learn of the short-term benefits of one mode of treatment and favor that over another that would have longer term gains. How the healthcare team shades the information can greatly affect the decision maker. Finally, when the patient is critically ill, the decision maker may be in crisis, emotionally overwhelmed and unable to process information. It is imperative that the nurse calm the decision maker, then explain the information repeatedly and listen to determine if it has been understood. Conflict is more likely to develop when healthcare providers, patients and families, or decision makers do not have a common understanding of the proposed treatment(s), or when they disagree about what the patient would have wanted or what is in the patient's best interests.

Reflect On

Have you cared for a patient you believed was undergoing or about to undergo a procedure to which he would not consent? How did you respond? What was the result? How would you respond if a similar situation arose in the future? Why?

End-of-Life Issues

There are a number of ethical/legal concerns that surround end-of-life care for the critically ill patient. These include advance directives, limitation of therapy, withdrawal of therapy, euthanasia, and organ donation from deceased donors and are discussed later in the text.

Issues Related to Organ Transplantation Recipients and Living Donors

As the need for organs for transplant expands, there are concerns about how organs should be allocated and who an appropriate living donor is. These issues are discussed in Chapter 16 .

Use of Restraints

In the past, it was common practice in the United States to physically restrain confused or frail patients to prevent them from harming themselves. Nurses' primary motivation in restraining these patients was beneficence, acting to prevent patients from the harm they might incur if they tried to pull out an endotracheal tube or IV, move around in bed unassisted, or inadvertently disconnect ventilator tubing. However, ethicists have been deeply concerned about the practice. They believe that the principle of nonmaleficence (or not harming a patient) should always take precedence over beneficence (attempting to do good for a patient). As researchers studied restraints, nurses learned that restraining a patient causes physical harm. When people are restrained, they are more likely to become weak, incontinent, constipated, and to develop nosocomial infections. They also recover more slowly. Thus, there is reason to believe that in acting to prevent a potential harm, such as the disconnection of an IV, the nurses may be causing actual physical harm as well as moral harm, such as the deprivation of the patients' autonomy.

However, critically ill patients are not fully aware. According to Bray et al. (2004), there is variability in what patients recall from the experience of being restrained, ranging from patients who remember very little of the experience to patients who describe the event as extremely unpleasant, even frightening. Critically ill patients often perceive the discomfort from an endotracheal tube but lack the capacity to realize its purpose and its necessity. Thus, they are likely to reach up and remove objects that cause them discomfort. How should critical care nurses respond? What is the appropriate balance between keeping a patient safe but possibly leaving them with frightening memories and allowing them freedom of movement? Maccioli et al. (2003) list the following recommendations developed by the American College of Critical Care Medicine Task Force for the use of restraining therapies:

- Institutions and practitioners should strive to create the least restrictive but safest environment for patients regarding restraint use.

- Restraining therapies should be used only in clinically appropriate situations and not as a routine component of therapy. When restraints are used, the risk of untoward events must outweigh the physical, psychological, and ethical risks of their use.
- Patients must always be evaluated to determine whether treatment of an existing problem would obviate the need for restraint use.
- The choice of restraint should always be the least invasive option capable of optimizing patient safety, comfort, and dignity.
- The rationale for restraint use must be documented in the medical record. Orders for a restraining order should be limited to a 24-hour period. New orders should be written after 24 hours if the restraining orders are to be continued.
- Patients should be monitored for development of complications from restraining therapies every 4 hours, more frequently if they are agitated.
- Patients and their significant others should receive ongoing education as to the need and nature of restraining therapies.

It is nurses who are left to try to balance their patients' safety needs against their legal and moral rights to be free from physical restraint. This is clearly a difficult balance since more than 80% of patients who self-extubate are restrained (Chang, Wang, and Chao, 2008; Curry, Cobb, Kutash, and Diggs, 2008). Cho, Kim, Kim, and Choi (2006) determined that the main factors in the nurses' decisions to use restraints were the Glasgow Coma Scale score, restless behavior, emotional state, discomfort factors, medical devices, and life-sustaining devices. In their study, 31% of patients were restrained, with more patients being restrained during the night than during the day. Martin and Mathisen (2005) compared the use of restraints in the United States with that in Norway and found dramatic differences. Restraints were used in 40% of patients in the United States, whereas none of the Norwegian patients were restrained. Seven incidents of unplanned removal of an invasive device (either IV or nasogastric tube) occurred, all in restrained patients in the United States.

There were other differences between the care of patients in the two countries. The nurse/patient ratio for the Norwegian sample was 1.05:1, whereas the ratio for the U.S. sample was 0.65:1. In Norway, nursing stations are decentralized and a "norm has prevailed for nurses to remain within a distance that allows for direct visual observation and eye contact with intubated patients, both to avoid isolation and to be alert for behavioral changes" (Martin & Mathisen, 2005, p. 139). However, the Norwegian patients received more sedation and pain medication than the patients in the United States.

This is concerning because patients who are more sedated have been demonstrated to spend more time on a ventilator, in the ICU, and in the hospital.

Cho et al. (2006) concluded and Chang et al. (2008) reaffirmed that better guidelines on the use of restraints should be developed, taught, and followed. Until then, nurses need to make the decision to restrain patients carefully, obtain consent if possible, follow available guidelines, and be diligent in their efforts to prevent untoward effects.

Reflect On

What factors influence your decision to restrain your patients?

Legal Issues

The Nurse Practice Act, enacted by the state legislature, defines the practice of nursing in each state and delegates the powers of enforcement for the act to the state Board of Nursing. The Board of Nursing has responsibility for interpreting and implementing the act in each state. Because the act varies from state to state, the critical care nurse needs to be cognizant of the rules and regulations for the practice of nursing of the state in which she is practicing. The easiest way to locate and review the Nurse Practice Act as well as the rules and regulations for any state is to check the Board of Nursing website. Most have the information readily available. In order to prevent potential legal problems, it is essential that the nurse know what functions are within the bounds of the practice of professional nursing as defined by the Nurse Practice Act. In addition, the nurse must have the education and experience to competently perform those functions and must perform them in congruence with the policies of the employing institution.

There are two major areas of law: criminal law and civil law. Criminal law involves situations whereby the local, state, or federal government has filed a suit against a nurse. Fortunately, these suits are rare for nurses, but they do occur. The most common types of criminal cases are criminal assault and battery, criminal negligence, and murder. The most common types of suits filed against nurses are civil suits involving tort law. Tort law concerns a wrong committed against a person or the person's property. Negligence and assault and battery are examples of torts. When nurses obtain consents from patients or proxies appropriately, before providing care or treatments, they protect themselves against charges of assault and battery.

Negligence

The most common reason for lawsuits against nurses is **negligence**. However, because the nurse is a medical

professional, the trend is to call it “malpractice.” Critical care nurses are held to the same criteria for negligence or malpractice as any other healthcare provider. In order to prove negligence, all of the following must be demonstrated:

- A duty was owed—A legal duty exists whenever a hospital or healthcare provider undertakes the care or treatment of a patient.
- The duty was breached—The provider failed to provide care in accordance with the existing, relevant standard of care. The standard of care can be proven by producing an existing standard, an expert testimony, or by obvious error (the thing speaks for itself).
- The breach of the duty was the proximate cause of an injury to the patient.
- Damages—Without harm to the patient (losses that need not be physical or financial but may also be

emotional), there is no basis for a claim, regardless of whether or not the medical provider was negligent.

Although an inadvertent error can result in negligence, the National Council of State Boards of Nursing has built a taxonomy that differentiates between errors due to system problems and those that occur from willful negligence or intentional misconduct (Day, 2010).

Standards of Care

Nurses are held accountable for practicing in conjunction with the applicable standards of care. This means that critical care nurses should be acquainted with the appropriate standards. There are standards developed by a variety of professional bodies. Perhaps the most appropriate for the critical care nurse are those propagated by the National Council of State Boards of Nursing and the AACN. The standards developed by the AACN are displayed in Box 1-1.

Box 1-1 Standards of Care for Acute and Critical Care Nursing

Standard of Care I: Assessment

The nurse caring for acute and critically ill patients collects relevant patient health data.

Standard of Care II: Diagnosis

The nurse caring for acute and critically ill patients analyzes the assessment data in determining diagnoses.

Standard of Care III: Outcome Identification

The nurse caring for acute and critically ill patients identifies individualized expected outcomes for the patient.

Standard of Care IV: Planning

The nurse caring for acute and critically ill patients develops a plan of care that prescribes interventions to attain expected outcomes.

Standard of Care V: Implementation

The nurse caring for acute and critically ill patients implements interventions identified in the plan of care.

Standard of Care VI: Evaluation

The nurse caring for acute and critically ill patients evaluates the patients' progress toward attaining expected outcomes.

Standard of Professional Practice I: Quality of Care

The nurse caring for acute and critically ill patients systematically evaluates the quality and effectiveness of nursing practice.

Standard of Professional Practice II: Individual Practice Evaluation

The practice of the nurse caring for acute and critically ill patients reflects knowledge of current professional practice standards, laws, and regulations.

Standard of Professional Practice III: Education

The nurse acquires and maintains current knowledge and competency in the care of acute and critically ill patients.

Standard of Professional Practice IV: Collegiality

The nurse caring for acute and critically ill patients interacts with and contributes to the professional development of peers and other healthcare providers as colleagues.

Standard of Professional Practice V: Ethics

The nurse's decisions and actions on behalf of acute and critically ill patients are determined in an ethical manner.

Standard of Professional Practice VI: Collaboration

The nurse caring for acute and critically ill patients collaborates with the team, consisting of patient, family, and healthcare providers in providing patient care in a healing, humane, and caring environment.

Standard of Professional Practice VII: Research

The nurse caring for acute and critically ill patients uses clinical inquiry in practice.

Standard of Professional Practice VIII: Resource Utilization

The nurse caring for acute and critically ill patients considers factors related to safety, effectiveness, and cost in planning and delivering patient care.

The Standards for Acute and Critical Care Nursing Practice resource is a product and the expanded version is available through the AACN Online Bookstore.

Factors Affecting the Well-Being of Critical Care Nurses

Critical care nurses are often placed in situations with higher levels of complexity, uncertainty, and decisional authority than other nurses. Although many critical care nurses derive satisfaction from working in these circumstances, others have the potential of developing moral distress or compassion fatigue. Nurses can utilize specific strategies to enhance their satisfaction with their role.

Moral Distress

In 1984, Jameton described a circumstance that he called **moral distress**, wherein a nurse would know the right thing to do, yet institutional constraints such as lack of resources or personal authority would prevent her from doing it. Jameton believed that the distress nurses experienced was serious because they were involved in situations that they judged were morally wrong. This state of affairs has not diminished with the passage of years. In 2006, Lutzen, Dahlquist, Sriksson, and Norberg found that nurses facing competing or contradictory moral imperatives felt burdened with a troubled conscience. In 2005, Elpern and colleagues learned that critical care nurses commonly encounter situations that are associated with high levels of moral distress. The primary source of moral distress for nurses in their study was providing aggressive care to patients whom the nurses did not believe would benefit from the care. Morris and Dracup (2008) identified the inability to achieve appropriate pain control or relief of other patient-specific symptom and aggressive treatment of dying patients as the major causes of moral distress for healthcare providers. Nurses consistently state that when they do not have a voice in the decision-making, they feel powerless and that they cannot find meaning in the patients' or families' suffering.

The AACN believes there is evidence that the moral distress experienced by critical care nurses has a substantial impact on healthcare. Whitehead, Herbertson, Hamric, Epstein, & Fisher (2015) found that critical care nurses had higher levels of moral distress and were more likely to report an intention to leave their current position than nurses working in other types of units. In addition, nurses who experience moral distress may lose the capacity to care for their patients and experience psychological and physiological problems.

To respond to this concern, the AACN (2004) developed a public policy statement on moral distress, and the Ethics Work Group of the AACN (2006) developed a framework called *Ask-Affirm-Assess-Act: The 4 A's to Rise Above Moral Distress*, the value of which was reaffirmed in 2015 by Savel and Munro. The steps of the four As are discussed in the next sections.

Ask: The nurse asks, "Am I or are members of my team feeling symptoms or showing signs of suffering?" or "Have others noticed these symptoms and behaviors in me?"

Affirm: The nurse recognizes that moral distress is present and makes a commitment to take care of herself, validate her perceptions, and affirm her professional responsibility to act.

Assess: The nurse identifies the sources of her distress by clarifying the circumstances under which the distress occurs. Is it a particular patient care situation? Is it a unit policy or practice? Does it result from lack of collaboration? The nurse next determines the severity of the distress, her readiness to act, and the risks and benefits of any action.

Act: Before acting, the nurse needs to develop an action plan including a self-care plan, a list of sources of support, and possibilities for outside sources of guidance and assistance. Finally, the nurse needs to take actions that will address the specific sources of distress within her work environment using polite but assertive communication. According to the AACN Ethics Work Group, the nurse's goal is to preserve her authenticity and integrity.

Gutierrez (2005) also suggested approaches that nurses could utilize on their unit or at the institutional level to respond to moral distress. These include:

- Improving communication between patients, families, and healthcare providers
- Improving communication between nursing staff and managers
- Providing support to families in their coping
- Developing a forum for ethical discussion
- Promoting moral and ethical dialogue between nursing and medical students
- Facilitating clinical practice guidelines on futility, ethical decision-making, and palliative care

Conscientious Refusal

When all else fails, if a nurse believes that he cannot ethically perform an action he is being asked to perform, he may utilize conscientious refusal and ask to be excused from participating in or assisting with the action. In such a situation, the supporters of the action can offer a justification for the action and state that it is legal, but the nurse does not find their reasoning convincing, believes the action is morally wrong and thinks he would experience moral distress if he participated in it. According to Benjamin and Curtis (1998), a thoughtful nurse would identify his conscientious refusal to participate:

- Based on personal moral standards
- As determined by a prior judgment of rightness or wrongness
- As motivated by personal sanction and not external control

According to Weinstock (2013), the ability to present a moral reason for the objection in a way that is

understandable to the healthcare team is the essential component of conscientious refusal.

Conscientious refusal is not an option a nurse should choose without very careful consideration. If the patient and family have developed a relationship with the nurse, they may wish the nurse to remain with them beyond the decision-making phase to see the planned action accomplished and to help them cope with the consequences of their decision. On the one hand, the nurse must consider the effect that disrupting the nurse-patient relationship will have on the patient and family. On the other hand, the nurse must consider what effect his disapproval of the planned action will have on his ability to deliver quality nursing care to this patient and subsequent similar patients. The nurse must also consider the amount of support he will receive from the administration of the institution. The repercussions for the nurse of employing conscientious refusal may range from nonexistent to dismissal from his nursing position. Institutions vary from being supportive of conscientious refusal and changing their institutional policies to support it, to being legally required by some states to allow nurses to utilize it, to being able to dismiss the nurse who utilizes it.

Compassion Satisfaction/Fatigue

In 1992, Joinson identified the concept of **compassion fatigue** in nurses. In *Compassion Fatigue: An Expert Interview with Charles R. Figley* (Medscape, 2005), Figley further defined the concept and expanded it to others who provide care to suffering individuals. In a review of the literature, Sabo (2011) determined that nurses working in intensive care have been shown to be particularly vulnerable to compassion fatigue. According to Figley, compassion fatigue is a “state of tension and preoccupation with the suffering of those being helped that is traumatizing for the helper.” It occurs in care providers who may be so selfless and compassionate that they fail to pay sufficient attention to their own needs.

The terminology for compassion fatigue has evolved. What was once called *compassion fatigue* was renamed *secondary traumatic stress*, while *compassion fatigue* continues to be used to describe the combined effect of secondary traumatic stress and burnout. Secondary traumatic stress differs from burnout in that it may develop suddenly in response to a specific incident of suffering, whereas burnout tends to develop slowly and insidiously in response to various stressors. Secondary traumatic stress is primarily a response to caring for people who are suffering, whereas burnout is often a response to other stressors such as poor morale in the work environment. Critical care nurses ages 20 to 29 are more likely to experience high levels of secondary traumatic stress, while nurses between the ages of 40 and 49 experience significantly higher levels of burnout (Sacco, Ciurzynski, Harvey, & Ingersoll, 2015). Scales such as the Professional Quality of Life Screening (ProQOL) have been developed to allow healers to reflect on their standings on these characteristics.

Symptoms

Caregivers who are experiencing compassion fatigue have many symptoms that often parallel the symptoms of the suffering patients with whom they are working. Some of the symptoms of compassion fatigue include:

- Intrusive thoughts or images of patients’ situations or traumas
- Difficulty separating work life from personal life
- Lowered tolerance for frustration and/or outbursts of anger or rage
- Dread of working with certain patients
- Depression
- Increase in ineffective and/or self-destructive self-soothing behaviors
- Hypervigilance
- Decreased functioning in nonprofessional situations
- Loss of hope

Sabo (2011) describes several concerns about Figley’s model of compassion fatigue. Of particular importance, she notes that the concept of empathy in Figley’s model is not well developed. She wonders if nurses’ ability to empathize may help them to ward off compassion fatigue rather than be more deeply affected and transported toward compassion fatigue. Also, she believes that Figley’s model does not address how to halt compassion fatigue’s process. She believes that the ways in which resilience, hope, and self-help can ward off compassion fatigue need to be further explored.

Nursing professional organizations are realizing how important it is for nurses to care for themselves in order to provide optimal care to their patients. Beginning with the revision of the Code of Ethics for Nurses in 2001 and continuing through the most recent revision (ANA, 2015), the code has included a provision that states nurses have duties to themselves as well as to others. These duties include the promotion of personal health and safety, preservation of integrity, maintenance of competence, and a commitment to personal and professional growth. A variety of self-care practices exist that critical care nurses should employ primarily for their own health and well-being, but also because such practices may aid in preventing compassion fatigue.

Standards of Self-Care

The Academy of Traumatology/Green Cross has proposed standards of self-care for caregivers. These standards were described by Figley in an interview with Medscape in 2005. The purpose of the guidelines is twofold: first, to ensure that practitioners do no harm to themselves when helping or treating others; and second, to encourage providers to attend to their own physical, social, emotional, and spiritual needs as a way of ensuring high-quality services to those who look to them for support as a human being (Medscape, 2005).

Included in the proposed guidelines are sections on establishing and maintaining wellness and an inventory of self-care practices. The following are selections from the standard for establishing and maintaining wellness:

- Make a commitment to self-care.
- Develop strategies for letting go of work.
- Develop strategies for acquiring adequate rest and relaxation.
- Plan strategies for practicing effective daily stress reduction.

Next, the proposed standards identify specific ways in which helpers ought to inventory their self-care practices and provide self-care. These are divided into physical, psychological, social/interpersonal, and professional strategies. The standards suggest that caregivers inventory themselves on each of these criteria then develop a prevention plan by selecting one goal from each category and implementing behavioral changes.

Caregivers who are experiencing compassion fatigue may have chosen to give up sleep to continue to care for the suffering person or may have engaged in inappropriate self-soothing behaviors such as misuse of alcohol and drugs and either excessive or inadequate intake of nourishment. Assessment and behavioral change for physical well-being, therefore, is particularly important.

Strategies for enhancing physical well-being include:

- Monitoring all parts of the body for tension and utilizing appropriate techniques to reduce tension
- Utilizing healthy methods that induce sleep and return to sleep
- Monitoring all food and drink intake with an awareness of their implication for health and functioning

Strategies for enhancing psychological well-being include:

- Sustaining a balance between work and play
- Developing effective relaxation methods
- Maintaining contact with nature or other soothing stimuli
- Developing methods of effective creative self-expression
- Maintaining effective skills for ongoing self-care such as:
 - Assertiveness
 - Stress reduction
 - Interpersonal communication skills
 - Cognitive restructuring
 - Time management

Strategies for enhancing social/interpersonal well-being include:

- Identifying at least five people (a minimum of two at work) who will be highly supportive when called on to deliver help and will respond quickly and effectively
- Knowing when and how to secure help both personally and professionally
- Being involved in addressing and preventing moral harm

Strategies for enhancing professional well-being include:

- Balancing work and home responsibilities—devoting sufficient time to each without compromising the other
- Establishing boundaries and setting limits concerning:
 - Overworking
 - Therapeutic/professional boundaries
 - Personal boundaries
 - Realism when differentiating between things that one can change and accepting things that one cannot change
- Obtaining support at work from peers, supervisors, and mentors
- Generating work satisfaction by noticing and remembering the joys and achievements of the work

Reflect On

How do you balance your personal and professional life?

Job Satisfaction

There is evidence that nurses who work with critically ill patients do so because they obtain satisfaction from the type of care they provide. Tummers, van Merode, and Landeweerd (2002) compared the work characteristics and psychological work reactions of nurses employed in critical care with those of nurses employed in nonintensive care nursing. They found that ICU nurses reported significantly higher levels of complexity, uncertainty, and decision authority than non-ICU nurses. But although Tummers et al. had anticipated that ICU nurses would experience higher levels of emotional exhaustion in response to these challenges, their study showed that the ICU nurses reported lower levels of exhaustion compared with their non-ICU counterparts. Le Blanc, de Jonge, de Rijk, and Schaufeli (2001) noted that although ICU nurses identified providing nursing and medical care as being very demanding, it also “drove their satisfaction.” In 2013, 87% of critical care

nurses responding to a survey agreed with the statement, “Overall, I am satisfied with my choice of nursing as a career” (AMN Healthcare, 2013).

Ulrich, Woods, Hart, Lavandero, Leggert, and Taylor, 2007 (2007) examined how collaboration, communication, leadership, and support for nurses’ professional growth related to nurses’ satisfaction with their critical care

nursing career. They concluded that nurses working in critical care units striving for excellence (as identified by a Beacon Award or Magnet status application or designation) reported healthier work environments and higher job satisfaction. Both healthier work environments for nurses and higher nursing job satisfaction have been associated with better patient outcomes.

Critical Care Summary

Critical care nurses are an essential part of the multidisciplinary team providing care to patients with life-threatening problems. Although the critical care environment is

stressful, the majority of critical care nurses experience satisfaction from the clinical competencies they possess and the care they are able to provide to their patients.

Why/Why Not?

A patient with an ischemic foot was medicated with 50 mg of IV Fentanyl 20 minutes ago and is sleepy. The surgeon arrives, explains to the patient the revascularization procedure planned and the potential for amputation of toes or possibly the foot to the patient then states that the surgery is urgent. The patient signs

the consent form with an X and returns to sleep without asking a question. Should the nurse sign as a witness to the consent form?

See answers to Why/Why Not? in the Answer Section.

Case Study

Allen Hale, 27 years old, was admitted to ICU after running a red light in his car and colliding with another car. He is being evaluated for evacuation of a left parietal subdural hematoma. His concomitant injuries include a flail chest with pulmonary contusions for which he is being ventilated and a fractured femur. His blood alcohol level was 200 mg/dL on admission, and there was evidence of marijuana on his toxicology screen. Currently, he is receiving propofol 30 mcg/kg/minute for sedation. He responds to noxious stimuli by withdrawal, and his pupils are equal and reactive to light.

The healthcare team is unable to obtain consent for treatment from him and is unable to reach a next of kin or anyone with healthcare proxy.

1. How should they proceed?

Two weeks later, Mr. Hale’s is delirious and hallucinating. The physicians have determined that he should have

a tracheostomy and a gastrostomy. The social worker has learned that Mr. Hale is not from the state, that he does not have a primary care provider, and that he has only one living relative—a sister he has not seen in 5 years. In this state, the next of kin may serve as a proxy decision maker. Mr. Hale’s sister is having a difficult time determining if she should give permission for the surgery.

2. How can the nurse help her to decide?

During the third week of Mr. Hale’s hospitalization, Angela Gibbons is assigned to care for him. For the previous 2 weeks, Angela has been providing care to the only person in the other car who survived the crash. Angela believes that she should not be required to care for Allen at this time.

3. Is she justified in her belief? If so, what should she do now?

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 1.1 What are the advantages of a “closed” critical care unit?
- 1.2 Why are critical care units one of the most common sites for healthcare errors?
- 1.3 What does the Synergy Model state about the relationship of patient and nurse?
- 1.4 Why do some healthcare providers believe that critically ill patients cannot give informed consent?
- 1.5 What issues must a nurse consider before physically restraining a patient?
- 1.6 Why is moral distress a significant concern for critical care nurses?
- 1.7 How can a nurse act to prevent compassion fatigue?

See answers to Chapter Review Questions in the Answer Section

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Chapter 2

Care of the Critically Ill Patient

Kathleen Perrin, PhD, RN, CCRN

Abbreviations

AACN	American Association of Critical-Care Nurses	CPOT	Critical Care Pain Observation Tool
BMI	Body Mass Index	ICU	Intensive Care Unit
CAM-ICU	Confusion Assessment Method of the Intensive Care Unit	PCA	Patient-Controlled Analgesia
CCFAP	Critical Care Family Assistance Program	SCCM	Society of Critical Care Medicine
CCFNI	Critical Care Family Needs Inventory	TPN	Total Parenteral Nutrition

✓ Learning Outcomes

Upon completion of this chapter, the learner will be able to:

1. Explain the characteristics of the critically ill patient described in the AACN Synergy Model.
2. Plan nursing responses to the concerns most commonly expressed by critically ill patients.
3. Describe nursing actions to meet some of the basic physiologic needs of critically ill patients.
4. Discuss ways to identify and meet the needs of families of critically ill patients.

Introduction

Critically ill patients are at high risk for life-threatening problems, and nurses must often focus on specific life-sustaining treatments. However, critically ill patients have basic needs as well.

Characteristics of Critically Ill Patients

The American Association of Critical-Care Nurses (AACN, n.d.) defines **critically ill patients** as “those patients who are at high risk for actual or potential life threatening health

problems. The more critically ill the patient is, the more likely he or she is to be highly vulnerable, unstable, and complex, thereby requiring intense and vigilant nursing care.” In the Synergy Model, the AACN postulates that when the needs of the patient and family drive the competencies required by the nurse, optimal patient outcomes can be achieved. The AACN continues by identifying and describing eight characteristics of critically ill patients.

The Synergy Model-Patient Characteristics

The Synergy Model-patient characteristics are scored on the health illness continuum from Level 1, which describes

a more compromised patient, to Level 5, which indicates a patient who is less compromised.

The characteristics are:

- **Resiliency:** “The ability to bounce back quickly after insult.” Patients range along the continuum from being unable to mount a response to having strong reserves.
- **Vulnerability:** “Susceptibility to actual or potential stressors.” Patients range from being fragile to being safe or “out of the woods.”
- **Stability:** “The ability to maintain a steady state equilibrium.” Patients vary from being unresponsive to therapies and at high risk for death to stable and responsive to therapy.
- **Complexity:** “The intricate entanglement of two or more systems (e.g., body, family).” Patients span the gamut from having atypical presentations of an illness or complex family dynamics to simple clear-cut and typical presentation.
- **Predictability:** “A characteristic that allows one to predict a certain course of events or course of illness.” Patients range from having an unusual or unexpected course of illness to following a critical pathway.
- **Resource availability:** “Extent of resources the patient, family, and community bring to the situation.” Patients may have few of the resources necessary for recovery available to them or may have extensive knowledge and skills.
- **Participation in care:** “Extent to which patient and/or family engage in care.” Patients and families may vary from being unable or unwilling to assist with care to being fully willing and able to participate.
- **Participation in decision-making:** “Extent to which patient and/or family engage in decision-making.” Patients and families may range from requiring surrogate decision makers to having full capacity and making decisions for themselves.

The Synergy Model proposes that when these characteristics of the patients are met by the competencies of the nurse, the patient will have the following outcomes:

- Comfort and healing
- Satisfaction with care
- Absence of complications
- Perceived change in function
- Perceived improvement in quality of life
- Decreased recidivism
- Effective cost resource utilization balance

Concerns of Critically Ill Patients

Critical care nurses have long focused on creating environments conducive to the comfort and healing of their patients. The noise, lights, and alarms in an intensive care unit (ICU) have been assumed to be sources of discomfort to many ICU patients, and nurses have tried to limit these stressors. However, when Cornock (1998) and Abuatiq (2015) examined what patients in ICUs rated as stressful and compared the findings with what nurses in the same ICUs believed was most stressful for patients, they learned that the environmental factors such as unfamiliar noises and bright lights were not the major sources of stress for patients. In both studies, patients described the most stressful items as:

- Being in pain
- Having tubes and lines in their body that restrict their movement
- Not being able to communicate
- Being unable to fulfill family roles
- Being unable to sleep
- Not being able to control themselves
- Being thirsty

Cornock (1998) noted that, overall, ICU nurses perceived the environment as more stressful than their patients. Nurses in Abuatiq’s study believed that the primary stressor for their patients was being in pain, but recognized that having tubes, not being able to control themselves, and not being able to communicate were other areas of concern. The following sections explore some of the issues identified as stressors by both patients and nurses: not being able to communicate, being anxious, not being able to sleep, being delirious (not in control), and being in pain.

Communication

Critical care nurses and ventilated patients indicate that because communication can be frustrating and difficult, it is one of their major stressors. It does not matter whether the patient is alert or appears to be unresponsive: Studies have documented that ventilated patients need assistance with communication from their healthcare providers, especially their nurses.

Essential for Patient Centered Care

Nurses must communicate with their patients whether they appear able to hear and comprehend or not. Communication allows for the patients’ “basic needs to be conveyed and decisions, wishes and desires about plan of care and end-of-life decision-making to be expressed” (Grossback, Stanberg, & Chlan, 2011, p. 46).

Sedated Patients

Ventilated patients who appear to be sedated and minimally responsive have been shown to recall the communication efforts of their nurses. A common finding among studies of such patients has been the need for effective verbal communication with them by healthcare providers (Elliot & Wright, 1999). Most studies have demonstrated that ICU nurses spend little time communicating with their sedated patients. Alasad and Ahmad (2005) believe that although nurses know that communication is important, it is easily forgotten in the context of other important patient care needs. Nurses do not ignore patients; rather, they have a tendency to try to anticipate patients' needs and respond to them before they present a problem. Alasad and Ahmad also note that nurses see communication with ventilated patients as discouraging because it is usually one-way communication. Nonetheless, patients often recall the presence, or absence, of such communication when they awaken.

When nurses communicate with their sedated patients, it is usually for a specific purpose (Elliot & Wright, 1999) usually to convey or obtain biopsychosocial information (Slatore et al., 2012). Nurses communicate with or around their sedated patients to:

- **Provide orientation or translate medical information:** Nurses believe that not knowing who is providing care, and where the patient is can be stressful to the patient. Grossback et al. (2011) also emphasizes this by saying that patients need reminders and explanations of what has happened to them and why they are intubated and in ICU.
- **State procedural and task intentions:** Studies indicate that nurses communicate more with their sedated patients when the patients are having procedures or tasks done. Patients state that they would prefer to have nurses ask their permission before such tasks and to give them some indication of what they will experience.
- **Provide reassurance:** Morse (2001) noted that nurses use reassuring words to help a patient hold on and endure a difficult procedure. The nurse might say, "You're doing well—it's almost done" even to a patient who appears unresponsive.
- **Apologize and/or recognize discomfort:** A nurse might say as he prepares to suction a patient, "I'm sorry, I know this is uncomfortable but I need to clear these secretions now. It is important to help your lungs to heal."
- **Obtain a response:** Determining if the patient is able to respond to a request is important. However, even when the patient does not respond, it is quite possible he can still hear the request and any other conversation.
- **Provide intentional and unintentional distractions:** Morse stated that nurses may use intentional distractions as a way of providing some comfort to patients who are undergoing procedures. For example, nurses

sometimes sing, hum, or joke with their unresponsive patients while they provide care.

- **Provide social information to colleagues:** Nurses sometimes have social conversations as they are caring for unresponsive, sedated, or ventilated patients. Patients have identified overhearing such conversations as a source of distress. They have felt removed from the situation and less than human.

As important to patients as the content of communication is how is conveyed. Patients identify the need to feel that nurses are "physically present at the bedside" (Patak, Gawlinski, Fung, Doering, & Berg, 2004). Patients see being "physically present" as being kind, patient, and attentive to their needs; offering frequent verbal reassurances; and knowing what information is important and providing it. Grossback et al. (2011) emphasizes creating a communication-friendly environment that includes making sure that the nurse is visible to the patient, speaking directly to the patient, and ensuring that lighting is adequate for the patient to see the speaker or communication board.

Responsive Patients

A majority of responsive, ventilated patients identify communication as highly frustrating (Patak et al., 2004). They frequently modify requests or avoid communication because the activity is so difficult (Magnus & Turkington, 2006). Intubated patients who are severely ill and are probably dying have higher levels of anger and frustration about their inability to communicate than do less ill patients (Happ, Tuite, Dobbin, DiVirgilio-Thomas, & Kitutu, 2004). Some of the patients' frustrations appear to center around the limited means utilized for them to communicate. Although there are a variety of possible communication adjuncts, such as letter boards, speaking valves, and prosthetic larynxes, they are infrequently used. The most common means of communication used by responsive ventilated patients are head nods in response to yes/no questions, mouthing words, gesturing, and writing. Hand and arm movements are required for both gesturing and writing, so the already limited communication from patients is reduced when patients' hands and arms are restrained. Happ et al. (2004) recommended limiting the use of such restraints, believing it may be the most effective way to facilitate communication with responsive ventilated patients.

Communication with ventilated patients centers around specific, predictable issues:

- The most common reason for communication between nurses and ventilated patients is the patients' experiences of **pain**.
- Other common reasons for communication are:
 - Identifying patients' emotions
 - Determining patients' symptoms
 - Responding to patients' needs for physical care

- The physical environment of the ICU
- The patients' home and family
- Treatment decision-making (Happ et al., 2004)

Patak et al. (2004) recommended evidence-based interventions to facilitate communication with mechanically ventilated, seriously ill patients. They stated that healthcare providers should do the following:

- Be educated about the frustration that mechanically ventilated patients experience when they are attempting to communicate their needs and desires.
- When communicating with ventilated patients, they should:
 - Routinely ask patients about their feelings and their state of mind.
 - Ask permission before beginning nursing care and procedures.
 - Evaluate patients' understanding of the information conveyed to them by asking simple yes/no questions.
- Demonstrate attention to the needs of their patients by informing them of their surroundings, plan of care, and when they will return after leaving the bedside.
- Approach each patient with a kind, patient manner; take the time to investigate and understand what the patient is communicating; and respond to the patient's communicated needs.

Grossbach et al. (2011) emphasize the use of alternative and augmentative communication strategies to enhance communication. Three common tools are writing materials, letter boards, and picture communication boards. These tools can be used if the patient is able to move his arms and point, blink yes or no when the appropriate word or symbol is pointed to by the nurse, or gaze at the intended object. When such communication strategies are utilized, the nurse should verify the chosen words/letters/symbols with the patient immediately so there is no doubt that the nurse understands the patient correctly.

Comfort

Patients, nurses and families consistently identify pain as one of the major problems for patients in critical care units. Barr et al. (2013) concluded that ICU patients routinely experience pain at rest, with care, and during procedures. Unfortunately, although it is assumed that all patients in critical care will experience some pain or discomfort, pain control is frequently inadequate, with 71% of patients remembering experiencing pain during their ICU stay (Stites, 2013). Obvious sources of pain are a patient's surgical site or traumatic injury. Many disease processes, such as myocardial infarction and subarachnoid hemorrhage, are clearly associated with severe pain. However, most critically ill patients also experience discomfort from their monitoring devices and routine nursing care. Immobility may be difficult for patients to endure, especially if they have arthritis. Conversely, turning may be extremely frightening and has been demonstrated to be one of the most painful procedures for critically ill adult patients (Puntillo et al., 2001). Routine procedures such as endotracheal suctioning can be particularly painful. One former patient described her sensations while being suctioned as "a red hot poker being slid down my throat that burned even more as it was withdrawn."

Unresolved pain does not just cause physical and psychic discomfort for the patient. It can impair the patient's recovery from his or her illness as well. Pain that is not adequately controlled can lead to anxiety, interfere with sleep, and contribute to the stress response, causing vasodilation, increased vascular permeability, immunosuppression, hypercoagulability, post-operative infections, persistent catabolism, and delayed healing (Glowacki, 2015). Unresolved pain can also contribute to pulmonary dysfunction through muscle rigidity and guarding. Thus, it is imperative that critical care nurses assess their patients for pain and manage their pain adequately.

Pain Assessment

Once the critical care nurse recognizes that nearly all her patients will experience some source of discomfort, one of

Gerontological Considerations

Happ, Tate, and Garrett (2006) offered several practical suggestions for communicating with older adults who are intubated. These include the following:

- Provide the patient with his eyeglasses or hearing aids so that it is easier for him to hear or lip-read.
- Enunciate slowly and pause while speaking.
- Ask only one question at a time and allow the patient time to respond.
- Establish a series of gestures so that the patient can communicate using them.
- Decide on gestures for yes and no and post them so they are visible to all who wish to communicate with the patient.
- Write out questions with potential answers so the patient can see and hear the question and choices simultaneously and then point to his choice.

her priorities should be to perform a pain assessment on a regular basis. Pain should be assessed and documented at least at the beginning of every shift and also with each assessment of the patient and each adjustment of pain medication. It has long been established that the most reliable indicator of pain is the patient's self-report of pain. McCaffery's (1968) classic definition of pain is "whatever the person says it is, existing whenever he says it does." Thus, when the patient is able to respond, the nurse should question him or her about the pain. A basic assessment should include at least the intensity and location of pain but, if possible, the nurse should ask the patient to:

- Rate the intensity of the pain on a scale of 0 to 10, with 0 being no pain at all and 10 being the worst pain one can imagine.
- Point to the location of the pain.
- Show where the pain radiates.
- Describe the characteristics of the pain: Is it burning or aching?
- Indicate if the patient has associated symptoms such as nausea, shortness of breath, or dizziness.
- State what aggravates the pain; for example, does it hurt more when the patient breathes?
- Consider what alleviates the pain; for example, does the pain decrease when the head of the bed is elevated?

Because the patient's self-report of pain is the most reliable indicator of the amount and type of pain that a patient is experiencing, the critical care nurse should always attempt to obtain the assessment from the patient. This means always asking the patient, even if the nurse thinks the patient may not be able to respond, using simple yes-or-no questions that call for only a blink of the eye or nod of the head to respond, and allowing adequate time for the patient to respond (Belden, 2006).

Unfortunately, many critically ill patients are unable to communicate their experiences of pain to their nurses. Therefore, critical care nurses must employ other ways to identify pain in these patients. In 1999, McCaffery and Pasero described a hierarchy of pain assessment techniques, including:

- Patient self-report.
- Search for a potential cause of a change in patient behavior.
- Observation of patient behaviors when patient self-report is not possible.
- Surrogate report of a patient's pain or patient's behavior change.
- Attempt of an analgesic trial. If one assumes pain is present, then an analgesic trial may be diagnostic as well as therapeutic (Herr et al., 2006). Attempt of an

analgesic trial or use of bispectral analysis may be the only way to determine the pain needs of a patient who is receiving paralytic agents or is deeply sedated.

With the realization that pain is a persistent problem for critically ill patients and that observation of patient behaviors is a valid approach to pain assessment, there has been a growing emphasis on developing behaviorally based tools for assessment of pain in critically ill patients. Although there are differences among the behaviors assessed in the tools, many include:

- **Facial expressions:** Is the patient showing a relaxed neutral position, or is the patient grimacing?
- **Body movements:** Is the patient lying completely still, slow and cautious in movement, pulling at tubes, or clenching his fists and rubbing parts of his or her body?
- **Muscle tension:** Does the patient resist passive movement?
- **Compliance with ventilation:** Is the patient ventilating easily, or out of synchrony with the ventilator?
- **Vocalization:** Is the patient speaking normally, or crying out with pain?
- **Physiologic parameters:** What are the patient's vital signs? Is the patient exhibiting diaphoresis or pupil dilation? Vital signs should not be used alone to identify pain in a critically ill adult patient because vital signs do not correlate with either patient self-report of pain or behavioral pain scores (Barr et al., 2013; Riker & Fraser, 2013). However, vital signs may indicate that further assessment for pain is needed.

Tools that are valid and reliable and can be used for both verbal and nonverbal patients are the Critical-Care Pain Observation Tool (CPOT) displayed in Table 2-1 (Gelinas, Fillion, Puntillo, Viens, & Fortier, 2006) and the Behavioral Pain Scale (BPS) (Barr et al., 2013). The CPOT is displayed in Table 2-1. It is imperative that each critical care nurse develop familiarity with whichever valid tool is utilized on his unit, use it consistently to assess his patients, and medicate his patients for pain in response to their documented pain levels.

Essential for Evidence-Based Practice

The critical care nurse should utilize a reliable, valid pain assessment tool to assess patients at regular intervals for the presence or relief of pain.

Pain Management

Analgesics are the appropriate medication if pain is the cause of a patient's discomfort. Ideally, analgesic medications should be administered before pain develops because it is

Table 2-1 Description of the Critical-Care Pain Observation Tool

Indicator	Description	Score
Facial expression	No muscular tension observed	Relaxed, neutral 0
	Presence of frowning, brow lowering, orbit tightening, and levator contraction	Tense 1
	All of the above facial movements plus eyelid tightly closed	Grimacing 2
Body movements	Does not move at all (does not necessarily mean absence of pain)	Absence of movements 0
	Slow, cautious movements touching or rubbing the pain site, seeking attention through movements	Protection 1
	Pulling tube, attempting to sit up, moving limbs/thrashing, not following commands, striking at staff, trying to climb out of bed	Restlessness 2
Muscle tension	No resistance to passive movements	Relaxed 0
Evaluation by passive flexion and extension of upper extremities	Resistance to passive movements	Tense, rigid 1
	Strong resistance to passive movements, inability to complete them	Very tense or rigid 2
Compliance with the ventilator (intubated patients)	Alarms not activated, easy ventilation	Tolerating ventilator or movement 0
OR	Alarms stop spontaneously	Coughing but tolerating 1
	Asynchrony: blocking ventilation, alarms frequently activated	Fighting ventilator 2
Vocalization (extubated patients)	Talking in normal tone or no sound	Talking in normal tone or no sound 0
	Sighing, moaning	Sighing, moaning 1
	Crying out, sobbing	Crying out, sobbing 2
Total, range		0–8

Source: American Association of Critical-Care Nurses (Gelinas, Fillion, Puntillo, Viens, & Fortier, 2006)

easier to prevent pain than to treat established pain. Thus, the nurse should administer analgesia before a chest tube is removed. However, anticipation of pain is not always possible, so pain medication should be administered as soon as possible after pain begins (Stanik-Hutt, 2003). Stanik-Hutt recommended that whenever possible, the nurse should utilize both opioid and non-opioid medications because they work differently to relieve pain and have complementary effects. Nonsteroidal anti-inflammatory medications and acetaminophen work in the peripheral nervous system to control pain, whereas opioids work in the dorsal horn of the spinal column and in higher levels of the central nervous system. However, for the moderate to severe pain that critically ill patients usually experience, IV opioids are the drugs of choice and should be used for initial therapy. Barr et al. (2013) state that all available IV opiates can be equally effective when titrated appropriately. See “Commonly Used Medications,” related to opioids.

General Principles for Administration of Pain or Sedative Medications

- For acute pain, analgesics should be administered intravenously for immediate onset of action to prevent

the imprinting of the painful experience on the nervous system.

- Subsequent doses may be given intravenously or orally on a regular schedule around the clock. Continuous pain management is essential since duration and efficacy of analgesic intervention may be as important as timing for treatment of pain (Helms & Barone, 2008).
- Intravenous infusions of analgesics start to act immediately; however, they will not provide significant analgesia until the infusion reaches “steady state.” The time to reach steady state is related to the duration of the effect of the drug, which is measured as a half-life. It generally takes three half-lives to approach steady state and five half-lives to achieve steady state.
- At the initiation of an infusion and when the infusion rate is increased, loading doses must be administered in order to provide immediate analgesia and maintain the desired analgesia until the infusion reaches steady state. Thus, a critically ill patient often will receive an IV bolus of an analgesic followed by an ongoing infusion of the pain medication with intermittent boluses and increases in infusion until the drug attains steady state and the patient experiences pain relief.

Commonly Used Medications

Morphine Sulfate, an Opioid

Introduction

Morphine has been the gold standard to which all other analgesics are compared and has been the narcotic of choice in critical care. It is potent, has a wide therapeutic range, acts rapidly when given intravenously, and is relatively inexpensive.

Desired Effects: The desired effects, which should become apparent 5 minutes after IV administration and last for 1 1/2 to 2 hours (longer in patients with renal or hepatic impairment), include analgesia, sedation, vasodilation, and relief of air hunger.

Nursing Responsibilities:

- Administration of a bolus dose of morphine for initial pain management. Initial doses are often 2 to 4 mg IV given every 5 minutes until the patient obtains relief. Later, intermittent doses may range from 2.5 to 15 mg every 2 to 4 hours. A dose of 15 mg should be given by IV push over 4 to 5 minutes.
- Initiation of an ongoing morphine infusion, if a constant supply of pain medication is needed, usually beginning

between 1 and 10 mg/hr; however, infusion rates vary greatly depending on the patient's tolerance and level of pain.

- Assessment of the patient's level of discomfort using a valid, reliable pain assessment tool at regular intervals.
- Assessment of the patient's hemodynamic response to the morphine dose.
- Consistent documentation of the patient's symptoms as an indication for additional morphine administration as well as a description of the patient's response to the initial dose and any necessary increases in dose.
- Weaning should occur in a controlled, planned manner.

Side and/or Toxic Effects: Dose reductions are needed in patients with renal failure and in the elderly. Morphine is metabolized by the liver with an active metabolite that produces analgesia and sedation. Because this active morphine metabolite is excreted renally, prolonged analgesia and sedation will occur in patients with renal failure.

Morphine can produce histamine release, causing vasodilation and hypotension.

- In response to anticipated painful procedures (e.g., turning) the patient might receive an additional bolus. When IV infusion rates are repeatedly increased versus administration of intermittent boluses as a means of responding to acute pain, the risk for excessive analgesia dosing exists.
- There are a variety of predictable situations when the doses of pain medications may need to be adjusted.
 - Elderly patients and patients with renal insufficiency usually need a decreased dose of medication.

Commonly Used Medications

Fentanyl (Sublimaze), an Opioid

Introduction

Fentanyl is one hundred times more potent than morphine. It has a faster onset of action than morphine and a shorter duration of action. It is the analgesic of choice in acutely distressed patients and patients with renal dysfunction, morphine allergy, or ongoing hemodynamic instability (Jacobi et al., 2002) because it does not produce histamine release, vasodilation, or hypotension. Fentanyl is metabolized by the liver and may accumulate in patients with liver disease. Prolonged drug effect does not occur in renal failure. Fentanyl has a wider margin of safety when compared with older opioids (Taylor, 2005).

Desired Effects: The onset of IV fentanyl is rapid, within 1 to 2 minutes, with a peak in 3 to 5 minutes and a duration of action of 1/2 to 1 hour. The onset of pain relief and sedation should be almost immediate.

Nursing Responsibilities:

- Administration of a bolus dose of fentanyl (IV bolus dose is dependent on the desired effect. For sedation/

analgesia in an adult, the dose may range from 0.5 to 1 mcg/kg/dose administered IV over 3 to 5 minutes).

- Starting an intravenous infusion of fentanyl with prn doses for severe breakthrough pain when frequent dosing is required.
- Assessment of the patient's level of discomfort using a valid, reliable pain assessment tool at regular intervals beginning 3 to 5 minutes after the dose.
- Monitoring the patient's respiratory rate and effort as well as his blood pressure throughout therapy.
- Weaning should occur in a controlled, planned manner.

Side and/or Toxic Effects: Too rapid administration of fentanyl may result in apnea or respiratory paralysis, muscle rigidity, bradycardia, or hypotension.

The respiratory depressant effects of fentanyl may outlast its analgesic effects.

- At the onset of therapy, until comfort is achieved, patients will often require higher doses of pain medication.
- Post-op and post-traumatic injury pain should decrease over time.

Weaning of analgesics may be attempted when the patient meets the following criteria:

- Pain control has been achieved.
- Patient is not receiving neuromuscular blocking agents.
- Patient is hemodynamically stable.
- Patient is stable on the ventilator.

Information concerning the management of pain and promotion of comfort for patients with specific disorders is discussed with each of the disorders. General information about the administration of the most commonly used analgesics follows.

As the patient improves and becomes responsive, he or she may be switched to patient-controlled analgesia (PCA). Later, using equivalent dosing, the patient may be switched from IV opioids to other medications prior to transfer to another unit or before discharge from the hospital. Details concerning pain management for specific conditions are located in each chapter. Nonpharmaceutical interventions should also be considered as they may reduce or relieve requirements for pain medications. Examples of nonpharmaceutical interventions include back rubs, repositioning, mouth care, music therapy, noise and lighting reduction, family visits, and facilitation of rest and sleep.

Essential for Evidence-Based Practice

Nonpharmacological interventions for pain should be utilized as adjunct therapy only in critically ill patients because of the lack of evidence to support the efficacy of their use for pain management in such patients (Belden, 2006).

Sedation: Guiding Principles

Sedatives are used in the critical care setting to treat **anxiety** and agitation and to provide amnesia. Although the use of sedatives can reduce the stress response and improve the patient's tolerance to interventions, sedatives should be used only after "providing adequate analgesia and treating reversible physiologic causes" (Jacobi et al., 2002). Besides pain, common reversible physiologic causes of anxiety and agitation include:

- Hypoxia
- Hypoglycemia

- Withdrawal
- Sleep deprivation
- Immobility
- Fear

A sedation goal or endpoint should be established early and redefined regularly for each patient (Jacobi et al., 2002). The most common goal of care for a sedated patient is that he or she is calm, awake, or easy to arouse with normal sleep-wake cycles. This is referred to as light sedation and is associated with improved clinical outcomes including shorter time on a ventilator and shorter stay in an intensive care unit (Barr et al., 2013). However, some patients—for example, those requiring advanced ventilator therapy—require deeper levels of sedation, and their sedation goals would vary accordingly.

Sedation Assessment

Just as it is essential to assess and document pain according to a valid, reliable scale, it is imperative to utilize a comparable scale for assessing agitation and sedation. Various scales are available, but there tend to be common factors assessed across sedation scales. According to De Jong et al. (2005) these include:

- Consciousness defined as patient awareness of self and surroundings often assessed by neurological examination. Sedation scales may use the Glasgow Coma Scale to evaluate consciousness.
- Agitation defined as patient restlessness characterized by nonpurposeful movement.
- Anxiety defined as a "subjective feeling of distress and anguish." Critical care nurses have a tendency to focus on the physiologic markers of anxiety (heart rate, blood pressure, and agitation), often failing to ask the patient himself what he was experiencing (Moser et al., 2003). Because anxiety, like pain, is a subjective experience, it is essential to question the patient concerning feelings of anxiety.
- Sleep
- Patient-ventilator synchrony defined as when there is coordination between the respiratory movements of the patient and the ventilator. Although patients are often sedated to ensure patient-ventilator synchrony, there is an association among continuous infusion of sedative, deeper sedation, and a prolonged requirement for ventilator support.

When caring for a critically ill patient requiring sedation, the critical care nurse should:

- Assess the patient for pain and treat the pain prior to beginning a sedation assessment. When pain is adequately treated, the patient may not require sedation.

Additionally, if sedation is provided prior to pain medication, sedation may mask the uncontrolled pain and prevent the patient from communicating about the pain (Clukey, Weyant, Roberts, & Henderson, 2014).

- Assess the patient and administer sedation in response to demonstrated findings on a valid, reliable
- Plan to assess and document the sedation level of each patient at least at the beginning of every shift, every

Table 2-2 American Association of Critical-Care Nurses Sedation Assessment Scale

Domain or subscale	Indicator	Score				
		Best 1	2	3	4	Worst 5
Consciousness	Awake and aware of self and environment	Spontaneously opens eyes and initiates interaction with others	Wakens and responds after light verbal or tactile stimuli May return to sleep when stimuli stop	Wakens and responds after strong or noxious verbal or tactile stimuli Returns to sleep when stimuli stop	Displays localization or withdrawal behaviors to noxious stimuli	Displays posturing or no response to strong or noxious stimuli
Agitation	Body movement, patient/staff safety*	Calm body movements and tolerance of treatments and restrictions Movements do not pose a significant risk for safety of patient or staff		Body movements or noncompliance with treatments or restrictions do not pose a significant risk for safety of patient or staff		Body movements or noncompliance with treatments or restrictions poses a significant risk for safety of patient or staff
	Noises of patient	No noises		Frequent moaning or calling out		Shouting, screaming, or other disruptive vocalizations
	Patient's statements†	Very calm				Very restless
Anxiety	Patient's perceived anxiety† (Faces Anxiety Scale‡)	No anxiety 				Extreme anxiety 
Sleep	Observed sleep	Looks asleep, calm, resting (eyes closed, calm face and body)	Looks asleep, periodically awakens and returns to sleep easily	Awake, naps occasionally for brief periods		Unable to sleep or nap
	Patient's perceived quality of sleep†	I slept well 		I slept fair 		I slept poorly 
Patient-ventilator synchrony	Breathing pattern relative to ventilator cycle	Synchrony of patient and ventilator at all times, patient cooperative and accepting ventilation Coordinated, relaxed chest movement		Occasional resistance to ventilation, or spontaneous breathing is out of synchrony with the ventilator Chest movement occasionally not coordinated with ventilator		Frequent resistance to ventilation, or spontaneous breathing not synchronous with the ventilator Uncoordinated chest and ventilator movements

Source: American Association of Critical-Care Nurses. De Jong et al. (2005)

*This component is assessed in all patients, regardless of the goal of sedation.

†Assumes the patient has the ability to understand directions and communicate his or her perceptions either verbally, in writing, or by pointing to words or pictures. If score is greater than 2 for this subscale, ask the patient if he or she needs something to help him or her relax.

‡Faces Anxiety Scale reprinted from McKinley et al., with permission of Blackwell Publishing.

4 hours unless the patient is sleeping, and whenever there are changes in the patient's level of sedation or agitation.

- Record sedation scale values with any sedative doses.
- Assess for delirium if the patient becomes more confused, disoriented, or agitated with sedation.

Sedation Administration

The learner should refer to the earlier section, "General Principles for Administration of Pain or Sedative Medication," for a brief review of duration, half-life, attaining therapeutic levels of continuous infusions, and intermittent dosing of medications. A specific application of this information for IV sedation is that ultra-short-acting agents such as propofol achieve steady state quickly and require few loading doses.

The dose of sedatives should be titrated to a defined goal (endpoint), then systematic tapering of the dose or daily interruption and awakening should be instituted to minimize the effects of prolonged sedation (Jacobi, et al., 2002). Daily weaning of sedatives may automatically be attempted when the patient meets the following criteria:

- Patient's SAS or RASS is at the target level.
- Sedation is not being used to treat delirium.
- Patient is not receiving neuromuscular blocking agents.
- Patient is hemodynamically stable.
- Patient is stable on the ventilator.

The sedatives most frequently used for ventilated patients are propofol and dexmedetomidine. Benzodiazepines such as lorazepam were previously administered as continuous infusions in ventilated patients. However, that method of administration is no longer recommended as first line sedation because it is associated with the development of delirium and other adverse effects (Barr et al., 2013). These medications are discussed in "Commonly Used Medications."

The major reason for the emphasis on lighter sedation and daily interruption or automatic weaning of sedation is evidence from researchers such as de Wit and Epstein (2003), who realized that patients receiving continuous infusions are likely to be oversedated and develop complications. In contrast, patients receiving continuous sedation who had their sedation levels regularly monitored by a valid sedation scale and were maintained at a lighter level of sedation had significantly shorter duration of mechanical ventilation, fewer tracheostomies, and shorter ICU as well as hospital stays (Jarman, Duke, Reade, & Casamento, 2013).

O'Connor, Bucknall, and Manias (2008) state that more robust research is required to assess the potential impact of sedation on the physical and emotional health of ICU survivors. Post-traumatic stress syndrome (PTSD) has been estimated to occur in 20% of critically

ill patients with higher prevalence in patients receiving benzodiazepines and having delusional memories of the ICU stay (Parker, Sricharoenchai, Raparla, Bienvenu, & Needham, 2015; Warlan & Howland, 2015). Reduced sedation levels do not significantly affect the outcome of PTSD (Croxall, Tyas, & Garside, 2014). This might be because "being awake while undergoing intensive care is not experienced as ordinary wakefulness but as an uneasy feeling of being dizzy and cognitively impaired" (Nortvedt, Kvarstein, & Jonland, 2005, p. 533). Thus, nurses must be aware of ongoing research concerning the effects of sedation. In the meantime, nurses must balance the needs of their critically ill patients for sedation with their needs for adequate ventilation and extubation as soon as possible.

Reflect On

Have you noted any indications that some of your patients have more difficulty during wake-up assessments than other patients? Which patients? How have you been able to balance a patient's wakefulness and need for sedation?

Issues related to anxiety and sedation of patients with specific disorders are discussed in the appropriate chapters.

Complementary and alternative therapies may be useful adjuncts to the management of anxiety in the critically ill, ventilated patient. Tracy and Chlan (2011) recommend the use of music therapy, imagery, and presence. Evidence-based music therapy calls for a critically ill patient to listen to at least 30 minutes of his or her preferred music through headphones on a daily basis. It is most beneficial if the music is without words and approximately 60 to 80 beats/minute. The patient will most likely need a skilled practitioner to utilize guided imagery to assist him to relax. However, no special equipment is needed and a relatively short, 10-minute session may be sufficient for anxiety reduction. Tracy and Chlan caution that the nurse should wait to use presence, the development of an empathic relationship, until prompted by cues from the patient.

Prevention and Treatment of Delirium

Delirium is very common among critically ill patients, developing in 50% to 80% of severely ill ventilated patients, and 20% to 50% of other ICU patients. Even though delirium results in 20% to 30% increased morbidity and mortality rates (Chang et al., 2008), nurses only recognize delirious patients 28% of the time (Swan, 2014). This means the disorganized thinking, inattention, and change in level of consciousness that underlie delirium went unrecognized in the majority of patients and almost certainly complicated their recovery.

Commonly Used Medications

Propofol (Diprivan), a Short-Acting General Anesthetic Agent

Introduction

Propofol has a very short duration of action. It is indicated when sedation is required but rapid awakening to perform neurological assessment or extubation is necessary (Zaccheo & Bucher, 2008). It may be utilized for patients with head injuries, although there is conflicting evidence about its ability to reduce intracranial pressure. It has no analgesic properties and appears to provide less amnesia than benzodiazepines.

Desired Effects: The onset of sedation with propofol is 40 seconds with an unknown peak of action and a duration of 3 to 5 minutes.

Nursing Responsibilities:

- A large vein is recommended for the infusion because propofol may result in burning and stinging at the IV site.
- It is administered as a continuous IV infusion due to the short duration of effect.
- The initial infusion dose is often 5 mcg/kg/min. The dose is titrated up slowly (increasing by 5 to 10 mcg/kg/min every 5 minutes) until the sedation goal is reached.
- The patient should be monitored for the development of hypotension, especially during the first 60 minutes.
- The dose should be temporarily reduced (but not stopped) daily to assess neurological and respiratory functioning. Most patients will awaken within 8 minutes.

- An analgesic will be required for pain management because it has no analgesic properties.
- Because propofol is not soluble in water, it is formulated in the same lipid emulsion used for TPN solutions. Therefore:
 - The caloric content of the infusion must be included in the total calories that the patient receives and adjustments should be made to the rate of any feeding to prevent overfeeding.
 - Lipid monitoring is required; triglyceride levels should be monitored after 2 days of propofol infusion.
 - Strict aseptic technique is essential because the lipid solution is an excellent medium for bacterial growth. All bottles must be discarded within 12 hours of being opened. IV tubing must be changed every 12 hours.

Side and/or Toxic Effects: Doses higher than 70 mcg/kg/hr for longer than 48 hours have been associated with propofol infusion syndrome. The syndrome occurs more frequently in patients who are also receiving catecholamines or glucocorticoids and includes lactic acidosis, rhabdomyolysis, and cardiogenic shock (Zaccheo & Bucher, 2008). Excessive doses may result in hypotension, which can be managed with fluids, repositioning, or vasopressors. Because the drug contains EDTA, a trace metal chelator, zinc deficiencies may occur in patients who have been receiving the drug for longer than 5 days.

Commonly Used Medications

Dexmedetomidine (Precedex), an α_2 Agonist

Introduction

Dexmedetomidine is indicated for short-term sedation of intubated patients during treatment in a critical care unit. It has anxiolytic, anesthetic, hypnotic, and analgesic properties. When administered IV, it has a rapid onset of action, is highly protein bound, and is metabolized in the liver. It is recommended for use for less than 24 hours, although it has been used for longer periods.

Desired Effects: Patients who are appropriately medicated with dexmedetomidine will be sedated but arousable. When awake, they will be comfortable, compliant, and able to comprehend and communicate with healthcare workers.

Nursing Responsibilities:

- A loading dose of up to 1 mcg/kg may be administered over 10 minutes but may not be necessary. Either hypertension or hypotension is possible with a loading dose and the rate of infusion should be slowed or stopped if hypotension develops. Typically, the hypertension resolves without treatment (Kemp, Henderlight, & Neville, 2008).
- A continuous infusion is usually started at 0.2 mcg/kg/hr. The rate of administration should be titrated to obtain the

desired level of sedation as determined by a sedation scale up to a maximum of 0.7 mcg/kg/hr. Dosages may need to be adjusted for patients in liver failure.

If the drug is abruptly withdrawn, patients may experience withdrawal symptoms including nervousness, agitation, and headaches followed by a rapid rise in blood pressure.

Side and/or Toxic Effects: The most common side effect of dexmedetomidine is hypotension and approximately 25% to 30% of patients develop it. To prevent hypotension, volume replacement may be necessary prior to initiation of the drug. If hypotension occurs, the dose of the drug may be decreased or vasopressors instituted. In contrast, approximately 16% of patients develop hypertension, especially if a loading dose is used. The drug may need to be slowed but often the hypertension will resolve. Slightly less than 10% of patients develop symptomatic bradycardia for which atropine is usually effective (Short, 2010). Additional side effects include nausea, vomiting, fever, hypoxia, and anemia. Overdoses may result in first- or second-degree heart blocks that generally resolve spontaneously.

Bariatric Considerations

In 2012, approximately 35% of adults in the United States were obese, defined as a body mass index (BMI) of 30 or greater, and the number of obese adults in the United States is still increasing (CDC, 2015). Since obesity is a major contributor to morbidity, especially cardiovascular disease and diabetes, critical care nurses are caring for increasing numbers of obese patients. Nurses need to carefully consider how they calculate pain medication and sedation requirements because the distribution, metabolism, protein binding, and clearance of drugs are altered in obese patients (Astle, 2009).

Obese patients have more adipose tissue than do adults of normal weight, resulting in a change in the lean-to-fat ratio. Lipophilic pain medications, such as fentanyl, are well absorbed by fat, and to obtain an effective dose, one should calculate the dose based on the patient's actual body weight. However, the elimination half-life may increase so that the effect of the drug on the patient may be prolonged.

Adipose tissue holds less water than muscle tissue; thus, hydrophilic medications *may not* be evenly distributed

through fat cells. Therefore, dosages of hydrophilic drugs, such as morphine, should be initially based on ideal body weight.

Dosage of the lipophilic sedative, propofol is also calculated based on the patient's actual body weight. Combining propofol with dexmedetomidine may be especially useful when sedating obese patients because the patient usually requires less narcotic for pain relief and has an improvement in spontaneous ventilation. However, no agent appears to be clearly superior to any other for sedating obese patients (King & Velmahos, 2010).

Nurses also need to be aware that obese patients can be malnourished. Missed or delayed identification of malnutrition in obese patients can result in increased morbidity and prolonged hospitalization (Mauldin & O'Leary-Kelly, 2015). Obese, malnourished patients whose renal and liver function are intact usually receive diets that are high in protein, low in calories. The goal is adequate nutrients for recovery but no emphasis on weight loss (Mauldin & O'Leary-Kelly, 2015).

Predisposing Factors for Delirium

There are three main neurotransmitters involved in the development of delirium: acetylcholine, dopamine, and aminobutyric acid. Dopamine normally excites the brain,

whereas the other two counterbalance the dopamine. Delirium probably results from an imbalance among these neurotransmitters in the brain, resulting in unpredictable and inconsistent neurotransmission. Thus, any disease

Commonly Used Medications

Lorazepam (Ativan), a Benzodiazepine

Desired Effects

Lorazepam has anxiolytic, sedative, and anticonvulsant effects. It also inhibits the ability to recall events. It has a slower onset but longer duration of action than midazolam. Lorazepam is appropriate for sedation of many patients and may be administered by intermittent or continuous IV dosing (Jacobi et al., 2002). When given by IV bolus, the onset of lorazepam's sedative effects as well as the peak effects should occur in 15 to 30 minutes with a duration of about 8 hours.

Nursing Responsibilities:

- The use of sedation guidelines or an algorithm to initiate and wean doses is recommended.
- Continuous sedation may be achieved by regular bolus doses (e.g., 2 mg IV every 4 hours) or a continuous infusion (no longer a preferred option for sedation).
- Lorazepam must be diluted immediately before it is injected with an equal volume of sterile water, dextrose

in water, or saline. To ensure that the drug is adequately mixed, the container should be gently inverted repeatedly.

- A 2 mg dose or fraction thereof should be administered slowly over 2 to 5 minutes.

Side and/or Toxic Effects: Use of large parenteral doses (equivalent to 5 to 10 mg/hr) may cause reversible renal tubular necrosis, lactic acidosis, and hyperosmolar states because of accumulation of the carrier solvents (polypropylene glycol and polyethylene glycol). Cumulative doses of 20 mg in 24 hours leads to delirium.

With too rapid IV administration, apnea and cardiac arrest as well as bradycardia and hypotension may occur. The patient may experience dizziness and excessive drowsiness. Both physical and psychological dependence can develop.

process, medication, or pattern of care that disrupts these neurotransmitters predisposes the patient to delirium. According to King and Gratrix (2009), Chang et al. (2008), and Burk, Grap, Munro, Schubert, & Sessler (2014) factors that predispose critically ill patients to delirium include:

- Patient characteristics such as:
 - Older age
 - Preexisting alcohol or sedative misuse or withdrawal
 - Preexisting dementia, depression, or psychiatric illness
 - Vision or hearing impairment
- Critical illness factors such as:
 - The presence of infection, especially if the patient develops sepsis
 - Organ dysfunction especially congestive heart failure or renal failure
 - Respiratory failure and the need for mechanical ventilation
- Pain
- Iatrogenic factors such as:
 - Polypharmacy (patient receiving three or more drugs) or the use of benzodiazepines
 - Disruptions in sleep pattern
 - Prolonged immobilization
- Restraints

Essential for Evidence-Based Practice

Nurses should assess patients routinely for the predisposing factors to delirium since patients who have three or more risk factors have a 60% chance of developing delirium (King & Gratrix, 2009).

Prevention of Delirium

Nonpharmacological strategies have been shown to be useful to prevent, and as adjunct strategies to treat, delirium (Rivosecchi, Smithburger, Svec, Campbell, & Kane-Gill, 2015). Reorienting the patient to his surroundings with each awakening and consistency of nurses caring for the patient are recommended. Utilizing some of the nonpharmacological strategies to promote a more restful sleep pattern as described in the next section help (Rivosecchi et al., 2015). Limiting unnecessary noise or providing the patient with music to filter out the noise helps some patients. Providing patients with eyeglasses and hearing aids so that they are less likely to misinterpret events in the environment also helps. Removal of physical restraints as soon as possible and early mobilization have been shown to be especially helpful (Rivosecchi et al., 2015). Vasilevskis

and colleagues (2010) recommend the adoption of a set of interventions known to decrease the likelihood of delirium: the ABCDE bundle. The bundle includes:

- Awakening and breathing coordination,
- Delirium monitoring
- Exercise/early mobility

Medications chosen for pain relief and sedation can also influence the development of delirium. Sedation with benzodiazepines, certainly lorazepam and probably midazolam, has been associated with the onset of delirium. The risk of development of delirium approaches 100% if a patient has received 20 mg of lorazepam (1 mg/hr) in a 24-hour period (Pandharipande, 2010). Dexmedetomidine may therefore be utilized for sedation instead of benzodiazepines especially in septic patients who are already prone to delirium. Reducing pain appropriately with morphine may also reduce the incidence of delirium. However, fentanyl does not appear to confer the same benefit (Pandharipande, 2010). Barr et al. (2013) do not recommend any medication be administered specifically for the prevention of delirium in the adult patient.

Manifestations of Delirium

Delirium is the sudden onset of disturbances in cognition, attention, and perception that fluctuate over time. Delirium can manifest as hyperactive (agitated), hypoactive (also known as quiet and often not identified at all or misdiagnosed as depression), or mixed. It is likely that the mixed type of delirium is the most prevalent in ICUs.

Delirium Assessment. Every critically ill patient should be screened for delirium at least once each shift. Initially an arousal or sedation/agitation assessment (e.g., RASS or SAS) should be performed. If appropriate, the patient should then be assessed using a delirium assessment scale such as the Confusion Assessment Method of the Intensive Care Unit (CAM-ICU) which has been demonstrated repeatedly to be both reliable and valid (Guenther et al., 2010). The CAM-ICU is displayed in ♦ Figure 2-1, and the instructions for scoring it are available at the ICU delirium website. To score positive for delirium on the CAM-ICU, the patient must display:

1. acute changes or a fluctuation in mental status (CAM-ICU Feature 1)
2. accompanied by inattention (Feature 2) and
3. either disorganized thinking (Feature 3) or
4. a level of consciousness other than alert (Feature 4)

Management of Delirium

Treatment of delirium includes the use of medication and environmental and supportive strategies. As soon as

Confusion Assessment Method for the ICU (CAM-ICU) Flowsheet

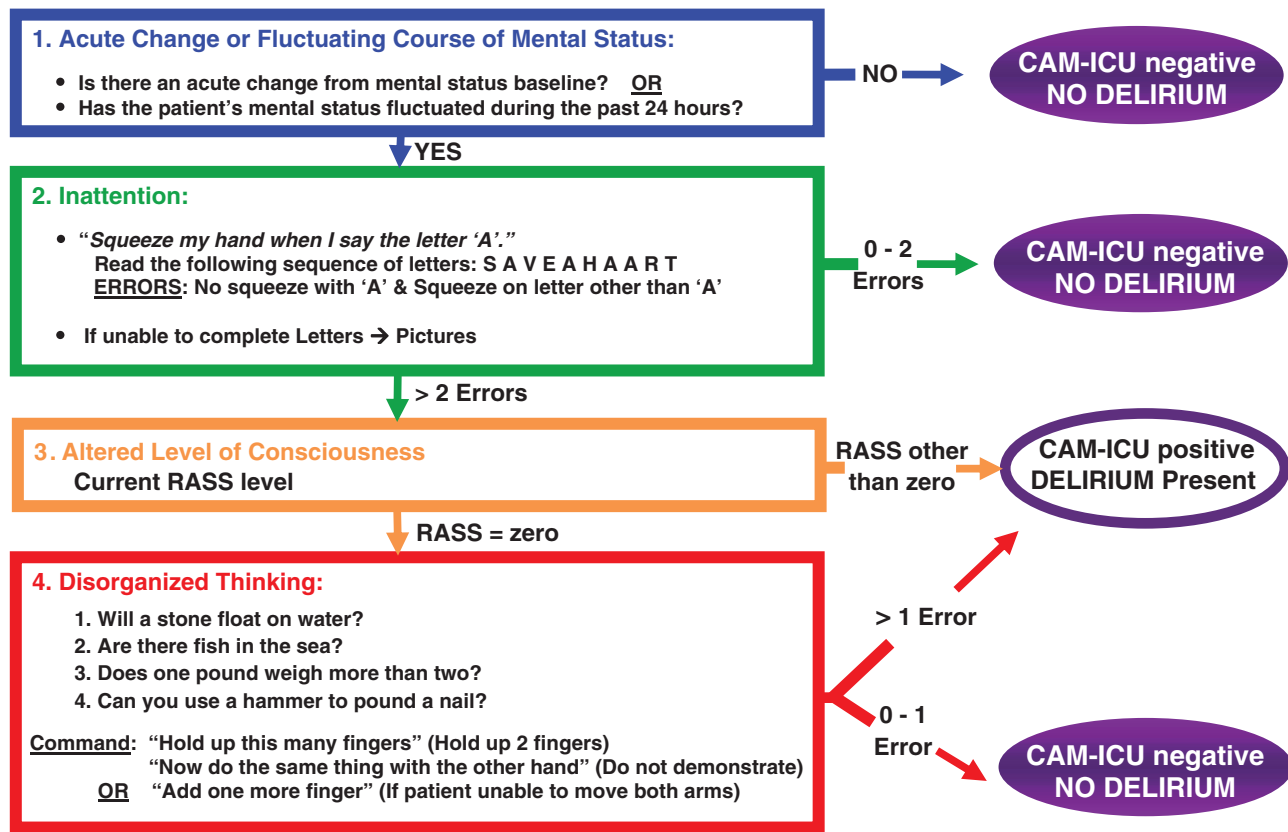


Figure 2-1 Confusion Assessment Method for the ICU (CAM-ICU) Flowsheet.

Source: ICU Delirium and Cognitive Impairment Study Group (www.ICUDelirium.org). Copyright 2002, E. Wesley Ely, MD, MPH and Vanderbilt University, all rights reserved.

delirium is suspected, the patient's medication regimen should be reviewed to determine if it is exacerbating the problem. Treatment with sedatives alone or the combination of sedatives and analgesics can worsen delirium because they do not solve the neurotransmitter imbalance and they can increase agitation. The dosages of any medications thought to be contributing to delirium should be decreased or the drugs discontinued.

Although haloperidol has been used in the past for treatment of delirium, there are no published studies that show it reduces the duration of delirium in critically ill adult patients (Barr et al., 2013). However, second generation atypical antipsychotics such as quetiapine may reduce the duration of delirium (Barr et al., 2013). The nurse monitors a patient receiving quetiapine for QT prolongation and dysrhythmias (torsades de pointes), which can result in sudden death. However, the QT prolongation is not as pronounced with quetiapine as with haloperidol. Common reported adverse effects include somnolence, hypotension, and dizziness. Quetiapine is not available in parenteral form and must be crushed and given by enteral tube if the patient is unable to swallow.

When IV medication is required, Barr et al. (2013) suggest a continuous infusion of dexmedetomidine be administered to decrease the duration of delirium. See "Commonly Used Medications" for more information.

Essential for Evidence-Based Practice

When administering dexmedetomidine, the nurse must monitor the patient for bradycardia and hypotension.

While the patient is experiencing delirium, the nurse and the patient's family will need to reorient the patient to her surroundings frequently. As one patient who was delirious for several days after a motor vehicle crash said, "All the time I was there, I never experienced any pain but the mental anguish I felt was terrible. I wish I could tell my nurses now how they could have helped me. I thought they were part of the Mafia and were trying to do me in at any time. I needed my nurse to say to me every time I woke up, 'You are in _____ Medical Center. You have been in a motor vehicle crash. At the moment, you have a tube in

your throat so you can't speak; we have your hands tied to keep you safe so that you do not pull out your tubes. You seem to be seeing and hearing things. These are wild dreams that are caused by the medications we are giving you. Try to remember that you are doing okay and we are trying to help you."

Essential for Patient Centered Care

Although delirious patients often act out and may frighten others, they are terrified and need nursing reassurance that they are safe.

Basic Physiologic Needs of Critically Ill Patients

Sleep

Despite extensive research indicating the value of sleep to critically ill patients, sleep disturbances continue to be common in critically ill patients contributing to delirium and other sources of morbidity (Tembo, Parker, & Higgins, 2013). According to Bourne and Mills (2004), there are a multitude of reasons why critically ill patients may have difficulty sleeping, including:

- **Environmental factors:** An ICU can be a bright, noisy place with alarms sounding and machinery such as ventilators or balloon pumps working appropriately.
- **Patient care activities:** When a nurse is trying to allow a patient to sleep but needs to monitor vital signs, the tightening of a blood pressure cuff may awaken the patient.
- Discomfort from the acute illness itself such as diaphoresis from a fever may cause sleep disruption.
- **Medications:** Sedative and analgesic combinations commonly used to facilitate mechanical ventilation are among the most sleep disruptive of drugs. Cardiovascular, gastric protective, antiasthma, anti-infective, antidepressant, and anticonvulsant medications have also been reported to cause sleep disorders.
- Withdrawal from prescribed and recreational drugs also may cause sleep disruption.

Sleep disruption is so common in the critically ill that very few mechanically ventilated patients have normal sleep patterns. King and Gratrix (2009) state that patients in ICU average only 2 hours of sleep a day. The sleep that they do manage to obtain is more likely to be in the early superficial levels of sleep than in deep, restorative REM sleep (Tracy & Chlan, 2011). This is a concern because lack of sleep has been associated with both serious physiologic and psychological effects. The consequences of inadequate sleep include changes in metabolism and immune response

as well as respiratory dysfunction. These may lead to delayed healing and prolong the need for mechanical ventilatory support (Tracy & Chlan, 2011). In addition, sleep disruption has long been thought to be a factor in the development of delirium.

Unfortunately, according to Bourne and Mills (2004), the majority of sleep disorders experienced by ICU patients cannot be resolved by the use of medications alone because patients experience disruptions in both the total amount of sleep and the quality of sleep. Critically ill patients suffer in particular from sleep fragmentation and reduced restful sleep. Bourne and Mills believe that there is limited evidence to support the use of drugs to treat sleep disorders in ICU patients and that all the available drugs have potential problems. Therefore, they recommend the following strategies:

- A short course of therapy for any drugs prescribed for acute sleep disturbances
- Appropriate use of sedation, sedation scoring systems, and vigilance for withdrawal syndromes to minimize sleep disturbances
- A combined approach incorporating environmental controls, appropriate treatment of the patient's acute illness, and rational drug use

After noting that it is a nursing challenge to recognize the importance of adequate sleep to critical care patients, Honkus (2003) listed the following methods that can be used to modify the environment and prepare a patient for sleep:

- Assess all intravenous lines for patency and level all transducers, placing the head of the bed in the desired position before the patient is settled for sleep. Obtain vital signs.
- Assess the patient for pain or discomfort and provide medication as needed.
- Decrease noise in the patient's environment as much as possible. To accomplish this, many units are now instituting designated quiet times, often twice daily, during which visitors are limited and controllable noise, light and activity are decreased to provide all patients on the unit an opportunity to sleep (Stafford, Haverland, & Bridges, 2014). If quiet times are not possible, there is some evidence that providing patients with ear plugs and eye masks might help the patient to sleep (Alway, Halm, Shilhanek, & St. Pierre, 2013).
- If it is necessary to disrupt the patient's sleep, try to space procedures so that the patient is able to obtain at least 2 to 3 hours of uninterrupted sleep at a time.

Tracy and Chlan (2011) reiterate Honkus's recommendations and add the following suggestions:

- Assess the patient's normal sleep patterns and habits (e.g., does the patient listen to music while falling

asleep?). Attempt to duplicate as many of these as possible.

- Ensure ventilator settings are in synchrony with the patient's breathing pattern.

Nutrition

Nearly all critically ill patients need a consult with a dietitian. It may be because they:

- Have had a cardiovascular event and should modify their diet
- Are in renal failure and require adjustment of nutrients in their diet
- Are intubated and require enteral or parenteral nutrition
- Have had surgery and require specialized nutrition for tissue repair
- Require nutritional therapy to modulate the metabolic response to stress
- Have increased caloric and other nutritional needs following trauma or burns

The precise nutritional assessment and requirements for patients with specific disorders are discussed in each of the chapters of this text. However, an overview and some general principles concerning nutrition for critically ill adults are discussed in this section.

Although severity of illness is the major factor in determining outcomes of critical illness, the patient's pre-existing nutritional status and adequacy of nutritional supplementation also contribute to them (Alliance to Advance Patient Nutrition, 2013). The benefits of adequate nutrition in patients include:

- Improved wound healing
- Decreased catabolic response to injury
- Improved gastrointestinal function
- Reduction in complications, length of hospital stay, and cost of stay

In addition, it has been postulated that undernourishment of ventilated patients results in diminished ventilatory drive, reduction in the production of surfactant, and difficulty in weaning due to muscle fatigue caused by diaphragmatic and skeletal muscle weakness. This has not been consistently demonstrated in research studies, however (Higgins, Daly, Lipson, & Guo, 2006).

Assessment

The first step in determining a patient's nutritional requirements is doing an assessment. A nutritional assessment includes:

- Obtaining a history including comorbid conditions
- Reviewing diagnostic studies

- Assessing function of the gastrointestinal tract
- Determining nutritional requirements

The amount of information that the nurse is able to obtain from the history may be limited due to the patient's physical condition. However, it is essential that the nurse learn:

- The patient's current height and weight, and if the patient has recently lost or gained a significant amount of weight
- Information about food allergies, especially allergies to shellfish for patients who may be undergoing studies that require contrast media
- If the patient has been consuming nutritional supplements because some supplements can alter the patient's electrolyte and metabolic balance
- If the patient has had any swallowing difficulties, nausea or vomiting, or constipation or diarrhea
- The amount of alcohol a patient admits to consuming
- Any type of diet the patient may be on, whether medically prescribed or self-determined

The nurse will need to verify the patient's weight and compare it to the patient's previous weights to identify excessive weight gain or loss. It is common for critically ill patients to have weight fluctuations due to fluid imbalances. A change in weight of 2 pounds (or about 1 kg) may represent the loss or retention of approximately a liter of fluid. The patient's weight should also be compared to an ideal body weight, or the patient's body mass index (BMI) should be calculated. (See Box 2-1 for information about BMI.) Patients who are obese or underweight when admitted to critical care have more complications and longer hospital stays.

A common measure used to identify protein nutritional status in critically ill patients is serum albumin. Critically ill patients often have low serum albumin levels (less than 3.5 g/dL or 35 g/L), most likely reflecting both their nutritional status and the physiologic stress of their illness. Levels of serum prealbumin (normal 20–30 mg/dL) are monitored less frequently but are more immediate indicators of physiologic stress and protein nutritional adequacy (Higgins et al., 2006). Additional studies that are frequently reviewed to determine nutritional adequacy and requirements are serum hemoglobin, magnesium, and phosphorus.

Once the patient's nutritional status has been assessed, the patient's nutritional requirements will be determined by the dietitian

- A first step is usually to calculate the patient's caloric, protein, and fluid requirements.
- A normal caloric intake is 25 kcal/kg/day for a person who is well nourished, nonstressed, and has a normal albumin level. The nitrogen requirement for the same patient would be 0.8 to 1 g protein/kg/day.

Box 2-1 Body Mass Index (BMI) Calculation

$$\text{BMI (kg/m}^2\text{)} = \frac{\text{Weight (kg)}}{\text{Height (m)}^2}$$

- As the stress of the critical illness mounts, the patient will require increasing amounts of calories and may require as much as 1.5 to 2 g/kg/day of protein.
- Fluid intake is closely tied to energy consumption and, normally, a person will require about 1 mL of fluid per calorie. However, fluid requirements are influenced by a wide variety of factors.
- Caloric, protein, and fluid requirements are usually calculated by a nutritionist because both underfeeding and overfeeding patients present problems.

The energy requirements of critically ill, ventilated patients are usually estimated by either a dietician or a physician. They may be estimated by using formulas that calculate energy demand based on body size composition, age, and gender, or they may be estimated based on calorimetry. The most commonly used equations to estimate energy requirements for critically ill patients are the Harris-Benedict equations (Box 2-2). Unfortunately, these equations tend to result in unpredictable errors when used to estimate energy requirements for some patients (Higgins et al., 2006) such as those who are morbidly obese, severely underweight, or have fluid imbalances. Therefore, indirect calorimetry may be utilized instead even though there is not sufficient evidence to recommend it in all circumstances (McClave et al., 2009).

Indirect calorimetry uses a metabolic cart to measure the heat production, oxygen consumption, and carbon dioxide production of the patient. These values are used to calculate basal energy expenditure in kilocalories. Resting energy requirements are usually calculated, so the measurement must occur when the patient has been without food for 2 hours, after the patient has been resting for 30 minutes, and when the patient is in a thermoneutral environment (Higgins et al., 2006). Once a patient's nutritional requirements have been established, a decision can then be made about how to meet them best.

Guidelines for Providing Nutritional Support

Whenever possible, patients should receive oral feedings. However, many critically ill patients are unable to eat and thus require nutritional support. Unfortunately, there is disagreement among available guidelines about the details of provision of nutritional support (Marshall et al., 2012). Most guidelines suggest that if a critically ill patient

Box 2-2 Harris-Benedict Equations for Calculating Basal Energy Expenditure (BEE)

For women, the BEE = $66.5 + (13.75 \times \text{kg}) + (5.003 \times \text{cm}) - (6.775 \times \text{age})$

For men, the BEE = $655.1 + (9.563 \times \text{kg}) + (1.850 \times \text{cm}) - (4.676 \times \text{age})$

The result is multiplied by a stress factor (1.2 to 2.5) for most patients.

is unable to consume adequate nutrition orally, nutritional support should be started within 24 to 48 hours of admission (McClave et al., 2009). Although a consult with a dietician is preferred before the start of nutritional support, it should not be delayed if a consult is not immediately available (Alliance to Advance Patient Nutrition, 2013). Starting nutritional therapy early is associated with improved calorie and protein intake, resulting in better nitrogen balance and a trend toward lower mortality. McClave et al. (2009) state that early nutritional support should be administered enterally if at all possible because it is associated with a much lower risk of infection and better outcomes than parenteral nutrition. When the patient is not tolerating enteral nutrition, there is insufficient evidence to state when parenteral nutrition should be started. However, the use of parenteral nutrition is appropriate for patients in whom it would be life sustaining such as patients with short bowel syndrome, perforated gut, or a high-output fistula.

Essential for Evidence-Based Practice

Whenever possible, critically ill patients should receive enteral nutrition within the first 24 to 48 hours of the onset of their illness.

Enteral Nutrition. **Enteral nutrition** is the delivery of nourishment by feeding tube into the gastrointestinal (GI) tract. Initially and for short-term use, enteral feedings may be delivered through a large-bore nasal or oral gastric tube. For long-term use, small-bore feeding tubes or gastrostomies are usually required. Feedings may also be delivered into the small bowel (postpyloric feeding); however, there is no evidence that placing tubes in the small bowel prevents aspiration and ensuring such placement may delay the onset of feeding. See ♦ Figure 2-2 for information about the various types of feeding tubes and sites.

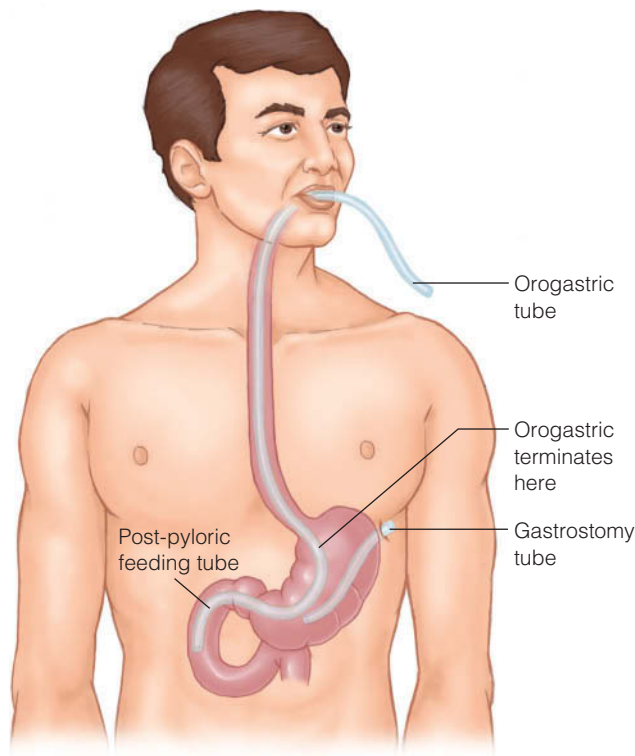


Figure 2-2 Locations of feeding tubes.

Enteral feeding is the preferred route for nutritional supplementation because it is associated with significantly lower rates of infection than parenteral nutrition. In part, this lower infection rate may occur because enteral feeding prevents translocation of bacteria from the GI tract, which can occur when the GI tract is inactive. Therefore, the recommendation to start feeding early, at 24 to 48 hours after admission, is not only to provide adequate nutrition immediately but also to decrease the likelihood of infection. The most common problems associated with enteral feeding are:

- High gastric residual volumes
- Bacterial colonization of the stomach
- Increased risk of aspiration pneumonia
- Unnecessary interruption of feeding resulting in prolonged fasting and underfeeding

Enteral feeding formulas are usually composed of proteins; a source of calories (often lactose free); vitamins; minerals; trace elements; and, possibly, fiber. There are standard formulas and ones that are intended for specific subsets of patients, such as patients with renal failure. The appropriate formula, amount of free water, and any adjuncts for a specific patient are usually determined by the dietitian in consultation with the physician because patients have differing nutritional needs. For example, the addition of glutamine is recommended for patients with severe burns and trauma. The amount of formula that the

patient will require to meet his nutritional requirements is dependent on the chosen product and is calculated by the dietitian and established as a target dose or goal.

Issues Associated with Enteral Feedings

Critical care nurses are responsible for attempting to provide the appropriate amount of nutritional intake while limiting the problems associated with enteral nutrition.

Optimizing Delivery of Feedings. Most ventilated, enterally fed patients receive only a portion of their prescribed caloric needs, primarily because their tube feedings are interrupted almost 5 hours each day (Stewart, 2014). The most common reasons for long-term interruption of tube feedings are technical problems with the tube such as misplacement or obstruction accounting for 25% of interruptions or discontinuation of the feedings when patients are about to be extubated or have a procedure performed accounting for 50% of interruptions (Stewart, 2014). Common reasons for shorter term interruption of tube feedings include the patient developing a high gastric residual or being repositioned.

Critically ill patients are often started on continuous gastric tube feedings because it is believed continuous feeding may cause less GI intolerance. There are varying philosophies on how best to avoid the development of GI intolerance while ensuring that the patient receives his target dose:

- To avoid GI distress, some physicians advocate starting at 10 mL/hr and increasing the rate every few hours if the tube feeding is tolerated until the patient is at the target dose.
- In order to attain the target dose more quickly, some physicians advocate starting the feeding at that rate.
- In the past, to prevent GI distress, nurses checked the patient for gastric residual every 2 to 4 hours and stopped the feeding if the residual was twice the amount of the hourly feeding.
- According to McClave et al. (2009) higher residual feeding protocols should be considered as a strategy to optimize patients' nutritional intake. They recommend continuing tube feedings unless the residual exceeds 500 mL. However, this recommendation is controversial (McClave, 2010). Metheny, Schallom, Oliver, and Clouse (2008) recommend checking residuals every 4 hours and then evaluating the patient, potentially stopping the tube feeding when residuals exceed 200 mL because aspiration occurs significantly more often then.
- Protocols using higher residual volumes are less likely to result in interruption of gastric feedings. However, most protocols that utilize higher residuals suggest that after a second residual greater than 200 or 250 mL

is aspirated, a motility agent, such as metoclopramide, be prescribed. Most importantly, the nurse needs to observe the patient for any indication of GI distress such as cramping, abdominal distention, increasing gastric residuals, and nausea. The feeding should be stopped if these develop as well as if the patient vomits, whatever the residual.

- The absence of bowel sounds or flatus should not forestall feeding. Providing enteral nutrition enhances GI motility and the return of bowel sounds (McClave et al., 2009).
- An alternative to continuous tube feedings are intermittent boluses of tube feeding. According to McClave et al. (2009), there is insufficient evidence to support the use of one mode of delivery of enteral feeding versus the other. When a bolus dose is delivered, it is usually a larger dose (250–400 mL) and is provided every 4 to 6 hours. Bolus tube feedings have historically been held if the residuals are half of the bolus dose. However, as with continuous feedings, some advocate administering the bolus as long as the residual is less than the amount described in a protocol (often 200–500 mL) and adding metoclopramide to the regimen if the residuals are high. Patients are more likely to complain of some discomfort such as cramping, bloating, and nausea with bolus doses.

Essential for Evidence-Based Practice

Assessment for gastric residuals should be based on evidence and protocols so that feedings are not interrupted unnecessarily for residuals less than 200 mL.

- Prevention of aspiration:
 - Prior to institution of enteral feeding, the nurse must ensure that the nasogastric tube is located in the stomach. Historically this has been accomplished by auscultation for an air bolus over the stomach. However, this method has been demonstrated to be inaccurate. The appropriate method for identifying placement of the feeding tube in the stomach is visualizing the tube in the stomach on an abdominal x-ray (AACN, 2010).
 - Placement should be verified every 4 hours by examining the position of the tube, the changes in the volume and type of aspirate, and measuring the pH of the aspirate. If the nurse has any concerns about the placement of the tube, an x-ray should be obtained to confirm the position (AACN, 2010).
 - Most authorities now advocate that to prevent aspiration of tube feeding contents and ventilator

associated pneumonia, patients receiving gastric feedings be placed in the semirecumbent position (have the head of the bed elevated between 30 and 45 degrees).

- When gastric residuals have been consistently high and/or the patient has not been tolerating gastric feedings, then there may be sufficient reason to switch to small bowel feedings.
- Prevention of bacterial colonization of the stomach:
 - Prepackaged tube feeding products are sterile. Although there is insufficient evidence to require their use, many nursing units are using closed enteral feeding systems, using sterile water for any additive, and opening the system aseptically. The systems are changed every 24 hours with the expectation that there will be less likelihood of colonization of the stomach.
- Prevention of diarrhea:
 - If the patient is receiving bolus feedings, switching to continuous feedings may decrease the occurrence of diarrhea.
 - If the patient is lactose intolerant, switching to a feeding that does not contain lactose may help.
 - Adequately diluting liquid medications with water may also help.
 - It is unclear whether using probiotics restores normal flora and decreases the incidence of diarrhea in the general ICU population. However, the use of probiotics does decrease infection in patients following transplantation, major abdominal surgery, and severe trauma.
 - If diarrhea develops, an attempt should be made to distinguish between infectious and osmotic diarrhea.
 - If noninfectious diarrhea develops, soluble fiber or small peptide formulations may be utilized.

Parenteral Nutrition. **Parenteral nutrition** is the infusion of nutrients using a venous catheter located in a large, usually central, vein. According to Simpson and Doig (2005), it is indicated in critically ill patients when nutritional supplementation is needed and enteral feedings cannot be initiated. It may be initiated within 24 hours for patients with preexisting nutritional deficiencies. However, in 2009, McClave et al. recommended that in patients who were previously healthy, if enteral nutrition would not be tolerated, initiation of parenteral nutrition could be delayed for 7 days. This recommendation emphasizes that enteral nutrition is preferred over parenteral nutrition and that strategies to maximize enteral nutrition should be used before the parenteral route is utilized. Parenteral nutrition solutions usually are formulated by combining dextrose, lipids,

protein (in the form of amino acids), electrolytes, water, vitamins, and trace elements. In addition, McClave et al. recommended that parenteral nutrition be supplemented with glutamine. The specific components of the infusion are prescribed for each patient by his physician, and the rate of administration is dependent on the patient's energy requirement. The major risks associated with the administration of parenteral nutrition are:

- Gut mucosal atrophy
- Overfeeding
- Hyperglycemia
- Increased risk of infectious complications
- Increased mortality

Critical care nurses are responsible for maintaining the ongoing infusion while attempting to limit the problems associated with parenteral nutrition. Some of the issues associated with parenteral feedings and the related nursing interventions are:

- **Access:** Parenteral nutrition is delivered through a catheter with its tip in the superior vena cava or the right atrium.
 - To aid in the prevention of catheter-related sepsis, full barrier precautions should be utilized during insertion of the catheter, and the site should be prepped with chlorhexidine.
 - The subclavian vein is the recommended access, so a chest x-ray to rule out pneumothorax and to ensure catheter placement is required following catheter insertion and before the IV is utilized.
 - A dedicated line is required for infusion of parenteral nutrition. No other IV infusions or boluses should be administered through the line and no blood should be drawn from it.
 - Policies vary from institution to institution but most require changing the parenteral nutrition solution bag and tubing aseptically every 24 hours.
- **Overfeeding:** Due to the high concentration of glucose and lipids, overfeeding can occur with parenteral nutrition. Patients who are receiving propofol for sedation should not receive lipids in their parenteral nutrition. McClave et al. (2009) suggested that all patients could maximize the benefits and minimize the risks of parenteral nutrition by not receiving lipids in the solution during the first week of hospitalization.
- **Hyperglycemia:** Critically ill patients who are receiving parenteral nutrition should be started on intensive glucose management and insulin therapy.
- **Hypoglycemia:** A sudden decrease in the rate of infusion of parenteral nutrition can cause the patient's blood sugar to plummet. Therefore, parenteral nutrition

is rarely stopped abruptly, and it is always infused using an infusion pump.

- **Risk of infection:**
 - Ensuring that the central line has been inserted utilizing full barrier precautions is the first step in decreasing the risk of infection.
 - Maintaining a dedicated line is the second step. The dressing on the access site should be aseptically applied, securely attached, and changed in accordance with agency policy.
 - Removing lipids from the parenteral nutrition is also helpful in preventing infection because lipids support the growth of many microorganisms.
 - The patient should be monitored for signs of sepsis, including fever, chills, elevated white blood count, and positive blood cultures.
 - If the catheter site is thought to be the source of infection, the catheter is discontinued and the tip is usually sent for culture and sensitivity.

Evaluation of Effectiveness of Nutritional Therapy

The effectiveness of the patient's nutritional therapy can be monitored by:

- Determining the patient's weight daily (expect weight stabilization or 1/4 to 1/2 pound weight gain per day with adequate nutrition)
- Examining the following lab studies:
 - Albumin or prealbumin
 - Hemoglobin and hematocrit
 - Electrolytes, including potassium
 - Magnesium
 - Phosphorus
- Assessing the patient's wounds for granulating tissue

Mobility

Since Olson, Johnson, and Thompson published the *Hazards of Immobility* in 1990, nurses have been aware of the risks to all patients of being immobile. However, critical care nurses, perhaps fearing the consequences of moving patients with multiple lines or poor oxygen saturation, have been slow to mobilize their patients. Now, it is becoming increasingly apparent that immobility is a grave hazard for critically ill patients and the challenges to mobilizing them must be overcome. The short-term consequences of immobility in critically ill patients include an increase in ventilator-associated pneumonia, delayed weaning from mechanical ventilation, development of pressure ulcers, and delirium (Vollman, 2010; Barr et al., 2013). Long-term

consequences of immobility include increased morbidity, mortality, and cost, as well as decreased quality of life (Vollman, 2010).

Instead, critically ill patients who are mobilized early in the illness with a structured, progressive plan show a decrease in the need for sedation, a decrease in the prevalence of delirium, and a decreased length of stay in both the intensive care unit and the hospital (Balas et al., 2012). Early mobilization is progressive, beginning with head of the bed elevation and passive lateral turns, continuing to passive and active range of motion, movement against gravity, dangling and balancing, transferring to a chair, chair positioning, then ambulating. Physical and occupational therapists are actively involved in deciding what level of activity is appropriate for each patient, providing therapy, and deciding when to progress the patient to a higher level.

Critical care units that are engaged in progressive mobilization usually have developed a protocol for mobilizing patients. The protocol is likely to include minimum criteria for instituting early mobility, reasons to halt mobility, and a mobility hierarchy (Balas et al., 2012). The nurse's role in progressive mobility depends on the protocol. In some protocols, it is the nurse's responsibility to initiate turning and passive range of motion then identify to the healthcare team when the next level is appropriate. In other protocols, progressive mobility is instituted automatically for patients in ICU. The nurse is responsible for determining when a patient is compromised and the activity should be halted. Often, the nurse will share responsibility for active and passive range of motion and transferring a patient from bed to chair with a physical therapist. Ambulating a ventilated patient is a more complex task and usually requires the assistance of multiple disciplines.

The Needs of Families of Critically Ill Patients

Families of critically ill patients often experience stress, anxiety, and depression (McAdam & Puntillo, 2009). Families' stress scores are high when the patient is admitted to the ICU but tend to decrease over time (McAdam, Fontaine, White, Dracup, & Puntillo, 2012). McAdam and Puntillo (2009) noted that between 15% and 35% of family members experience depression, with wives of patients being the most likely to be depressed. Anxiety is particularly high in families of critically ill patients, with 35% to 73% of family members being anxious especially wives and families of patients with unexpected admissions. Such stressed and anxious families clearly have needs, and nurses are often called on to respond to them.

Molter (1979) did seminal work on the needs of families of patients in critical care, leading to the formation of the Critical Care Family Needs Inventory (CCFNI). Since that time, her work has been validated by numerous other researchers (Kinrade, Jackson, & Tomnay, 2009). Based on her work, the following have been identified as needs of families of critically ill patients:

- Feel there is hope
- Feel hospital personnel care about the patient
- Have a waiting room near the patient
- Be called at home about changes in the patient's condition
- Know the prognosis
- Have questions answered honestly
- Know specific facts about the patient's prognosis
- Receive information about the patient at least once a day
- Have explanations given in understandable terms
- Be allowed to see the patient frequently

Leske (1991) was one of the researchers who validated Molter's findings, and she grouped the needs of the families into the following five domains:

- Support
- Comfort
- Proximity
- Information
- Assurance

Although these quantitative studies list the needs of the families, the qualitative work of other nurse researchers captures what it is to be a family member of a critically ill patient. Jamerson et al. (1996) described the proximity phase of families' experiences as "hovering." This is a time when family members feel confused and tense as they wait until they can gain access to information about the patient or access to the patient. It has been described by Fulbrook, Allan, Carrol, and Dawson (1999) as maintaining a vigil—a phase during which the family feels a need to watch over the patient because they could not forgive themselves if anything happened while they were away. During this phase, the patient takes precedence, and family members want to be with him, to touch him, to talk to him, or even just to sit quietly near him. They want to provide comfort and obtain comfort for the patient.

As the patient's critical illness progresses, the family begins searching actively for information. "Not knowing is the worst part," according to Verhaeghe, Defloor, Van Zuuren, Duijnste, and Grypndonck (2005), and intensive anxiety can persist until the family believes sufficient information has been obtained. The family may then enter a "tracking phase" in which they observe, analyze, and evaluate the patient's care. It is a phase during which family

members want to observe the patient's care and be reassured that he is being cared for appropriately. McAdam and Puntillo (2009) summarize the findings from quantitative studies as "family members have negative emotions and multiple stressors that could affect family relationships, roles, and communication" (p. 205).

How Critical Care Nurses Best Meet the Needs of Patients' Families

Medina (2005) states, "In the critical care setting, families appear to have a profound beneficial impact on the response of the critically ill patient to the illness" (p. 1005). Families probably confer this beneficial impact by buffering the patient from some of the stress of the environment and illness as well as by serving as a resource for patient care. However, families are not able to perform these important functions if they are under severe stress. Therefore, it is important to the patient, not just to the family, that critical care nurses meet the needs of families. Health-care providers have been attempting to meet the needs of families of critically ill patients since they were identified almost 30 years ago. Some of the families' needs and evidence-based strategies being employed to meet these needs are described next.

Families' Need for Information

Information is the primary need identified by families of critically ill patients (Verhaeghe et al., 2005). Families have been reported to experience panic when patient updates are not provided in a timely fashion and they are scared to make caregiving decisions when they do not have current information (Riley, White, Graham, & Alexandrov, 2014). However, not all family members desire the same amount and type of information. Hickman, Daly, Douglas, and Clochesy (2010) discovered that some family members, described as monitors, needed much more information than other family members, described as blunters. The monitors were constantly on alert scanning the environment and wanting information. Only when they had received sufficient, consistent information could they relax. Blunters attempted to distract themselves from information they found threatening, and Hickman postulates that detailed information could be harmful to them. Thus, the nurse must carefully assess how much information family members desire and can assimilate.

Most families expect the physician to initially explain the patient's condition, prognosis, and treatment. However, they expect the nurse to clarify the information, provide information about the daily care and treatment of the patient, what the role of the family should be, and whether the patient is ready for transfer. To encourage family sense-making of the experience, Davidson (2010) recommends

that during a family visit, the nurse explain what is happening to the patient and at the end of the visit ask the family to explain their understanding of the patient's situation.

Essential for Collaboration

Families search out information from multiple sources, so a unified message from all members of the team is essential (Davidson, 2010).

When choosing a clinician to seek information, families prefer clinicians who spoke to them in understandable terms and were sensitive, honest, and unhurried. They remember clinicians who talked directly to them and kept their word more than clinicians who had the most information. Miracle (2006) summarized strategies that might be utilized with families:

- Regular family conferences to discuss patient goals and progress toward the goals can be helpful. Jacobowski, Girard, Mulder, and Ely (2010) state that since 50% of families do not understand either the patient's diagnosis, treatment, or prognosis, the conference should begin with the physician providing a summary in lay language of the patient's situation followed by an opportunity for the family to ask questions.
- Written instructional guides to provide information about the critical care unit, equipment, location of waiting areas, and phone numbers for the unit.
- A way to contact family members and for family members to contact the patient's nurse and primary physician should be established. If the family member has a cell phone this might be utilized; otherwise a pager can be provided.
- If at all possible, there should be consistency in the nurse providing care to the patient. Families indicate that it is not only important to receive an update from a nurse but that they prefer to receive it from the same nurse each time.
- Some hospitals provide written and verbal instructions to families to speak with the nurse whenever they have a question they want answered.

Families' Need for Proximity

Families consistently state that they would prefer open visiting hours, and patients and families are more satisfied when the unit has flexible rather than limited visiting hours (Sims & Miracle, 2006). Sims and Miracle stated there is abundant evidence that visitation is not stressful for patients and that it does not impede the delivery of nursing care. In fact, they described the following benefits of open visitation:

- Family members are more satisfied. They can arrange their schedules to their convenience and see the patient

whenever they need to. Therefore, families report they are less tired with a flexible visiting policy.

- Communication is facilitated between patient, family, and staff. Families believe they receive more information about the patients' conditions because they see the nurse more frequently and are able to ask questions.
- Open family visitation allows families more opportunity to provide emotional support and reassurance to patients, thus assisting with patients' recoveries.
- There is some evidence that open family visitation may have physiologic benefits for the patient by increasing the amount of patient rest while decreasing the patient's heart rate, blood pressure, and intracranial pressure.
- Flexible visiting hours allow more opportunities for nurse-family interaction and family teaching.

Sims and Miracle (2006) summarized by stating that a liberal visitation policy has been shown to improve patient outcomes. Hart, Hardin, Townsend, and Mahrle-Henson (2013) recommend tailoring a plan for family visitation at the time of patient admission to the ICU so that the family can have open access to the patient during the times when they are most available. However, critical care nurses must remain the gatekeepers for visitors. It becomes the responsibility of the critical care nurse, not a policy, to determine when a patient is benefiting from a visitor and when the patient would do better without a visitor present.

Some critical care nurses ask family members to leave while a procedure is being performed. However, there is accumulating evidence that it may benefit both the patient and family to have a family member available for the patient during intrusive procedures. Other ICUs are partnering with family members by encouraging family members to provide basic care such as applying lip moisturizer or combing a patient's hair. Mitchell, Chaboyer, Burmeister, and Foster (2009) found that such partnering had a significant positive effect on the families' experience. However, Agard and Maindal (2009) found that there are major differences in nurses' willingness to allow families present during intrusive procedures and involvement in basic care.

Reflect On

What has been your experience with having families partner in patient care? When have you asked a family to leave so that you could provide care? Were there specific patient or family behaviors that led you to believe that the patient was not benefiting from family presence? How did you ask the family to leave?

Families' Needs for Support and Assurance

Henneman and Cardin (2002) explained these needs by saying that families want to believe the patient is being

cared for in the "best way possible" and that everything that ought to be done is being done. When nurses demonstrate competence and caring in their dealings with the patient and family, families are reassured. When a nurse explains what care is being delivered to the patient, focusing on how and why it is expected to help the patient, this offers hope, support, and reassurance to the family.

Families are concerned with the comfort of the patient. They want to be reassured that every effort is being made to keep the patient comfortable. They also have comfort needs of their own. However, most studies indicate that families place their own comfort among the least important of their concerns (Verhaeghe et al., 2005). Issues related to comfort for families are access to telephones and bathrooms, availability of coffee and food while they are waiting, comfortable chairs and sources of distraction in the waiting room, a place to sleep that is in or near the hospital, and information about services available from the hospital.

Continuing Concerns of Families

Unfortunately, despite identifying the needs of families of critically ill patients nearly 30 years ago, critical care personnel have not been able to meet them with uniformity. Various gaps in meeting the needs of families still exist. According to Lederer, Goode, and Dowling (2005), the most commonly noted gaps in service include the following:

- There is a discrepancy regarding the amount and adequacy of information shared. Whereas healthcare providers believe that information is generally available and communication is adequate, family members indicate that communication of patient information is sporadic and inconsistent.
- Family members want more members of the family involved in decisions about patient treatment and care. Healthcare providers generally prefer fewer family representatives.
- Family members indicate that some support services are not offered or are offered inadequately. Specifically, these include a place for family members to sleep or rest, an opportunity for families of critically ill patients to speak with each other, assistance with travel arrangements, a supply of coffee near the waiting area, and better preparation for home care.

Attempts to Improve Families' Ongoing Concerns

Recognizing that family needs were not being met, the Critical Care Family Assistance Program (CCFAP) was developed. Its goal is to respond to the unmet needs of families of critically ill patients in hospital ICUs through the provision of educational and family support materials

(Lederer et al., 2005, p. 69S). CCFAP immediately identified that improvements in communication, environment, and provision of educational and informational materials were required.

Communication. Waiting for news about the patient is time consuming and anxiety provoking for families. Family members wait for long periods in a less than pleasant environment just to hear a few words from a doctor or nurse. CCFAP identified several events that consistently caused anxiety and irritation for families: “surgery being cancelled and the family not being informed; an extensive stay in the recovery room after surgery and the family kept unaware of the reason; contradictory information being received from different sources; and the inability to locate or talk to the physician” (Lederer et al., 2005, p. 73S).

Interventions developed by CCFAP units to allay these communication difficulties include:

- Designating specific individuals to be responsible for knowing each family, thus ensuring the family receives necessary information, that questions are answered, and appropriate conferences take place in a timely manner.
- Identifying who is responsible for handling the logistics and scheduling of family or interdisciplinary meetings, especially when several physicians are treating the patient.
- Facilitating weekly group meetings for the families of ICU patients. Some units have meetings that are facilitated by a social worker and a member of the pastoral care team in an attempt to make the ICU environment less frightening. The facilitators offer explanations about equipment, describe medical procedures, identify whom the family should contact if information is coming too slowly, and provide guidance on ways to be appropriately assertive without being disruptive.

Environmental changes to enhance family comfort. All of the hospitals involved in CCFAP determined that their waiting areas required improvement (Lederer et al., 2005, p. 73S).

They attempted to provide families a more relaxed, less institutional space by expanding the waiting area to make it less cramped, brightening the look of the room, or providing more comfortable furniture. Hospitals also sought to find space for sleeping rooms for families traveling great distances, providing coffee and soft drinks, supplying telephones, and placing televisions or videocassette recorders (VCRs) in the waiting area. Some units also designed and created consultation rooms for family conferences with physicians and staff.

Education and Informational Materials. “The CCFAP model seeks to assist ICUs in the important task of delivering unambiguous, but compassionate, information to families of ICU patients. Families require this information, first, to cope with their distress, but primarily to participate in making decisions about family members who cannot speak for themselves about critical care decisions” (Lederer et al., 2005, p. S73). The education provided is of two very different types: general medical information and very specific information about the family member. In addition, many sites supply material to assist family members in becoming familiar with staff so that family members can identify who is caring for the patient, what their responsibilities are, and how to contact the patient’s physicians. Also available might be pamphlets with frequently asked questions, information about advance directives, maps, and diagrams of the hospital, and simple explanations of the medical and surgical problems most commonly seen in the ICU. This information is also available in an electronic format at an information kiosk. Providing information in a variety of formats allows the family to access it when they are ready and to consult it repeatedly while they are considering their options.

Critical care nurses need to realize that families state they are not receiving adequate, consistent information. This does not mean that healthcare providers are not providing the information. Rather, it means that families may need critical care personnel to repeat information several times and provide it in multiple formats while the family attempts to assimilate it.

Critically Ill Patient Summary

Critically ill patients are physically unstable and require physiologic support. However, the patients have other needs as well, including the needs for comfort, relief of anxiety, and assistance with communication. Families of critically ill patients are often in crisis and have been shown to

need assurance, information, comfort, support, and proximity to the patient. When nurses’ competencies are matched with the patients’ needs and the multidisciplinary critical care team functions collaboratively to meet the needs of patients and families, patients’ outcomes are enhanced.

Why/Why Not?

A patient with a gunshot wound to the chest is ventilated and has an oro-gastric tube in place with tube feeding running at 50 mL/hr. If the patient tolerates the feeding, it is to be advanced by 10 mL every 6 hours. The feeding was last increased 6 hours ago. The nurse verifies the location the tube and aspirates 200 mL of residual. Four hours ago, the

nurse also aspirated 200 mL. The patient is not complaining of any discomfort or nausea. Should the tube feeding be advanced? Why or why not?

See answers to Why/Why Not? in the Answer Section.

Case Study

Harold Grier is a 45-year-old (200-pound) man who suffered a gunshot wound to his chest. On arrival in ICU from the PACU, he is intubated, has a ventilator, and a chest tube. His blood pressure is 170/100, heart rate 90, and he is grimacing and agitated. His last pain medication was morphine sulfate 4 mg IV 1 hour ago in the PACU. He is mouthing “Help me” to his nurse and pulling at his IV and ET tube. His blood gases and oxygen saturation are acceptable. The physician orders 4 mg morphine IV push now followed by a morphine infusion beginning at 4 mg/hr. In addition, he orders midazolam 0.05 mg/kg and a midazolam infusion to be started at 0.1 mg/kg/hr.

1. What is the dose of midazolam that the nurse will give IV push?
2. How many mg/hr will the nurse set the midazolam infusion?
3. Are these appropriate doses? Why or why not?

The nurse notes the patient’s levels of pain and agitation using the appropriate scales and administers the IV push

doses of morphine and midazolam. Then she begins to set up the IV infusions of morphine and midazolam. The morphine is available in the standard concentration for the hospital of 1 mg/mL and the nurse sets the infusion at 4 mL or 4 mg/hr. The midazolam available from the pharmacy is 50 mg/50 mL. When she adjusts the new pump, she does not notice that the standard concentration for that pump is 50 mg/100 mL. She allows the pump to do the calculation and sets the dose at the calculated mg/hr of midazolam.

Three hours later, at the change of shift when the oncoming nurse evaluates the patient, she is unable to arouse him and notes he is no longer initiating any respiratory effort. She reviews the settings on the pump and realizes that the midazolam has been infusing at the wrong dose and rate.

4. What should the nurse do?
5. What factors contributed to the error?
6. How could they have been prevented?
7. What factors compounded the error?

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 2.1 What does the AACN say are the characteristics of critically ill patients?
- 2.2 What are the priority needs identified by critically ill patients?
- 2.3 What strategies can a nurse utilize to communicate with a ventilated patient?
- 2.4 How can a nurse determine if a nonverbal patient is experiencing pain?
- 2.5 How should a nurse determine when a patient has received adequate sedation?
- 2.6 Why is enteral nutrition preferred over parenteral nutrition in the critically ill patient?
- 2.7 What are the advantages and disadvantages of open visiting hours in critical care?
- 2.8 How can a nurse meet the needs for information of families of critically ill patients?

See answers to Chapter Review Questions in the Answer Section.

References

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Chapter 3

Care of the Patient with Respiratory Failure

Destiny R. Brady, MSN, RN, CCRN

Abbreviations

ALI	Acute Lung Injury	IRV	Inspiratory Reserve Volume
ARDS	Acute Respiratory Distress Syndrome	NIV	Noninvasive Mechanical Ventilation
ARF	Acute Respiratory Failure	PEEP	Positive End-Expiratory Pressure
BIPAP	Bilevel Positive Airway Pressure	PSV	Pressure Support Ventilation
COPD	Chronic Obstructive Pulmonary Disease	RV	Residual Volume
CPAP	Continuous Positive Airway Pressure	SIMV	Synchronized Intermittent Mandatory Ventilation
ERV	Expiratory Reserve Volume	TLC	Total Lung Capacity
ET	Endotracheal Tube	VAP	Ventilator-Associated Pneumonia
FiO ₂	Fraction of Inspired Oxygen	VC	Vital Capacity
FRC	Functional Residual Capacity	V _T	Tidal Volume
FRV	Functional Residual Volume	VQc	Ventilation Perfusion
HFO	High-Frequency Oscillation		

Learning Outcomes

Upon completion of this chapter, the learner will be able to:

1. Briefly describe the normal anatomy and physiology of the respiratory system.
2. Compare and contrast types of respiratory failure and identify common causes of each type.
3. Evaluate the appropriate use of technology to support respiratory function including artificial airways, oxygen delivery devices, and mechanical ventilation.
4. Identify the clinical signs and symptoms of ALI/ARDS and prioritize appropriate nursing care for affected patients.

Introduction

Acute respiratory failure (ARF) is one of the most common disorders treated in critical care units and occurs when the pulmonary system is unable to adequately

exchange oxygen and remove carbon dioxide. ARF has many causes, but the most severe respiratory disorders are **acute lung injury (ALI)** and **acute respiratory distress syndrome (ARDS)** in which diffuse alveolar damage causes profound hypoxia. First identified in Ashbaugh,

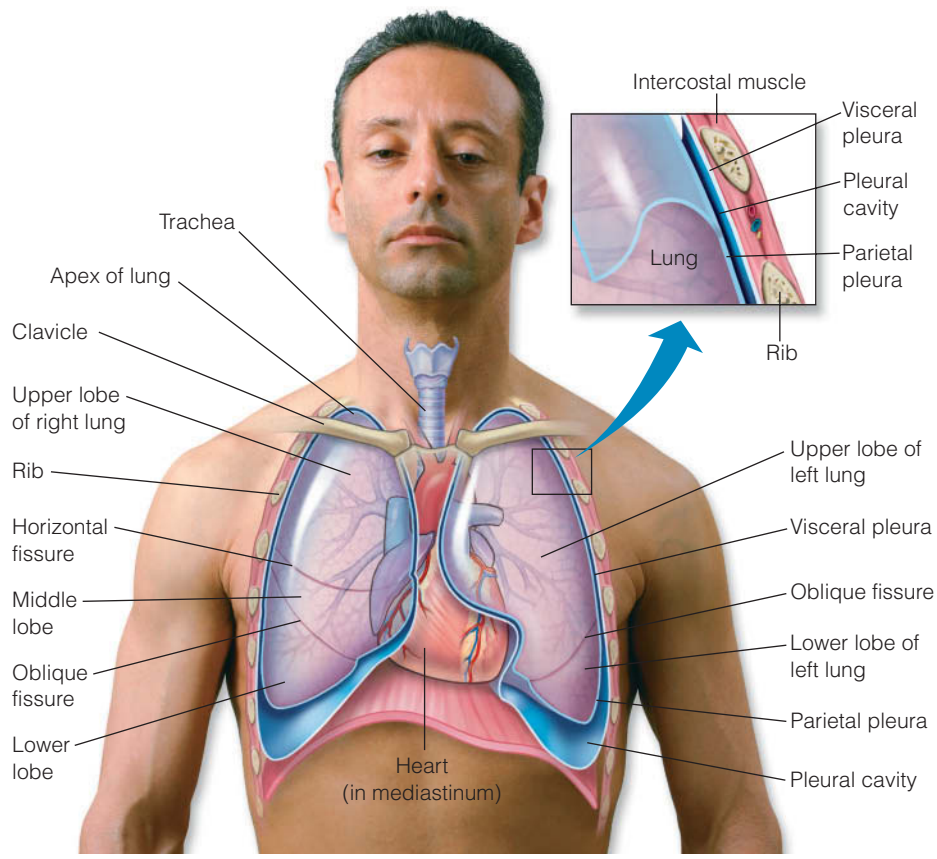


Figure 3-1 Anterior view of thorax and lungs.

Bigelow, Petty, & Levine (1967), ARDS will affect nearly 200,000 people in the United States each year with an estimated 39% mortality rate (Harman, 2014). This chapter will assist the nurse in caring for patients with ARF requiring advanced ventilatory support.

Anatomy and Physiology Review

In order to appropriately care for a patient with respiratory failure, it is important to understand normal anatomy and physiology of the respiratory system. Review the anterior view of the thorax and lungs (♦ Figure 3-1).

The lower airway comprises the **bronchial tree** and **bronchioles**, which branch into even smaller passages (♦ Figure 3-2). The lower airways are protected by the mucociliary escalator, a complex system of cilia that continuously propel respiratory secretions into the upper airways for expulsion. Further protection is provided by the cough reflex, triggered by the presence of foreign substances in the airways. Touching the carina is one method for stimulating this reflex. At the end of the bronchioles are over 300 million **alveoli**. It is within these small balloon-type structures that the exchange of gases between alveoli and capillaries occurs (♦ Figure 3-3).

Bronchial circulation does not participate in gas exchange. The left bronchiole circulation is rooted from the aorta, and right-sided bronchioles are supplied by arteries that originate in the intercostal, subclavian, or internal mammary arteries. Bronchial veins empty into the vena cava. Pulmonary circulation originates from the pulmonary artery, and the associated capillaries provide the conduits for gas exchange. Venules carry the newly oxygenated blood back to the pulmonary vein for circulation to the rest of the body.

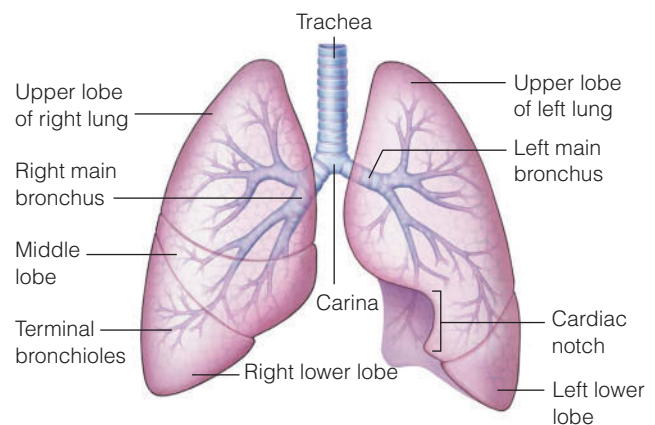


Figure 3-2 Respiratory passages.

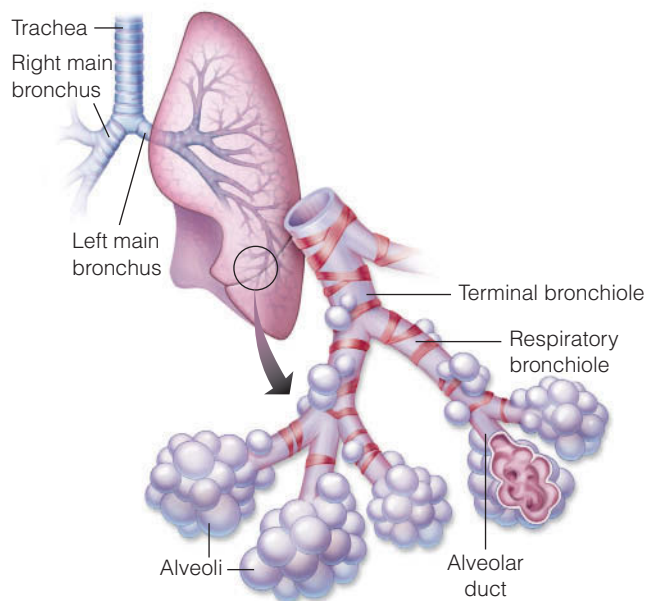


Figure 3-3 Lobule of the lung.

Physiology

It is important for the nurse to discriminate between the two functions of the respiratory system, which are ventilation and respiration. **Ventilation** is the mechanical act of moving air into and out of the respiratory tree and involves the musculoskeletal and nervous systems. **Respiration** involves the transport of oxygen and carbon dioxide between the alveoli and the pulmonary capillaries. Disruption of either of these processes results in respiratory failure.

Ventilation

When the diaphragm contracts, it flattens, increasing the volume of the thoracic cavity and creating a relative *negative*

intrapulmonary pressure, that is, a pressure less than atmospheric pressure. This change in intrapulmonary pressure is supported by the assistance of the intercostal muscles during normal respiration. During respiratory distress, the use of the intercostals becomes more pronounced, and the scalenes and sternocleidomastoid assist. These muscles may be referred to as accessory muscles. Additional effort of the accessory muscles increases the **work of breathing (WOB)**.

Compliance refers to the ability of the lungs and thorax to stretch and expand given a change in pressure. Compliance is decreased in pulmonary fibrosis and increased in emphysema and COPD. In conditions with low compliance, the lungs need more pressure to expand so the muscles of breathing must work harder. WOB increases in patients with chronic lung diseases that affect chest wall structure and/or decreased compliance. As described in the “Gerontological Considerations,” the normal changes of aging adversely affect WOB.

Normal pulmonary volumes and capacities are depicted in ♦ Figure 3-4. **Total lung capacity (TLC)** is the maximum volume the lungs can hold. **Tidal volume (V_T)** is the volume of one inhalation/exhalation cycle. **Functional residual capacity (FRC)** is the volume remaining in the lungs after a normal exhalation, whereas inspiratory capacity is the V_T plus the **inspiratory reserve volume (IRV)**. IRV is the volume of air one is able to inhale in addition to the V_T . The **residual volume (RV)** is the volume of air that remains in the lungs following forced expiration beyond normal expiration, also referred to as the **expiratory reserve volume (ERV)**. **Vital capacity (VC)** is the sum of the ERV, V_T , and IRV.

The brainstem controls autonomic respiration, whereas the cerebral cortex controls voluntary ventilatory effort. Central **chemoreceptors** detect the level of the hydrogen ion in the blood, whereas peripheral chemoreceptors are sensitive to oxygen, carbon dioxide, and

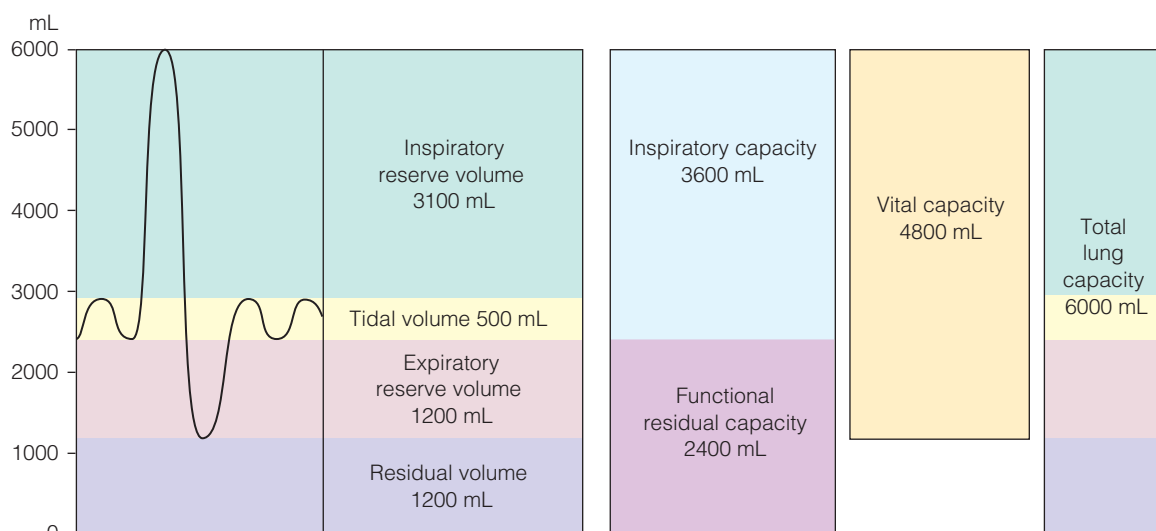


Figure 3-4 Lung volume measurements.

Gerontological Considerations

There are a number of age-related changes in respiratory function. These include:

- Decreased cough and laryngeal reflexes
- Decline in mucociliary escalator function
- Increased VQ mismatches
- Decrease in alveoli
- Increased AP diameter
- Increase in residual volume
- Decrease in respiratory muscle strength

For the healthy older adult, these changes do not appreciably affect normal activities. However, they contribute to a decreased respiratory reserve in the older

adult and make it more difficult for the older adult to respond when demand exceeds capacity.

When older adults develop respiratory failure, they often manifest nonspecific symptoms of hypoxemia such as agitation, confusion, disorientation, lethargy, dyspnea, and chest pain.

The chest x-ray of an older adult with ARDS, especially one with preexisting lung disease, may not clear for a period of days to weeks after the ARDS has begun to resolve. Therefore, chest x-rays should not be used to guide therapy.

Mortality and incidence of ARDS increases with advancing age (Harman, 2014; Villar, Sulemanji, & Kacmarek, 2014).

hydrogen ion levels. The chemoreceptors respond to changes in these levels by stimulating acceleration or slowing of the ventilation rate.

Respiration

Respiration is the process of transporting oxygen (O_2) and carbon dioxide (CO_2) across the alveolar capillary membrane. These gases are transported by the process of diffusion, which causes molecules to move from areas of higher concentration to areas of lower concentration. The elevated concentration of O_2 in the alveoli causes O_2 to diffuse into the capillaries, and the reverse is true for CO_2 . This process must occur through the several components of the respiratory membrane (♦ Figure 3-5). Alterations in any of these structures negatively impacts gas exchange.

Ventilation-Perfusion Relationships

Gas exchange relies on adequate perfusion of the alveoli. Normally, the volume in the conducting airways, such as the trachea and bronchioles, do not participate in gas exchange. This volume is known as anatomical dead space, because these structures are not designed for gas exchange. However, when an alveolus is not perfused, this alveolar cluster is termed an alveolar **dead space unit** because no gas exchange is occurring (♦ Figure 3-6). In dead space units, ventilation exceeds perfusion. Examples include pulmonary embolism or pulmonary infarct. Conversely, when an alveolus is inadequately ventilated in the presence of perfusion, this cluster is referred to as a **shunt unit**. Therefore, in shunt units, perfusion exceeds ventilation, such as with pneumonia or atelectasis. However, when ventilation and perfusion are impaired, the result is a **silent unit**. Silent units occur in severe ARDS or pneumothorax.

Oxyhemoglobin Dissociation Curve

O_2 is carried in the blood in two ways: About 97% is bound to hemoglobin (Hgb), known as O_2 saturation (SaO_2), and about 3% is dissolved in serum (PaO_2) (♦ Figure 3-7).

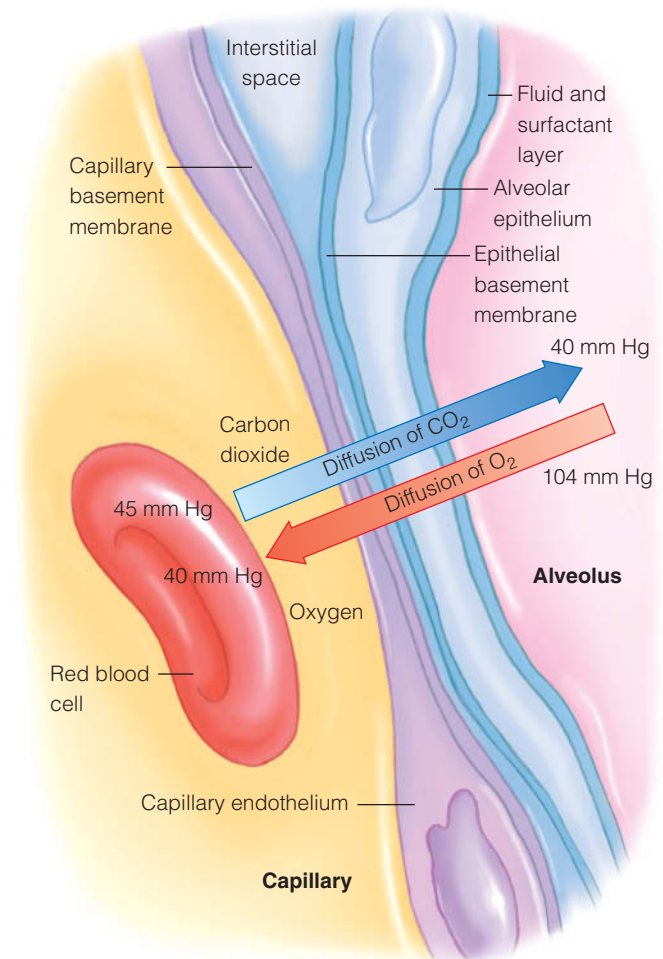


Figure 3-5 Ultrastructure of the respiratory membrane.

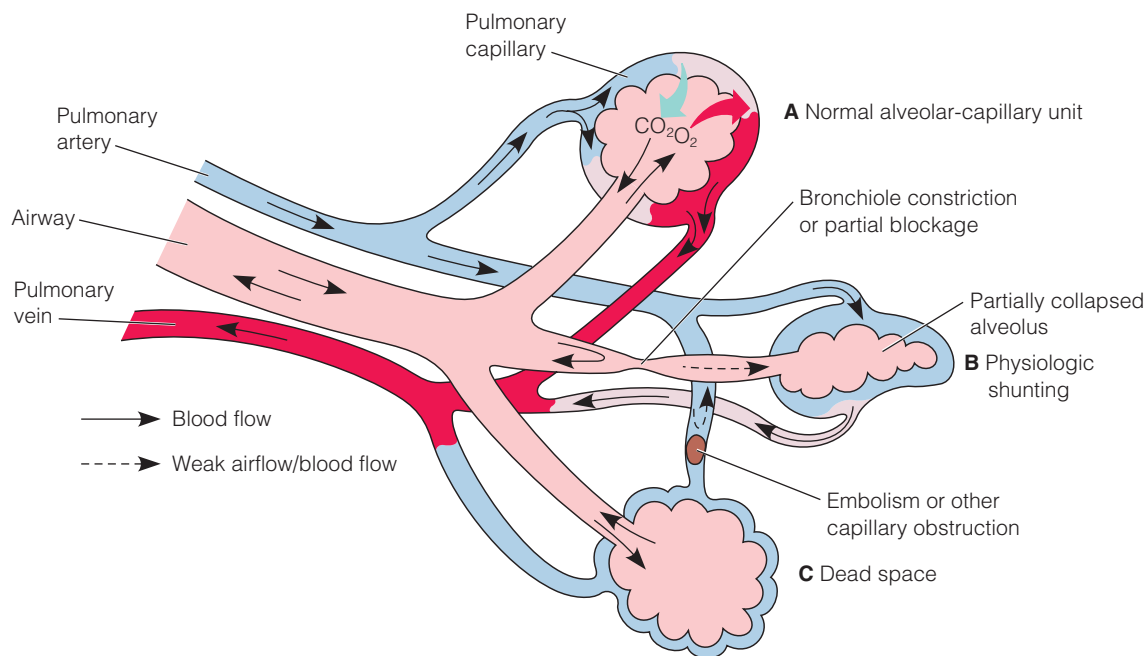


Figure 3-6 Ventilation-perfusion relationships. **A**, Normal alveolar-capillary unit with an ideal match of ventilation and blood flow. Maximum gas exchange occurs between the alveolus and blood. **B**, Physiologic shunting: A unit with adequate perfusion but inadequate ventilation. **C**, Dead space: A unit with adequate ventilation but inadequate perfusion. In the latter two cases, gas exchange is impaired.

The **oxyhemoglobin dissociation curve** describes the balance of these two transport methods. A shift in the curve means that the normally predictable affinity of Hgb toward O_2 is altered. This shift means that there is a change in the way O_2 is taken up by the Hgb molecule at the alveolar level

as well as a change in the way it is delivered to the tissues. With a shift to the right, there is a decrease in the O_2 saturation for any given PaO_2 (i.e., Hgb has less affinity for O_2). However, this increases O_2 delivery to the tissues (for a brief time) because the hemoglobin **R**eleases the O_2 more **R**eadily.

Factors that shift the curve to the **R**ight:

- Rise in body temperature (fever)
- Reduced pH (acidosis)
- Rise in CO_2 (hypercapnea)
- Rise in 2,3 diphosphoglycerate

A shift to the left means there is an increased O_2 saturation for any PaO_2 ; thus, the delivery of O_2 to the tissues is impaired. In other words, the Hgb **L**atches onto the O_2 .

Factors that shift the curve to the **L**eft:

- Low temperature (hypothermia)
- aLkalosis
- Low CO_2
- Low 2,3 diphosphoglycerate

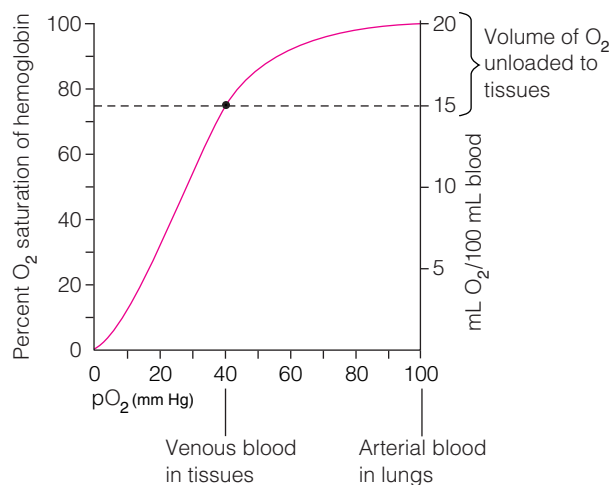


Figure 3-7 Oxygen-hemoglobin dissociation curve. The percent O_2 saturation of hemoglobin and total blood oxygen volume are shown for different oxygen partial pressures (pO_2). Arterial blood in the lungs is almost completely saturated. During one pass through the body, about 25% of hemoglobin-bound oxygen is unloaded to the tissues. Thus, venous blood is still about 75% saturated with oxygen. The steep portion of the curve shows that hemoglobin readily off-loads or on-loads oxygen at pO_2 levels below about 50 mm Hg.

The oxyhemoglobin dissociation curve is clinically important because it helps the clinician understand the patient's tissue oxygenation. Further, it reminds the clinician that saturations greater than 90% do not necessarily guarantee adequate oxygen delivery to the tissues for every patient. It is important to note the steep decrease in PaO_2 once the SaO_2 falls below 90%. These levels of O_2 are unsafe, and the nurse should evaluate the patient closely with the goal of restoring optimal oxygenation as soon as

Table 3-1 Arterial Blood Gases: Normal Adult Values

PaO ₂	80–100 mm Hg
SaO ₂	93–99%
pH	7.35–7.45
PaCO ₂	35–45
HCO ₃	22–26

possible. 2,3 Diphosphoglycerate (2,3 DPG) is normally found in red blood cells and increases in response to an acute need for more tissue O₂ (causing the Hgb to release the O₂ more readily). Decreases in 2,3 DPG may occur in septic shock, hypophosphatemia, and blood transfusions.

Arterial Blood Gases (ABG). An ABG directly measures the pH of the blood, along with the partial pressure of O₂, CO₂, bicarbonate ion, and saturation of Hgb. This information is crucial to adequate treatment of respiratory illness. When these values are abnormal, they can be significant clues to respiratory problems as well as needed changes in ventilator or oxygen settings. Normal values are listed in Table 3-1. Interpretation of ABGs is a skill easily mastered with sufficient practice. One should approach these results in a systematic fashion; these steps are described in Box 3-1.

To evaluate oxygenation, consider the PaO₂ and the SaO₂. The PaO₂ reflects the partial pressure of O₂ in arterial blood, whereas the SaO₂ represents saturation of Hgb with O₂. When interpreting these values, consider how much oxygen the patient is receiving. Is the patient receiving

enough FiO₂? Are the values very low despite receiving high FiO₂? Oxygen diffusion can be disrupted by several factors that alter ventilation-perfusion relationships including disease process, sedation, and metabolic needs.

Next, consider the pH, which is a reflection of the concentration of the hydrogen ion in serum. Low values indicate acidosis, whereas elevated values indicate alkalosis. If the PaCO₂ is elevated while the pH is decreased, this indicates respiratory acidosis. If the PaCO₂ is decreased while the pH is elevated, this indicates respiratory alkalosis. If the HCO₃ is decreased along with the pH, the likely cause is metabolic acidosis, whereas metabolic alkalosis is seen when the HCO₃ is elevated along with the pH.

The body’s ability to function normally requires tight control of the pH level. Compensatory mechanisms help return the body to a normal acid-base balance as quickly as possible. For example, if a patient is experiencing an acute metabolic acidosis, the chemoreceptors detect the increase in hydrogen ions and stimulate the brainstem to increase respiratory rate, essentially using the respiratory system to compensate for the affected metabolic state. The net effect is a decrease in PaCO₂, as the body “blows off” CO₂, resulting in a shift of acid-base balance toward the alkalotic side, righting the balance to the desired range of 7.35 to 7.45. Thus, a normal pH may indicate a compensated state if in the context of abnormal PaCO₂ and HCO₃ values. However, compensation is not an instantaneous process. If a patient is in an acute respiratory acidosis, the metabolic compensation may take several days. If ABGs are evaluated during this time frame, the pH may still be abnormal along with the CO₂ level; however, the HCO₃ level will demonstrate an increase as the base tries to counteract the excess acid. These ABGs demonstrate a partially compensated state.

Example:A patient on room air has the following blood gas results:

pH = 7.30
PaCO₂ = 58
PaO₂ = 95
HCO₃ = 24

In this example, the PaO₂ is greater than 90 and is considered adequate. The pH is less than 7.35 and is thus acidic. The PaCO₂ is high, and because it is the “acid,” this results in acidosis. The HCO₃ is within normal limits. Because the pH remains outside normal limits, and the PaCO₂ is elevated, this result would be analyzed as respiratory acidosis.

Measurements of Gas Exchange Adequacy

Additional factors are important to consider if a patient has a severely compromised respiratory status, such as ARDS. One factor is the comparison of the alveolar oxygen content

Box 3-1 Steps for Interpreting Arterial Blood Gases

- Evaluate Oxygenation:**
Consider PaO₂ and SaO₂. Is there an oxygen problem?
- Evaluate pH:**
Is it alkaline, acidic, or normal?
- Evaluate PaCO₂:**
Is it too low (alkaline), too high (acidic), or normal? Is my patient ventilating too much or too little? Is this a “respiratory” or ventilation problem?
- Evaluate HCO₃:**
Is it too high (alkaline), too low (acidic), or normal? Is this a “metabolic” problem?
- Reconsider pH:**
Has compensation occurred? Is the pH normal despite the presence of abnormal HCO₃ or PaCO₂ (full compensation)? Is the PaCO₂ or HCO₃ balancing the other out (i.e., HCO₃ high and PaCO₂ high) despite an abnormal pH?

(PAO_2) to arterial oxygen content, or PaO_2 . Many ABGs report this value and the calculation is not routinely required.

Calculation of Alveolar Oxygen Content:

$$\text{PAO}_2 = \text{FiO}_2(\text{P}_\text{B} - \text{P}_{\text{H}_2\text{O}}) - \text{PaCO}_2/\text{RQ}$$

Where FiO_2 is expressed as a decimal, P_B is barometric pressure (760), $\text{P}_{\text{H}_2\text{O}}$ is pressure of water vapor (47), and RQ is respiratory quotient (0.8).

A patient on 50% O_2 has the following blood gas results:

$$\begin{aligned}\text{pH} &= 7.30 \\ \text{PaCO}_2 &= 64 \\ \text{PaO}_2 &= 95 \\ \text{HCO}_3 &= 24\end{aligned}$$

At first glance, the PaO_2 of 95 seems acceptable. However, this equation demonstrates why it is crucial to include O_2 therapy when reporting blood gases:

$$\begin{aligned}\text{PAO}_2 &= 0.5(760 - 47) - 64/0.8 \\ &= 0.5(713) - 80 \\ &= 356.5 - 80 \\ &= 276.5\end{aligned}$$

This means that 276.5 mm Hg of O_2 is delivered to the alveoli; however, only 95 torr is detected in the artery.

A normal difference (gap or gradient), between the alveolus and artery is less than 20 mm Hg. Comparing the O_2 content of the alveoli to the arterial content, or the A-a gradient, provides a perception of the presence and severity of impaired gas exchange. However, it is not diagnostic

of cause. Nevertheless, it is a useful tool in determining if a ventilation-perfusion mismatch is present.

Calculation of A-a Gradient:

$$\begin{aligned}\text{A-a gradient} &= \text{PAO}_2 - \text{PaO}_2 \\ (\text{Normal value is less than } 20) \\ 276.5 - 95 &= 181.5\end{aligned}$$

Thus, a significant impairment of gas exchange exists. This gradient, or gap, may be tracked to trend gas exchange issues and monitor therapy for outcomes.

Due to the limitations of the A-a gradient, nurses are more commonly estimating intrapulmonary physiologic shunting by calculating a **P/F ratio**. This ratio represents the relationship between the PaO_2 and the FiO_2 ($\text{PaO}_2/\text{FiO}_2$) with normal values in healthy adults ranging from 400 to 500 mm Hg. As the ratio grows smaller, the patient's shunt is more severe, such as occurs in ARDS.

Results of ABG analysis are essential to assist the clinician in proper treatment of underlying disorders. Table 3-2 lists the causes as well as signs and symptoms of acid-base disturbances. All critical care nurses should be proficient in analysis of this foundational diagnostic study.

Types of Respiratory Failure

Respiratory failure is classified as one of two types linked to failures of ventilation or respiration (Table 3-3).

Type I respiratory failure, or hypoxemic failure, stems from a disruption of O_2 transport from the alveolus to arterial flow. Respiration is impeded. It may be a blockage of

Table 3-2 Possible Causes, Signs, and Symptoms of Acid-Base Disturbances

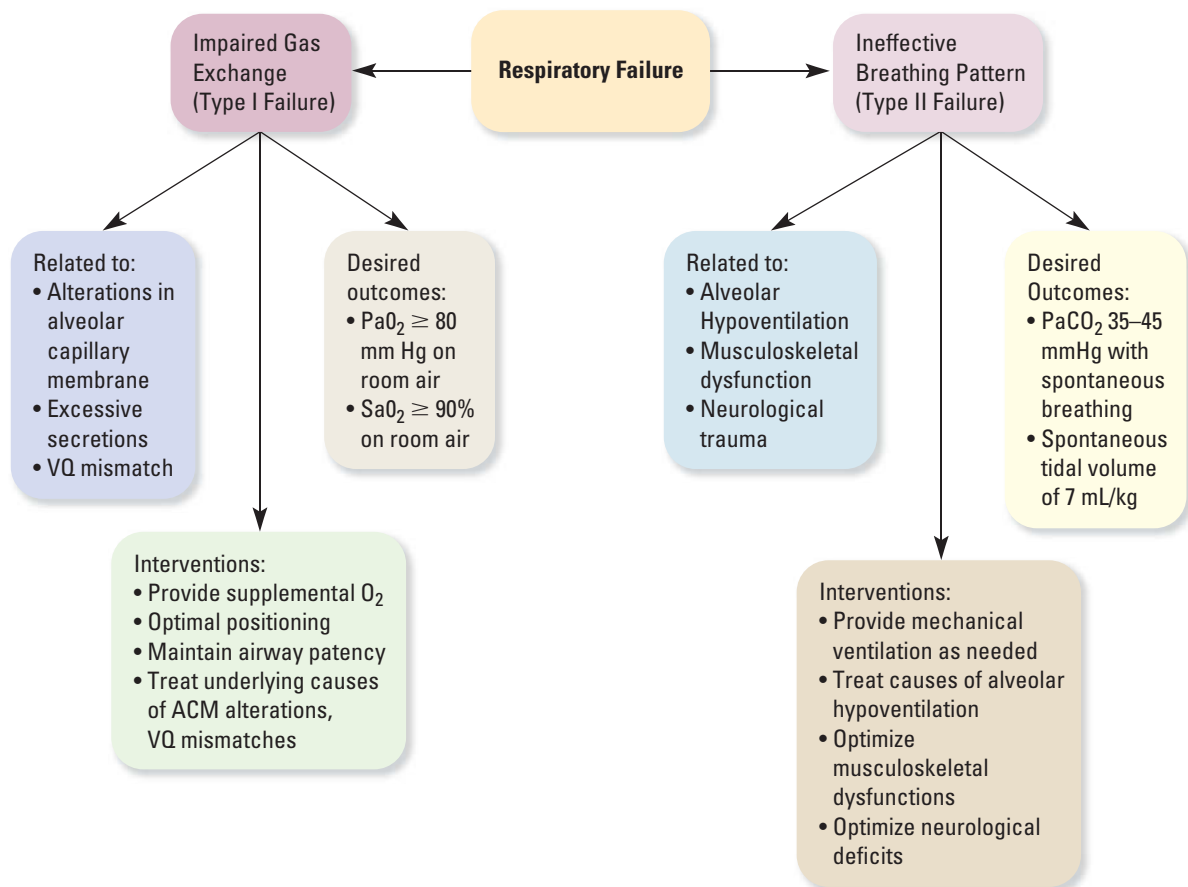
Disturbance	Frequent Causes	Signs and Symptoms
Respiratory Acidosis $\text{PaCO}_2 > 45$ mm Hg $\text{pH} < 7.35$	Central nervous system depression Hypoventilation Atelectasis Bronchial obstruction Severe pulmonary infections Heart failure and pulmonary edema Pulmonary embolism	Restlessness Confusion Lethargy Decreased responsiveness Tachycardia Dysrhythmias Dyspnea Respiratory distress
Respiratory Alkalosis $\text{PaCO}_2 < 35$ mm Hg $\text{pH} > 7.45$	Central nervous system lesions Hyperventilation Anxiety, fear, or pain Fever and sepsis	Lightheadedness Confusion Tingling in hands Dysrhythmias and palpitations
Metabolic Acidosis $\text{HCO}_3 < 22$ mEq/L $\text{pH} < 7.35$	Excess acid Anaerobic metabolism (shock) Renal failure Ketoacidosis Base deficit Diarrhea Intestinal fistulas	Mental status changes Hyperventilation (possibly Kussmaul respirations) Dysrhythmias
Metabolic Alkalosis $\text{HCO}_3 > 26$ mEq/L $\text{pH} > 7.45$	Base excess Lactate administration Excessive administration of bicarbonate Acid deficit Vomiting or nasogastric suction Hypokalemia and hypochloremia	Mental status changes Hypoventilation Muscle cramps and twitching

Table 3-3 Types of Respiratory Failure

Type	Causes	Nursing Diagnoses	Signs and Symptoms	Treatment
Type I Hypoxemic	Pneumonia, pulmonary edema, ARDS, aspiration, atelectasis	Impaired Gas Exchange	• Decreased SaO₂ • Decreased PaO₂ • Increased respiratory rate	• Oxygen therapy • Treat underlying cause
Type II Hypercapneic Hypoxemic	COPD, neurological system failure to stimulate respiration, muscular failure, or skeletal alterations	Ineffective Breathing Pattern	• Increased PaCO₂ • Decreased pH • Decreased SaO₂ • Decreased PaO₂ • RR may be increased or decreased	• Improve ventilation • May require mechanical ventilation • Treat underlying cause as able

mucus or fluid or disease in the basement membrane of the capillaries. This type of failure is linked to the nursing diagnosis of Impaired Gas Exchange. Causes of hypoxemic failure include pneumonia, cardiogenic pulmonary edema, ARDS, aspiration, and atelectasis. **Type II respiratory failure**, or hypoxemic hypercapneic failure, originates in musculoskeletal or anatomical lung dysfunction or suppression. Ventilation is not sufficient. This may be due to alterations in lung anatomy (COPD), failure of the neurological system to stimulate respiration (excess narcotic administration, head injury), muscular failure (muscular dystrophy, amyotrophic lateral sclerosis, or Guillain-Barré),

or skeletal alterations (kyphosis or fractures due to trauma). These conditions result in failure to adequately blow off CO₂ and take in oxygen-rich air from the environment. CO₂ is trapped in the alveoli, resulting in the rise of PaCO₂. Although the body is capable of O₂ transport, the high concentration of CO₂ in the alveolus causes **hypercapnea**, and the inability to replace the CO₂ with O₂ results in **hypoxemia**. Nursing diagnoses linked to Type II failure include Impaired Spontaneous Ventilation or Ineffective Breathing Pattern. (See “Nursing Diagnoses.”) The conditions may coexist, as in a patient with chronic emphysema who has an acute pneumonia.



Visual Map 3-1 Respiratory Failure Summary

Nursing Diagnoses

Mechanically Ventilated Patients

- Impaired gas exchange related to alveolar capillary membrane changes and ventilation perfusion imbalance
- Ineffective breathing pattern related to hypoventilation, hypoxia, and/or inflammatory process
- Ineffective airway clearance related to presence of artificial airway and secretions in the bronchi
- Impaired spontaneous ventilation related to respiratory muscle fatigue
- Impaired verbal communication related to presence of artificial airway
- Dysfunctional ventilatory weaning response related to ineffective airway clearance, sleep pattern disturbance, inadequate nutrition, uncontrolled pain, or fatigue
- Decreased cardiac output related to altered preload and stroke volume
- Risk for Infection related to presence of artificial airway

Building Technology Skills for Respiratory Failure

O₂ administration, airway maintenance, and management of mechanical ventilation are essential skills for the nurse caring for a patient with respiratory failure.

Oxygen Administration

Oxygen therapy is the administration of supplemental oxygen to relieve signs and symptoms of hypoxia. Oxygen therapy is indicated for individuals with hypoxia as evidenced by decreased PaO₂, SaO₂, or SpO₂ levels. Signs and symptoms of hypoxia include tachypnea, tachycardia, SOB, increased WOB, restlessness, hypertension, confusion, lethargy, and dysrhythmias. The goal of O₂ therapy is to correct alveolar and tissue hypoxia. This may be demonstrated by an increase in the PaO₂ to acceptable levels with a concurrent reduction in the respiratory rate and work of breathing. By increasing the concentration of oxygen inspired, oxygen will be more likely to diffuse into the blood stream.

Oxygen Delivery

There are a wide variety of methods for administering O₂. These may include either low-flow or high-flow oxygen delivery devices. Low-flow devices are common, but depending on the severity of illness, they may not meet the oxygenation needs for all patients. Some patients may require a pharyngeal airway to maintain airway patency.

Low-Flow Oxygen Therapy

The simplest low-flow device is the **nasal cannula**, also known as nasal prongs due to the two prongs designed for inserting into the nares. Nasal O₂ is well tolerated and can be given at rates up to 6 liters per minute (6 LPM). Advantages of nasal O₂ include ease of administration and patient comfort. Disadvantages include the inability to quantify the FiO₂ being administered due to variation in breathing patterns.

Simple masks allow for higher FiO₂, but precise calculation of FiO₂ is again dependent on the patient's respiratory pattern. Flow rates administered via simple masks range from 8 to 12 LPM. The partial rebreather mask has a reservoir bag to capture the 100% O₂ coming through the tubing that blends with expired air. Vents on either side of the mask allow the majority of exhaled air to escape, which provides between 40% and 60% FiO₂. **Non-rebreather masks** have valves over the ports that allow exhaled air to escape but prevent room air from being inhaled. This mask is capable of delivering up to 100% O₂. The **venturi mask** provides more precise FiO₂ levels when used properly. This device has an adjustable dial imprinted with the desired FiO₂ and LPM flow. A port or blender is attached around the connection of the O₂ tubing and the adapter. This port must remain in place and uncovered in order to ensure accurate O₂ delivery.

High-Flow Oxygen Therapy

High-flow oxygen therapy is designed to deliver oxygen at a higher flow rate than the patient's inspiratory flow rate (Fealy et al., 2015; Maggiore et al., 2014). Low-flow devices are unable to exceed this flow rate, and therefore room air is mixed into the inspired gas at variable rates depending on the patient. These devices may also have some positive pressure effect and can create an oxygen reservoir in the physiologic dead space of the upper airways (Maggiore et al., 2014). This therapy can be delivered through a wide nasal cannula, nasal pillow, or face-mask (♦ Figure 3.8). The delivered gas is heated and humidified and may help improve comfort and promote the mobilization of secretions (Maggiore et al., 2014). It is important to clear the condensation from the tubing periodically and position the fluid collection reservoir below the level of the face to reduce the risk being splashed with the condensation.

Pharyngeal Airways. Some patients are unable to maintain ventilation due to airway issues, such as ineffective cough or bronchial spasm. Pharyngeal airways are designed to prevent the tongue from occluding the upper airway. Choosing an oral or nasal airway of an appropriate length and proper placement important to the function of the airway.

Nasopharyngeal airway tubes are made of either plastic or rubber and are available in sizes ranging from



Figure 3-8 Optiflow mask.

infant to adult (♦ Figure 3-9A). To select the correct size for an adult, the nurse should measure the airway and ensure it reaches from the earlobe to the tip of the nose. The tube should be lubricated prior to insertion and guided through the nares toward the posterior pharynx. One benefit of the nasopharyngeal airway is that it can be used in a conscious patient.

The **oropharyngeal airway** is made of rigid plastic and is available in sizes for all age groups (♦ Figure 3-9B). Proper size is crucial. The nurse should measure the size by placing the airway at the corner of the mouth and seeing that it reaches to the corner of the jaw. Oropharyngeal

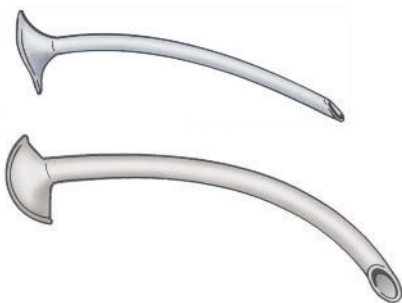


Figure 3-9A Pharyngeal airways: Nasopharyngeal airways.

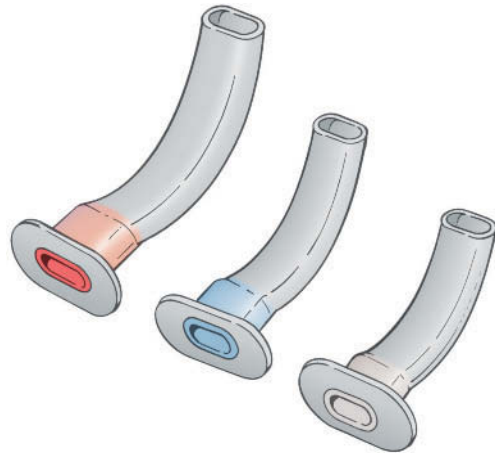


Figure 3-9B Pharyngeal airways: Oropharyngeal airway.

airways are contraindicated in patients who are alert and have an intact gag reflex. To insert this airway, the nurse displaces the patient's tongue downward using a tongue depressor and slides the airway over the patient's tongue into the oropharynx. It is easier to insert the airway with the distal end pointed up toward the nose and flip it 180 degrees once inside the mouth.

Mechanical Ventilation

Mechanical ventilation assists the breathing process in patients who cannot effectively ventilate independently. Current technology allows for noninvasive and invasive ventilatory support. While ventilation does not guarantee improved gas exchange by ensuring ventilation, oxygenation may improve, and carbon dioxide levels may be reduced.

The desired outcomes of mechanical ventilation are:

- Relief of symptoms of respiratory distress
- Rest for fatigued muscles of respiration
- Decrease in the work of breathing
- Improvement in oxygenation
- Improvement in ventilation
- Restoration of acid-base balance
- Stabilization of the chest wall
- Provision of sedation/anesthesia

Noninvasive Mechanical Ventilation

Noninvasive mechanical ventilation (NIV) supports the ventilatory efforts of patients without the insertion of invasive airway devices. Early use of NIV may circumvent the need for invasive mechanical ventilation (McNeill & Glossop, 2012). NIV has been used effectively for exacerbation of COPD, obesity hypoventilation syndrome,

cardiogenic pulmonary edema, and lung contusions (McNeill & Glossop, 2012). It is contraindicated in patients with apnea, recent airway or gastrointestinal surgery (Cross, 2012; Kato, Suda, & Kasai, 2014). NIV should be used cautiously in patients with an increased risk of aspiration, excessive secretions, swallowing impairment, inadequate airway protection, severe hypoxemia or acidosis, multi-organ failure, hepatic congestion, low cardiac output, and when an adequate interface fit is not possible (Kato et al., 2014).

NIV provides mechanical ventilation without the use of an artificial airway. This requires a machine that provides ventilation through the upper airway using an interface with a tight seal such as nasal pillows, an oronasal mask, or a full-face mask. Individual anatomy must be accommodated. Alternatively, a helmet may be used to improve patient tolerance but increased carbon dioxide rebreathing may occur (Esquinas Rodriguez, Papadakos, Carron, Cosentini, & Chiumello, 2013). The most commonly used interface is the oronasal mask (♦ Figure 3-10).

Two modes are available for NIV: **continuous positive airway pressure (CPAP)**, and **bilevel positive airway pressure (BiPAP)**. CPAP delivers one continuous positive pressure throughout inspiration and expiration. Typical pressure settings are between 5 and 15 cm H₂O (Cross, 2012). CPAP helps recruit or open alveoli and prevents atelectasis during expiration (Cross, 2012). This allows more lung units to be available for gas exchange and increases the partial pressure of oxygen in the alveoli facilitating oxygen diffusion into the bloodstream (Cross, 2012).

BiPAP allows the clinician to set a higher **inspiratory pressure (IPAP)** and a lower **expiratory pressure (EPAP)**. Frequently these settings are reported as a ratio of IPAP/

EPAP, for example 15/5, is 15 cm H₂O IPAP and 5 cm H₂O EPAP. Theoretically, adding IPAP reduces the work of breathing further and is more effective than CPAP, but studies have not shown a significant difference in outcomes other than an increase in patient comfort due to the reduced exhalation effort (Cross, 2012).

Complications. NIV reduces preload and afterload by decreasing venous return and decreasing left ventricular systolic wall stress (Kato et al., 2014). These hemodynamic effects improve stroke volume without increasing myocardial oxygen consumption, but can reduce cardiac output and lead to hypotension, especially in hypovolemic patients (Cross, 2012). Other complications of NIV include aspiration, skin breakdown, gastric insufflation, barotrauma, dryness, discomfort, and anxiety (Kato et al., 2014).

Nursing Care: Noninvasive Mechanical Ventilation

It is important to monitor respiratory pattern, depth, effort, and oxygenation status. The effects of NIV on gas exchange are evident in ABGs within 15 minutes (Cross, 2012).

Enhancing Comfort

The positive pressure and tight fit of the mask may cause anxiety in some patients. Nurses should stay with the patient during initiation of therapy and administer ordered anxiolytics when appropriate. Mucous membrane dryness can occur and patients will need regular oral hygiene. The tight fit of the mask puts the patient at risk for tissue ischemia. Pressure-induced ischemia at 35mmHg for longer than 2 hours can cause tissue necrosis (Ahmad, Venus, Kisku, & Rayatt, 2012). Pressure ulcers on the bridge of the nose are common and great care should be taken to assess and protect the skin. Silicone gel sheets or other appropriate dressings applied under the mask may reduce injury (Ahmad et al., 2012). Periodic removal of the mask should be considered when possible to off-load the pressure on the skin.

Promoting Safety

Anxiety and restlessness related to hypoxemia and respiratory distress is common in patients who require NIV. Nurses should stay near the patient during the initiation of therapy until ventilation is stabilized. The nurse should ensure that the patient is alert enough to manage airway clearance and has the ability to remove the mask if emesis should occur. Restraints are inappropriate for these patients.



Figure 3-10 Sleep Apnoea Syndrome

SOURCE: Amelie-Benoist/BSIP/Science Source

Placing the head of the bed at 45 degrees maximizes respiratory effort and minimizes the risk of aspiration. Forcing the ventilatory volume into the oropharynx makes gastric insufflation a potential complication of NIV. The nurse monitors the patient for gastric distention. In the event the patient is unable to tolerate NIV or ventilation cannot be stabilized, invasive mechanical ventilation is indicated.

Providing Nutrition

NIV is easily removed and reapplied to facilitate communication, eating, drinking, and airway clearance (Cross, 2012). When the patient's conditions allow it, the mask can be removed temporarily to allow for meals and oral hygiene. The patient may need a nasal cannula or high-flow oxygen device according to his or her tolerance during these periods. The nurse should carefully assess swallowing ability and mucous membrane condition prior to allowing meals. Close supervision and continuous monitoring of oxygen saturation and respiratory status should be performed.

Invasive Mechanical Ventilation

Invasive mechanical ventilation is provided by a positive-pressure ventilator. An example is displayed in ♦ Figure 3-11. In invasive mechanical ventilation, positive pressure is applied at the patient's airway by means of a nasotracheal, endotracheal, or tracheostomy tube.



Figure 3-11 Positive-pressure ventilator.

Nasotracheal and Endotracheal Tubes. **Endotracheal tubes** are placed into the trachea via the mouth in order to establish a patent and stable airway for patients. **Nasotracheal tubes** are placed into the trachea via the nose in circumstances when anatomy, trauma, or the procedure prohibits placement directly via the mouth. There are numerous sizes available, from neonate to adult. The size is dictated by the patient's anatomy and the preference of the provider inserting the tube. The tube has a 15-mm adapter for adults, which connects to conventional resuscitation bag systems, as well as to the ventilator tubing. A radiopaque line that will appear prominently on x-ray runs the length of the tube, assisting assessment of tube location.

The distal end has an inflatable cuff, inflated via a port balloon located at the proximal end of the tube (♦ Figure 3-12). The cuff serves two purposes. First, it essentially seals the trachea so that air entering via the tube cannot escape between the tube and trachea. Second, it helps secure the level of the tube within the trachea. Finally, the cuff helps protect the airway from aspiration of gastric contents. Some have subglottic suctioning capability to clear secretions that gather around the inflated cuff to prevent aspiration.

Prior to non-emergent intubation, the nurse should ensure that the patient and family understand the procedure. The physician may choose to sedate patients, and the nurse should have ready access to needed medications. Common medications for this purpose include, but are not limited to, etomidate, succinylcholine, rocuronium, propofol, and midazolam. Many physicians utilize rapid sequence intubation when intubating an unstable patient. Equipment needed for intubation is listed in Box 3-2.

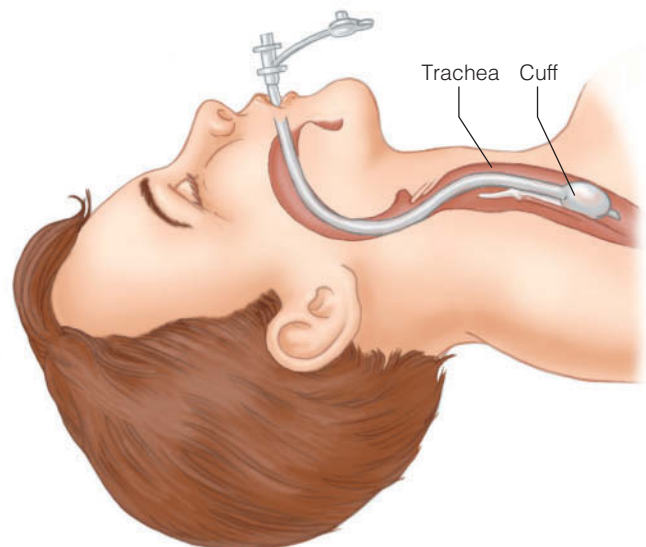


Figure 3-12 Endotracheal tube in place.

Box 3-2 Equipment Needed for Endotracheal Intubation

- Laryngoscope (test to ensure bulb is functional)
- Endotracheal tube of choice
- 10-cc syringe to inflate bulb
- Sterile lubricant (anesthetic gel may be used)
- Stylet (to help tube hold its shape)
- Stethoscope to auscultate for placement
- Device or tape to secure tube following placement
- Personal protective equipment
- Suction with orotracheal suction device
- Bag-valve mask
- Oxygen source available
- Medications for sedation
- CO₂ detector

The placement of a nasotracheal or endotracheal tube is referred to as **intubation**. During the intubation procedure, the nurse is responsible for monitoring oxygenation status as well as cardiovascular status. The nurse should alert the physician to decreases in arterial oxygen saturation (SaO₂) below 90% as this change should halt the procedure, and the patient should be manually ventilated until saturations exceed 90%. Immediately following the intubation, the nurse should ensure that the lungs are auscultated to check for accurate placement of the tube; air movement should be heard in the apices and bases of the lungs bilaterally. A CO₂ detector should be used to ensure the tube is in the trachea and not the esophagus. The stomach does not routinely contain CO₂; therefore, if CO₂ is detected, the tube is accurately placed in the trachea. Once these informal tests verify placement, the cuff is inflated, and the tube secured. The nurse should note the marking on the tube at the lip and document this finding as this enables the staff to monitor the stability of tube placement. Afterward, a portable chest radiograph (chest x-ray) should be ordered immediately to obtain verification of optimal tube placement. Optimal location for the distal tip of the endotracheal (ET) tube is 2 to 3 cm above the carina but beneath the clavicles. The nurse should monitor for the patient to “awaken” as the sensation of choking may create anxiety. The section on sedation in Chapter 2 [∞](#) has further information on maintaining sedation in mechanically ventilated patients. Although life-saving, endotracheal intubation still presents potential complications, including oral-pharyngeal or tracheal trauma, bronchospasm, loss of teeth, vocal cord dysfunction, ulcerations, and infections.

Tracheostomy Tubes. A **tracheostomy** is indicated for patients requiring long-term mechanical ventilation (greater than 21 days) or airway protection and patients with airway obstruction (Morris, Whitmer, & McIntosh, 2013). The procedure involves an incision in the neck at the cricothyroid membrane, thus accessing the trachea and creating a stoma through which the tracheostomy tube is inserted. The procedure may be done at the bedside or in the operating suite. Advantages of the tracheostomy tube are decreasing laryngeal injury as well as improving comfort and promoting activities of daily living (Morris et al., 2013). Tracheostomies may shorten the duration of mechanical ventilation (Dawson, 2014).

The tracheostomy tube is usually constructed of plastic and may have one or two lumens (♦ Figure 3-13). Either type has a cuff at the distal end of the tube, which is inflated through a pilot balloon. An obturator functions as a stylet to facilitate replacement of the tube should it become dislodged unintentionally. The single lumen type provides decreased resistance to airflow, thus allowing the patient to breathe with less effort. The double lumen tube consists of an outer cannula that remains in place with a removable inner cannula. This inner cannula is useful in secretion management, as built-up secretions may be removed with the cannula for cleaning or, if disposable, discarded and replaced with a new sterile cannula.

Complications of tracheostomy tubes include bleeding, infection, ulceration, dysphonia, tube obstruction, and the development of fistulas. Nurses are responsible for providing tracheostomy care according to agency protocol to clean the stoma and change the dressings or ties as needed. It is important for the nurse to ensure that emergency

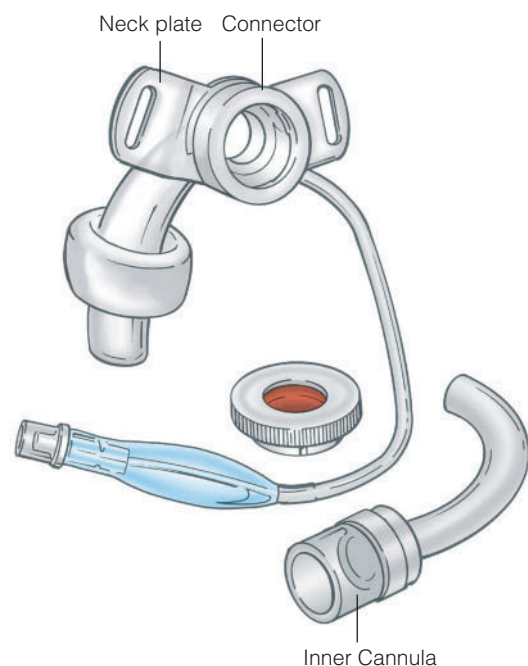


Figure 3-13 Tracheostomy tube.

Box 3-3 Bedside Equipment

- Extra tracheostomy tubes (one of the same size/type, and one size smaller)
- Obturator
- Suctioning system
- Yankauer and suction catheters
- Manual resuscitation bag and oxygen
- Tracheostomy dressings or drain sponges
- Tracheostomy cleaning kit
- Disposable inner cannulas when indicated
- New tracheostomy holder or ties
- Saline
- 10 ml syringe for cuffed tracheostomy tubes

SOURCE: Dawson, 2014; Morris et al., 2013.

equipment is at the bedside (Box 3-3). The nurse must remember that the patient will experience a change in body image, and families may struggle to accept this change in the patient's appearance as well. The nurse must assist the family and patient to remain positive.

Technological Requirements. Ventilators have a variety of settings with which the nurse must be familiar in order to provide safe, effective care. Table 3-4 summarizes common settings. The first is the **fraction of inspired oxygen (FiO₂)**. This is the percentage of O₂ in the mix of air delivered to patients. The FiO₂ of room air is 21%. FiO₂ is also expressed as a decimal: for example, room air is 0.21. Current ventilator strategies call for the lowest FiO₂ setting to achieve PaO₂

and SaO₂. High oxygen settings may cause tracheobronchitis, absorptive atelectasis, and diffuse alveolar damage if used for prolonged periods (Byrd & Roy, 2015). The nurse should remember that oxygen is a drug and should be titrated by physician order or according to standing orders.

The next setting is the **tidal volume (V_T)** which is the volume of gas delivered in one ventilatory cycle. V_T is normally 7 mL/kg of body weight or around 500 mL. In mechanical ventilation, the volume is kept at 5–8 mL/kg in order to prevent trauma to lung tissues. In ALI, lower volumes are preferred. The rate is the number of breaths the ventilator provides in 1 minute. The mode and patient's CO₂ level factors into determining the best rate. When CO₂ is elevated, the rate may be increased to allow the patient to "blow off" excess.

The **inspiratory:expiratory ratio (I:E Ratio)** compares the time spent in inspiration to that in expiration. Normally, a person spends twice as much time in exhalation as in inspiration, so the ratio would be 1:2. In disorders where the lungs are "stiff" or noncompliant, the I:E ratio may be reversed in order to allow a slower delivery of the V_T. This more gradual introduction permits the lungs to adjust to the distention while decreasing the risk of barotrauma. Called **inverse ratio ventilation**, this setting is very uncomfortable for the patient, and nurses should ensure the patients are adequately sedated.

The **flow rate** of the ventilator determines the speed at which the V_T is delivered. Normal rates are 60 to 100 liters/minute, generally estimating the peak flow at four times the minute volume. **Minute volume** is determined by totaling the V_Ts over 1 minute and is indicative of readiness to wean. When patients are experiencing ventilator asynchrony, adjusting the peak flow may increase comfort and improve ventilator tolerance.

Table 3-4 Settings on Mechanical Ventilators

Setting	Description	Standard Range
Oxygen Concentration (FiO ₂)	Amount of oxygen in gas delivered to patient	21–60%; lowest setting allowing for SaO ₂ over 90%, and PaO ₂ greater than 60
Tidal Volume (V _T)	Volume of gas delivered in one cycle	6–10 mL/kg of predicted body weight
Rate	Minimal number of breaths per minute	6–12 breaths per minute
Inspiratory: Expiratory Ratio	Ratio of time of inspiration to time of expiration; may be reversed in conditions where lungs are noncompliant	1:2
Flow Rate	Rate at which tidal volume is delivered to patient	60–100 liters/minute
Sensitivity	The negative inspiratory pressure or flow a patient must generate in order to trigger the ventilator to deliver a breath	–1 to –2 cm H ₂ O
High-Pressure Limit	Ventilator will not exceed this pressure in delivering volume; pop-off mechanism prevents excessive pressure	10–20 cm H ₂ O above peak inspiratory pressure
Pressure Support	Positive pressure used to decrease patient's work of breathing	5–10 cm H ₂ O
Positive End-Expiratory Pressure (PEEP)	Positive pressure left in lungs at end of expiration; prevents atelectasis and may enhance oxygenation at higher levels	3–10 cm H ₂ O

Source: Byrd & Roy, 2015; Bristle, Collins, Hewer, & Hollifield, 2014.

Sensitivity determines how much negative pressure the patient must generate before the ventilator will deliver a breath. This setting prevents the ventilator from hyperventilating a patient taking very shallow breaths and helps synchronize the patient's intrinsic respiratory rate with mechanical support. This synchrony increases the patient's tolerance of the ventilator.

The **high-pressure limit** sets a cap on the amount of pressure that can be delivered to the lungs in a breath. If this set amount of pressure is increased, whether by secretions, coughing, or lung compliance, a pop-off valve allows the excess pressure to escape to the atmosphere. This safety setting is very important for protecting the patient's lungs from barotrauma. High-pressure alarms should signal the nurse to check the patient to determine if suctioning is needed, whether the patient is biting the tube or has rolled onto it, and to determine if a patient is experiencing bronchospasm or mucous plug. **Peak inspiratory pressure (PIP)** is the highest pressure during inspiration, whereas **plateau pressure** stays relatively constant through inspiration.

PEEP, positive end expiratory pressure, is just what it sounds like: positive pressure left in the lungs at the end of expiration. This setting usually ranges from 3–10 cm H₂O, and the pressure helps keep the alveoli open, preventing atelectasis. Higher levels of PEEP may be used to recruit collapsed alveoli and improve oxygenation in patients with severe lung dysfunction but this comes with an increased risk of barotrauma. This higher pressure distends the alveolus, providing a greater surface area for gas exchange. Nurses must monitor patient blood pressure during increases in PEEP as the increased intrathoracic pressure may cause compression of the vena cava and decrease preload.

Whereas PEEP is at the end of expiration, **pressure support** occurs at the beginning. To understand PEEP, one might envision blowing up a party balloon and remember how hard it is to do so before the pressure inside the

balloon exceeds atmospheric pressure. But once enough air is pushed into the balloon and atmospheric pressure is overcome, the balloon becomes much easier to inflate. The lungs are very similar in that the majority of the work of breathing coincides with moving the ribs and overcoming atmospheric resistance to chest expansion. By providing positive pressure at the beginning of inspiration, the work of breathing decreases, allowing the patient to expand the chest more fully while expending less energy. Pressure support is commonly prescribed during the weaning process.

Ventilator Modes. When positive-pressure mechanical ventilation began, patients were given a set number of breaths at a set volume, no more and no less. This older, "controlled mandatory ventilation" mode is difficult to tolerate and is rarely used in critical care.

However, as technology improved, other modes of ventilation (Table 3-5) such as **assist control** were developed. In this mode, the patient regains some control of the rate of breathing. While still receiving a set V_T , the patient is able to take additional breaths over the set rate as desired. With each breath, the ventilator will deliver the set V_T . This is the most commonly used mode in current practice.

Another common mode is **synchronized intermittent mandatory ventilation (SIMV)**. In this mode, the patient receives a minimum number of breaths with a set V_T , but may take additional breaths as determined by the patient. In these additional breaths, the patient determines how much volume to inspire and is able to use their respiratory muscles. The mandatory breaths are spaced evenly throughout each minute, timed or synchronized with the patient's inspiratory effort. This feature explains the *intermittent* in the name of the mode. This prevents the machine from trying to deliver a breath at the same time a patient is trying to exhale and makes this mode easier to tolerate.

Continuous positive airway pressure (CPAP) is used frequently as patients become ready for extubation to wean,

Table 3-5 Common Modes of Mechanical Ventilation

Mode	Description	Target Population	Nursing Implications
Controlled Mandatory Ventilation	Tidal volume delivered at a set rate independent of patient effort; rarely used	Apneic patients or those with little or no respiratory drive	Patients may be heavily sedated or have induced paralysis
Assist-Control Ventilation (AC)	Tidal volume delivered at a set rate in response to patient effort; if patient fails to breathe at predetermined time, ventilator will deliver a breath	Patients able to breathe spontaneously with weak respiratory muscles	Patient is able to hyperventilate if volume controlled or may become hypercapneic if pressure controlled
Synchronized Intermittent Mandatory Ventilation (SIMV)	Tidal volume delivered at a low set rate in response to patient effort while allowing spontaneous breaths between	Patients who cannot sustain spontaneous ventilation for extended periods	Provides better synchrony and preserves some respiratory muscle function
Continuous Positive Airway Pressure	Applies positive pressure during spontaneous breaths	Effective as a weaning trial or training mode in patients capable of spontaneous ventilation	Patient must be monitored for respiratory distress

Source: Bristle, Collins, Hower, & Hollifield, 2014.

and to determine ability to wean. In this mode, positive pressure is continually applied to the airway. CPAP increases functional residual capacity by keeping alveoli open. In this mode, the patient controls both rate and volume; in other words, the patient is essentially breathing on his or her own. CPAP is often used in conjunction with pressure support ventilation (PSV) to offer bilevel ventilation pressures. When used with CPAP, PSV serves to help the patient overcome the small diameter and length of the endotracheal tube. These two settings cannot be used unless the patient has a ventilatory effort, and caution should be used with sedation in this mode. Most ventilators, however, do have a backup *apnea* mode in case the patient fails to take an adequate number of breaths on his or her own.

Pressure control ventilation is less frequently used and provides a set pressure instead of tidal volume during the respiratory cycle. On inspiration, the ventilator delivers gas until a pressure limit is reached. An adequate tidal volume is not ensured and must be monitored frequently. This mode is used in spontaneously breathing patients to reduce the risk of barotrauma by reducing airway pressures but it may prolong the duration of mechanical ventilation (Bristle, Collins, Hewer, & Hollifield, 2014).

Other Ventilator Modes. Other ventilator modes include high frequency oscillation (HFO), airway pressure release ventilation (APRV), proportional assist ventilation (PAV), and neurally adjusted ventilatory assist (NAVA). HFO requires the use of a special ventilator that delivers very small tidal volumes at rapid rates. The rapid frequency or oscillation of gas delivery holds the mean airway pressure constant and allows gas exchange to occur throughout the entire respiratory cycle (Scott & Gentile, 2012). APRV applies two pressure settings, a high pressure and a low pressure, while the patient breathes spontaneously. The ventilator alternates between a high pressure and a low pressure, during which exhalation occurs and is not timed with the patient's ventilation cycle but allows spontaneous breathing to occur at any time (Cordioli, Akoumianaki, & Brochard, 2013). PAV alternates the pressure support according to the patient's inspiratory muscle pressure theoretically improving comfort and sleep (Cordioli et al., 2013). NAVA uses measurement of diaphragm electrical activity to determine appropriate positive pressure (Cordioli et al., 2013). The effectiveness and appropriate use of these settings are currently being investigated.

The physician prescribes ventilator settings, but in some facilities, respiratory therapists or nurses adjust settings following a physician-approved protocol. Regardless, the healthcare professional making setting changes should clearly document the alteration in a single, easily accessible location to enhance communication between nurses, respiratory therapists, and physician.

Weaning from Mechanical Ventilation. **Weaning** is the process in which support from the mechanical ventilator is reduced and the work of breathing is assumed by the patient. Removal of the nasotracheal or endotracheal tube is referred to as **extubation**. Determining readiness to wean is a complex process requiring the consideration of many factors for readiness, including the indication for mechanical ventilation. Prolonged ventilator duration is associated with increased morbidity, mortality, and longer ICU and hospital stays, however premature extubation also increases these risks (Jeganathan, Kaplan, & Balk, 2015; Shaikh, Morales, & Laghi, 2014). Post-extubation distress affects 10% to 20% of patients, a third within 12 hours post extubation and 42% after 24 hours (Shaikh et al., 2014). Protocols and daily weaning screens have been instituted to facilitate the weaning process but have not been shown to improve weaning outcomes (Shaikh et al., 2014).

Several clinical factors are known to have a predictive value in the success of extubation. Patients with impaired neurological status, increased secretions, weak cough, hemoglobin < 10g/L, rapid shallow breathing index >105, advanced age, fluid overload, high APACHE II scores, and chronic respiratory or cardiac conditions are at high risk for extubation failure (Jeganathan et al., 2015). Consistent daily screening for readiness followed by spontaneous breathing trials can reduce the duration of mechanical ventilation (Shaikh et al., 2014). Several indicators to determine readiness for weaning have been studied, including the **rapid shallow breathing index (RSBI)**, **maximum inspiratory pressure**, B-type natriuretic peptides (BNP), and diaphragm ultrasonography (Shaikh et al., 2014). The most frequently used measures are the RSBI and maximum inspiratory pressure.

RSBI is determined by dividing the respiratory rate (f) by tidal volume (V_T) in liters. The resulting value is represented in breaths per minute per liter. For example a patient with a respiratory rate of 16 and a tidal volume of 0.5 L would have an RSBI score of 32. Scores equal to or greater than 105 are associated with a higher extubation failure risk. Typically, the RSBI is obtained either on CPAP of 5 mm H₂O or a T-piece. With a T-piece, the patient is removed from the ventilator and an oxygen source is connected to the endotracheal tube. RSBI scores are as much as 20 to 50% lower with ventilator support than a T-piece and should be interpreted cautiously (Shaikh et al., 2014). Maximum inspiratory pressures less than -30cm H₂O have been associated with success and pressures higher than -20cm H₂O have been associated with failure, but sensitivity and specificity of this measure has not been reliable (Shaikh et al., 2014). Large increases in BNP may indicate a patient is unlikely to tolerate extubation (Jeganathan et al., 2015). Diaphragmatic dysfunction was associated with longer weaning and ventilator duration, but requires further research to determine its usefulness in identifying patients at high risk for extubation failure (Shaikh et al., 2014).

Box 3-4 Indicators of Readiness for Weaning from Mechanical Ventilation

1. RSBI ≤ 105
2. Respiratory rate ≤ 35
3. Maximal inspiratory pressure ≤ -20
4. V_T greater than 5 mL/kg
5. Vital capacity greater than 10 mL/kg
6. $SpO_2 > 90$ with FiO_2 of 40% or less
7. PaO_2/FiO_2 ratio of 150 or higher

Following extubation, the patient is placed on cool humidified oxygen via a mask (cool aerosol) or nasal cannula. The cool aerosol may decrease airway swelling and promote expectoration of secretions. The nurse must carefully monitor vital signs and assess for signs of respiratory distress including stridor that might indicate laryngeal edema.

Traditionally, weaning consisted of slowly lowering ventilator settings over time to allow the patient to gradually accept the work of breathing. A patient might progress from AC mode to SIMV with progressively lower mandatory rates to CPAP and finally to a T-piece and extubation. SIMV is being used less for weaning as it may prolong time to extubation (Shaikh et al., 2014). Instead, a daily screen for readiness followed by a spontaneous breathing trial is attempted. Indicators of readiness are described in Box 3-4. If the patient is suitable for weaning, they are either placed on CPAP at 5 cm H_2O or a T-piece for 2 hours. An RSBI or maximal inspiratory pressure is obtained and the clinician decides if extubation is appropriate based on the patient's condition and disease process.

Essential for Evidence-Based Practice

Nurses are important members of the multidisciplinary team and are often responsible for the management of sedation in mechanically ventilated patients. Nurses often have the power to employ sedation strategies that improve patient outcomes. Several important studies guide collaborative practice regarding sedation of mechanically ventilated patients and assessment of readiness to wean.

1. Daily "sedation vacations" may reduce the duration of mechanical ventilation, length of ICU stay, and length of hospitalization, while increasing the number of ventilator-free days. (Kress, Pohlman, O'Connor, Hall, 2000)
2. Timing sedation vacations with daily spontaneous breathing trials has demonstrated a significant reduction in mortality (Girard et al., 2008)

3. Daily "sedation vacations" in addition to nurse directed sedation protocols that target light sedation levels were not associated with shorter duration of mechanical ventilation. The key to reducing the duration of mechanical ventilation may rely on consistently using protocols that reduce excessive sedation (Mehta et al., 2012).

These key studies guide current practice of maintaining light sedation when possible and performing daily sedation lightening or wake-ups with concurrent assessments of readiness for extubation.

Complications of Mechanical Ventilation

Mechanical ventilation can cause several complications. The most significant is **ventilator-associated pneumonia (VAP)**. Nursing plays an important role in the prevention of VAP. The reader is referred to the discussion of VAP in Chapter 17 . However, nurses may employ specific interventions to assist in prevention of VAP as noted in Box 3-5.

Ventilator-induced lung injury (VILI) can result from excessive pressure (barotrauma) or excessive volume (volutrauma). **Barotrauma** is the perforation or rupture of an alveolus that allows air to enter the pleural space or the mediastinum (Byrd & Roy, 2015). **Volutrauma** is overdistention of the alveoli that results in inflammation and can reduce the patient's ability to recover respiratory function (Byrd & Roy, 2015). High tidal volumes, elevated pressures, and patient-ventilator dyssynchrony can lead to increased risk for these types of injury. This lack of

Box 3-5 Strategies to Prevent VAP

The key components of the IHI ventilator bundle are

- Elevation of the head of the bed at 30 degrees or higher
- Daily "sedation vacations" and assessment of readiness to extubate
- Peptic ulcer disease prophylaxis
- Daily oral care with chlorhexidine
- Deep venous thrombosis prophylaxis

Additional interventions recommended to prevent VAP include

- Washing hands before and after contact with each patient
- Use of continuous subglottic suctioning
- Change of the ventilator circuit no more often than every 48 hours or no longer routinely changing the patient's ventilator circuit

coordination between the perceived need and the function of the ventilator has many causes including disease process, mode, settings, and anxiety. Working with the respiratory therapist, the nurse may be able to adjust the ventilator settings and alleviate problems such as excessive V_T or insufficient expiratory time.

Another complication of mechanical ventilation is cardiovascular compromise. The presence of positive pressure in the thorax, particularly in the presence of higher levels of PEEP, may decrease venous return, thus decreasing preload and cardiac output. This may result in hypotension or decreased perfusion to tissues particularly in hypovolemic or preload-dependent patients (Kato et al., 2014).

Nursing Care for the Mechanically Ventilated Patient

Caring for the mechanically ventilated patient requires the nurse to be vigilant and aware of potential complications. Nurses must also carry out independent nursing interventions designed to minimize time on the ventilator as well as preventing complications. The mechanically ventilated patient requires an interdisciplinary team to maximize outcomes, and the nurse must have full participation on the team. The nurse should ensure that a manual resuscitation bag and alternate O_2 supply is available at the bedside at all times. The bedside stock should contain an adequate supply of suction and oral care supplies. The nurse must respond quickly to ventilator alarms. Common alarms and ways the nurse might troubleshoot the alarms are listed in Table 3-6.

Routine nursing assessments should include the level of the tube at the patient’s lip line, size of the tube, oxygen

saturation levels, level of consciousness (or sedation), breath sounds, tolerance of the ventilator, and vital signs. When suctioning or moving the patient, the nurse must monitor O_2 saturations and modify activities to ensure adequate oxygenation at all times. The nurse should analyze ABGs (if ordered) and evaluate the adequacy of gas exchange in order to ensure the patient is receiving optimal ventilation and oxygenation. The nurse should verify that the prescribed settings are delivered via the ventilator and that tubing is free of kinks, and work with respiratory therapists to maintain appropriate temperature of the delivered gas to prevent mucus plugs. The nurse should ensure the ventilator is connected to an emergency electrical outlet so that, if the power fails, the ventilator will receive power from an alternate power source. Unnecessary disconnection of the ventilator circuit should be avoided. Sputum should be monitored for color, consistency, odor, and amount, and documentation must reflect these characteristics. The nurse should monitor the mouth or nose for signs of ulceration from tube pressure. Where possible, nurses should also evaluate the daily chest x-ray for tube position and the appearance of potential complications. Common nursing diagnoses are listed in the “Nursing Diagnoses” feature presented earlier in this chapter.

Promoting Safety: Airway Maintenance

To facilitate airway patency, the nurse must suction the patient’s endotracheal tube to clear secretions as needed. Suctioning reduces the risk of consolidation and atelectasis, but also carries risks including hypoxemia, dysrhythmias, and tracheal tissue trauma. Suctioning should be performed only when necessary to reduce these complications. Indications for suctioning include coarse crackles over the trachea, elevated PIP, decreased tidal volume, decrease in oxygen saturation, visible secretions, coughing or inability to generate a cough, acute respiratory distress, suspected aspiration and the need to obtain a sputum specimen (AARC, 2010). The need for suctioning should be assessed every 2–4 hours using the indications listed above (Sole, Bennett, & Ashworth, 2015). Two methods of suctioning include open suctioning with single use systems and closed suctioning with an in-line catheter. Prior to suctioning, the nurse applies 100% FiO_2 . Hyperoxygenation prior to and after suctioning for 30–60 seconds reduces the risk of hypoxemia (AARC, 2010). Instillation of saline during suctioning is controversial, and is not recommended as routine practice (AARC, 2010). Due to the risk of trauma to tissues and atelectasis, the lowest possible level of suction should be used, typically 80–100 mm Hg (AARC, 2010). For deep suctioning, the catheter is advanced until resistance is met then withdrawn 1–2 cm before application of continuous suction (AARC, 2010). Application of suction should not last longer than 15 seconds (AARC, 2010).

Table 3-6 Troubleshooting Ventilator Alarms

Apnea	Check to be sure that the patient is connected to the ventilator. Ensure patient is ventilating adequately and is not oversedated. Patient may require a higher level of ventilatory support.
High Pressure	Ensure tubing is not kinked or compressed. Assess need for suctioning and check for presence of ventilator dyssynchrony and coughing. Report alarm to responsible clinician if not quickly remedied.
Low Minute Volume	Check to be sure that the patient is connected to the ventilator. Ensure patient is ventilating adequately and is not oversedated. Patient may require a higher level of ventilatory support.
High Minute Volume	Assess patient for anxiety. Check vital signs and assess patient for signs of hypoxia (tachycardia, hypertension, restlessness, cyanosis, accessory muscle use, etc.). Patient may require a higher level of ventilator support, anxiolytics, sedation, or increased FiO_2 .

Safety Initiatives

Preventing Ventilator-Related Deaths and Injuries

PURPOSE: Ensuring that ventilator alarms are appropriately set and that clinically significant alarms are responded to in a timely and appropriate manner

RATIONALE: A decade ago, The Joint Commission (2002) issued a sentinel event alert titled “preventing ventilator-related deaths and injuries.” The alert included a review of 23 reported ventilator-related deaths or injuries. At that time, the commission determined that in the majority of the cases, there was malfunction, misuse, or inadequate setting of at least one ventilator alarm. Alarm safety continues to be a key goal of The Joint Commission and in 2013, the following plan was announced for which hospitals would be held accountable (The Joint Commission, 2013).

PHASES OF THE 2013 NATIONAL SAFETY GOAL:

1. As of January 2014, hospitals are required to make alarm safety a priority and to identify the alarms that are most important to manage within their own institutions based on patient risk and frequency of alarms.
2. Beginning in January 2016, hospitals will be required to have policies and procedures in place for alarm management that address:
 - a. Clinically appropriate alarm parameters
 - b. When alarm parameters can be changed and/or disabled
 - c. Who has the authority to set or change alarm parameters and disable alarms
 - d. Monitoring and responding to clinical alarms
 - e. Checking individual alarm signals for proper functioning
3. In January 2016 hospitals will need to ensure that staff and independent practitioners are educated about the purpose and operation of alarms and their responsibilities in respect to those alarms.

ALARM RECOMMENDATIONS:

1. Set realistic, actionable alarm parameter defaults based on the unit's patient population following unit specific review.
2. Set alarms for the specific patient based on the patient's lung status and likely values. For example, the alarms on pulse oxygenation monitors should be set appropriately for a patient with COPD. “One size fits all” is not appropriate.
3. Standardize alarm volumes and sounds based on priority. Many sources recommend standardization of the alarm sounds between makers of products. High-priority alarms would include apnea, high airway pressure, and electrical malfunction. Mid-level alarms would include PEEP alarms. Lower priority alarms would be low or high concentrations of oxygen during inspiration, or low or high temperature.
4. Ensure that clinically significant alarms can be heard either by increasing the alarm volume or distributing the speakers.

Sources: Korniewicz & Kenney, 2011; The Joint Commission, 2002; The Joint Commission, 2013.

Bariatric Considerations

Obesity has major negative effects on the respiratory system. Lung volume is decreased along with FRC, compliance, and respiratory muscle function. Moderate to severe obesity causes micro-atelectasis and gas trapping within the lungs (Mandal & Hart, 2012, O'Donnell, Ciavaglia, & Neder, 2014).

Obesity decreases the quality of the chest x-ray and may obscure the distinction between pulmonary infiltrates and pulmonary edema.

To assess the breath sounds of an obese patient, it may be necessary to displace all of the skin folds. Breath sounds are most likely to be heard clearly in an obese patient in dependent areas closest to the chest wall.

Intubation of the obese patient may be problematic due to the excess cervical fat that makes positioning the head difficult. Moreover, obese patients who have a tracheostomy have twice the number of complications of

non-obese patients. The most serious and common of these complications is loss of airway. The next most common complication is deterioration of the skin of the neck and resultant infections in neck creases.

When obese patients are ventilated, most authors suggest calculations of tidal volume should be based on ideal body weight rather than actual weight in order to minimize barotrauma. However, some authors recommend higher inspiratory pressures and PEEP recruitment strategies for obese patients because they believe their alveoli are more likely to collapse.

Obese patients who are ventilated have significantly longer courses of mechanical ventilation and more frequent complications associated with ventilation. However, they are not more likely to die following mechanical ventilation than non-obese patients.

SOURCE: Mandal & Hart, 2012; O'Donnell, Ciavaglia, & Neder, 2014.

Reflect On

Your patient with a COPD exacerbation is placed on a spontaneous breathing trial consisting of CPAP mode. Fifteen minutes after the wean from the ventilator begins, the high minute ventilation alarm sounds. What assessments will you make?

Promoting Safety: Use of Restraints

Many facilities use protocols to provide care for mechanically ventilated patients, and these protocols frequently include the use of restraints to prevent accidental extubation. Nurses believe they must restrain a patient to “protect the airway,” but evidence suggests that the opposite is true (Kiekkas, Aretha, Panteli, Baltopoulos, & Filos, 2012).

Enhancing Communication

One of the most frustrating aspects of caring for a mechanically ventilated patient is the inability to ascertain what the patient is trying to say. Lip reading can be challenging for the most experienced nurse, and the presence of an oral endotracheal tube renders lip reading even more difficult. Patients experience discomfort and frustration when they cannot communicate their needs to staff (Bergbom-Engberg & Haljamae, 1989). Establishing a means of communication is essential to providing good nursing care. Common strategies are blinking eyes (one blink for yes, two for no) or hand squeezes. Once a strategy has been established, it should be communicated among all caregivers to eliminate confusion for the patient. Picture and letter boards can be helpful in decreasing frustration and conserving patient energy. For those patients who are able, providing a pen and clipboard with paper expedites communication.

Reflect On

You get report on Mr. Smith, a 48-year-old intubated patient with amyotrophic lateral sclerosis (ALS), a disease in which muscle atrophy progressively prevents the patient from being able to move or maintain adequate ventilation. Mr. Smith can move only his facial muscles and his left thumb. The nurse giving you report tells you that Mr. Smith has been withdrawn and tearful. What communication methods might be most appropriate for him?

Providing Patient and Family Education

Patients and families need to understand why mechanical ventilation is necessary as well as the potential risks of consenting to or refusing the therapy. Explaining why suctioning causes the patient to gag may not make it less uncomfortable, but understanding that it is “normal” may

decrease the anxiety. Establishing an emergency stop signal may also be valuable in helping the patient feel some sense of control. Explaining the alarm systems for the ventilator, ECG monitoring, and infusion pumps may decrease anxiety related to the noise for patients and families. Families need to understand that intubated patients are able to hear and why they cannot speak. Many families are afraid to talk to patients, and encouraging them to touch and speak to the patients is very important. If the patient is going home with a tracheostomy, nurses can begin to teach family members tracheostomy care while in the critical care area. Visitation regulations may need to be adapted to allow the family access to the patient and promote psychological health of both the patient and family.

Providing Nutrition

The role of adequate nutrition in critical care cannot be understated. The gastrointestinal (GI) tract, if functioning, is the preferred route for providing nutrition to the mechanically ventilated patient. Early enteral nutrition reduces the risk of nutritional shortages that could worsen mortality in critically ill patients (Blot, Alfonso, & Labeau, 2015). Nurses play a role as advocates on the multidisciplinary team, and a nutritional consult is an important start to ensuring that the patient receives adequate nutritional support. Nurses must routinely assess GI system function.

Aspiration is a major problem in tube-fed patients requiring mechanical ventilation. Feeding may be administered through nasogastric, nasoduodenal, or nasojejunal routes. Post-pyloric (duodenal or jejunal) feeding may be indicated in patients with pancreatitis, who are at high risk of aspiration (Compton, Bojarski, Siegmund, & van der Giet, 2014). Insertion of enteral feeding tubes can result in complications including respiratory placement and perforation of trauma of tissues. Often, insertion of these enteral tubes is done blindly by nurses, and placement is confirmed prior to use. Radiography is the gold standard for confirming placement (Blot et al., 2015). Many facilities use carbon dioxide detectors and an air bolus to detect proper placement during enteral tube insertion. Carbon dioxide detectors may be helpful in detecting tracheal placement, and an air bolus may detect gastric placement, but neither method can replace radiography (Blot et al., 2015). These tubes may be used to administer medications, and the nurse must be very aware of what medications can be safely crushed. Tablets must be thoroughly crushed, and the tube must be well flushed after administration. Nurses should consult with physicians or pharmacists to supply liquid forms of medication when available.

Numerous nutritional formulas and supplements have been designed to address compromised organ systems. Protein supplements and probiotics may be ordered. Feedings may be administered as bolus feedings or

continuously. The nurse must monitor the patient's tolerance of the feedings by monitoring for diarrhea, gastric residual as appropriate, and hyperosmolar dehydration. The head of the bed should be elevated to at least 30 degrees, and feedings should be discontinued during turning, suctioning, and extubation. If enteral feedings cannot be tolerated, parenteral feedings must be considered.

Acute Lung Injury and Acute Respiratory Distress Syndrome

ARDS is a serious and deadly type of respiratory failure. This heterogenous syndrome can be caused directly, such as in aspiration and pneumonia, or indirectly, as occurs in sepsis, systemic inflammatory response syndrome, or pancreatitis (Box 3-6). ARDS is an inflammatory disorder that damages the alveolar capillary membrane throughout the lung, interfering with gas exchange. Sepsis is the most common cause of ARDS (Harman, 2014). Prior to 2011, ARDS was considered a severe form of acute lung injury (ALI). The more recent Berlin definition of ARDS encompasses ALI as mild form of ARDS (Thompson & Moss, 2013). The criteria for ARDS require acute onset

hypoxemia, despite administration of PEEP, with diffuse bilateral opacities on chest radiography, which cannot be fully explained by heart failure (Thompson & Moss, 2013). The degree of hypoxemia determines whether the ARDS is mild, moderate, or severe. A P/F >200 and ≤ 300 despite PEEP or CPAP of at least 5cm H₂O is mild. Moderate ARDS has a P/F ratio >100 and ≤ 200 with a PEEP greater than 5cm H₂O and severe ARDS has a P/F ratio ≤ 100 with a PEEP of at least 5cm H₂O (Thompson & Moss, 2013).

Pathophysiology

ALI begins with injury to the lungs that stimulates an inflammatory response. The progression to ARDS can be divided into three overlapping stages: exudative, fibroproliferative, and recovery. During these stages, a complex series of extreme immune and inflammatory responses damages the alveolar capillary membrane (ACM). This injury results in a cascade of cellular mediators and an amassing of neutrophils, macrophages, and platelets in the damaged ACM. Humoral mediators follow the cellular mediators, resulting in further damage to the ACM. (The way that this process results in damage to the endothelial and alveolar membranes is further described in Chapter 18  and displayed in Figure 18-1 .)

Capillary permeability is an important step in ARDS pathophysiology. ( Figure 3-14).

During the exudative phase (usually the first 2–4 days) the capillary membrane begins to leak, and protein-rich fluid fills the alveoli, reducing gas exchange. This fluid appears on radiography as bilateral infiltrates, but because ALI does not affect left ventricular function directly, pulmonary occlusive pressures stay below 18 mm Hg. As capillary permeability continues to worsen, neutrophils begin to attach to the damaged membrane and may cross into the alveoli, further impairing gas exchange. A key to this stage is the formation of “hyaline membranes” in the alveoli and conduction airways from the consolidation of proteins.

The fibroproliferative stage is characterized by the production of type II pneumocytes, macrophage-mediated destruction of hyaline membranes, and resolution of neutrophilic-mediated inflammation. Type II alveolar cells sustain damage, limiting the production of **surfactant**, resulting in further loss of alveolar function. This decrease in available alveolar surface area results in a ventilation perfusion (V/Q) mismatch and hypoxemia. Patients struggle for air due to an increase in the work of breathing, which results from decreases in FRC and lung compliance. Hypoxemia becomes increasingly severe as the increased work of breathing contributes to alveolar hypoventilation. The patient also develops pulmonary hypertension due to the structural damage suffered by the alveoli. The resultant

Box 3-6 Antecedents of ALI/ARDS

Direct injury

- Chest trauma
- Pneumonia
- Aspiration of pneumonitis
- Pulmonary contusion
- Near drowning
- Inhalation injury
- Pulmonary embolus
- Radiation
- Eclampsia of pregnancy

Indirect injury

- Sepsis
- Burns
- Severe trauma with multiple blood transfusions
- Drug overdose
- Cardiopulmonary bypass
- Acute pancreatitis
- Intracranial hypertension
- Drug overdose

Acute respiratory distress syndrome (ARDS) is a severe form of acute respiratory failure that occurs in response to pulmonary or systemic insults. ARDS is characterized by noncardiogenic

pulmonary edema caused by inflammatory damage to alveolar and capillary walls. Many disorders may precipitate ARDS, although sepsis is the most common.

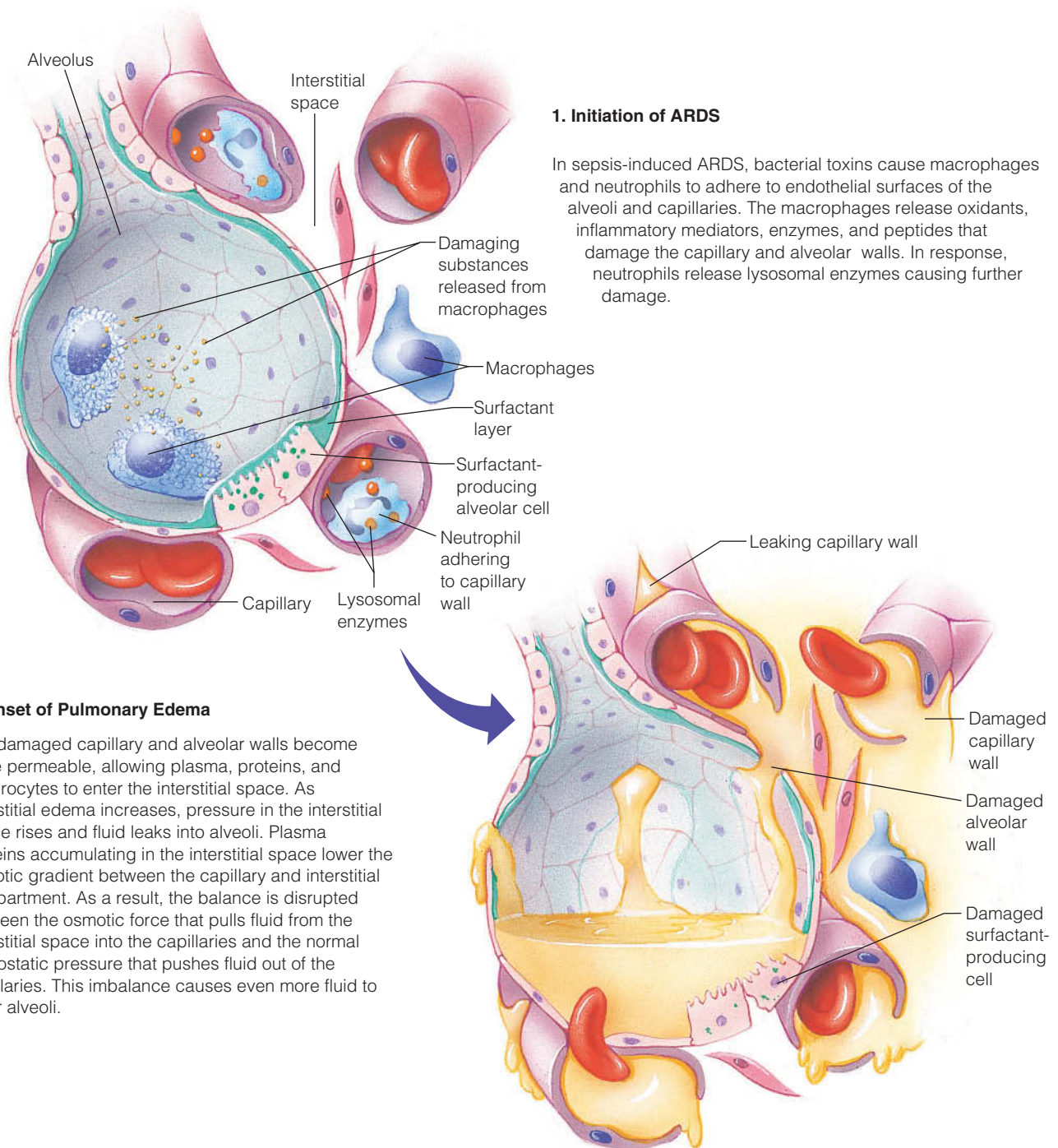
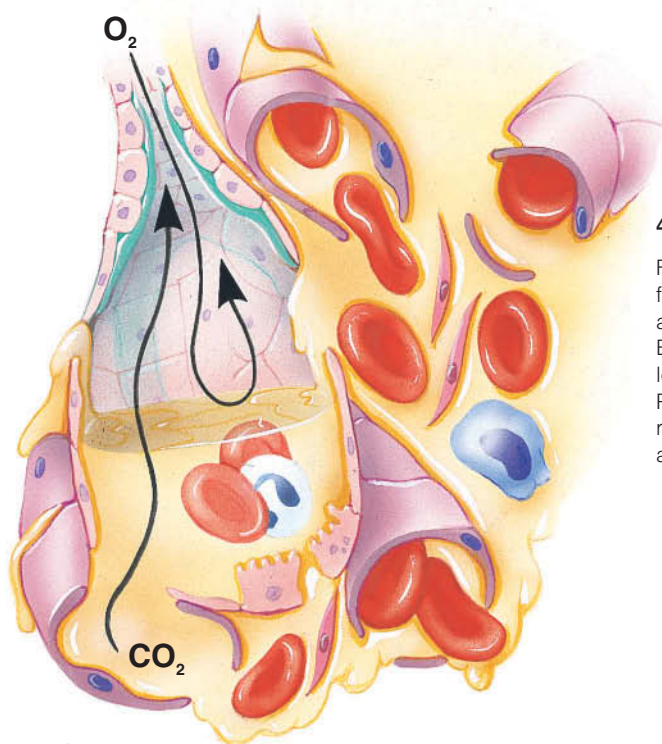
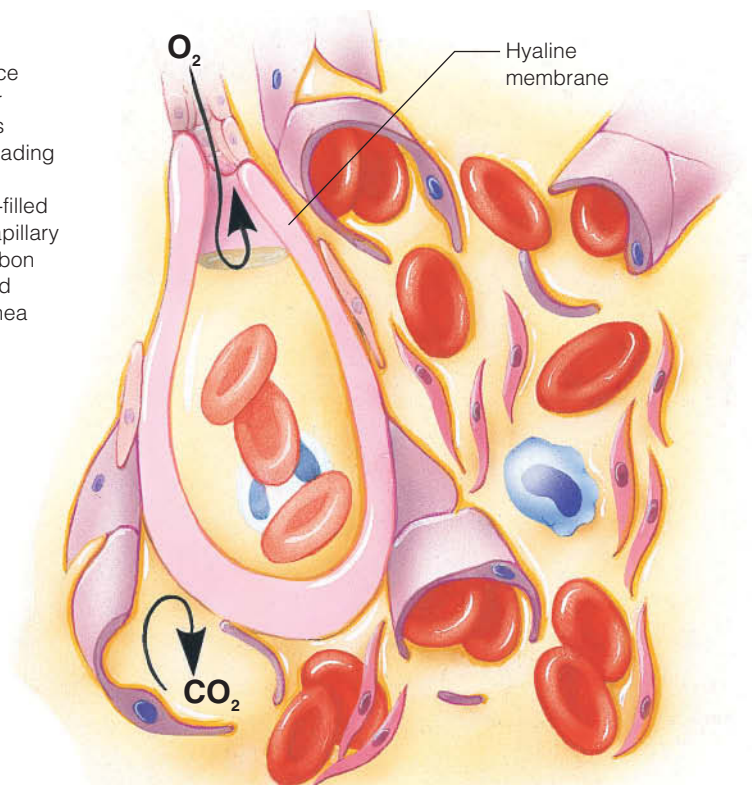


Figure 3-14 Acute Respiratory Distress Syndrome.

3. Alveolar Collapse

Protein-rich fluid accumulates in the alveoli, inactivating surfactant and damaging type II alveolar cells that produce surfactant. (Surfactant is important in maintaining alveolar compliance—the ability of tissue to stretch or distend.) As active surfactant is lost, the alveoli stiffen and collapse, leading to atelectasis, which increases breathing effort.

Decreased alveolar compliance, atelectasis, and fluid-filled alveoli interfere with gas exchange across the alveolar-capillary membrane. Blood oxygen (PaO_2) levels fall. Because carbon dioxide diffuses more readily than oxygen, however, blood carbon dioxide (PaCO_2) levels also fall initially as tachypnea causes more CO_2 to be expired.



4. End-Stage ARDS

Fibrin and cell debris from necrotic cells combine to form hyaline membranes, which line the interior of the alveoli and further reduce alveolar compliance and gas exchange. Because CO_2 cannot diffuse across hyaline membranes, PaCO_2 levels now begin to rise while PaO_2 levels continue to fall. Rising PaCO_2 levels can lead to respiratory acidosis. Without respiratory support, respiratory failure will develop. Even with aggressive treatment, almost 50% of patients with ARDS die.

Figure 3-14 (continued).

Table 3-7 Stages of ARDS, Associated Pathophysiology, and Assessment Findings

Phase	Pathophysiology	Assessment Findings
Exudative Phase	Capillary membranes leak Protein-rich fluid fills alveoli Gas exchange is disrupted Type I alveolar cells are destroyed Hyaline membranes are formed	Chest x-ray may be normal or show dependent infiltrates Tachypnea and dyspnea Use of accessory muscles but lung sounds may be clear PAWP may be less than 18 mmHg Change in level of consciousness
Fibroproliferative Phase	Type II alveolar cells are damaged Surfactant production declines Peak inspiratory pressure increases Compliance declines Decreased FRC Further loss of alveolar function Ventilation/perfusion mismatch	Chest x-ray shows diffuse bilateral infiltrates and elevated diaphragm Refractory hypoxemia with hypercarbia despite hyperventilation Dramatically increased WOB Crackles on auscultation PA and PAWP pressures increase RA pressure increases Right-sided heart failure develops Agitation
Recovery/Fibrotic Phase	Development of fibrotic tissue in the ACM resulting in alveolar disfigurement Decreased lung compliance Worsening pulmonary hypertension Increased dead space ventilation	Leukocytosis and fever Worsening infiltrates on chest x-ray Worsening hypoxemia and hypercarbia Decreased tissue perfusion Increasing heart rate with decreasing blood pressure Lactic acidosis End-organ dysfunction

increase in right ventricular afterload contributes to right-sided failure. This phase of ARDS may last as long as 1 month.

The recovery phase is often called the fibrotic phase because the altered healing process may result in development of fibrotic tissue in the alveolar capillary membrane or destruction of alveoli. It is a period of reorganization of lung tissue. The more type II alveolar cells salvaged the more functional the repaired alveolar tissue will be. The disease course varies according to individual factors and comorbidities, and the level of recovery also varies accordingly. Direct causes tend to have more severe lung epithelial damage and indirect causes tend to have more endothelial injury and inflammation (Caffee et al., 2015). In the future, biomarkers that differentiate the types of ARDS may help researchers develop more targeted forms of treatment (Caffee et al., 2015; Chen & Ware, 2015). The phases of ARDS are correlated with the physical symptoms in Table 3-7.

Patient History and Assessment

Due to the severity of the patient's illness, the collection of history depends on review of extant records and accounts of significant others. However, an accurate history is crucial in the identification and removal of potential causative factors.

Initial clinical assessment may reveal a patient with tachypnea, tachycardia, and increases in heart rate and respiration that seem incongruent with the remainder of the exam. Such findings should trigger the nurse to perform a detailed assessment for ARDS and notify the physician in

order to begin treatment promptly. Additional early symptoms of ARDS include:

- Dyspnea and tachypnea
- Rapid, shallow respiratory pattern
- Use of accessory muscles
- Mottling or cyanosis of skin
- Abnormal breath sounds
- Dry cough
- Change in level of consciousness
- Confusion
- Restlessness
- Retrosternal discomfort
- Tachycardia
- Fever

Diagnostic confirmation will require ABGs and chest radiography but may include a computerized tomography (CT) exam. ABGs will initially demonstrate hypoxia despite oxygen delivered (low PaO_2 and SaO_2). The pH may be alkalotic or acidotic depending on the cause of lung injury and hyperventilation present. The PF ratio is important in diagnosis.

Early radiographic results may be normal or reveal some dependent infiltrates, but serial analysis reveals increasingly diffuse bilateral infiltrates. Inclusion of CT examinations in diagnostic testing for ARDS has been met with increasing favor. In addition to allowing earlier diagnosis, the scans provide the healthcare team with more detailed information regarding illness severity and response to treatment. The advantages of CT examination

must be carefully weighed against the risk to the patient of being transported for the study.

Reflect On

A patient diagnosed with ARDS is receiving paralytics and his high-pressure alarm sounds. How might you approach troubleshooting the alarm? What are the implications of not solving this problem as quickly as possible?

COLLABORATIVE CARE

Despite numerous studies, no effective therapy for treating ARDS at the pathophysiologic level is available. Key strategies for management of ARDS are identifying and treating the underlying disease and avoiding additional tissue damage.

Mechanical Ventilation in Acute Lung Injury / Acute Respiratory Distress Syndrome

ARDS causes severe hypoxia; it is important to maintain adequate oxygenation by providing supplemental oxygen and recruiting collapsed alveoli to participate in gas exchange. Mechanical ventilation is frequently required. A trial of NIV may be reasonable, but failure rates range from 36% to 61%, and the nurse should be prepared for emergent intubation (Frat et al., 2015). Researchers continue to develop new understandings and manipulate the various components of mechanical ventilation to improve outcomes in ARDS. Mechanical ventilation with positive pressure is the key treatment for ARDS, but can also further injure lung tissue and worsen the condition if not used properly.

Ventilator management in ARDS focuses on low V_T s, PEEP, and careful monitoring of plateau pressures. In ARDS, normal V_T can cause overdistention of functioning alveoli and shearing forces on opening areas of atelectasis. Plateau pressures should be monitored at a minimum of every four hours and with each titration of V_T and PEEP. PEEP can be titrated higher to improve oxygenation and reduce FiO_2 but should be used judiciously to prevent overdistention, shearing, and shunting of blood away from the open alveoli. The ideal level of PEEP in ARDS is not known (Fares & Carson, 2013). High levels of PEEP have not shown an improvement in outcomes (Chang et al., 2013). All efforts should be made to reduce FiO_2 to below 60% to reduce the risk of toxicity (Table 3-8).

Permissive Hypercapnea. The ARDS network study (2000) examined the use of lower V_T s to prevent VILI in patients with ARDS and found that V_T s of 4 to 6 mL/kg of body weight reduced mortality as well as decreased days on the ventilator. The lower V_T s result in alveolar hypoventilation, resulting in a rise in serum CO_2 levels.

Table 3-8 Ventilator Management Goals in ARDS

Goal	Purpose
Tidal volume between 4 and 8 mL/kg	Prevent ventilator-induced lung injury
Plateau pressure ≤ 30 cm H ₂ O	Prevent ventilator-induced lung injury
Keep $FiO_2 \leq 60\%$	Reduce risk of oxygen toxicity
Judicious use of PEEP at 5–15 cm H ₂ O	Recruit alveoli and improve oxygenation
Permissive hypercapnea ($PaCO_2$ 60–100)	Prevent ventilator-induced lung injury
Maintain SpO_2 between 88% and 95%	Support tissue oxygenation

Therefore, hypercapnea is an expected outcome of using lower V_T s or pressures, and permissive hypercapnea is a deliberate exchange to protect lungs from VILI as opposed to maintaining normal $PaCO_2$ levels. Aside from the physiologic problems with hypercapnea, it is permissible unless otherwise contraindicated, such as in elevated intracranial pressure or renal failure (Marhong & Fan, 2014).

Extracorporeal Life Support

Extracorporeal life support (ECLS) has recently received more attention as a rescue therapy for ARDS, given the 2009 epidemic of H1N1. It is used in cases of ARDS with severe hypoxemia, uncompensated hypercapnea, pH <7.15 or end expiratory plateau pressures >35 – 45 cm H₂O who have failed a prone positioning trial (Leligdowicz & Fan, 2015). The technology is essentially a lung bypass machine—blood is oxygenated and carbon dioxide is removed outside the body. Use of ECLS comes with many risks, and while some success was found with ARDS related to H1N1, there is a lack of strong evidence supporting its use (Leligdowicz & Fan, 2015).

Pharmacological Support

There is currently no generally accepted pharmacological treatment for ARDS, but there are a few interventions that have shown some benefit in subgroups of patients with ARDS (Spieth & Zhang, 2014). These include neuromuscular blocking agents, statins, corticosteroids, vasodilators, surfactant therapy, β_2 agonists, angiotensin-converting enzyme inhibitors, fluid therapy, nutrition therapy, stem-cells, and gene therapy.

Neuromuscular Blocking Agents

In severe ARDS, neuromuscular blocking agents such as cisatracurium may improve oxygenation and reduce mortality when used within the first 48 hours (Hraiech, Forel, & Papazian, 2012; Spieth & Zhang, 2014). Theoretically, these

agents reduce ventilator-induced lung injury and may have an anti-inflammatory effect (Hraiech et al., 2012). Risks associated with neuromuscular blockers include ICU-acquired weakness and post-traumatic stress disorder (Hraiech et al., 2012). Adequate sedation is required when neuromuscular blockers are used.

Statins

This class of antilipemics have some immunomodulatory and anti-inflammatory effects that show some possible protective benefits in critically ill patients with ARDS but need further testing in randomized controlled trials (Spieth & Zhang, 2014).

Corticosteroids

The anti-inflammatory properties of corticosteroids seemed a logical solution to the cytokine-mediated inflammatory response in ARDS, and studies investigating corticosteroids have been ongoing since the 1980s. The use of corticosteroids is controversial with conflicting studies on its effectiveness in improving outcomes. If used, low doses may be preferable to higher doses (Spieth & Zhang, 2014).

Vasodilators

Two vasodilators have been in ARDS nitric oxide (NO) and epoprostenol. NO is produced in the lungs and relaxes pulmonary vascular smooth muscle, therefore playing a role in ventilation perfusion ratios. Inhaled NO improves oxygenation by promoting blood flow to well-ventilated areas of the lung but has not been proven to improve outcomes at this time. Epoprostenol is a synthetic prostacyclin, which in addition to vasodilation, has anti-platelet effects. It is proposed as a rescue therapy for refractory cases of ARDS (Arumpanayil, 2013).

Surfactant and β -Agonists

In ARDS, surfactant production is impaired, which increases the surface tension of alveoli resulting in atelectasis. Theoretically, instilling surfactant into the lungs would help reduce surface tension, but this practice has been found effective only in the pediatric population and does not seem to improve outcomes in adults. Use of β -agonists such as albuterol had appeared promising due to their ability to reduce pulmonary edema and inflammation and up-regulate sodium and water transport. Unfortunately, recent studies do not support their use; β -agonists may actually be harmful (Singh et al., 2014).

Fluid Management

Managing fluids in the ARDS patient remains an area of controversy as well. While conservative fluid management

has not been proven to reduce mortality, it has been shown to improve oxygenation reduces the duration of mechanical ventilation (Gattinoni, Cressoni, & Brazzi, 2014). Conservative fluid strategies have a possible connection to late cognitive impairment (Gattinoni et al., 2014). It is important that fluid management be tailored to the patient's condition. Fluid resuscitation should be goal directed and fluid overload avoided. Crystalloid solutions with lower chloride content should be used when possible (Gattinoni et al., 2014). Albumin and furosemide may be helpful in further reducing edema, but should be done cautiously to avoid volume depletion and subsequent organ dysfunction (Gattinoni et al., 2014).

Nutrition

Adequate nutrition plays an important role in protecting the critically ill patient from gastrointestinal complications, infection, and reducing inflammation. Omega-3 fatty acid supplementation theoretically modulates the immune response in ARDS and showed some promise in early research, but recent studies have shown inconsistent results (Garcia de Acilu et al., 2015; Spieth, & Zhang, 2014). Trophic feeding, or "trickling" in the tube feeding was suggested as having an immune-modulating effect, but a recent randomized controlled trial showed no benefit of trophic feeding over full enteral feeding (Rice et al., 2012).

NURSING ACTIONS

Nursing care of patients with ALI/ARDS is supportive of the medical goals of maintaining tissue perfusion and protecting the lungs from injury.

Prevention

As always, prevention is the ideal treatment method for any syndrome. Simple nursing actions such as hand washing can greatly reduce transmission of microorganisms. Attention to line care and oral care and keeping the head of the bed in the proper position all contribute to prevention of nosocomial infections. VAP prevention is key, as sepsis and pneumonia are common causes of ARDS.

Sedation

The nurse should attempt to minimize the patient's O₂ demand and improve ventilator synchrony. Table 3-9 lists the impact of various activities on O₂ consumption. To minimize these effects, sedation is a standard of care. Commonly used agents include propofol, fentanyl, and midazolam. Current practice allows for paralysis when sedation alone is not effective.

Table 3-9 Activities That Increase Oxygen Consumption

Activity	Percent Increase in vo_2
Dressing change	10
Physical exam	20
Bath	23
Chest x-ray	25
Suctioning	27
Increased work of breathing	40
Position change	31
Linen change—occupied bed	22

Source: Vollman, 2007.

Positioning

Prone positioning relieves cardiac compression and allows normally dependent (dorsal) areas of the lung to participate in gas exchange, reaching uninjured alveoli (Gattinoni, Taccone, Carlesso, & Marini, 2013). The prone position is

recommended for severe ARDS as defined by the Berlin definition and should be considered whenever the P/F ratio is less than 150mmHg, PEEP of 5 cm H_2O , and FiO_2 is greater than 60% (Gattinoni et al., 2013). Recent studies suggest that when combined with lung protective ventilation, for patients with severe ARDS, prone positioning may improve survival (Gattinoni, et al., 2013). It is not recommended for mild cases of ARDS.

Contraindications for prone positioning include spinal injuries, elevated intracranial pressure, abdominal compartment syndrome, and hemodynamic instability. Complications included transient hypotension and desaturation, accidental extubation, and catheter dislodgment. Longer periods in the prone position may have additional complications including increased sedation, vomiting, pressure sores, and facial edema (Gattinoni et al., 2013). Before proning, the patient's eyes are lubricated and secured, and great attention is given to the security of all lines and drains. Safety during this process requires several staff members, specialized equipment, and an organized approach.

Respiratory Failure Summary

Caring for patients with acute respiratory failure (ARF) provides a significant challenge for the healthcare team. However, the array of supportive modalities, such as mechanical ventilation, requires skilled nursing care and the constant application of critical thinking. Healthcare researchers, including nurses, continue to research interventions that will optimize the care of patients with ARF. The needs of these patients and their families require the entire healthcare team, including spiritual caregivers. The nurse functions as the coordinator for this team and ensures the provision of holistic, optimal care to patients with ARF. The care these patients require is summarized in the visual map.

The nurse caring for a patient in acute respiratory failure must understand the pathophysiologic processes underlying the disease. Advanced ventilatory support requires close and careful assessments and monitoring. The nurse must consider the implications, consequences, and evidence supporting every intervention. Care should be taken to assist the patient and family in understanding the care provided and allow for optimal communication strategies. Using evidence-based practice enables the nurse to prevent complications and improve outcomes.

Why/Why Not?

The nurse has received report from another unit on Ms. Jones, a patient with a history of severe COPD and oxygen dependence admitted for pneumonia. Ms. Jones is being transferred to the ICU for worsening respiratory distress and is currently obtunded. Her BP is 92/56(67) and RR 38, O_2 saturation is 86%. The provider has ordered NIV with

bilevel positive airway pressure with IPAP 16 cm H_2O and EPAP 8 cm H_2O . Determine if this is an appropriate intervention to manage her respiratory failure. Why or why not?

See answers to Why/Why Not? in the Answer Section.

Case Study

Mark Donnelly is a 54-year-old patient admitted 2 days ago in severe DTs. Immediately prior to his admission, he vomited, had a seizure, and probably aspirated. His previous

medical history includes hypertension, COPD, and an MI treated with angioplasty and a stent. According to his wife, he was taking Toprol and Lipitor regularly prior to admission

and has smoked a pack a day for the past 30 years. He had been drinking a fifth of vodka daily until 3 days prior to admission when he stopped completely. He is 5'11" and weighs approximately 180 pounds. On arrival in the ED, his blood pressure was 193/124 with a heart rate of 150. He was tremulous, nauseated, and agitated. Lorazepam was started according to CIWA protocol, but he began to vomit and had several seizures. He was sedated with propofol and Zemuron, emergently intubated, ventilated, and transferred to the ICU. On arrival in the ICU, his blood pressure was 80/52.

1. What probably caused his blood pressure to drop?
2. What strategies should the nurse institute immediately to prevent VAP?
3. Will his current BP affect the implementation of any of the strategies?
4. Would his DTs and mental status affect the implementation of any of these strategies?

During the initial assessment on his second day of hospitalization, his ET tube was noted to be 24 cm at the lips. His nurse heard crackles and rhonchi in the bases, especially on the right. His chest x-ray showed that his ET tube was

approximately 5 cm above the carina. He had infiltrates and a consolidation on the right with an elevated diaphragm. The nurse suctioned him for copious amounts of pale yellow secretions.

5. What, if any, inferences can the nurse draw from these assessment findings and the results of the chest x-ray?
6. What should the nurse do about these?
7. What factors could predispose Mr. Donnelly to the development of ARDS?

Mr. Donnelly was being mechanically ventilated. On ventilator settings of SIMV rate 14, tidal volume 600, FiO₂ 50%, PEEP 8, his respiratory rate was 15, he was maintaining an oxygen saturation of 94%, and his peak inspiratory pressures were 22 to 28. His ABGs revealed pH 7.42, PaO₂ 96, PaCO₂ 48, and HCO₃ 29.

8. What information, if any, do his blood gases reveal?
9. If his ventilator is set for 14 breaths per minute with a mode of SIMV, how is he able to breathe at a rate of 15? Why is this useful for a patient?

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 3.1 Explain the following ventilator settings:
 - a. Oxygen concentration (FiO₂)
 - b. Tidal volume
 - c. Rate
 - d. Inspiratory:expiratory ratio
 - e. High-pressure limit
 - f. Pressure support
 - g. Positive end-expiratory pressure (PEEP)
- 3.2 What conditions may cause ALI/ARDS? Why can so many different conditions result in the development of ALI/ARDS?

- 3.3 How does ALI/ARDS progress?
- 3.4 What are the early manifestations of ALI/ARDS? What is the hallmark of ARDS?
- 3.5 What factors are associated with increased mortality for patients with ARDS?
- 3.6 What does the evidence suggest are the best ventilator settings for patients with ARDS? Why?
- 3.7 What are three nursing priorities when caring for the ventilated patient with ARDS?

See answers to Chapter Review Questions in the Answer Section.

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Chapter 4

Interpretation and Management of Basic Dysrhythmias

Carrie Edgerly MacLeod, PhD, APRN

Abbreviations

ACLS	Advanced Cardiac Life Support	ICD	Implantable Cardioverter-Defibrillator
AED	Automated External Defibrillator	NSR	Normal Sinus Rhythm
CRT	Cardiac Resynchronization Therapy	RFCA	Radiofrequency Catheter Ablation
ECG, EKG	Electrocardiogram	TEE	Transesophageal Echocardiogram

Learning Outcomes

Upon completion of this chapter, the learner will be able to:

1. Articulate the basic electrophysiology of the cardiac system.
2. Interpret the basic components of the electrocardiogram.
3. Interpret sinus dysrhythmia waveforms and prioritize effective collaborative care.
4. Interpret atrial dysrhythmia waveforms and prioritize effective collaborative care.
5. Interpret atrioventricular dysrhythmia waveforms and prioritize effective collaborative care.
6. Interpret ventricular waveforms and prioritize effective collaborative care.
7. Compare electrophysiology treatment options for dysrhythmias.

Introduction

To understand basic cardiac dysrhythmias, an understanding of the electrical activity of the heart and the normal heart rhythm is needed. The cardiac rhythm is usually determined by recording and examining an electrocardiogram, or ECG, strip. Another common abbreviation for electrocardiogram is EKG, which comes from the German word *elektrokardiogramm*. A normal heart rhythm, referred

to as normal sinus rhythm, is seen on the ECG when an electrical impulse begins at the heart's primary pacemaker, the sinus node or sinoatrial (SA) node, and travels through the heart's electrical conduction system unimpaired at a normal rate and rhythm. If there are disturbances of the cardiac electrical impulse formation or conduction, or both, an abnormal rhythm, or cardiac dysrhythmia, results. The terms "dysrhythmia" and "arrhythmia" are used interchangeably; however, some people prefer to use

arrhythmia to refer to the loss of rhythm, as the prefix “a” means “without.” Dysrhythmias may be life threatening, markedly altering the cardiac output and causing deterioration in vital signs, or they may be benign, or somewhere in between. To interpret and manage basic dysrhythmias, an understanding of basic cardiac electrophysiology is necessary, including the special properties of cardiac cells, the conduction system, and the electrocardiogram.

Basic Electrophysiology

Cardiac electrophysiology is the study of the electrical properties of the cardiac muscle. These properties ensure that electricity flows through the heart in a regular, measured pattern and results in contraction of the cardiac muscle.

Properties of Cardiac Cells

Specialized cardiac cells have unique characteristics that allow them to regulate heart rate and rhythm. These characteristics include automaticity, excitability, conductivity, and contractility.

Automaticity is the pacing function or ability of cardiac pacemaker cells to spontaneously initiate an electrical impulse. Normally only the primary pacemaker cells, the sinoatrial SA node, the atrioventricular (AV) junction, and the Purkinje fibers possess the characteristic of automaticity. ♦ Figure 4-1 depicts the electrical conduction system of the heart.

Under certain conditions, however, such as myocardial infarction (MI), electrolyte imbalances, hypoxia, and drug toxicity, any cardiac cell may exhibit this characteristic and initiate electrical impulses, generating dysrhythmias.

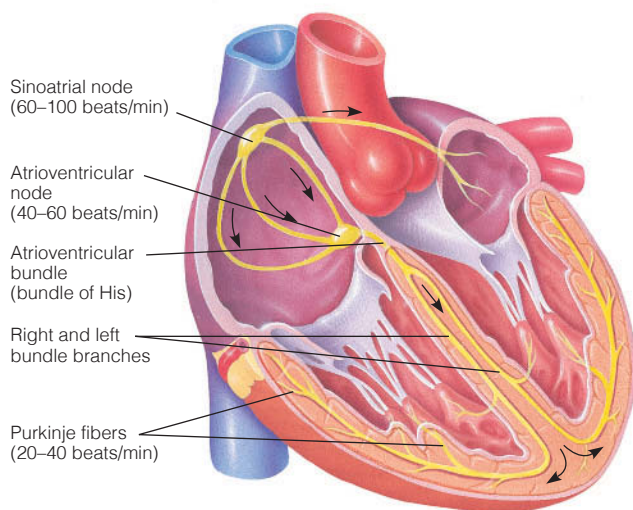


Figure 4-1 The electrical conduction system of the heart.

Excitability is a characteristic shared by all cardiac cells that refers to the ability to respond to an electrical impulse generated by pacemaker cells or other external stimulus, such as from a chemical, mechanical, or electrical source. These stimuli cause depolarization, which occurs when the cells become electrically excited due to an ionic imbalance across the membrane of the cells.

Conductivity is the ability of the cardiac cells to transmit the electrical impulse to adjacent cardiac cells. This allows an electrical impulse in any part of the myocardium to spread throughout the heart as excitable cells depolarize in rapid succession. The resultant wave of depolarization creates the deflections seen on the ECG. Disturbances in conduction can result in electrical impulses that travel too fast or too slow, that are blocked, or that follow abnormal electrical pathways, generating dysrhythmias.

Contractility is the ability of the cardiac cells to shorten in response to electrical stimulation. Contractility is a mechanical event rather than an electrical event. The wave of depolarization initiates the contraction of the cardiac cells, creating the mechanical activity of the heart and propelling the blood forward. Certain medications, such as digoxin, dopamine, and epinephrine, can help to enhance the contractility of the heart. Conductivity without contractility is a medical emergency known as pulseless electrical activity (PEA). This happens when the electrical activity of the heart can be seen on the ECG, but the patient does not have a pulse because there is no mechanical activity in response to the electrical stimuli or the heart cannot contract or fill due to mechanical issues such as tension pneumothorax or cardiac tamponade.

Basic Electrophysiology—Cardiac Action Potential

Cardiac action potential refers to the change in the electrical charge inside the cardiac cell when it is stimulated. This electrical change is caused by the flow of specific ions, or electrically charged particles, into and out of the cardiac cell. This electrical change creates a series of events to occur known as polarization, depolarization, and repolarization.

Polarization is the electrical state that exists at the cardiac cell membrane when the cell is at rest. In this state no electrical activity is occurring, and the ECG displays a flat, isoelectric line. When the cardiac cell is polarized, the inside of the cell is more negative than the outside of the cell because of the ions found inside it. During the resting state, potassium ions leak out of the cell, leaving the negatively charged ions inside.

Depolarization is the opposite of polarization and occurs when there is a reversal of the electrical charges

at the cell membrane. The inside of the cell becomes more positive due to the rapid influx of sodium ions into the cell. On the ECG, the P wave represents atrial depolarization and the QRS complex represents ventricular depolarization. Depolarization is the electrical event that results in a contraction of the cardiac muscle, a mechanical event.

Repolarization is the restoration of the polarized state at the cell membrane. Repolarization can be thought of as the recovery of the cell to its original polarized state. After the cell depolarizes, the diffusion of sodium ions into it stops and potassium ions diffuse out, leaving mostly negatively charged ions inside the cell. On the ECG, the ST segment and the T wave represent ventricular repolarization.

Refractory Periods

Refractory is a term that refers to the resistance of the cell membrane to respond to a stimulus. The refractory period extends beyond the length of the cardiac contraction and protects the cardiac muscle from spasm or tetany. There are three refractory periods: the absolute refractory period, the relative refractory period, and the supernormal period.

The **absolute refractory period** is the brief period during depolarization when the cells will not respond to further stimulation, no matter how strong the stimulus. This period corresponds with the onset of the QRS complex to the peak of the T wave.

The **relative refractory period** is also known as the vulnerable period. During this period some cardiac cells have repolarized, and if they receive a stronger than normal stimulus they may respond. This response is known as the “R-on-T phenomenon,” because it occurs when a stimulus, causing “QRS” depolarization, lands on the vulnerable downslope of the “T” wave. This phenomenon may cause a life-threatening dysrhythmia known as ventricular fibrillation.

The **supernormal period** comes after the relative refractory period. During this period a weaker than normal stimulus can cause depolarization of cardiac cells. On the ECG, this period corresponds to the end of the T wave. Cardiac dysrhythmias may also occur during this period.

♦ Figure 4-2 depicts the cardiac cell action potential: polarization, depolarization, polarization as well as refractory periods.

The Cardiac Conduction System

The cardiac conduction system is composed of the SA node, the AV node, the bundle of His, the left and right bundle branches, and the Purkinje fibers. This system of specialized cardiac cells is responsible for the generation

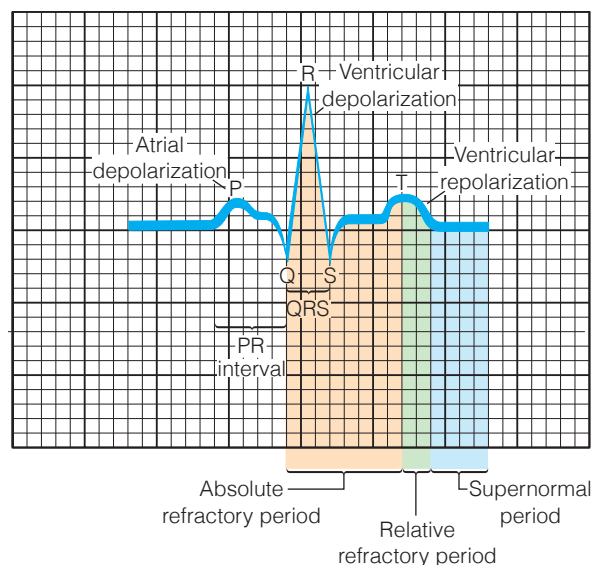


Figure 4-2 Cardiac Cell Action Potential.

and conduction of electrical impulses that cause atrial and ventricular depolarization (refer to Figure 4-1).

The **sinoatrial (SA) node**, also referred to as the sinus node, is the heart's dominant pacemaker and is located in the upper-posterior wall of the right atrium. The SA node generates stimuli at regular intervals that result in a wave of depolarization. This electrical activity is recognized on the ECG monitor as “Sinus Rhythm.” The SA node possesses the greatest degree of automaticity of the cardiac cells and spontaneously generates electrical impulses at a rate of 60 to 100 beats per minute. The wave of depolarization disperses away from the SA node, via slow and fast conduction pathways, in all directions throughout both atria, causing them to contract. The waves of depolarization are created by movement of the positively charged sodium ions into the cardiac cells. This positive charge is seen on the ECG monitor as an upward wave, which represents atrial depolarization. This upward wave is called the P wave. The depolarization of the atria should lead to atrial contraction. This atrial contraction forces blood through the atrioventricular valves (mitral and tricuspid) located between the atria and ventricles.

When the wave of atrial depolarization enters the atrioventricular junction area, the AV node, conduction markedly slows down. The **atrioventricular (AV) node** is located on the posterior wall of the right atrium just behind the tricuspid valve and is the only electrical conduction pathway between the atria and ventricles. The slowed conduction is attributed to the slow-moving calcium ions that carry the electrical stimuli through the AV node. This slowed conduction allows time for the atria to contract and the ventricles to fill with blood. This creates an

electrical pause on the ECG monitor, which is seen as a flat line after the P wave known as the PR interval. The AV junction, if not electrically stimulated from the SA node, can spontaneously generate electrical impulses at a usual rate of 40 to 60 beats per minute and act as the pacemaker for the heart. This is known as an “escape mechanism” because this secondary pacemaker tries to compensate for the failed SA node.

Conduction speeds up again as the wave of depolarization enters the ventricular conduction system. The ventricular conduction system originates at the **bundle of His**, which penetrates the AV valves, then bifurcates into the right and left bundle branches to the terminal filaments of the **Purkinje fibers**. The entire ventricular conduction system is composed of Purkinje fibers, which use fast-moving sodium ions for the conduction of depolarization. This allows for rapid depolarization of the ventricles, which is recorded on the ECG monitor as the QRS complex. Ventricular depolarization causes ventricular contraction, sending the blood out of the ventricles into the aorta and pulmonary artery. Occasionally, some of the cells that make up the Purkinje fibers will demonstrate automaticity, initiating an electrical impulse and causing ventricular beats or rhythms. These cells can generate electrical impulses at a rate of 20 to 40 beats per minute. If there are no electrical impulses being generated from the SA or AV nodes, the Purkinje fibers may try to compensate by acting as a tertiary pacemaker.

The Electrocardiogram

The electrocardiogram (ECG) provides a graphic image of the electrical activity of the heart. The electrical waves of depolarization and repolarization are weakly transmitted to the body surface, where they are picked up by the conductive gel within the electrode pads and transmitted to the ECG machine. Besides monitoring a patient’s heart rate, the ECG can provide information related to conduction disturbances, electrical impulse disturbances, the presence of myocardial ischemia, and electrical effects of medications and electrolyte imbalances.

The Leads

Most cardiac monitors will show selected leads or views of the heart’s electrical activity. The standard ECG provides 12 leads, or separate views of the same myocardial electrical activity. The lead is a graphic image of the electrical current flowing between two electrodes. The 12 leads are divided into two main types: six limb leads and six chest leads. The limb leads are identified as Leads I, II, III, and aV_R , aV_L , and aV_F . The chest leads are identified by V_1 through V_6 . ♦ Figure 4-3 shows the leads of the 12-lead ECG.

The limb leads provide a view of the frontal plane of the heart, or the heart viewed from the front of the body. The chest leads provide a view of the horizontal plane of the heart, or the heart viewed in a cross-sectional slice, as if it were sliced in half horizontally. All the leads are recording the same electrical activity, but the ECG tracing looks different in each lead because the electrical activity is viewed from different positions. The placement of the positive electrode determines the portion of the heart viewed by that lead; in other words, it acts as the camera. Table 4-1 describes where the positive electrode for each lead is placed on the body, and which aspect of the heart is electrically visualized by that lead.

Each lead also has a negative and a grounding electrode, but to emphasize the relationship between the positive electrode and the aspect of the heart that is visualized, only the positive lead is shown in the table.

The ECG Paper

The electrical activity that is viewed from the various leads is recorded on ECG paper. The paper is graph paper made up of small and large boxes, with each small box measuring 1 mm high and 1 mm wide. Each large box has the width and height of 5 mm as shown in ♦ Figure 4-4.

The horizontal axis of the paper represents time. All ECG monitors record at a standard speed of 25 mm/sec. At this speed, each small horizontal box represents 0.04 second ($25 \text{ mm/sec} \times 0.04 \text{ sec} = 1 \text{ mm}$). Each large box contains five small boxes, which is equivalent to

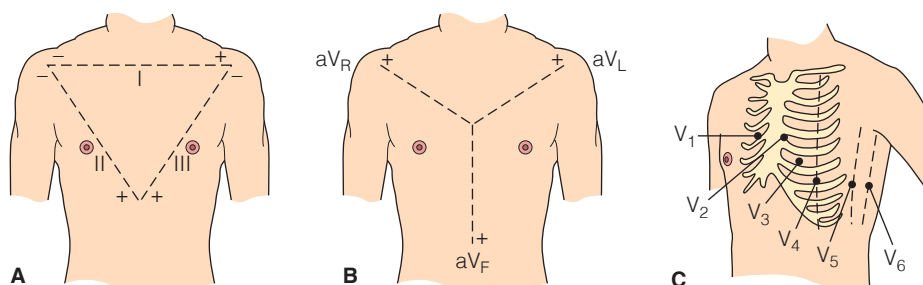


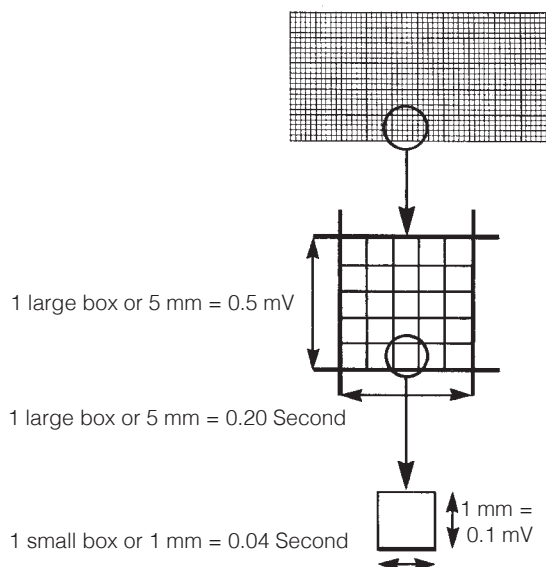
Figure 4-3 Leads of the 12-lead ECG.

Table 4-1 Position of Lead and Heart Surface Viewed

Lead	Position of Positive Electrode	Heart Surface Viewed
Limb Lead 1	Left arm or under the left clavicle	Lateral
Limb Lead 2	Left leg or lowest rib, left midclavicular line	Inferior
Limb Lead 3	Left leg or lowest rib, left midclavicular line	Inferior
Limb Lead aV _R	Right arm or under the right clavicle	None
Limb Lead aV _L	Left arm or under the left clavicle	Lateral
Limb Lead aV _F	Left leg or lowest rib, left midclavicular line	Inferior
Chest Lead V ₁	Right side of sternum, fourth intercostal space	Septum
Chest Lead V ₂	Left side of sternum, fourth intercostal space	Septum
Chest Lead V ₃	Midway between V ₂ and V ₄	Anterior
Chest Lead V ₄	Left midclavicular line, fifth intercostal space	Anterior
Chest Lead V ₅	Left anterior axillary line, horizontal to V ₄	Lateral
Chest Lead V ₆	Left midaxillary line, horizontal to V ₄	Lateral

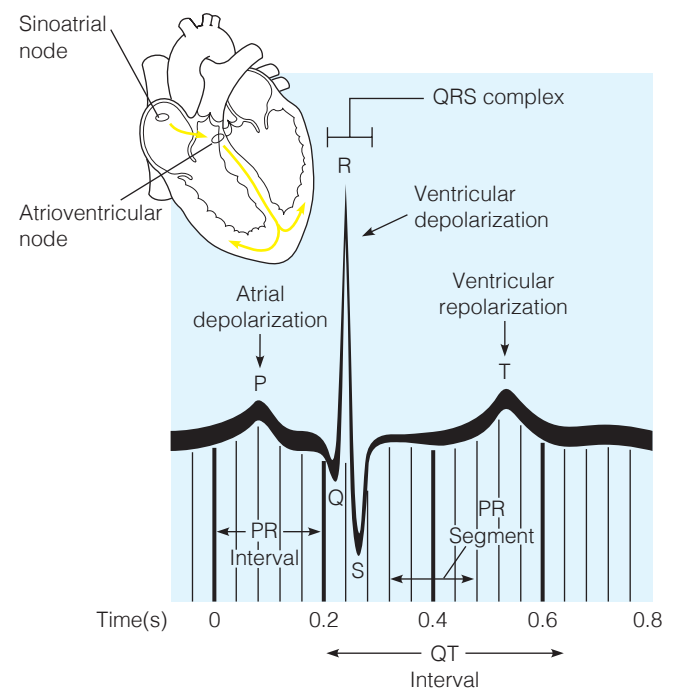
0.20 second ($5 \times 0.04 \text{ sec} = 0.20 \text{ sec}$). Following this same logic, five large boxes represent 1 second ($5 \times 0.20 \text{ sec} = 1 \text{ sec}$). Vertical lines, or hash marks, at the top or bottom of the ECG strip denote 15 large block segments, representing 3-second strips. This information is useful when ECG strips are analyzed to determine the cardiac rate.

The vertical axis of the ECG paper represents voltage or amplitude of the waveforms. The height and depth of a waveform are measured vertically from the baseline in millimeters (mm). The waveform is a deflection either upward or downward from the baseline of the ECG recording. When a wave of depolarization (positive charges) moves toward a positive electrode, this produces a positive or upward deflection on the ECG. This is a measurement of the wave's voltage. Conversely, when a wave of depolarization moves away from a positive electrode, this produces a negative or downward deflection on the ECG.

**Figure 4-4** Time and voltage measurements on ECG paper recording at a speed of 25 mm/second.

ECG Waveforms

A full cardiac cycle of depolarization and repolarization creates particular waveforms on the ECG. The cardiac cycle is composed of waveforms, segments, intervals, and complexes. ♦ Figure 4-5 depicts a normal cardiac cycle. As discussed earlier, a waveform is a deflection either upward or downward from the ECG baseline, such as the P wave or T wave. A segment is a line between waveforms that usually falls on the ECG baseline, such as the PR segment and the ST segment. An interval is a waveform and an adjoining segment, such as the PR interval and the QT interval. A complex is made up of multiple deflections, such as the QRS complex.

**Figure 4-5** The normal cardiac cycle with ECG waveforms, segments, and intervals.

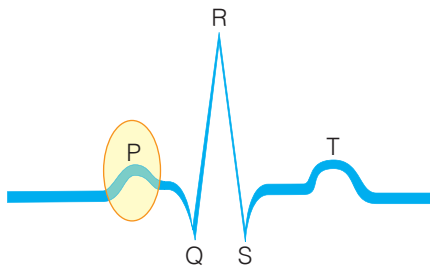


Figure 4-6 P wave.

The P Wave

The cardiac cycle starts with the P wave (♦ Figure 4-6). The P wave results from electrical firing of the SA node, and the resulting atrial depolarization. P waves are usually round and smooth positive deflections on the ECG. However, in certain leads, the P waves may be negative or biphasic deflections. For the sake of this chapter, however, Lead II is referred to in the examples and discussions of wave deflections because this lead is quite commonly used and normally has a positively deflected P wave, QRS complex, and T wave. When all of the P waves originate from the SA node, they all have the same configuration, so they all look the same. If an ectopic pacemaker, or different cardiac cell, fires and stimulates atrial depolarization, the P wave will have a different configuration from the other P waves.

The PR Segment

After the P wave, there is a short segment that usually falls on the isoelectric line (or ECG baseline) (♦ Figure 4-7). This results from the short pause in electrical conduction as the wave of depolarization slows down while traveling through the AV junction. The duration of the PR segment depends on the length of time it takes for the electrical impulse to conduct through the AV junction.

The PR Interval

The entire P wave and PR segment make up the PR interval (♦ Figure 4-8). Electrically speaking, the PR interval reflects depolarization of the right and left atria (P wave) and the impulse delay through the AV junction (PR segment). This interval measures from the beginning of the P wave

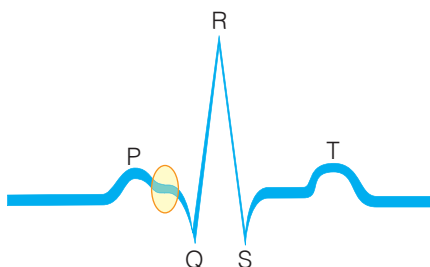


Figure 4-7 PR segment.

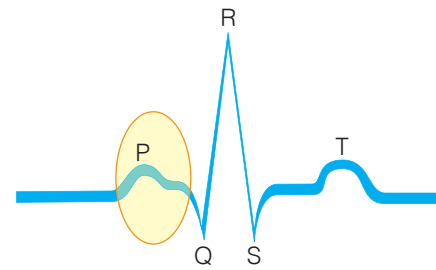


Figure 4-8 PR interval.

to the beginning of the QRS complex. It normally measures 0.12 to 0.20 second, or three to five small boxes on the ECG paper. A PR interval longer than 0.20 second usually indicates a delay in impulse conduction through the atria or AV junction. A PR interval shorter than 0.12 second may indicate that the impulse originated from an ectopic pacemaker in the atria, rather than the SA node, that was located closer to the AV node.

The QRS Complex

The QRS complex is made up of three waveforms (♦ Figure 4-9).

The three waveforms are not all visualized in all leads, so the QRS complex may actually have one, two, or three waveforms. The Q wave is the first downward deflection after the P wave. The R wave is the first upward deflection in the QRS complex. The S wave is the downward deflection following the R wave. The QRS complex represents the depolarization of both ventricles. The QRS is larger than the P wave because the ventricles have a much larger muscle mass to depolarize than the atria. The QRS complex is measured from the point where the first waveform of the complex deviates from baseline to the point where the last waveform begins to level out. The QRS complex normally measures less than 0.12 second, or less than three small boxes. A QRS complex that is wider than 0.12 second usually is the result of an electrical impulse originating from an ectopic pacemaker in the ventricles. An abnormally deep Q wave (such as one that is more than 1/3 of the entire height of the QRS complex) may indicate cardiac cell necrosis from a previous myocardial infarction.

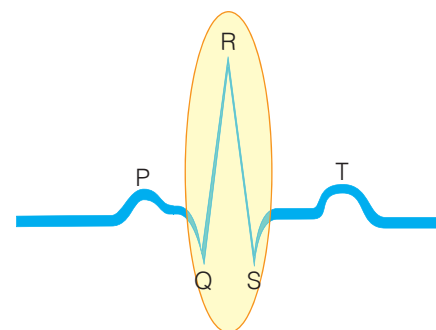


Figure 4-9 QRS complex.

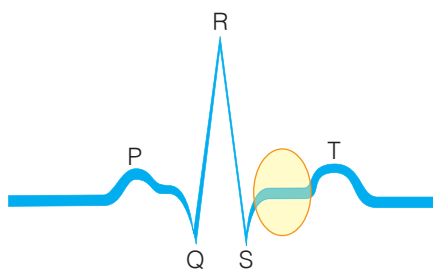


Figure 4-10 ST segment.

The ST Segment

The ST segment is usually an isoelectric line between the QRS complex and the T wave. It begins at the end of the QRS complex and ends at the beginning of the T wave (♦ Figure 4-10). The ST segment represents early repolarization of the ventricles. The ST segment is considered elevated if it deviates above the isoelectric line, and it is considered depressed if it deviates below the isoelectric line by more than about 1 mm, or one small box. ST segment depression may be indicative of myocardial ischemia; ST segment elevation may be indicative of myocardial injury and may occur during an MI.

T Wave

The T wave follows the ST segment and represents ventricular repolarization. The T wave is usually rounded and deflected in the same direction as the preceding QRS complex (♦ Figure 4-11). A negative T wave following a positive QRS complex is suggestive of myocardial ischemia. Tall and peaked T waves may be suggestive of hyperkalemia. If an ectopic stimulus excites the ventricles during the downward slope of the T wave, the vulnerable period, it may cause ventricular irritability and a resulting lethal dysrhythmia. This is known as the R-on-T phenomenon.

The QT Interval

The entire QRS complex, ST segment, and T wave make up the QT interval (♦ Figure 4-12). The QT interval represents the total time required for ventricular depolarization to repolarization. It is measured from the beginning of the QRS complex to the end of the T wave. The length of the

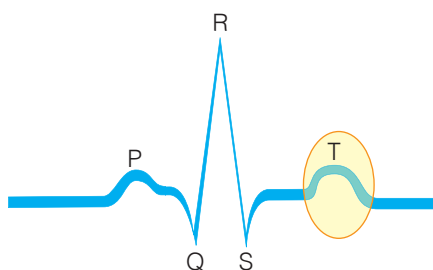


Figure 4-11 T wave.

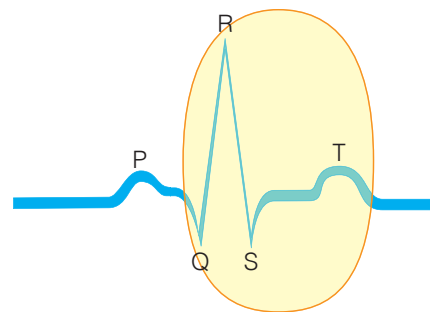


Figure 4-12 QT interval.

QT interval varies with the patient's age, gender, and heart rate but, typically, it is 0.38 to 0.42 second. The interval becomes shorter with faster heart rates, and longer with slower heart rates; therefore, the corrected QT (QTc) is usually calculated taking into account the patient's heart rate. This calculation is the QT interval/ $\sqrt{\text{R-R interval}}$ and estimates the QT interval at a heart rate of 60 bpm. Most cardiac monitors will calculate the QTc. A prolonged QT interval may be congenital or as a result of certain medications and electrolyte imbalances such as some antidysrhythmic drugs and low levels of potassium, calcium, or magnesium. A prolonged QT interval causes premature action potentials during the late phases of depolarization and may develop into fatal dysrhythmias such as ventricular tachycardia, torsades de pointes, or ventricular fibrillation.

Interpreting Cardiac Rhythm

Until the nurse has gained experience at reading ECG rhythm strips, it is important to approach reading them in a step-by-step manner. The seven steps of interpreting a cardiac rhythm are:

1. Determine the heart rhythm.
2. Measure the heart rate.
3. Examine the P waves.
4. Examine the P to QRS ratio.
5. Measure the PR interval.
6. Examine the QRS complex.
7. Interpret the rhythm.

Step One: Determine the Heart Rhythm

The first step is to determine whether the cardiac rhythm is regular or irregular. Irregular rhythms can be regularly irregular, mostly regular with some irregularity, or totally irregular. The SA node generates impulses at regular intervals, producing waves of depolarization in regularly repeating cycles. If the ventricular rhythm is regular, the R to R interval will be relatively the same for all of the cardiac cycles. If the atrial rhythm is regular, the P to P interval will be relatively similar for all of the cardiac cycles.

A dysrhythmia that does not have a regular rhythm may be caused by the firing of ectopic pacemakers, resulting in a rhythm that is mostly regular with some irregularity. Atrial fibrillation is a common dysrhythmia that usually causes a totally irregular cardiac rhythm. Once the rhythm has been determined, then it is time to calculate the heart rate. For a regular heart rhythm, any of the three methods discussed next can be used to calculate the rate. For irregular rhythms, only the first method (the 6-second method) will be accurate.

Step Two: Measure the Heart Rate

As mentioned earlier, in adults the SA node generates electrical impulses at a rate of 60 to 100 beats per minute; therefore, the normal heart rate is 60 to 100 beats per minute. A rate that is less than 60 beats per minute is considered bradycardic, and a rate that is greater than 100 is considered tachycardic. Both the atrial and ventricular rates should be the same if there are no problems with the conduction and impulse generation. There are many different ways to calculate the cardiac rate. Following are some of the most common methods.

- **6-Second Method:** Most ECG paper is printed with a 3-second hash mark at the top or bottom of the rhythm strip. The fastest way to determine the cardiac rate is to count the number of R waves (usually the most prominent deflection of the QRS complex) in a 6-second period and multiply it by 10 to find the ventricular heart rate in 1 minute. For example, refer to the rhythm strip on page 86 in which there are five R waves in the 6-second ECG strip; multiply 5 by 10 and the result is a ventricular rate of about 50. This method gives a quick average of the cardiac rate and is most appropriate for irregular cardiac rhythms. For regular rhythms the large box method may also be used.
- **Large-Box Method:** This method is appropriate for regular cardiac rhythms, but it would not be appropriate for irregular heart rhythms. To determine the atrial rate, the number of large boxes between two consecutive P waves is counted. To determine the ventricular rate, the number of large boxes between two consecutive R waves is counted. The number of large boxes between the waveforms is then divided into 300, because there are 300 large boxes in 1 minute. For example, if there are two large boxes between two consecutive R waves, the ventricular rate would be 150 ($300/2 = 150$). This method is similar to the memory method.
- **Memory Method:** To determine the ventricular rate, the nurse should find an R wave that falls on one of the dark lines of a large box. Then, with the first dark line that the R wave falls on being 0, the nurse should recite the following numbers while counting each large box line after that R wave until the second R wave is

reached: 300, 150, 100, 75, 60, 50, 43, 38, 33, and 30. This method is the same as the large-box method, except that the math is already figured out if both of the R waves fall on dark lines; however, if the second R wave does not fall on a dark line, the nurse can make a guesstimate of the difference between the two or calculate a more exact heart rate. The calculation is done by taking the difference of the two memorized numbers between which the second R wave falls and dividing it by 5. This number is used as the value of each little box that lies between the second R wave and the last dark line that immediately precedes it. From that last dark line, the number of small boxes is counted between it and the second R wave. That value is added to the memorized number above to get the heart rate. For example, if the second R wave lies between the memorized numbers of 60 and 50, the nurse would calculate the difference, $60 - 50 = 10$, then divide 10 by 5 to determine the value of each small box, which would be 2. If the second R wave lies two small boxes beyond the dark line that represented 60, the value of 2 would be added for each of the two boxes to 60; therefore, the heart rate would be 64.

Step Three: Examine the P Waves

As the SA node initiates the wave of depolarization in the atria, the P wave is produced. If all of the P waves are similar in size and shape, and occurring regularly, it can be assumed that they are all originating from the SA node. If there are differently shaped P waves, it can be assumed that they are originating from irritable foci in the atria. If the P waves are negatively deflected or absent, the rhythm may be originating from the AV junction, or the ventricles. Premature atrial contractions (PACs) are an example of a dysrhythmia that may cause irregularly shaped P waves.

Step Four: Examine the P to QRS Ratio

Each P wave should be followed by a QRS complex, signifying that the wave of depolarization has successfully moved from the atria to the ventricles. If there are more P waves than QRS complexes, it can be assumed that a dysrhythmia exists.

Step Five: Measure the PR Interval

The PR intervals should be the same for all cardiac cycles on the monitor strip, measuring approximately 0.12 to 0.20 second. AV blocks are an example of dysrhythmias in which the PR interval may be longer than normal.

Step Six: Examine the QRS Complex

All of the QRS complexes, like the P waves, should be similar in shape and size and should occur with regularity

across the ECG strip. All of the QRS complexes should measure approximately less than 0.12 second. A wide QRS complex measuring greater than 0.12 second probably originated in the ventricles. An example of this would be a premature ventricular contraction (PVC). A narrow QRS complex measuring less than 0.12 second probably originated above the ventricles, for example, in the SA node or AV node.

Step Seven: Interpret the Rhythm and Evaluate the Patient's Reaction

The final step is to interpret the cardiac rhythm and the assumed pacemaker site of origin. For example, “the monitor pattern reads sinus tachycardia at a rate of 120 beats per minute.” However, when interpreting the cardiac monitor always remember to examine and treat the patient, not the monitor! The nurse needs to determine how the patient is tolerating the cardiac rate and rhythm. The patient in sinus tachycardia at 120 beats per minute may be symptom-free and not require any treatment at all, aside from determining the cause of this tachycardia and resolving the cause. On the other hand, the patient may be showing signs of distress in this tachycardic rhythm and may need immediate interventions to decrease the cardiac rate. With any dysrhythmia, treatment will depend on the patient's reaction to the rhythm, which ultimately is determined by the patient's underlying health, length of time in the dysrhythmia, and amount of hemodynamic compromise being caused by the dysrhythmia.

Essential for Safety

Do not base treatment decisions solely on rhythm interpretation, neglecting clinical evaluation—treat the patient, not the monitor.

Hemodynamic Consequences of Dysrhythmias

Hemodynamics refers to the study of the forces that aid in circulating blood throughout the body (see Chapter 5 , *Cardiodynamics*). If any of these forces are compromised, the cardiovascular system is affected. The hemodynamic status of a patient is monitored by frequently assessing the blood pressure, pulse, mental status, urinary output, and cardiac output. A deviation from the patient's normal values may indicate hemodynamic compromise. With both tachydysrhythmias and bradydysrhythmias, a patient may experience hemodynamic compromise because the cardiac output may be affected. Cardiac output is a product of stroke volume and heart rate. A decrease in either stroke volume or heart rate will decrease the cardiac output. In tachydysrhythmias, the rate may be too fast to enable the ventricles to fill completely, causing a shortened

diastole and thereby decreasing stroke volume. As a result, after an initial increase, cardiac output and blood pressure will begin to decrease. In bradydysrhythmias, the rate may be too slow to provide adequate blood pressure and cardiac output. In both cases, coronary perfusion may be compromised.

There are many symptoms that may indicate a compromised hemodynamic status. These symptoms include, but are not limited to, hypotension, chest pain, congestive heart failure, decreased urine output, altered mental status, and other signs of shock. Symptoms may also include dizziness, diaphoresis, palpitations, shortness of breath, and syncope. After interpreting the rhythm, it is important to evaluate the clinical significance of the rhythm by determining the patient's reaction to the cardiac rhythm. With many dysrhythmias, the intervention will be dependent on the patient's reaction to the cardiac rhythm. As the following dysrhythmias are discussed, when reference is made to patients being symptomatic, it is important to refer to the above-mentioned symptoms. Also, it is important to remember that dysrhythmias may look somewhat different from patient to patient so following the steps of interpreting ECGs will be important as the heart rhythms are read.

Sinus Rhythms and Dysrhythmias

Sinus rhythms and dysrhythmias refer to rhythms that result from electrical impulses generated from the SA node. These rhythms typically have similarly shaped P waves because they all originate from the SA node ( Figure 4-13). The QRS complex follows each P wave, and the QRS complexes are usually narrow, or less than 0.12 second. The most common rhythms in this category include normal sinus rhythm, sinus arrhythmia, sinus bradycardia, and sinus tachycardia.

Sinus Rhythm

Sinus rhythm (SR), also referred to as normal sinus rhythm (NSR), is the rhythm that originates at the SA node. The SA

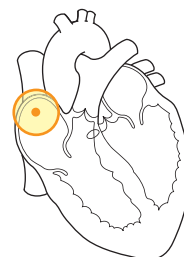


Figure 4-13 Origination of sinus rhythms.

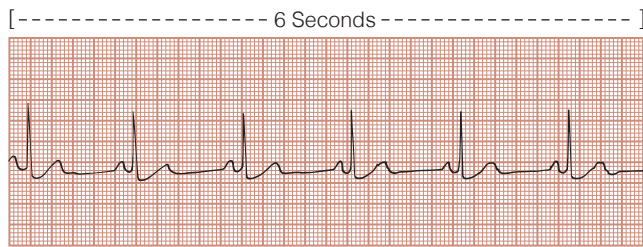


Figure 4-14 Normal sinus rhythm.

node typically initiates electrical impulses at a rate of 60 to 100 beats per minute; therefore, the atrial and ventricular rate for SR is 60 to 100. The P to P intervals and R to R intervals are regular. The P waves are uniform and upright, with one P wave preceding each QRS complex. The PR interval is between 0.12 and 0.20 second and remains constant throughout the monitor strip. The QRS complexes are less than 0.12 second and are uniform and upright.

♦ Figure 4-14 shows an example of sinus rhythm. In the 6-second strip there are six QRS complexes; multiply this by 10 seconds and the result is a rate of 60. The PR interval measures approximately three small boxes, and each small box is 0.04 second. Using the calculation of $3 \times 0.04 = 0.12$ second, the PR interval is found to be within normal parameters. The QRS complex spans about two small boxes, so using the calculation of $2 \times 0.04 = 0.08$ second, the QRS complex is also within normal limits.

Evaluation

In summary, for NSR the ECG interpretation will be as follows:

1. Heart rhythm—regular
2. Heart rate—60 to 100 beats per minute
3. P waves—uniform and upright
4. P to QRS ratio—one to one
5. PR interval—0.12 to 0.20 second
6. QRS complex—narrow, less than 0.12 second

Sinus Arrhythmia

Sinus arrhythmia is a variation of normal sinus rhythm and not a true arrhythmia in the sense that arrhythmia means “without rhythm.” Sinus arrhythmia is also referred to as respiratory sinus arrhythmia because it occurs with respiration. The heart rate increases during inspiration and decreases during expiration. The rhythm still originates at the SA node but is irregular with a repetitive variation in cycle length. The difference between the shortest and longest R to R interval is usually more than 0.12 second. The rate is usually 60 to 100 beats per minute but may be slightly faster or slower with inspiration and expiration. For example, ♦ Figure 4-15 shows sinus arrhythmia with a slower than normal rate of 50 beats per

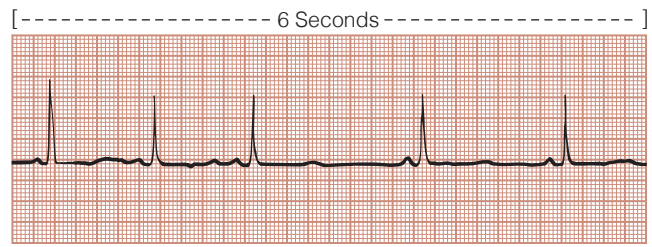


Figure 4-15 Sinus arrhythmia.

minute. The PR interval and the QRS duration are normal and consistent.

Evaluation

In general, for sinus arrhythmia the ECG interpretation will be as follows:

1. Heart rhythm—irregular, heart rate increases gradually with inspiration and decreases with expiration
2. Heart rate—60 to 100 beats per minute
3. P waves—uniform and upright
4. P to QRS ratio—one to one
5. PR interval—0.12 to 0.20 second
6. QRS complex—narrow, less than 0.12 second

Etiology

This normal phenomenon may be a result of reflex enhancement of sympathetic tone during inspiration and parasympathetic tone with expiration. Sinus arrhythmia is commonly seen in healthy children but may be seen in any age group. Occasionally sinus arrhythmia may be due to nonrespiratory causes as a result of medications such as digitalis and morphine.

COLLABORATIVE CARE

Sinus arrhythmia usually does not require any treatment because it seldom produces symptoms. If it is accompanied by a bradycardic heart rate that causes hemodynamic compromise, it can be treated as symptomatic sinus bradycardia as described in the next section.

Sinus Bradycardia

In sinus bradycardia (SB), the rhythm still originates at the SA node but at a decreased rate of less than 60 beats per minute for both the atria and ventricles. The P to P intervals and R to R intervals are regular. The P waves are uniform and upright, with one P wave preceding each QRS complex. The PR interval is between 0.12 and 0.20 second and remains constant throughout the strip. The QRS complexes are less than 0.12 second and are uniform

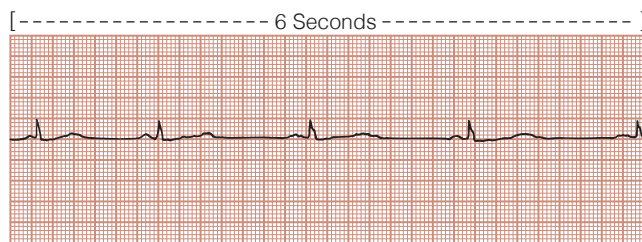


Figure 4-16 Sinus bradycardia.

and upright. ♦ Figure 4-16 shows an example of SB that has a rate of 50 beats per minute.

Evaluation

In summary, for SB the ECG interpretation will be as follows:

1. Heart rhythm—regular
2. Heart rate—less than 60 beats per minute
3. P waves—uniform and upright
4. P to QRS ratio—one to one
5. PR interval—0.12 to 0.20 second
6. QRS complex—narrow, less than 0.12 second

Etiology

SB may be normal during sleep and in well-trained athletes. It also results from excessive vagal or parasympathetic stimulation. Excessive vagal stimulation may be caused by vomiting, carotid sinus massage, suctioning, and Valsalva maneuvers such as bearing down for a bowel movement, choking, or gagging. SB may also result from an acute myocardial infarction, hypoxia, and medications such as digitalis, beta blockers, and calcium channel blockers.

COLLABORATIVE CARE

Treatment of SB is needed only if the patient is exhibiting signs of hemodynamic compromise. If any of these symptoms exist (as discussed earlier under “Hemodynamic Consequences of Dysrhythmias”), immediate intervention should be provided. All patients should receive oxygen, IV access, and continuous monitoring. The unstable patient should be immediately prepared for temporary external pacing (see the discussion of selected technologies later in this chapter). The drugs to consider while awaiting the temporary pacemaker include atropine (see “Commonly Used Medications”) and epinephrine or dopamine infusions (Neumar et al., 2015).

Sinus Tachycardia

In sinus tachycardia (ST), the rhythm still originates at the SA node but at an increased rate of greater than 100 beats per minute for both the atria and ventricles. The heart rate is usually between 100 and 180 beats per minute. The P to P intervals and R to R intervals are regular. The P waves are uniform and upright, with one P wave preceding each QRS complex. The PR interval is between 0.12 and 0.20 second and remains constant throughout the strip. The QRS complexes are less than 0.12 second and are uniform and upright. In ♦ Figure 4-17 there are 15 QRS complexes in the 6-second strip; multiplied by 10, this gives a rate of 150 beats per minute. The PR intervals are equal to three small boxes, or $3 \times 0.04 = 0.12$ second. The QRS complexes measure approximately two small boxes or $2 \times 0.04 = 0.08$ second. This strip is an example of sinus tachycardia.

Commonly Used Medications

Atropine

Introduction

Atropine is the first-line drug of choice for symptomatic bradycardia of any origin. Atropine is not recommended for routine use in the management of pulseless electrical activity (PEA) or asystole (Neumar et al., 2015).

Desired Effect: Atropine is an anticholinergic drug that blocks the cholinergic and parasympathetic stimulation of the heart, resulting in an increased heart rate. This effect should be seen within 1 to 2 minutes of the IV bolus administration.

Nursing Responsibilities:

- Administration of the therapeutic dose. The usual dose of atropine is 0.5 mg IV boluses repeated every 3 to

5 minutes to a total dose of 3 mg or six 0.5 mg doses (Neumar et al., 2015). Atropine may be given down an endotracheal tube or intraosseous cannulation (into a long bone) if IV access is unobtainable.

- Atropine administration should not delay the implementation of temporary external pacing for patients with poor perfusion.
- Assessment of cardiac rhythm for increase in heart rate.

Side and/or Toxic Effects: Because of the parasympathetic-blocking effect, side effects of atropine can include mouth dryness, urinary retention, blurred vision, and increased intraocular tension. Doses of less than 0.5 mg boluses may further slow the heart rate and should be avoided.

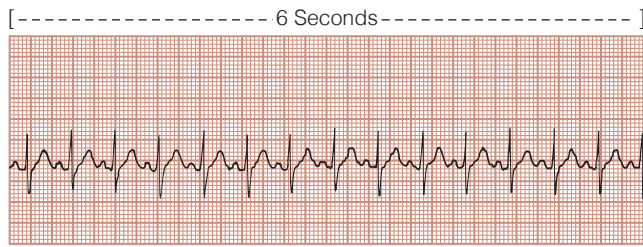


Figure 4-17 Sinus tachycardia.

Evaluation

In summary, for ST the ECG interpretation will be as follows:

1. Heart rhythm—regular
2. Heart rate—greater than 100 beats per minute
3. P waves—uniform and upright
4. P to QRS ratio—one to one
5. PR interval—0.12 to 0.20 second
6. QRS complex—narrow, less than 0.12 second

Etiology

ST is usually the result of a normal physiologic response to physical exercise, fever, anxiety, pain, hypoxia, congestive heart failure (CHF), acute MI, infection, sympathetic stimulation, anemia, or other stressors that may increase the body's requirement for increased oxygen. Certain medications such as epinephrine, atropine, and dopamine may cause sinus tachycardia. Caffeine, nicotine (a catecholamine), and cocaine may also cause ST.

COLLABORATIVE CARE

Treatment of sinus tachycardia is directed toward identifying and correcting the underlying cause. Interventions may include decreasing the fever, anxiety, or pain; treating the infection, CHF, or MI; or removing the offending medication or substance. According to the Neumar et al., (2015), no emergent drug treatment is needed for ST, and in patients where cardiac function is poor, the cardiac output may be dependent on a compensatory tachycardia. In this case, normalizing the heart rate could be detrimental to the patient. However, the nurse should be prepared to place an IV and perform a 12-lead ECG in case the patient becomes unstable.

Sick Sinus Syndrome

Sick sinus syndrome (SSS) is actually a combination of a few different dysrhythmias caused by an unhealthy sinus node. SSS is most commonly identified by SB with periods of sinus arrest without the normal escape mechanisms of atrial and junctional foci, which are also dysfunctional. Sinus arrest, also called sinus pause, occurs when the sinus node fails to pace for at least one complete cycle.

Etiology

SSS most often occurs in elderly patients with heart disease. The SB is usually a result of excessive parasympathetic activity, which innervates the SA node and depresses the pacing rate of the node. The atrial and junctional foci are depressed as well and fail to fire as an escape mechanism when sinus arrest occurs resulting in periods of asystole. Depending on the degree of SB and the sinus arrest episodes, the patient may experience hemodynamic compromise.

COLLABORATIVE CARE

If hemodynamic compromise is present, IV atropine 0.5 mg may be given. If the episodes of sinus arrest are frequent and lasting longer than a few seconds, insertion of a permanent pacemaker may be required.

Atrial Dysrhythmias

Atrial dysrhythmias refer to those dysrhythmias or complexes that originate in the atria but outside the SA node (♦ Figure 4-18). The P wave configuration will be upright but shaped differently from the P waves originating in the SA node, as the electrical conduction will follow a different pathway to the AV node. The common dysrhythmias in this category include premature atrial complexes, atrial tachycardia, atrial flutter, and atrial fibrillation. The term **supraventricular tachycardia (SVT)** is often used interchangeably with atrial tachycardia (AT); however, it is actually a general term to describe tachydysrhythmias that originate above the ventricles, either of atrial or junctional origin, or of uncertain origin but with a rapid ventricular rate and narrow QRS complexes.

Premature Atrial Complexes

Premature atrial complexes (PAC) are early beats that most often originate from an irritable focus in the atria. PACs are single early beats within an underlying rhythm rather than an abnormal rhythm in and of itself. The rhythm is regular except for the premature beats occurring earlier than would

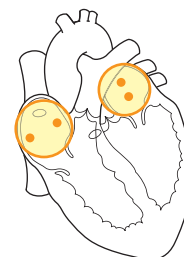


Figure 4-18 Atrial dysrhythmia.

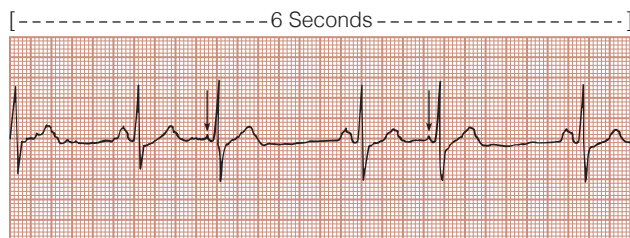


Figure 4-19 Premature atrial complex (PAC).

(From Jacobs, S. H. [1976]. *Intensive coronary care multimedia learning system: ECG exercises*. Van Nuys, CA: Sutherland Learning; Dubin, D. [2000]. *Rapid interpretation of EKG's* [6th ed.]. Tampa, FL: Cover; Kinney, M. R., & Packa, D. R. *Andreoli's comprehensive cardiac care* [8th ed.]. Boston, MA: Mosby.)

otherwise be expected. There is a P wave before each QRS; however, as shown in ♦ Figure 4-19, the P wave with the early beat may have a different configuration because it is not initiated in the SA node. The P wave may also be hidden in the preceding T wave; therefore, if no P wave is identified, the T wave should be examined for changes in size and shape compared with the other T waves. The PR interval of the PAC may be normal or prolonged. QRS complexes are usually narrow and similar in both the PAC beats and normal beats as they are all conducted through the AV node; however, occasionally the PAC may have a wide (aberrant) QRS complex or no QRS complex. The wide QRS complex may occur if the PAC is conducted abnormally down one of the bundle branches first and down the second bundle branch afterward. This would be called an aberrantly conducted PAC, and the QRS would look wider than the QRSs from the normal beats. Occasionally a QRS complex does not follow the premature P wave because it occurred so early that the AV junction was still in the refractory period and was unable to conduct the impulse to the ventricles. This is referred to as a nonconducted PAC and will appear as a P wave on the monitor that is not followed by a QRS complex. A pause usually occurs following the PAC and represents the time delay during which the SA node resets its rhythm for the next beat. There are two types of pauses following premature complexes: compensatory, meaning complete; or noncompensatory, meaning incomplete. Noncompensatory pauses usually follow a premature atrial complex, and compensatory pauses usually follow a premature ventricular complex (PVC). When a PAC is wide due to aberrant conduction, examining the pause may help identify the origin of the premature beat. To distinguish between the two types of pauses, the distance between three normal beats is measured, and that measurement is compared with the distance between three beats that includes the premature complex. If the premature beat has a noncompensatory pause, then the measurement of the three complexes that includes the premature complex will be less than the measurement containing the three normal beats. If the premature beat has a compensatory pause, the two measurements will be the same.

Evaluation

In summary, for PACs the ECG interpretation will be as follows:

1. Heart rhythm—regular except for the premature beats
2. Heart rate—usually within normal range
3. P waves—the P waves of the early beat may be upright, flattened, notched, or lost in the preceding T wave
4. P to QRS ratio—one to one
5. PR interval—0.12 to 0.20 second or prolonged in the PAC
6. QRS complex—narrow, less than 0.12 second

Etiology

PACs can be caused by emotional stress, anxiety, fatigue, infection, or lack of sleep, or from substances such as nicotine, caffeine, alcohol, and stimulants. PACs may also be caused by medications, CHF, electrolyte imbalances, and myocardial ischemia. Patients may have no symptoms, or they may complain of palpitations, feelings of skipped beats, or an unpleasant awareness of their heart beating. PACs do not usually cause hemodynamic compromise.

COLLABORATIVE CARE

Usually no treatment for premature atrial complexes is necessary other than treating the cause, such as stress reduction, rest, avoidance of substances known to cause atrial irritability, correcting electrolyte imbalances, or treating CHF.

Reflect On

You are working in the health clinic at the local community college. It is final exam week and a student comes in complaining of “feeling skipped beats in my heart.” An ECG is done on the student that confirms frequent PACs. What questions might you ask the student to assess the cause of the PACs? What teaching might you do with this student to help prevent future episodes of PACs?

Supraventricular Tachycardia

Supraventricular Tachycardia (SVT) is a rapid dysrhythmia that originates above the ventricles in either the atria or the AV junction. When the origin of the rhythm is clearly in the atria, it may be referred to as atrial tachycardia (AT). It may also be referred to as paroxysmal atrial tachycardia (PAT) or paroxysmal supraventricular tachycardia (PSVT) when its abrupt onset or cessation is observed via cardiac monitoring. The rhythm in SVT is usually very

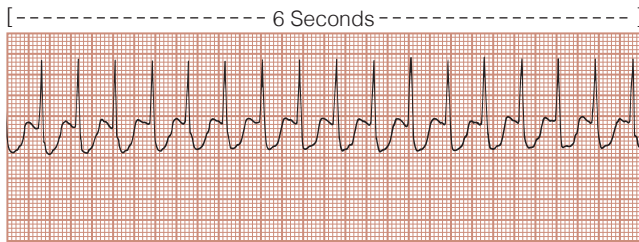


Figure 4-20 Supraventricular tachycardia (SVT).

regular. SVT has an atrial and ventricular rate of more than 120 beats per minute, typically 150 to 250 beats per minute. The P waves are hidden in the preceding T waves because of the rapid rate; therefore, the PR interval is usually not measurable. The QRS interval is usually narrow, less than 0.12 second. ♦ Figure 4-20 shows an example of SVT; it has a rate of 170 beats per minute.

Evaluation

In general, for supraventricular tachycardia the ECG interpretation will be as follows:

1. Heart rhythm—regular
2. Heart rate—150 to 250 beats per minute
3. P waves—may differ in shape from sinus P waves; may be hidden in preceding T wave
4. P to QRS ratio—one to one
5. PR interval—may be difficult to measure
6. QRS complex—narrow, less than 0.12 second

Etiology

SVT is often initiated by PACs, so the factors that cause PACs can also cause AT. Runs of SVT are common in young, healthy people with no evidence of cardiac disease, as well as in people with hypertension or atherosclerosis.

COLLABORATIVE CARE

The intervention for a supraventricular tachycardia depends on the patient's reaction to the dysrhythmia. Stable patients can be treated with oxygen, IV access, cardiac monitoring, and vagal maneuvers. Vagal maneuvers may successfully terminate SVT in about 20% to 25% of the patients (Neumar et al., 2015). These maneuvers include gagging, bearing down, and carotid sinus massage. Carotid massage should be performed only by the physician.

If the vagal maneuvers are unsuccessful or give only transient relief from SVT, adenosine is the first-line drug of choice (See "Commonly Used Medications"). If the patient becomes unstable, immediate synchronized cardioversion should take place. This is discussed later in the chapter.

Essential for Patient-Centered Care

Hypoxemia is a common cause of tachycardia, so initial evaluation of any patient with tachycardia should evaluate work of breathing and pulse oximetry (Neumar et al., 2015).

Reflect On

You are working in the emergency department (ED) and are assigned to the Cardiac Room. You receive a 38-year-old female, Mrs. Owens, who is complaining of feeling her heart pounding fast. She states she feels like she is going to pass out. You quickly hook her up to the cardiac monitor and get her vital signs. The monitor shows SVT at a rate of 210, her BP is 90/50. What are your top priorities in caring for Mrs. Owens? The doctor tells you to get out the crash cart and get the adenosine ready. Considering its short half-life, where should you start the IV on Mrs. Owens and how will you prepare the adenosine? After two boluses of adenosine, her rhythm converts to NSR with a rate of 86. If her rhythm had not converted with the adenosine, what other medications or procedures might have been attempted?

Atrial Flutter

In atrial flutter, the atrial rhythm is usually regular and the ventricular rhythm can be regular or irregular depending on the AV conduction. Atrial flutter is usually initiated by an irritable focus in the atria, which causes rapid depolarization at an atrial rate of about 250 to 350 beats per minute. The ventricular rate will be slower due to a protective mechanism in the AV node, which does not allow all of the atrial impulses to be conducted to the ventricles. There are usually no identifiable P waves; instead there are "flutter" waves that have a saw-toothed, or picket fence top, appearance. The rhythm is often described in terms of the number of flutter waves for each QRS complex, for example, a 3 to 1 flutter would refer to a pattern with three flutter waves for every one QRS complex. The PR interval is usually hard to measure. The QRS duration is usually narrow. In ♦ Figure 4-21, the ventricular rate is about 90; however, the atrial rate is approximately 250. There are two to three flutter waves for each QRS complex.

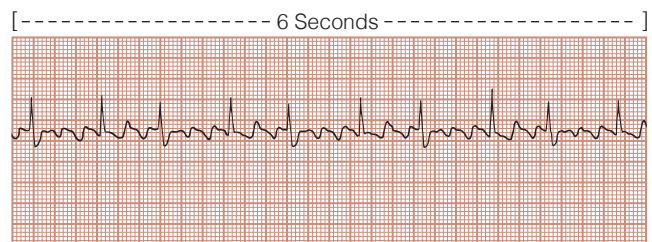


Figure 4-21 Atrial flutter.

Commonly Used Medications

Adenosine (Brand Names: Adenocard, Adenoscan)

Introduction

Adenosine is recommended in the treatment of (a) stable, narrow-complex regular tachycardias, (b) unstable narrow-complex regular tachycardias, and (c) stable, regular, monomorphic, wide complex tachycardia as a therapeutic and diagnostic maneuver. Adenosine should not be used for *irregular* wide-complex tachycardias because it may cause degeneration of the rhythm to ventricular fibrillation (Neumar et al., 2015).

Desired Effect: Adenosine is an antiarrhythmic drug. When administered intravenously, adenosine causes a transitory block at the AV node. This blockade results in a short run of asystole when the patient has no heart rhythm. An effect from adenosine should be seen within 1 to 2 minutes.

Nursing Responsibilities:

- Administration of a therapeutic dose. The dosage of adenosine is 6 mg rapid IV push, followed by a second bolus of 12 mg within 2 minutes if the dysrhythmia does not convert back to normal sinus rhythm. A third bolus of 12 mg may follow in another 2 minutes if the SVT continues.

- Adenosine has a very short half-life; therefore, it must be given rapidly over 1 to 3 seconds through an IV in the antecubital vein followed by a 20 mL saline flush and elevation of the arm; a lower dose may be used if it is given through a central line.
- HR, BP, and cardiac rhythm pattern should be monitored constantly while administering adenosine. There may be a significant conversion pause if the medication successfully cardioverts the rhythm. The nurse should be prepared for a code situation by having a code cart, oxygen, suction, and defibrillator readily available.

Side and/or Toxic Effects: Many individuals experience facial flushing, diaphoresis, lightheadedness, chest pain, and a sense of doom after administration of adenosine. These symptoms usually last less than 1 minute. If the dysrhythmia returns or does not convert with adenosine, a longer-acting AV node blocking agent, such as a calcium channel blocker or beta blocker, is the second-line drug of choice (Neumar et al., 2015).

Commonly Used Medications

Calcium Channel Blocker

[Diltiazem (Brand Name: Cardizem) or Verapamil (Brand Name: Isoptin)]

Introduction

Diltiazem and verapamil are recommended in the treatment of (a) stable, narrow-complex tachycardias if rhythm remains uncontrolled or unconverted by adenosine or vagal maneuvers, or if supraventricular tachycardia (SVT) is recurrent; and (b) to control ventricular rate in patients with atrial fibrillation or atrial flutter (Neumar et al., 2015).

Desired Effect: Diltiazem and verapamil are both calcium channel blockers and work by slowing the conduction of electrical activity of the heart and increasing the refractory period of the AV node. The slowing of conduction through the AV node causes a decrease in the heart rate and may terminate the AT/SVT, which depends on the AV node for its depolarization circuit.

Nursing Responsibilities:

- Ensuring appropriate dosage: Diltiazem is given by slow IV push at a dose of 15 to 20 mg. A repeat bolus of 20

to 25 mg may be given after 15 minutes if needed. An IV infusion dose of 5 to 15 mg/hr may also be started and titrated to the desired heart rate. Verapamil is also given slow IV push at a dose of 2.5 to 5 mg. If there is no response to the verapamil, repeat doses of 5 to 10 mg may be given every 15 to 30 minutes to a total dose of 20 mg.

- Each single dose of diltiazem or verapamil is administered slow IV push over at least 2 minutes.
- Cardiac rhythm, HR, and BP should be monitored during administration.

Side and/or Toxic Effects: Diltiazem and verapamil may cause hypotension, bradycardia, AV heart block, flushing, burning or itching at the injection site, and CHF.

Commonly Used Medications

Beta Blockers

[Metoprolol (Brand Name: Lopressor), Atenolol (Brand Name: Tenormin), Esmolol (Brand Name: Brevibloc), Propranolol (Brand Name: Inderal), and Sotalol (Brand Name: Betapace)]

Introduction

These beta blockers are indicated in the treatment of (a) stable, narrow-complex tachycardias if rhythm remains uncontrolled or unconverted by adenosine or vagal maneuvers or if SVT is recurrent; (b) to control ventricular rate in patients with atrial fibrillation or atrial flutter; and (c) in certain forms of polymorphic ventricular tachycardia (VT).

Sotalol is a potassium channel blocker and a beta blocker that is indicated in the treatment of hemodynamically stable monomorphic VT (Neumar et al., 2015).

Desired Effect: Beta blocker is an abbreviated name for “beta-adrenergic blocking agent.” Beta blockers decrease the heart rate and blood pressure by reducing the effects of circulating catecholamines. Beta blockers also have cardio-protective effects for MI patients.

Nursing Responsibilities:

- Administration and dosage depend on the beta blocker being administered; however, beta blockers

are administered via slow IV push, and the dose is often checked with another nurse prior to administration.

- Continuous monitoring of HR, BP, and cardiac rhythm is mandatory during IV administration.
- Beta blockers should not be administered to patients with heart rate of less than 50 or those with second- or third-degree AV block.

Side and/or Toxic Effects: Side effects include bradycardia, hypotension, AV conduction delays, AV heart block, or heart failure.

Note: Synchronized cardioversion (see the discussion of selected technologies later in this chapter) may be needed if the above-mentioned medications do not successfully convert the patient back to sinus rhythm or if the patient is not tolerating the dysrhythmia due to hemodynamic compromise.

Evaluation

In general, for atrial flutter the ECG interpretation will be as follows:

1. Heart rhythm—atrial regular, ventricular may be regular or irregular
2. Heart rate—atrial rate 250 to 350 beats per minute; ventricular rate is usually slower
3. P waves—flutter waves that look like the top of a picket fence
4. P to QRS ratio—more flutter waves than QRSs
5. PR interval—not measurable
6. QRS complex—normal unless flutter waves are buried in the QRS, which would make the QRS appear wider

Etiology

Atrial flutter is usually seen in the presence of heart disease, CHF, cardiac valvular disease, and following cardiac surgery. The rhythm may be paroxysmal, suddenly converting back to NSR or to atrial fibrillation either spontaneously or with treatment. Expert consultation may be

needed because the dysrhythmia may be difficult to distinguish from other SVTs. Prolonged or recurrent atrial flutter will often require antithrombotic therapy with Coumadin (see further details under “Atrial Fibrillation”).

COLLABORATIVE CARE

The treatment for atrial flutter depends on the patient’s condition. The patient is usually asymptomatic; however, the more rapid the ventricular rate, the more likely the patient is to be symptomatic. The goal with rapid ventricular response to atrial flutter is to slow down the ventricular rate. To decrease the heart rate, medications such as calcium channel blockers or beta blockers (as described under “Supraventricular Tachycardia”) may be used. Antiarrhythmic medications such as amiodarone or synchronized cardioversion may be attempted to convert the rhythm back to normal sinus. If the patient has a permanent pacemaker or temporary epicardial pacing wires, rapid atrial pacing may be attempted to override the irritable focus and allow the sinus node to take over.

Essential for Patient-Centered Care

Establish IV access and administer sedation if the patient is conscious before cardioversion whenever possible.

Atrial Fibrillation

Atrial fibrillation (AF, Afib) occurs from the rapid firing of multiple irritable foci in the atria, resulting in no effective atrial contraction because there is no uniform wave of depolarization. The loss of an effective atrial contraction results in the loss of the “atrial kick” and a subsequent decrease in cardiac output. Atrial kick refers to the blood that is pushed out of the atria into the ventricles after they depolarize and contract together. The loss of the atrial kick can result in a 20% to 30% reduction in cardiac output. The rapid and erratic fibrillation of the atria also results in a risk for an embolic event due to the pooling and clotting of blood in the atria. The atrial rate is around 350 to 600 beats per minute. There are no consistently identifiable P waves; instead there are fibrillatory waves causing an erratic and wavy baseline. The ventricular rate is variable and has an irregular rhythm. In an untreated or uncontrolled AF, the ventricular rate may be between 100 and 160 beats per minute; this is referred to as a rapid ventricular response. When the ventricular rate is less than 100, the dysrhythmia is usually referred to as controlled atrial fibrillation. There is difficulty in measuring the PR interval. The QRS duration is usually narrow. In the example of atrial fibrillation in ♦ Figure 4-22, the ventricular rate is 70 and irregular.

Evaluation

In general, for AF the ECG interpretation will be as follows:

1. Heart rhythm—ventricular rhythm is usually consistently irregular
2. Heart rate—atrial rate is 350 to 600 beats per minute; the ventricular rate is variable
3. P waves—no consistently identifiable P wave, fibrillatory waves
4. P to QRS ratio—more fibrillatory waves than QRSs
5. PR interval—not measurable
6. QRS complex—usually normal but may appear wider if there is a conduction delay

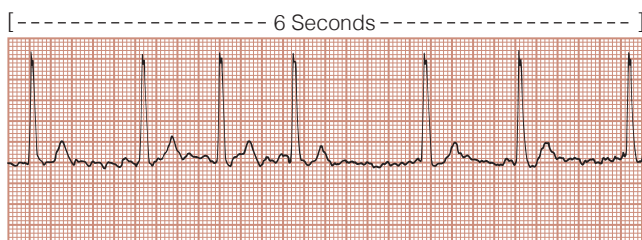


Figure 4-22 Atrial fibrillation.

Etiology

AF is a common dysrhythmia and is usually seen in patients with underlying heart disease, hypertension, valvular disease, CHF, MI, recent cardiac surgery, and electrolyte imbalances.

COLLABORATIVE CARE

The intervention for AF is composed of two areas: rate control and rhythm control. Rate control focuses on the use of diltiazem or beta blockers to keep the ventricular rate less than 100 beats per minute. Rhythm control focuses on conversion of AF back to NSR either by pharmacological means or synchronized cardioversion. These patients are frequently put on medications for anticoagulation, such as warfarin (brand name: Coumadin), due to the high risk of embolic events. Patients with prior stroke, transient ischemic attack, or a CHA2DS2-VASc score of 2 or greater require antithrombotic therapy (Moss & Cifu, 2015). Patients who have been in AF for longer than 48 hours are also at increased risk for an embolic event such as a stroke or thromboembolism and should have anticoagulation treatment before rhythm control is attempted. These patients may undergo emergency synchronized cardioversion only if they are hemodynamically compromised or the absence of an atrial thrombus is confirmed by a transesophageal echocardiogram (Neumar et al., 2015). AF that does not reconvert to a sinus rhythm may be treated with the MAZE procedure in the operating room or with radiofrequency catheter ablation (RCFA) in the electrophysiology lab (see the “Building Technology Skills” section of this chapter for a discussion of these two procedures).

Atrioventricular Dysrhythmias and Blocks

AV junctional dysrhythmias are those dysrhythmias that originate from the AV node or bundle of His (♦ Figure 4-23a). The AV junction may take over as the primary pacemaker

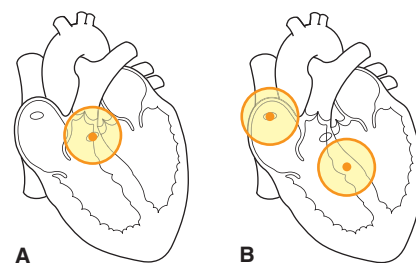


Figure 4-23 A, Atrioventricular (AV) junctional dysrhythmias. B, Atrioventricular (AV) Blocks

Gerontological Considerations

AF is the most commonly sustained dysrhythmia in older adults because the incidence of AF increases with age and heart disease. Approximately 20% of individuals who have a stroke have permanent atrial fibrillation and it is thought that this number is actually doubled due to undetected AF. Stroke prevention is usually accomplished through anticoagulants; however, the elderly are also at increased risk of bleeding from being on anticoagulants because of increased falls, functional impairments, poor nutrition, changing physiologic function and cognitive dysfunction. The CHA₂DS₂-VASc score calculates stroke risk in patients in atrial fibrillation. Points are assigned based on a patient's age; sex; history of heart failure; prior stroke, transient ischemic attack or thromboembolic event; vascular disease and diabetes (Ishola, Hite, Smith, & Hasan, 2013). Despite their increased risk of bleeding, the benefits of using an anticoagulant generally outweigh the risks in older adults with AF.

The most recent guidelines for the treatment of AF were published by the American Academy of Family Physicians and the American College of Physicians and include the following (January et al., 2014):

1. Rate control with chronic anticoagulation is recommended for most AF patients.
2. Anticoagulation is recommended for CHA₂DS₂-VASc score >2. If the atrial fibrillation is not associated with valvular disease, use of a direct thrombin or factor Xa inhibitor (dabigatran, rivaroxaban, or apixaban) may be recommended over Coumadin.
3. Beta blockers (atenolol, metoprolol) and calcium channel blockers (diltiazem, verapamil) are recommended for rate control as an outpatient; digoxin should be considered as second-line therapy for patients who do not respond to these medications. Digoxin works by slowing conduction through the AV node. It also increases the strength of myocardial contraction, resulting in a slower, stronger heartbeat with increased cardiac output.
4. For patients requesting cardioversion, either synchronized cardioversion or pharmacological conversion is appropriate.
5. To minimize the risk of embolic stroke in patients electing cardioversion, either transesophageal echocardiogram (TEE) or 3 weeks of anticoagulation prior to cardioversion is acceptable. Both strategies are followed by 3 to 4 weeks of anticoagulation.
6. Antiarrhythmic medications are generally not recommended as their risks are likely to outweigh the benefits.

of the heart if the SA node fails to discharge an impulse, or the impulse is blocked and does not reach the AV node, or if the SA node is pacing slower than the impulse generation of the AV node. When the AV node paces the heart, the impulse generated may travel backward to depolarize the atria and forward to depolarize the ventricles. Depending on the timing of the atrial and ventricular depolarization, the P wave may be inverted and appear before the QRS complex, it may be buried in the QRS complex, or it may be inverted and appear after the QRS complex. Some AV junctional dysrhythmias include premature junctional complexes (PJCs) and junctional escape rhythm.

With AV blocks, the focus shifts to conductivity problems of the heart. An AV block exists when the electrical conduction through the AV node or bundle of His is delayed or blocked (♦ Figure 4-23b). The blocks are differentiated by their level of severity or degree of block, becoming more acute with each additional degree: first-degree AV block; second-degree AV block, type I and type II; and third-degree AV block, or complete heart block.

Premature Junctional Complexes

PJCs are early beats that arise from the AV junction within an underlying rhythm. The atrial and ventricular rates are usually within normal limits but may be faster or slower than normal depending on the underlying rhythm. The P waves are usually inverted, as shown in ♦ Figure 4-24, and may occur before, during, or after the QRS complex. If the P wave occurs before the QRS complex, the PR interval will probably be shorter than normal (<0.12 second). The QRS duration is usually narrow (<0.12 second) because the

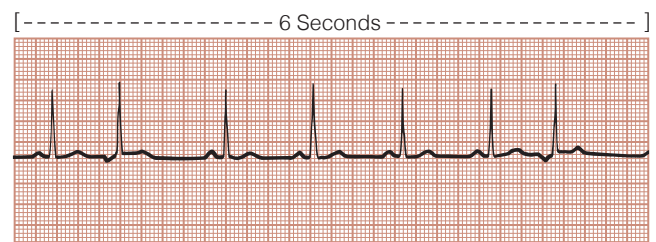
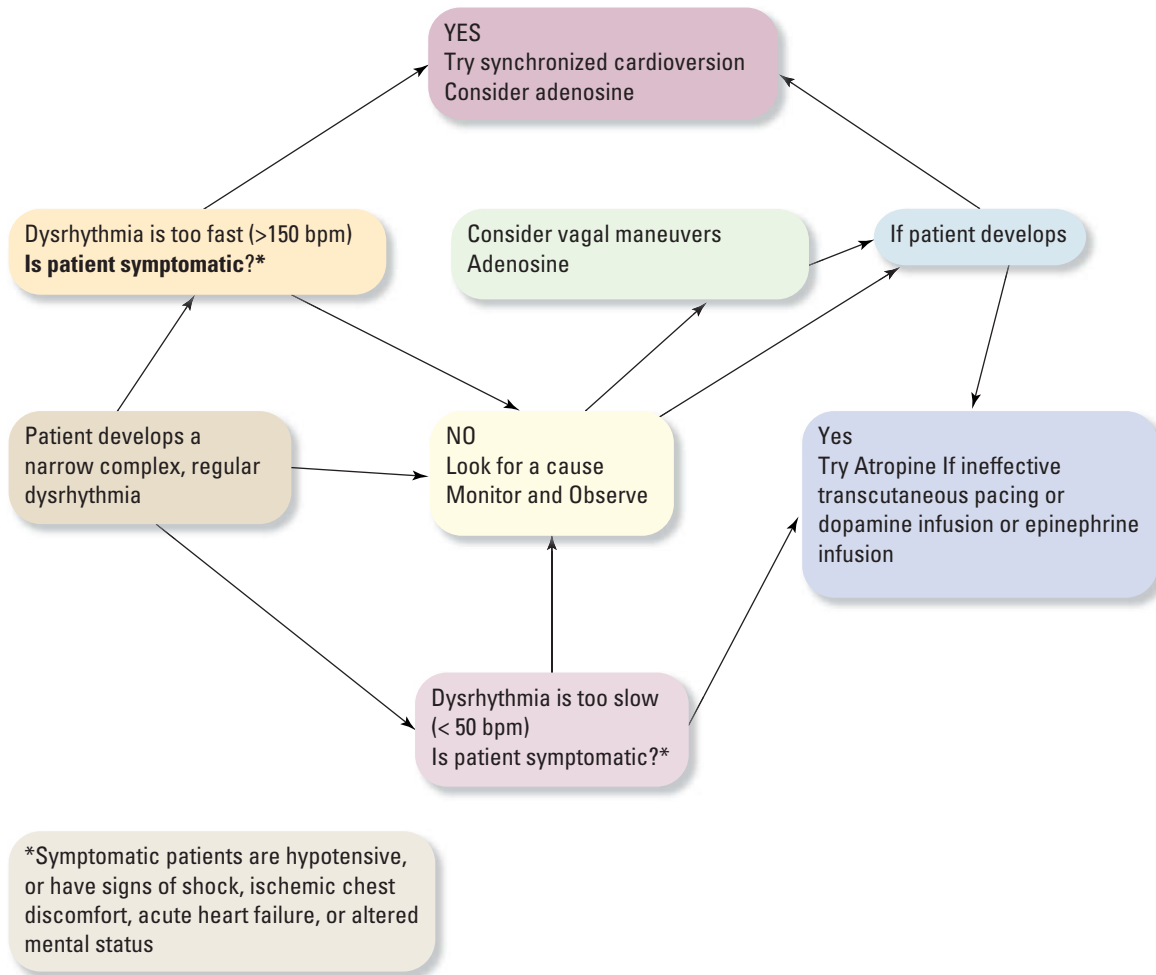


Figure 4-24 Premature junctional complex (PJC).



Visual Map 4-1 Narrow Complex Dysrhythmia

impulse travels through the ventricles along the normal conduction pathway.

Evaluation

In summary, for PJs the ECG interpretation will be as follows:

1. Heart rhythm—regular except for the premature beats
2. Heart rate—usually within normal range
3. P waves—the P waves of the early beat may occur before, during, or after the QRS and may be inverted
4. P to QRS ratio—one to one
5. PR interval—usually normal or shorter than normal, or may not be able to be measured if the P wave does not appear before the QRS
6. QRS complex—usually narrow, less than 0.12 second, unless there is a conduction delay

Etiology

PJCs may be caused by the same stimuli that cause PACs; however, they are less common than PACs or PVC.

COLLABORATIVE CARE

Premature junctional complexes usually do not cause symptoms; therefore, treatment for them is rarely necessary. If symptoms do occur, PJCs are treated in the same way as PACs.

Junctional Escape Rhythm

Junctional escape rhythm occurs because the SA node has failed to pace the heart or the impulse has failed to reach the AV node. The atrial and ventricular rate is 40 to 60 beats per minute because that is the rate of automaticity of the nodal cells at the AV junction. The rhythm is regular. The P wave is usually inverted and may occur before, during, or after the QRS complex. In ♦ Figure 4-25, the junctional escape rhythm has a rate of 50, and the P wave looks like an inverted notch and occurs immediately after the QRS complex. If the P wave occurs before the QRS, the PR interval will probably be shorter than normal. The QRS complex will be normal and narrow.

Safety Initiatives

Prevent Harm from High-Alert Medication: Warfarin

PURPOSE: To prevent harm from high-alert medications by implementing the changes in care recommended in the Institute for Healthcare Improvement (IHI) guide. High-alert (or high-hazard) medications are medications that are likely to cause significant harm to the patient, even when used as intended.

RATIONALE: Warfarin (Coumadin) has a very narrow therapeutic index, making appropriate dosing and monitoring critical. Patients and their families are usually involved in administration; therefore, they must understand how to take the medication, avoid specific medications, and recognize symptoms that indicate likely problems. The IHI recommends changes in three areas to decrease adverse events. These areas for change are standardization of dosing, ensuring adequate monitoring, and ensuring partnering with patients and their families.

HIGHLIGHTS OF RECOMMENDATIONS:

THE IHI RECOMMENDS: Changes designed to ensure standardization such as standardizing protocols and dosing of warfarin but also vitamin K dosing guidelines.

Changes designed to ensure adequate monitoring such as making lab results available on the unit within 2 hours and plotting the INR results and dose changes on a chart.

Changes designed to ensure better partnering with patients and families such as developing appropriate educational programs that include self-monitoring, how to avoid drug and food interactions, and carrying an accurate list of medications.

Source: Institute for Healthcare Improvement.

Evaluation

In summary, for junctional escape rhythm the ECG interpretation will be as follows:

1. Heart rhythm—regular
2. Heart rate—40 to 60 beats per minute
3. P waves—may occur before, during, or after the QRS and may be inverted
4. P to QRS ratio—one to one
5. PR interval—usually normal or shorter than normal, or may not be able to be measured if the P wave does not appear before the QRS
6. QRS complex—usually narrow, less than 0.12 second, unless there is a conduction delay

Etiology

Junctional escape rhythm may be seen in acute MI, valvular disease, SA node disease, post-cardiac surgery, and in patients taking certain medicines such as digitalis, beta blockers, and calcium channel blockers. The junctional rhythm may serve as a safety mechanism if the primary pacemaker fails to pace the heart.

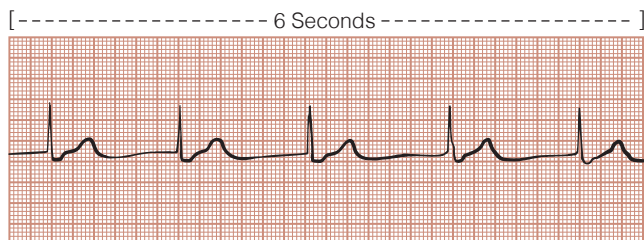


Figure 4-25 Junctional escape rhythm.

COLLABORATIVE CARE

The treatment for junctional escape rhythm depends on the patient's symptoms and the underlying cause of the dysrhythmia. If the dysrhythmia is due to a medication, such as digitalis toxicity, the medication should be held. If the patient is hemodynamically unstable because of the slow heart rate, the same intervention as for sinus bradycardia may be used.

Essential for Patient Safety

The therapeutic goal for any bradycardias and tachycardias is to quickly identify and treat patients who are hemodynamically symptomatic.

First-Degree Atrioventricular Block

First-degree AV block is really a conduction delay rather than a block. Every impulse is conducted to the ventricles but in a delayed manner. The rate is usually within normal limits but depends on the underlying rhythm; for example, the underlying rhythm may be sinus bradycardia with first-degree AV block. The rhythm is regular. The P waves are normal configuration with one P wave preceding each QRS complex, and the QRS duration is usually narrow. The distinguishing feature of first-degree AV block is that the PR interval is greater than 0.20 second due to the conduction delay. All PR intervals are consistent in that they all measure roughly the same length. ♦ Figure 4-26 gives an example of sinus rhythm with a first-degree AV block in which the PR interval is approximately six small boxes, or 0.24 second.

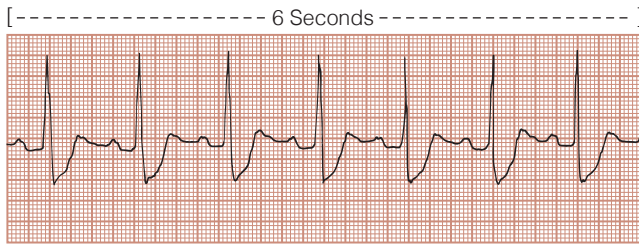


Figure 4-26 Sinus rhythm with first-degree AV block.

Evaluation

In summary, for first-degree AV block the ECG interpretation will be as follows:

1. Heart rhythm—regular
2. Heart rate—usually normal, but depends on the underlying rhythm
3. P waves—normal in size and shape
4. P to QRS ratio—one to one
5. PR interval—consistently prolonged from cycle to cycle, greater than 0.20 second
6. QRS complex—usually narrow, less than 0.12 second, unless there is a conduction delay

Etiology

First-degree AV block may be a normal finding for some individuals, or it may be due to a diseased heart. Some causes of first-degree block include AV nodal ischemia, which may occur from an inferior MI; digitalis toxicity or toxicity to other medications such as beta blockers, calcium channel blockers, and narcotics; excessive vagal stimulation; post-cardiac surgery due to edema in the AV junction; or calcification of the AV node.

COLLABORATIVE CARE

Generally, first-degree atrioventricular block does not cause hemodynamic compromise, so the patient remains without symptoms. If the dysrhythmia is due to a medication, the drug should be stopped or the dosage decreased. If the dysrhythmia is due to an acute MI, the patient should be monitored closely for progression to a more severe AV block.

Second-Degree Atrioventricular Blocks

In a second-degree block, not all of the impulses from the atria are conducted to the ventricles. The SA node is firing at regular intervals, but some of the impulses are blocked at the AV junction. There are two types of second-degree AV blocks: Mobitz Type I, also called Wenckebach, and Mobitz Type II, which is more serious than type I. Just as the PR interval was the distinguishing factor in first-degree block, the PR interval is the distinguishing factor differentiating type I from type II second-degree block.

Second-Degree Atrioventricular Block, Mobitz Type I (Wenckebach)

The rhythm appears irregular due to the nonconducted P waves, or P waves that are not followed by a QRS complex. The atrial rate is greater than the ventricular rate due to the nonconducted P waves. The P waves are normal, but some P waves are not followed by a QRS complex. The QRS duration is narrow. The PR interval lengthens with each consecutive cardiac cycle until a P wave is not followed by a QRS complex. The pattern of progressively longer PR intervals and a dropped QRS repeats itself. The impulses from the SA node take an increasingly longer time to conduct through the AV node until one is unable to conduct to the ventricles. The conduction pause then allows the AV node time to recover so that the next impulse starts the cycle over again with an initial shorter conduction time. ♦ Figure 4-27 shows an example of second degree AV block, type I. After the first QRS in the strip, there is a P wave that is not conducted into the ventricles and, therefore, is not followed by a QRS. The PR interval of the second cardiac cycle is 0.24 second, the PR interval of the third cardiac cycle is 0.28 second, the PR interval of the fourth cardiac cycle is 0.36 second, and then a nonconducted P wave is seen again.

Evaluation

In summary, for second-degree AV block, type I, the ECG interpretation will be as follows:

1. Heart rhythm—regularly irregular due to repeating patterns with a dropped QRS complex
2. Heart rate—the atrial rate is greater than the ventricular rate due to the dropped QRS complex
3. P waves—normal in size and shape
4. P to QRS ratio—some P waves are not followed by a QRS complex
5. PR interval—lengthens with each cycle until a P wave is not followed by a QRS complex
6. QRS complex—usually narrow, less than 0.12 second

Etiology

The conduction delay through the AV node is thought to be caused by AV nodal ischemia from occlusion to the right

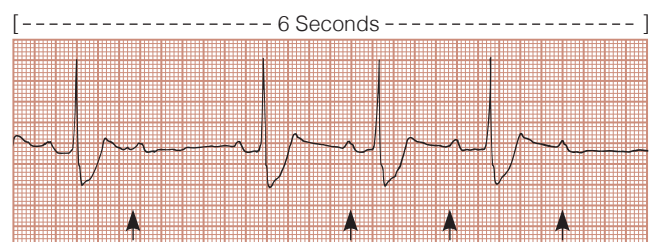


Figure 4-27 Second-degree AV block, type I (Wenckebach).

coronary artery, from acute inferior wall MI, or from increased parasympathetic stimulation. It is usually transient and rarely progresses to second-degree AV block, type II, or third-degree AV block.

COLLABORATIVE CARE

This dysrhythmia is usually asymptomatic as the cardiac output is not significantly affected. If the patient does become symptomatic and the rhythm is slow, treatment will follow that for sinus bradycardia, using atropine as the first-line drug of choice. Temporary pacing should be considered depending on the patient's symptoms.

Second-Degree Atrioventricular Block, Mobitz Type II

Second-degree AV block, type II, is less common than type I. It is more serious than type I because it may suddenly progress to a third-degree AV block and because it is usually bradycardic in nature. The block occurs below the bundle of His in the bundle branches of the conduction system. The atrial and ventricular rhythms are usually regular. The atrial rate is greater than the ventricular rate because of the nonconducted P waves. The ventricular rate is often slow. If the nonconducted P waves are blocked in a random pattern, the ventricular rate will be irregular. When the P waves are blocked at regular intervals, such as in a 2 to 1 block (two P waves for every one QRS), the ventricular rate will be regular. The P waves are normal in size and shape. Unlike type I block, the PR interval is constant for the conducted beats. The QRS duration may be normal, or it may be wider than normal (>0.12 seconds) because of a delay or blockage in the conduction below the bundle of His. ♦ Figure 4-28 shows an example of a second-degree AV block, type II. Notice that there is a nonconducted P wave and a conducted P wave before each QRS complex.

Evaluation

In summary, for second-degree AV block, type II, the ECG interpretation will be as follows:

1. Heart rhythm—usually regular, but may be irregular for the ventricular rhythm

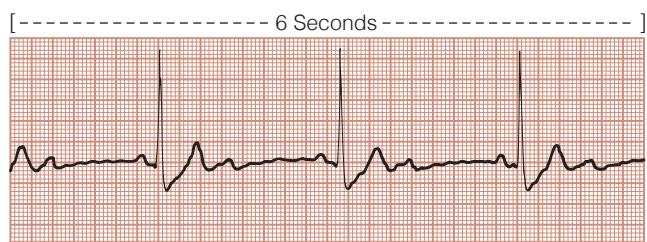


Figure 4-28 Second-degree AV block, type II.

2. Heart rate—the atrial rate is greater than the ventricular rate due to the dropped QRS complexes
3. P waves—normal in size and shape
4. P to QRS ratio—some P waves are not followed by a QRS complex
5. PR interval—normal or slightly prolonged and constant for the conducted P waves
6. QRS complex—may be narrow and normal, or widened due to coexisting bundle branch block

Etiology

Second-degree AV block, type II, may occur in patients with left coronary artery disease, such as an anterior wall MI, because the bundle branches receive their blood supply from the left coronary artery. Type II block may also result from other forms of heart disease. The patient may experience hemodynamic compromise if there are frequently dropped P waves, resulting in a slow ventricular rate and inadequate cardiac output.

COLLABORATIVE CARE

The treatment for unstable patients with second-degree AV block, type II, is immediate temporary pacing. Atropine 0.5 mg IV may be considered while waiting for the pacer, but its administration should not delay temporary pacing. If temporary pacing is ineffective, preparations should be made for a permanent pacemaker, and expert consultation should be obtained. (See the section on pacemakers under “Building Technology Skills.”)

Reflect On

You are observing in the ED as a student nurse and the call comes in from EMS that they are bringing in a witnessed arrest. The patient is an 87-year-old male who passed out at grocery store. When it was determined that the patient was nonresponsive, the grocery store workers quickly attached an AED, which analyzed the rhythm and advised defibrillation. The patient has been defibrillated twice, and CPR is in progress. They will be in your ED momentarily. The charge nurse asks if you will do the compression on the patient. Have you ever done CPR on someone before? How will you respond to the charge nurse? What can you do to stay ready and prepared to help in a code situation?

Third-Degree Atrioventricular Block (Complete Heart Block)

Second-degree AV blocks are incomplete blocks because some of the impulses are conducted through the AV junction to the ventricles and some of the impulses are

blocked. Third-degree AV block is referred to as complete heart block because none of the impulses from the atria are conducted through the AV junction to the ventricles. The atria and ventricles beat independently of each other and it may also be referred to as “AV dissociation.” Both the atrial and ventricular rhythms are usually regular, but they have no relationship to each other. The atrial rate is greater than the ventricular rate. The ventricular rate is determined by the site of the ventricular pacemaker. The atria may be paced by the SA node or by an ectopic focus in the atria; the ventricles may be paced by the AV node, the bundle of His, or the Purkinje fibers. The P waves are normal in shape and size and are equal distances from each other. The QRS complexes may be narrow or wide depending on the location of the escape pacemaker. Narrow QRS complexes are indicative of an escape pacemaker at the AV junction and will usually have a rate of 40 to 60 beats per minute, as in the junctional escape rhythm. Wide QRS complexes are indicative of an escape pacemaker in the ventricles and will usually have a rate of less than 40 beats per minute. The QRS complexes are equal distances apart, but there is no relationship between the P waves and the QRS complexes. There is no consistent PR interval, because the atria and ventricles are beating independently of each other. Thus, all the PR intervals are different. Complete heart block may deteriorate into a fatal dysrhythmia such as ventricular asystole, ventricular tachycardia, or ventricular fibrillation. ♦ Figure 4-29 shows an example of complete heart block. Notice how the P waves march out independently of the QRS complexes. The atrial rate is 60, whereas the ventricular rate is 30.

Evaluation

In summary, for third-degree AV block the ECG interpretation will be as follows:

1. Heart rhythm—the atrial and ventricular rhythms are regular, but there is no relationship between the two rhythms
2. Heart rate—the atrial rate is greater than the ventricular rate
3. P waves—normal in size and shape
4. P to QRS ratio—there are usually more P waves than QRS complexes
5. PR interval—none, as the atria and ventricles beat independently of each other
6. QRS complex—narrow or wide depending on the location of the escape pacemaker; narrow indicates a junctional pacemaker, whereas wide indicates a ventricular pacemaker

Etiology

Complete heart block may be caused by medications such as digitalis toxicity and by degenerative heart disease. It may also be caused by acute MI and myocarditis.

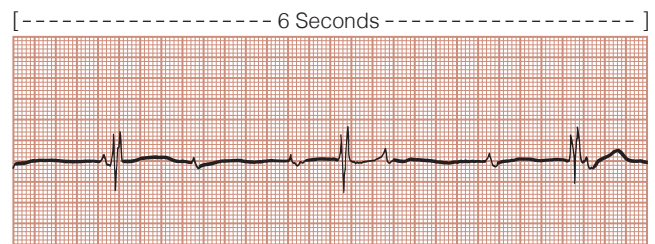


Figure 4-29 Third-degree AV block (complete heart block).

Commonly Used Medications

Epinephrine (Brand Name: Adrenalin)

Introduction

Epinephrine infusion may be used to treat symptomatic bradycardia that is unresponsive to atropine (Neumar et al., 2015).

Desired Effect: Epinephrine is a catecholamine and as such causes vasoconstriction. Epinephrine is also a potent cardiac stimulant, strengthening myocardial contraction and increasing heart rate.

Nursing Responsibilities:

- An infusion may need to be prepared according to hospital policy (usually 1 mg of 1:1,000 concentration in 500 mL NS) and hung on an infusion pump at a rate of 1 to 5 mL/min to equal 2 to 10 mcg/min (Wilson, Shannon, & Shields, 2016); most drug books provide drug calculation charts to help calculate the drug dosage.

- Epinephrine is rapidly destroyed in alkaline solutions, so alkaline medications such as sodium bicarbonate should be administered through a separate IV line.
- The patient's response to the medication should be assessed by monitoring the ECG, BP, and HR every 5 minutes.
- The epinephrine label must be checked because the medication comes in a 1:1000 solution and a 1:10,000 solution.

Side and/or Toxic Effects: Many side effects are transitory and can include anxiety, dizziness, dyspnea, and palpitations. Overdose can include bradycardia, tachycardia, cerebrovascular hemorrhage, fibrillation, pulmonary edema, renal failure, and death (Wilson et al., 2016).

COLLABORATIVE CARE

The nurse should prepare for emergent temporary pacing in patients with symptomatic third-degree AV block. Atropine 0.5 mg IV may be given, while awaiting the pacemaker, to a total of 3 mg. Epinephrine or dopamine infusion may also be considered while awaiting the pacemaker or if the pacing is ineffective (Neumar et al., 2015). If temporary pacing is ineffective, the nurse should prepare for permanent pacemaker insertion. Third-degree AV block with a narrow QRS is usually better tolerated than third-degree block with a wide QRS.

Bundle Branch Block

Bundle branch block (BBB) is a type of intraventricular conduction block. After the AV node, the electrical impulse enters the bundle of His, which bifurcates into the right and left bundle branches. BBB is a conduction delay or block in one of the two main bundle branches. Instead of both ventricles being depolarized simultaneously, the unblocked bundle branch conducts normally, then the wave of depolarization is spread from the first ventricle, cell by cell, to the second ventricle. The electrical impulses spread much slower through the cardiac cells than through the specialized bundle branch cells. This slowed depolarization prolongs the QRS duration to 0.12 second or longer. Its appearance varies depending on the affected bundle branch (♦ Figure 4-30). A right bundle branch block (RBBB) is more common than a left bundle branch block (LBBB) and a BBB may be either incomplete or complete.

Evaluation

In general, for BBB, the ECG interpretation will be as follows:

1. Heart rhythm—depends on the underlying rhythm, usually sinus in origin
2. Heart rate—depends on the underlying rhythm

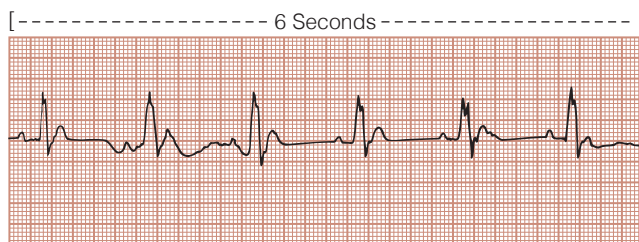


Figure 4-30 Normal sinus rhythm with a bundle branch block.

3. P waves—not affected, usually normal
4. P to QRS ratio—not affected, usually one to one
5. PR interval—not affected
6. QRS complex—usually 0.12 second or wider; usually appears notched with an R and R prime wave or as a widened QRS with two peaks. The second notch, or peak, represents the delay in ventricular depolarization

Etiology

BBB may be a temporary or permanent conduction disorder. A temporary BBB may occur after acute conditions such as MI or heart failure. A permanent BBB may occur with congenital heart disease, rheumatic heart disease, cardiomyopathy, severe aortic stenosis, or other cardiac diseases that may cause scarring of the conduction system. Typically, the patient with a BBB is asymptomatic. New onset of a left BBB may be indicative of acute myocardial infarction.

COLLABORATIVE CARE

BBBs do not usually require intervention; however, a new block should be reported to the healthcare provider, and the patient should be assessed for signs of hemodynamic compromise. In the event of hemodynamic compromise, treatment may include a ventricular pacemaker.

Commonly Used Medications

Dopamine (Brand Name: Intropin)

Introduction

Dopamine infusion may be used to treat symptomatic bradycardia that is unresponsive to atropine (Neumar et al., 2015).

Desired Effect: Dopamine is a catecholamine agent, a cardiac stimulant, and a vasopressor. Dopamine causes an increase in heart rate and blood pressure in similar fashion as epinephrine, and it also increases contractility of the heart and cardiac output. An effect should be seen in less than 5 minutes.

Nursing Responsibilities:

- A dopamine infusion should be started at 2 to 10 mcg/kg/min and titrated to the patient's response. Drug calculation charts for dopamine are available in most drug books.

- An infusion may need to be prepared according to hospital policy (a 400-mg or 800-mg ampule must be diluted in 250 to 500 mL of IV solution).
- The slowest possible rate should be used to maintain adequate heart rate and blood pressure.
- Blood pressure and heart rate should be checked every 5 minutes to avoid hypertension.
- A central line is preferred for infusion as infiltration into the tissues can cause necrosis and sloughing of the tissue.

Side and/or Toxic Effects: Side effects may include bradycardia, dyspnea, ectopic beats, hypertension, hypotension, palpitations, tachycardia, and widened QRS complex (Wilson et al., 2016).

Ventricular Dysrhythmias

Ventricular dysrhythmias are those dysrhythmias that originate in the ventricles (♦ Figure 4-31). The ventricles may take over as the primary pacemaker of the heart if:

- the SA node fails to discharge an impulse
- the impulse is blocked and does not reach the ventricles
- the SA node and AV node are pacing slower than the impulse generation of the ventricles
- an irritable site in one of the ventricles produces a rapid rhythm

When one of the ventricles paces the heart, the impulse generated must first spread a wave of depolarization through one ventricle then depolarize the other ventricle. This creates a QRS complex that is wide (>0.12 second) and abnormally shaped. The impulse is usually blocked in the AV junction and does not depolarize the atria. Tachycardic rhythms that have narrow QRS complexes (<0.12 second) usually originate above the ventricles, whereas tachycardic rhythms that have wide QRS complexes (>0.12 second) usually originate in the ventricles. Some common ventricular dysrhythmias include PVC, ventricular escape rhythm, ventricular tachycardia (VT), ventricular fibrillation (VF), ventricular asystole, and PEA.

Premature Ventricular Complexes

A PVC is an early beat within an underlying rhythm that are initiated by an irritable focus in one of the ventricles. There is no PR interval with the PVC because the beat originated in the ventricles and does not have a P wave. The QRS duration is wide (>0.12 second) and bizarre. The T wave is usually deflected in the opposite direction of the QRS complex. A pause usually occurs following the PVC. As discussed in the section on PACs, there are two types of pauses following premature complexes: compensatory (meaning complete) and noncompensatory, meaning incomplete. Noncompensatory pauses usually follow a PAC, and compensatory pauses usually follow a PVC. When a PAC is wide due to aberrant conduction, examining the pause may help identify the origin of the premature beat. ♦ Figure 4-32a

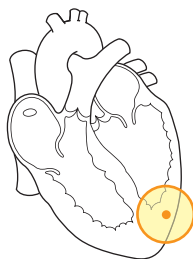


Figure 4-31 Ventricular dysrhythmias.

shows an example of a PVC with an irregular underlying rhythm of AF. Multiple PVC may be described as unifocal or multifocal. Unifocal PVC mean they look the same and originate from the same irritable focus in the ventricles. Multifocal PVC mean they look different and may originate from different irritable foci in the ventricles. When the rate is fast, PVC may occur in pairs, called couplets. A run of three or more PVC at a fast rate is known as VT. PVC may also appear in repetitive patterns known as bigeminy or trigeminy. Bigeminy refers to a repetitive pattern in which every other beat is a PVC. Trigeminy refers to a repetitive pattern in which every third beat is a PVC. ♦ Figure 4-32b shows an example of bigeminy.

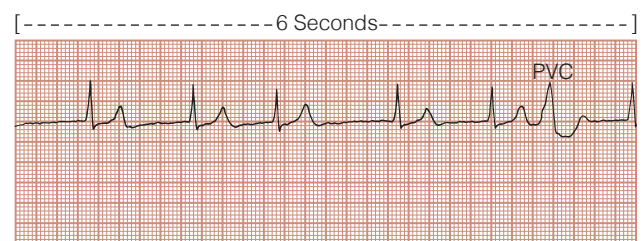
Evaluation

In general, for PVC the ECG interpretation will be as follows:

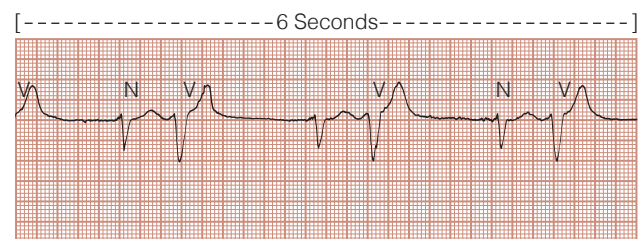
- Heart rhythm—depends on the underlying rhythm, essentially regular with premature beats
- Heart rate—depends on the underlying rhythm
- P waves—usually absent
- P to QRS ratio—the PVC does not have a P wave preceding it
- PR interval—none
- QRS complex—greater than 0.12 second, wide and bizarre, with T waves in the opposite direction as the QRS complex

Etiology

PVC can occur in healthy patients, and the incidence of PVC increases with age. PVC may produce no symptoms or may cause a feeling of skipped beats, racing heart, or



A



B

Figure 4-32 A, A premature ventricular complex (PVC) with an underlying rhythm of atrial fibrillation. B, Ventricular bigeminy.

chest and neck discomfort. PVC may or may not produce a palpable pulse. PVC may be caused by hypoxia; myocardial ischemia or infarction; CHF; stress; medications such as digitalis, epinephrine, and dopamine; or electrolyte imbalances such as hypokalemia or hypomagnesemia. PVC may also be caused by ingestion of caffeine, alcohol, and tobacco, and by an increase in catecholamines. PVC may also be caused by successful fibrinolytic therapy and by insertion of a PA line.

COLLABORATIVE CARE

Occasional PVC in otherwise healthy patients do not need intervention, except to eliminate contributing factors such as medications or caffeine. Treatment of post-MI patients with frequent PVC includes correction of hypoxia, pain relief, and correction of electrolyte imbalances and heart failure. Occasionally an antiarrhythmic medication, such as amiodarone or lidocaine, may be needed. However, due to the potential adverse effects of many antiarrhythmic medications, the healthcare practitioner needs to weigh the costs and benefits of each particular patient in treating PVC with medications.

Ventricular Escape Rhythm (Idioventricular Rhythm)

Ventricular escape rhythm is also called idioventricular rhythm because it occurs in the ventricles without any association with the atria. This rhythm may exist when both the SA and AV nodes fail to pace the heart or when their impulses are blocked. The ventricular rate is 20 to 40 beats per minute, and the ventricular rhythm is usually regular. P waves are usually absent. The QRS duration is greater than 0.12 second, and the T wave is deflected opposite the QRS complex. ♦ Figure 4-33 shows an example of ventricular escape rhythm with a rate of 40 beats per minute.

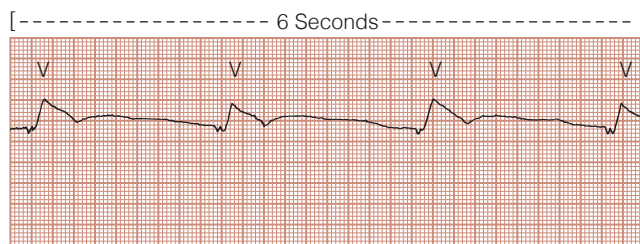


Figure 4-33 Ventricular escape rhythm (idioventricular rhythm).

Evaluation

In general, for ventricular escape rhythm the ECG interpretation will be as follows:

1. Heart rhythm—usually regular
2. Heart rate—20 to 40 beats per minute
3. P waves—usually absent
4. P to QRS ratio—the PVC does not have a P wave preceding it
5. PR interval—none
6. QRS complex—greater than 0.12 second; T waves are frequently in the opposite direction as the QRS complex

Etiology

Ventricular escape rhythm indicates significant slowing of the supraventricular pacemakers and is often seen in the dying heart. The rhythm is typically too slow to provide adequate perfusion.

COLLABORATIVE CARE

Ventricular escape rhythm usually requires immediate resuscitation efforts, including cardiopulmonary resuscitation (CPR) and advanced cardiac life support (ACLS) measures. If there is no pulse with the rhythm, it is treated as PEA and the interventions for PEA are followed. If there is a pulse, the patient should be treated for symptomatic bradycardia with immediate temporary pacing, and the use of atropine, epinephrine, and dopamine should be considered.

Bariatric Considerations

Changes in the ECG of the obese patient often result from changes in the morphology of the myocardium. If the myocardium has hypertrophied, the PR, QRS, and QT intervals may all be increased (Peavy, 2009).

A variety of nonspecific ECG changes may result from the increased distance between the myocardium and the heart.

The anatomical landmarks for ECG placement and cardiac monitoring may be obscured by the increase in

the obese patient's chest size. Variable placement for leads can alter the characteristics of waveforms for alarm triggers and dysrhythmia analysis (Winkelman & Maloney, 2009).

Premature ventricular contractions may be more common in an overweight patient due to left ventricular hypertrophy, and atrial fibrillation occurs more frequently in obese patients.

Commonly Used Medications

Amiodarone (Brand Name: Cordarone)

Introduction

Amiodarone is indicated in (a) stable irregular narrow complex tachycardia, such as atrial fibrillation, (b) stable regular narrow-complex tachycardia, (c) to control rapid ventricular rate due to accessory pathway conduction in pre-excited atrial arrhythmias, (d) hemodynamically stable monomorphic VT, and (e) polymorphic VT with normal QT interval (Neumar et al., 2015).

Desired Effect: Amiodarone is an antiarrhythmic agent. It has actions similar to calcium channel blockers on slowing conduction and prolonging refractoriness at the AV node. Amiodarone also prolongs the duration of cardiac action potential via the sodium and potassium channels, raising the threshold for SVT and ventricular fibrillation, and may prevent its recurrence (Wilson et al., 2016).

Nursing Responsibilities:

- Ensuring administration of therapeutic dose. Amiodarone 150 mg IV (diluted in 100 mL D₅W) is administered over 10 minutes and repeated as needed to a maximum dose of 2.2 grams in 24 hours. If the amiodarone is effective in converting the tachydysrhythmias back to NSR, the patient is maintained on an IV infusion of amiodarone. Amiodarone infusion is dosed at 1 mg/min over 6 hours, followed by 0.5 mg/min maintenance infusion over 18 hours. In cardiac arrest the dosage is increased from 150 mg to an initial dose of 300 mg IV, which can be followed by a second dose of 150 mg (Neumar et al., 2015).

- The preferred route of administration is through a central venous line due to risk of vein phlebitis; use of a 0.2 micron in-line filter is recommended, along with a glass bottle for infusions that will take over 2 hours to infuse, because amiodarone may leach out chemicals in plastics (Wilson et al., 2016).
- PO Load: 800–1600 mg PO qDay for 1–3 weeks until response; once adequate arrhythmia control achieved, reduce dose to 600–800 mg/day for 1 mo; then reduce to maintenance dose of 400–600 mg per day
- Continuous monitoring of HR, BP, QTc (QT corrected) and cardiac rhythm pattern is essential during IV administration.
- If a patient is taking Warfarin, concomitant administration of Amiodarone will increase Warfarin properties. Warfarin doses need to be reduced in order to maintain a therapeutic INR.

Side and/or Toxic Effects: Side effects include hypotension, bradycardia, or QT interval prolongation; amiodarone may cause torsades de pointes, a type of ventricular tachycardia. Amiodarone has a long half-life; therefore, side effects to the drug may continue even after the drug has been discontinued. In the elderly, clearance of amiodarone is slower and half-life may be doubled to up to 47 days (Wilson et al., 2016). Baseline pulmonary status evaluation, liver and thyroid function tests should be obtained if possible.

Ventricular Tachycardia

Ventricular tachycardia (VT or VTach) is a run of three or more PVC in rapid succession. The ventricular rate is usually between 110 and 250 beats per minute, and the rhythm is usually regular. The P waves are usually absent, or hidden in the QRS complexes. The QRS duration is greater than 0.12 second. The rhythm is referred to as non-sustained VT if it lasts less than 30 seconds and sustained VT if it persists for more than 30 seconds. ♦ Figure 4-34 shows an example of ventricular tachycardia.

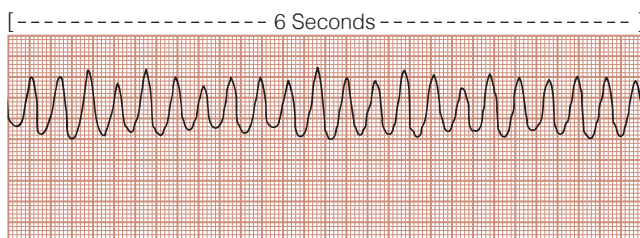


Figure 4-34 Ventricular tachycardia.

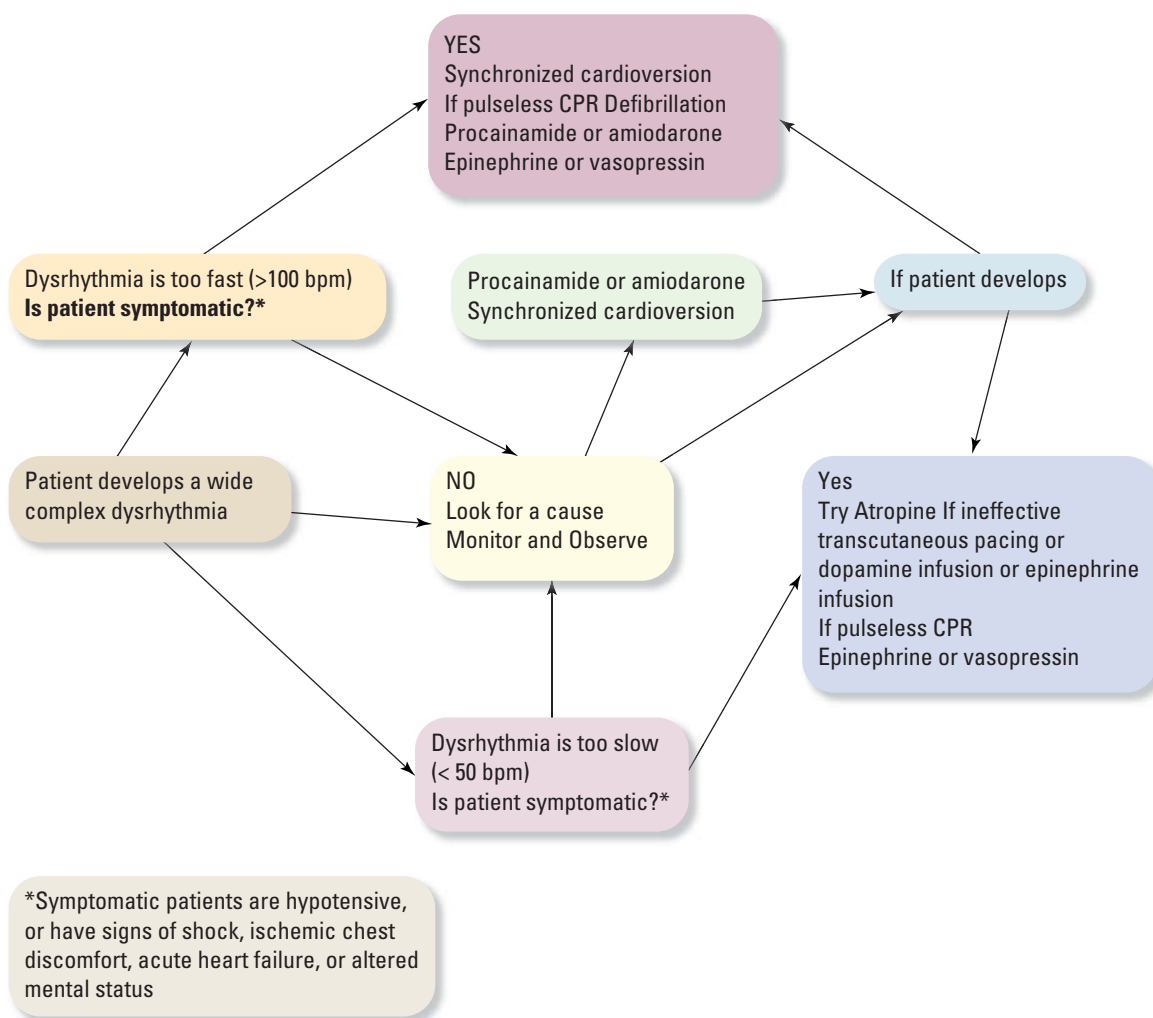
Evaluation

In general, for VT the ECG interpretation will be as follows:

1. Heart rhythm—usually regular
2. Heart rate—110 to 250 beats per minute
3. P waves—usually absent
4. P to QRS ratio—the PVC does not have a P wave preceding it
5. PR interval—none
6. QRS complex—greater than 0.12 second; the shape, width, and amplitude of QRS complexes are all similar; T waves are often difficult to distinguish from QRS complexes

Etiology

VT is usually indicative of significant cardiac disease. VT can be seen in patients with acute MI, coronary artery disease, cardiomyopathy, heart failure, and valvular heart disease. It may also be seen in electrolyte imbalance such as hypokalemia or hypomagnesemia. This rhythm can be mimicked by artifact, such as by scratching or tapping the electrodes, or by shivering. When a dysrhythmia is seen on



Visual Map 4-2 Wide Complex Dysrhythmia

the monitor, the nurse should always assess the patient, not just the monitor, before initiating treatment! Disconnected monitor leads, loose electrode patches, and patient shivering can all mimic dysrhythmias.

COLLABORATIVE CARE

Patients in VT may present with a pulse and in a relatively stable condition, or without a pulse and in significant hemodynamic compromise. The treatment will depend on the patient's presentation. A stable patient in sustained VT may be successfully treated with an antiarrhythmic drug such as amiodarone, procainamide, or sotalol, or with synchronized cardioversion. If antiarrhythmic therapy is unsuccessful, cardioversion and expert consultation should be considered (Neumar et al., 2015).

Magnesium may be effective in terminating a particular type of VT called torsades de pointes. *Torsades de pointes* is a French term that means "twisting of the points." It is a polymorphic VT characterized by long QT intervals and QRS complexes that change shape, width, and amplitude.

The strip in ♦ Figure 4-35 shows the changing of the amplitude in torsades de pointes as the complexes gradually increase and then gradually decrease, giving the tracing a wavy appearance. It may be caused by certain medications, hypomagnesemia, and other conditions that prolong the QT interval.

Ventricular Fibrillation

Ventricular fibrillation (VF) represents severe electrical chaos in the ventricles. Multiple irritable foci fire in an erratic and disorganized manner, resulting in no

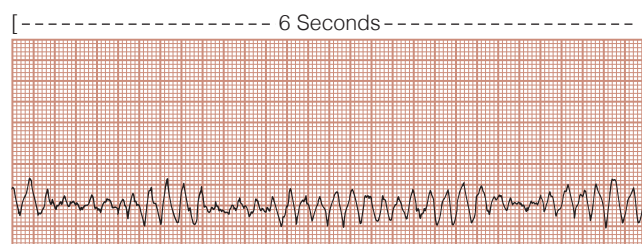


Figure 4-35 Torsades de pointes.

Commonly Used Medications

Lidocaine (Brand Name: Xylocaine)

Introduction

Lidocaine is less effective in terminating VT than procainamide, sotalol, and amiodarone; therefore, it should be considered a second-line antiarrhythmic therapy for monomorphic VT (Neumar et al., 2015).

Desired Effect: Lidocaine is an antiarrhythmic drug that blocks the sodium channels, decreasing cardiac automaticity and depolarization.

Nursing Responsibilities:

- The initial dose is 1 to 1.5 mg/kg IV. If ventricular ectopy persists, repeat doses of 0.5 to 0.75 mg/kg IV may be

given at 5–10-minute intervals to a maximum dose of 3 mg/kg (Neumar et al., 2015). A maintenance IV infusion of 1 to 4 mg/min is usually hung if the bolus was able to terminate the ventricular dysrhythmia.

- The nurse should continuously monitor the ECG.

Side and/or Toxic Effects: Many side effects are transient because of the short duration of action of lidocaine; these include confusion, dizziness, numbness of tongue and lips, hallucinations, and vomiting. Major side effects include anaphylaxis, bradycardia, seizures, cardiac arrest, hypotension, respiratory depression, and cardiovascular collapse.

Commonly Used Medications

Magnesium

Introduction

Magnesium is indicated in polymorphic VT associated with QT prolongation, such as torsades de pointes (Neumar et al., 2015).

Desired Effects: Magnesium is an electrolyte replenisher, a central nervous system (CNS) depressant, and a cardiac muscle depressant. It acts as an anticonvulsant and anti-dysrhythmic. It can reverse VT or VF caused by hypomagnesemia. Its onset of action is immediate, and it is effective for about 30 minutes.

Nursing Responsibilities:

- Ensuring accurate dosage: 1 to 2 grams is administered diluted in 10 mL D₅W over 5 to 20 minutes slow IV push.
- The nurse should continuously monitor the ECG and the magnesium level.

Side and/or Toxic Effects: Side effects usually result from magnesium intoxication and include CNS depression, respiratory depression, hypotension, complete heart block, circulatory collapse, and cardiac arrest (Wilson et al., 2016).

organized depolarization of the ventricles and, therefore, no ventricular contraction, resulting in no cerebral, myocardial, or systemic perfusion. Without successful termination of this dysrhythmia, death will follow within minutes. The ventricular rate cannot be determined because there are no discernible waveforms. The rhythm is rapid and chaotic and is described as coarse or fine VF depending on the amplitude of the irregular undulations.

♦ Figure 4-36a shows an example of VF. ♦ Figure 4-36b shows an example of a sinus rhythm that deteriorated into a few beats of VT and then into coarse VF, maybe due to the R-on-T phenomena.

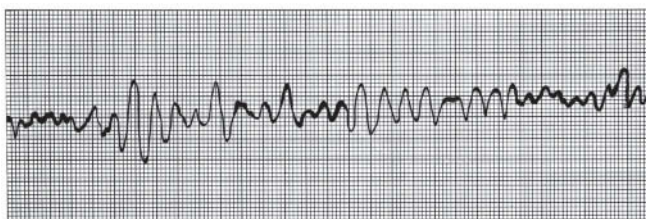


Figure 4-36A Ventricular fibrillation.

Evaluation

In general, for VF the ECG interpretation will be as follows:

- Heart rhythm—regular or irregular
- Heart rate—unable to determine, no identifiable wave patterns
- P waves—undetectable
- P to QRS ratio—none
- PR interval—none
- QRS complex—undetectable

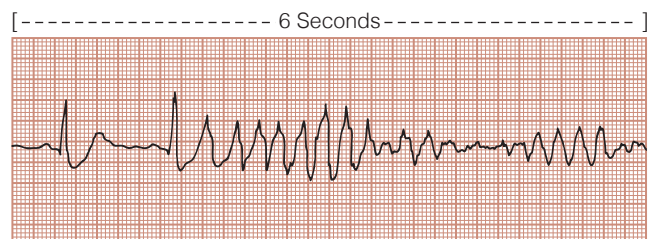


Figure 4-36B Sinus rhythm that deteriorated into a few beats of ventricular tachycardia, and then into ventricular fibrillation.

Commonly Used Medications

Procainamide (Brand Name: Pronestyl)

Introduction

Procainamide is indicated in hemodynamically stable monomorphic VT (Neumar et al., 2015).

Desired Effect: Procainamide is an antiarrhythmic agent that blocks sodium channels and prolongs the cardiac action potential. This results in slowed conduction of both atrial and ventricular dysrhythmias and a reduction in myocardial irritability. An effect from the medication should occur in 2 to 3 minutes.

Nursing Responsibilities:

- Procainamide is given via IV infusion as a loading dose at 20 mg/min until the arrhythmia is suppressed, hypotension ensues, the QRS complex widens by 50%, or a total of 17 mg/kg of the drug has been given. A maintenance dose is started after the dysrhythmia is suppressed or a maximum dose is reached at 1 to 4 mg/min.
- For the loading dose, 1 gram of procainamide can be diluted in 50 mL D₅W to yield 20 mg/mL. This is

administered via an infusion pump at 1 mL/min to get the desired dose of 20 mg/min. A total loading dose for a patient weighing 70 kg at 17 mg/kg would be 1,190 mg, or the entire 1 gram, unless the loading dose needs to be discontinued due to hypotension, widened QRS complex, or suppression of the dysrhythmia. Wilson and colleagues (2016) suggest the use of dopamine to correct hypotension.

- The nurse should continuously monitor the ECG and BP, assessing for widened QRS and hypotension.

Side and/or Toxic Effects: Side effects include blood dyscrasias, hypotension, PR interval prolongation, QRS complex widening, QT interval prolongation, and ventricular asystole (Wilson et al., 2016).

Note: If the patient becomes unstable during the administration of an antiarrhythmic drug, immediate synchronized cardioversion is performed. If the rhythm progresses to a pulseless VT at any time, the rhythm is treated as ventricular fibrillation, and the patient is immediately defibrillated with an unsynchronized shock.

Etiology

The patient in VF rapidly loses consciousness and becomes pulseless and apneic. Respiratory and metabolic acidosis develop. Death rapidly follows if the VF is not converted to an organized rhythm that can sustain a cardiac output. VF is often associated with coronary artery disease, MI, and cardiomyopathy. As with any pulseless arrest, the healthcare professionals need to be searching for and treating possible causes of the arrest outlined in the ACLS protocols. These possible contributing factors include hypovolemia, hypoxia, acidosis, hyper- or hypokalemia, hypoglycemia, hypothermia, toxins, cardiac tamponade, tension pneumothorax, thrombosis, or trauma.

COLLABORATIVE CARE

The immediate need of the patient in VF includes restoring cardiac output; therefore, CPR with minimal interruption in chest compressions is essential while a defibrillator or automated external defibrillator (AED) is being accessed. The ACLS algorithm for ventricular fibrillation and pulseless ventricular tachycardia must be initiated as soon as possible. The algorithm calls for one shock, via defibrillation, as soon as it can be accomplished, then CPR is immediately resumed for five cycles. If VF or pulseless VT persists, another shock is delivered and CPR again resumed. If VF continues, a vasopressor,

epinephrine, can be given. CPR should not be interrupted to give medications; rather, the drugs should be administered during CPR to minimize interruptions in chest compressions.

Epinephrine promotes vasoconstriction by means of alpha-stimulated peripheral vascular resistance. This allows the blood to be shunted from the extremities to the body's core. This can increase coronary and cerebral perfusion pressure during CPR. In cardiac arrest, epinephrine may be administered via IV, down the endotracheal tube, or via intraosseous cannulation (IO). The appropriate dose of epinephrine is 1 mg IV/IO every 3 to 5 minutes. (The learner will recall that epinephrine was discussed in conjunction with symptomatic bradycardia; however, the dose for that is much less at 2 to 10 mcg/min infusion.)

If VF or pulseless VT persists after two or three defibrillations, CPR and IV administration of epinephrine, an antiarrhythmic drug, may be considered. Amiodarone is the antiarrhythmic drug of choice followed by lidocaine. Amiodarone is also used for VT with a pulse (and discussed under that section), but in cardiac arrest the dosage is increased (see section on "Amiodarone").

Essential for Collaboration

The treatment for ventricular fibrillation and *pulseless* ventricular tachycardia is defibrillation: "D Fib for V Fib."

If VF or pulseless VT is not converted back to an organized rhythm with the above measures, it may deteriorate into asystole or pulseless electrical activity.

Reflect On

You are working in the ICU caring for Mr. Fogg, a 65-year-old post-MI patient. The monitor technician tells you that she just noticed Mr. Fogg's rhythm change from NSR to second-degree heart block. You quickly go to assess Mr. Fogg; what are you going to assess? You notify Mr. Fogg's cardiologist of the rhythm change. What interventions do you anticipate will be needed for Mr. Fogg? How do you feel about your performance in stressful situations such as this? What can you do to become more confident in stressful situations?

Ventricular Asystole

Ventricular asystole is also referred to as cardiac standstill. It is the absence of any electrical activity in the heart. There is no ventricular activity and, therefore, no QRS complexes, no ventricular contraction, and no cardiac output. There may still be some electrical firing from the SA node, producing P waves on the ECG, but there is usually no conduction or firing from the ventricles. ♦ Figure 4-37 shows an example of ventricular asystole.

Evaluation

In general, for asystole the ECG interpretation will be as follows:

1. Heart rhythm—none present
2. Heart rate—none
3. P waves—usually none present
4. P to QRS ratio—none
5. PR interval—none
6. QRS complex—none

Etiology

The causes of asystole are usually the same as those listed for VF. As with VF, these patients are unresponsive, pulseless, and apneic. The survival rate from this dysrhythmia is very poor.

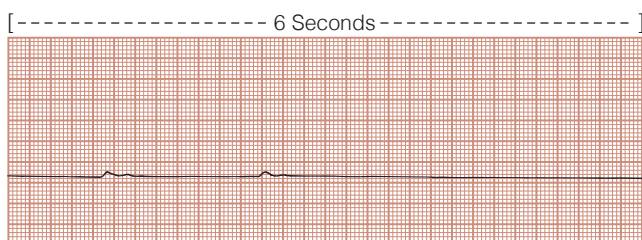


Figure 4-37 Ventricular asystole.

COLLABORATIVE CARE

Immediate CPR, with minimal interruptions, should be initiated. Patients in ventricular asystole will not benefit from defibrillation because there is no electrical activity to attempt to reorganize. After five cycles of CPR, the rhythm should be rechecked; if asystole is confirmed, CPR is resumed and epinephrine 1 mg IV/IO may be administered every 3 to 5 minutes. After each drug delivery and approximately five cycles of CPR, the rhythm is rechecked to see if it has converted to a shockable rhythm, such as VF or pulseless VT. If a shockable rhythm is present, the guidelines for VF should be followed; if an organized rhythm with a pulse is present, post-resuscitation care is initiated. If the patient remains in asystole, the nurse should consider family presence, if it has not already been addressed, and termination of resuscitation efforts should also be considered.

Essential for Evidence-Based Practice

For the best chance of survival, three actions must occur within minutes of cardiac arrest: (a) activation of the emergency medical services, (b) high-quality CPR, and (c) operation of an AED (Neumar et al., 2015).

Pulseless Electrical Activity

PEA is a clinical situation in which organized electrical activity is present, but the patient is pulseless due to absent or inadequate mechanical activity or the inability of the heart to fill or contract. The electrical activity may produce contractions, but the contractions are too weak to produce a pulse or BP. PEA has a poor prognosis unless a reversible underlying cause can be rapidly identified and treated.

Etiology

As listed under VF, the causes of PEA include hypovolemia, hypoxia, acidosis, hyper- or hypokalemia, hypoglycemia, hypothermia, toxins, cardiac tamponade, tension pneumothorax, thrombosis, and trauma.

COLLABORATIVE CARE

The intervention for pulseless electrical activity follows the same ACLS algorithm as described for asystole. However, it is essential that the resuscitation team be looking for a treatable cause of the PEA. For example, if hypovolemia is suspected, a fluid bolus should be given; if the patient is hypothermic, warming should be initiated.

All pulseless arrest patients should be provided with high-quality CPR and competent ACLS efforts following

the AHA guidelines (Neumar et al., 2015), unless prior provisions for a “do not resuscitate” order have been made. The decision to terminate resuscitation efforts is never an easy one; however, AHA (Neumar et al., 2015) suggests that in an advanced life support (ALS) response in the field, termination of resuscitation be considered when ALL of the following criteria apply: (a) arrest was not witnessed, (b) no bystander CPR was provided, (c) no return of spontaneous circulation occurred after full ALS care in the field, and (d) no AED shocks were delivered.

Building Technology Skills

Some dysrhythmias do not convert back to a sinus rhythm with conservative measures and drug therapy alone. Some of the potentially life-threatening dysrhythmias may need defibrillation or synchronized cardioversion. There are also many other procedures that can be performed in an **electrophysiology (EP)** lab that may help restore the normal cardiac rhythm. Electrophysiology is the study and intervention of cardiac dysrhythmias. Some of the common procedures besides synchronized cardioversion include pacemakers, implantable cardioverter/-defibrillators, and radiofrequency catheter ablation. These technologies are discussed in this section.

Defibrillation and Cardioversion

A defibrillator can provide two types of shocks: unsynchronized shocks (defibrillation) and synchronized shocks (cardioversion). In both cases electrical current is delivered through the chest and heart to depolarize myocardial cells in an attempt to allow the SA node to regain control of the heart. **Defibrillation**, however, typically uses a higher energy level than cardioversion. Defibrillation, as the name states, is the de-*fibrillation* of a heart that is in *ventricular fibrillation*, or “D-fib for V-fib” (Cummins, 2001). Defibrillation is used for the shockable pulseless arrest dysrhythmias, which include VF and pulseless VT. Defibrillators are classified as either monophasic or biphasic waveform defibrillators. The original defibrillators were the monophasic type, and they are still used in many facilities. The newer defibrillators are the biphasic type, meaning that energy is delivered in two phases with each shock. The biphasic defibrillators are capable of effectively terminating VF with lower energy levels than are required by the older monophasic models. The defibrillator operator selects the shock energy level depending on the type of defibrillator. The self-adhesive pads are firmly applied to the patient’s chest. To protect the patient from burns, the pads should not be placed over ECG wires or medication patches. CPR is continued while the preparations for defibrillation are carried out.

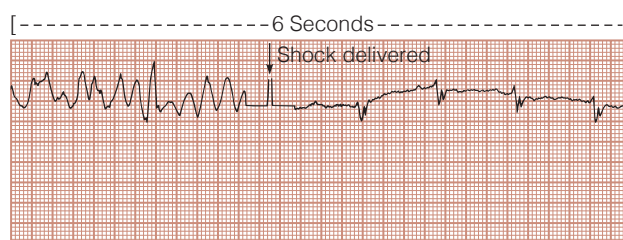
Once the defibrillator is fully charged, the defibrillator operator calls out the “all clear” order and checks to be sure no one is touching the bed or the patient; the shock is then delivered and CPR is resumed. ♦ Figure 4-38a gives an example of a pulseless ventricular fibrillation that was successfully defibrillated into a sinus rhythm.

Synchronized cardioversion is a shock that is synchronized with the QRS complex. The “synchronize” button on the defibrillator must be pressed to put the defibrillator in the synchronized mode. In this mode, the defibrillator will recognize the QRS complexes and shock on an R wave, thus avoiding discharging during the vulnerable period of the T wave, which could cause VF. Synchronized cardioversion is indicated for unstable SVT, AF, atrial flutter, and VT with a pulse. The procedure should be explained to the patient and the consent signed, unless the cardioversion is an emergency because the patient is hemodynamically unstable. The patient should be pre-medicated with sedation and pain medication.

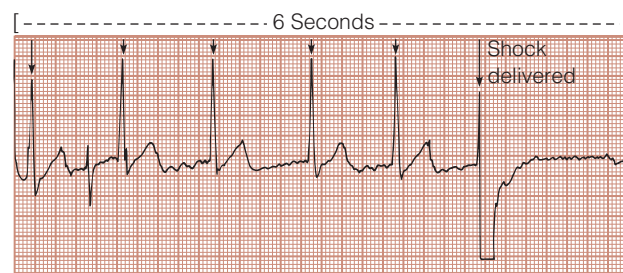
♦ Figure 4-38b shows an example of AF that was successfully cardioverted into a sinus rhythm.

Essential for Safety

Do not place pads or paddles directly over transdermal medication patches because a patch may block delivery of energy from the electrode pad to the heart and may cause burns to the skin. The anterior (negative) pad is placed to the left of the sternum and the posterior (positive) electrode on the left side of the thoracic spine, opposite the anterior pad, or along the left axilla near the bottom of the rib cage.



A



B

Figure 4-38 A, Pulseless ventricular tachycardia/ventricular fibrillation defibrillated into a sinus rhythm. B, Atrial fibrillation cardioverted into a sinus rhythm.

Pacemakers

A pacemaker is a device designed to assist in stimulating the heart when the natural pacemaker is too slow or its impulses are blocked from reaching the ventricles. There are two different methods of pacing: temporary and permanent. There are also two different modes of pacing: fixed-rate and demand pacing. There are pacemaker codes that are used to describe the functions of the pacemaker, and there are malfunctions of pacers. This section discusses these areas. The two major methods of pacing include temporary and permanent pacemakers.

Temporary Pacemaker

A temporary pacemaker can be internal or external.

External Temporary Pacemaker. An external temporary pacemaker is the type commonly used in emergency situations for patients with unstable bradycardia, including unstable second-degree type II block or third-degree block. It is the least invasive type of pacing, and it is quick and effective until a more permanent pacemaker can be inserted. The external temporary pacemaker is also referred to as *transcutaneous pacing* because it is performed through the skin by placing two self-adhesive pacing pads on the chest wall. The anterior (negative) pad is placed to the left of the sternum and the posterior (positive) electrode on the left side of the thoracic spine, opposite the anterior pad, or along the left axilla near the bottom of the rib cage. Most emergency defibrillators can also function as external pacemakers, using the same pads to monitor the heart rhythm, defibrillate, and/or pace the patient. The pacer rate is set as ordered and the threshold is set by finding the lowest current that achieves electrical and mechanical capture. Electrical capture indicates that the electrical impulse from the pacemaker successfully caused depolarization so that on the ECG (a) the pacer spike is followed by a QRS complex, (b) the QRS complex is wide, and (c) the T wave is deflected in the opposite direction of the QRS complex. Mechanical capture is determined by the presence of pulse, indicating cardiac output. The electrical current from the pacemaker travels from one pacer pad to the other through the chest wall and heart, electrically stimulating the muscles in its path. The chest wall will contract with each paced beat. If the patient is conscious, the stimulation of the chest wall muscles may be uncomfortable. Analgesics or sedatives should be given as prescribed to provide comfort.

Internal Temporary Pacemaker. The other type of temporary pacemaker is the internal temporary pacemaker, also called *transvenous* or *epicardial* pacing. If the external pacing is ineffective or if the patient will need temporary pacing for any length of time (more than 24 hours),

transvenous pacing should be initiated. Transvenous pacing usually involves insertion of a pacing wire into either the right atrium or right ventricle via a central venous catheter. The other end of the pacer wires are attached to an external, battery-operated pulse generator. The electrical impulses, at the set rate and current, are emitted from the generator and flow through the lead wire to hopefully depolarize the cardiac cells. The electricity is sent to the cells, but the cells must be capable of conducting the electricity. Epicardial pacing wires may be placed during an open chest, cardiac procedure. There are general two wires attached to the atrium and two attached to the ventricle. The wires exit just below the xiphoid process and are easily removed by a trained professional. Transvenous pacing is not as uncomfortable as transcutaneous pacing; however, if the patient does experience some discomfort, it is usually alleviated by decreasing the pacer current. Often, the QRS complex is wide due to the fact that one ventricle is being depolarized at a time.

Permanent Pacemaker. The permanent pacemaker is used to resolve dysrhythmias that are not temporary, such as third-degree heart block, or SSS (the malfunctioning of the SA node). A permanent pacemaker involves placing one or more wires into the heart chambers. One end of each wire is attached to the heart muscle in the atria and/or ventricles, and the other end is attached to the pacemaker generator. The generator is usually inserted below the subcutaneous fat of the chest wall, usually in the subclavicular space.

Modes of Pacing

The two basic modes of pacing are demand (synchronous) pacing and fixed-rate (asynchronous) pacing.

Demand Pacemaker. A demand pacemaker is synchronized with the patient's heart rate, meaning that it fires (delivers an electrical stimulus) only when the patient's rate drops below a preset rate. The temporary pacemakers often use the demand mode. ♦ Figure 4-39 is an example of ventricular demand pacing. It should be noted there are no pacing spikes before the patient's natural cardiac beats.

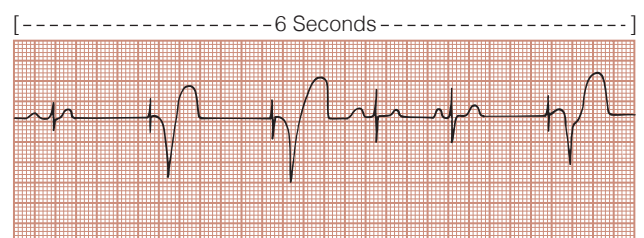


Figure 4-39 Ventricular demand pacing.

Table 4-2 NASPE Pacing Codes

I	II	III	IV	V
Chamber(s) Paced	Chamber(s) Sensed	Mode(s) of Response	Programmable Functions	Antitachycardia Functions
V = Ventricle	V = Ventricle	T = Triggered	R = Rate modulated	O = None
A = Atrium	A = Atrium	I = Inhibited	C = Communicating	P = Paced
D = Dual (A&V)	D = Dual (A&V)	D = Dual (Triggered and Inhibited)	M = Multiprogrammable	S = Shocks
O = None	O = None	O = None	P = Simple Programmable	D = Dual (P&S)

Source: Bernstein et al. (2002). NASPE/BPEG pacing codes.

Fixed-Rate Pacemaker. Fixed-rate pacing is not synchronized with the patient’s heart rate, meaning it fires continually at a preset rate regardless of the patient’s intrinsic heart rate. This pacing mode is not used often because if the patient’s heart rate increases above the pacer set rate, competition will occur. VF may result if the pacer fires an impulse during the vulnerable period of repolarization, causing the pacer spike to fall on the T wave. If the nurse observes pacer competition, the pacemaker should be changed to the demand mode. This mode may be used in a severely bradycardic patient.

Codes in Pacing

The functions of a pacemaker have been coded into three to five-letter codes to describe the main features of the pacemaker. The pacemaker codes have been used since 1974, and have been updated and expanded most recently by the North American Society for Pacing and Electrophysiology (NASPE) and the British Pacing and Electrophysiology Group (BPEG). Refer to Table 4-2, NASPE Pacing Codes. To understand how the codes are used, assume a patient has a DDD pacemaker. This would mean the pacemaker can pace both chambers, can sense both chambers, and is a demand pacemaker with a dual mode of response. This is also known as a dual-chamber pacemaker. ♦ Figure 4-40 is an example of DDD pacing, or dual-chamber pacing. The atria are paced first then the ventricles; therefore, this is also referred to as AV sequential pacing.

A biventricular pacemaker (BVP) paces both the left and right ventricles and is also known as cardiac resynchronization therapy (CRT). By pacing both ventricles, the pacemaker can resynchronize the heart to depolarize the

ventricles at the same time. This improves the functional ability of the heart for patients with moderate to severe heart failure (see Chapter 7, Care of the Patient Experiencing Heart Failure, for further details).

Malfunctions of Pacemakers

The four types of pacemaker malfunctions that the nurse can check for while reading the rhythm strip include failure to pace, failure to capture, failure to sense (undersensing), and oversensing. ♦ Figures 4-41a, b, and c give examples of some of these pacemaker malfunctions.

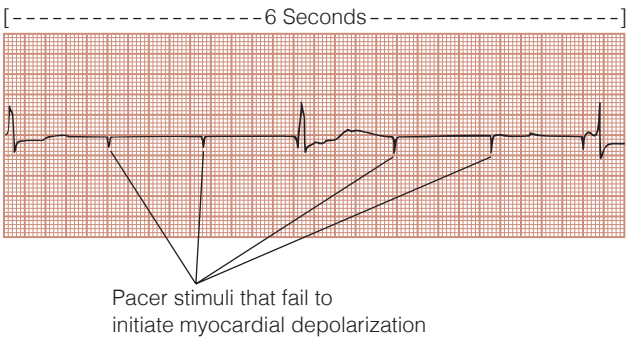


Figure 4-41A Pacemaker malfunction: Failure to capture (noncapture). Notice the pacemaker spikes that are not followed by QRS complexes.

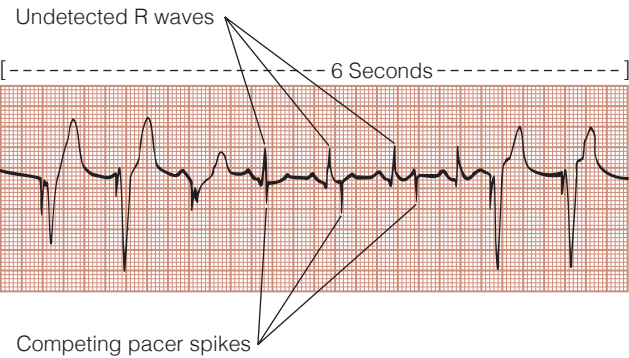


Figure 4-41B Pacemaker malfunction: Failure to sense (undersensing). Notice the pacemaker spikes immediately following two of the patient’s own cardiac beats.

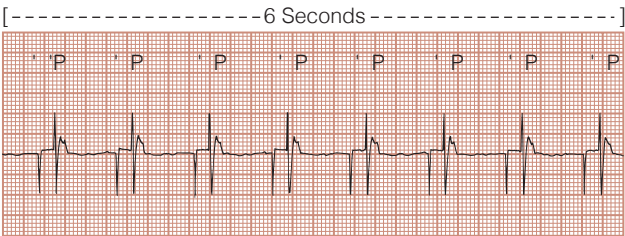


Figure 4-40 Dual-chamber or DDD pacing.

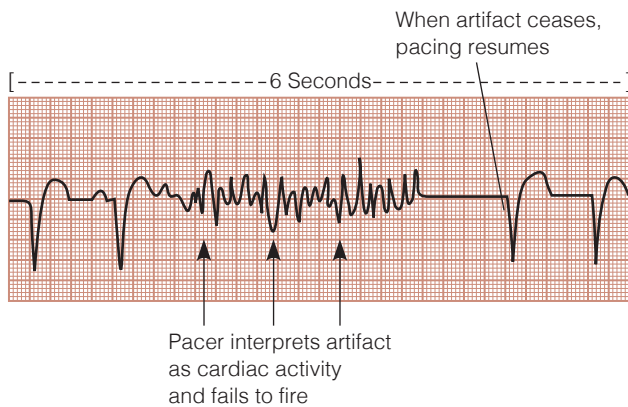


Figure 4-41C Pacemaker malfunction: Oversensing. Notice that there are no QRS complexes during the artifact because the pacemaker interprets this activity as the patient's own cardiac beats.

Failure to Pace. Failure to pace occurs when the pacemaker fails to deliver an electrical stimulus (fire) when it should have fired. This can be seen on the ECG as an absence of pacer spikes when the patient's heart rate is less than the demand pacer rate is set, indicating the pacemaker did not fire. The patient may become bradycardic and hypotensive, and hemodynamically compromised. Causes of failure to pace may include a loose connection between the pacer wire and the generator, a broken pacing wire, or battery failure. The healthcare provider should be notified because the patient will probably need to return to the EP lab for a pacemaker check.

Failure to Capture (Noncapture). Failure to capture occurs when the pacemaker fires, but the chamber that is being paced (atria or ventricles, or both) does not depolarize. This is seen on the ECG strip as a pacer spike that is not followed by a P wave or QRS complex. Again, the patient may become bradycardic and hypotensive. Common causes of failure to capture include displacement of the pacing wire or the energy level set too low. The patient may need to be repositioned or the energy level may need to be slowly increased until capture occurs. Failure to capture can also be seen when the patient expires and has an asystolic rhythm with the pacemaker still on. See Figure 4-41a.

Failure to Sense (Undersensing). Failure to sense occurs when the pacer fails to recognize, or sense, the heart's natural electrical activity. This is seen on the ECG as pacer spikes that occur too closely behind the patient's QRS complex. This can result in a pacemaker spike that falls in the vulnerable period of the T wave and causes an R-on-T phenomenon. Causes of failure to sense include the same problems as noted in the malfunctions above; however, the most common cause is displacement of the electrode tip, or fibrosis at the electrode tip. Treatment may include increasing the sensitivity or replacing the pacing lead. See Figure 4-41b.

Oversensing. Oversensing occurs when the pacer senses extraneous electrical stimuli, or artifact, for actual atrial or ventricular depolarization and therefore fails to fire. This is seen on the ECG as pacemaker spikes that are at a slower rate than the pacemaker's preset rate or no paced beats, even though the patient's heart rate is slower than the pacer's preset rate. The patient should be taught to avoid strong electromagnetic fields, such as magnetic resonance imaging (MRI) machines, and arc welding equipment because electromagnetic interference (EMI) may affect the pacemaker settings. EMI is caused by devices that work on radiofrequency waves of 50 to 60 Hz. This includes such common devices as cellular phones and iPods, and less common devices such as arc welders, antitheft devices, HAM radios, and airport radar. In the hospital the devices that can cause EMI include electrocautery, defibrillation, radiation, radiofrequency ablation, and MRI. These devices can affect the pacemaker in many ways, including oversensing, undersensing, inappropriate reprogramming, mode changes, and pacing rate changes. To avoid EMI with the pacemaker, iPods and cellular phones should not be carried in a shirt pocket over an implanted pacemaker, and the ear on the opposite side of the pacemaker should be used when talking on the cellular phone. Pacer pads for cardioversion or defibrillation should be placed at least 4 to 6 inches away from the implanted pacemaker. To avoid EMI from airport radar, the patient should inform airport security workers about the pacemaker and have an individual security check performed. If any of these devices are used on a patient with a pacemaker in the hospital setting, the pacemaker can be interrogated or checked to verify correct pacemaker settings after the procedure. See Figure 4-41c.

Essential for Evidence-Based Practice

Pacing is not effective for asystolic cardiac arrest and may delay or interrupt chest compressions.

Implantable Cardioverter-Defibrillator

The **implantable cardioverter-defibrillator (ICD)** is a device similar in size to a pacemaker, and implanted in a patient similar to a pacemaker. It is used in patients who are at risk of sudden cardiac death due to VF and those patients who have experienced one or more episodes of VT or VF unrelated to an MI. An ICD constantly monitors the patient's heart rate and rhythm and is multiprogrammable to deliver pacing, cardioversion, and/or defibrillation according to the patient's needs. The ICD is programmed to deliver an electric shock if the patient goes into VF. Most are also programmed to provide overdrive pacing, or fast pacing, if the patient declines into VT in an attempt to break the tachycardia before it progresses to VF. The ICD may also be programmed to pace the heart if severe bradycardia develops.

Radiofrequency Catheter Ablation

Radiofrequency catheter ablation (RFCA) is useful in the treatment of many tachydysrhythmias. RFCA is a high-frequency form of electrical current that creates small necrotic lesions in the heart by means of thermal injury. The high-frequency energy is delivered through a catheter, and the tip of the catheter delivers the current to the site of origin of the dysrhythmia. RFCA has successfully treated SVT, atrial flutter, and AF. RFCA has also successfully treated VT in patients who have ICDs that frequently discharge in response to a VT rhythm. RFCA is performed in the EP lab under IV conscious sedation. Cardiac medications, such as beta blockers, calcium channel blockers, digoxin, and warfarin, are often held prior to the procedure so an assessment of the best response of the heart can be detected. In the procedure, two to five electrode catheters are inserted via the femoral or internal jugular veins and positioned in the right and left side of the heart. For left heart catheterization, the catheter may be placed retrograde across the aortic valve, or transseptally via the interatrial septum. Multiple catheters are used prior to the ablation to map the various tachydysrhythmia pathways. Anticoagulation with IV heparin is used during the procedure to reduce the risk of thromboembolism.

Major complications occur in approximately 3% of patients who have an ablation procedure. These complications include death in less than 0.3% of all procedures; cardiac complications, such as AV block, cardiac tamponade, and pericarditis; vascular complications, such as bleeding, hematoma, thromboembolism, and stroke; and pulmonary complications, such as pneumothorax (Reynolds et al., 2015).

MAZE Procedure

The MAZE procedure is the precursor to RFCA therapy and was developed in the 1980s to eliminate AF. The procedure

was originally performed as an open heart procedure and has developed into a minimally invasive procedure using endoscopy and radiofrequency ablation. The MAZE procedure is done by making a series of incisions arranged in a maze-like pattern in the atria. It is hoped that the incisional scars block the electrical impulses that cause AF. The MAZE procedure is still employed for patients in chronic AF who are scheduled for an open heart procedure for other cardiac problems such as valve repair or coronary artery bypass, thereby eliminating the need for the RFCA procedure.

Cardiac Mapping

To diagnose and treat cardiac dysrhythmias, cardiac mapping may be utilized. Cardiac mapping is also referred to as electrophysiologic mapping and is performed in the EP lab. To determine the origin or origins of a dysrhythmia, multiple flexible wires, called electrode catheters, are threaded into the heart. These catheters can track and stimulate the heart's electrical impulses, allowing the physician to identify the dysrhythmia foci. Other types of catheters may be used to create three-dimensional pictures of the heart. These catheters have multiple miniature electrodes on them that act as radio antennae; they receive signals from all around the heart chamber and relay the signals to a computer. The computer processes the signals into a three-dimensional picture of the heart chamber. By determining the cause of the dysrhythmia, the physician is able to determine if the patient would be best treated by medication, an ICD, a pacemaker, or an ablation procedure. If an ICD or pacemaker is to be inserted or if an ablation procedure is to be preformed, it can usually be done at the time of the mapping and with the guidance of the mapping graphics. Complications of cardiac mapping are similar to those of catheter ablation.

Why/Why Not?

Mr. M is a 90 year-old male who had a coronary bypass surgery three days ago. His epicardial pacing wires have been removed. His postoperative course has been significant for atrial fibrillation, which is new for him. When he is in AF, his BP has been intermittently compromised as his EF is 20%. He has been started on an Amiodarone PO load and continues on his Toprol SR 50 mg po daily. After two days on this combination, he

has converted back into sinus rhythm. On his 12 lead EKG this morning, you note the QTc is 550 ms; previously his QTc was 415 ms. He is scheduled to receive his Toprol and Amiodarone. Should you administer these two medications? Why or why not?

See answers to Why/Why Not? in the Answer Section.

Case Study

As you enter the ICU for your oncoming shift, you are told to go immediately to your assigned room because your patient, a 55-year-old male, is in full cardiac arrest. You quickly learn that your patient had open heart surgery 2 days ago for a coronary bypass graft and mitral valve repair. He is on the ventilator and has a central line for IV medications. You enter the cardiac room and find the code team performing CPR and charging the monitor for another defibrillation. You see ventricular tachycardia on the monitor; however, the patient has no pulse and no blood pressure. The team leader tells you that this is the second shock and no medications have been given yet.

The team leader asks you to be the medication nurse for the code.

1. What medication do you assume will be given first?
2. What is the implication of giving this/these medication(s)?
3. After a second defibrillation, the monitor reveals sinus bradycardia with a rate of 48, what should be done next?
4. What medication(s) should be given next?
5. What might have caused this ventricular dysrhythmia, and what labs might be drawn to help determine the cause of this dysrhythmia?

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 4.1 Tachycardias may be classified as narrow QRS complex or wide QRS complex. What are some examples of each type and how do the electrical pathways differ?
- 4.2 Why is the development of atrial fibrillation significant for a patient?
- 4.3 Why is anticoagulation important for the patient in atrial fibrillation?
- 4.4 What are the common treatment modalities for atrial fibrillation?

- 4.5 What are the characteristics of third-degree heart block?
- 4.6 Which tachycardias may be treated with amiodarone, and what is its mechanism of action?
- 4.7 What are four ways in which pacemakers can malfunction?

See answers to Chapter Review Questions in the Answer Section.

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Chapter 5

Cardiodynamics and Hemodynamic Regulation

Erin McDonough, DNP, AGACNP-BC

Abbreviations

CI	Cardiac Index	RAP	Right Atrial Pressure
CO	Cardiac Output	SaO ₂	Oxygen Saturation
CVP	Central Venous Pressure	SmO ₂	Muscle Oxygen Saturation
MAP	Mean Arterial Pressure	ScvO ₂	Central Venous Oxygen Saturation
PA	Pulmonary Artery	SV	Stroke Volume
PCWP	Pulmonary Capillary Wedge Pressure	SvO ₂	Oxygen Saturation of Venous Blood
PPV	Pulse Pressure Variation	SVR	Systemic Vascular Resistance
PVR	Pulmonary Vascular Resistance	SVRI	Systemic Vascular Resistance Index
PVRI	Pulmonary Vascular Resistance Index	SVV	Stroke Volume Variation



Learning Outcomes

Upon completion of this chapter, the learner will be able to:

1. Distinguish cardiac anatomy and the cardiac cycle.
2. Interpret the basic concepts of hemodynamics.
3. Compare and contrast hemodynamic monitoring technology and the associated nursing responsibilities.
4. Effectively interpret invasive pressure monitoring waveforms.

Introduction

Cardiodynamics and hemodynamic regulation deal with the forces the heart must develop to circulate blood through the cardiovascular system. These hemodynamic forces demonstrate themselves as blood pressure and blood flow. This chapter concentrates on systemic hemodynamics—the blood pressure and blood flow at the output of the left heart.

Heart Anatomy and the Cardiac Cycle

The heart consists of four chambers: the right and left atrium and the right and left ventricles. Major vessels lead into and out of the chambers, moving blood within the systemic and pulmonary systems. Deoxygenated blood returns from the body via the inferior and superior vena

cavae into the right atrium. Blood then moves through the tricuspid valve into the right ventricle. The right ventricle, which is structurally thinner than the left ventricle, moves blood through the pulmonary valve into the pulmonary artery and pulmonary circulation. In the lungs oxygen is diffused into pulmonary circulation from the alveoli, and carbon dioxide is diffused into the alveoli from the pulmonary capillaries for exhalation. Once oxygenated the blood returns to the left atrium via the pulmonary veins and through the mitral (bicuspid) valve into the left ventricle, then leaves the left ventricle through the aortic valve into the aorta, and continues into the systemic circulation. Once in systemic circulation, blood travels to the capillaries where the oxygen and carbon dioxide are exchanged at the tissue level (♦ Figure 5-1). One of the important functions of the cardiopulmonary circulation is tissue perfusion.

The Cardiac Cycle: Diastole and Systole

The **cardiac cycle** is the sequence of events related to the flow of blood from the beginning of one heartbeat to the beginning of the next. It is divided into two phases: diastole and systole (♦ Figure 5-2). The diastolic phase is further divided into the passive phase and the active phase. During the passive phase, when the heart is at rest, blood returning to the right and left atria creates a pressure exceeding that in the ventricles. At this point the tricuspid and mitral valves open, allowing blood to passively fill the right and left ventricles. The active phase immediately follows this passive filling. During this phase the atria contract and empty, fully filling the ventricles. This atrial contraction makes up 20% to 30% of the cardiac output and is an important contribution to the filling of the ventricles. The atrial contraction is stimulated by the electrical impulse originating in the sinoatrial node or atria and traveling through the atrial conduction pathways. This electrical

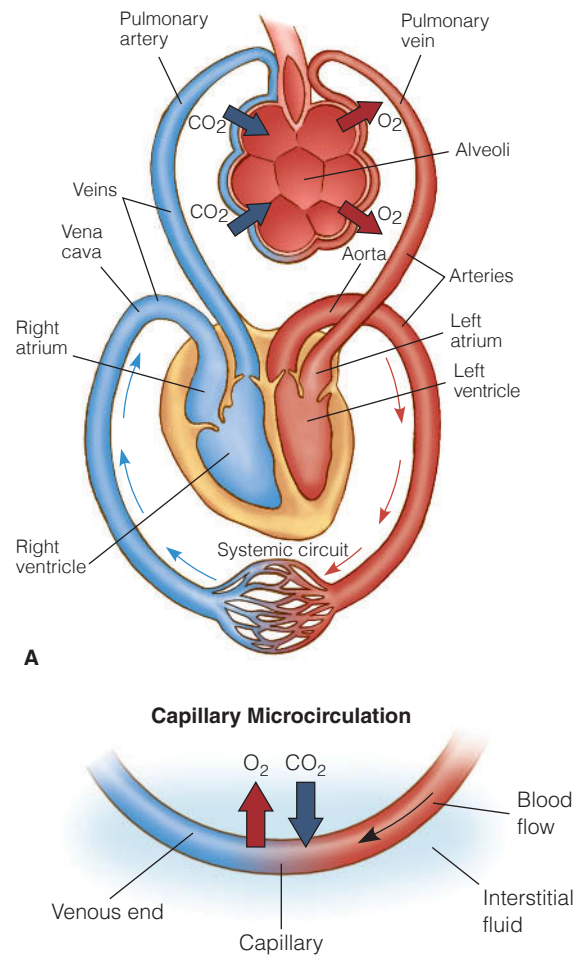


Figure 5-1 Cardiopulmonary circulation: *A*, The deoxygenated blood (blue in color) returns to the right side of the heart, exiting through the pulmonary artery into the pulmonary circulation where carbon dioxide is released into the alveoli and oxygen diffuses into the pulmonary circulation. The oxygenated blood (red in color) returns to the left side of the heart and exits through the aorta. *B*, In the capillaries, the oxygen is released to the tissues.

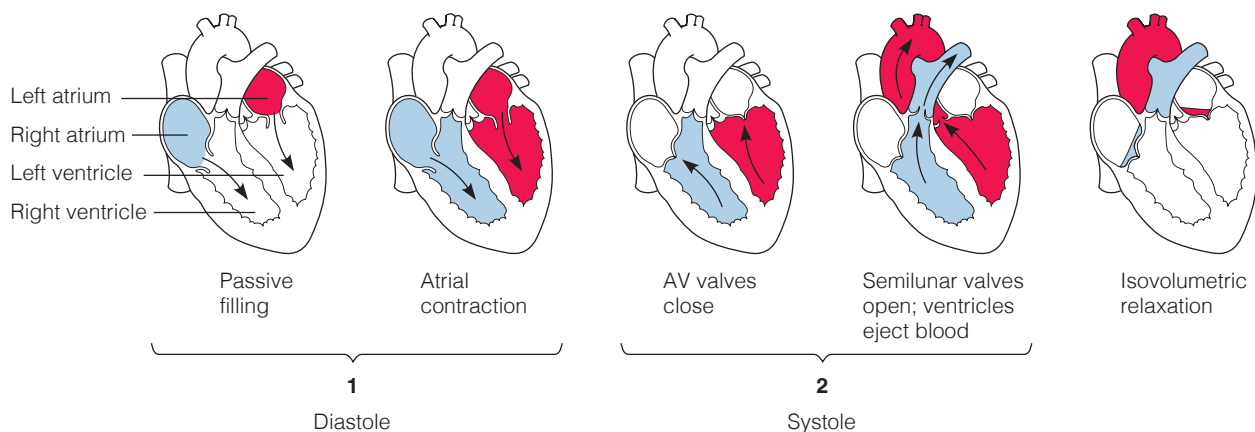


Figure 5-2 The cardiac cycle. The cardiac cycle is divided into diastole and systole. During diastole there is passive flow of blood from the atria to the ventricles followed by an active phase of atrial contraction. Once the ventricles are fully filled, blood is ejected into the aorta and pulmonary circulation.

impulse is seen on the electrocardiogram as the P wave (see Chapter 4 ∞ for discussion of ECG waveform). The nurse should be aware that if the patient is in a rhythm that does not have a P wave (i.e., atrial fibrillation) or if the P wave is positioned abnormally before the QRS (i.e., junctional rhythms), the patient may exhibit signs of decreased cardiac output. This is due to the loss of the “atrial kick” associated with the 20%-30% contribution to the cardiac output.

Once the ventricles are fully filled and the pressure in the ventricle exceeds the pressure in the atria, the tricuspid and mitral valves close. Pressure builds up in the ventricles, opening the pulmonic and aortic valves. Blood is ejected into the pulmonary circulation from the right ventricle and into the systemic circulation from the left ventricle. This is the systolic phase.

Concepts in Basic Hemodynamics

In order to understand basic hemodynamics, it is essential to understand basic cardiac anatomy, the cardiac cycle, blood pressure, cardiac output, preload, afterload, and contractility.

Blood Pressure

The blood pressure is determined by the flow through the vessels and the vessel resistance. The peak pressure is known as the systolic pressure and the lowest point of the pressure is known as the diastolic pressure. The difference between the systolic and diastolic pressure is known as the **pulse pressure**. The **mean arterial pressure (MAP)** is the average pressure over one cardiac cycle. The MAP is thought to be a very good estimate of the perfusion seen by the organs. In many situations, the MAP is a better indicator of overall perfusion than the systolic pressure. A MAP of greater than 60 mmHg indicates adequate perfusion. Most patient monitoring systems (noninvasive and invasive)

automatically calculate the MAP. The formula for calculating the MAP reflects (at normal heart rates) the extended time the heart is in diastole during the cardiac cycle.

$$\text{MAP} = \frac{(\text{systolic pressure}) + (\text{diastolic pressure} \times 2)}{3}$$

Cardiac Output

The **cardiac output (CO)** is the volume of blood ejected by the left ventricle every minute. The normal value is 4 to 8 L/min. The **cardiac index (CI)** is the CO divided by the body surface area, and is the preferred value because it reflects the CO related to patient size. The normal CI is 2.8 to 4.2 L/min/m². Most bedside monitors automatically calculate the CI once height and weight are entered.

CO is determined by the heart rate (HR) × **stroke volume (SV)**. HR most significantly affects the CO when it is either too slow or too fast. Tachycardic rhythms shorten the time in diastole when the heart fills, therefore reducing the amount of blood available for ejection, thus lowering CO. Patients who are bradycardic have a reduced CO simply due to reduced ejection time. Stroke volume is the volume of blood ejected from each ventricle with each heartbeat. The right and left ventricles eject about the same amount, which normally is between 60 and 100 mL per heartbeat (Table 5-1). The primary factors that determine stroke volume are preload, afterload, and contractility.

Preload

Preload is the stretch on the ventricular myocardium at end diastole. Preload is determined by volume left in the ventricles. Pressures are used to evaluate volume at the end of diastole; these are also known as the “filling pressures” of the ventricles. The volume of blood at end diastole is determined by the circulating blood volume and the atrial contraction. Patients who have a reduced circulating volume (e.g., trauma or gastrointestinal [GI] bleed) or who are in an electrocardiogram (ECG) rhythm that does not have an

Table 5-1 Hemodynamic Calculations

Parameter	Calculations	Normal Values
Mean arterial pressure (MAP)	$\text{MAP} = \frac{(\text{systolic pressure}) + (\text{diastolic pressure} \times 2)}{3}$	70–105 mmHg
Cardiac output	Heart rate × stroke volume	4–8 L/min
Cardiac index	Cardiac output ÷ body surface area	2.8–4.2 L/min/m ²
Stroke volume	Cardiac output × 1000 ÷ heart rate	60–100 mL/beat
SVI	SV/BSA	30–65 mL/beat/m ²
SVR	$\text{MAP} - \text{CVP/RA} \times 80 \div \text{CO}$	900–1400 dynes/s/cm ⁻⁵
SVRI	$\text{SVR} \div \text{BSI}$	2000–2400 dynes/s/cm ⁻⁵
PVR	$\text{PA mean} - \text{PCWP} \times 80 \div \text{CO}$	100–250 dynes/s/cm ⁻⁵
PVRI	$\text{PVR} \div \text{BSI}$	255–315 dynes/s/cm ⁻⁵

effective atrial contraction (e.g., atrial fibrillation) may present with a reduced preload. Additionally, end-diastolic volume is also determined by the distribution of blood volume. Increases in intrathoracic pressure and venodilation, as occurs in patients who are mechanically ventilated or sometimes as a side effect of medications such as nitroglycerin, can reduce the volume of blood returning to the heart and therefore reduce the preload. Preload exists for the right and left ventricles. Both the right and left preload are decreased in states of reduced circulating blood volume (e.g., trauma or GI bleed). As the preload decreases, the CO/CI may be reduced. Right heart preload is elevated in patients with volume overload, tricuspid stenosis or insufficiency, pulmonary stenosis or insufficiency, pulmonary hypertension, and right ventricular infarction. Left heart preload is elevated in left-sided heart failure, volume overload, and mitral stenosis. Both right and left preload are elevated in conditions that cause increases in pericardial pressures such as pericardial tamponade or constrictive pericarditis (Table 5-2).

Afterload

Afterload is the pressure (resistance) against which the right or left ventricle has to pump to eject the blood. The right ventricular resistance is the pulmonary vascular resistance (PVR). Elevations in PVR occur in pulmonary hypertension, hypoxia, pulmonary embolism, and pulmonary stenosis. The left ventricular resistance is the **systemic vascular resistance (SVR)**. Elevations in SVR occur in hypertension, vasopressor use, aortic stenosis, and hypothermia. Decreased afterload may occur in cases of septic shock, anaphylactic or neurogenic shock, use of vasodilators, and side effects to some medications (e.g., narcotics) (Table 5-3). If the PVR is elevated, less blood is available to the left ventricle, potentially decreasing the CO/CI. If the SVR is elevated, less blood is able to be ejected from the left ventricle, which may decrease the CO/CI.

Table 5-2 Causes of Elevated and Decreased Preload

	Right Ventricle	Left Ventricle
Elevated preload	• Volume overload • Tricuspid stenosis • Tricuspid insufficiency • Pulmonary stenosis • Pulmonary insufficiency • Pericardial tamponade • Constrictive pericarditis • Right ventricular infarction • Pulmonary hypertension	• Left-sided heart failure • Volume overload • Mitral stenosis • Mitral insufficiency • Cardiomyopathy • Myocarditis • Pericardial tamponade • Constrictive pericarditis
Decreased preload	• Hypovolemia • Venodilation	• Hypovolemia

Table 5-3 Causes of Elevated and Decreased Afterload

	Elevated	Decreased
Systemic vascular resistance (SVR)	• Hypertension • Vasopressor use • Aortic stenosis • Hypothermia	• Septic shock • Anaphylactic shock • Neurogenic shock • Use of vasodilators • Side effects of some medications
Pulmonary vascular resistance (PVR)	• Pulmonary hypertension • Hypoxia • Pulmonary embolism • Pulmonary stenosis	• Use of vasodilators • Side effects of some medications

Contractility

Contractility is the ability of the myocardium to shorten the muscle fibers. Contractility of the heart is influenced by electrolytes (particularly potassium and calcium), acid-base abnormalities, and myocardial oxygen supply/demand abnormalities. Decreased contractility may be found in patients with hyperkalemia, hypocalcemia, myocardial ischemia, administration of negative **inotropic** medications, hypercapnea, hypoxia, and acidosis. Increased contractility may be found in patients with hypercalcemia, administration of positive inotropic medications, or sympathetic stimulation (Table 5-4).

A relationship, known as the Frank-Starling Law of the heart, exists between end-diastolic volume or PCWP and cardiac output. As the end-diastolic volume increases, the myocardial fibers stretch, thus increasing the force of contractions and increasing the cardiac output. Adequate cardiac output is dependent on adequate end-diastolic volume to optimize the contractility. As contractility decreases (such as in heart failure) or afterload increases, there is a reduction in cardiac output (♦ Figure 5-3).

Tissue Oxygen Supply and Demand

The primary goal of the cardiovascular system is to maintain a balance between oxygen supply to the tissues and

Table 5-4 Causes of Elevated and Decreased Contractility

Elevated	Decreased
• Hypercalcemia • Administration of positive inotropic medications • Sympathetic stimulation	• Hyperkalemia • Hypocalcemia • Myocardial ischemia • Administration of negative inotropic medications • Hypercapnea • Hypoxia • Acidosis

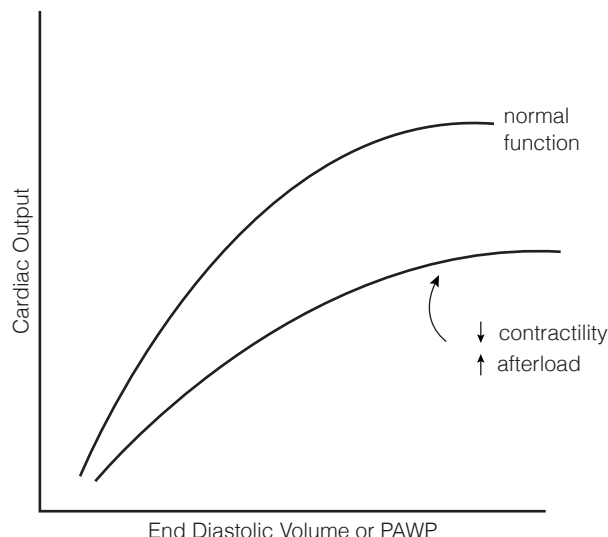


Figure 5-3 Frank-Starling Curves. There is a relationship between cardiac output and end diastolic volume or pressure. As end diastolic volume increases, cardiac output increases. As contractility decreases and afterload increases, cardiac output is reduced.

oxygen demand of the tissues at any given time. An imbalance of oxygen supply and demand can lead to tissue hypoxia, cellular death, and eventual organ failure.

Oxygen supply to the tissues is dependent on how much oxygen is in the arterial bloodstream (oxygen content) and how effectively that oxygen content is being delivered to the tissues.

Oxygen Content

The two determinants of oxygen content in the arterial blood are the oxygen saturation (SaO_2) of the hemoglobin and the oxygen dissolved in the blood (pO_2). The majority of oxygen in the blood is in the form of oxygen bound to hemoglobin. An adequate amount of hemoglobin is required for effective oxygen-carrying capacity. The SaO_2 value indicates what percentage of available hemoglobin is saturated. The nurse must always evaluate the patient's hemoglobin level when assessing oxygen supply. If a patient has a low hemoglobin level, the SaO_2 may appear adequate, but the overall oxygen-carrying capacity will be reduced due to the low hemoglobin level. A small percentage of oxygen is dissolved in the blood and is indicated by the pO_2 value. At the bedside, the arterial oxygen saturation can be estimated by pulse oximetry or measured via an arterial blood gas analysis. The pO_2 is measured as part of an arterial blood gas analysis. Oxygen content may also be calculated on an arterial blood gas report.

Cardiac Output

The oxygen content is delivered to the tissues by the CO. An adequate oxygen supply to the tissues is dependent on a normal CO.

Oxygen Demand

Oxygen demand is the tissue's need for oxygen. Oxygen demand is increased when the patient's metabolic rate is increased such as in infection, hyperthyroidism, shivering, or seizure activity. Stressful states can also increase oxygen demand such as with surgery, pain, and trauma. Oxygen demand cannot be assessed directly; therefore, the nurse must utilize measures to assess the balance of oxygen supply and demand.

Evaluating the Balance of Oxygen Supply and Demand

Tissue oxygenation is adequate if the oxygen supply and demand is balanced (♦ Figure 5-4a). An imbalance can occur with the following conditions: decreased arterial oxygen saturation, decreased cardiac output, decreased hemoglobin shown in ♦ Figure 5-4b or increased cellular demand (over the supply) shown in ♦ Figure 5-4c. Commonly used methods to determine an imbalance of supply and demand are laboratory measurements of lactic acid and measurement of mixed venous oxygen saturation.

Lactate

Under normal conditions when oxygen is available to the tissues, glucose is metabolized into carbon dioxide (CO_2)

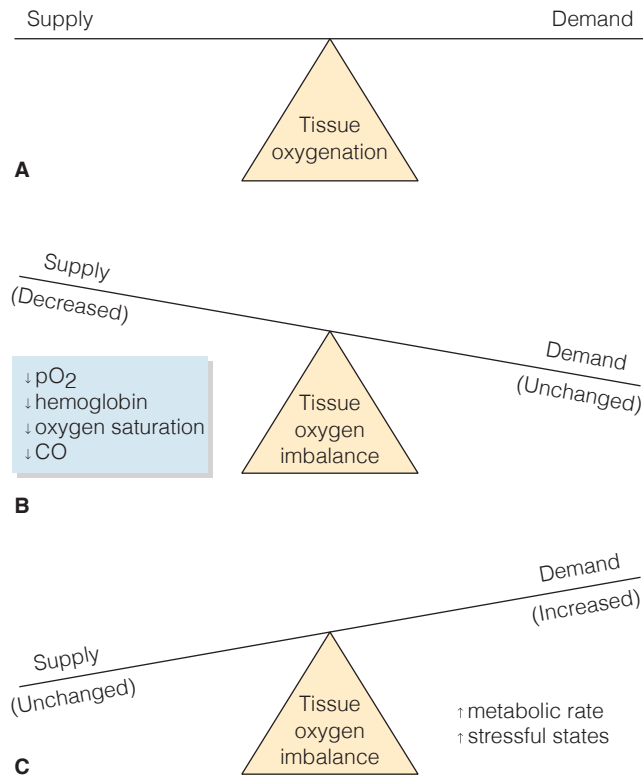


Figure 5-4 Tissue oxygenation supply and demand. A, Balanced supply and demand. B, A decreased supply compared to demand, creating an imbalance of tissue oxygenation. C, An increased demand with an unchanged supply creates an imbalance of tissue oxygenation.

and water. When the cells experience an oxygen debt, anaerobic metabolism of glucose occurs, resulting in lactate formation instead of CO₂ and water. Liver dysfunction is a contributing factor to elevated lactate levels as lactate clearance is delayed. Lactate accumulates in the blood during conditions of tissue hypoxia; therefore, elevated levels of lactate (serum lactate greater than 4 mmol/L) are a reliable indicator of tissue hypoxia. **Lactate clearance**, or the trending of lactate values towards normal, is measured during resuscitation from oxygen supply and demand imbalance. Rapid improvement is associated with better patient outcomes, while failure to improve is associated with adverse clinical outcomes (Zhang & Xu, 2014).

Mixed Venous Oxygen Saturation

The oxygen saturation returning from the tissues to the venous system is a reflection of the oxygen supply and demand. The oxygen saturation of venous blood (SvO₂) and the central venous oxygen saturation (ScvO₂) are influenced by the concentration of hemoglobin and the oxygen saturation of the hemoglobin, CO, and the amount of oxygen extracted and utilized by the tissues. The percentage of saturation in the venous system is normally lower than the arterial system due to consumption at the tissue level and the hemoglobin giving up the oxygen at the tissue level. The SvO₂, sometimes referred to as “mixed venous saturation,” is normally 60% to 75%. SvO₂ can be continuously monitored by the nurse using a specialized (fiber-optic) catheter that is positioned in the pulmonary artery, or it can be intermittently evaluated by drawing a sample from a similar pulmonary artery catheter. The ScvO₂, or central venous saturation, is generally about 5% higher than the SvO₂ levels and is obtained by continuously or intermittently sampling the saturation from a catheter positioned near the right atrium. The variation between SvO₂ and ScvO₂ can be greater in patients with sepsis (Van Beest, 2012). Although SvO₂ was once considered the primary marker of tissue oxygenation in patients with shock, ScvO₂

is now used more commonly because it only requires placement a central venous catheter, which is more frequently indicated in critically ill patients than pulmonary artery catheters. Regardless of which method is used, the trending of this data can give the nurse valuable information regarding oxygen supply and demand. Decreased values of venous oxygen saturation indicate increased oxygen extraction by the tissues. This may be due to either a decrease in oxygen delivery or an increase in tissue oxygen demands. As the arterial oxygen saturation or hemoglobin level is reduced or the cardiac output decreases, less oxygen returns to the venous system after tissue extraction. This in turn reduces the SvO₂ or ScvO₂. If the hemoglobin, arterial oxygen saturation, and CO remain the same but the tissue demand increases, this will also reduce the SvO₂ and ScvO₂ (Table 5-5). Occasionally, the oxygen supply exceeds the demand of the tissues or the tissue extraction is dysfunctional as in late sepsis. In this case the SvO₂ and ScvO₂ may be elevated.

Muscle Oxygen Saturation

While lactate, SvO₂ and ScvO₂ remain the most commonly used measurements to determine an imbalance of oxygen supply and demand, new technologies are constantly

Table 5-5 Causes of Low Mixed Venous Oxygen Saturation

Cause	Condition
Low CO/CI	• Hypovolemia • Left ventricular failure
Low hemoglobin	• Bleeding • Dyshemoglobinemia (carbon monoxide poisoning, methemoglobinemia)
Low SaO ₂	• Pulmonary disease • Low inspired oxygen
High O ₂ demand	• Elevated metabolic rate (fever, hyperthyroidism, shivering, or seizure activity) • Stressful states (surgery, pain, and trauma)

Bariatric Considerations

In obese patients, an inappropriate blood pressure cuff size can result in a falsely elevated systolic blood pressure, as high as 30 mmHg above the true blood pressure (Palatini & Parati, 2011). Ideally, invasive arterial pressure monitoring is utilized for obese patients who are highly unstable. When that is not practical, a cuff with a bladder that is 80% of the patient’s arm circumference should be chosen or an adult cuff may be used on the patient’s forearm. Alterations in hemodynamics in obese individuals result from the increase in blood

volume required to perfuse their body mass and include increases in cardiac output, right atrial pressure, right ventricular pressure, and pulmonary artery mean and occlusive pressures. The cardiac index is cardiac output adjusted for the patient’s body mass and is a more accurate indication of cardiac performance in the obese patient; however, the obscured vascular anatomy in obese patients can make invasive monitoring devices more challenging, and thus riskier, to place (Alpert et al., 2014).

evolving to evaluate critically ill patients noninvasively. One such technology is **muscle oxygen saturation (SmO₂)**. SmO₂ refers to the amount of oxygen in venous blood after tissue oxygen extraction has occurred, and is measured through a near-infrared spectroscopy sensor placed on the patient's shoulder, thigh, or calf. Originally used for elite athletes to maximize training and for military trauma patients as an early indicator of hemorrhagic shock, SmO₂ now shows promise for medical critical care applications. New research demonstrates that SmO₂ correlates to both ScvO₂ and CO in patients suffering from shock (Bohula May et al., 2014).

Hemodynamics Technology and Nursing Responsibilities

The technologies covered in this section assist the nurse in determining the hemodynamic status of the patient. As with many technologies utilized in healthcare, interpretation of the values must be done in context with the patient's condition and the nurse's physical assessment. Many of the invasive technologies used to assess hemodynamic status rely on pressure monitoring systems to display the data.

Overview of Pressure Monitoring Systems

The basic components of any invasive hemodynamic pressure monitoring system are the flush system, transducer, and monitor. The catheter is attached to the fluid-filled flush system. The mechanical pressure through the catheter is communicated to the transducer. The transducer converts the mechanical pressure into an electrical signal. The monitor converts the electrical signal to a waveform and digital number for the nurse to see (♦ Figure 5-5). Tubing is connected from the catheter to the transducer, and a flush system is attached to a flush solution of normal saline. About 30% of institutions use solutions that also contain heparin; however, recent data demonstrates no difference in catheter patency whether heparinized solutions or normal saline are used (Schallom, Prentice, Sona, Micek, & Skrupky, 2012). Many facilities now utilize normal saline only to decrease the risks associated with heparin, such as bleeding and heparin-induced thrombocytopenia, or HIT. The solution is placed in a pressure bag that is inflated to 300 mmHg. At this pressure, the flush system continuously delivers a small amount of solution (e.g., 3 mL/hr) through the catheter, thus maintaining patency.

Zero and Level the Pressure Monitoring System

In order to obtain accurate readings, the pressure monitoring systems must be zeroed and leveled periodically.

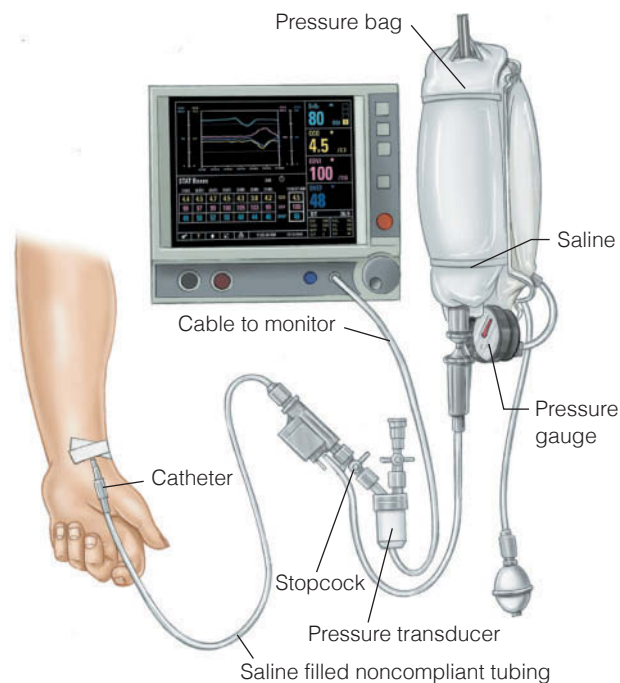


Figure 5-5 Pressure monitoring system. The system consists of a flush system, transducer, and monitor. The flush system is attached to solution in a pressure bag and connected to the patient using noncompliant tubing. The transducer is connected to a monitor via a cable.

The pressure monitoring system must be leveled from the air-fluid interface to the patient's left atrium. Leveling the transducer to the location of the left atrium corrects for changes in the readings due to hydrostatic pressure above and below the heart. The level of the left atrium, in the supine position, is the fourth intercostal space halfway between the anterior-posterior diameter of the chest. This is known as the **phlebostatic axis**. The nurse should mark this site on the chest wall and use a commercial level to properly level the transducer air-fluid interface to the phlebostatic axis. The air-fluid interface on the pressure monitoring system is at the level of the stopcock closest to the transducer (♦ Figure 5-6). Re-leveling is done anytime the patient's position is changed. In some settings, the flush/transducer system is attached directly to the patient at the phlebostatic axis, preventing a need to relevel after a change in the patient's position. As long as the patient is supine, studies have found that measurements are accurate when the head of the bed is elevated between 0 and 60 degrees (AACN, 2009).

Once the system is leveled it can be zeroed. Zeroing is the process of negating the influence of atmospheric air. The zeroing procedure is done by turning the stopcock on the pressure monitoring system off to the patient and open to air. The nurse then activates the zero button on the monitor. Zeroing must be done for each pressure monitoring system used. It is done initially during setup and when the monitoring cable is disconnected from the flush system.

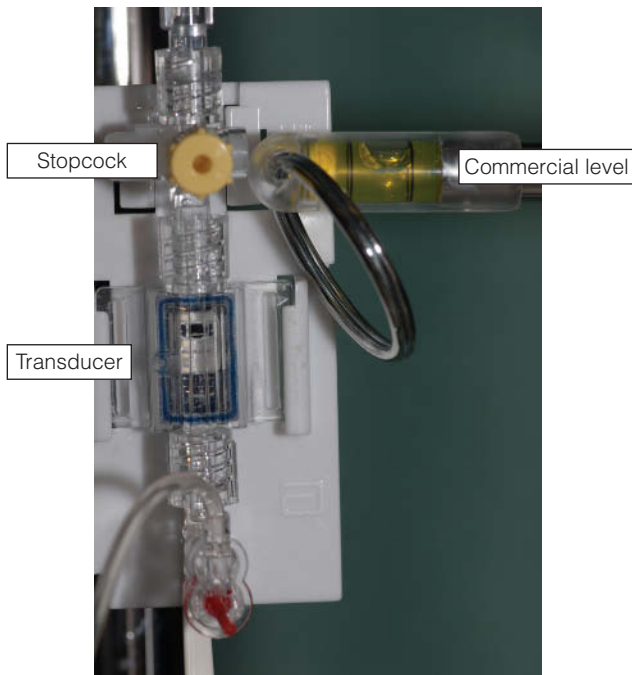


Figure 5-6 The nurse levels the transducer to the phlebostatic axis on the patient and the air-fluid interface. The air-fluid interface on the pressure monitoring system is at the level of the stopcock closest to the transducer.
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Zeroing is also customarily done at the beginning of each shift and when the values do not fit the clinical picture.

The Square Wave Test. The **square wave test** allows the nurse to test the ability of the pressure monitoring system to accurately reflect pressure values. The test is accomplished by activating the fast flush device on the pressure monitoring system and observing the monitor for the resultant pressure waveform (♦ Figure 5-7). The nurse should perform the square wave test on the initial system setup, on every shift, after opening the system (i.e., when drawing blood), and when the values are suspected to be inaccurate (AACN, 2009).

Inaccurate values in pressure monitoring systems can be a result of an overdampened (pressures are underestimated) or underdampened (pressures are overestimated) system. Factors that can cause overdampened values are catheter patency, air bubbles or blood in the tubing, and kinks in the tubing. Factors that can cause underdampened values are excessive tubing length and use of multiple stopcocks (Table 5-6).

Arterial Pressure Monitoring

Invasive arterial pressure monitoring is used in situations where blood pressure must be continuously monitored, as occurs with a hemodynamically unstable patient or one who is receiving vasoactive intravenous medications such



Pulling this fast flush device results in the square wave waveform

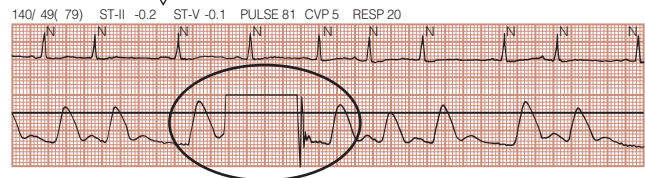


Figure 5-7 Square wave test. By activating the fast flush device, a square wave is displayed on the monitor. The waveform should have a sharp upstroke, a plateau, and a rapid downstroke extending below the baseline followed by one or two small waveforms lasting no more than 0.12 second.

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as vasopressors or vasodilators. The placement of an arterial line, or a-line, also provides the opportunity to easily sample arterial blood for arterial blood gas analysis.

Insertion and Management

An arterial catheter for blood pressure monitoring can be placed in the radial, femoral, brachial, axillary, or dorsalis pedis arteries. Radial and femoral arteries are most commonly used, and radial arteries are preferred due to decreased risk of infection and complications. The dorsalis pedis artery is used infrequently due to its distance from central circulation, which can make pressure readings less accurate. Prior to inserting a catheter in the radial artery, an Allen's test should be performed to test the patency of the radial and ulnar arteries in the event that the radial artery becomes occluded (♦ Figure 5-8). To perform the test, the patient opens and closes his or her fist while the radial and ulnar arteries are compressed. The patient then opens his

Table 5-6 Troubleshooting Overdamped and Underdamped Pressure Monitoring Systems

	Cause	Nurse Corrective Action
Overdamped waveforms	Reduced catheter patency	• Ensure the pressure bag is inflated to 300 mmHg to maintain continuous flow of saline solution through the catheter. • Ensure there is adequate saline solution in the flush bag. • Inspect for kinks in the tubing preventing the flush solution from reaching the catheter. • Heparinize the solution per institution policy.
	Air bubbles	• Ensure air is removed from the flush system on initial setup.
	Blood in the tubing	• Ensure the pressure bag is inflated to 300 mmHg because a reduction in inflated pressure can cause blood to back up in the tubing. • Ensure that there is adequate saline solution in the flush bag. • Fully clear the pressure system after blood is drawn from the line.
	Kinks in the tubing	• Inspect the tubing periodically to ensure that no kinks are present.
Underdamped waveforms	Incorrect pressure scale on the monitor	• Use of a pressure scale on the monitor significantly higher than the actual monitored pressure can cause the waveform to appear overdamped.
	Excessive tubing length	• Do not add tubing to the premanufactured flush system.
	Use of multiple stopcocks	• Utilize stopcocks available with the premanufactured flush system. Reduce the number added to the system.

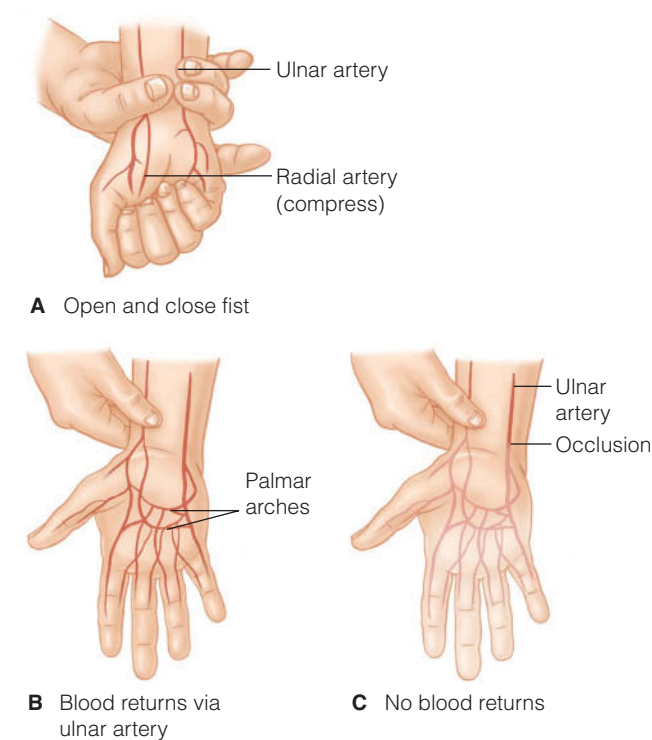


Figure 5-8 Allen's test. *A*, Compress both the radial and ulnar arteries and have the patient open and close the fist. *B*, Release the ulnar artery compression and blood should return to the hand. *C*, Ulnar artery compression is released and no blood returns. Occlusion in the ulnar artery. Do not use this radial artery for arterial line insertion.

or her hand, which will appear blanched. Ulnar compression is then released, and the hand is observed for return of color. If color returns in 5 seconds or less, the test is considered positive and the radial artery may be used for catheter placement. If restoration of perfusion to the hand with release of the ulnar artery takes longer than 5 seconds, the

test is negative and the radial artery should not be used since this indicates that the ulnar artery does not have sufficient blood flow to supply the hand in the event of a radial artery occlusion.

Once a location for the arterial line is safely chosen and the catheter is placed and attached to a pressure monitoring system, a waveform and digital value will be displayed on a monitor.

Waveform Interpretation

The arterial waveform has a rapid upstroke as ventricular ejection occurs and the aortic pressure rises (♦ Figure 5-9). The peak of the waveform is the systolic pressure. The ventricular pressure then falls to a point below the aortic pressure and the aortic valve closes. This point is called the **dicrotic notch**. The dicrotic notch signals the end of systole. The pressure continues to fall to the lowest pressure on the waveform, which is the diastolic pressure.

The average pressure during the entire cardiac cycle is the MAP. Identification of the dicrotic notch is one indication that the waveform is accurate. As the waveform becomes overdamped, the dicrotic notch is lost. A waveform that appears overdamped can be a problem with the pressure monitor system or a hypotensive episode in the

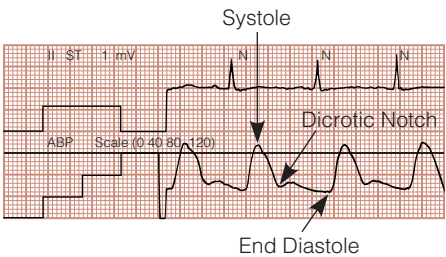


Figure 5-9 Arterial waveform.

(Courtesy of Elliot Hospital, Manchester, NH.)

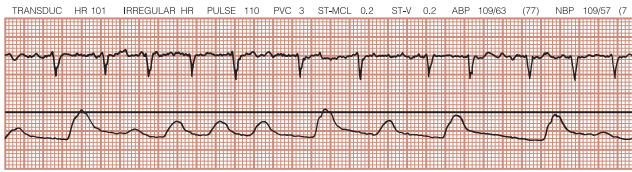


Figure 5-10 Effect of atrial fibrillation on the arterial waveform. Note the varying arterial pressures due to erratic cardiac filling and rhythm irregularity.

(Courtesy of Elliot Hospital, Manchester, NH.)

patient. The nurse should immediately evaluate the arterial system for accuracy and perform a square wave test. The nurse can easily identify the effects of dysrhythmias on blood pressure by observing the simultaneous invasive arterial pressure waveforms (♦ Figure 5-10).

NURSING ACTIONS

The arterial system should be routinely checked to ensure that the connections are tight, that sterile nonvented caps are present on the flush system, that the pressure bag is at 300 mmHg, and that there is an adequate volume of solution in the flush system. The nurse should perform a neurovascular assessment in the cannulated extremity immediately after insertion and periodically according to institution protocol. The monitor pressure alarm should be activated at all times to alert the nurse of changes in hemodynamic stability or unintentional dislodgment, which could result in hemorrhage. The nurse should observe the insertion site for signs of infection. Periodic dressing changes, tubing flushes, and solution changes, as dictated by institution protocol, all help to prevent infection.

Essential for Safety

The monitor pressure alarm should be activated at all times to alert the nurse of changes in hemodynamic stability or unintentional dislodgment.

Complications

Complications of arterial pressure monitoring include:

- Hemorrhage from the site of insertion
The nurse should apply direct pressure and notify the provider.
- Thrombosis in the cannula
A thrombosis in the cannula is usually suspected when the waveform is damped. The nurse should aspirate and discard blood (and the thrombus) from the cannula, then fast flush to clear the tubing of blood. If the thrombus cannot be aspirated, the catheter will need to be removed.

- Tissue ischemia in the cannulated extremity

Notify the provider. The nurse should continue to assess for worsening of the ischemia indicated by delayed capillary refill, pallor or cyanosis, reduction in pulses distal to the cannula, and cool temperature.

- Nerve injury

This complication can occur with any arterial catheter site; however, the consequences are more severe if a more central location is chosen. For example, radial nerve damage may result in a carpal-tunnel syndrome, whereas axillary nerve damage may result in brachial nerve plexus injury and distal paresthesia.

- Infection

Notify the provider. The nurse should anticipate that the provider will discontinue the infected cannula and should be prepared for an arterial insertion in another location.

Central Venous Pressure Monitoring

The **central venous pressure (CVP)** or the **right atrial pressure (RAP)** measures the right-sided preload also known as the right filling pressures. CVP values are helpful in determining volume status, pulmonary status (e.g., pulmonary hypertension or pulmonary embolism), right-sided cardiac function, and medication effects, particularly those that decrease preload such as nitroglycerin.

Insertion and Management

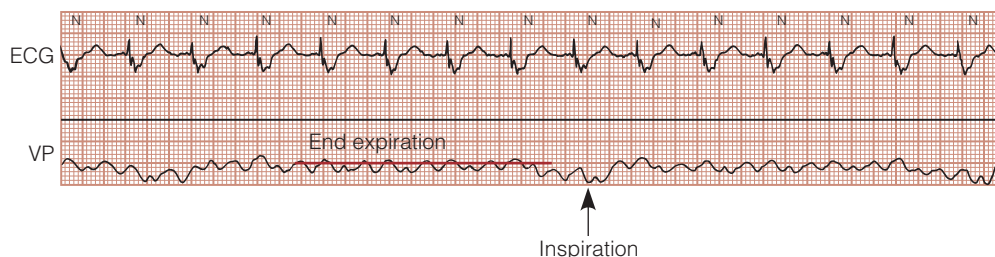
A central venous catheter is placed within a central vein near the right atrium, such as the internal jugular or subclavian vein. Once the catheter is connected to a monitoring device, a CVP value is obtained. If a pulmonary artery (PA) catheter is in place, the RAP is obtained from the proximal port of the catheter (see discussion of PA catheters in the section “Pulmonary Artery Pressure Monitoring”). At most institutions, the CVP value is obtained through a pressure monitoring system; a manometer is used with less frequency.

Waveform Interpretation

After ensuring that the system is leveled and zeroed, the nurse must measure the CVP or RAP at the end-expiration in the patient’s breathing cycle. It is at this point that the effects of intrathoracic pressure variations during respiration are minimized. Because the digital readouts on the monitor reflect pressures throughout respiration, a graphic tracing will allow more accurate identification of end-expiration pressures. (♦ Figure 5-11).

Figure 5-11 CVP tracing in a patient who is spontaneously breathing. Read the CVP value at end exhalation.

(Courtesy of Elliot Hospital, Manchester, NH.)



Normal CVP/RAP are approximately 2 to 6 mmHg (mean). CVP/RAP that are lower than normal usually indicate volume deficit or venodilation. Conditions causing elevation of the CVP/RAP include:

- Volume excess
- Right ventricular failure
- Pulmonary hypertension
- Tricuspid stenosis or insufficiency
- Pulmonic stenosis or insufficiency
- Pulmonary embolism
- Mechanical ventilation, especially when high PEEP (positive end-expiratory pressure is used)

NURSING ACTIONS

The nurse should record the CVP/RAP values hourly or more frequently based on patient condition. As with all hemodynamic values, the trending of the values over time is most valuable. The CVP system should be routinely checked to ensure that the connections are tight, that sterile nonvented caps are present on the flush system, that the

pressure bag is at 300 mmHg, and that there is an adequate volume of solution in the flush system. The monitor pressure alarm should be activated at all times to alert the nurse of changes in hemodynamic stability or unintentional dislodgment. The nurse should observe the insertion site for signs of infection. Periodic dressing changes, tubing flushes, and solution changes should occur per institution protocol to prevent infection. A daily review to assess whether or not the central venous catheter is still indicated should be performed by the nurse in conjunction with the interdisciplinary team.

Complications

Complications for the CVP/RAP measurements are primarily related to central line placement:

- Pneumothorax/hemothorax

Signs and symptoms of pneumothorax/hemothorax include dyspnea, tachypnea, tachycardia, decreased breath sounds on the affected side, possible hypotension, decreased oxygen saturation, and possible tracheal deviation away from the affected side. The nurse should notify the provider, provide supplementary oxygen, and

Safety Initiatives

Prevent Central Line Infections

PURPOSE: To prevent catheter-related bloodstream infections by implementing the five components of care called the “central line bundle.”

RATIONALE: Central lines (catheters that terminate in great vessels) are used frequently in hospitalized patients. Numbers vary considerably among different intensive care units (ICUs) and specialties, but it is estimated that approximately 43% to 80% of critically ill patients have a central line at some point during their hospitalization (Ricard et al., 2013). This is significant considering one of the most expensive and deadly healthcare-associated infections in the United States is the central line-associated blood stream infection, or CLABSI (Institute for Healthcare Improvement, 2016). These infections are particularly dangerous because they may cause hemodynamic changes, organ dysfunction, and even death. It is estimated that about 62,000 deaths occur annually due to central line infections (WHO, 2016).

Therefore, the institution of a group of best practices, known as a central line bundle, is recommended to prevent central line infections.

HIGHLIGHTS OF RECOMMENDATIONS: The central line bundle has several key components:

- Hand hygiene
- Maximal barrier precautions on line insertion
- Chlorhexidine skin asepsis
- Daily review of line necessity with prompt removal of unnecessary lines

The site selected for central venous access has long been considered in the analysis of infection rates, with femoral lines being the site at highest risk for CLABSI. More recent literature supports no statistically significant difference in infection rates among the three major central venous access sites: internal jugular, subclavian, and femoral (Marik, Flemmer, & Harrison, 2012).

prepare for assisting the provider with a needle decompression or chest tube insertion. In severe cases, an untreated pneumothorax or hemothorax can result in cardiac arrest.

- Air embolism

If an air embolism is suspected, the patient should be immediately placed in a Trendelenburg position on their left side to trap the air in the apex of the heart. Notify the provider immediately.

- Laceration of major vessels

The nurse may suspect laceration of major vessels if a hematoma/swelling occurs around the site of insertion. Apply direct pressure and notify the provider. Closely monitor blood pressure, heart rate, respiratory rate, and pulse oximetry.

- Catheter malposition possibly leading to dysrhythmias

The nurse and the provider should review the chest x-ray to evaluate the placement of the catheter. The provider will reposition the catheter if necessary. If dysrhythmias occur, notify the provider and treat the dysrhythmias according to standing orders if available.

- Thrombus formation occurring at the tip of the catheter

A thrombus in the cannula is usually suspected when the waveform is damped. The nurse should aspirate and discard blood (and the thrombus) from the cannula, then fast flush to clear the tubing of blood. If the thrombus cannot be aspirated, the catheter will need to be removed.

Pulmonary Artery Pressure Monitoring

The PA catheter is used to:

- Provide support for a diagnosis, such as heart failure in a hemodynamically unstable patient
- Guide therapy such as volume repletion, or monitor for medication effects of vasopressors, inotropes and vasodilators
- Monitor cardiac function in patients undergoing high-risk procedures, such as cardiac surgery or repair of aortic aneurysm.
- Assess cardiac output and the balance of oxygen supply and demand, such as with SvO_2

Catheters

The standard PA catheter is 7.5 Fr and 110 cm long. It has four ports, and when inserted into the heart, it has an opening in the right atrium that corresponds to the proximal port, an opening in the pulmonary artery that corresponds to the distal port, a port for inflating a balloon at the distal

tip, and a thermistor port for obtaining a cardiac output. Additional ports are available in some catheters to provide supplemental volume or medication administration.

Insertion and Management

Prior to PA catheter insertion, an introducer is placed through the internal jugular, subclavian, or femoral vein by a physician or advanced practice nurse. The PA catheter is then threaded through the introducer and into the right atrium. Once the catheter reaches the right atrium, the balloon at the distal tip is inflated and the catheter is advanced over the tricuspid valve into the right ventricle, then through the pulmonary valve into the pulmonary artery. The catheter remains in this position for the routine monitoring of PA waveforms and pressures (♦ Figure 5-12). During insertion, the ECG must be continuously displayed and the nurse must monitor the ECG to identify any dysrhythmias. Some providers utilize fluoroscopy for guidance when placing PA catheters.

Waveform Interpretation

The following pressures and waveforms can be obtained from the PA catheter: right atrial (RA), PA systolic (PAS), PA diastolic (PAD), PA mean, and **pulmonary capillary wedge pressure (PCWP)**. After ensuring that the system is leveled and zeroed, the measurement of the PA pressures must be done at end-expiration in the patient's breathing cycle as described in the section on CVP.

Waveform Interpretation During Pulmonary Artery Catheter Insertion

Insertion of the PA catheter is guided by waveform analysis. (♦ Figure 5-13).

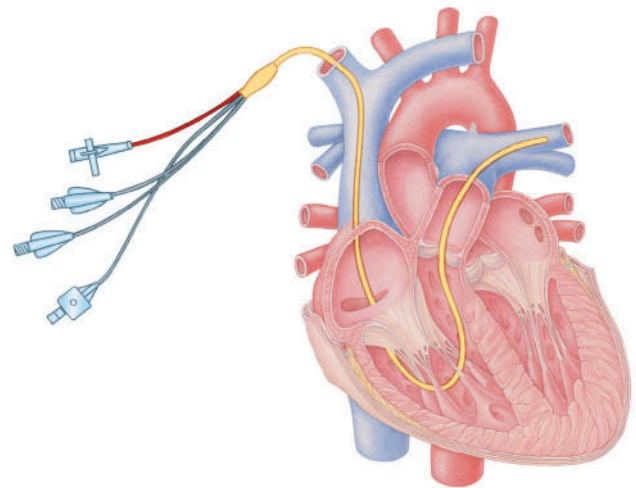


Figure 5-12 Pulmonary artery catheter positioned in the heart. The proximal port communicates with an opening in the right atrium for RA pressures. The distal port communicates with an opening at the end of the catheter for pulmonary artery pressure. The balloon at the end of the catheter is inflated for PCWPs.

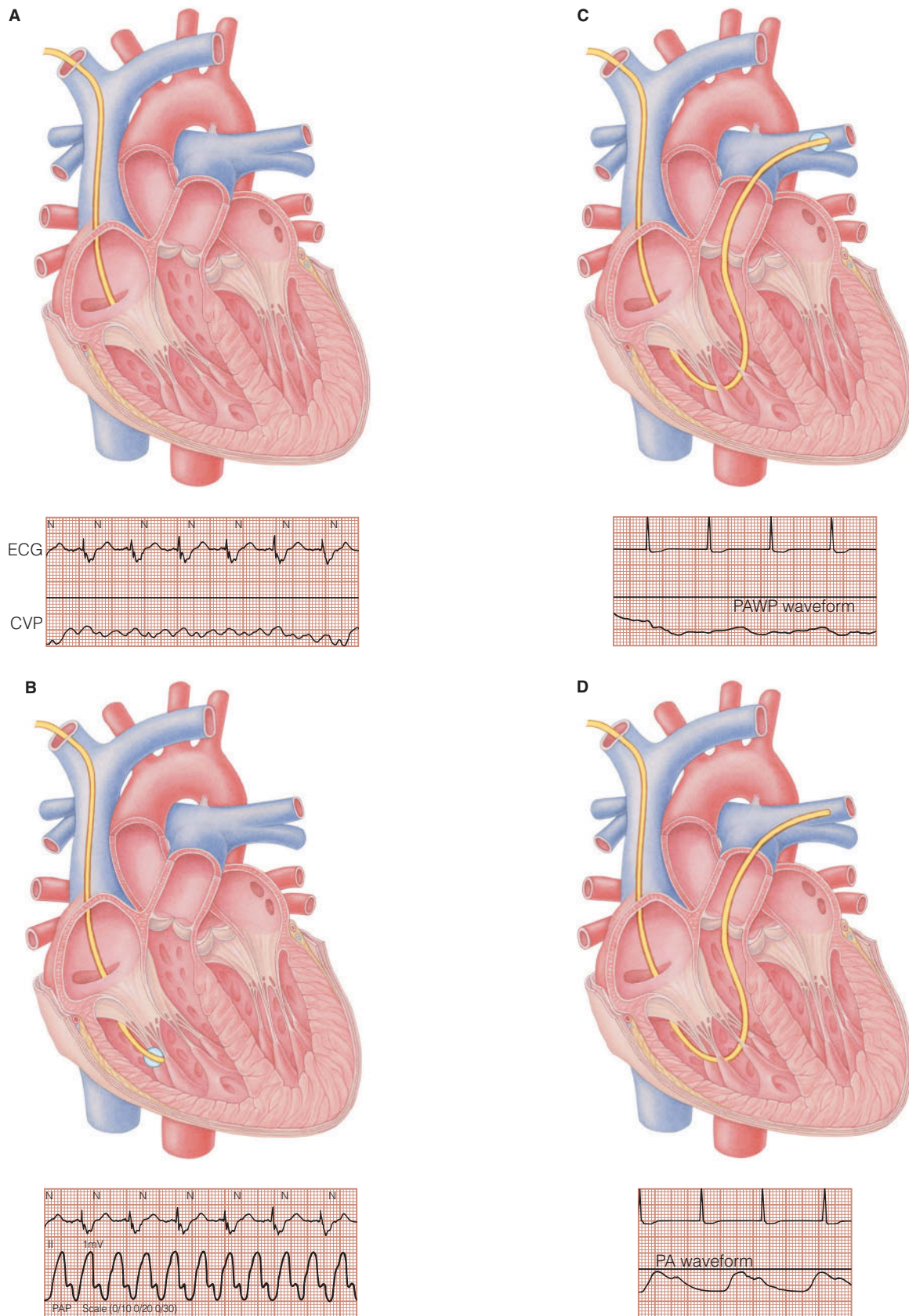


Figure 5-13 Insertion of the pulmonary artery catheter. *A*, During insertion the catheter is first positioned in the right atrium, displaying an RA pressure tracing. *B*, The distal balloon is inflated and the catheter is advanced into the right ventricle, displaying the RV pressure tracing. Note the elevation in systolic pressure. *C*, The catheter is advanced into the pulmonary artery where the distal balloon is wedged in a capillary, displaying the PCWP tracing. *D*, The balloon is deflated, displaying the PA tracing. Note the dicrotic notch.

As the catheter is advanced from the central vessel into the right atrium, the balloon is inflated to facilitate movement through the heart, allowing the catheter to float along the same pathway as blood moving through the heart. In the right atrium, the RAP is obtained. The normal RAP value is 2 to 6 mmHg. Conditions causing elevated or decreased RAPs are found under the CVP section.

As the catheter is advanced over the tricuspid valve, there is a marked increase in systolic pressure. The nurse must pay close attention to the ECG monitor during the passing of the catheter through the right ventricle (RV), as ventricular dysrhythmias, such as premature ventricular contractions or ventricular tachycardia, can occur. Normal RV pressure is 15 to 25 mmHg (systolic) and 0 to 8 mmHg (diastolic). The RV pressure is usually obtained only during insertion of the catheter. A low RV pressure most often indicates hypovolemia. Conditions causing elevation of RV pressures include:

- Pulmonary hypertension
- Pulmonic valve stenosis
- Increased pulmonary vascular resistance
- Volume excess
- Cardiac tamponade
- Right heart failure

Once the catheter leaves the RV it is advanced across the pulmonary valve to the PA. The PA waveform has a dicrotic notch that identifies the closure of the pulmonary valve. The systolic pressure is fairly consistent with the RV systolic pressure; however, the diastolic pressure increases once the catheter is in the PA. Normal PA pressure value is 15 to 25 mmHg (systolic), 5 to 15 mmHg (diastolic), 9 to 16 mmHg (mean). Conditions causing elevation of PA pressures include:

- Increased pulmonary vascular resistance
- Pulmonary hypertension
- Mitral stenosis or insufficiency
- Left heart failure
- Volume excess

Conditions causing decreased PA pressure include:

- Volume deficit
- Pulmonic stenosis
- Tricuspid stenosis

Pulmonary Capillary Wedge Pressure

During insertion, the catheter can be advanced farther into the PA until the PCWP is obtained. With the balloon inflated, the catheter floats into, or wedges, in a smaller pulmonary arteriole or capillary and temporarily stops forward blood flow in that area. When the balloon is inflated,



Figure 5-14 When the distal balloon is inflated the catheter is wedged in a small pulmonary capillary, stopping forward flow. The opening at the distal tip (see insert) “looks” forward through the pulmonary circulation and the pulmonary veins and into the left atrium. If the mitral valve is normal, left ventricular end-diastolic pressures can be obtained.

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the opening at the end of the catheter looks through the pulmonary circulation into the pulmonary veins and into the left atrium (♦ Figure 5-14).

The waveform obtained when the balloon is inflated is the PCWP or pulmonary artery occlusion pressure (PAOP). This pressure reflects the left atrial pressure. During diastole, when the normal mitral valve is open, the PCWP also reflects the left ventricular end-diastolic pressure. Thus, the PCWP gives the nurse an estimate of the preload of the left ventricle. Once the PCWP is obtained, the position of the catheter is confirmed by deflating the balloon and observing a PA tracing. Normal PCWP value is 4 to 12 mmHg (mean). When a PCWP is lower than normal, it usually indicates volume deficit. Conditions causing elevation of PCWP pressures include:

- Volume excess
- Left ventricular failure
- Mitral stenosis or insufficiency
- Aortic stenosis or insufficiency

See Table 5-7 for normal values of the PA catheter pressures.

Table 5-7 Pulmonary Artery Catheter Pressures—Normal Values

Pressure	Normal Value
RA	2–6 mmHg
RV systolic/diastolic	15–25 mmHg/0–8 mmHg
PA systolic/diastolic	15–25 mmHg/5–15 mmHg
PA mean	9–16 mmHg
PCWP	4–12 mmHg

Waveform Interpretation and Management

Right Atrial Pressure

The RAP is obtained from the proximal port of the PA catheter. Per institution policy, the waveform and pressure may be continuously monitored or monitored periodically.

Right Ventricular Pressure

The right ventricular (RV) pressure is not routinely monitored. It is important for the nurse to know the features of the RV waveform because, occasionally, the catheter can slip from the PA back to the RV. The hallmarks of the RV pressure include the lack of a dicrotic notch and a low diastolic pressure. Prompt identification of potential catheter migration is important to reduce the time the catheter spends in the RV, as it can potentially cause a ventricular dysrhythmia. The procedure for advancement of the catheter back into the PA depends on institution policy. In many institutions, the nurse must notify the physician or advanced practice nurse to advance the catheter back to the PA. Some institutions allow the bedside nurse to inflate the balloon, utilizing the blood flow within the heart to float the catheter back into the PA.

Pulmonary Artery Pressures

The PA pressures, including the PA systolic, PA diastolic, and mean are obtained from the distal port of the PA catheter and routinely documented.

Pulmonary capillary wedge pressure

The PCWP is obtained by inflating the balloon, and should be performed according to institution policy or at the frequency ordered by the physician or advanced practice nurse. The balloon must be inflated using only the syringe that is provided by the manufacturer. This unique syringe has a stop on it, preventing inflation of more than 1.5 mL of air. Overinflation of the PA balloon can result in inaccurate values, and more importantly, can cause pulmonary capillary or pulmonary artery rupture, which can be fatal. The nurse should slowly inflate the balloon with the air until the PA waveform changes to a PCWP (♦ Figure 5-15). Slight resistance should be felt as the

balloon is inflated. The nurse uses only the volume of air necessary to obtain a waveform change. The balloon is kept inflated for no more than 15 seconds because longer inflation times may also lead to pulmonary infarction and rupture. Once a PCWP is obtained, the nurse allows the air to passively escape from the balloon, then disconnects the syringe and removes the air. Lastly, the nurse places the syringe back on the inflation port and closes the stopcock on the balloon inflation valve, preventing inadvertent inflation of the balloon.

Essential for Safety

The balloon is kept inflated for no more than 15 seconds.

Pulmonary Artery Diastolic

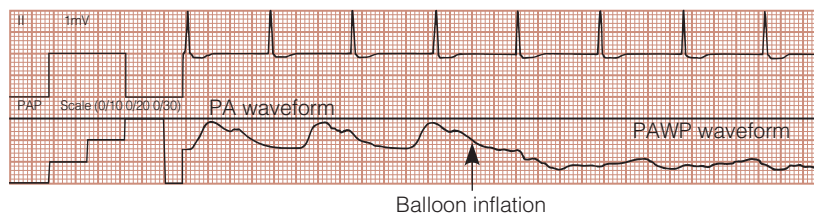
The pulmonary artery diastolic (PAD) pressure can be used to estimate the left ventricular end-diastolic pressure. In most patients, the PCWP is about 1–4 mmHg less than the PAD. When this correlation exists, the PAD can be monitored as an estimate of left ventricular end-diastolic pressure when a PCWP is not done. The presence of pulmonary embolism, pulmonary hypertension, and severe COPD may result in a greater disparity between the PAD and PCWP. In these patients, the PAD cannot be used as a surrogate for PCWP measurements.

Occasionally, the PA catheter cannot be wedged. This may occur if the catheter has migrated back into the pulmonary artery, in which case the physician or advanced practice nurse should be notified to advance the catheter. If mild resistance during inflation is not felt, the balloon may be ruptured. If balloon rupture is suspected, the nurse should (a) notify the physician or advanced practice nurse, (b) tape the balloon inflation port close, (c) label the port to identify that the port should not be used.

Sometimes the catheter independently migrates to the PCWP position. The nurse must closely monitor the patient with a PA catheter to identify such complications quickly. When a continuously wedged waveform is seen, the nurse should ensure that all of the air is out of the syringe, and then the nurse should try repositioning the patient. The PA catheter should never be flushed when the catheter is stuck in wedge because this can lead to PA rupture. The physician or advanced practice nurse should be notified immediately to reposition the catheter back into the PA. Some institutions

Figure 5-15 Pulmonary artery pressure to PCWP pressure with balloon inflation.

(Courtesy of Elliot Hospital, Manchester, NH.)



have policies that allow the bedside nurse to withdraw the PA catheter until the PA waveform appears.

NURSING ACTIONS

During insertion, the nurse assists the physician or advanced nurse practitioner by priming the pressure monitoring tubing, preparing the insertion kits, connecting the system to appropriate solutions and pressure cables, leveling and zeroing the system, placing a sterile occlusive dressing on the insertion site, documenting the waveforms and values, and setting the appropriate alarms. The nurse should ensure that a chest x-ray has been done to verify catheter placement.

The nurse should record the RA, PA systolic, PA diastolic, and PA mean values hourly or more frequently based on patient condition. As with all hemodynamic values, the trending of the values is most valuable. The PA system should be routinely checked to ensure that the connections are tight, that sterile nonvented caps are present on the flush system, that the pressure bag is at 300 mmHg, and that there is solution in the flush system. The PA pressure alarms should be activated at all times to alert the nurse of changes in hemodynamic stability or unintentional dislodgment. Periodic dressing changes, tubing flushes, and solution changes performed per hospital protocol can prevent infection. The PA pressures should be continuously displayed on the monitor (bedside and central). The nurse is responsible for monitoring the PA waveform to ensure that it does not migrate into the PCWP position.

Essential for Safety

The PA pressures should be continuously displayed on the monitor.

Complications

The nurse should be aware of complications from the insertion or use of the PA catheter, including:

- Ventricular dysrhythmias
The provider should be notified immediately. If the patient is unstable, administer antiarrhythmics per standing orders if available.
- Pulmonary capillary rupture, pulmonary infarction, and pulmonary artery rupture.
Signs and symptoms include blood-tinged or frank bloody sputum, decreased SpO₂, and possible respiratory distress. Notify the provider immediately. The nurse should provide supplementary oxygen and be prepared to support ventilation if necessary. Pulmonary artery rupture is a surgical emergency and can quickly result in death.

- Pneumothorax/hemothorax

Signs and symptoms of pneumothorax/hemothorax include dyspnea, tachypnea, tachycardia, decreased breath sounds on the affected side, possible hypotension, decreased oxygen saturation, and possible tracheal deviation away from the affected side. The nurse should notify the provider, provide supplementary oxygen, and prepare to assist the provider with a needle decompression or chest tube insertion.

- Infection

Notify the provider. The nurse should anticipate that the provider will discontinue the infected cannula and should be prepared for a catheter insertion in another location.

- Hematoma

The nurse may suspect laceration of major vessels if a hematoma/swelling occurs around the site of insertion. Apply direct pressure and notify the provider. Closely monitor blood pressure, heart rate, respiratory rate, and pulse oximetry.

- PA catheter knotting

If the PA catheter is knotted, the nurse should never attempt to remove the PA catheter. Notify the provider.

Cardiac Output Measurement

Cardiac output measurements are used to assess the hemodynamic function of the patient. They are also used to guide therapy and evaluate patient responses to pharmacological interventions and mechanical ventilation.

Cardiac Output Measurement—Thermodilution Method

The thermodilution method of obtaining cardiac output measurements relies on a volume (usually 5 to 10 mL) being injected through the proximal port of the PA catheter. That volume undergoes a temperature change as it moves through the RV and into the PA. This temperature change is picked up by the thermistor on the end of the PA catheter and is converted to a temperature-time curve on the monitor. The monitor calculates a CO from that curve. The CO measurement can have significant variations depending on the rate and smoothness of the injection. Therefore, between three and five injections are necessary to reliably select several values to average for the CO. Room temperature injectates can be used in most situations because research has proven that they reliably measure CO. The nurse should be aware that the injectate fluid may need to be replaced if erroneous values are being calculated, particularly if a warming lamp or other device is being used for the patient that could alter the temperature of the fluid.

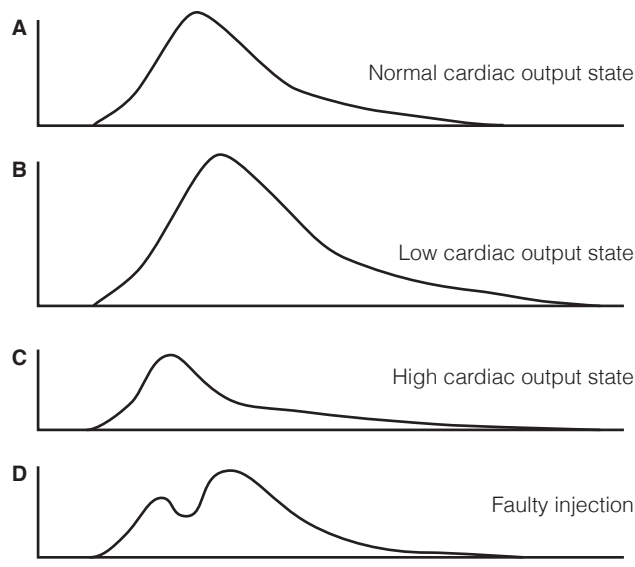


Figure 5-16 Thermodilution cardiac output curves. A, Normal curves showing a smooth injection technique. B, An abnormal curve showing low cardiac output C An abnormal curve showing high cardiac output D An abnormal curve caused by erratic injection.

Substitute for Figure 5-19 (Hollenberg, 2015)

Cardiac Output Curve Interpretation

The CO curve should have a smooth upstroke followed by a gradual downslope to the baseline. Common errors with the curve are uneven upstrokes and long injection times (♦ Figure 5-16).

Derived Hemodynamic Values

Once the CO value is obtained, several other derived hemodynamic values can be calculated. The CI and SV are commonly calculated. Indicators of afterload such as SVR and PVR as well as indicators of contractility such as stroke work index can also be calculated. Commonly, the bedside monitor system performs these calculations.

NURSING ACTIONS

Prior to the beginning of the procedure, the nurse must ensure that the computation constant entered into the bedside monitor is appropriate for the catheter size and volume of injectate. Any discrepancy in this value can lead to inaccurate readings and erroneous calculations. The nurse should inject at end-expiration to limit variability in CO measurements related to respiratory cycle. Injections should be done smoothly and as fast as possible, over less than 4 seconds. It is important to maintain the patency of the proximal port through a continuous infusion, because a clotted proximal port renders the catheter of no use for thermodilution CO measurement. Toward that end, at the completion of the CO procedure, the nurse should return the stopcock of the proximal port to its original position and ensure that infusions are flowing through the proximal port.

Alternatives to Thermodilution Cardiac Output Monitoring

As data continues to demonstrate that pulmonary artery catheter use does not positively influence patient outcomes, their use has steadily decreased. Only about 6.2% of patients in ICUs have a PA catheter, and the majority of these are surgical patients (Gershengorn & Wunsch, 2013). Minimally invasive and noninvasive methods of determining cardiac output have grown in popularity over recent years, and include pulse contour analysis and doppler-based monitoring.

Arterial Pressure-Based Cardiac Output Monitoring

This system of pulse contour analysis continuously estimates CO and stroke volume (SV) by analyzing the arterial pressure waveform obtained from an arterial line. Pulse contour analysis devices can provide stroke volume variation (SVV), and pulse pressure variation (PPV) can also be obtained by some bedside monitoring systems. SVV and PPV are thought to be better predictors of fluid responsiveness than CVP monitoring alone. SVV and PPV work on the principle that cardiac preload is influenced by changes in intrathoracic pressure created by mechanical ventilation. These changes are more pronounced in hypovolemia as variations increase in the systolic arterial pressure (PPV) and in the pulse contour analysis (SVV). An optimal arterial waveform is essential for the accuracy of these types of device. Unfortunately, arterial pressure-based monitoring devices cannot be broadly used in the critical care setting because they require very specific clinical parameters. The patient must be mechanically ventilated with minimal spontaneous breaths, and have a tidal volume of at least 8 mL/kg, which is higher than recommended for some patient populations at risk for lung injury from mechanical ventilation. These devices also necessitate that the patient have an arterial line and central venous catheter. Presently, arterial pressure-based cardiac output monitoring has limited reliability in patients who are hemodynamically unstable, which is often the population of patients that most benefits from such monitoring. They also cannot be used in patients with dysrhythmias, such as atrial fibrillation, or those with aortic insufficiency as this alters the shape of the arterial waveform. (Thiele, Bartels, & Gan, 2015).

Doppler-Based Monitoring

Minimally invasive or noninvasive CO monitoring can be estimated via transthoracic or esophageal ultrasound devices. Transthoracic devices use information from the pulmonary and aortic valves to intermittently provide CO. In esophageal Doppler monitoring, a small transducer probe is placed into the esophagus via the nose or mouth. This probe transmits and receives a signal that picks up the velocity and

distance of the blood flow in the descending aorta to continuously estimate CO. The esophageal Doppler cannot directly measure the cardiac output because about 30% of blood flow is diverted prior to reaching the descending thoracic aorta.

Research suggests that this form of measuring CO is slightly less accurate than thermodilution technique; however, its major advantage is being low risk and noninvasive. (Prekker, Scott, Hart, Sprenkle, & Leatherman, 2013).

Cardiodynamic Summary

Understanding cardiodynamics and hemodynamic regulation helps the nurse and other members of the healthcare team to determine the status of the patient. The technologies

described in this section must be utilized carefully. The values they supply should be interpreted in conjunction with an accurate and thorough assessment of the patient.

Why/Why Not?

A patient is admitted to the intensive care unit from the cardiology clinic with acute decompensated heart failure. He was unable to fill his prescription for lasix, and now has gained approximately 5.0 kg over the last 4 days. He is also having difficulty breathing; his SpO₂ is 84% and his wife states that

he has had to sleep in a chair all week because he cannot breathe while lying flat. The team is discussing whether or not to place a PA catheter for monitoring. Why or why not?

See answers to Why/Why Not? in the Answer Section.

Case Study

A 73-year-old male was seen in the emergency department for abdominal pain. The following day he goes to surgery for a laparoscopic cholecystectomy. In the recovery room he develops pulmonary edema secondary to an intraoperative non-ST elevated myocardial infarction. He is transferred to the ICU on a mechanical ventilator. The patient is receiving diuretics to manage pulmonary edema and dopamine 5 mcg/kg/min to support blood pressure. The ejection fraction is 20%. He has a history of diabetes, chronic renal failure, hypertension, hyperlipidemia, and coronary bypass surgery. A PA catheter is placed in the ICU.

1. What are the indications for placement of the PA catheter in this patient?

The following waveform is obtained from the distal port of the PA catheter (♦ Figure 5-17).

2. Identify the waveform. Identify the dicrotic notch.

You enter the patient's room and notice that the PA waveform is no longer on the screen. Instead, the following waveform is on the monitor (♦ Figure 5-18).

3. Identify the waveform.
4. Explain how the waveform appeared without any manipulation by the nurse.
5. Identify the corrective action by the nurse.

A thermodilution cardiac output is done and hemodynamic values are calculated.

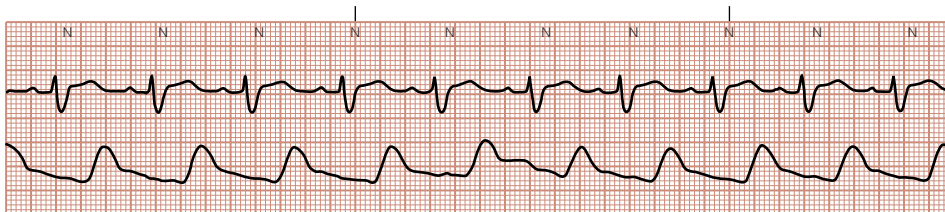


Figure 5-17 PA catheter waveform.

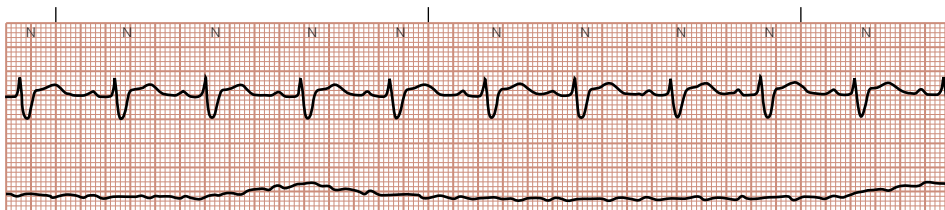


Figure 5-18 PA catheter waveform.

CI: 1.66 L/min/m²
 SVI: 15.8 mL/m²
 RA: 12 mmHg
 PCWP: 38 mmHg
 SVRI: 3,951 dynes/s/cm⁻⁵
 PVRI: 337 dynes/s/cm⁻⁵

6. Which two parameters are used to analyze the patient's preload? Evaluate this patient's preload.
7. Which two parameters are used to analyze the patient's afterload? Evaluate this patient's afterload.

It is always important to assess the balance of oxygen supply and demand to the tissues. The patient's current arterial blood gas on FiO₂ of 0.40 is pH 7.45/pO₂ 86/pCO₂ 38/Bicarb 26. The ScVO₂ is 54%.

8. Evaluate the oxygen supply and demand to the tissues.

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 5.1 What are the four determinants of cardiac output?
- 5.2 What is the relationship between preload and contractility?
- 5.3 Upon entering a patient's room the nurse notices blood backing up in the tubing leading from the arterial catheter to the flush/transducer system. What are

- the nursing actions to correct this? Will this cause the arterial waveform to be overdamped or underdamped?
- 5.4 What conditions would cause an increase in RA pressure?

See answers to Chapter Review Questions in the Answer Section.

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Chapter 6

Care of the Patient with Acute Coronary Syndrome

Carrie Edgerly MacLeod, PhD, APRN-BC

Abbreviations

ACS	Acute Coronary Syndrome	NSTEMI	Non-ST-Segment Elevation Myocardial Infarction
CABG	Coronary Artery Bypass Graft	PTCI	Percutaneous Transluminal Coronary Intervention
CAD	Coronary Artery Disease	PTCA	Percutaneous Transluminal Coronary Angioplasty
CK-MB	Creatine phosphokinase–myocardial band	STEMI	ST-Segment Elevation Myocardial Infarction
DAPT	Dual antiplatelet therapy	tPA	Tissue Plasminogen Activator
ECG	Electrocardiogram	USA	Unstable Angina
LIMA	Left Internal Mammary Artery		
MI	Myocardial Infarction		
MIDCAB	Minimally Invasive Direct Coronary Artery Bypass		



Learning Outcomes

Upon completion of this chapter, the learner will be able to:

1. Articulate the pathophysiology, classifications, and risk factors of acute coronary syndrome.
2. Interpret the manifestations, diagnostic testing, and collaborative care of the patient with angina pectoris, ST-elevation myocardial infarction, and non-ST-elevation myocardial infarction.
3. Compare invasive therapies/interventions and distinguish the nursing care associated with each.
4. Detect the potential complications associated with acute coronary syndrome.

Introduction

Acute coronary syndrome (ACS) is an inclusive term for conditions that cause chest pain due to insufficient blood supply to the heart muscle. ACS thus covers the spectrum of clinical conditions ranging from **ST-segment elevation**

myocardial infarction (STEMI) to non-ST-segment elevation (NSTEMI) ACS, which includes the diagnosis of unstable angina and **non ST-segment elevation myocardial infarction (NSTEMI)**. These life-threatening disorders are a major cause of emergency medical care and hospitalization in the United States.

Coronary artery disease (CAD) is the leading global cause of death accounting for 17.3 million deaths annually (American Heart Association, 2015). In the past few years, the death rate has started to decline, most likely due to improved prevention and treatment. Despite this decline, CAD is the number one cause of death in the United States and is also the leading cause of death for women, accounting for the loss of more lives than all types of cancer combined (Mozaffarian et al., 2015).

Pathophysiology, Classifications, and Risk Factors of Acute Coronary Syndromes

ACS is the clinical manifestation of coronary heart disease, which includes the development of fatty plaques in the coronary arteries. The growth of the atherosclerotic plaques narrows the vasculature, which then limits the blood flow and delivery of oxygen to the coronary muscle (♦ Figures 6-1 and ♦ 6-2). In the early stage of atherosclerosis, small lipoprotein particles penetrate the vascular endothelium, where they are coalesced into the intimal lining. This leads to an accumulation of plaque in the coronary arteries resulting in a less serious condition known as **stable angina**. A more serious condition exists when the plaque

ruptures or becomes unstable in the vasculature. The unstable plaque is characteristically composed of a large lipid core, a large number of lipid-laden macrophages, and a thin fibrous cap. Rupture of this cap exposes the thrombogenic contents of the plaque into a patient's circulation. This evolution of foreign material causes circulating platelets to adhere to the subendocardial collagen, thus causing more platelets to activate and aggregate at the site of injury (Srikanth & Ambrose, 2012).

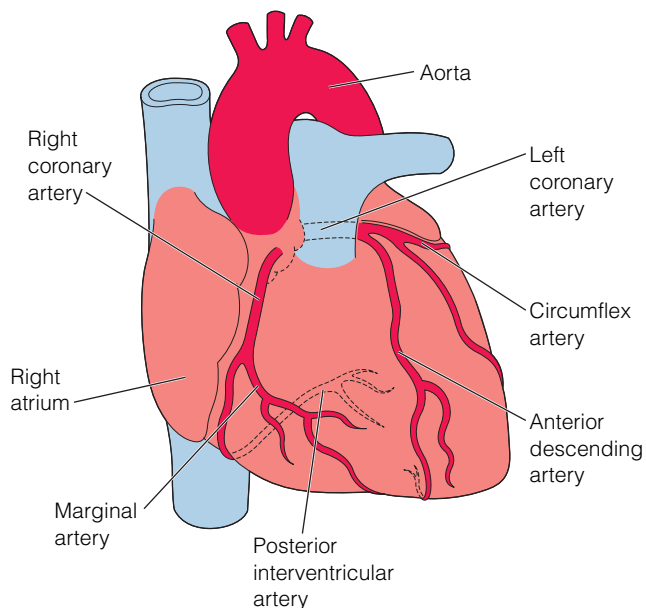


Figure 6-1 Coronary circulation, coronary arteries.

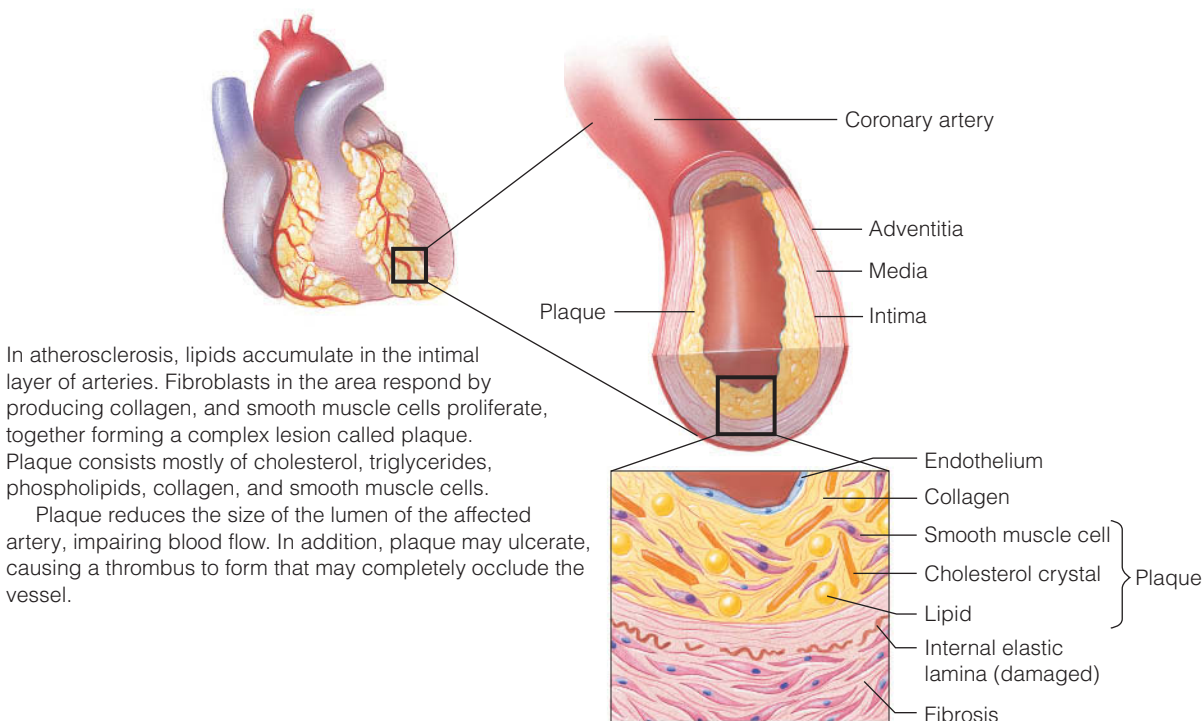


Figure 6-2 Atherosclerosis.

The formation of platelet-rich thrombi is the underlying cause of adverse ischemic events across the full spectrum of ACS. At first, the growth of the platelet-rich thrombi causes an occlusion, which leads to myocardial ischemia and the diagnosis of **unstable angina (USA)**. As the platelets continue to aggregate, they can cause blockage of smaller blood vessels resulting in a NSTEMI. The plaque rupture also induces the formation of thrombin, which converts fibrinogen into fibrin. This formation of fibrin along the area of ruptured plaque assists the thrombus to stabilize and fully occlude the coronary vessel. The result of this full occlusion is a STEMI.

Risk Factors for Acute Coronary Syndrome

Through extensive research the American Heart Association has articulated several factors that place a patient at risk for developing CAD (Eckel et al., 2014). Some of these risk factors can be modified, treated, or controlled, and others cannot. The more risk factors a person has, the greater the chance of developing an ACS sometime in the person's lifetime.

Modifiable Risk Factors

Modifiable risks are those that the patient has some control over, for example, smoking, high blood pressure, sedentary life style, obesity, cholesterol level, and diabetes.

Tobacco Smoke. Cigarette smoking has an adverse effect on the lipid profile, clotting factors, and platelet function. Smokers' risk of coronary heart disease is two to four times that of nonsmokers. Cigarette smoking has been linked to sudden cardiac death as well as ACS. People who smoke cigars and pipes also have a high risk of dying from CAD, but it is still less than that of cigarette smokers. Exposure to secondhand smoke for nonsmokers also increases the risk of having an ACS event. Cessation of smoking can reduce the risk of a coronary event by 50% within the first 1–2 years of quitting (U.S. Department of Health, 2014) (Box 6-1).

Box 6-1 Interventions to Achieve Tobacco use Cessation

- Ask about tobacco status.
- Advise to quit if still smoking.
- Assess willingness to quit.
- Assist in developing a plan for quitting.
- Arrange for a smoking cessation class.
- Avoid environmental exposure.

Box 6-2 Interventions to Achieve Goal Blood Pressure

- Initiate lifestyle modification, including weight control; increased physical activity; alcohol moderation; sodium restriction.
- Include a diet of fresh fruits and vegetables and low-fat dairy products.
- If blood pressure is elevated, control with medication—beta blockers and/or ACE inhibitors and diuretics.

Hypertension. Hypertension increases the heart's workload, causing increased stress and oxygen consumption by myocardial muscle. There is a definite correlation between the degree of blood pressure (BP) elevation and CAD risk. The 20 mmHg average increase in systolic BP that comes with age increases the occurrence of CAD two to three times for individuals between the ages of 30 and 65 years. Recent modifications in the AHA recommendations for individuals with CAD call for maintenance of a SBP < 140 mmHg and a DBP < 90 mmHg. However, an excessive lowering of the DBP for patients with ACS may impair coronary blood flow and oxygen supply. In addition, patients with ACS often have vasomotor instability with an increased tendency to exaggerated responses to antihypertensive therapy (Rosendorff et al., 2015) (Box 6-2).

Physical Activity. Sedentary lifestyles are strong risk factors for heart disease. Regular, moderate to vigorous physical activity has been shown to prevent heart and blood vessel disease. Exercise has also been demonstrated to assist in controlling or eliminating other risk factors such as hypertension, hyperlipidemia, diabetes, and obesity (Box 6-3).

Obesity. The measurement commonly used to determine whether a person is obese is the body mass index (BMI). A BMI of 25.9 kg/m² to 29.9 kg/m² is considered overweight,

Box 6-3 Interventions to Achieve Physical Activity Goal

- Assess risk with physical activity history and/or exercise test.
- Encourage 30–60 minutes of activity every day.
- Encourage resistance training 2 days per week.
- Encourage cardiac rehabilitation program for people at high risk.

Bariatric Considerations

Extreme obesity (EO), defined as BMI ≥ 40 kg/m², is correlated with a higher risk of procedural and surgical complications. A recent study of 133 EO patients examined complications one year after percutaneous coronary intervention (PCI) and coronary artery bypass

surgery (CABG). The results indicated that a higher BMI and CABG surgery were associated with greater risk of major complications. Interestingly enough, both PCI and CABG procedures demonstrated the same rates for stroke, death and myocardial infarction (Ramirez et al., 2015).

and a BMI of over 30 kg/m² is considered obese. Excess weight causes added stress on the heart muscle, which increases coronary workload. Obesity is also linked to hypertension, hyperlipidemia, a decrease in high-density lipoproteins (HDL), impaired glucose metabolism, and inflammation. Numerous studies indicate the presence of the “obesity paradox,” which is present when lean patients with established CAD have a worse clinical prognosis than do their more overweight/obese counterparts with the same CAD (De Schutter, Lavie, & Milani, 2014) (Box 6-4).

Dyslipidemia. Dyslipidemia is defined as a disorder of lipoprotein metabolism, including lipoprotein overproduction or deficiency. The most commonly measured blood levels include total cholesterol, low-density lipoprotein (LDL) cholesterol, triglycerides, and high-density lipoprotein (HDL) cholesterol. Dyslipidemia is highly associated with the formation of atherosclerotic plaque, the precursor to ACS, and can be either familial and/or nonfamilial. The nonfamilial form is exacerbated by foods containing excessive trans fats, saturated fats, and cholesterol. The familial form is an autosomal dominant disorder that causes marked

Box 6-5 Interventions to Achieve Goal Serum Lipid Levels

- Start dietary therapy. Reduce intake of saturated fats to <7% of total calories.
- Add fiber to the diet.
- Encourage consumption of omega-3 fatty acids.
- Initiate lipid-lowering medication if fasting lipid levels are abnormal.

elevation of total cholesterol (TC) and LDL, also known as *bad cholesterol*, due to the absence or reduced number of LDL receptors. HDL is often called *good cholesterol* because it is responsible for removing excess cholesterol from blood vessels and transport it to the liver for metabolism. The ratio between the TC and HDL is referred to as the cardiac risk index (Goff et al., 2013). Refer to Table 6-1 for normal serum levels and cardiac risk index (see also Box 6-5).

Box 6-4 Interventions to Achieve Goal Body Mass Index

- Assess risk with physical activity history and/or exercise test.
- Encourage 30–60 minutes of activity every day.
- Encourage resistance training 2 days per week.
- Encourage cardiac rehabilitation program for people at high risk.

Diabetes. Diabetes increases both risk and mortality associated with ACS. Diabetes often coexists with the risk factors of dyslipidemia, hypertension, and obesity. The patient often shows signs of insulin resistance before being diagnosed with diabetes. This grouping of risk factors is known as metabolic syndrome and has been shown to increase the incidence of ACS (Box 6-6).

Metabolic syndrome, also known as insulin resistance syndrome, includes a group of abnormalities. First, insulin levels are elevated and fasting blood glucose is greater than 110 mg/dL. Insulin receptor sites in the skeletal muscle, fat, and liver become unresponsive to the action of insulin.

Table 6-1 Lipid Levels and TC/HDL Ratio

	Total Cholesterol	LDL	HDL	TG	TC/HDL
Desirable	<200 mg/dl	100–129 mg/dl	>45 mg/dl	<150 mg/dl	<3
Borderline	200–239 mg/dl	130–159 mg/dl	30–45 mg/dl	150–199 mg/dl	3–4
high risk	>240 mg/dl	>160 mg/dl	<35 mg/dl	>200 mg/dl	>4

Source: Martin, S. S., et al. (2014). 2013 ACC/AHA cholesterol treatment guideline: What was done well and what could be done better. *Journal of the American College of Cardiology*, 63 (24), 2674–2678.

Box 6-6 Interventions to Achieve Goal Serum Glucose Levels

- Initiate lifestyle and pharmacotherapy to achieve a normal HbA1c.
- Lifestyle changes as indicated in other risk factors.
- Coordinate care between other medical providers.

As this occurs, glucose cannot enter the cells in sufficient quantities. Initially, the pancreas responds by producing larger amounts of insulin. People with this condition are often asymptomatic though they have both hyperglycemia and hyperinsulinemia. Other abnormalities that are present in metabolic syndrome include central obesity, defined as a waist circumference larger than 40 inches in men or larger than 35 inches in women; high triglyceride levels, over 150 mg/dL; high LDL; and low HDL, less than 40 mg/dL in men and less than 50 mg/dL in women. These factors greatly increase the risk of heart disease.

Nonmodifiable and Other Contributing Risk Factors

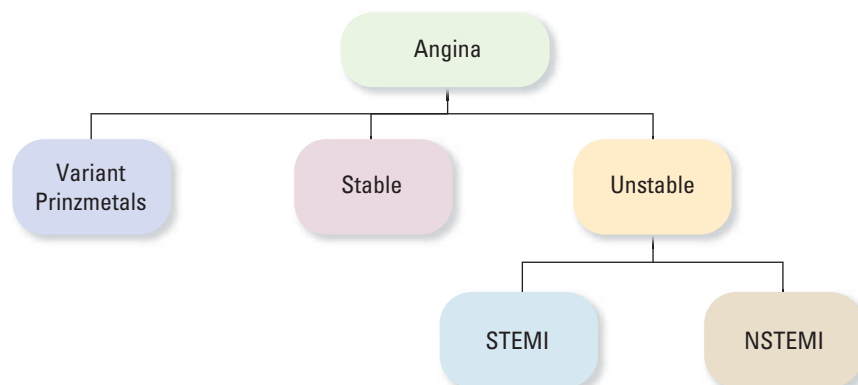
- **Age:** Plaque builds up over time so the incidence of CAD occurs more as we age. The risk for women increases after age 55 years and the risk for men increases after age 45 years.
- **Gender:** Men have a greater risk of MIs than women, and they tend to experience MIs earlier in life. In the 35 to 44 year old age group, white men are 6.1 times more likely to die of CAD than white women. There is less difference among nonwhites.
- **Heredity:** Children of parents with heart disease are at greater risk for developing it themselves. African Americans, due to the comorbidity of hypertension,

have a higher risk than Caucasians. Due to the obesity factor, it has been found that Mexican Americans, American Indians, and some Asian Americans are also at increased risk for developing a heart disease-related syndrome (AHA, 2015).

- **Stress:** People who are under extreme stress tend to participate in other unhealthy activities. This includes lack of exercise, overeating, and smoking. Stress also increases the workload on the cardiovascular system. Stress has the tendency to increase BP and heart rate (HR) and also contributes to the blood's ability to form clots, putting the patient at increased risk for a coronary event.
- **Alcohol Consumption:** Alcohol, when taken in large quantities, has been attributed to increasing a person's BP. It also contributes to an increase in triglycerides, an important component in the development of CAD.
- **Menopause:** Early menopause, at age 39 years or younger, and late menopause, at age 56 years or older, increases cardiovascular risk. By age 75, a woman's risk of death from CAD is equal to or greater than the risk for a man. Women who are taking estrogen therapy and who have vascular disease can continue this therapy if it is prescribed for other well-established indications. However, there is no clinical evidence for adding or continuing estrogens in postmenopausal women with CAD in an effort to prevent or retard progression of the disease (Whayne & Debabrata, 2015).

Angina Pectoris

Angina pectoris literally means "strangling of the chest." It is a general medical term used to define types of chest pain caused by myocardial ischemia. Due to plaque buildup and atherosclerosis, the myocardium can become deprived of blood and oxygen. Thrombus formation and further plaque formation eventually narrow the coronary arteries, causing ischemia and death of myocardial tissue. Ischemia occurs if the demand for oxygen exceeds its supply. Demand for



Visual Map 6-1 Relationship Among Types of Angina

myocardial oxygen increases with isometric and isotonic exercise, stress, exposure to cold, cigarette smoking, and sympathetic nervous system stimulation (Visual Map 6-1).

Classification

Angina can be classified into three different phases: stable, unstable, and variant. The angina that is of the most concern to the development of ACS is the unstable angina prototype. The stable and variant types are included to assist in the differentiation of the major anginal prototypes. Many factors can precipitate anginal pain (Box 6-7).

Stable Angina. Stable angina is chest pain that occurs predictably on exertion. This type of angina is associated with stable plaque buildup in the coronary arteries. The stenotic coronary vessel reduces coronary artery blood flow to a critical level. Symptoms of stable angina, although caused by decreased myocardial oxygen, can change from time to time depending on stress, oxygen consumption, and temperature extremes. Stable angina is often treated with short term nitroglycerin preparations and rest. Beta blockers are recommended as the initial agent to relieve symptoms in most patients with stable angina. If beta blockers are not well tolerated, patients may be placed on calcium channel blockers or long-acting nitrates. If the patient is known to have had a previous myocardial infarction or concurrent coronary artery disease, treatment should also include antiplatelet therapy, such as aspirin, clopidogrel, or a combination of the two. ACE inhibitors should be prescribed for patients with diabetes or left ventricular dysfunction unless contraindicated (Fihn et al., 2012). Risk factor modification and lifestyle changes are also included in the management of stable angina.

Reflect On

A patient starts experiencing left-sided chest pain. He associates it with “heartburn” and does not seek further evaluation. As the nurse, what would you suggest to this patient? What could be the consequences of the patient’s actions?

Box 6-7 Precipitating Causes of Anginal Pain

- Physical exertion
- Temperature extremes
- Emotions (stress and anger)
- Consumption of a large meal
- Tobacco use
- Sexual activity
- Stimulants (caffeine, cocaine, amphetamines)
- Circadian rhythm patterns

Variant (Prinzmetal’s) Angina

Variant angina is a less common form of angina. It is characterized by episodes of chest pain that occur at rest. Unlike stable and unstable angina, variant angina is caused by coronary artery vasospasms, which can cause an increase in myocardial oxygen demand and a transient ST-segment elevation. This type of angina is often treated with calcium channel blockers and long-acting nitrate preparations. Patients with Prinzmetal’s angina are younger and have fewer risk factors, so a cardiac examination is often unremarkable in the absence of ischemia. Cardiac catheterization may or may not demonstrate transient coronary spasms, with minimal plaque formation (Cannon & Braunwald, 2012).

Unstable Angina

Unstable angina is pain that occurs more often and in unpredictable patterns. It can occur while the patient is at rest, as well as with minimal exertion, and often causes the patient to limit his activity. The 2014 AHA key recommendations emphasize the continuum between unstable angina and NSTEMI (Amsterdam & Wenger, 2014) (Table 6-2).

Myocardial Infarction (MI)

An MI occurs when the heart muscle is abruptly deprived of oxygen. When the heart is deprived of oxygen, it proceeds through several phases of tissue injury (♦ Figure 6-3). The first phase is the area of ischemia. If treatment is not immediate, the tissue damage will continue on to injury and then necrosis. Coronary plaque rupture initiates the events

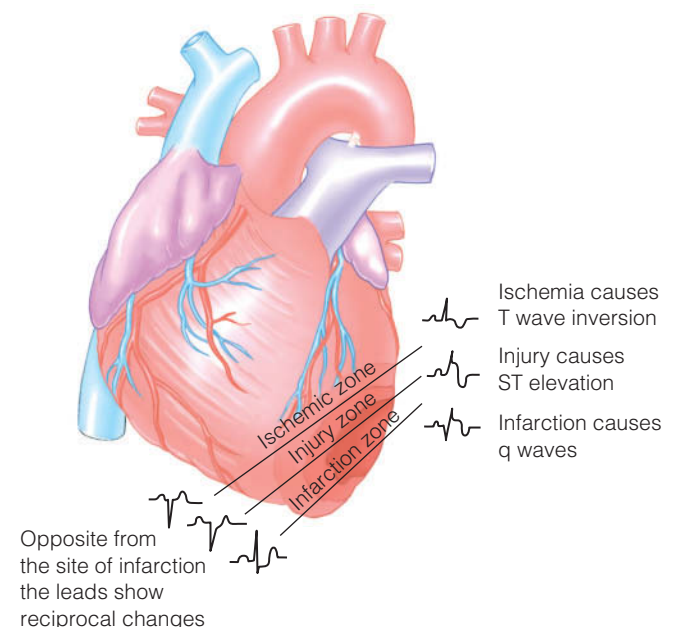


Figure 6-3 The evolution changes in acute MI.

Table 6-2 Classification and Treatment of Anginal Prototypes

Classification	Etiology	Symptoms	Treatment
Stable	Myocardial ischemia ACS	• Episodic pain lasting 5–10 minutes • Aggravated by exertion, exposure to cold, eating, emotional stress • Relieved by rest or nitroglycerin	• NTG • Aspirin • Beta adrenergic blockers • Calcium channel blockers • ACE inhibitors
Unstable	Ruptured or thickened plaque with platelet and fibrin thrombus, causing an increase in coronary obstruction	• Angina of increasing intensity, frequency, or duration • Occurs at rest or with minimal activity • Pain unresponsive to NTG	• NTG • Beta blockers tissue plasminogen activators (tPA), oxygen • Clopidogrel • Morphine • Statins • Aspirin
Variant	Coronary vasospasm	• Occurs at rest without provocation • Triggered by smoking Transient ST-segment elevation during pain. Often with associated AV block or ventricular arrhythmias • Occurs with or without the presence of CAD	• Calcium channel blockers

of an acute MI. Myocardial cells become ischemic within 10 seconds of coronary occlusion. During ischemia, cells experience a temporary shortage of oxygen, hypoxia, and downgrade their activity to conserve energy. Several minutes after the loss of contractile ability, the myocardium is deprived of its glucose source necessary for aerobic metabolism. This causes anaerobic processes to take over, and a buildup of lactic acid ensues. During this period the cells of the myocardium do not fully polarize. If this buildup continues without restoration of aerobic processes, an MI occurs. Because infarcted cells cannot respond to electrical impulses, they cannot help the heart contract. The buildup of scar tissue from the ensuing necrotic heart tissue can lead to **ventricular remodeling**, which changes the shape and function of the heart muscle.

Location of Infarction

Myocardial injury and infarction occur mostly in the left ventricle because it has the greatest oxygen demand and is therefore more affected by occlusions of the coronary arteries. An MI is categorized by the wall of the ventricle on which it occurs. This can be the anterior, lateral, septal, inferior, or posterior wall, or a combination of these walls.

An anterior wall MI is caused by occlusion of the left anterior descending artery (LAD). The higher or more proximal the occlusion, the more muscle damage ensues. Occlusion to the LAD can also cause an anterolateral or anteroseptal MI. Damage for this particular MI includes the anterior wall and anterior portion of the septal wall. Anterior wall MIs account for 25% of MIs and have the highest mortality rate. These patients often suffer from serious complications of ACS, elaborated on later in the chapter.

A lateral wall MI usually occurs with an occlusion of the circumflex artery or the diagonal branch of the LAD. Muscle damage occurs in the free, lateral wall of the left ventricle. Because the circumflex artery also supplies a portion of the sinoatrial (SA) node and atrioventricular (AV) node, patients often present with the complications of sinus dysrhythmias. An inferior wall MI is most commonly due to an occlusion of the right coronary artery (RCA). The muscle damage is seen in the right ventricle and posterior and inferior left ventricle. More than half of all inferior wall MIs are associated with an occlusion of the proximal portion of the RCA, which can lead to significant damage to the right ventricle. (Further information can be found later in the chapter in the discussion of the anatomical location of MIs.)

Myocardial injury to the posterior wall of the left ventricle is uncommon due to the dual blood supply from the RCA and circumflex artery. If an infarction occurs in this portion of the heart muscle, it will also cause significant damage to the right ventricle.

Non-ST-Segment Elevation Myocardial Infarction (NSTEMI)

NSTEMI (previously termed subendocardial infarction or non-Q-wave myocardial infarction) and unstable angina occur as a result of a subtotal occluding thrombus. This type of infarction is associated with ST segment depression or T wave inversion on the ECG (♦ Figure 6-4). There are no acute ST-segment elevations or Q waves. NSTEMI and unstable angina may be undistinguishable with ECG studies. They differentiate only by the serologic evidence of myocardial necrosis.

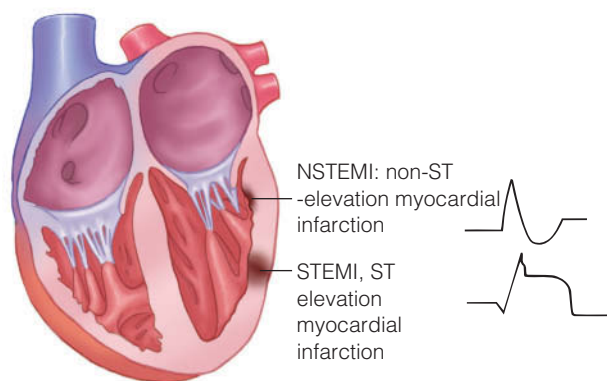


Figure 6-4 ECG and myocardial findings in Q-wave and non-Q-wave MIs.

(Courtesy Barbara Ritter, EdD, FNP-BS, CS.)

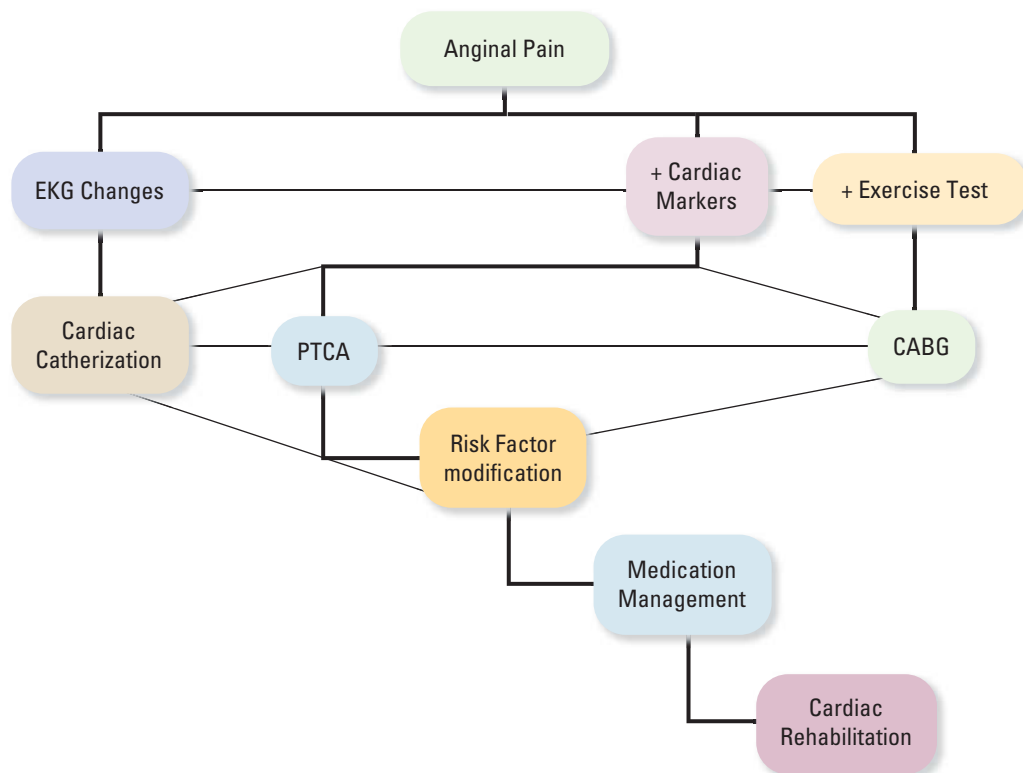
Initial Focused Assessment and treatment includes (Amsterdam & Wenger, 2014):

- Hospital admission
- Serial ECGs and cardiac enzymes
- Rest for 24–48 hours
- Continuous bedside ECG
- Analgesics
- Supplemental oxygen
- Pharmacological therapy
 - Sublingual nitroglycerin to start, if pain reoccurs should be placed on a NTG intravenous drip

- Oral beta blockers should be instituted to decrease heart rate, blood pressure, and myocardial oxygen demand
- Calcium antagonists are used for patients unresponsive to NTG and beta blockers
- Aspirin, P2Y₁₂ inhibitor, anticoagulation with unfractionated heparin or bivalirudin
- High intensity statin therapy
- Urgent/immediate invasive strategy

ST-Segment Elevation Myocardial Infarction (STEMI)

The most common cause of STEMI, formerly known as Q-wave MI, is reduced myocardial perfusion from an occlusive thrombus that developed on a ruptured portion of atherosclerotic plaque. Associated ECG changes will be discussed later in this chapter but briefly include ST segment elevation, Q waves, and a new left bundle branch block. Nursing actions are similar to the NSTEMI actions and will be further elaborated upon in the next session (Abid et al., 2015). Reperfusion therapy should be administered to all eligible patients with STEMI with symptom onset within the prior 12 hours, and PCI is the recommended method of reperfusion when it can be performed in a timely fashion by experienced operators (O’Gara et al., 2013).



Visual Map 6-2 Assessment and Management of the Patient with Anginal Pain

Manifestations, Diagnostic Testing, and Collaborative Care of the Patient with Acute Coronary Syndrome

The classic symptom a patient experiences during a cardiac event is pain. It is important for the nurse to differentiate the pain of an MI or angina attack from a multitude of other pain syndromes that can mimic a coronary event. **Noncardiac chest pain (NCCP)** may be caused by other cardiovascular issues including pericarditis, aortic aneurysm or dissection. Non-cardiovascular causes may originate from organs in close proximity to the heart. Gastrointestinal causes may include peptic ulcer disease, reflux, pancreatitis or esophageal rupture. Pulmonary causes may include pulmonary embolus, pneumothorax, pleuritis or pneumonia. Musculoskeletal causes may include costochondritis, rib fracture or disc injury. Patients commonly describe the discomfort as crushing, oppressive, or constricting or as a pressure that may radiate to the left arm, neck, jaw, infrascapular area, or epigastric region. Transient symptoms that last less than 15 minutes and disappear at rest are classified as angina. The discomfort associated with an MI typically lasts more than 30 minutes, is not relieved by rest or nitroglycerin, and may or may not be severe. The patient is asked to evaluate the level of pain using the universal 0 to 10 scale, with 10 being the highest level of discomfort.

Additional signs and symptoms of an MI include nausea, vomiting, diaphoresis, palpitations, and dyspnea. The patient may have cool, clammy skin due to vasoconstriction and a low-grade fever caused by the systemic response to inflammation (Table 6-3).

Special populations may be challenging to assess correctly. The elderly and patients with diabetes may not

Table 6-4 Symptoms of Heart Disease in Special Populations

Women	The most commonly encountered symptom is shortness of breath. Other atypical signs and symptoms include nausea or vomiting, diaphoresis, palpitations, gastrointestinal problems
Diabetics	Atypical and silent signs and symptoms due to autonomic dysfunction
Elderly	Generalized weakness, stroke or cerebral vascular accident, syncope or change in mental status, silent ischemia, especially if having a history of heart failure.

Source: Adapted from Fihn, S. D., et al. (2012). ACCF/AHA/ACP/AATS/PCNA/SCAI/STS guideline for the diagnosis and management of patients with stable ischemic heart disease: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, and the American College of Physicians, American Association for Thoracic Surgery, Preventive Cardiovascular Nurses Association, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. *Journal of the American College of Cardiology*, 60(24), e44–e164.

experience chest discomfort but may present with only dyspnea and palpitations. Women often arrive in the ED complaining of shortness of breath, fatigue, lethargy, indigestion, and anxiety. Women also tend to experience pain in the back rather than substernally or in the left side of the chest and do not characterize it as pain, but as a numbness or tingling sensation (Fihn et al., 2012). Refer to Table 6-4, “Symptoms of Heart Disease in Specialized Populations.”

Focused Assessment and Management

A mnemonic device used for the evaluation of chest pain is “APQRST” (Table 6-5). A history must be ascertained from the patient. This should include symptomatology of the anginal pain, history of CAD, and presence of CAD risk factors (Loscalzo, 2012). Therapeutic hypothermia should be started as soon as possible in comatose patients with STEMI and out-of-hospital cardiac arrest caused by ventricular fibrillation or pulseless ventricular tachycardia (O’Gara et al., 2013).

Table 6-3 Differentiation Between Cardiac and Noncardiac Pain

Characteristics	Cardiac	Noncardiac
Timing	Usually occurs in the AM	End of day
Pain Quality	Pressure, heaviness, constricting, burning, squeezing. Deep pain, not superficial. Radiates to arms, jaw, back, below diaphragm. Cannot pinpoint; described as diffuse pain	Sharp, more localized. Easy to pinpoint
Etiology	Pain preceded by exertion: exercise, heavy meals, extreme weather. May awaken patient from sound sleep	“out of the blue” pain directly associated with injury
Associated Symptoms	Dyspnea, nausea/vomiting, diaphoresis, palpitations. Feelings of impending doom.	
Pain Duration	Lasts with exertion but declines or stops with rest.	May be fleeting or last several hours.
Pain Relief	Stops with rest. Nitroglycerin Sitting and leaning forward Laying down increases pain	Rest or exercise Simple analgesics Antacids Reassurance
Risk Factors	Hypertension, diabetes, obesity, dyslipidemia, smoking	

NURSING ACTIONS

Upon arrival to the emergency department (ED), the nurse needs to perform the following interventions to alleviate pain and anxiety and increase the myocardial oxygen level.

- The patient is placed in a semi-Fowler’s position to allow for comfort and proper cardiovascular functioning.
- An ECG and cardiac enzymes should be assessed within 10 minutes.
- An intravenous line should be started to keep the vein open for immediate access for the administration of emergency drugs.
- Medications and treatments are then started in a standard progression.
- Aspirin is given first and consists of four “baby strength” aspirin or the equivalent of 325 mg. Baby aspirin is the treatment of choice because the patient can chew or have the medication dissolve in the mouth without a bitter taste. It is used to prevent platelet aggregation and the formation of blood clots in the coronary vasculature. A loading dose of a P2Y₁₂ receptor inhibitor, clopidogrel, ticagrelor, or prasugrel, should be given as early as possible or at time of primary PCI (O’Gara et al., 2013).
- Nitroglycerin—May be administered in either the sublingual tablet or translingual spray, which will reduce the pain that is currently present, or the intravenous form to prevent future episodes.
- Supplemental oxygen—According ACLS Guidelines (AHA, 2015), oxygen should be applied only if the oxygen-hemoglobin concentration is less than or equal to 94%.
- Morphine—Morphine sulfate is given in a dose of 2 to 5 mg intravenous every 5 to 30 minutes as needed. Morphine is used for both pain management and as an antianxiety medication. If this medication is given first, it can mask the patient’s perception of pain and mask the underlying cause of ischemia.
- Beta blockers—should be promptly instituted because they decrease heart rate and blood pressure levels. These drugs will also decrease left ventricular contractility, thereby reducing myocardial oxygen demand.

- Vital Signs—should be taken at frequent intervals.
- Ace Inhibitor—should be started, especially for patients with STEMI in the anterior location, heart failure, or ejection fraction less than or equal to 40.

Diagnostic Criteria

Diagnostic criteria are extremely important for the patient with an MI and ACS. This information helps to determine the location of the muscle damage as well as the extent of the damage. Several diagnostic tests are included as the mainstay of MI or ACS diagnosis.

Electrocardiogram

The 12-lead **electrocardiogram (ECG)** is one of the most crucial diagnostic tools available for ACS. In a normal ECG, the ST segment is level with the tracing’s isoelectric line. Any deviation of the ST segment from this isoelectric line will determine the amount of damage to the heart muscle. Patients presenting to an ED with chest discomfort should have a 12-lead ECG performed within 10 minutes of arrival and should also be evaluated with telemetry monitoring. The presence of severe and prolonged elevation of the ST segment, or, less frequently, new Q waves, or new left bundle branch block (LBBB) indicates that the patient is likely experiencing a STEMI. Patients with NSTEMI may present with ST depression, T-wave inversion, transient ST-segment elevation, or with any ischemic ECG abnormalities.

Ischemia. Myocardial ischemia is the result of an imbalance between the myocardial oxygen supplies versus the myocardial oxygen demand. If this supply and demand scenario is not corrected in a timely fashion, cell death will occur. There are several key ECG changes that coincide with myocardial ischemia. The first is a T-wave inversion. Other ECG changes are ST-segment depression of greater than 0.5 mm, an ST segment that remains on the baseline longer than 0.12 second, and inverted U waves.

Injury. Myocardial injury is most often indicated by ST-segment elevation of 1 mm or more above the baseline.

Table 6-5 “APQRST” Mnemonic for the Evaluation of Chest Pain

A = Associated symptoms	Dyspnea, nausea/vomiting, diaphoresis, palpitations, feelings of impending doom
P = Precipitating factors	Exertion; exposure to cold; following meals; movement; relieved by rest, nitroglycerin, or positional changes
Q = Quality	Heaviness; tightness; pressure; sharp, stabbing, burning
R = Region, radiation, risk factors	Radiates to arm, jaw, back, below diaphragm Region is substernal, left lateral, right chest Risk factors include hypertension, diabetes, obesity, dyslipidemia, smoking
S = Severity	Pain scale of 0–10
T = Timing	Onset and duration: nocturnal, constant or intermittent

Source: Adapted from Reigle, J. (2005). Evaluating the patient with chest pain: The value of a comprehensive history. *Journal of Cardiovascular Nursing*, 20(4), 226–231.

The T wave may also become taller and pointed in configuration. There is also symmetric T-wave inversion. ST segment elevation of greater than 0.2mmV in more than two chest leads, ST segment elevation more than 0.1mmV in more than two contiguous limb leads or a new complete left bundle branch block (LBBB) are indications for immediate coronary intervention.

Infarction. Infarction is suspected when the ECG shows either new Q waves or a deepening of existing Q waves. A Q wave on an ECG is the first negative deflection in a QRS complex. Q waves appear within hours of pain onset and usually remain for the remainder of a patient's lifetime. A Q wave that is either 0.04-second-wide or that has a depth of at least 25% of the size of the R wave is considered pathologic.

When a Q wave appears on an ECG, the MI tends to be transmural or through the entire thickness of the myocardium. T-wave inversion occurs within hours of an infarction and can last for several months after the complete evolution of the MI.

Specific 12-Lead Changes

An anterior MI is correlated to ST elevation in leads V₂ through V₄. An MI caused by the occlusion of the left main artery is characterized by ST-segment elevation in leads V₁ through V₆. Elevation in V₁ through V₆ represents a large amount of damage to the myocardium. An anterior lateral MI caused by an occlusion of the LAD shows ST-segment elevations in V₃ through V₆ and leads I and aVL. An anteroseptal MI shows ST elevations in leads V₁ through V₄ (♦ Figure 6-5).

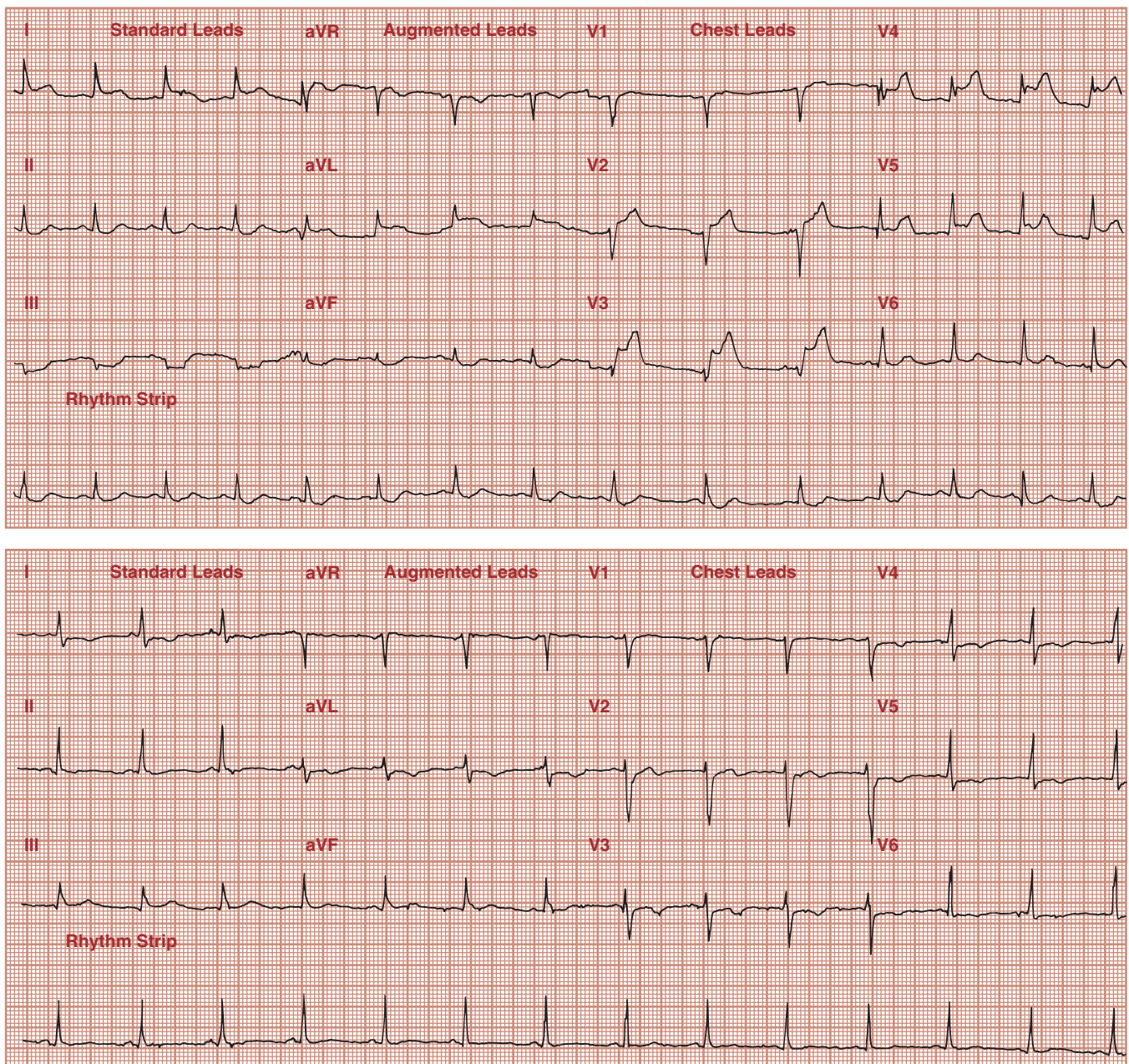


Figure 6-5 12-lead ECG of patient with an anterior MI before and after reperfusion.

Table 6-6 ECG Changes in MI

Site Of Infarction	Changes Seen	Possible Occlusion
Large anterior wall	V ₁ –V ₆ : ST-segment elevation <i>II, III, aVF: ST-segment depression</i>	Left main coronary artery
Anterior wall	V ₂ –V ₄ : ST-segment elevation <i>II, III, aVF: ST-segment depression</i>	Left anterior descending artery
Anteroseptal	V ₁ –V ₄ : ST-segment elevation <i>II, III, aVF: ST-segment depression</i>	LAD and branches supplying blood to the septal wall
Anterolateral	I, aVL, V ₃ –V ₆ : ST-segment elevation <i>II, III, aVF: ST-segment depression</i>	LAD and branches supplying blood to the lateral wall
Lateral wall	V ₅ , V ₆ , I, aVL: Pathologic Q wave, ST-segment elevation, inverted T wave <i>III, aVF: ST-segment depression</i>	Left circumflex (LCx) and/or LAD
Inferior wall	II, III, aVF: Pathologic Q wave, ST-segment elevation, inverted T wave <i>ST-depression AVL + Lead 1</i>	RCA and/or LCx
Posterior wall	V ₁ –V ₃ : ST-segment depression, tall, upright, symmetrical R wave, and tall, symmetrical T wave	RCA and/or LCx

Source: Adapted from Ellis, K. (2007). *EKG plain and simple*. Upper Saddle River, NJ: Prentice Hall.

ST-segment changes in leads I and aVL indicate a high lateral wall infarct, whereas elevations in leads V₅ and V₆ reveal a lower lateral infarct. Inferior wall MI is shown with elevations in leads II, III, and aVF.

ECGs look primarily at the front of the heart; therefore, in order to assess damage to the posterior wall, a nurse needs to look for reciprocal changes in the pattern. These changes are italicized in Table 6-6, and include ST depression in the contralateral leads.

Reflect On

A 65-year-old woman on the cardiac unit complains of chest pain. The ECG reveals changes indicative of acute anterior STEMI. What would be your nursing priorities?

Laboratory Assessment

The patient who is suspected of having acute myocardial injury should have blood drawn to measure blood counts including a CBC, metabolic profile and coagulation studies. Cardiac biomarkers are released when myocardial inflammation, injury, and necrosis are present. These biomarkers are used to confirm diagnosis of an MI and assist with risk stratification and outcome prediction. Blood work should be performed within 10 minutes of arrival to the ED and obtained serially as initial levels may be normal.

Troponin. **Cardiac troponin** assays are very useful in the diagnosis of an acute MI. These are highly sensitive tests that can determine myocardial damage. There are two cardiac-specific troponin markers: Troponin T and troponin I. Troponin I is the most accurate marker of myocardial injury. Data have suggested that a troponin T concentration measured 72 hours after an acute MI may be predictive of MI size, independent of reperfusion. The guidelines consider values greater than 0.01 ng/mL as elevated and

values of 0.1 ng/mL as markedly elevated and indicating a high risk of death. Cardiac troponins may not be released until 6 hours after symptoms begin; therefore, the ACC/AHA (Amsterdam & Wegner, 2014) recommends that patients who have a negative troponin level within 6 hours of symptom onset be retested at 8 to 12 hours after onset.

Creatinine Kinase. **Creatine kinase** may not be helpful in immediate diagnosis of an MI due to the 4 to 6 hours needed to see a significant rise. It is believed that the CK levels are more important in gauging the size and timing of an acute MI than the actual diagnosis. **Creatine phosphokinase-myocardial band (CK-MB)** is used to determine myocardial damage. These levels increase within 3 to 6 hours, peak at 24 hours, and return to normal within 36–72 hours after infarction. The CK-MB is considered positive when it is greater than 3% of the total CK. This test can be an effective biomarker in the detection of reinfarction (McLean & Huang, 2012).

Myoglobin and C-Reactive Protein (CRP). Both of these biomarkers lack cardiac specificity and need to be used in conjunction with other definitive laboratory tests. A doubling of the myoglobin level in 2 hours strongly suggests MI, and a negative myoglobin within 4 to 8 hours after the onset of symptoms is used to rule out an MI. The CRP level correlates with peak levels of the MB isoenzyme. This peak usually occurs 1 to 3 days later. Failure of the CRP to normalize may indicate ongoing damage to the heart. A baseline CRP is a good marker for future cardiovascular events (Abid et al., 2015). Refer to Table 6-7 for comparison of these biomarkers.

Reflect On

A patient arrives at the emergency room complaining of chest pain. Cardiac enzymes 1 hour post the event are normal. Can you say the patient did not suffer from a STEMI? What else does the nurse need to assess?

Table 6-7 Cardiac Biomarker Comparison

	Blood Levels	Initial Elevation and Peak	Duration
Troponin I	<0.03 ng/mL >1.5 ng/mL consistent with MI	Initial: 4–12 hrs Peak: 12 hrs	5–14 days
CPK-MB	3–6%	Initial: 3–6 hrs Peak: 12–24 hrs	Up to 48 hours

Chest X-ray

A chest x-ray upon admission can be used to determine signs of impending heart failure. It can also be useful in ruling out pulmonary and vascular causes for chest pain such as pericardial effusion, pneumothorax, pulmonary effusions or consolidation, pulmonary vessel congestion from pulmonary embolism and to evaluate for cardiomegaly.

Exercise Testing

Exercise testing with continuous electrocardiographic monitoring is a widely used and reliable method of evaluating patients who have or are at risk for developing an ACS. This type of evaluation focuses on the changes in the electrocardiograph tracing during exercise as well as gives information about dysrhythmias, HR, BP, exercise-perceived exertion rate, and exercise capacity, myocardial oxygen uptake and VO_2 , oxygen uptake and ventilator threshold (Fletcher et al., 2014). The most common testing is done while the patient is on a treadmill or exercise bicycle.

Pharmacological stress agents may be used if a patient is unable to walk a treadmill or perform exercise on the exercise bicycle. Patients with musculoskeletal disorders, vascular diseases, and chronic pulmonary diseases or the elderly are prime candidates for this type of stress testing. Pharmacological stressing is also used in patients with left bundle branch block and ventricular pacing, which often interfere with testing and yield false positive results. Pharmacological stress agents currently in use are dipyridamole, adenosine, and dobutamine.

Single photon emission computed tomography (SPECT) myocardial perfusion imaging is widely used in the assessment of patients with known or suspected CAD. Comparison of images obtained during rest and stress allows diagnosis of the reversibility, severity, and extent of the myocardial perfusion defects. This is an important piece in the management decision-making of the patient with CAD. The timing of the exercise stress test in patients with ACS depends on the specific risk. The AHA states it is reasonable for patients with possible ACS who have normal serial ECGs and cardiac troponins to have a treadmill ECG, stress myocardial perfusion imaging, or stress echocardiography before discharge or within 72 hours after discharge. It is strongly suggested that the patient be clinically stable before undergoing any type of exercise stress testing (Amsterdam & Wenger, 2014).

Diagnostic Imaging

In patients with a suspected STEMI, the two-dimension echocardiography reveals abnormalities in wall motion and determines left ventricular function. A TTE (transthoracic) or TEE (transesophageal) approach may be used.

Doppler echocardiography allows assessment of both direction and velocity of blood flow within the heart and great vessels. This can be used to evaluate valve integrity. The two-dimensional echocardiography and Doppler echocardiography are often used in conjunction with exercise or pharmacological stress testing to detect regional wall motion abnormalities during exercise.

Cardiac Catheterization

A cardiac catheterization or angiography is the gold standard for diagnosis of cardiac pathology. It is used to determine the exact location of the myocardial injury and specific obstructions to the coronary vasculature. A left heart catheterization permits the visualization of coronary artery anatomy, while a right heart catheterization demonstrates intracardiac pressures. This test determines if coronary artery occlusion is amenable to nonsurgical intervention such as percutaneous coronary intervention (PTCI) or surgical intervention such as coronary artery bypass graft (CABG). See specific care of patient undergoing these procedures later in this chapter.

COLLABORATIVE CARE

The immediate treatment goals for the patient with MI are to restore coronary artery blood flow, limit the infarction size, and balance myocardial oxygen supply and demand.

Pharmacological therapy remains an important intervention for patients with ACS. There are many therapies involved including those that will decrease ischemia, assist in a decrease in platelet aggregation, and provide pain relief. See “Commonly Used Medications” for key information regarding anti-ischemic, antithrombotic, and platelet aggregation inhibitor therapies.

Invasive Therapy/Interventions

Patients who have definitive ischemic ECG changes and/or elevated cardiac markers are typically managed with an early invasive strategy. This strategy involves the use of diagnostic catheterization, followed by a PCI or CABG surgery as soon as possible. Procedural and nursing care for the cardiac catheterization patient are similar to the PCI, so they will be included in the discussion later.

Commonly Used Medications

Anti-Ischemic Therapy: Increase Myocardial Perfusion and Decrease Pain

Nitroglycerin

Nitroglycerin is an arterial and venous vasodilator. It increases coronary blood flow by dilating the coronary arteries. It is first given as a 0.4-mg sublingual tablet or spray, and may be followed with intravenous administration.

Desired Effects: Reduces systolic, diastolic, and mean BP and has antianginal, anti-ischemic, and antihypertensive effects. When administered sublingually to a patient with angina, it usually results in a reduction in pain in 3 to 5 minutes.

Nursing Responsibilities:

- Ensuring administration of a therapeutic dose: Sublingual: 0.3 to 0.4 mg q 3 to 5 min (maximum dose is three tablets in 15 minutes); Intravenous: Start infusion at 5 mcg/min and titrate every 3 to 5 minutes until the desired response is achieved.
- Ensuring use with extreme caution in patients with hypotension; monitoring for unresponsiveness and cardiac dysrhythmias.
- Monitor blood pressure every 3 to 5 minutes if on intravenous dose to maintain SBP > to 90 mmHg.
- If the patient's blood pressure does decrease, lowering the head of the bed, increasing the rate of IV fluids, or decreasing the dose of nitroglycerin temporarily should resolve the problem.
- Assess patient for a headache (50% of patients develop this effect).

Side and/or Toxic Effects: Symptoms of overdose include hypotension; tachycardia; warm, flushed skin; headache; confusion, nausea; convulsions; and cardiovascular collapse. These symptoms may occur because ethanol is the diluent in IV nitroglycerin. When high doses of IV nitroglycerin are administered, the patient may experience ethanol intoxication (Wilson, Shannon, & Shields, 2016).

Morphine Sulfate

Morphine sulfate, an opioid analgesic, is used when chest discomfort is not relieved with nitroglycerin, when the patient becomes agitated, or when he or she is experiencing the complication of pulmonary congestion.

Desired Effects: Morphine helps to increase the coronary blood flow by dilating arteries and veins while it controls severe pain and anxiety. It also reduces preload, which, in effect, decreases the workload of the myocardial tissue.

Nursing Responsibilities:

- Providing a therapeutic dose, usually 2 to 10 mg IV in divided doses.

- Obtaining baseline respiratory rate, depth and rhythm, and size of pupils.
- Recording pain level and duration of analgesia.
- Continuing to monitor for respiratory depression.

Beta Blockers

Blocking beta-1 receptors located primarily in the myocardium and beta-2 receptors in the vasculature and bronchial smooth muscle decreases myocardial contractility, slows heart rate, and decreases systolic BP, thus lowering the cardiac oxygen demand. A slower heart rate will also assist in increasing the ventricular filling time and coronary artery blood flow, so more oxygen will reach the myocardial tissue. Initiate oral beta blockers within the first 24 hours of ACS in the absence of HF, low-output state, risk for cardiogenic shock, or other contraindications to beta blockade. Receiving beta blockers within 24 hours of hospital arrival after an MI reduces mortality in the first week by 14% (Amsterdam & Wenger, 2014).

Beta-blocker therapy is contraindicated when the patient has a heart rate less than 60 beats per minute, systolic blood pressure less than 100 mmHg, moderate or severe left ventricular failure, shock, PR interval on the electrocardiogram greater than 0.24 second, second or third-degree heart block, and active asthma and/or reactive airway disease.

Desired Effects: Beta blockers block the cardiac effects of beta-adrenergic stimulation; as a result, heart rate, myocardial irritability, and force of contraction decrease. They have also been associated with a decrease in platelet aggregability. When administered intravenously the effects occur within 1 to 2 minutes.

Nursing Responsibilities:

- Administering a therapeutic dose once the patient's hemodynamic condition has stabilized following an MI. AHA recommends an initial intravenous dose of 5 mg of metoprolol followed by two additional doses at 5-minute intervals for a total of 15 mg. The patient is converted to oral dosing if IV is tolerated.
- Observing monitor pattern, heart rate, respiration, blood pressure, and circulation closely during administration. Administration is stopped if bradycardia, heart block, or hypotension develop.
- Monitoring for signs and symptoms of heart failure.

Side and/or Toxic Effects: Bradycardia, hypotension, and heart blocks are serious side effects. Bronchospasm and dyspnea may also occur as may indications of poor perfusion such as syncope, confusion, or dizziness.

Commonly Used Medications

Antithrombotic Therapy Prevention of Thrombin-Mediated Fibrin Deposition and Stabilization of Platelet-Rich Thrombi

Aspirin

Giving aspirin as soon as possible inhibits platelet activity and interrupts platelet aggregation at the site of plaque rupture.

Desired Effects: It inhibits platelet aggregation, reducing the ability of blood to clot. It is used to prevent recurrence of MI, and in prophylaxis against MI in patients with unstable angina.

Nursing Responsibilities:

- Administration of a therapeutic dose. If a patient does not take aspirin regularly the initial dose should be 160 to 325 mg. The patient should chew the dose in the acute phase to increase absorption. If a patient regularly takes an aspirin daily, check with the physician for orders on appropriate dosage.
- Monitoring platelet count, prothrombin time and international ratio unit in patients with concurrent anticoagulant therapy.
- Using enteric-coated tablets to reduce gastrointestinal disturbances.

Side and/or Toxic Effects: The most common side effects are bleeding, GI discomfort, and tinnitus.

P2Y₁₂ Inhibitor: Clopidogrel or Ticagrelor

Both clopidogrel (Plavix) and ticagrelor (Ticlid) are drugs that inhibit platelet aggregation. This therapy is used to prevent thrombotic events in patients who have had a myocardial infarction with acute ST-segment elevation. Either clopidogrel or ticagrelor, in addition to aspirin, should be administered for up to 12 months to all patients with NSTEMI-ACS without contraindications who are treated with either an early invasive or ischemia-guided strategy (Amsterdam & Wenger, 2014).

Desired Effect: The desired effect is a prolongation of bleeding time and prevention of MI in patients with recent MI or unstable angina.

Nursing Responsibilities:

- Administration of a therapeutic dose: clopidogrel: 300–600 mg, followed by 75 mg daily. Ticagrelor: 180-mg loading dose, then 90 mg twice daily.
- Observing for bleeding episodes, especially GI bleeding.
- Reviewing periodic platelet count, bleeding time, and lipid profile.
- Should hold clopidogrel or ticagrelor for at least 5 days before CABG.

Side and/or Toxic Effects: The most serious adverse effects include bleeding and purpura.

Unfractionated Heparin (UFH)

Desired Effects: UHF inhibits formation of new clots but does not lyse already existing thrombi. It prevents thrombotic complications during the acute stage of an MI.

Nursing Responsibilities:

- Initial loading dose of 60 IU/kg (maximum 4000 IU) with initial infusion of 12 IU/kg per hour (maximum 1000 IU/h) adjusted per activated partial thromboplastin time (aPTT) to maintain therapeutic anticoagulation.
- Continued for 48 hours or until PCI or CABG is performed.
- Monitor aPTT levels closely.
- Checking platelet count for heparin induced thrombocytopenia (HIT).
- Observing for signs and symptoms of internal and external bleeding.

Side and/or Toxic Effects: The most common problematic adverse effect is bleeding.

Low Molecular Weight Heparins

Low molecular weight heparins (LMWH) have a relatively low degree of binding to plasma proteins and cells; they also have a more predictable anticoagulant effect, a longer half-life, and a lower incidence of thrombocytopenia than UHF. The low molecular weight heparin enoxaparin (Lovenox), given subcutaneously, is preferred. Enoxaparin has a longer half-life than heparin and provides more sustained and predictable anticoagulation.

Contraindications for both forms include hypersensitivity to heparin, previous heparin-induced thrombocytopenia, uncontrollable active bleeding, and the inability to monitor platelet counts.

Desired Effects: As with UHF, enoxaparin inhibits the formation of new clots but does not lyse existing clots. Antithrombotic properties are due to its anti-factor Xa and antithrombin (antifactor IIa) in coagulation activity.

Nursing Responsibilities:

- Administration of a therapeutic dose: usually 1 mg/kg every 12 hours for 2 to 8 days; given concurrently with aspirin 100 to 325 mg per day.
- Monitoring baseline coagulation studies.
- Watching platelet count closely and withholding the drug if the level of platelet count is less than 100,000 mm³.
- Assessing for signs of unexplained bleeding.

Reperfusion Therapy

Percutaneous Transluminal Coronary Intervention

Percutaneous Transluminal Coronary Intervention (PTCI) is recommended method of reperfusion when it can be performed in a timely manner. An ideal first medical contact to device time should be 90 minutes or less (O’Gara et al., 2013). PTCI is the placement of a small mesh tube called a stent into an infarcted or narrowed coronary artery. The procedure consists of cardiac catheterization and insertion of a catheter with a balloon tip that is inflated to open the artery, called percutaneous transluminal coronary angioplasty (PTCA), followed by a stent placement. Drug-eluting stents are coated with medications that prevent restenosis by reducing inflammation and the formation of thrombin.

Other PCTI procedures include percutaneous atherectomy and thrombus aspiration. To be most effective, these procedures should be performed within 90 minutes of the patient’s arrival in the ED and medical evaluation. The goal of treatment is to open the blockage in the coronary artery and reperfuse the myocardium, limiting the amount of damage. PCI is indicated in coronary arteries that have at least 70% narrowing.

During a PTCI, a catheter with a balloon is inserted into an arterial access (femoral, brachial, or radial artery) and guided to the desired site. The balloon is inflated, expanding the stent, which in turn squeezes the atherosclerotic plaque against the vessel wall and widens the arterial lumen. After the stent is in place, the balloon is deflated and removed. The stent remains in place, holding the plaque against the arterial walls and providing structural support (♦ Figure 6-6).

Patients with stent placement usually require dual antiplatelet therapy (DAPT) with aspirin and clopidogrel for one year. During this time, the endothelium grows over the stent, incorporating the device into the artery and reducing the tendency to clot. DAPT is maintained after stenting to reduce the risk of vessel thrombosis (Amsterdam & Wenger, 2014). (♦ Figure 6-7).

Fibrinolytic Therapy

If fibrinolytic therapy is used as the primary reperfusion strategy, administration should occur within 30 minutes of arrival. This may occur if the patient is at a non-PCI-capable facility, or if the time to intervention is greater than 120 minutes. Individuals who receive primary fibrinolytic therapy should also receive adjunctive antiplatelet and anticoagulant therapy (O’Gara et al., 2013).

Fibrinolysis is achieved with the use of tissue plasminogen activators (tPA) to quickly lyse the thrombus. These thrombolytic drugs are the most common used to

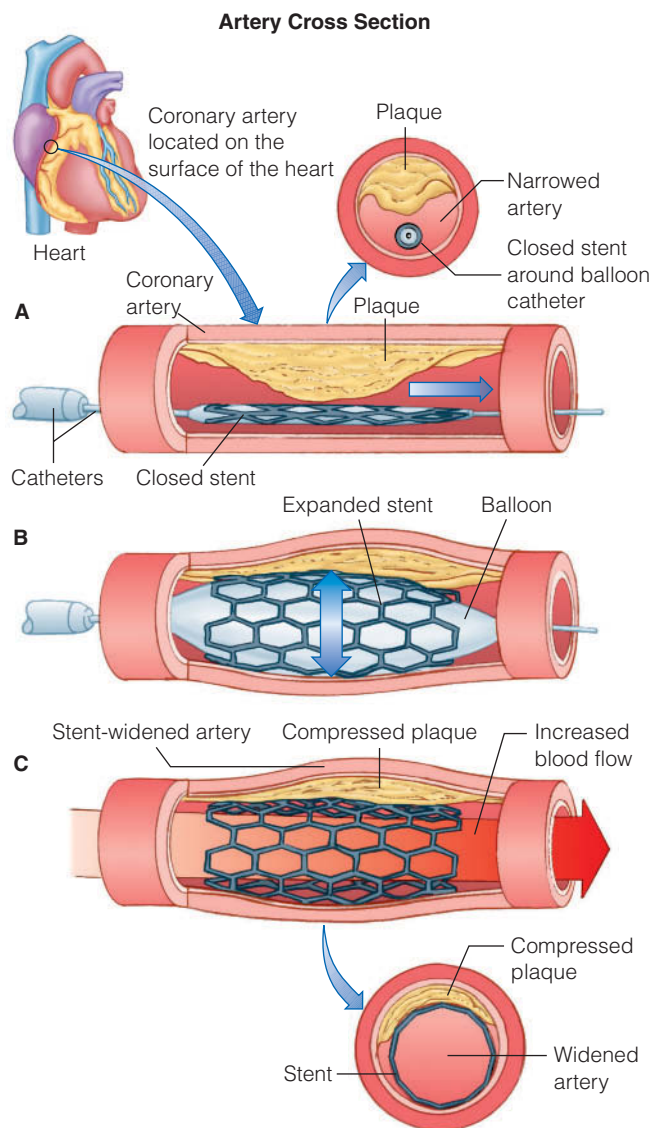


Figure 6-6 Stent placement following angioplasty.

treat acute coronary syndromes. The most commonly used thrombolytic drugs include alteplase (Activase), reteplase (Retavase), and tenecteplase (TNK-tPA).

The physician may also give the patient platelet GP IIb-IIIa inhibitors. These inhibitors may be used for patients with unstable angina, NSTEMI or in conjunction with PCTI. GP IIb-IIIa medications prevent platelet aggregation by keeping fibrinogen from binding to the GP IIb-IIIa receptors on the platelet surface. See “Commonly Used Medications” for more information regarding these medications. Contraindications to fibrinolytic therapy are displayed in Box 6-8.

NURSING CARE

The nurse prepares the patient for the procedure. The first step is to ascertain that the patient understands the procedure and that the consent form is signed. A medication

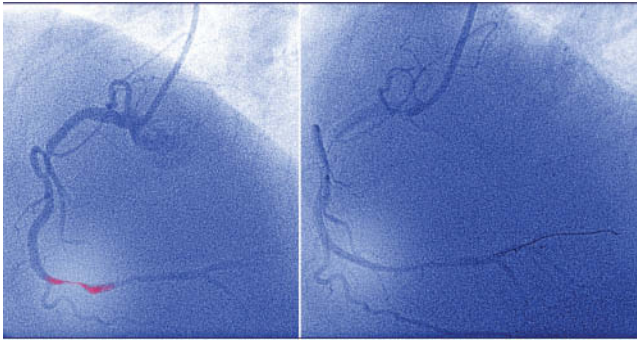


Figure 6-7 Coronary artery before angioplasty with stent insertion.

history, and any allergic reactions, especially to radiographic contrast media, aspirin, or clopidogrel, must be obtained. Routine pre-procedure laboratory work should be reviewed and abnormal values reported to the physician. Hydration should be considered an important component prior to contrast administration which may have a nephrotoxic impact on the kidneys (Box 6-9). Intravenous access must then be established with fluid at a keep-vein-open rate. The nurse should document peripheral pulses using a 0 to 4 scale regardless of access site, which may include radial, brachial or femoral arteries.

The nurse should explain to the patient what to expect during the procedure. The patient will be awake

Box 6-8 Contraindications for the Use of Fibrinolytic Therapy

Absolute Contraindications

- Active internal bleeding
- Known history of cerebral aneurysm or arteriovenous malformation (AVM)
- Previous cerebral hemorrhage
- Pregnancy
- Recent ischemic stroke
- Severe uncontrolled hypertension
- Suspected aortic dissection
- Traumatic CPR

Relative Contraindications

- Active peptic ulcer disease
- Current use of anticoagulants
- Prior ischemic stroke
- Dementia
- Recent surgery
- Recent internal bleeding

but will receive analgesics and sedatives to keep her relaxed and comfortable. The patient should be notified that she might experience mild chest pain during balloon inflation.

Commonly Used Medications

GP IIB-IIIa Inhibitors

Introduction

Three different agents fall into this category: eptifibatide (Integrilin) and tirofiban (Aggrastat), which are small molecular agents, and abciximab (ReoPro), which is a monoclonal antibody fragment. These drugs should be given as soon as possible and can be given prior to catheterization or PCI. The GP IIb-IIIa inhibitors are contraindicated for patients with bleeding diathesis, acute abnormal bleeding, or history of stroke within the last month. Renal dialysis, a history of major surgery within 6 weeks, or a platelet count of less than 100,000 mm³ are also contraindications (Wilson et al., 2016).

Desired Effects: The desired effect is inhibition of platelet aggregation, and effectiveness is indicated by minimizing thrombotic events during treatment of ACS.

Nursing Responsibilities:

- Administering the appropriate doses as prescribed: Abciximab (ReoPro)—Intravenous dose of 0.25 mg/kg bolus over 5 minutes followed by a continuous infusion of

0.125 mcg/kg/min for the next 12 hours; eptifibatide (Integrilin)—Intravenous infusion of 180 mcg/kg initial bolus followed by a 2 mcg/kg/min drip for up to 72 hours; tirofiban (Aggrastat)—Intravenous loading dose of 0.4 mcg/kg/min for 30 minutes followed by 0.1 mcg/kg/min for 12 to 24 hours.

- Monitoring platelet count, hemoglobin, and hematocrit prior to initiation of therapy and throughout treatment.
- Monitoring partial thromboplastin time (PTT) and international ratio (INR).
- Withholding the medication if thrombocytopenia is confirmed.
- Monitoring for signs and symptoms of internal and external bleeding at all invasive sites but especially at the sheath insertion site by observing the site and palpating the site and pulses distal to it.

Side and/or Toxic Effects: The most significant adverse effect is bleeding.

Box 6-9 Risk Factors for Contrast-Induced Acute Kidney Injury

Creatinine level ≥ 1.5 mg/dL
 Glomerulofiltration rate (GFR) $<$ or equal to 60 mL/min
 Diabetic nephropathy
 Congestive heart failure
 Hypovolemia
 High-dose contrast medium

SOURCE: From Marenzi, G., Assanelli, E., Marana, I., Lauri, G., Campodonico, J., Grazi, M., . . . Veglia, F. (2006). N-Acetylcysteine and contrast induced nephropathy in primary angioplasty. *New England Journal of Medicine*, 354, 2773–2782.

Peri-Procedure

Throughout the procedure, the patient's vital signs will be monitored as well the pulse oximetry. The patient needs to be assessed for chest pain and shortness of breath, signs and symptoms of bleeding, changes in cardiac rhythm, and presence of ST-segment or T-wave abnormalities. The nurse must also monitor for neurological status and peripheral vascular status, especially in the limb used for the procedure.

Vasovagal reactions can occur during sheath insertion and removal. If the patient's BP and HR drop and the patient starts to complain of dizziness, nausea, and possible vomiting, the nurse should administer IV fluids and atropine according to advanced cardiac life support guidelines until the HR and BP return to normal.

Post-Procedure

After the procedure the patient needs to be monitored for signs and symptoms of myocardial ischemia, thrombosis, and bleeding. Myocardial ischemia and possible thrombosis are assessed by chest pain; ST-segment changes, especially elevation on the telemetry; and shortness of breath. Changes in vital signs may include decreased oxygen saturation, a drop in BP, or a decrease or increase in HR. Dysrhythmias are assessed, especially if they cause hemodynamic compromise. The most common dysrhythmias are ventricular tachycardias, atrial bradycardias, and heart blocks.

Bleeding can include hematuria as well as bleeding from the catheterization site or other body orifices. The formation of a hematoma at the catheter insertion site can be a sign of internal bleeding. This is of particular concern with the femoral approach. The most common form of internal bleeding is retroperitoneal. Vital signs can also indicate more serious internal bleeding problems. The patient's hemoglobin, hematocrit, and platelet levels should also be

monitored. The platelet count may indicate the patient's tendency to bleed, especially if the patient has been given glycoprotein IIb/IIIa receptor inhibitors and/or tPA because these can cause thrombocytopenia. If the patient should start to bleed from the insertion site, the nurse can either apply manual pressure to the site, or use a special clamp that can be placed over the insertion site to maintain appropriate pressure.

Femoral access site closure may be accomplished by the use of Angio-Seal, which is a collagen sheath or patch. This method provides a secure method of achieving hemostasis following a femoral artery puncture. It allows the patient to achieve early ambulation and hospital discharge. The patient needs to be monitored for peripheral thrombosis and ischemia in the leg used for the procedure. Any change in pulses from the pre-procedure assessment should be reported immediately to the interventionalist. Upon return to the unit, the patient must keep the catheterized leg straight and remain on bedrest for 4 to 6 hours until femoral artery hemostasis occurs. Early sheath removal reduces post-procedure vascular complications.

Several devices have been developed for radial artery access to assist in maintaining pressure on the wrist; most are bands that allow easy modification of hemostatic pressure. The radial approach is the preferred access as it allows for improved patient comfort, early mobilization and decreased access-site complications. See Box 6-10 for a summary of Post PTCI/Catheterization Nursing Care.

Discharge Teaching

The patient should report any signs or symptoms of an MI or angina to the physician. The patient also needs to notify

Box 6-10 Post-PTCI/Catheterization Nursing Care

Monitor for signs of myocardial ischemia, thrombosis, and bleeding from the catheterization or sheath site.
 Monitor for reperfusion dysrhythmias.
 Monitor vital signs and the catheterization insertion site frequently. Report any significant changes to the interventionalist.
 Monitor the patient's hemoglobin, hematocrit, and platelet counts.
 Monitor the extremity below the insertion site for pulses. Report any changes to the interventionalist.
 Maintain the patient on bedrest with the catheterized extremity straight for 4–6 hours as ordered if femoral approach used.

the interventionalist if any signs or symptoms of infection or bleeding occur. These can include fever, swelling, oozing, or extension of bruising around the catheter insertion site. Pain, numbness, or tingling of the leg used for the procedure must also be reported.

Nursing Diagnoses

Patient with Acute Coronary Syndrome

- Acute pain related to imbalance between myocardial oxygen supply and demand
- Activity intolerance related to imbalance between oxygen supply and demand
- Anxiety related to situational crisis and life-threatening illness
- Decreased cardiac output related to alterations in preload and dysrhythmias
- Ineffective individual and family coping related to situational crisis of new diagnosis
- Ineffective tissue perfusion: myocardial
- Risk for ineffective therapeutic regimen management due to complexity of medical regimen

The patient will be discharged on antiplatelet medication, either aspirin or clopidogrel, a statin to lower lipids, and a beta blocker to reduce the cardiac workload. (These medications will be discussed later in the chapter.) Instructions on the proper administration of these drugs should be given to the patient. If the patient remains on antiplatelet medications, he must notify the physician of record so adjustments in the medication regime can be made prior to any medical procedure.

For the first 2 weeks at home, the patient should avoid excessive pressure on the puncture site or lifting items heavier than 8 pounds. The patient should not drive for 3 to 4 days or as directed. The patient can usually resume normal exercise, housework, and employment after a week.

Reflect On

A patient returned to the coronary care unit post PCI with an infusion of eptifibatide. Initial blood work post PCI showed a platelet count of 398,000. A second set of blood work sent 6 hours post procedure showed a platelet count of 150,000. What should the nurse do at this point?

Coronary Artery Bypass Grafting

Coronary artery bypass grafting (CABG) is a procedure in which the patient's diseased coronary arteries are bypassed with the patient's own venous (saphenous vein) or arterial (internal mammary artery or radial) vessels.

CABG is indicated for patients with ACS to relieve symptoms, improve quality of life, and, it is hoped, prolong life. Proper preparation of the patient and significant others increases the likelihood of a positive outcome for the patient. CABG is indicated for the patient who:

- Has failed PTCL.
- Greater than 70% stenosis of three major coronary arteries.
- Greater than 50% left main artery stenosis.
- Mild to moderate LV dysfunction (LVEF 35-50%) and proximal LAD disease.
- Multi-vessel coronary artery disease with diabetes mellitus (Hillis et al., 2012).

Pre-Operative Phase

Education is an important aspect of preparing the patient prior to surgery. When a patient knows what to expect, it assists with recovery, increases patient contentment, and decreases postoperative complications. Because cardiac surgery is anxiety producing, it is important for the nurse to assess the individual for needs and provide the information in a timely manner to decrease that anxiety. The experienced professional nurse individualizes preoperative instruction to meet the specific needs of the patient.

Information that may be included in preoperative teaching includes sights and sounds that will be experienced, invasive lines that will be inserted, anticipated pre-operative medications, and anticipated length of the operation. The nurse should also provide information on the expected postoperative course. Teaching should also include proper techniques to prevent respiratory complications, including the proper splinting of the incision for effective turning, coughing, and deep breathing. Patients also need to be reassured that pain will be managed during the postoperative period.

Teaching about insertion of an endotracheal tube during surgery should be stressed. The patient needs to know that he may not be able to speak when he wakes after surgery, but a nurse will be available close by to anticipate the patient's needs at that time. Increasingly patients are extubated in the operating room. If the patient is intubated after surgery, he needs to be informed that the endotracheal tube will be removed as soon as it is no longer needed. Pulmonary care should be stressed. The patient needs to be told that use of the incentive spirometer as well as coughing and deep breathing despite incisional pain are needed to ensure positive postoperative outcomes.

It is appropriate at this time to teach about the sternal precautions immediately after CABG. The patient should not lift more than 10 pounds or push or pull with her arms when moving in bed or getting out of bed. The patient should avoid deep bending and be cautioned not to hold her breath during activity. It is important to stress proper splinting of the chest when coughing or sneezing.

Nursing interventions for the significant others should include how communication will be handled during the intraoperative period and about the patient's expected appearance once the surgery is completed. The

Box 6-11 Important Preoperative Teaching Points

Sights and sounds of the peri-operative environment
 Insertion of monitoring lines
 Pre-operative medications and anticipated sensations
 Use of incentive spirometer
 Length of the operation
 Expectations of the post-operative environment
 Postoperative pain management
 Splinting of incision for pain control
 Sternal precautions
 Postoperative presence of an endotracheal tube
 Communication issues
 Postoperative activity
 Preparation of significant others

patient may appear pale, cool, and edematous due to the cooling of the body during surgery and the blood loss. The nurse should also explain the equipment they will see during the intensive care and postoperative course, including chest tubes, ventilator, nasogastric tubes, urinary catheter, and multiple intravenous and other invasive lines (Box 6-11).

Intraoperative Phase

Cardiac surgical patients undergo the insertion of a large-bore peripheral intravenous catheter, a central line catheter, an arterial line, and, if deemed necessary, a pulmonary artery catheter (Swan-Ganz). These are needed for the monitoring and stabilization of fluid balance and hemodynamics. The patient is also intubated. A TEE is obtained during surgery to assess optimal hemodynamics and guidance of inotropic therapy.

The standard surgical approach is via a median sternotomy. Sources of grafts or conduits can be the internal mammary artery, the saphenous vein, and/or the radial artery (♦ Figure 6-8).

Heparin is administered during surgery, and anticoagulation studies are performed at specific intervals to assess and guide the amount of heparin to be administered. The patient is placed on cardiopulmonary bypass, and cardioplegia is introduced. Cardioplegia is a cold solution that has a high concentration of potassium that is used to assist in stopping the heart.

Rewarming of the body occurs after the surgery to offset the surgically induced hypothermia. The intrinsic heart rhythm is often spontaneously reestablished once the rewarming starts and the cross-clamp is removed from the patient. Once the patient has a good return of BP and pulse, the cardiopulmonary bypass is removed and protamine sulfate is administered to reverse the effects of

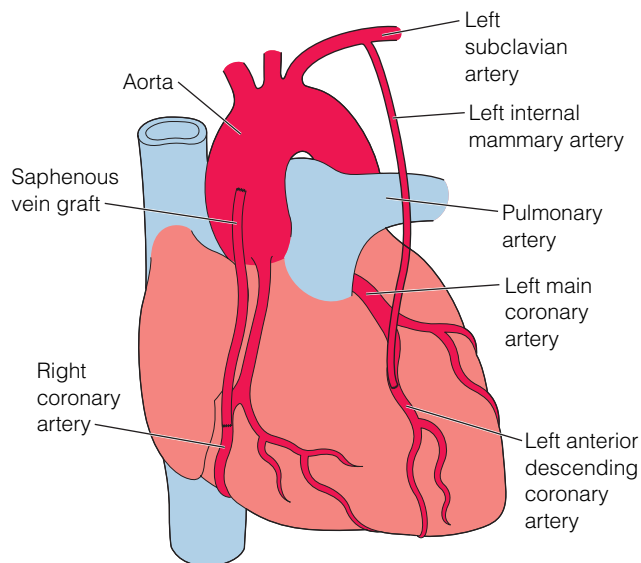


Figure 6-8 Coronary artery bypass graft showing internal mammary and saphenous vein graft.

the intraoperative heparin. Epicardial atrial and ventricular pacing wires are inserted at this time. Mediastinal and pleural chest tubes will also be inserted to drain mediastinal and pleural fluid. The sternum is then wired, the incision is sutured, and the patient is transported to the intensive care unit.

COLLABORATIVE CARE

The patient must be monitored carefully because changes are subtle and occur rapidly during this initial postoperative period. It is important for the nurse to anticipate the possible complications so the proper interventions can be initiated in a timely manner to ensure a positive outcome for the patient. While the admitting nurse is doing an overall assessment of the patient and connecting the monitoring equipment, the operating room nurse and anesthesiologist report on the patient's condition to a receiving nurse. The patient is transported immediately from the operating room to the Intensive Care Unit. Standard monitoring upon arrival to the critical care setting should include continuous ECG monitoring, pulse oximetry and invasive arterial BP monitoring.

- Attach the patient to the bedside cardiac monitor and notes the rate, rhythm and/or presence of epicardial pacing.
- Attach all pressure devices to the monitor; transducers are leveled and zeroed, noting pressure values and waveforms. If there is a PA catheter in place, obtain a cardiac output and index, noting the presence of inotropic or vasoactive drips. Obtain a cuff BP to correlate with arterial wave form.

- If the patient is extubated prior to transfer, oxygen should be continued via face mask. If the patient is intubated, lung sounds should be auscultated. The patient must be monitored for oxygen saturation via pulse oximetry.
- Check for peripheral pulses and perfusion signs.
- Monitor the chest tubes and note the character of the drainage, amount, color, and flow into the collecting chambers.
- Check urine output.
- Obtain initial laboratory studies: ABG, hemoglobin, potassium, glucose, coagulation profile, or per institutional protocol.

Pulmonary Management

Pulmonary dysfunction is a common complication of CABG surgery. Patient history and intraoperative complications must be considered for proper management postoperatively.

Some patients will be intubated and mechanically ventilated when they first arrive in the intensive care unit. Early extubation, adequate ventilation, and oxygenation are the desired outcomes for the patient. Studies have shown that there is an increase in pulmonary complications, especially ventilator-associated pneumonia (VAP) if the patient is intubated for longer than 16 hours postoperatively. It is the goal to have the patient extubated within 4 hours after surgery (Stephens & Whitman, 2015).

Postoperative management includes accurate and frequent assessment; arterial blood gas analysis and pulse oximetry; pulmonary care, including suctioning if intubated; and incentive spirometry after extubation. Early mobilization has also been shown to be important to decrease pulmonary complications postoperatively.

The nurse must assess for patient readiness for extubation. Extubation is considered when the patient is arousable, able to follow commands, hemodynamically stable, and initiating spontaneous ventilations without excessive respiratory effort. Specific hospital protocols and parameters must be followed for extubation. During the weaning process, the nurse should assess the patient for an increase in respiratory effort and HR, use of accessory muscles, fatigue, and color changes. These changes may indicate the patient is not ready for extubation (Box 6-12).

There will be times when a patient cannot be extubated in a timely manner. To prevent complications the Institute for Healthcare strategies to prevent ventilator associated pneumonia (VAP). See discussion in Chapter 3, "Care of the Patient with Respiratory Failure."

Post-Operative Management of Hemodynamics

Nearly all post cardiac surgery patients have myocardial dysfunction, vasomotor tone dysfunction and

Box 6-12 Indicators of Readiness for Weaning from Mechanical Ventilation

1. RSBI < 105
2. Respiratory rates
3. Maximal inspiratory pressure ng from
4. VT greater than 5 mL/kg,
5. Vital capacity greater than 10 mL/kg
6. $\text{SaO}_2 > 90$ with FiO_2 of 40% or less,
7. $\text{PaO}_2/\text{FiO}_2$ ratio of 150 or higher

decreased ventricular compliance. It is essential to monitor and treat preload, afterload and inotropic abnormalities. The overall goal of is to maintain adequate organ perfusion and oxygen delivery while protecting surgical anastomoses.

The nurse must monitor the volume in the circulatory system, which is reflected by BP, central venous pressure (CVP) and pulmonary artery pressures, cardiac index (CI) and systemic vascular resistance (SVR) if there is a Swan-Ganz line.

Low Blood Pressure. If the BP, CI, and CVP/PCWP are low, the patient will need replacement volume. Volume replacement should be with packed cells or a colloid solution depending on the cause of volume depletion. If the BP and CO are low but the CVP/PCWP is high, the patient may be experiencing a decreased contractility, and inotropic support with an agent such as milrinone should be instituted. If the BP is low, CI is adequate or elevated, the systemic vascular resistance (SVR) may be low, and the patient may need a vasoconstrictive agent such as phenylephrine or vasopressin. Some patients may experience rhythm disturbances that will cause a decrease in blood pressure. If the patient should become symptomatic, the nurse will need to initiate pacing with epicardial wires if bradycardic. The nurse should always assess the patient for adequate perfusion by assessing peripheral pulses, limb warmth, urine output and if necessary checking a lactate level (goal <2mmol/L) (Stephens & Whitcomb, 2015).

High Blood Pressure. Surgical anastomosis or cannulation sites can be disrupted in the setting of elevated BP. Nitroprusside, a vasodilator, is often administered to lower BP. Nitroglycerin may also be used to cause vasodilation as described previously in this chapter, and to lower the BP. These medications need to be started slowly and carefully titrated according to patient response. If the patient is responding poorly to either one of these

Box 6-13 Causes of Post-Operative Dysrhythmias

- Hypothermia
- Inhaled anesthetics
- Electrolyte disturbances (hypokalemia, hypomagnesemia, hypocalcemia, and hypercalcemia)
- Metabolic acidosis
- Manual manipulation of the heart
- Myocardial ischemia
- Increased catecholamine levels

medications, Cardene (nicardipine hydrochloride) is a viable alternative treatment.

If the patient is not normothermic, this may be accomplished with the use of warm blankets, warm humidified oxygen, convective air mattresses, and other procedures as dictated by individual hospitals. The negative effects of hypothermia include ventricular dysrhythmias, vasoconstriction, and depression of clotting factors.

Post-Operative Dysrhythmias. There are many factors that can cause dysrhythmias post-operatively, and management depends on the type of dysrhythmia and the patient's clinical response (Box 6-13). A BP should be obtained in the setting of a dysrhythmia to assess how the patient is tolerating the change in rhythm and/or rate. Because surgeons place epicardial pacing wires during surgery, temporary pacing can be instituted to override an improper intrinsic rhythm so adequate BP can be maintained. The intensive care nurse needs to utilize the protocols of the individual institution as well as current advanced cardiac life support protocols.

Atrial fibrillation continues to be a common dysrhythmia after coronary artery bypass grafting, especially when it is combined with coronary valve replacement surgery. This can occur in 20–40% of the patients. Postoperative atrial fibrillation generally peaks in prevalence on postoperative day two or three. Treatment for this dysrhythmia includes amiodarone, beta blockers, and anticoagulation (Stephens & Whitcomb, 2015).

Ventricular dysrhythmias are uncommon and should raise concern about ongoing ischemia. Amiodarone remains the drug of choice for these dysrhythmias. If ventricular dysrhythmias persist, an electrophysiology consult should be initiated, particularly for those individuals with a depressed ejection fraction (less than 40%).

Bradycardic dysrhythmias may occur due to trauma to conduction tissue. Atrial pacing with temporary epicardial pacing wires is the treatment of choice. If a bradycardic rhythm persists for greater than five days, a permanent pacemaker may be required.

Post-Operative Management of Bleeding

Many factors need to be considered when assessing the patient's potential for bleeding. Patients who were on anticoagulants and antiplatelet agents prior to surgery may be at risk for post-operative bleeding. These should be discontinued if the CABG at least five days before an elective case.

The nurse should monitor the chest tube drainage every 15 minutes initially then, if stable, hourly. A sudden increase or cessation of drainage should be reported immediately. Acceptable hourly chest tube drainage amounts vary per institution but should be well known to the nurse. The nurse should also monitor for signs of bleeding from surgical sites as well as clinical signs of hypovolemia due to blood loss. Hemoglobin and hematocrit should be monitored at regular intervals during the postoperative period according to physician orders and institutional protocol. Because bleeding increases the risk for blood to accumulate in the pericardium, the nurse needs to be cognizant of the signs and symptoms of cardiac tamponade. The clinical manifestations of tamponade include lack of chest tube drainage, decreased BP, narrowed pulse pressure, increased HR, jugular venous distention, elevated central venous pressure (CVP), and muffled heart tones. The patient may require an emergent opening of the chest and return to the OR to reverse the cause of tamponade.

Pharmacological intervention for abnormal coagulation studies may include protamine sulfate to reverse heparin, or antifibrinolytic agents such as aminocaproic acid or desmopressin (DDAVP) may be ordered. Blood products such as fresh frozen plasma, packed red cell, cryoprecipitate, platelets and other factor concentrates may also be ordered, especially when the patient is actively bleeding. If the patient is bleeding and all of the coagulation studies are within normal limits, there may be either a surgical bleed or continued bleeding from the sternal marrow. This requires an immediate return to the OR for surgical re-exploration.

Transfusion of blood products carries its own risks for cardiac surgery patients. Mortality and adverse cardiac events increase after only one or two units and is additive with additional transfusions. Most institutions utilize restrictive transfusion protocols with target hemoglobin of greater than 7.0 g/dL (Stephens and Whitman, 2015).

Post-Operative Neurological Management

CABG patients are at risk for neurological complications including delirium and cerebrovascular accident (CVA). Delirium occurs in about 50% of patients after cardiac surgery and has been associated with profound long-term consequences, including cognitive decline and mortality. Risk factors include benzodiazepine use and prolonged immobilization. CVAs can be caused by hypoperfusion

or an embolic event during or after surgery. The risk of CVA is approximately 2.5%.

The nurse needs to routinely perform a complete neurological assessment of the patient. This should include pupil size and reactivity, the ability to follow commands, equality of strength of the extremities, and the ability to move all extremities. The patient should be assessed for orientation to time, place, and person.

Post-Operative Renal Management

Renal insufficiency can be caused by advanced age, hypertension, diabetes, decreased left ventricular function, and the length of time on cardiopulmonary bypass. Normal urinary output should be at least 0.3 mL/kg/hr. The patient will have a foley catheter in place and the urinary output should be monitored hourly for the first 24 hours postoperatively. The patient's potassium level, BUN and creatinine should be measured closely. Intravenous potassium may be administered via a central line to keep levels within normal limits.

Post-Operative Glycemic Control

Adequate control of postoperative hyperglycemia reduces the risk of deep sternal wound infection, all infections, sepsis and mortality (Stephens & Whitman, 2015). Current guidelines support maintaining serum glucose less than 180 mg/dL for the first 24 hours. Insulin infusions are initiated in the OR then coverage is transitioned to subcutaneous sliding scale after the first 24 hours.

Post-Operative Gastrointestinal Management

Complications can include gastritis, perforated ulcer, pancreatitis, cholecystitis, bowel ischemia, and liver dysfunction. The risk factors for gastrointestinal (GI) dysfunction include age over 70, history of GI disease, history of alcohol abuse, emergent operation, prolonged cardiopulmonary bypass, postoperative hemorrhage, use of vasopressors, and low post-operative CO.

The nurse should monitor the patient's bowel sounds, abdominal distention, and nausea and vomiting. The patient will have a nasogastric tube in place, so placement and assessment of the amount, color, and characteristics of drainage should be noted. After discontinuance of the nasogastric tube, the nurse should continue to assess for potential complications. The nurse can administer antiemetics, histamine-blocking agents, and proton pump inhibitors as ordered by the physician. When the nasogastric tube is removed, the patient will be started on a clear liquid diet. The diet will be advanced as the patient tolerates food and fluids.

Post-Operative Pain Control

Effective pain control is important for patient comfort, hemodynamic stability, and to prevent pulmonary complications.

Without adequate analgesia, the HR and BP can increase and the blood vessels constrict, causing an increase in cardiac workload and myocardial oxygen demand.

Pain control is achieved with intravenous push opioids in the immediate postoperative period, then switched to oral forms by the second or third postoperative day, as tolerated. If sedation is needed, initial use of intravenous propofol (Diprivan) or dexmedetomidine (Precedex) are both appropriate choices. Analgesics, positioning, distraction, and relaxation techniques can also be used to control pain until the patient is able to tolerate and maintain control with oral narcotic agents. The nurse must assess for respiratory depression when the patient is using narcotic pain relievers. In addition to opioid analgesics, a nonsteroidal anti-inflammatory agent, ketorolac (Toradol), can be used as an adjunct and to decrease the amount of narcotic pain relievers administered. The patient needs to be monitored for renal status and bleeding while on ketorolac, and discontinue use if the serum creatinine is too high or the platelet count is too low.

Post-Operative Infection

There is a systemic inflammatory response and leukocyte activation associated with the use of cardiopulmonary bypass which contributes to the possibility of postoperative infection. Additional sources may include respiratory, incisional, invasive lines, and tubes. The nurse must monitor for increased temperature, leukocytosis, drainage or redness from incisions, increased production or change in color of sputum, or complaints consistent with urinary tract infection. Risk factors for infection include diabetes, malnutrition, and patients undergoing emergent or prolonged surgery.

Minimally Invasive Direct Coronary Artery Bypass

If a patient presents with lesions to the LAD only, the patient may be referred for a minimally invasive direct coronary artery bypass (MIDCAB). This is also called off-pump bypass or beating heart surgery. This approach utilizes a thoracotomy approach, and cardiopulmonary bypass is not required during the procedure. During the procedure, a 2-inch incision is made at the level of the fourth rib. This rib is then removed for access to the **left internal mammary artery (LIMA)**. The LIMA is then dissected and anastomosed to the level below the lesion in the LAD while the heart is still beating (Hillis et al., 2012). After the surgery the patient needs to be monitored for signs of acute graft closure, including a new presence of Q waves and ST-segment changes in leads V₂ through V₆.

Post-operative care is focused on pain management, assessing for dysrhythmias or ischemia, and adequate oxygenation. Although the patient does not go on bypass, respiratory function may be compromised by the surgical

incision and pleural chest tube. The patient recovers faster from the MIDCAB, spending less time in and is often discharged from the hospital within 2 to 3 days.

Transmyocardial Laser Revascularization (TMR)

TMR is a surgical procedure pioneered in the 1990s that provides angina relief to patients with diffuse coronary disease. The theory behind improvement of angina symptoms involves myocardial angiogenesis leading to increased perfusion. This may be an isolated procedure or in used combination with other procedures, such as CABG. If used as a sole procedure, TMR may be done without the use of cardiopulmonary bypass or anticoagulation. The patient will receive general anesthesia with a double-lumen endotracheal tube to isolate the left lung. Exposure to the left ventricle is through a left anterolateral thoracotomy. A laser beam is then utilized to create long, narrow channels into the left ventricle. These will allow oxygenated blood to travel to the cardiac muscle during diastole. In most cases, the patient is extubated in the operating room. Research is currently focusing on using adjuvant stem cell therapy to better improve functional outcomes in patients (Kindzelski, Zhou, & Horvath, 2015).

Innovative Therapies

Several active areas of investigation hold promise for future advances in cardiovascular science and medicine, including genetics and genomics. Molecular targeting focuses on molecular pathways and is essential to the development of therapeutics. Pharmacogenomics is the investigation of genetic variability in patients' responsiveness to a drug in order to inform clinical decisions about dosing and selection, stem-cell biology and regenerative medicine. There is also continued progress in the use of robotics in cardiac surgery.

NURSING CARE

The care of the patient with ACS not only includes the hospitalization, but many months of recuperation and rehabilitation. The patient will be monitored by the cardiologist and nursing staff, and the plan of care will include medications, anxiety control, exercise management, and nutrition management. As with any program that involves a lifestyle change, frequent contact with healthcare providers is highly suggested to provide time for evaluation and encouragement to continue with the promoted changes.

Pharmacological Therapies

The patient will be discharged from the hospital after an acute coronary event on multiple medications. These medications will include ones that control platelet adhesion, prevent the expenditure of myocardial oxygen, and prevent ventricular remodeling so that the complication of congestive heart failure is less likely to develop. See "Commonly Used Medications."

Risk Factor Modification

The strategy of changing lifestyle habits has been demonstrated to be successful in the patient with coronary syndromes. Patients need interactions with healthcare providers in the form of personal contact, phone surveys, and support groups. Therapeutic lifestyle changes, including smoking cessation and weight control with exercise, along with medications, are found to be independent, but both contribute to the patient having successful outcomes.

Cardiac Rehabilitation Programs

The particular goal of a cardiac rehabilitation program is to promote lifestyle changes and secondary prevention of CHD. The programs strive to improve both the quantity

Safety Initiatives

Patterns of Statins Initiation, Intensification, and Maximization Among Patients Hospitalized with Acute Myocardial Infarction

PURPOSE: Assess statin use at admission and discharge among acute myocardial infarction patients.

RATIONALE: Intensive statins are superior to moderate statins in reducing morbidity and mortality after an acute myocardial infarction. Although studies have documented rates of statin prescription as a quality performance measure, variations in hospitals' rates of initiating, intensifying, and maximizing statin therapy after acute myocardial infarction are unknown.

HIGHLIGHTS OF RECOMMENDATIONS: Rates of statin intensification and maximization were low and varied substantially across hospitals. Given that more intense statin therapy is associated with better outcomes, changing the existing performance measures to include the intensity of statin therapy may improve care.

Source: Arnold et al. (2014), Patterns of statin initiation, intensification, and maximization among patients hospitalized with an acute myocardial infarction. *Circulation*, 129(12), 1303–1309.

and the quality of life by alleviating the psychological and physiologic constraints in the form of reducing risks, managing symptoms, and having patients regain control of their lives. The programs consist of a monitored exercise program and an educational program on lifestyle changes. The length of the programs varies from program to program.

The monitored exercise program is usually supervised by a physiotherapist and a registered nurse who assesses the patient's HR, BP, ECG, and cardiac symptoms during the exercise session. This is to provide data for compliance and exercise tolerance so complications can be avoided.

The programs consist of three phases. Phase 1 is provided on an inpatient basis. It is started a few days after the coronary event and ends at discharge. Phase 2 is an outpatient exercise/educational program and can last for

8 to 12 weeks. After completing the outpatient program, some patients elect to continue on to a Phase 3 or maintenance program. In this program the patients are in a controlled setting for exercise but are no longer monitored for ECG changes. If the patient does not elect to go on to a maintenance program in the hospital, a home exercise program is given to maintain the lifestyle change of physical activity.

Physical Activity. Physical activity provides a multitude of benefits for the cardiac patient. An exercise program can include aerobic and resistance training. Aerobic exercise has been found to positively affect lipid levels, hypertension, diabetes, weight, and obesity. Resistance training has a positive correlation with muscle mass, bone density, and functional capacity. Physical activity also has a positive correlation with psychological health.

Commonly Used Medications

At Discharge Following an Acute Coronary Event

Aspirin

Long-term aspirin therapy significantly reduces the risk of MI, stroke, and vascular death among patients with CAD. Taking a maintenance dose of 81 to 325 mg/day significantly reduces the risk of reinfarction. Any STEMI patient without aspirin sensitivity should take a maintenance dosage indefinitely, unless contraindications are present.

Desired Effects: Inhibit platelet aggregation and prevent reoccurrence of MI or ACS.

Nursing Responsibilities:

- Monitoring the platelet count, especially in patients with concurrent antithrombotic therapy.
- Monitoring the patient for signs and symptoms of bleeding.

Beta Blockers

Continued beta-blocker use post ACS reduces long-term mortality by 23%. These medications should be continued indefinitely in all patients after an MI and ACS. Drug selection is based on the desired mechanism for the patient. Beta blockers such as metoprolol and atenolol are cardioselective. Others, including labetalol, have both alpha- and beta-blocker properties and may be prescribed for concurrent management of such disorders as hypertension.

Desired Effects: See "Commonly Used Medications" earlier in the chapter. When effective, the patient has a slower heart rate, less cardiac irritability, and is able to be more active without chest discomfort.

Nursing Responsibilities:

- Administering the appropriate dose of the indicated medication such as carvedilol (Coreg); atenolol (Tenormin);

metoprolol tartrate (Lopressor); metoprolol succinate (Toprol); propranolol hydrochloride (Inderal); and labetalol.

- Obtaining a history to rule out asthma and chronic obstructive pulmonary disease (COPD).
- Monitoring apical pulse, respiration, BP, and circulation to extremities closely during dose adjustment.
- Monitoring for signs and symptoms of heart failure.
- Instructing the patient not to fast for more than 12 hours because the drugs can cause hypoglycemic effects.

Side and/or Toxic Effects: Common long-term adverse effects are fatigue and weakness that, if accompanied by impotence, may cause a patient to stop complying with therapy. Bradycardia and heart failure potentially resulting in pulmonary edema are some of the most serious side effects. However, because beta blockers may mask the symptoms of hypoglycemia, it can be dangerous if it develops.

ACE Inhibitors

By causing vasodilation, these drugs reduce afterload, the pressure against which the heart must pump, and thus its workload. ACE inhibitors help decrease ventricular remodeling after MI and may decrease the risk of heart failure. Long-term use also may improve the endothelial integrity in abnormal segments of the coronary arteries. These drugs have also been shown to be effective for BP control and stroke prevention.

Desired Effects: ACE inhibitors improve CO and exercise tolerance due to inhibition of ACE. They decrease peripheral resistance or afterload and pulmonary vascular resistance. Effectiveness may be noted by a decrease in blood pressure for a hypertensive patient, fewer symptoms of CHF, and survival following MI or ACS.

(Continued)

Commonly Used Medications (*Continued*)

Nursing Responsibilities:

- Administering the appropriate doses of benazepril hydrochloride (Lotensin); captopril (Capoten); enalapril maleate (Vasotec); lisinopril (Prinivil, Zestril); ramipril (Altace).
- Watching for postural hypotension when initiating therapy.
- Monitoring blood pressure before dosing and holding per agency protocol if BP is too low.
- Monitoring for cough, which may occur at any time during therapy.
- Monitoring creatinine, potassium and sodium. The patient may develop hyperkalemia, hyponatremia and increase in creatinine.

Side and/or Toxic Effects: The most common adverse effects are hypotension (which occurs most commonly in patients who are hyponatremic), an increase in serum potassium, and cough.

Cholesterol-Lowering Medications

Achieving and maintaining normal serum lipid levels produces reductions in recurrent MI, coronary death, stroke, and coronary artery procedures. The most commonly prescribed dyslipidemia medications are the statins including simvastatin (Zocor), rosuvastatin (Crestor), and atorvastatin calcium (Lipitor). Dosing is determined by serum levels and the presence of heart disease or other major risk factors (Martin et al., 2014). Other medications may include bile sequestrants, nicotinic acid and fabric acids.

Drug: HMG-COA Reductase Inhibitors (Statins)

Desired Effects: These medications increase HDL cholesterol while decreasing LDL cholesterol and total cholesterol synthesis. They are used for patients with hypercholesterolemia and familial hypercholesterolemia; when effective, they reduce the risks of coronary death and nonfatal MI.

Nursing Responsibilities:

- Administering the appropriate dose of the following medications: lovastatin (Mevacor) simvastatin (Zocor); atorvastatin (Lipitor); rosuvastatin (Crestor).
- Obtaining baseline and periodic liver function tests.
- Monitor cholesterol levels.
- Monitoring INR: If the patient is on concurrent anticoagulants, the INR may be prolonged.
- Reporting unexplained muscle pain.
- Instructing the patient to not take the medication with grapefruit juice.

Nicotinic Acid (Nicobid, Niaspan)

Desired Effects: Nicotinic acid inhibits hepatic synthesis of very low density lipoproteins (VLDL), cholesterol, and

triglyceride, and indirectly LDL. Large doses effectively reduce elevated serum cholesterol and total cholesterol in hypercholesterolemia. It is used as adjuvant therapy to patients with hyperlipidemia who do not respond to diet and weight loss.

Nursing Responsibilities:

- Ensuring administration of therapeutic dose 1.5 to 3 grams daily with a maximum dose of 6 grams daily.
- Monitoring for therapeutic effectiveness.
- Monitoring diabetics for increase in blood sugar.
- Telling patients they may feel warm and experience a flushed face within 2 hours after ingestion.

Fibric Acid Derivatives (Fibrates)

Desired Effects: Fibrates decrease VLDL and triglyceride synthesis, produce a moderate increase in HDL cholesterol levels, and reduce levels of total and LDL cholesterol and triglycerides. They are used for patients with high triglycerides who have not responded to diet restrictions.

Nursing Responsibilities:

- Ensuring administration of the therapeutic dose.
- Monitoring cholesterol and triglyceride levels.
- Notifying the physician if the patient presents with signs and symptoms of cholelithiasis or cholecystitis.

Nitroglycerin

Action and Expected Outcomes: Nitroglycerin increases the coronary blood flow by dilating the coronary arteries. Nitroglycerin has also been shown to potentiate the hypotensive effects of patients who take drugs for erectile dysfunction including sildenafil (Viagra), tadalafil (Cialis), or vardenafil (Levitra).

Desired Effects: Nitroglycerin reduces systolic, diastolic, and mean BP and produces antianginal, anti-ischemic, and antihypertensive effects. When effective, nitroglycerin prevents angina pectoris, controls CHF related to MI, and controls hypertension in the cardiac patient.

Nursing Responsibilities:

- Ensuring administration of a therapeutic dose: Sublingual: 0.3 to 0.4 mg q3–5 min (maximum dose three tablets in 15 minutes); Transdermal: Apply once every 24 hours (12 hours on–12 hours nitro free); Topical: 1 to 1½ inches of ointment every 4 to 6 hours.
- Teaching the patient to sit or lie down before taking sublingual nitroglycerin and to keep sublingual tablets in a dark bottle with the top tightly sealed to prevent moisture from deactivating the medication.
- Checking BP until the patient's response is known if the patient recently initiated treatment.

Side and/or Toxic Effects: The most common adverse effects are headache and hypotension.

The recommendation is for all adults to participate in a program of 30 minutes of moderate physical exercise for at least 5 days a week.

Smoking Cessation. The patient with an ACS is often forced to quit smoking during hospitalization. Many patients can continue with the elimination of smoking post-cardiac event, but it has been found that some patients relapse into the smoking habit after about 3 months at home. Predictive factors of failure of a smoking cessation program include a previous medical event of a coronary nature; use of sedatives and antidepressants; nonparticipation in a cardiac rehabilitation program, and a higher level of cigarette consumption prior to the coronary event.

Patients who participate in a cardiac rehabilitation program have been reported to have greater success at smoking cessation. This can be attributed to the patient's motivation to ensure a healthier lifestyle as well as peer support throughout the program.

Cigarette consumption per day is considered an indicator of nicotine dependency. It has been found that the higher average consumption is associated with a patient's continuing to smoke after a coronary event, whereas a patient who smokes a few cigarettes, or is not as dependent on nicotine, has a better chance of being successful in a smoking cessation program.

With short-term stay hospitalizations, it is often difficult for nurses to find time to broach the subject of smoking cessation with the patient. Literature supports that counseling by nurses and other health professionals during a patient's admission will increase the chances of the patient being successful at smoking cessation. Healthcare professionals need to invest more time and effort in assisting patients to stop smoking, and provide the proper follow-up with the patient to ensure adherence to the program.

Nutrition

Nutrition and cardiovascular health education should include dietary changes to control lipids, hypertension, diabetes, and

Box 6-14 Dietary Recommendations

- Use up at least as many calories as you take in.
- Aim for at least 40 minutes of physical activity on most, if not all, days.
- Eat a diet rich in vegetables and fruit
- Choose whole-grain, high-fiber foods.
- Eat skinless poultry and fish.
- Limit how much saturated fat, trans fat, and cholesterol you eat.
- Select fat-free, 1-percent fat, and low-fat dairy products.
- Cut back on foods containing partially hydrogenated vegetable oils to reduce trans fat in your diet.
- Cut back on beverages and foods high in calories and low in nutrition, such as soft drinks and foods with added sugar.
- Choose and prepare foods with little or no salt.
- If you drink alcohol, drink in moderation.

SOURCE: Eckel, R. H., Jakicic, J. M., Ard, J. D., de Jesus, J. M., Miller, N. H., Hubbard, V. S., . . . Yanovski, S. Z. (2014). 2013 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Journal of the American College of Cardiology*, 63 (25_PA), 2960–2984.

overall obesity. There are many diets on the market for a patient to follow, but ones recommended by the AHA and the American Diabetes Association appear to be the healthiest and easiest to meet the patient's particular needs (Box 6-14).

Dyslipidemia. LDL cholesterol lowering can be achieved by reducing saturated fats to less than 7% of total calories and consuming less than 200 mg of cholesterol. The National Cholesterol Educational Panel (NCEP) also recommends an intake of monounsaturated fatty acids of up to 20% of total calories. Carbohydrates have been shown to be effective when used to replace fats in the diet; however, they also tend to increase triglyceride levels and decrease HDL. Therefore, it is recommended that

Gerontological Considerations

The leading cause of death in patients 65 years of age and older in the United States is cardiovascular disease. It is also one of the major causes of ED visits, hospitalization, morbidity, and disability in older adults. Older adults, especially older women, may manifest acute coronary events differently than younger adults and men. Older women may be less likely to experience the classic chest discomfort and more likely to have nonspecific discomfort, dyspnea, diaphoresis, and fatigue.

Because age often excludes patients from clinical trials and research studies, there is not sufficient evidence to say whether cardiovascular disease ought to be managed differently in older adults than younger individuals. However, secondary prevention strategies have been shown to be effective. When older adults participate in light to moderate physical activity and have hypertension managed aggressively, their outcomes are better. Older adults are more likely to become depressed and isolated following a cardiovascular event.

Commonly Used Medications

Medications Used for Smoking Cessation

Nicotine

Desired Effect: To reduce withdrawal that accompanies smoking cessation. It is used in conjunction with a medically supervised behavior modification program and a temporary and alternate source of nicotine by the nicotine-dependent smoker who is withdrawing from cigarette smoking.

Nursing Responsibilities:

- Ensuring administration of the appropriate dose: Gum: Chew 1 piece of gum whenever there is an urge to smoke; do not exceed 20 pieces of gum per day; Intranasal: 1 dose = 2 sprays, 1 to 2 doses every hour with a maximum of 5 doses per hour (40 doses per day); Topical: Apply one patch every 24 hours in gradually decreasing strengths.
- Monitoring for skin reactions when using the transdermal approach.
- Reviewing with the patient the explicit instructions of drug use; gradually decreasing doses of all nicotine replacement forms.
- Instructing the patient to not smoke while using the transdermal approach.

Bupropion Hydrochloride (Wellbutrin, Zyban)

Desired Effect: Bupropion is an antidepressive agent used for smoking cessation.

Nursing Responsibilities:

- Administering the drug as indicated: Start with 150 mg once daily for 3 days, then increase to 150 mg twice a day for 7 to 12 weeks.
- Monitoring for therapeutic effect; do not abruptly discontinue the drug.

Varenicline Tartrate (CHANTIX)

Desired Effect: By binding to nicotinic acetylcholine receptor subtypes, it reduces the craving to smoke and nicotine withdrawal symptoms. If effective, the patient will be able to quit smoking.

Nursing Responsibilities:

- Administering the appropriate dose 0.5 mg once a day on days 1 to 3, followed by 0.5 mg twice a day in the morning and evening on days 4 to 7, then 1 mg twice a day from day 8 until the end of the 12-week course of treatment.
- Teaching the patient to start 1 week before he or she plans to stop smoking.
- Instructing the patient to take the drug with a full glass of water after eating.

carbohydrate intake be limited to 60% of total calories. Fiber has also shown positive effects on lipid management, and it is recommended that the patient receive a minimum of 5 to 25 grams of soluble fiber per day. Americans typically eat six or seven meals away from home each week, so they must also be aware of how to eat reasonably when eating out.

Hypertension. The Dietary Approaches to Stop Hypertension (DASH) study showed that a diet low in saturated fat, cholesterol, and total fat and one that emphasizes fruits, vegetables, and low-fat dairy products reduces BP. The DASH diet also provided information on sodium control in the diet. It was found that the largest reduction in BP is with a sodium intake of less than 1500 mg per day. In order to adhere to these dietary constraints the patient needs to be taught how to read food labels, avoid processed food, and use an alternative seasoning to salt.

Diabetes. Because diabetic patients present with a higher risk for ACS, they should receive individualized attention on dietary management. Because they usually present with comorbidities of increased cholesterol, hypertension,

and obesity, following the above recommendations plus management of glucose control will give these patients some control over their dietary habits. The diabetic patient must maintain tight glycemic control. The goal is to maintain a hemoglobin A_{1c} (HbA_{1c}) of less than 7% (American Diabetes Association, 2015).

Weight Management

Obesity is a risk factor highly suggestive of second coronary events, especially in the older population. The obese patient presents with the comorbidities of hypertension, hypercholesterolemia, and diabetes. By controlling weight with a medically supervised weight loss program, the patient should also find success in controlling the comorbid conditions.

Anxiety/Depression

Depression has emerged as a risk factor for ACS. It has been estimated that up to 30% of patients experience depression after a coronary event. Depression is associated with reduced participation in cardiac rehabilitation programs. The thoughts of a person who is depressed often

accompany feelings of worthlessness, hopelessness, and even death. Anxiety often coexists with depression in a patient with ACS. Anxiety may present itself with feelings of tension and impending catastrophe as well as insecurity, inadequacy, and helplessness. In a recent study, it was found that a primary depressive episode after an MI signals a considerably greater risk for a future coronary event.

It is important for the nurse to understand depression and anxiety in order to enhance patient recovery from the coronary event. Patients often feel that they are to blame for their health situation and that they have no control over what is going to happen over the course of rehabilitation.

Patients' anxiety and depression can be controlled by medications, but often they feel that having a nurse or medical team member keeping them informed of the situation gives them more control. Patients need to have an emphasis on psychoeducational programs, such as stress management and relaxation techniques, in the rehabilitation setting. It is important for the nurse to identify the patients' current coping strategies. The nurse can assist patients in attaining the appropriate coping mechanisms by keeping them informed and helping them sort out the important information they need to be successful in the rehabilitation process. Nurses also can assist patients to adapt and use the appropriate defense mechanisms for healthy control of depression and anxiety.

Medications commonly used to treat depression and anxiety in the post-ACS patient include the serotonin selective reuptake inhibitors (SSRI), anxiolytics, and benzodiazepines.

Sexuality Concerns

Patients are often confused about returning to sexual relations after either an MI or a cardiac surgical intervention. Most patients can return to a healthy sexual life after a cardiac event. If a patient successfully finishes a cardiac rehabilitation program, the risk of experiencing an additional risk as a result of sexual activity is 20 in a million. The energy expended during a sexual encounter is equivalent to walking at a fast pace on level ground or climbing a flight of stairs. It is currently believed that a patient should be ready to resume sexual activity within 6 to 8 weeks of an MI or after being cleared by a post-myocardial treadmill test. Many patients wait longer because of fear of another event. Patients should be warned to avoid sexual activity for several hours after eating a heavy meal, after excessive alcohol intake, in extreme temperatures, or while wearing restrictive clothes. Sexual dysfunction after an MI is often attributed to depression and/or medication side effects.

Prevention Guidelines for Women

Along with the routine lifestyle and drug therapies, daily administration of omega-3 fatty acids and screening for

depression is recommended for women. Also, the potential risks and benefits of hormone replacement therapy, antioxidant vitamins, folic acid supplementation, and aspirin as a preventative should be discussed.

Complications

Many complications can occur as the result of an episode of ACS. The four main conditions are congestive heart failure, cardiogenic shock, dysrhythmias, and pericarditis.

Heart Failure

Heart failure is one of the more common complications of a coronary event. The more common events that lead to heart failure are left ventricular dysfunction due to decreased blood supply and decreased ventricular wall motion; the rupture of the intraventricular septum; the rupture of the papillary muscles, causing valvular dysfunction; and a right ventricular infarction. These etiologies cause a reduction in the amount of blood the heart can eject.

Cardiogenic Shock

Cardiogenic shock needs to be assessed and treated quickly. This is the leading cause of mortality among the complications of ACS and has a mortality rate of 50% to 60% even with maximal therapy. It often occurs in patients when 40% or more of the left ventricle is damaged. The patient usually presents with:

- Tachycardia.
- Hypotension.
- Decreased urine output (less than 0.5 mL/kg/hr).
- Cold, clammy skin.
- Agitation.

Dysrhythmias

Dysrhythmias can occur in up to 95% of all patients with an MI and are frequently the cause for post-MI mortality. The location of the MI is important to determine the type of dysrhythmias the patient will demonstrate. Careful hemodynamic monitoring of the patient is essential for proper treatment of the patient.

Anterior MIs are associated with the release of catecholamines, which increase HR, contractility, and BP. This type of MI often produces tachydysrhythmias, including sinus tachycardia and supraventricular tachycardias (SVT). The supraventricular tachycardias can include atrial fibrillation, atrial flutter, paroxysmal atrial tachycardia, and accelerated AV junctional rhythms. Ventricular irritability is also common for the patient with an anterior

Table 6-8 Differentiation Between Angina, Myocardial Infarction, and Pericarditis

Angina	Myocardial Infarction	Pericarditis
Substernal chest discomfort radiating to left arm	Substernal chest discomfort radiating to left arm, jaw, or back	Substernal chest discomfort increasing with inspiration
Pain relieved with rest or nitroglycerin	Pain relieved with morphine sulfate	Pain relieved with ASA and NSAIDs
No specific associated symptoms	Associated symptoms include nausea, diaphoresis, dyspnea, fear and anxiety, fatigue, dysrhythmias	Associated symptoms include friction rub, elevated white count, elevated temperature, dysrhythmias

MI. A common cause for ventricular irritability is hypokalemia. The patient needs to be monitored frequently for the potassium level, and replacement should be given if the level falls below 3.5 mg/dL.

Inferior and posterior infarctions are associated with a parasympathetic response. In addition to the signs and symptoms of nausea, vomiting, and hypotension, this type of MI produces bradycardias and junctional escape rhythms.

AV blocks are also associated with acute MIs. These conduction defects are also associated by location of the myocardial damage. First-degree and second-degree type 1 occur often in inferior MIs. These rhythms are often transient and require no treatment.

Second-degree type II AV blocks are more likely to occur with anterior or anteroseptal MIs. The third degree of complete heart block is associated with inferior and anterior MIs. These blocks are less common, but more life-threatening for the patient, because they indicate that a large portion of the left ventricle has been damaged. Second-degree and third-degree heart blocks are often treated with a temporary pacemaker until the dysrhythmia resolves or the patient is able to receive a permanent pacemaker.

Pericarditis and Post-Pericardiotomy Syndrome

Pericarditis is an inflammation of the pericardium, and it is often characterized by chest pain and ECG changes. The pain is located in the anterior precordium, which can radiate to the upper abdomen, upper arms, and back. The pain usually increases with inspiration and is relieved by having the patient lean forward (Table 6-8). When pericarditis occurs after a cardiac surgical procedure, it is often called post-pericardiotomy syndrome. In addition to pericarditis the patient also presents with a low-grade fever, pericardial effusions, pleuritis, and an elevated white count. It is often thought to be an autoimmune response.

Pericarditis and post-pericardiotomy are often accompanied by pericardial friction rubs. This syndrome presents days to weeks after an MI or cardiac surgery and usually resolves without intervention, but many healthcare professionals treat it with the use of anti-inflammatory medications (aspirin [ASA], NSAIDs, and corticosteroids) to reduce the inflammation and pain.

Acute Coronary Syndrome Summary

Although CHD remains the leading cause of death in the United States, dramatic improvements in patient outcomes and survival rates have occurred over the past 20 years. This is primarily due to the advancements in cardiac reperfusion strategies and advances in medication therapy. The

nurse has essential responsibilities for the early identification and management of the patient experiencing an acute coronary event, care of the patient following intervention and/or surgery, and preparing the patient for cardiac rehabilitation and self-care.

Why/Why Not?

Ms. M, a 63-year-old woman presents to the emergency department with complaints of intermittent epigastric and chest discomfort that has been occurring over the past week. She also complains of fatigue, periods of shortness of breath and lightheadedness. On exam, her BP is 190/100 and HR 90 beats per minute. She has not been taking her antihypertensive meds. Her 12-lead EKG

shows no sign of ischemia but did show a pathologic Q wave in leads II, III, aVf. Initial troponin I is borderline. Should Ms. M go directly to the cardiac catheterization lab? Why or why not?

See answers to Why/Why Not? in the Answer Section.

Case Study

A 59-year-old male and successful business manager is transported to the hospital after experiencing chest pain at work. Upon arrival to the ED, you notice the patient clutching his chest and moaning. He states that the pain is in the middle of his chest and radiating to his jaw and left arm. You obtain a partial history from the patient.

Subjective data

- States he has been working overtime for the past week, trying to obtain a multimillion dollar contract for his firm
- States he has been told to lose weight but has not had the time to go on a diet or exercise
- Recently divorced
- Has two children in college and one teenage son
- Is borderline diabetic

Objective data

- Weight: 250 lb (114 kg)
- Height: 5 feet 11 inches
- Diaphoretic, pale, and short of breath
- BP 170/100
- Pulse 110 beats/min and slightly irregular
- Respiratory rate 24

- Temperature 99
- Oxygen saturation 96%

Diagnostic studies

- ECG shows ST elevation in leads V₁–V₄
- Cardiac-specific enzyme troponin I = 12
- Total cholesterol level 275 mg/dL, LDL 120 mg/dL, HDL 35 mg/dL
- Creatinine phosphokinase 300
- Blood glucose 130

1. What are some of the causes of his chest pain?
2. What is the significance of the ECG results?
3. What is the possible pathophysiology of the patient's condition? How is it different from ACS?
4. What factors are contributing to the patient's medical condition?
5. What other lab values would the nurse need to evaluate? Why?
6. What should the immediate management strategies be for this patient? Why?

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 6.1 Why is aspirin given to the patient immediately upon arrival to the ED?
- 6.2 What is troponin and why is it a useful test in patients with chest pain?
- 6.3 What are the electrolyte disturbances the nurse needs to be aware of with the ACS patient? Which is the most important? How is it treated?
- 6.4 How is pain managed in the ACS patient?
- 6.5 Why would a patient undergo a CABG surgery rather than a PCI?
- 6.6 What are the advantages of fibrinolytic therapy for the patient?
- 6.7 What are the complication of an MI? What are the causes of these complications?

See answers to Chapter Review Questions in the Answer Section.

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Chapter 7

Care of the Patient Experiencing Heart Failure

Carrie Edgerly MacLeod, PhD, APRN-BC

Abbreviations

ADHF	Acute Decompensated Heart Failure	HF_rEF	Heart Failure with Reduced Ejection Fraction
BNP	B-Type Natriuretic Peptide	LVEF	Left Ventricular Ejection Fraction
CO	Cardiac Output	RAS	Renin-Angiotensin-Aldosterone System
HF	Heart Failure	SNS	Sympathetic Nervous System
HF_pEF	Heart Failure with Preserved Ejection Fraction		



Learning Outcomes

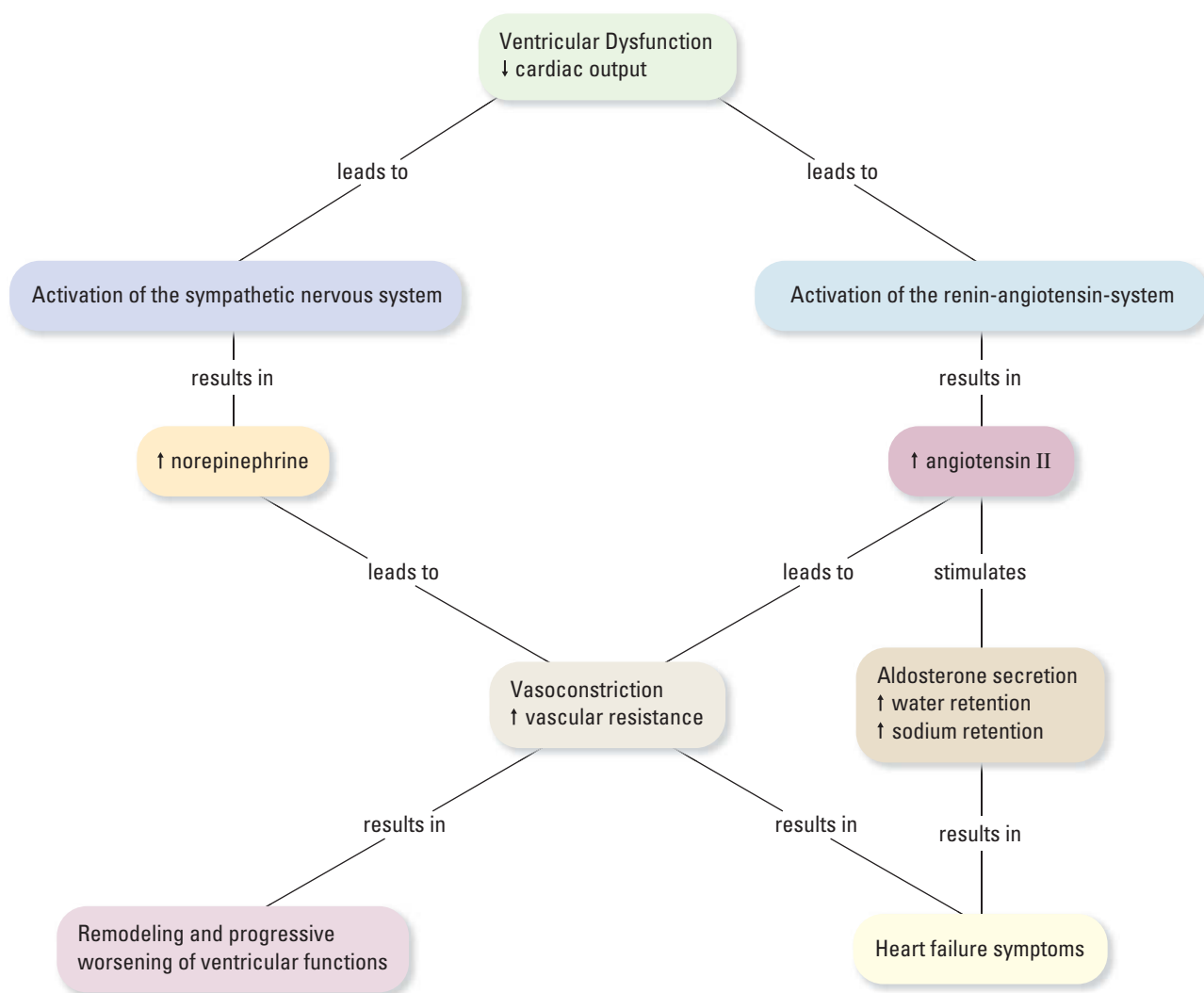
Upon completion of this chapter, the learner will be able to:

1. Determine the pathophysiologic and neurohormonal mechanisms of heart failure.
2. Compare and contrast systolic/diastolic dysfunction and left/right ventricular failure including diagnostic findings.
3. Prioritize collaborative management strategies appropriate for the patient with heart failure.
4. Prioritize collaborative management strategies appropriate for the patient with complications due to acute decompensated heart failure.

Introduction

Heart failure (HF) is a syndrome characterized by high mortality, frequent hospitalization, reduced quality of life, and a complex therapeutic regimen. Approximately 5.1 million individuals over the age of twenty years in the United States have heart failure. Of those individuals living with HF, only 50% are expected to live five years after initial diagnosis due to progressive pump failure and congestion, sudden cardiac death, or end-organ failure resulting from inadequate systemic organ perfusion, particularly

to the kidneys (Centers for Disease Control, 2014). Heart failure occurs more frequently among the elderly, and as the general population continues to age, an increase in the incidence of this syndrome is expected. In fact, with each additional decade beyond the age of 65, there is a twofold increase for the chance of hospitalization for these individuals. There is a male predominance until age 75, but then the occurrence of HF equalizes across genders. The cost of healthcare services, medications, and lost productivity due to HF is staggering, creating an annual economic burden in the United States of \$32 billion (Go et al., 2012).



Visual Map 7-1 Heart Failure

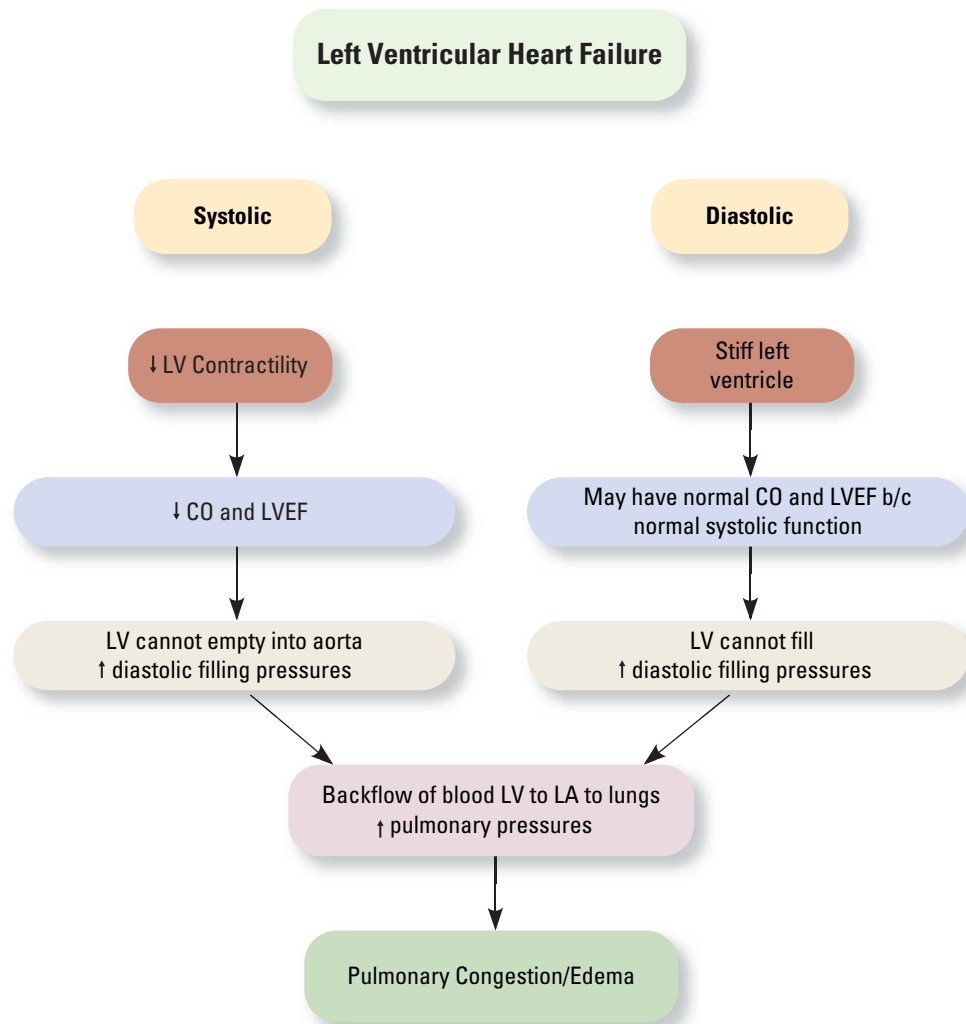
Pathophysiology of Heart Failure

Pathophysiology

Heart failure is an acute or chronic clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the heart to fill with or eject adequate blood throughout the body. As a result, tissues are inadequately perfused, leading to cellular hypoxia and disorders in metabolism. As heart function fails and the **cardiac output (CO)** decreases, compensatory mechanisms are activated. The role of these compensatory mechanisms is to maintain perfusion to the vital organs. The primary compensatory neurohormonal mechanisms involved in response to HF are the

sympathetic nervous system (SNS) and the **renin-angiotensin-aldosterone system (RAS)**. In response to a decreased CO, the SNS increases heart rate and contractility, causes arteriolar vasoconstriction in nonessential vascular beds, and stimulates secretion of renin from the juxtaglomerular apparatus of the kidney. Activation of the RAS via SNS stimulation and decreased renal perfusion further increases vasoconstriction through the release of angiotensin II, a potent vasoconstrictor, and water and sodium reabsorption through the release of aldosterone (refer to Visual Map 7-1 Heart Failure).

While these compensatory mechanisms are beneficial in the short term, long-term activation of these mechanisms can be deleterious, leading to a process called remodeling. Remodeling is defined as alteration



Visual Map 7-2 Physiology of LV Systolic and Diastolic Dysfunction

in the structure (dimensions, mass, shape) and function of the heart in response to hemodynamic load and/or cardiac injury in association with neurohormonal activation. Remodeling leads to progressive worsening of ventricular dysfunction due to ventricular dilation and hypertrophy. Ventricular chamber dilation causes increased wall tension, worsens subendocardial myocardial perfusion, and can provoke ischemia in patients with coronary atherosclerosis. Furthermore, dilation of the left ventricle (LV) chamber can cause mitral annular dilatation and mitral regurgitation leading to pulmonary congestion (Konstam, Kramer, Patel, Maron, & Udelson, 2011).

The understanding of the role of neurohormonal factors and cardiac remodeling in the development of heart failure has improved in the past several years. It is imperative that the nurse comprehend the pathophysiology of heart failure in order to understand the rationales for both pharmacological and nonpharmacological treatment.

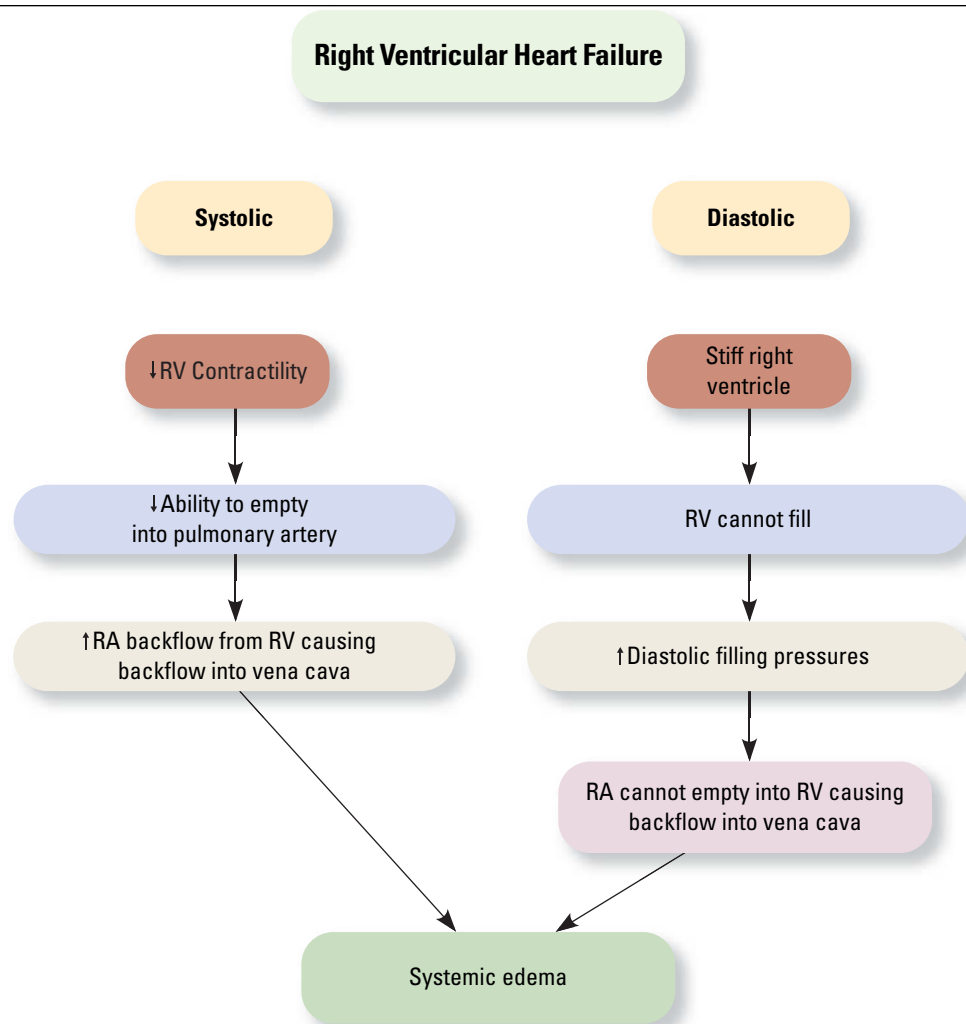
Systolic/Diastolic Dysfunction and Left/Right Ventricular Failure

Classifications and Etiologies

The classifications for heart failure are organized into systolic versus diastolic dysfunction, which can involve the left ventricle (LV), right ventricle (RV), or both; staging that monitors the progression of failure; and function, or how well the patient can live with the disease.

Systolic Versus Diastolic Heart Failure

The **left ventricular ejection fraction (LVEF)** is the percentage of blood ejected from the left ventricle with each contraction—55% to 75% is normal. Systolic dysfunction is the impairment of ventricular contractility. The most common type of HF is LV systolic dysfunction comprising



Visual Map 7-3 Physiology of RV Systolic and Diastolic Dysfunction

about 60% of the HF population. When the LV is involved, impaired contractility leads to a reduction in the LVEF and CO resulting in poor peripheral perfusion. This is known as **heart failure with reduced ejection fraction (HFrEF)** where the LVEF is less than 40%. Diastolic dysfunction occurs when the ventricle is normal size but hypertrophied, leading to loss of ventricular diastolic

relaxation, distensibility, and ability to fill. The individual with diastolic dysfunction may or may not have a **heart failure with preserved ejection fraction (HFpEF)**, which is defined as an LVEF greater than 50%. The failure of ventricular relaxation in diastolic dysfunction results in elevated end-diastolic pressures. The end result of diastolic dysfunction is identical to systolic dysfunction.

Gerontological Considerations

More than 80% of the people in the United States with heart failure are older than 65 years of age, with the majority of people being older than 75. The increased incidence among the elderly is directly related to the structural and functional changes that occur with the aging heart. These changes include arterial wall thickening and stiffening, a deterioration of cardiomyocyte functioning, increased incidence of atrial arrhythmias stiffening of the heart valves, and a slowing of the cardiac conduction system.

In addition, there may be an increase in lack of medication adherence and dietary restriction among the elderly (Alosco et al., 2012). These factors, in addition to the increased comorbidities in elderly heart failure patients such as depression, dementia, arthritis, chronic kidney disease, hypertension, coronary artery disease, and diabetes, may present challenges in effective patient education, diagnosis and management of failure, and recognition of escalating symptoms of failure.

Bariatric Considerations for Heart Failure

Obesity contributes greatly to adverse effects on left ventricular (LV) structure, diastolic and systolic function, and other risk factors for heart failure, including hypertension and coronary heart disease. Although HF incidence and prevalence are markedly increased in obese patients, numerous studies have documented an “obesity paradox” in which overweight and obese patients, defined by body mass index, percent body fat,

or central obesity, demonstrate a better prognosis compared with lean or underweight HF patients. This may be related to the fact that overweight and mildly obese HF patients may be somewhat protected from cardiac cachexia.

Vest, A., & Young, J. (2014). Should we target obesity in advanced heart failure? *Current Treatment Options in Cardiovascular Medicine*, 16(284).

In **left ventricular failure**, the pumping action (systolic) or ability of the LV to fill (diastolic) is impaired, allowing blood to back up from the LV to the left atrium (LA) and into the pulmonary veins. Fluid eventually builds up in the lungs and pleural spaces, causing pulmonary symptoms. Refer to Visual Map 7-2 for the physiology of LV systolic and diastolic dysfunction. In **right ventricular failure**, the blood volume backs up from the right ventricle (RV) to the right atrium (RA) and out to the systemic circulation, causing peripheral and organ edema. Right ventricular HF is often a consequence of left ventricular HF, but can also be a primary cause of HF. Refer to Visual Map 7-3 for the physiology of RV systolic and diastolic dysfunction.

A less common form of HF is high output failure, which is primarily a systolic dysfunction. This is when the LV becomes hyperdynamic causing the CO to be normal or elevated. High output failure occurs in response to a decreased afterload or systemic vascular resistance. This type of failure is seen when the body has an increased metabolic need, decreased oxygen-carrying capacity or change in the peripheral vasculature. The etiologies behind heart failure vary greatly depending on systolic or

diastolic dysfunction, left versus right ventricular failure, or high output failure (Table 7-1).

Reflect On

How would you explain a diagnosis of heart failure to a patient who states “But the doctor tells me my heart pumps fine.”?

Classifications: Staging and Function

The American College of Cardiology (ACC) and American Heart Association (AHA) have developed a classification of heart failure based on stages of the syndrome and emphasize the development and progression of the disease.

The New York Heart Association (NYHA) classifies exercise capacity and symptomatic status. The difficulty with this classification is that it is variable depending on what medications the patients are on or off, and the degree of patient compliance with the medications and diet. The ACC staging system is useful in that Stage A encompasses “pre-heart failure”—a stage where intervention with treatment can presumably prevent progression to overt symptoms. See Table 7-2 for how these classifications correspond with each other.

Table 7-1 Etiologies of Heart Failure

Left Heart Systolic Dysfunction	Left Heart Diastolic Dysfunction	Right Heart Systolic Dysfunction	Right Heart Diastolic Dysfunction	High Output Systolic Dysfunction
• Coronary artery disease	• Coronary artery disease	• Left ventricular failure	• Pericardial constriction	• Chronic severe anemia
• Hypertension	• Hypertension	• RV infarction	• Cardiac tamponade	• Thyrotoxicosis
• Diabetes mellitus	• Diabetes mellitus	• Coronary artery disease	• RV hypertrophy	• Sepsis
• Valvular disease	• Valvular disease	• Valvular disease		• AV fistulas
• Arrhythmias	• Hypertrophic cardiomyopathy	• Chronic pulmonary disease		• Pregnancy
• Myocarditis	• Restrictive cardiomyopathy	• Pulmonary embolism		• Paget's disease
• Systemic inflammatory diseases	• Constrictive pericarditis	• Pulmonary hypertension		
• Other: Peripartum cardiomyopathy, drugs, congenital disease, idiopathic cardiomyopathy				

Table 7-2 Comparison of ACCF/AHA Stages of HF and NYHA Functional Classifications

ACCF/AHA Stages of HF		NYHA Functional Classification	
A	A: At high risk for HF but without structural heart disease or symptoms of HF	None	
B	B: Structural heart disease but without signs or symptoms of HF	I	No limitation of physical activity. Ordinary physical activity does not cause symptoms of HF.
C	C: Structural heart disease with prior or current symptoms of HF	I	No limitation of physical activity. Ordinary physical activity does not cause symptoms of HF.
		II	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in symptoms of HF.
		III	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes symptoms of HF.
		IV	Unable to carry on any physical activity without symptoms of HF, or symptoms of HF at rest.
D	D: Refractory HF requiring specialized interventions	IV	Unable to carry on any physical activity without symptoms of HF, or symptoms of HF at rest.

Focused Assessment of the Patient

The assessment of the acutely ill patient with heart failure demonstrates signs, symptoms, and laboratory alterations from the effects of basically two major dysfunctions: decreased CO leading to volume overload, and end-organ hypoperfusion.

Volume overload leads to:

- Dyspnea on exertion.
- Orthopnea.
- Paroxysmal nocturnal dyspnea (PND).
- Peripheral edema.
- Abdominal pain and distention.
- Weight gain.
- Crackles on pulmonary auscultation.
- Elevation of central pressures (CVP, PCWP).
- Presence of S₃ or S₄ upon cardiac auscultation.

End-organ hypoperfusion is manifested by:

- Decreased exercise tolerance.
- Fatigue.
- Dizziness.
- Confusion.
- Syncope or near syncope.

- Palpitations.
- Hypotension.
- Tachycardia.
- Cool extremities.
- Delayed capillary refill.
- Decreased urinary output.

Reflect On

Your patient is in a rapid atrial fibrillation. Reflect on how he or she would manifest worsening cardiac function.

The nurse looks for evidence of worsening of the heart failure, including increasing dyspnea, tachypnea, tachycardia, fatigue, and exercise intolerance. Dysrhythmias such as ventricular ectopy or atrial fibrillation may exist in the presence of worsening disease. Atrial fibrillation in the setting of a low ejection fraction can precipitously drop a patient's cardiac output. Atrial kick, which may supply as much as nearly 20% of the volume entering the ventricle, is compromised and contributes to a lessening ventricular volume and subsequent decreased CO. Refer to Table 7-3 Physical Manifestations of Left and Right Ventricular Heart Failure.

Diagnostic Criteria

History and Physical Exam

It is crucial that an accurate patient history is obtained. Focus should be placed on cardiac and noncardiac disorders or behaviors that may affect HF, medications, use of illicit drugs, use of alternative therapies, and ability to perform routine activities of daily living. The physical exam should focus on the patient's fluid and perfusion status including blood pressure, weight, temperature of extremities, and presence or absence of abnormal physical findings.

Essential for Evidence-Based Practice

Symptoms and signs are important in monitoring a patient's response to treatment and stability over time. Persistence of symptoms despite treatment usually indicates the need for additional therapy, and worsening of symptoms is a serious development and merits prompt medical attention (McMurray, Adamopoulos, Anker, Auricchio, & Bohm, 2014).

Noninvasive Cardiac Testing

An **echocardiogram** should be performed in all patients initially presenting with heart failure. This may be either a TTE (transthoracic echocardiogram) or a TEE (transesophageal echocardiogram). This test determines LV/RV size and function, wall motion abnormalities, valvular function

Table 7-3 Physical Manifestations of Left and Right Ventricular Heart Failure

	Left Ventricular Heart Failure	Right Ventricular Heart Failure
GENERAL	Fatigue; weakness; lethargy; exercise intolerance	Easily fatigued
PULM	Orthopnea; rhonchi; crackles; wheezes; dyspnea; frothy, pink sputum; tachypnea; hacking cough worse at night; hypoxia; respiratory acidosis	Dyspnea present but not as prominent
CARDIAC	Tachycardia with exertion; dizziness; palpitations; irregular rhythm; S3/S4 summation gallop.	Palpitations; irregular rhythm; JVD
	Cardiomegaly	↑ CVP, PCWP, PAP
	↑ PCWP; PAP; CVP	↑/↓ BP
	Angina	
	↑/↓ BP	
NEURO	Confusion; restlessness	
GI	Present but not as prominent	Anorexia; nausea; hepatosplenomegaly; distended abdomen; constipation
RENAL	Oliguric during the day	Polyuria at night; weight gain
EXTREMITIES	Skin cool, moist; cyanosis; pallor; ↓ Capillary refill; mild to moderate edema	Moderate to severe edema

and abnormalities, diastolic function, presence or absence of pericardial abnormalities or intracardiac masses, and evaluates intracardiac filling pressures.

An estimate of the LVEF is also done to determine if the patient has a reduced ejection fraction (less than 40%). An echocardiogram is considered the gold standard for the diagnosis and ongoing evaluation of the patient with heart failure.

A 12-lead EKG (electrocardiogram) should also be performed to assess for arrhythmias such as sinus tachycardia at rest, atrial fibrillation, premature atrial and ventricular contractions, bundle branch blocks, ischemia (as evidenced by ST segment abnormalities), electrical dyssynchrony, and LV hypertrophy.

Chest X-ray

A chest x-ray is done to assess for cardiomegaly, pulmonary congestion and may show cardiac chamber enlargement, increased pulmonary venous pressure, interstitial or alveolar edema, valvular or pericardial calcification, or coexisting thoracic diseases. A chest x-ray should not be the sole determinant of the specific cause of HF, as it has a low sensitivity and specificity.

Laboratory Work

The following laboratory work is recommended:

- Complete Blood Count: Assess for anemia and infection.
- Serum electrolytes, BUN, creatinine: Renal dysfunction, elevation of neurohormonal substances, activation of the renin-angiotensin-aldosterone axis, and response to medications represent the major contributory factors to altered electrolytes, BUN, and creatinine. The following laboratory results are the most commonly found in the HF patient: hyponatremia, elevated BUN and

serum creatinine, hypokalemia or hyperkalemia, and hypomagnesemia.

- **B-type natriuretic peptide (BNP):** BNP and N-terminal pro-BNP levels can be used to evaluate patients with dyspnea for heart failure. BNP is secreted by the atria and ventricles in response to stretching or increased wall tension. Normal range for BNP is less than 100 pg per mL and N-terminal proBNP less than 125 pg per mL in patients younger than 75 years. BNP levels increase with age, are higher in women and blacks, and can be elevated in patients with renal failure. BNP appears to have better reliability than N-terminal pro-BNP, especially in older populations (King, Kingery, & Casey, 2012). It is important to recognize that there are variables other than HF that may cause an elevated BNP. Refer to Table 7-4 Causes of Elevated BNP.
- Liver function tests: Elevated transaminases and LDH as well as total bilirubin levels may be present due to liver congestion.
- Arterial blood gas: Alterations in oxygenation and acid-base balance may occur due to decreased perfusion and/or fluid overload.
- Other laboratory results: It is recommended that patients with no apparent etiology of HF or no specific clinical features suggesting unusual etiologies undergo additional directed blood and laboratory studies to determine the cause of HF.

Invasive Cardiac Testing

Left-sided heart catheterization and coronary angiography should be considered when the etiology of heart failure cannot be determined by clinical or noninvasive imaging methods. Coronary angiography is particularly helpful in patients with LV systolic dysfunction and who also have

Table 7-4 Causes of Elevated BNP

Cardiac Etiologies	Noncardiac Etiologies
Heart failure, including RV syndromes	Advancing age
Acute coronary syndrome	Anemia
Heart muscle disease, including LVH Valvular heart disease	Renal failure
Pericardial disease	Pulmonary: obstructive sleep apnea, severe pneumonia, pulmonary hypertension
Atrial fibrillation	Critical illness
Myocarditis	Bacterial sepsis
Cardiac surgery	Severe burns
Cardioversion	Toxic-metabolic insults

known or suspected coronary artery disease. Myocardial ischemia can play a major role in the reduction of LV systolic function and the worsening of heart failure. An endomyocardial biopsy may be performed on patients when a specific diagnosis is suspected that may influence therapy.

Invasive Hemodynamic Monitoring

Invasive hemodynamic monitoring should only be performed in patients with severe clinical decompensation requiring escalating pressor therapy, dependence on intravenous inotropic infusions, or persistent severe symptoms despite adjustment of recommended therapies. The nurse monitors hemodynamic values for elevated filling pressures, hypoperfusion, and altered vascular tone.

Endomyocardial Biopsy

Endomyocardial biopsy can be useful when seeking a specific diagnosis that would influence therapy, and biopsy should thus be considered in patients with rapidly progressive clinical HF or worsening ventricular dysfunction that persists despite appropriate medical therapy. Endomyocardial biopsy should also be considered in patients suspected of having acute cardiac rejection status after heart transplantation.

Nursing Diagnoses

Patient Presenting with Heart Failure

- Excess fluid volume
- Impaired gas exchange (acute decompensated heart failure)
- Decreased cardiac output related to decreased myocardial contractility
- Fatigue related to decreased cardiac output
- Activity intolerance related to imbalance between oxygen supply and demand
- Decreased tissue perfusion

Essential for Collaboration

Implementation of evidence-based heart failure guidelines, improved interdisciplinary coordination of care, patient education, self-management skills, and transitional care at the time of discharge improved overall heart failure outcome measures. The collaboration between disciplines improved continuity of care, patient compliance to their discharge regimen, and adequate discharge follow-up (White & Hill, 2014).

Collaborative Management Strategies

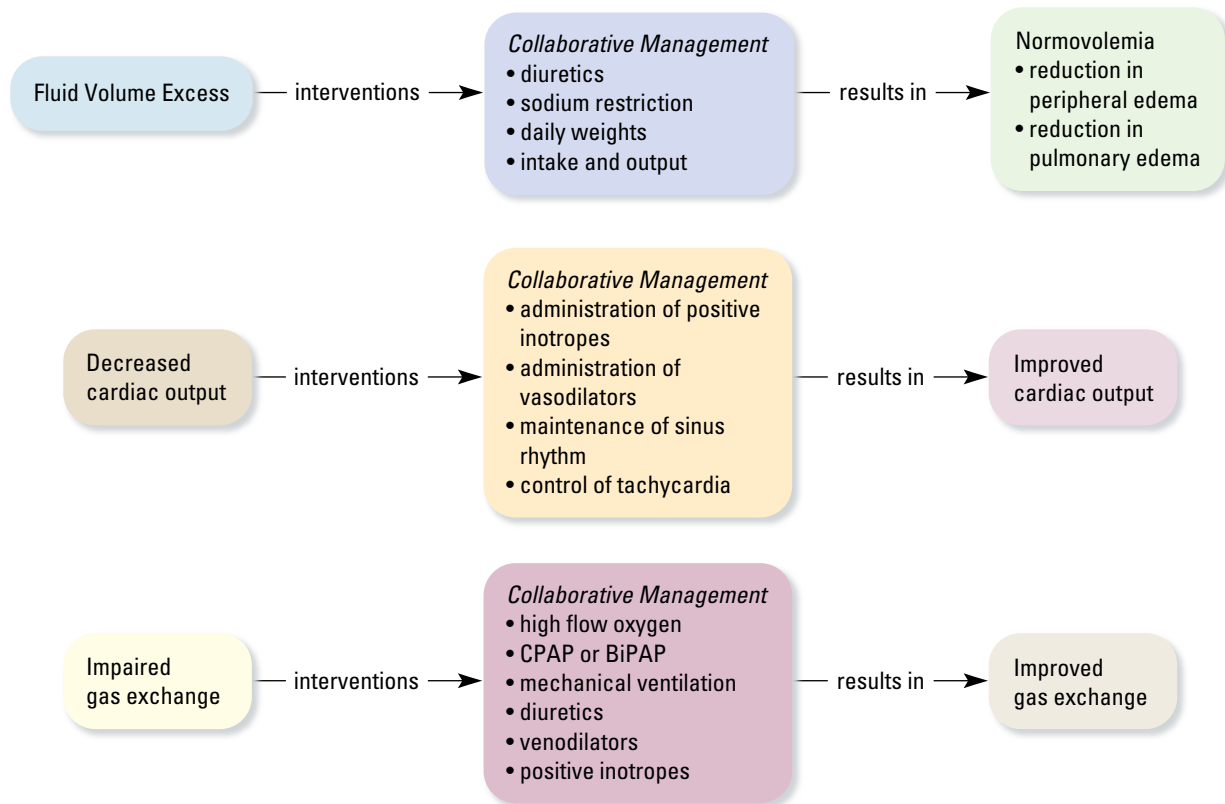
The nurse works collaboratively with the physician and other members of the healthcare team to manage the symptoms of heart failure, reduce the progression of the disease, and prolong survival. The management of the patient involves both nonpharmacological and pharmacological measures that focus on fluid volume excess, decreased cardiac output, and impaired gas exchange. Refer to Visual Map 7-4 Heart Failure Collaborative Management.

Nonpharmacological Measures

Nonpharmacological measures significantly impact patient stability, functional capacity, mortality, and quality of life. These measures include smoking cessation, restriction of alcohol consumption and other cardiotoxins, dietary salt restriction of 1.5–3 g/day and weight reduction in obese patients. Fluid restriction should be initiated in the setting of hyponatremia (Na < 130) and for those whose fluid status is difficult to control despite sodium restriction and the use of high-dose diuretics. Caloric supplementation is recommended for patients with evidence of cardiac cachexia (Yancy, Jessup, Bozkurt, Butler, & Casey, 2013). Aerobic exercise training, particularly long term, can reverse left ventricular remodeling in clinically stable heart failure patients (Chen, Li, Zhu, & Cao, 2012). Limitation of activities may be advised for individuals in acute decompensated failure and most patients should not participate in exhaustive sports or heavy labor. Patients with heart failure can benefit from attention to exercise, diet, and nutrition.

Pharmacological Measures

The mainstays of pharmacological treatment for the heart failure patient are medications directed at the neurohormonal activation of the sympathetic nervous system and the renin-angiotensin-aldosterone system as well as diuretics to reduce blood volume and venous pressures. Refer to Visual Map 7-5 Pharmacological Sites of Action of Beta



Visual Map 7-4 Heart Failure Collaborative Management

Blockers, ACE I, and ARBs. By blocking the neurohormonal activation through the use of angiotensin-converting enzyme (ACE-I) inhibitors, beta blockers, angiotensin II receptor blockers (ARBs), and aldosterone inhibitors, symptoms of heart failure are reduced and progression of the disease is slowed (Werner & Bohm, 2008). ACE inhibitors and beta blockers are considered first-line drug management directed toward management of the SNS and RAS in HF (Table 7-5). Furthermore, attention must be paid to the patient's comorbid processes that may affect the progression and management of HF.

NURSING ACTIONS

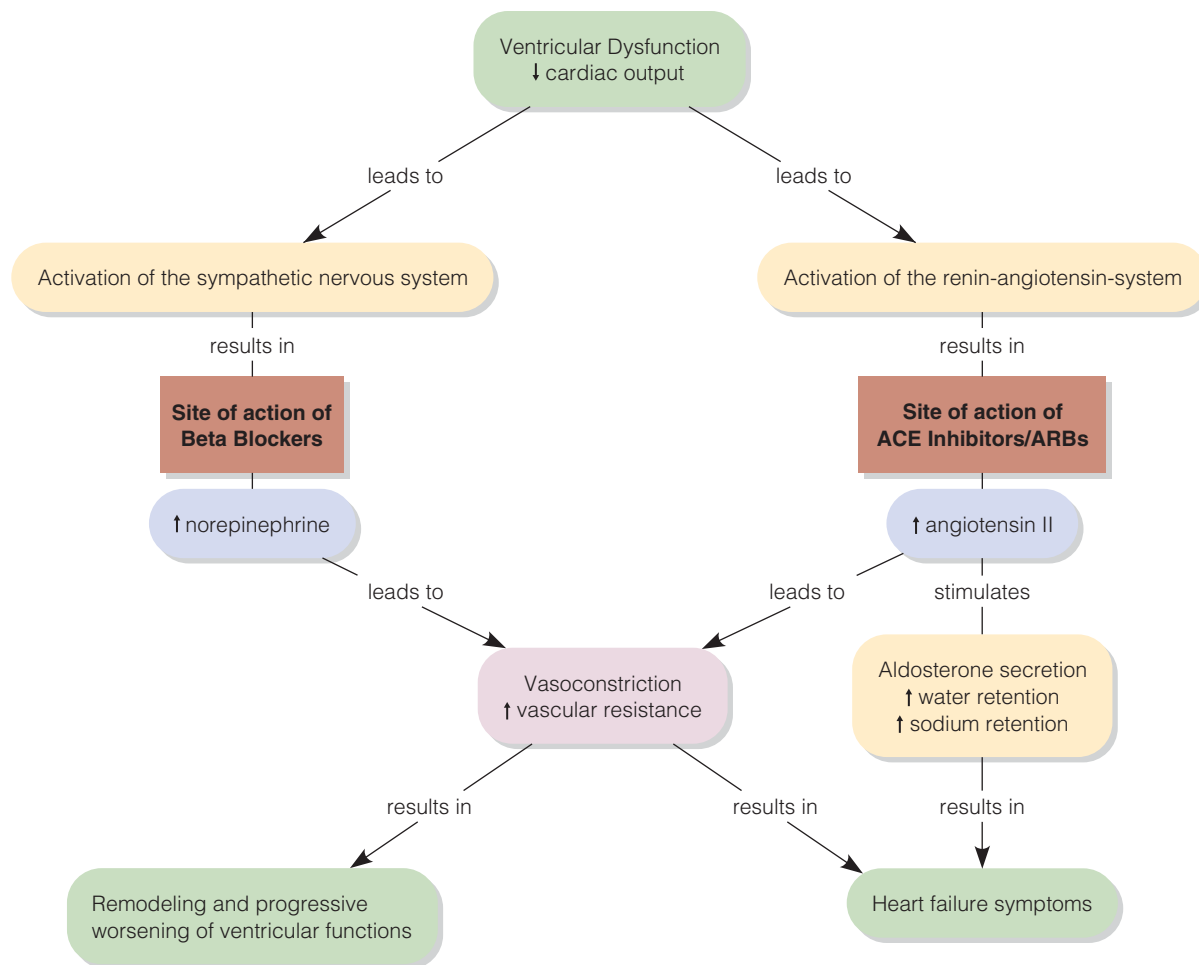
ACE Inhibitors/Angiotensin II Receptor Blockers

The benefits of these medications are seen in patients with mild, moderate, or severe symptoms of HF, and should be prescribed to all patients with HFrEF. Occasionally, ARBs and ACE-I are used together, but for the most part, ARBs are used in patients who are unable to tolerate ACE-I due to side effects (Go et al., 2013). These classes cause suppression of the RAS resulting in vasodilation. With a decreased systemic vascular resistance (SVR), it is easier

for the LV to unload volume to the systemic circulation. Contraindications to ACE-I include but are not limited to renal failure (serum creatinine greater than 3 mg/dL), hyperkalemia (greater than 5.5 mmol/L), angioedema, or very low systolic pressure, usually less than 80 mmHg or the presence of side effects. Patients with diastolic dysfunction may not be on these medications because the resultant hypotension can exacerbate the slow diastolic

Table 7-5 Commonly Used Medications to Block Neurohormonal Processes

ACE-Inhibitors	ARBs	Beta Blockers	Aldosterone Antagonists
captopril (Capoten)	candesartan (Atacand)	carvedilol (Coreg)	spironolactone (Aldactone)
enalapril (Vasotec)	valsartan (Diovan)	metoprolol (Toprol XL)	eplerenone (Inspra)
fosinopril (Monopril)		bisoprolol (Zebeta)	
lisinopril (Prinivil) (Zestril)			
quinapril (Accupril)			
ramipril (Altace)			



Visual Map 7-5 Pharmacological Sites of Action of Beta Blockers, ACE I, and ARBs

filling. With the use of ACE inhibitors, particular care should be given to the patient's volume status, renal function, and concomitant medications.

Nursing Responsibilities

- Assess for side effects; the two most common are dizziness and hypotension. Prior to administering the medication, the nurse should evaluate the patient's blood pressure ensuring that it is adequate before administration. Up to 20% of patients will experience an ACE-I induced cough.
- Renal function and serum potassium should be assessed within 1 to 2 weeks of initiation of therapy, looking for hyperkalemia and worsening renal function. The nurse should monitor for a change in urine output, as this is an indicator of renal function.
- Avoid concomitant administration of nonsteroidal anti-inflammatory drugs (NSAIDs). NSAIDs may reduce the vasodilation and unloading effects of ACE inhibitors as well as promote additional potential renal injury.

- Patient education should include avoiding rapid changes in position to prevent orthostatic changes and the importance of calling a healthcare provider to report a new cough or evidence of facial swelling (angioedema).
- Abrupt withdrawal of treatment with an ACE-I can lead to clinical deterioration and should be avoided.
- Patients should not be started on ACE-I if they are pregnant or plan to become pregnant.

Beta Blockers

Long-term treatment with beta blockers can lessen the symptoms of HF, improve the patient's clinical status, enhance the patient's overall sense of well-being, and are recommended for all patients with current or prior symptoms of HFrEF, unless contraindicated. By inhibiting the effects of the SNS stimulation, beta blockers reduce heart rate, blood pressure, and cardiac contractility. Beta blockade reduces cardiac hypertrophy, dysrhythmias, and tachycardia leading to heart failure. The earlier this therapy is prescribed, the more positive effects there may

be on the decreasing disease progression and the improvement of systolic function. Initiation of beta-blocker treatment may produce adverse reactions that require attention and management including fluid retention and worsening HF, fatigue, bradycardia or heart block, and hypotension.

Nursing Responsibilities

- Assess for hypotension, bradycardia, new atrioventricular blocks, dizziness, and fatigue. Prior to administering the medication, the nurse should evaluate the patient's blood pressure and pulse, ensuring that they are adequate for that patient before administration.
- Assess weights, increasing edema, dyspnea, and auscultate for crackles.
- Monitor the patient's blood sugar, particularly in the diabetic patient, as beta blockers worsen glucose control in those with diabetes and also blunt warning symptoms when hypoglycemia occurs.
- Observe the patient with asthma and COPD for increasing dyspnea.

Aldosterone Receptor Antagonists

Aldosterone receptor antagonists are recommended in patients with NYHA class II–IV HF and who have HFrEF, unless contraindicated, to reduce morbidity and mortality. Aldosterone antagonists enhance the lowering of serum aldosterone, thus reducing water and sodium retention while conserving potassium. Eplerenone (Inspra) selectively blocks the mineralocorticoid receptor and therefore has fewer side effects such as gynecomastia and sexual dysfunction compared with spironolactone (Aldactone). Renal insufficiency is a relative contraindication for this group of medications. Creatinine should be 2.5 mg/dL or less in men, or 2.0 mg/dL or less in women, and potassium should be less than 5.0 mEq/L.

Nursing Responsibilities

- Closely monitor the potassium levels and renal function. Under circumstances where these lab values cannot be monitored, it may be prudent to stop this medication.
- Monitor blood pressure closely.
- Monitor urine output as this is an indicator of renal function.

Reflect On

Your patient with HFrEF is due for lisinopril and Coreg. His BP is 90/50, HR 56. Which medication would you prioritize as the one to administer?

Table 7-6 Commonly Used Diuretics

Loop	Thiazides	Potassium Sparing
Bumetanide (Bumex)	Chlorothiazide (Diuril)	Amiloride (Midamor)
Furosemide (Lasix)	Chlorthalidone (Thalitone)	Spironolactone (Aldactone)
Torsemide (Demadex)	Hydrochlorothiazide	Triamterene (Dyrenium)
	Indapamide (Lozol)	
	Metolazone (Zaroxolyn)	

Diuretics

Diuretics should be prescribed to all patients who have evidence of or a prior history of fluid retention. They are usually combined with an ACE inhibitor, beta blocker, and aldosterone antagonist. Few patients with HF will be able to maintain target weight without the use of diuretics. Diuretics are usually started at low dose and the dose is increased until urine output increases and weight decreases, generally by 0.5 to 1.0 kg daily. Further increases in the dose or frequency of diuretics may be required to maintain an active diuresis and sustain weight loss. Typically, a loop diuretic like furosemide (Lasix) is initiated with usual starting doses of 20 to 40 mg PO daily. High doses of dietary sodium, agents that block the effects of diuretics or impaired renal function may lead to poor response to high doses of diuretics. Intermittent intravenous dosing or continuous drip administration may overcome diuretic resistance. Refer to Table 7-6 Commonly Used Diuretics.

Nursing Responsibilities

- Monitor for electrolyte imbalances: hypokalemia (less than 3.5 mmol/L), hypomagnesemia (less than 1.5 mg/dL), and metabolic alkalosis.
- Monitor for signs of excessive diuresis, which will lead to volume contraction resulting in hypotension and renal insufficiency.
- Concomitant administration of NSAIDs, which may blunt the renal effects of diuretics and promote renal insufficiency.
- The nurse should weigh the patient daily to detect fluid accumulation. An increase in more than 2 kg in 1 day suggests fluid volume excess or diuretic resistance. Careful assessment of intake and output will identify an imbalance leading to fluid volume excess.

Evidence-Based Interventions for Impaired Gas Exchange

The impaired gas exchange in HF is generally the result of pulmonary edema from elevation of left-sided filling

pressures. See further discussion of pulmonary edema in the “Acute Decompensated Heart Failure with Pulmonary Edema” section under “Prevention and Detection of Complications.”

Evidence-Based Interventions for Arrhythmias in Heart Failure

Maintenance of sinus rhythm in HF is important in optimizing CO. Supraventricular dysrhythmias like atrial fibrillation can exacerbate HF by reducing CO through the absence of the “atrial kick,” and if the ventricular rate is tachycardic, ventricular filling may decrease during diastole. Both of these developments are particularly concerning in the patient with diastolic dysfunction who relies on an adequate filling volume to maintain CO. Once considered the cornerstone of heart failure therapy, the drug digoxin (Lanoxin), a cardiac glycoside, is now used less frequently. It is still used for patients who continue to be symptomatic despite ACE inhibitor, beta-blocker, and diuretic use. Digoxin has a twofold purpose: to increase contractility, and to act as an antiarrhythmic. Many patients with HF have ventricular dysrhythmias including ventricular premature beats and nonsustained and sustained ventricular tachycardia. The nurse must closely monitor the patient’s rhythm as the presence of these types of

dysrhythmias is a predictor for increased mortality. Most antiarrhythmic drugs are proarrhythmic, cause an increase in dysrhythmias, and have a negative inotropic effect, making them ineffective and dangerous for heart failure patients. Amiodarone and dofetilide are the only antiarrhythmic agents to have neutral effects on mortality in clinical trials of patients with HF, and thus are the preferred drugs for treating arrhythmias in this patient group. Surgical insertion of an implantable cardioverter defibrillator can immediately treat these ventricular dysrhythmias and prevent the development of sudden cardiac death. Cardiac resynchronization therapy (CRT) may improve ventricular contraction and enhance CO (see discussions in “Building Technology Skills”).

Acute Decompensated Heart Failure (ADHF)

Early identification and management of stable heart failure, patient compliance with lifestyle modifications, and appropriate pharmacologic therapies may decrease the progression of heart failure and prevent the development of ADHF. ADHF is characterized by the development of dyspnea associated with the rapid accumulation of fluid within the

Safety Initiatives

Transforming Care at the Bedside: Creating an Ideal Transition to Home for Patients with Heart Failure

PURPOSE: Significantly improve care and reduce readmissions for patients with heart failure.

RATIONALE: Numerous studies have demonstrated the benefit of prompt post-discharge support and follow-up, and that hospitals can play a pivotal role in initiating a strong ambulatory care plan and reducing readmissions.

HIGHLIGHTS OF RECOMMENDATIONS: It is recommended that discharge planning encompasses the key components emphasized by the Institute for Healthcare Improvement to prevent the risk of adverse effect associated with deficiencies in health literacy, patient education, communication among healthcare providers within and between sites of care, appropriate medical follow-up, and any number of issues related to medications:

- Provision of an ACE-inhibitor or an angiotensin receptor blocker at discharge for HFrEF (LVEF less than 40%)
- Anticoagulant at discharge for CHF patients with chronic or recurrent atrial fibrillation
- Smoking cessation advice and counseling
- Discharge instructions that address all of the following: activity level, diet, discharge medications, follow-up

appointment, weight monitoring, and what to do if symptoms worsen

- Influenza immunization (seasonal)
- Pneumococcal immunization

In addition, emphasis should be placed on the following:

- **Enhanced admission assessment for post-discharge needs:** Include family caregivers and community providers in the discharge plan; medication reconciliation; plan of care based on needs assessment
- **Enhanced teaching and learning:** Design the plan of care to improve learner understanding; follow up calls
- **Patient and family-centered handoff communication:** Provide real time critical information to next care providers including medication reconciliation
- **Post-acute care follow-up:** Follow-up phone calls and next-care providers visits scheduled within 48 hours for high-risk patients and within 5 days for moderate-risk patients

Source: Nielsen, G. A., Bartely, A., Coleman, E., Resar, R., Rutherford, P., Souw, D., & Taylor, J. (2008). *Transforming care at the bedside how-to guide: Creating an ideal transition home for patients with heart failure*. Institute for Healthcare Improvement. Retrieved from <http://www.ihl.org/resources/pages/tools/tcabhowtoguidetransitionhomeforhf.aspx>

Table 7-7 Etiologies of Acute Decompensated Heart Failure

Nonadherence with medication regimen, sodium and/or fluid restriction
Acute myocardial ischemia
Uncorrected high blood pressure
Atrial fibrillation and other arrhythmias
Recent addition of negative inotropic drugs
Pulmonary embolus
Initiation of drugs that increase salt retention
Excessive alcohol or illicit drug use
Endocrine abnormalities
Concurrent infections
Additional acute cardiovascular disorders

lungs' interstitial and alveolar spaces that is the result of acutely elevated cardiac filling pressures (cardiogenic pulmonary edema). Studies reveal an equal percentage of patients with ADHF have HF_rEF and HF_pEF, and the overall morbidity and mortality for both groups is high. Hospitalized patients with HF can be classified into important subgroups: patients with acute coronary ischemia, accelerated hypertension and acutely decompensated HF, shock, and acutely worsening right HF (Yancy et al., 2013).

Refer to Table 7-7 for etiologies of ADHF. Diagnostic workup should include CXR, EKG, labs, echocardiogram, and possible PA catheter placement for continual monitoring of hemodynamics. The nurse assesses the patient for signs of congestion at rest and low perfusion, including:

- **Pulmonary:** dyspnea at rest, paroxysmal nocturnal dyspnea, reduced oxygen saturation, crackles on lung auscultation, worsening cough
- **Cardiac:** JVD, tachycardia, hypotension, new arrhythmias, elevation of PCWP, reduction in CO and cardiac index, S3, S4
- **Systemic:** fatigue, increased weight and edema, cool extremities, poor urine output

COLLABORATIVE CARE

It has been demonstrated that continuation of ACE inhibitors or ARBs and beta blockers for most patients is well tolerated and results in better outcomes in ADHF. However, intravenous administration of loop diuretics remains the foundation in the management of ADHF. The patient with ADHF who routinely takes loop diuretics may need to be treated with increased IV doses or a continuous infusion in the acute setting. If the nurse does not observe diuresis within 30 minutes of IV administration, higher doses of the same diuretic or addition of another classification of diuretics may be necessary (Yancy et al., 2013). For the patient in ADHF who also has renal insufficiency or

poor response to IV diuretics, renal replacement therapy may be utilized to achieve fluid removal. There are three forms of renal replacement therapy (RRT): intermittent hemodialysis, continuous renal replacement therapy, and peritoneal dialysis. The principles underlying RRT are diffusion, osmosis and ultrafiltration. This therapy is used to correct electrolyte imbalances, restore fluid balance, adjust acid-base balance, and remove wastes.

NURSING ACTIONS

CPAP/BiPAP

As fluid builds up in the interstitial spaces and alveoli, gas exchange is impaired, leading to hypoxia and hypercarbia. Short-term positive pressure ventilation such as continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP) should be considered first-line treatment of acute cardiogenic pulmonary edema. In ADHF, both modalities have shown a faster improvement in gas exchange and physiologic parameters with respect to conventional oxygen therapy including improvement of lung compliance, a decrease in work of breathing, and an increase in alveolar size, increasing tidal volume and providing more surface area for gas exchange, thus improving oxygenation and reducing hypercarbia. CPAP may also reduce preload and afterload, increasing cardiac output in some patients.

CPAP delivers one continuous positive pressure throughout inspiration and expiration. CPAP helps open alveoli and prevents atelectasis during expiration. This allows more lung units to be available for gas exchange and increases the partial pressure of oxygen in the alveoli facilitating oxygen diffusion into the bloodstream.

BiPAP allows the clinician to set a higher inspiratory pressure (IPAP) and a lower expiratory pressure (EPAP). Theoretically, adding IPAP reduces the work of breathing further and is more effective than CPAP, but studies have not shown a significant difference in outcomes other than an increase in patient comfort due to the reduced exhalation effort.

Vasodilators: Morphine Sulfate, Nitroglycerin

These medications are used to reduce the signs and symptoms of pulmonary edema and improve gas exchange through the reduction of preload and PCWP. In addition, morphine sulfate can be used to reduce patient anxiety. Prior to each intravenous dose, and at the peak of action, the nurse must assess blood pressure, respiratory rate, oxygen saturation, and level of consciousness.

Patients who do not respond to oxygen, diuretics, or morphine sulfate may be treated with intravenous nitroglycerin to further reduce preload and PCWP. Intravenous

Table 7-8 Commonly Used Medications in Acute Decompensated Heart Failure

Medication	Action	Expected Outcomes	Dosing	Side Effects
Diuretic				
Furosemide (Lasix)	Loop diuretic Inhibits reabsorption of sodium and chloride	↑ Urine output ↓ Edema ↓ Intravascular volume ↓ PCWP	Start at 20 mg IV push over 1–2 min Continuous infusion	Circulatory collapse ↓ K ⁺ , ↓ Mg ⁺⁺ , ↓ Ca ⁺⁺ , ↓ Na ⁺ Tinnitus Renal insufficiency
Vasodilators				
Nitroglycerin	Relaxes vascular smooth muscle	↓ CVP ↓ BP	Start at 5 mcg/min on infusion pump Titrate as tolerated	Hypotension Headache Circulatory collapse
Morphine	Relaxes vascular smooth muscle Anxiolytic	↓ Respirations ↓ PCWP ↑ O ₂ saturations ↓ Dyspnea	2 to 4 mg IV push over a 3-minute period; may repeat q 5–15 minutes	Anaphylaxis Respiratory depression/arrest Bradycardia Hypotension
Brain Natriuretic Peptide				
Nesiritide (Natrecor)	Inhibits ADH Vasodilator	↑ Urine output ↓ PCWP ↓ Preload ↓ dyspnea	IV bolus 2 mcg/kg followed by a continuous infusion of 0.01 mcg/kg/min	Hypotension Arrhythmias Circulatory collapse Electrolyte imbalances Renal insufficiency
Positive Inotropes				
Dopamine	Alpha and beta adrenergic agonist	↑ CO ↑ Systolic BP ↓ PCWP ↓ SVR	2–5 mcg/kg/min, titrate up to 20 mcg/kg/min	↑ Heart rate Arrhythmias Hypotension
Dobutamine	Beta adrenergic agonist	↑ CO ↓ PCWP ↓ SVR ↑ AV node conduction	Start infusion 0.5–1.0 mcg/kg/min. Titrate to 2.5–15 mcg/kg/min	↑ Heart rate Hypertension Anginal pain
Milrinone (Primacor)	Inhibits cyclic-AMP phosphodiesterase	↑ CO ↓ PCWP ↓ SVR	Loading dose; 50 mcg/kg/min over 10 min 0.375–0.75 mcg/kg/min	Ventricular arrhythmias Hypotension ↓ K ⁺ Anginal pain

nitroglycerin should always be administered via an infusion pump and with special tubing to prevent excessive absorption of the drug. Prior to initiation, before each titration, and at the peak action of nitroglycerin, the nurse must assess blood pressure, heart rate, respiratory rate, and oxygen saturation. Refer to Table 7-8 for further information regarding these medications.

Brain Natriuretic Peptide

Nesiritide (Natrecor) is a human B-type natriuretic peptide. It inhibits antidiuretic hormone (ADH), thus producing urine sodium loss and diuresis and also produces both venous and arterial vasodilation thereby decreasing preload and reducing the PAWP. Currently, nesiritide is indicated only for patients with ADHF who have dyspnea at rest. It is not to be used to replace diuretics or to enhance diuresis (Wilson, Shannon, & Shields, 2013). The nurse should closely monitor the patient's hemodynamic profile, electrolytes, and renal status during the course of this therapy. Refer to Table 7-8 for further information regarding these medications.

Intropic Therapy

Patients with ADHF who present primarily with low cardiac output or systolic dysfunction pose a more difficult clinical challenge. The nurse should assess the patient for signs of cardiogenic shock:

- Cardiac index less than 2.0 L/min
- Systolic blood pressure less than 90 mmHg
- PCWP above 18 mmHg

Patients who remain in pulmonary edema in the setting of low CO may be placed on intravenous inotropic therapy. Dobutamine and milrinone (Primacor) are the primary inotropes used in heart failure.

Dobutamine acts on beta 1 and beta 2 adrenergic receptors to increase contractility, decrease PCWP, and reduce afterload, thereby improving cardiac output. It also decreases AV conduction and has less potential for precipitating arrhythmias (Wilson et al., 2013). Dobutamine should always be administered via an infusion pump. Prior to initiation, before each titration, and at the peak action of dobutamine, the nurse must assess blood pressure, heart rate,

respiratory rate, and oxygen saturation. Frequent assessment of these parameters should continue throughout the infusion period.

Milrinone is a phosphodiesterase inhibitor that acts as a positive inotrope and vasodilator and has minimal chronotropic activity. Expected actions of milrinone would be an increase in cardiac output, reduction in PAWP, and reduction in vascular resistance (Wilson et al., 2013). Milrinone should always be administered via an infusion pump. Prior to initiation and at the peak action of milrinone, the nurse must assess blood pressure, hemodynamic profile, heart rate, respiratory rate, and oxygen saturation. Frequent assessment of these parameters should continue throughout the infusion period. Refer to Table 7-8 for further information regarding these medications.

Complex cases of ADHF and cardiogenic shock with progressive end-organ hypoperfusion or persistent hypotension may require more aggressive therapies such as ventricular support and heart transplantation.

Building Technology Skills

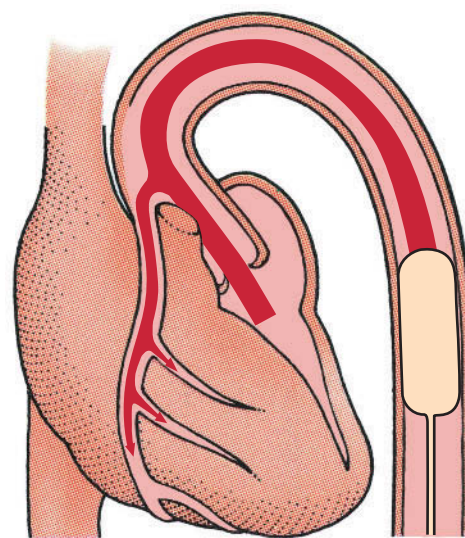
Technologies applied in the treatment of cardiogenic shock include intra-aortic balloon pump (IABP), implantable cardioverter defibrillator (ICD), cardiac resynchronization therapy (CRT), and ventricular assist devices.

Intra-Aortic Balloon Pump

IABP provides counterpulsation therapy, which increases coronary perfusion and decreases left ventricular workload. During inflation, the blood in the aorta is pushed toward the coronary arteries providing retrograde perfusion and thus improving the oxygen supply to the myocardium. During deflation, the heart pumps against the reduced resistance left by the balloon deflation, thus decreasing afterload and demand on the heart (♦ Figures 7-1A and B). The expected effects of IABP therapy are to improve CO/CI, improve MAP, and reduce PCWP.

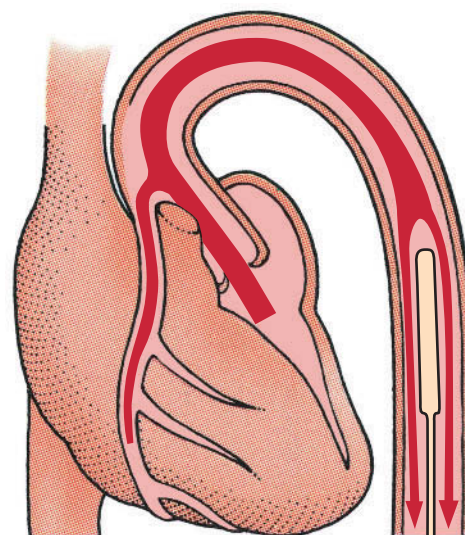
Access and Technological Requirements

A balloon catheter is inserted in the femoral artery and advanced into the aorta with the tip of the catheter just distal to the left subclavian artery and just above the renal arteries. The patient's ECG and arterial waveform are continuously monitored on the IABP console. Using the ECG as a trigger and the dicrotic notch as the timing indicator, the balloon is inflated during diastole and deflated during systole. Weaning from the IABP is dependent on the recovery of cardiac function and is achieved by decreasing the frequency of inflation or reducing the IAB volume (Lewis, Ward, & Courtney, 2009).



IAB Inflation

Figure 7-1A Inflation of the intra-aortic balloon.



IAB Deflation

Figure 7-1B Deflation of the intra-aortic balloon.

Nursing Responsibilities

It is important for the nurse to consider multi-organ assessment when evaluating the effects of IABP therapy. Nursing responsibilities include the following:

- Assess balloon timing within the cardiac cycle by evaluation of the arterial pressure values and tracings.
- Monitor alarms for indication of balloon leak, loss of augmentation.
- Monitor for blood in extender tubing.
- Maintain stability of the balloon catheter by maintaining the head of the bed less than 30 degrees and avoiding hip flexion.

- Monitor urine output q1h to assess for improvement of renal function and evidence that IABP is not occluding renal arteries.
- Assess for complications including limb ischemia, excessive bleeding from insertion site, thrombocytopenia, balloon immobility, balloon leak, infection, and aortic dissection.
- Assess the patient for hemodynamic stability during the weaning process.

Reflect On

How do you maintain HOB < 30 degrees when your patient has an IABP and is intubated?

Implantable Cardioverter Defibrillator

Sudden death in HFrEF is substantially decreased by neurohormonal antagonists that alter disease progression and also protect against arrhythmias. However, patients with HFrEF remain at increased risk for sudden cardiac death due to ventricular tachyarrhythmias. Implantation of an ICD can improve survival in certain subsets of heart failure patients and has been shown to be superior to antiarrhythmic drug therapy in preventing sudden death (Yancy et al., 2013). The ICD is a generator implanted in the tissue below the collarbone and is connected to the heart with insulated wires. The ICD detects dysrhythmias such as ventricular fibrillation and ventricular tachycardia and automatically delivers a shock to the heart. The shock may be in the form of cardioversion or defibrillation depending on the rhythm detected. Many ICDs also are able to perform antitachycardia pacing. This feature delivers bursts of pacing pulses at a rate faster than the ventricular tachycardia, and can successfully terminate the tachycardic rhythms. If the rhythm becomes too slow, a pacemaker feature can restore the normal heart rate. Patients who are appropriate candidates for ICD therapy include cardiac arrest survivors, sustained or inducible VT, ischemic cardiomyopathy, and dilated cardiomyopathy.

Nursing responsibilities in the acute setting include:

- Continuous monitoring of the ECG.
- Identification of the active features and current settings.
- Utilization of external defibrillation if the ICD fails to fire.
- Knowledge of policies and resources for temporarily suspending function in the event that the device is misfiring.

Cardiac Resynchronization Therapy (CRT)

Approximately 33% of HF patients have a substantial prolongation of the QRS interval. CRT or biventricular pacing can improve ventricular contractile function, decrease secondary mitral regurgitation, reverse ventricular remodeling, and sustain improvement in LVEF. All of these measures may improve blood pressure and allow increased titration of neurohormonal antagonist medications that may further contribute to improvement. In heart failure, there is a delay in atrioventricular synchrony with a reduced atrial systole, a delay in ventricular conduction causing disorganized ventricular contraction, and a decreasing pumping efficiency. CRT is thought to improve the synchrony by coordinating atrial systole and simultaneously pacing the ventricles. It is recommended that CRT therapy be initiated for patients who have LVEF $\leq 35\%$, sinus rhythm, left bundle-branch block (LBBB) with a QRS duration of ≥ 150 ms (Yancy, 2013). The goal is to optimize the atrioventricular (AV) delay through consistent timing of the AV interval and simultaneously pace the ventricles 100% of the time. Optimizing the AV delay decreases mitral regurgitation and increases diastolic filling time. Simultaneous activation of the septum and LV lateral wall improves LV contraction, leading to increased CO and increased ejection fraction. Additional outcomes are improved quality of life, exercise tolerance, and survival. CRT can be combined with an ICD as a single device if the patient meets criteria for both therapies, as is often the case (Cooper, 2015).

Technological Requirements

The biventricular pacing system consists of one atrial lead, one right ventricular lead activating the septum, and activation of the lateral wall of the left ventricle via a lead placed in the coronary sinus and advanced to the lateral cardiac vein. Simultaneous pacing in the ventricles displays only one ventricular pacing spike for each ECG complex (♦ Figure 7-2).

Nursing Responsibilities

Nursing responsibilities in the acute setting include:

- Continuous monitoring of the ECG.
- Measurement of the QRS duration. Widening of the QRS duration from the baseline may indicate a loss of ventricular synchronization.
- Identification of the active features and current settings.

Ventricular Assist Devices (VAD)

VAD therapy is a treatment option for individuals with end-stage or Class IV HF. Studies indicate that therapy with a VAD doubled the one-year survival rate of patients with end-stage heart failure as compared with drug treatment

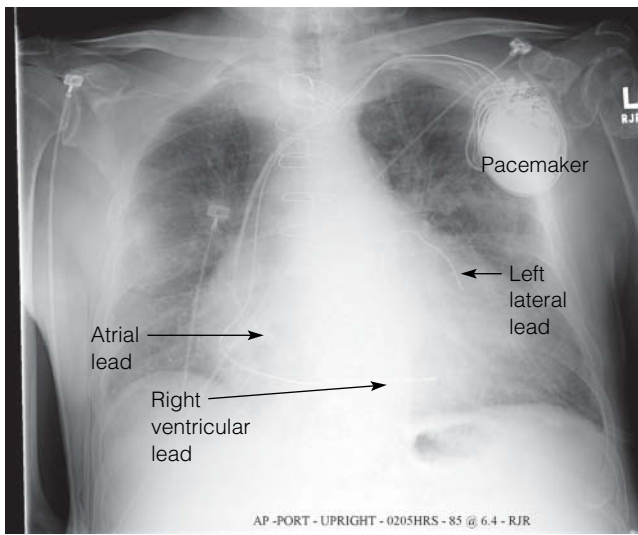


Figure 7-2 Biventricular pacemaker.

(© Elliot Hospital. All rights reserved.)

alone. These devices are used in patients who are no longer responsive to conservative medical treatment, who are not candidates for a heart transplantation (destination therapy), who are awaiting a heart transplantation, and who have acute heart failure and whose myocardial function is expected to return to normal. The therapeutic goals of VAD therapy include decreasing the workload of the heart, restoring blood flow to the body, and relieving or eliminating heart failure symptoms. Complications following VAD implantation are common and often life threatening. These include thromboembolism, infection, perioperative coagulopathy and bleeding, compromised immunity, malnutrition, device malfunction, and multisystem organ failure. Newer rotary continuous flow LVADs have proven to be more durable and are associated with fewer complications.

Technological Requirements

The VAD may be placed to assist the left ventricle (LVAD), right ventricle (RVAD), or both ventricles (BiVAD). The pump is placed in the upper part of the abdomen. The inflow cannula of a VAD is connected to the apex of the ventricle. Blood is mechanically pumped by the device via the outflow cannula to the ascending aorta, bypassing the patient's own ventricular function. Another tube, called the driveline, is brought out of the abdominal wall to the outside of the body and attached to the pump's battery and control system.

Nursing Responsibilities

Nursing management of the patient with a VAD include issues unique to advanced heart failure and mechanical circulatory support, and include the following (O'Shea, 2012):

- Monitoring of hemodynamic, renal, and hepatic status
- Adjusting and monitoring heart rate, stroke volume, and mode of operation
- Assessing driveline exit site for signs of infection
- Documenting flow rates
- Monitoring alarms
- Ensuring adequate power source: battery; hand pump
- Anticoagulation
- Patient education
 - How to change the batteries
 - Daily maintenance
 - Alarm recognition
 - Troubleshooting

Heart Failure Summary

Heart failure is a common syndrome that carries high morbidity and mortality. The nurse has a responsibility to promptly recognize escalating signs and symptoms of worsening failure, and to have knowledge of appropriate

pharmacological and nonpharmacological management strategies. The nurse should be an active participant in patient education and management of HF as well as the prevention of complications.

Why/Why Not?

A patient is admitted with acute decompensated heart failure (ADHF) and started on a milrinone drip. It is currently running at 0.5 mcg/kg/min. He has a pulmonary artery catheter in place. The patient's cardiac index is 2.1, pulmonary pressures 22/13 and systemic vascular resistance of 870. In the

setting of stable hemodynamics, the nurse receives an order to discontinue the Milrinone. Should the nurse agree with this order and shut the drip off? Why? Why not?

See answers to Why/Why Not? in the Answer Section.

Case Study

Mrs. E is a 76-year-old female admitted to the cardiac care unit (CCU). She is transferred from the emergency department (ED) where she presented with c/o SOB, chest discomfort, and palpitations for 2–3 days. Her health history includes hyperlipidemia, HTN, DM Type II, obesity, and hypothyroidism. Medications include simvastatin 20 mg qhs, levothyroxine 0.125 mg po daily, glipizide 5 mg po bid, HCTZ 25 mg po daily.

On admission to the CCU, a 12-lead ECG confirms new atrial fibrillation, no acute ST/T wave changes. VS: T 37, P 116 irregular, BP 100/48, RR 24, O₂ sat 90% on 6LNC. A brief exam reveals (+) JVD, crackles 2/3 up bilaterally, nonproductive cough, peripheral pulses are 2+ throughout, and 3+ pitting edema ankle to midcalf bilaterally, (+) hepatojugular reflux. Laboratory values: WBC 7.0, Hbg 9.0, Hct 27.0, platelets 213, Na 134, K 4.6, Cl 110, CO₂ 26, BUN 50, creat 2.2. BNP 1100. Thyroid panel WNL. Liver function tests WNL, ABG pH 7.29, pO₂ 63, pCO₂ 56, HCO₃ 20. CXR reveals pulmonary edema and cardiomegaly. An echocardiogram is done which reveals LVEF 25%, mild LV hypertrophy, valvular function intact.

1. The medical resident places the patient on BiPAP and inserts a pulmonary artery catheter line. Discuss why the resident has chosen to insert the PA catheter and the nurse's role in the insertion and safe management of a central line and PA catheter.
2. Mrs. E's BP has decreased to 80/44 and her HR has increased to 140, atrial fibrillation. The cardiologist determines that Mrs. E should be cardioverted. Why is atrial fibrillation so detrimental for Mrs. E?
3. The physician prescribes Lasix 20 mg IV push. What should you check before and during the administration of this drug? How do you evaluate if this intervention is successful? What would be your expectation if the patient did not respond appropriately to the medication?
4. Mrs. E's BP has decreased to 80/44 and her HR has increased to 140, atrial fibrillation. The cardiologist determines that Mrs. E should be cardioverted. Why is atrial fibrillation so detrimental for Mrs. E?
5. Mrs. E is started on dobutamine. Why is this an appropriate pharmacological intervention? What parameters would you assess to determine if this intervention is successful?
6. The PA catheter is removed and she is transferred to the step down unit. The physician prescribes lisinopril 10 mg po daily, Coreg 6.25 mg po bid, and Lasix 40 mg po daily. Discuss why these drugs are appropriate for Mrs. E. Present essential patient education including the rationales for starting these medications.

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 7.1 How do neurohormonal mechanisms contribute to the development of heart failure and how can these mechanisms be pharmacologically blocked?
- 7.2 What are the physiologic differences between HFrEF and HFpEF?
- 7.3 Your patient is without HF symptoms at rest but becomes dyspneic with less than normal activity. In which NYHA classification does she fall?
- 7.4 What are the indications that a patient with chronic heart failure is experiencing an acute decompensation with pulmonary edema?
- 7.5 What are the essential collaborative interventions for the patient with acutely decompensated heart failure?
- 7.6 What are the nursing priorities for the patient with acutely decompensated heart failure?

See answers to Chapter Review Questions in the Answer Section.

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Chapter 8

Care of the Patient Experiencing Shock

Shirley Jackson, DNP, RN, CCRN, CCNS

Abbreviations

CO	Cardiac Output	IABP	Intra-aortic Balloon Pump
CI	Cardiac Index	SV	Stroke Volume
CVP/RAP	Central Venous Pressure/Right Atrial Pressure	SVI	Stroke Volume Index
MAP	Mean Arterial Pressure	SVR	Systemic Vascular Resistance
PCWP	Pulmonary Capillary Wedge Pressure	SNS	Sympathetic Nervous System
		SCI	Spinal Cord Injury

Learning Outcomes

Upon completion of this chapter, the learner will be able to:

1. Discuss the etiologies and pathophysiology of hypovolemic, cardiogenic, obstructive, and distributive shock.
2. Integrate assessment, diagnostic and hemodynamic findings, and collaborative management of patients experiencing hypovolemic shock.
3. Integrate assessment, diagnostic and hemodynamic findings, and collaborative management of patients experiencing cardiogenic shock.
4. Integrate assessment, diagnostic and hemodynamic findings, and collaborative management of patients experiencing distributive shock.
5. Integrate assessment, diagnostic and hemodynamic findings, and collaborative management of patients experiencing obstructive shock.

Introduction

Shock is a condition in which a systemic decrease in perfusion to tissues and organs leads to poor gas and nutrient exchange. The nurse must be able to recognize the impending shock state and administer treatment in a timely fashion. Delays in recognition and treatment can lead to irreversible shock and multisystem organ failure.

Pathophysiology of Shock

Shock is a condition of impaired tissue perfusion and oxygenation. When oxygen demands exceed oxygen supply, there is a progressive deterioration of cellular dysfunction and organ failure. There are four stages of shock: initial, compensatory, progressive, and refractory. During the initial stage, hypoperfusion leads to a reduction in oxygen and

the cells switch to an anaerobic metabolism. In this anaerobic state, lactic acid is produced. During the compensatory state, the body tries to reverse the effects of the impaired tissue perfusion. The acidosis produced by lactic acid build up is moderated by hyperventilation to rid the body of carbon dioxide (CO₂) to normalize the pH. Baroreceptors in the arch of the aorta and in the carotid arteries react to a drop in circulating volume and reduction in blood pressure by increasing the heart rate and vascular resistance. As the vasoconstriction occurs, blood is shunted away from the skin, kidneys, and gut to the major organs such as the brain, heart, and lungs. The vasoconstriction that occurs as part of this compensatory process increases the diastolic pressure and narrows the pulse pressure. This increased vasoconstriction also results in cold, clammy skin and contributes to organ hypoperfusion (Society of Critical Care Medicine, 2012). At the tissue level, more oxygen is extracted which results in less oxygen returning to the heart, reflected in a lower SvO₂ or ScvO₂. The reduced blood flow to the kidneys stimulates the renin-angiotensin aldosterone system, leading to more vasoconstriction and conservation of water and sodium, maximizing the intravascular volume but further reducing the urine output. These compensatory mechanisms can keep the blood pressure and cardiac output normalized for a short period; however, as circulating volume is further reduced the compensatory mechanism fails. Unless the shock state is rapidly identified and treated, it will continue into the progressive stage. During this stage, cellular function continues to deteriorate, anaerobic metabolism continues increasing the acidosis state. The prolonged vasoconstriction leads to further tissue ischemia and organ damage. The final stage of shock is known as refractory. In this stage multiple organs have failed, and the shock can no longer be reversed.

There are four main categories of shock: hypovolemic, cardiogenic, distributive, and obstructive (refer to Table 8-1) (Society of Critical Care Medicine, 2012). These are explained below.

Table 8-1 Categories of Shock

Categories	Etiologies
Hypovolemic	Hemorrhage Burns Third spacing Diabetes insipidus
Cardiogenic	Cardiac ischemia/infarction Valve dysfunction Dysrhythmias Blunt cardiac trauma
Distributive	Spinal cord injury Anaphylaxis Severe sepsis
Obstructive	Pulmonary embolism Cardiac tamponade Tension pneumothorax Pericarditis

Hypovolemic Shock

Hypovolemic shock results from a decreased circulating volume. Etiologies of a reduced intravascular volume include hemorrhage, dehydration, gastrointestinal, or urinary losses. Third space losses which occur as fluid redistributes to transcellular areas may occur in burns, trauma, pancreatitis and prolonged surgical cases. Loss of approximately 15% to 30% or more of the normal blood volume can produce hypovolemic shock.

Cardiogenic Shock

Cardiogenic shock is a result of the heart's inability to deliver adequate circulation to the tissues due to cardiac pump failure. This occurs in the presence of adequate circulating volume. Etiologies of cardiac pump failure include damage to the myocardium as myocardial infarction or cardiomyopathy, structural defects to the cardiac valves or septum, and arrhythmias. In cardiogenic shock, stroke volume and cardiac output is reduced leading to poor tissue perfusion. Decreased stroke volume causes increased filling pressures in the cardiac chamber with a resultant increase in myocardial oxygen demand. The increased filling pressures in the left ventricle are reflected back into the left atrium and the pulmonary vasculature, leading to vascular congestion and pulmonary edema with impaired gas exchange (Warise, 2015).

Distributive Shock

Distributive shock is characterized by vasodilation and redistribution of blood volume. Etiologies of distributive shock are sepsis, spinal cord injury, and anaphylaxis, with sepsis being the most common.

Septic Shock

Septic shock is defined as the presence of sepsis with refractory hypotension. Collaborative management of septic shock includes identification of the likely source of infection, provision of antibiotics, and volume replacement. The earlier that the septic patient can be identified and the sepsis resuscitation bundle initiated, the better the patient outcome is likely to be.

Neurogenic Shock

Neurogenic shock (also called *spinal shock*) occurs when a spinal cord injury (SCI) to the cervical and upper thoracic spinal cord causes a temporary interruption in the sympathetic innervation, leaving the parasympathetic innervation unopposed (Hickey, 2013). This loss of sympathetic nervous system (SNS) innervation leads to immediate loss

of autonomic and motor reflexes below the level of the injury. Specifically, the loss of the autonomic reflexes results in vasodilation and redistribution of the blood volume, loss of cutaneous control of sweat glands resulting in warm, dry skin and parasympathetic regulation of the heart rate functioning unopposed resulting in bradycardia (Emergency Nurses Association, 2015).

Anaphylactic Shock

Anaphylactic shock is an overwhelming allergic reaction to an antigen with a rapid onset which may lead to death without appropriate interventions. Interaction of antigen with IgE on basophils and mast cells triggers release of histamine, leukotrienes, and other mediators that cause diffuse smooth muscle contraction (bronchoconstriction, vomiting, diarrhea) and vasodilation with plasma leakage (Girotra & Lalkhen, 2014). This vasodilation leads to profound hypotension. Fluid extravasation causes hypovolemia leading to reduced preload and reduction in cardiac output. Diffuse smooth muscle contraction leads to bronchoconstriction and a potentially compromised airway.

Obstructive Shock

Obstructive shock occurs as a result of impairment in cardiac ventricular filling or impairment of ventricular emptying. Obstructive shock can occur from such etiologies as cardiac tamponade, tension pneumothorax, and massive pulmonary embolism. Cardiac tamponade occurs when fluid accumulates in the pericardium compressing the atrial and ventricular chambers preventing the filling of the chambers with blood. Types of fluid likely to accumulate are blood or serous fluid from inflammatory processes such as pericarditis. This restriction to cardiac filling results in decreased cardiac output, hypotension and reduced tissue perfusion. Rarely, the pericardium becomes fibrotic and stiff from an inflammatory process. This constrictive pericarditis results in impaired ventricular filling, decreased output and reduced tissue perfusion. Tension pneumothorax occurs when a unilateral pneumothorax becomes so large that it shifts the mediastinum and compresses the cardiac chambers. This also results in impaired filling, decreased cardiac output, hypotension and reduced tissue perfusion. Obstructive shock can also occur from a massive pulmonary embolism which blocks blood flow through the pulmonary circulation. This obstruction increased the afterload of the right ventricle causing right ventricular failure. The embolus prevents adequate blood flow from the pulmonary circulation to the left ventricle. This obstruction coupled with right ventricular failure reduces left ventricular preload, decreasing the cardiac output, causing hypotension and reducing tissue perfusion (♦ Figure 8-1).

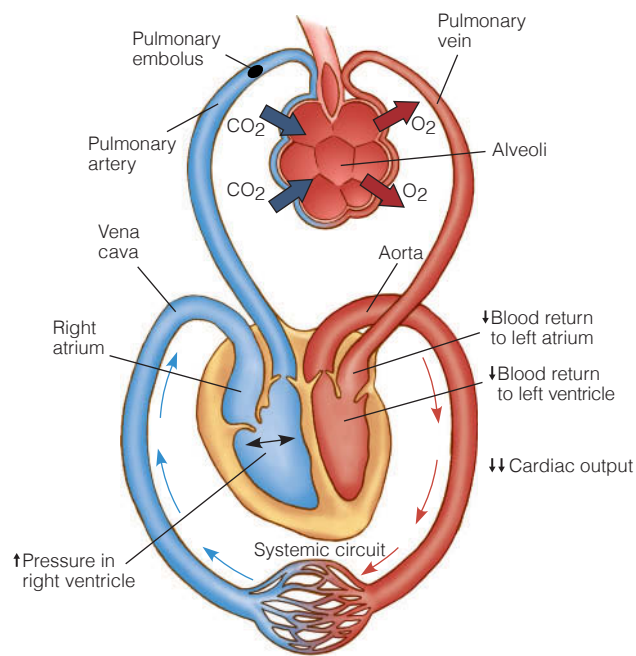


Figure 8-1 Pulmonary embolus restricting blood flow to the left ventricle and increasing pressure in the right ventricle which may lead to failure.

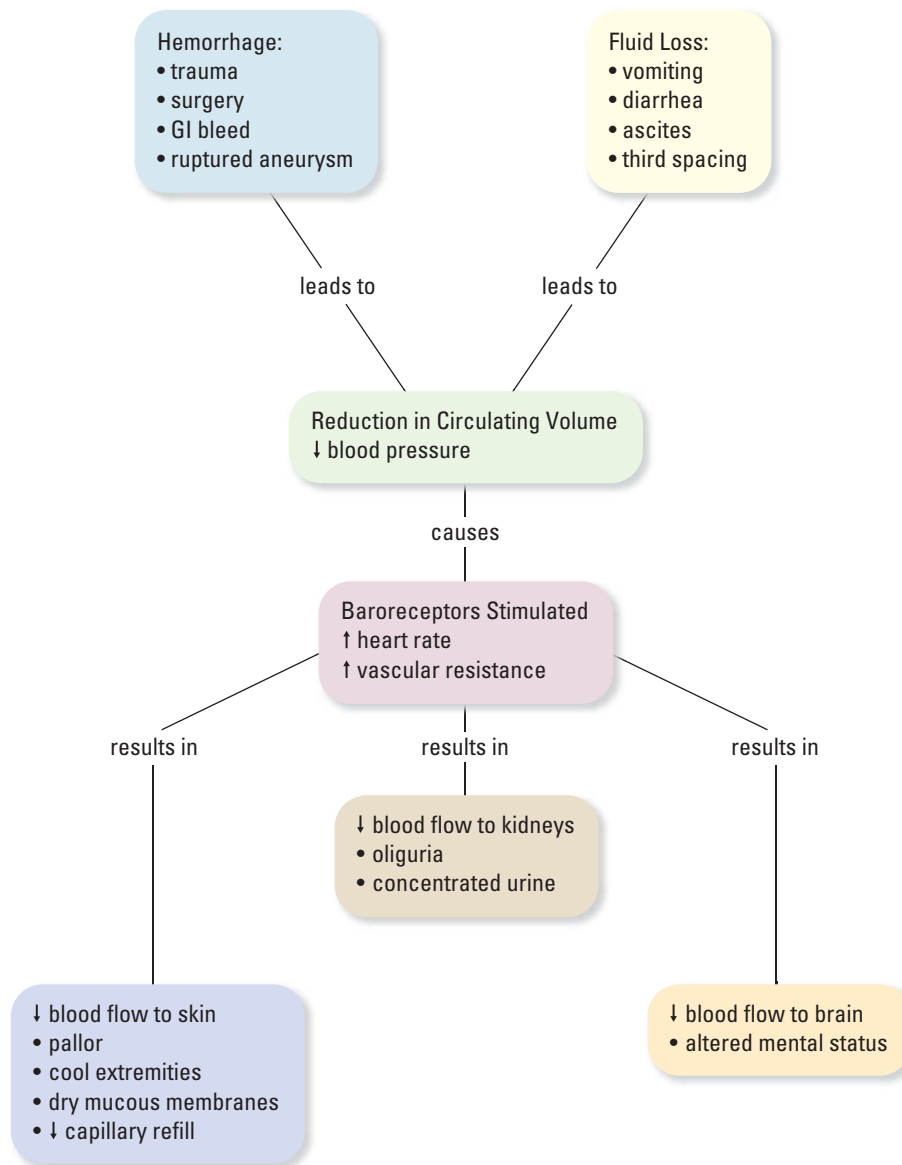
Hypovolemic Shock: Assessment and Management

The patient experiencing hypovolemic shock has either a hemorrhage or intravascular fluid loss. Causes of hemorrhage include GI bleeding, trauma, surgery, or ruptured aneurysm. Fluid loss may occur from vomiting or diarrhea, burns, or loss of fluids internally in cavities (e.g., ascites). In some situations, the intravascular fluid is redistributed into the interstitial space (third spacing), resulting in a deficit in intravascular volume such as occurs in sepsis. Failure of mechanisms regulating water balance can lead to a fluid volume deficit. Diabetes insipidus (DI) results from a deficiency in antidiuretic hormone (ADH) release from the posterior lobe of the pituitary gland. Trauma or surgery to the pituitary gland can lead to decreased ADH. This deficiency in ADH leads to excessive urine output and, if left unchecked, **hypovolemic shock**. (Visual Map 8-1).

Assessment

The nurse assesses the patient for the signs and symptoms indicating hypovolemia:

- Hypotension
- Orthostatic hypotension
- Tachycardia
- Delayed capillary refill



Visual Map 8-1 Hypovolemic Shock

- Dry mucous membranes
- Poor skin turgor
- Thirst
- Weight loss
- Oliguria
- Concentrated urine
- Altered mental status

Hemodynamic Findings

In hypovolemic shock, the heart rate is increased and the **mean arterial pressure (MAP)**, central venous pressure/right atrial pressure (CVP/RAP), and pulmonary artery wedge pressure (PAWP) are decreased, reflecting a reduced preload. A reduction in circulating volume may also lead

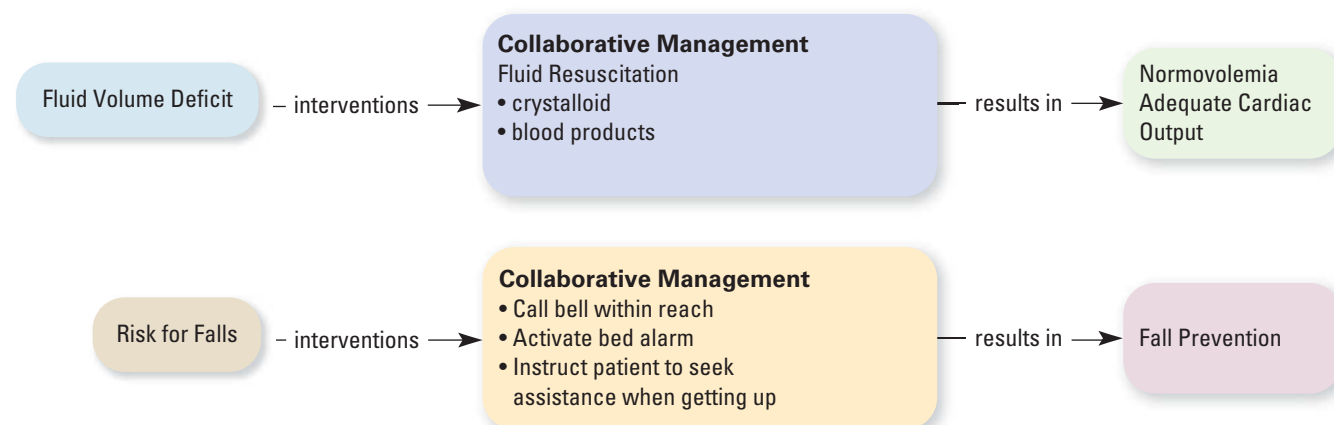
to a reduction in **stroke volume (SV)** and **cardiac output (CO)** or **cardiac index (CI)**. An elevated systemic vascular resistance index (SVRI) may be present reflecting the vasoconstriction due to compensatory mechanisms (refer to Table 8-1).

The nurse reviews laboratory studies for:

- Hyponatremia (serum levels above 145 mEq/L).
- Increased hematocrit (Hct) due to hemoconcentration (if patient is actively bleeding, Hct may drop).
- Arterial blood gases: metabolic acidosis.
- Elevated serum lactate (serum lactate greater than 4 mmol/L).
- Reduced mixed venous oxygen saturation (SvO₂ less than 60%; ScvO₂ less than 70%).

Table 8-2 Hemodynamic Changes and Interventions

Type of Shock	Etiologies	Cardiac Output	Central Venous Pressure	Systemic Vascular Resistance/SVRI	Pharmacological Interventions
Hypovolemic	Volume Depletion	↓	↓	↑	Isotonic solution (0.9% normal saline) Colloid (i.e., blood products)
Cardiogenic	Pump failure	↓	↑	↑	Dopamine Norepinephrine If pulmonary edema present: diuretics, preload reducers (i.e., nitroglycerin)
Neurogenic	Loss of nerve impulse communication	↓	↓	Normal or ↑	Crystalloids first Dopamine, norepinephrine, or phenylephrine if necessary Atropine for bradycardia
Anaphylactic	Allergic response	↓	↓	Normal or ↑	Epinephrine Diphenhydramine Ranitidine or famotidine
Septic	Presence of bacteria/toxins	Normal or ↑	Normal or ↓	↓	Norepinephrine Dopamine Phenylephrine Vasopressin

**Visual Map 8-2** Hypovolemia Collaborative Management

COLLABORATIVE CARE

The nurse works collaboratively with the physician to identify patients at risk for hypovolemic shock. Early identification of the patient in hypovolemic shock is key to a successful patient outcome. The goal of treatment is to expand the intravascular volume to restore a normal circulating volume to the microcirculation and oxygen transport. A MAP of greater than or equal to 65 mmHg is usually recommended (Society of Critical Care Medicine, 2012).

Restoration of volume status in the patient requires administration of fluid in the form of an isotonic solution or colloid through large-bore intravenous catheters. Standard, large (e.g., 14- to 16-gauge) peripheral intravenous (IV) catheters or central venous catheters are adequate for most fluid resuscitation. The advantage of a central venous catheter is that it may be used to monitor CVP, thus giving

the clinicians information on progress of volume resuscitation. Examples of isotonic solutions include 0.9% normal saline and lactated Ringer's solution. Administration of normal saline has been associated with metabolic acidosis (Myburgh & Mythen, 2013). These isotonic solutions are infused into the vascular space and stay there long enough to expand the intravascular space. (Visual Map 8-2). Hypotonic solutions, such as Dextrose 5% or 0.45% normal saline, have no place in fluid resuscitation for hypovolemic shock (Society of Critical Care Medicine, 2012). Hypotonic solutions do not stay in the intravascular space long enough to expand the circulating volume. Some references suggest the use of colloids in volume resuscitation. Randomized controlled trials have failed to demonstrate a reduction risk of death with the use of colloids (Perel, Roberts, & Ker, 2013). The utility and safety of hypertonic solution has not been established (Myburgh & Mythen, 2013).

The goal of volume resuscitation is to maintain tissue perfusion. The exact protocol for volume resuscitation varies. In large volume losses, it may be necessary to infuse large amounts of crystalloids or blood products. Some institutions utilize rapid infusers that also warm the fluid to prevent hypothermia (♦ Figure 8-2). Fluid resuscitation starts with 500–1000 mL boluses of an isotonic crystalloid titrated to monitored parameters (Society of Critical Care Medicine, 2012). Repeat if goal parameters are not met. Since administration of large amount of crystalloid may result in a dilutional coagulopathy the nurse should anticipate the administration of blood products (packed red blood cells, platelets and/or fresh frozen plasma). If the patient is in hypovolemic shock from hemorrhage, the nurse should anticipate orders for packed red blood cells (RBCs). In addition to crystalloid, packed RBCs administration is necessary in significant bleeding to increase the oxygen carrying capacity. Despite the trend of giving fewer blood transfusions, RBC transfusion is indicated for patients with evidence of hemorrhagic shock (Carson et al., 2012).



Figure 8-2 Rapid fluid infuser. Note the two solutions that can be infused together. The solutions are warmed by the heating cylinder.

(Carrie Edgerly MacLeod)

Rapid infusion of large amounts of citrated plasma or blood can lead to hypocalcemia (serum total calcium less than 8.5 mg/dL; serum ionized calcium level less than 4.5 mg/dL or 1.0 mmol/L). Citrate is used in banked blood to prevent coagulation. Once infused, the citrate binds with the calcium in the patient's blood, leading to low serum calcium levels.

Volume resuscitation in hypovolemic shock is only a temporary measure until the etiology can be identified and corrected. In the case of hypovolemia caused by trauma, GI bleeding, or ruptured aneurysm definitive surgery or other intervention is needed to correct the hemorrhaging. In the case of burns, fluid resuscitation must be continued until the patient is hemodynamically stable and the burn area is stable. In DI, administration of replacement ADH may be indicated to reduce the excessive urinary output.

NURSING ACTIONS

- Ensure a large, functioning intravenous access
- Monitor fluid resuscitation parameters. A positive response to fluid therapy would be indicated by a MAP of greater than or equal to 65 mmHg, urine output greater than 0.5 to 1 mL/kg/hr, and a decreasing lactate level
- The nurse must assess the patient closely during fluid resuscitation for signs of respiratory compromise from pulmonary congestion. Signs and symptoms indicating pulmonary congestion and inadequate oxygenation include tachypnea, crackles on auscultation, and decreased oxygen saturation
- Monitoring for complications such as renal insufficiency and cerebral ischemia are the primary concerns for complications. Renal insufficiency or renal failure due to the prerenal etiology of hypovolemia is a serious complication. Early identification and correction of the fluid volume deficit in hypovolemic shock is necessary to prevent cerebral ischemia. As the brain receives less blood flow as a result of hypovolemia, the reduction in end-organ perfusion leads to mental status changes. Mental status changes appear along a continuum from agitation and confusion to lethargy and coma. Identification of the early signs of cerebral ischemia can alert the nurse to the need for immediate volume resuscitation.

NURSING CARE

Maintaining Safety: The patient who is experiencing a reduction in fluid volume will be at risk of falling. The presence of orthostatic hypotension may lead to dizziness, lightheadedness, and syncope when sitting up or ambulating. The nurse should remind the patient to seek

Safety Initiative

Careful Management of Red Blood Cell (RBC) Transfusions

PURPOSE: Assuring that healthcare providers are aware of recognized risks, the re-evaluation of the benefits, and the more cautious indications for RBC transfusions.

RATIONALE: In 2007, the Joint Commission recommended limiting the use of RBC transfusions. In the recommendation, it emphasized the following risks of RBC transfusions:

- **Immune suppression:** Transfusion related immunomodulation occurs each time a person receives a unit of blood.
- **Reactions:** Multiple types of reactions are possible and reactions that were thought to be rare such as transfusion related lung injury are now recognized as being more common.
- **Infection:** In the early stages of infection, many viral illnesses cannot be detected and may be transmitted through RBC transfusions.

- **Risk of error:** The leading cause of death from RBC transfusion is the administration of incompatible blood.

Since that time, the AABB (formerly the American Association of Blood Banks) issued a clinical practice guideline addressing best practices for red blood cell transfusions (Carson et al., 2012).

HIGHLIGHTS OF RECOMMENDATIONS:

1. Transfusion decisions should be influenced by symptoms as well as Hb concentration
2. A restrictive strategy of RBC transfusion (transfuse for Hb less than 7g/dL) is effective in most critically ill hemodynamically stable patients. The exception may be patients with preexisting cardiovascular disease who may benefit from transfusion of RBCs for an Hb 8 g/dL or less.

assistance when getting up. The call bell should be placed within easy reach and the patient should be instructed to get up slowly. The bed or chair alarm should be activated to alert the nurse if the patient gets up independently.

Essential for Safety

Patients with a reduction of fluid volume or hypovolemic shock are at risk of falling. Implement nursing care strategies to prevent falling.

Reflect On

Your patient has just been admitted to the critical care area with massive trauma to both lower extremities. He is a post coronary stent patient on clopidogrel and aspirin. He presents in hypovolemic shock due to significant bleeding. His family states he is a Jehovah Witness. Consider what the treatment options are for this patient. Reflect on your response to the patient and family.

Cardiogenic Shock: Assessment and Management

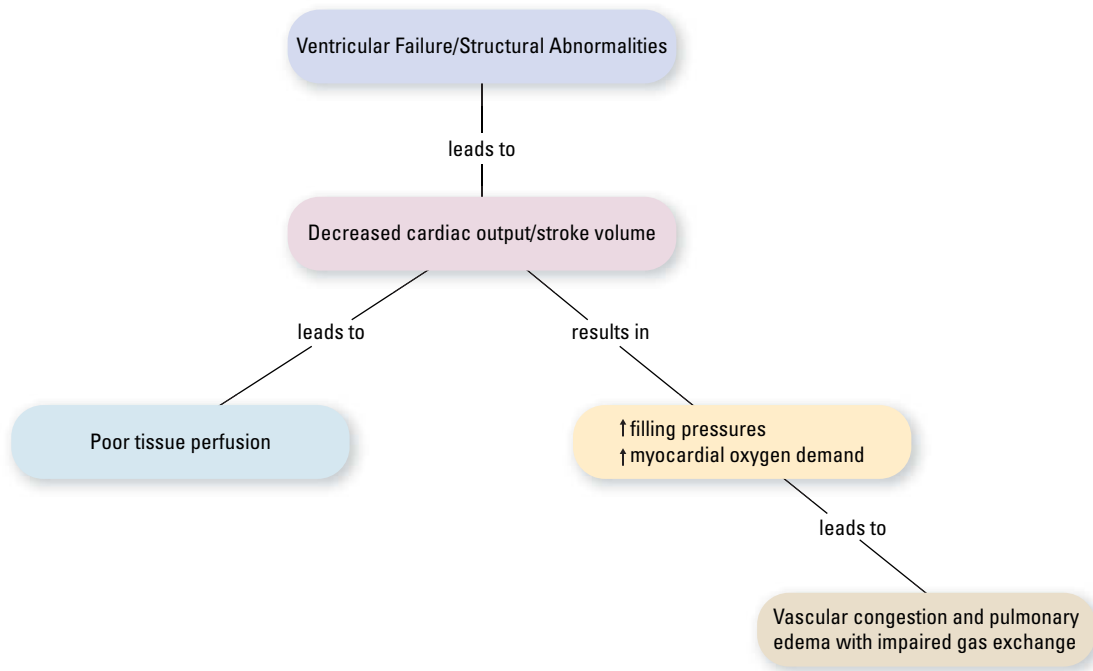
The most common cause of cardiogenic shock is left ventricular failure due to myocardial infarction. Etiologies of cardiogenic shock such as myocardial infarction, cardiomyopathy, and myocarditis lead to cardiac pump failure and the inability to eject blood. Structural abnormalities such as ventricular septal defect and acute mitral regurgitation lead to an acute redistribution of blood within the chambers of

the heart preventing an adequate volume of blood leaving the left ventricular. In an acute rupture of the mitral valve, the valve becomes incompetent so during systole blood shifts into the left atrium instead of moving out through the aortic valve into the systemic circulation. A ventricular septal defect creates a left to right ventricular shift of blood, reducing the amount of blood available for ejection into the systemic circulation. (Visual Map 8-3).

Assessment

The nurse assesses for signs and symptoms of cardiogenic shock. (Refer to Table 8-2):

- Hypotension (systolic blood pressure less than 90 mmHg)
- Tachycardia
- Delayed capillary refill
- Cool, mottled extremities
- Elevated jugular venous distention
- If pulmonary edema is present, the patient will exhibit dyspnea and crackles upon auscultation of the lungs
- Oliguria
- Concentrated urine
- Altered mental status
- Hemodynamic findings include:
 - Reduced cardiac index
 - Reduced stroke volume
 - Elevated PAWP, CVP/RA, SVRI



Visual Map 8-3 Cardiogenic Shock

- Lab findings include:
 - Arterial blood gases: metabolic acidosis, hypoxemia
 - Elevated serum lactate (serum lactate greater than 4 mmol/L)
 - Reduced mixed venous oxygen saturation (SvO_2 less than 60%; $ScvO_2$ less than 70%)

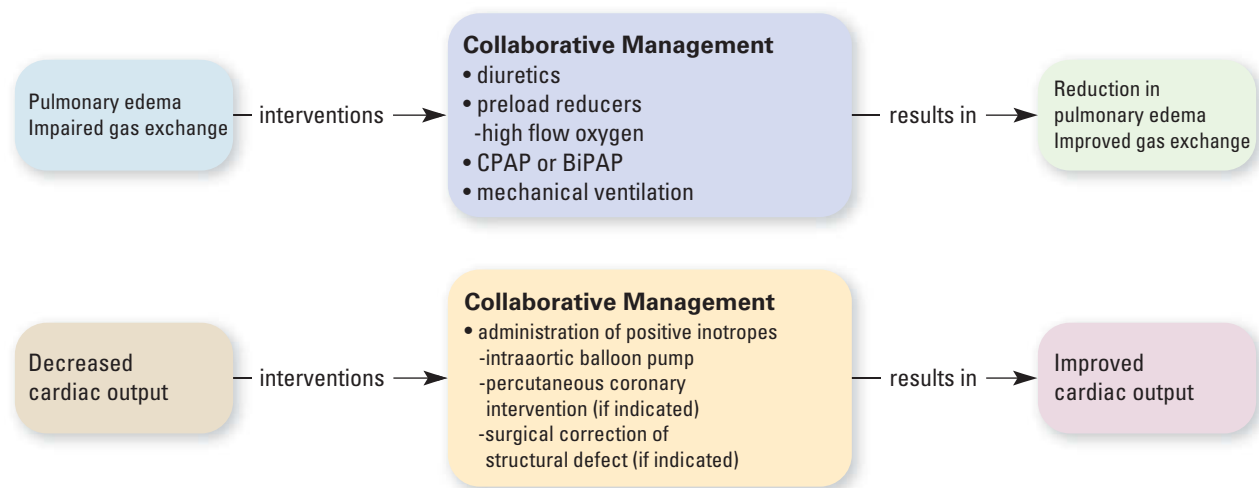
COLLABORATIVE CARE

The nurse works collaboratively with the provider to identify patients at risk for cardiogenic shock. The goal of treatment is to correct the underlying cause if possible, improve myocardial contractility and cardiac output, and manage complication such as pulmonary edema and dysrhythmias. Reperfusion of the myocardium during a myocardial infarction may be accomplished mechanically through percutaneous coronary intervention (PCI) or pharmacologically through administration of a thrombolytic. Acute mitral regurgitation due to valve dysfunction may be corrected by repairing or replacing the valve. Ventricular septal rupture may also be repaired by surgically repairing the defect in the septum. The **intra-aortic balloon pump (IABP)** may be used to increase coronary perfusion and decrease afterload in the patient experiencing a myocardial infarction. The IABP may also be used as a bridge to surgery in the acute mitral valve dysfunction or acute ventricular septal rupture. In these instances, the IABP reduces systemic afterload and facilitates the movement of blood out of the left ventricle into the circulation instead through the incompetent valve or through the septal defect. Inotropic and vasopressor agents

are used to reverse hypotension and improve cardiac contractility. Examples of inotropic and vasopressor agents include dopamine, dobutamine, and norepinephrine. Norepinephrine or dopamine are often initially ordered to treat the hypotension. At moderate doses, dopamine has a stronger positive inotropic effect than norepinephrine, thus improving cardiac contractility. Dopamine can be limited however by tachydysrhythmias, particularly at higher doses. Dobutamine is another positive inotropic infusion that could be added to further improve contractility. Treatment of acute pulmonary edema includes diuretics, ventilatory support, and use of preload reducers like nitroglycerin. These treatments must be used cautiously in the hypotensive patient as they may further lower blood pressure and cardiac output. (Visual Map 8-4).

NURSING ACTIONS

- Assess frequently blood pressure, heart rate, respiratory rate and pulse oximetry.
- Assess cardiodynamics (cardiac index, pulmonary artery pressures).
- Administer medication to support blood pressure, reduce chest pain and reduce pulmonary edema if present.
- Assess the patient closely signs of respiratory compromise from pulmonary congestion. Signs and symptoms indicating pulmonary congestion and inadequate oxygenation include tachypnea, crackles on auscultation, and decreased oxygen saturation.



Visual Map 8-4 Cardiogenic Shock Collaborative Management

- If indicated, prepare patient for percutaneous coronary intervention (PCI) including obtaining informed consent.

NURSING CARE

Facilitating communication: Collaborative management of the patient with cardiogenic shock requiring excellent communication between all of the healthcare professionals. If the patient is to be transferred to the cardiac catheterization lab for a PCI or IABP insertion, it is essential that the nurse communicate all pertinent information upon hand off of the patient to the lab staff. The pertinent information includes: allergies, current medications including dose of infusions, current lab data, current vital signs, and physical assessment information. The family and/or significant others must also be kept informed. The nurse and provider work together to explain the procedures, rationale for use and expected outcomes.

Distributive Shock: Assessment and Management

Distributive shock is characterized by vasodilation and redistribution of blood volume. Etiologies of distributive shock are sepsis, spinal cord injury, and anaphylaxis, with sepsis being the most common.

Septic Shock

Septic shock is defined as the presence of sepsis with refractory hypotension. Collaborative management of septic shock includes identification of the likely source of infection,

provision of antibiotics, and volume replacement. The earlier the septic patient can be identified and the sepsis resuscitation bundle initiated, the better the patient outcome is likely to be. A detailed description of the pathophysiology of septic shock as well as the assessment and collaborative care of the patient with septic shock including when and how to initiate the sepsis resuscitation bundles can be found in Chapter 18.

Neurogenic Shock

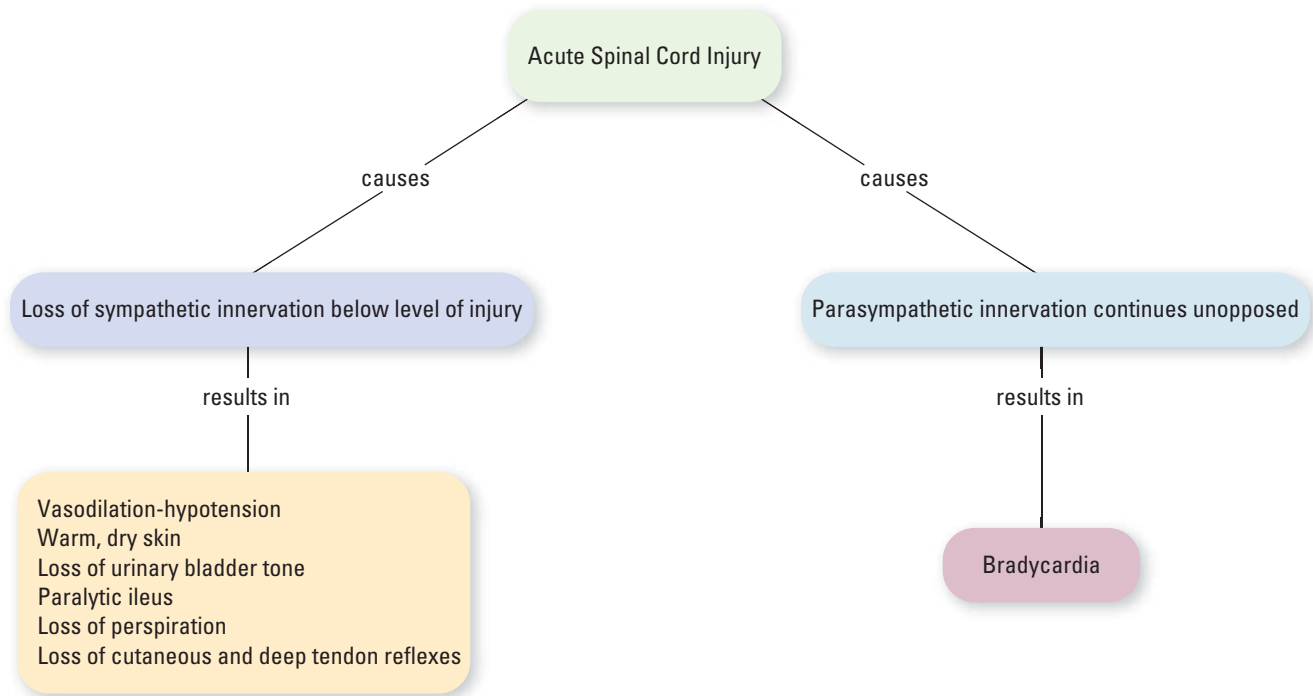
Neurogenic shock (also called *spinal shock*) occurs when a spinal cord injury (SCI) to the cervical and upper thoracic spinal cord causes a temporary interruption in the sympathetic innervation leaving the parasympathetic innervation unopposed (Hickey, 2013). This loss of sympathetic nervous system (SNS) innervation leads to immediate loss of autonomic and motor reflexes below the level of the injury. Specifically, the loss of the autonomic reflexes results in vasodilation and redistribution of the blood volume, loss of cutaneous control of sweat glands resulting in warm, dry skin, and parasympathetic regulation of the heart rate functioning unopposed resulting in bradycardia (Emergency Nurses Association, 2014).

Assessment

The most common cause of neurogenic shock is trauma to the spinal cord from events such as falls and motor vehicle collisions. Other causes of neurogenic shock can be from tumors, infectious conditions and vertebral fractures from osteoporosis (Hickey, 2013).

The nurse assesses for signs and symptoms of neurogenic shock (Visual Map 8-5):

- Hypotension without compensatory tachycardia
- Bradycardia



Visual Map 8-5 Neurogenic Shock

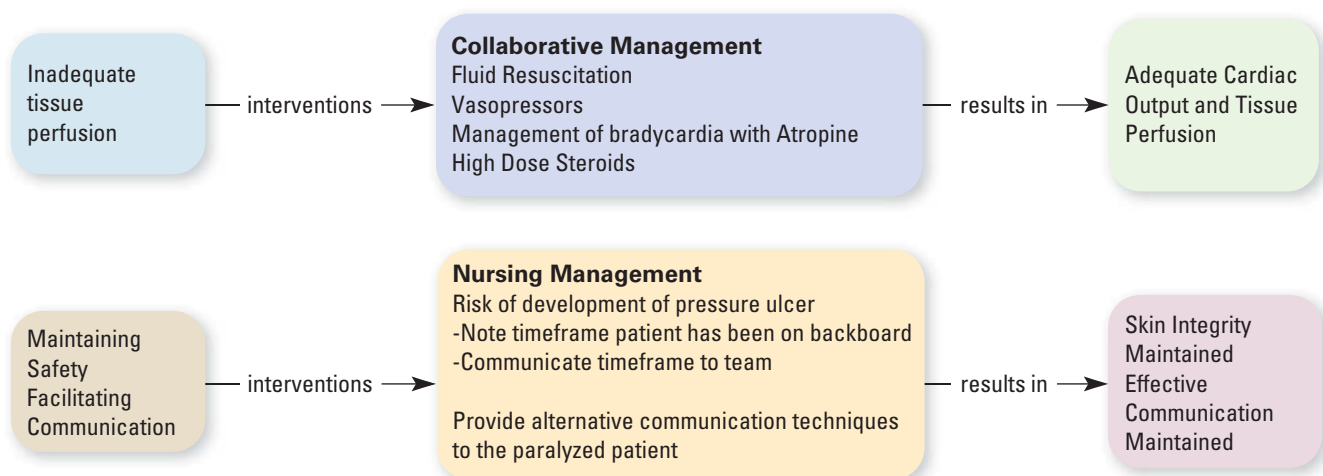
- Warm, dry skin
Associated physical findings include:
- Flaccid paralysis below level of injury.
- Loss of cutaneous and deep tendon reflexes below level of injury.
- Bowel and bladder dysfunction.
- Priapism.

Hemodynamic Findings. In neurogenic shock the MAP, CVP/RAP, and PAWP are decreased, reflecting a reduced

preload due to vasodilation. The nurse reviews the results of imaging studies with the healthcare team to identify the extent of spinal cord bone and ligament injury (Hickey, 2013). (Refer to Table 8-2).

COLLABORATIVE CARE

The goal of treatment is to restore adequate tissue perfusion by implementing interventions to correct the vasodilation and bradycardia. Since these patients with neurogenic shock have had a spinal cord trauma, it is important to

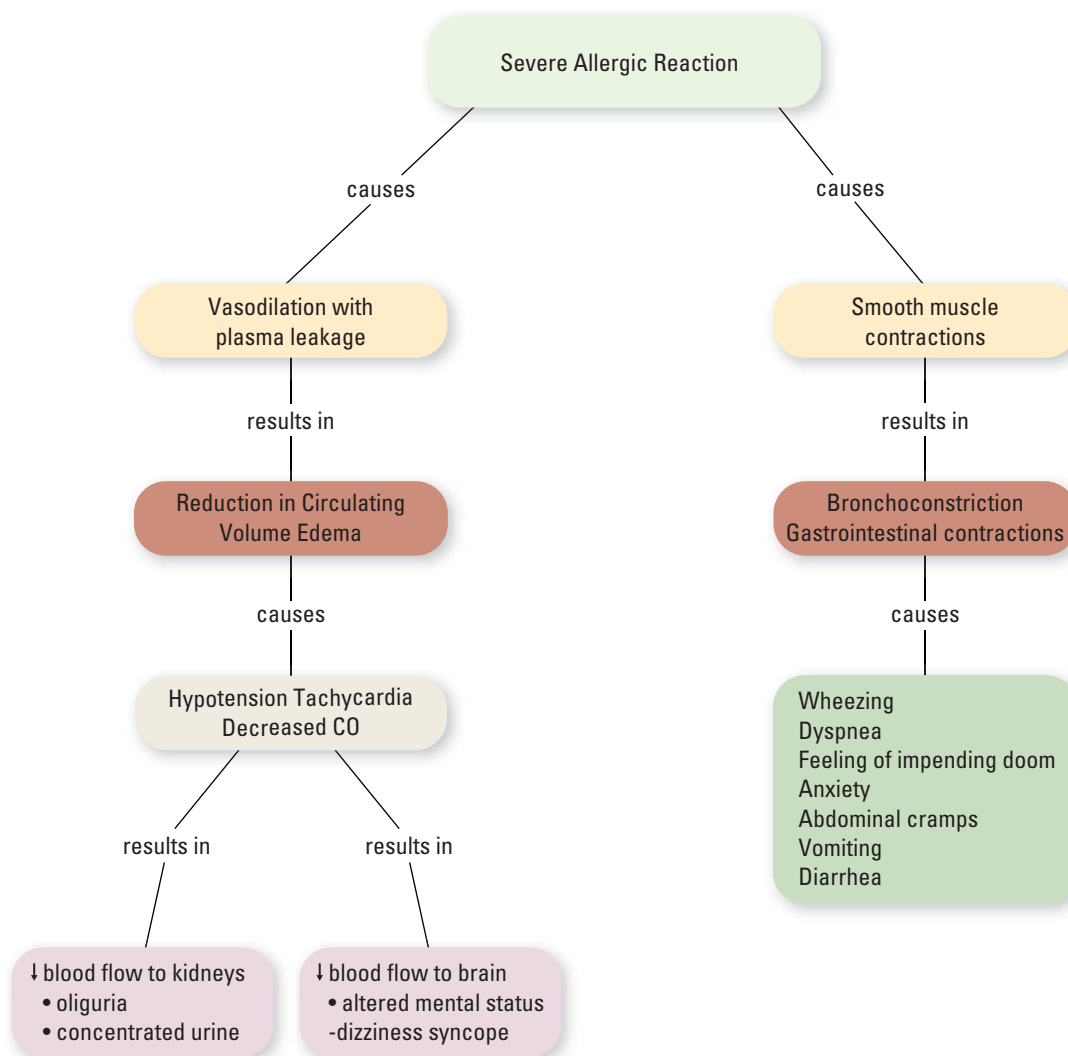


Visual Map 8-6 Neurogenic Shock Collaborative Management

maintain spinal immobilization, maintain the airway, and assist ventilation as needed. Interventions to correct the vasodilation initially include crystalloid administration. If volume resuscitation does not correct the hypotension, then vasopressors administered intravenously should be instituted. Vasopressor infusions such as dopamine, norepinephrine and phenylephrine are preferred as they stimulate the alpha receptors resulting in vasoconstriction. The bradycardia is corrected by the administration of atropine 0.5 mg IV (every 5 min for a total dose of 3.0 mg). Dopamine and norepinephrine infusions also stimulate the beta receptors which may also increase the heart rate. Administration of IV methylprednisolone for the treatment of acute SCI is not recommended, though some recent studies show there may be some benefit in upper cervical injuries (Harrop, 2014). (Visual Map 8-6).

NURSING ACTIONS

- Maintain spinal immobilization at rest and with patient movement.
- Ensure an adequate airway and monitor for adequate respiration, assist if necessary.
- Monitor blood pressure and heart rate. A positive response would be a MAP greater than 65 mmHg and a heart rate within normal limits.
- Administer intravenous vasopressors as ordered. Infuse vasopressors through a central line if possible. If infusion through a peripheral line is absolutely necessary, inspect the site hourly for signs and symptoms of extravasation. Extravasation of vasopressor agents can be extremely damaging to the surrounding tissue. Notify



physician immediately. The intravenous site will need to be changed and anticipate an order from the physician to administer a vasodilatory agent to the affected tissue.

Essentials for Safety

Closely monitor any peripheral intravenous site that has a vasopressor infusing. Notify physician immediately if extravasation is suspected.

NURSING CARE

Maintaining Safety: Following acute SCI, patients are immobilized on a backboard during their evaluation. Recognizing the risk for development of a pressure ulcer, the nurse should note how long the patient has been on the board and alert the team to the elapsed time and risk (Hickey, 2013).

Facilitating Communication: Depending on the level of SCI, the patient may be unable to communicate if intubated and also may be unable to move his/her upper extremities. Alternative means of communication (such as blinking eyes in response to questions) will need to be reviewed with the patient and family.

Anaphylactic Shock

Common antigens that trigger anaphylaxis include:

- Drugs.
- Foods.

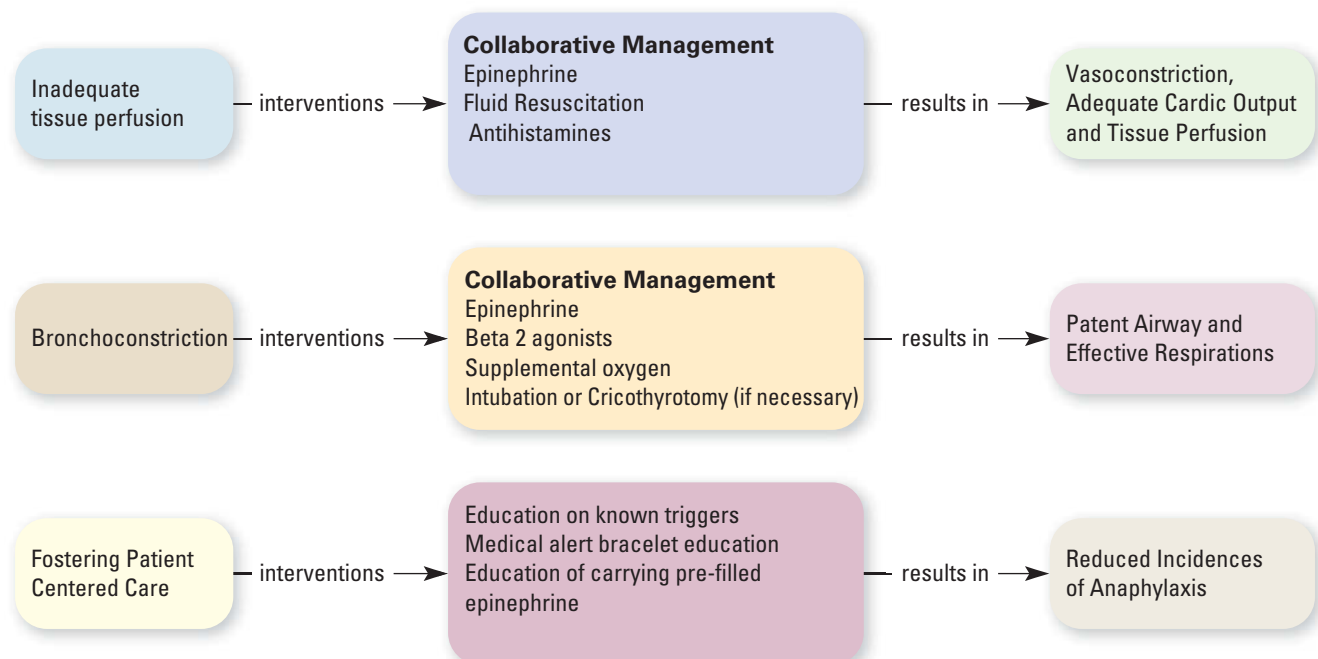
- Contrast for imaging.
- Proteins.
- Venoms like bee stings.
- Latex.

Often an anaphylactic response is not the first exposure for the patient. The re-exposure to the antigen triggers the overwhelming response. Refer to Visual Map 8-7 for Anaphylactic Shock pathophysiology.

Assessment

The nurse assesses for signs and symptoms of anaphylactic shock:

- Wheezing
- Dyspnea
- Flushing
- Urticaria
- Nausea/vomiting
- Diarrhea
- Abdominal cramps
- Palpitations
- Dizziness
- Hypotension
- Tachycardia
- Syncope
- Anxiety
- Feeling of impending doom



Visual Map 8-8 Anaphylactic Shock Collaborative Management

COLLABORATIVE CARE

The symptoms of anaphylaxis can come on quickly and progress to severe compromise and death within a short period of time. Usually there is little time to do definitive lab studies and other tests. The diagnosis is based on history and clinical presentation. The goals of care are to establish a patent airway and restore circulating volume and tissue perfusion. Epinephrine should be considered the first pharmacological therapy for anaphylaxis due to its quick action and ability to act on multiple receptors to block the mediators of the allergic reaction (Song & Lieberman, 2015). Epinephrine produces bronchodilation to improve the respiratory status, and causes peripheral vasoconstriction thus improving the blood pressure and tissue perfusion. The route of administration of epinephrine is usually IM. A 1:1000 solution of epinephrine is used for IM injection. The recommended dose for epinephrine is 0.01 mg/kg of a 1 mg/mL (1:1000) dilution to a maximum dose of 0.5 mg in an adult. If there is inadequate response, the dose can be repeated every 5–15 minutes (Song & Lieberman, 2015). In the event of circulatory collapse, intravenous epinephrine (1:10,000) may be considered. Concurrent administration of intravenous isotonic crystalloid boluses of 1–2 liters are recommended in the hypotensive patient. Other treatments include administration of supplemental oxygen. If the patient is unresponsive to epinephrine, the nurse should anticipate the need for intubation or, on rare occasion, a cricothyrotomy. A variety of antihistamines will also be ordered for the patient in an effort to block the histamine effects. H₁ blockers such as diphenhydramine in a dose of 25–50 mg IV and H₂ blockers such as ranitidine 50 mg IV or famotidine 20 mg IV may be administered (Zilberstein, McCurdy, & Winters, 2014). H₁ blockers do not reverse the cardiovascular, pulmonary or gastrointestinal symptoms of anaphylaxis. For that reason, it is important to recognize that epinephrine should be given first in an anaphylactic reaction. Corticosteroids and B₂ agonists are commonly prescribed, but have limited evidence of effectiveness (Zilberstein, McCurdy, & Winters, 2014). Patients on beta blockers may not respond to epinephrine. Glucagon 1–2 mg IV over 5 minutes followed by infusion of 5–15 micrograms per minutes to counteract the beta blocking effect may be needed. (Visual Map 8-8).

NURSING ACTIONS

- Closely monitor for a patent airway and respiratory drive. Notify provider immediately with worsening respiratory status.
- Assist provider with intubation or cricothyrotomy if indicated.

- Monitor blood pressure and heart rate. A positive response would be a MAP greater than 65 mmHg and a heart rate within normal limits.
- Administer epinephrine as ordered without delay.

Essentials for Safety

The nurse should note the concentration of epinephrine. The ordered routes (IM, IV) require different concentrations.

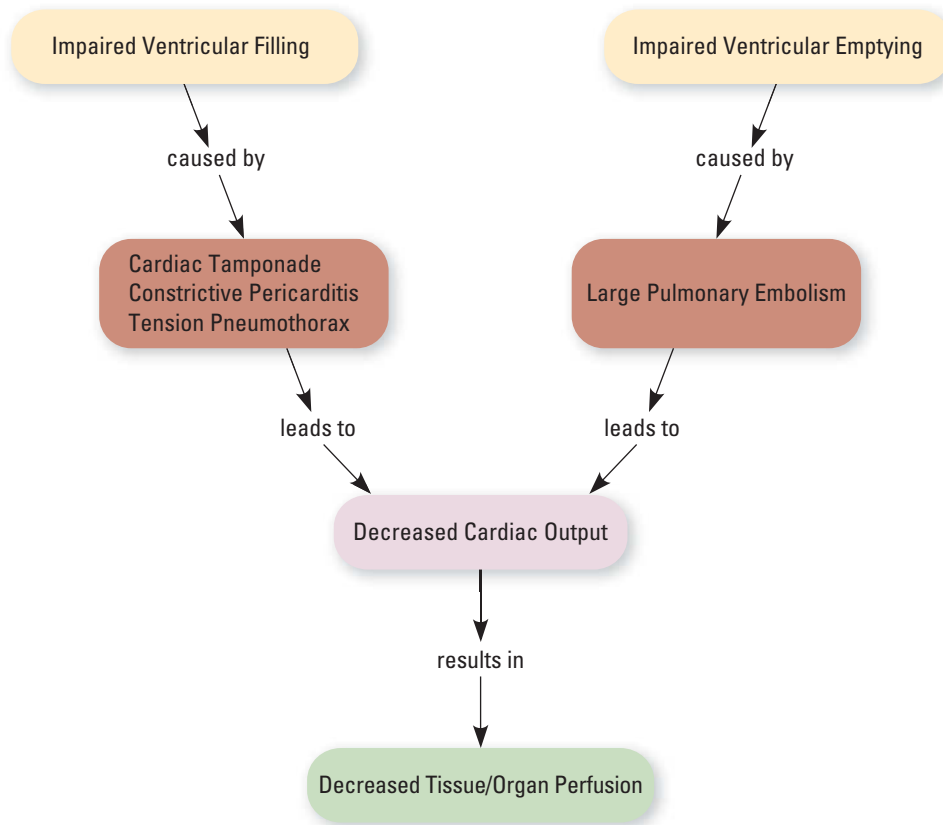
NURSING CARE

Fostering Patient-Centered Care: One of the most important care elements for the patient at risk for anaphylaxis is prevention. Engaging the patient in prevention strategies is essential. The nurse should counsel the patient to avoid known triggers. Provide information and encourage the patient to wear a medical alert bracelet. Encourage the patient to discuss with physician the need to carry a pre-filled epinephrine syringe for prompt self-administration with exposure to anaphylactic trigger.

Obstructive Shock

Patients at risk for developing cardiac tamponade include those who have experienced a blunt or penetrating trauma to the chest. Acute pericarditis and the development of fluid accumulation in the pericardial space may result from viral or bacterial infection, autoimmune disorders like rheumatoid arthritis, uremia, post myocardial infarction, post cardiac surgery, irradiation, or tumors (Foerg & Gardner, 2015). The development of fibrosis or constrictive pericarditis can occur as a result of any of the above conditions. The diagnosis of cardiac tamponade is often through echocardiography. If the patient is very unstable, this will be done at the bedside. (Visual Map 8-9).

Some of the more common risk factors for developing pulmonary embolus are immobilization, pregnancy, hypercoagulable disorders, venous thrombosis, advanced age, atrial fibrillation, and estrogen replacement. Screening tests will include electrocardiogram, chest radiograph, arterial blood gas, troponin and d-dimer (Ouellette, 2012). If the patient has a high probability of pulmonary embolus the patient will be sent for a computed tomography (CT) pulmonary angiography. A CT procedure to determine a pulmonary embolus uses contrast dye, which is contraindicated in patients with renal failure and patients with an allergy to contrast dye. Patients with renal failure may be sent for a ventilation-perfusion scan as an alternative to CT (Ouellette, 2012). A ventilation-perfusion scan uses radioactive materials inhaled through an aerosol and also injected



Visual Map 8-9 Obstructive Shock

through an intravenous catheter rather than contrast dye. Normal ventilation with decreased uptake of the isotope in the circulation may indicate a pulmonary embolus. If the patient is too unstable to travel to these tests, bedside echocardiography may be performed. On echocardiogram, the provider will be looking for right ventricular dysfunction indicating a pulmonary embolism large enough to cause impairment of the right ventricle function.

Assessment

The nurse assesses the patient for the signs and symptoms indicating obstructive shock due to reduced cardiac filling:

- Hypotension
- Pulsus paradoxus (a drop on systolic BP greater than 10 mmHg on inspiration)
- Tachycardia
- Muffled heart tones (cardiac tamponade)
- Reduced capillary refill
- Tachypnea
- Elevated JVD (depending on volume status)
- Crackles
- Unilateral absence of breath sounds (tension pneumothorax)

- Tracheal deviation (tension pneumothorax)
- Oliguria
- Altered mental status

Hemodynamic Findings. In obstructive shock due to filling impairment, the heart rate is increased and the MAP is reduced. The CVP/RAP and PAWP are increased reflecting impaired atrial filling. In fact, in cardiac tamponade, the CVP/RA and PAWP may equilibrate. A reduction in circulating volume due to impaired filling may also lead to a reduction in SV and CO/CI. An elevated SVR may be present reflecting the vasoconstriction due to compensatory mechanisms. Refer to Table 8-2.

The nurse assesses the patient for the signs and symptoms indicating obstructive shock due to increased afterload specifically massive pulmonary embolus:

- Hypotension
- Tachycardia
- Chest pain
- Reduced capillary refill
- Tachypnea
- Pulse oximetry less than 92%
- Arterial blood gases: pO₂ less than 80. Respiratory alkalosis initially.

- Elevated D-dimer
- Lungs may be clear initially
- Signs and symptoms of right ventricular failure (peripheral edema, dull pain right upper quadrant from hepatic congestion)
- Oliguria
- Altered mental status

Hemodynamic Findings

In obstructive shock due to pulmonary embolus, the heart rate is increased and the MAP is reduced. The CVP/RAP will be elevated with a reduced or normal PAWP reflecting an increase in right ventricle pressure. A reduction in circulating volume due to reduced preload to the left ventricle may also lead to a reduction in SV and CO/CI. An elevated SVR may be present reflecting the vasoconstriction due to compensatory mechanisms.

COLLABORATIVE CARE

The goal of care in obstructive shock is to remove the obstruction as quickly as possible. A cardiac tamponade requires a pericardiocentesis in which a provider inserts a needle or catheter into the pericardium to remove the accumulated fluid. In a tension pneumothorax, a provider inserts a large bore needle (i.e., 14-gauge) or chest tube into the pulmonary space causing immediate evacuation of air, therefore reexpanding the lung. Once the lung is re-expanded, the heart is not compressed and filling will be improved. A pulmonary embolism large enough to cause shock is treated with a thrombolytic such as tissue plasminogen activator or removed during a surgical embolectomy or fragmented and aspirated through percutaneous mechanical thrombectomy. These mechanical procedures

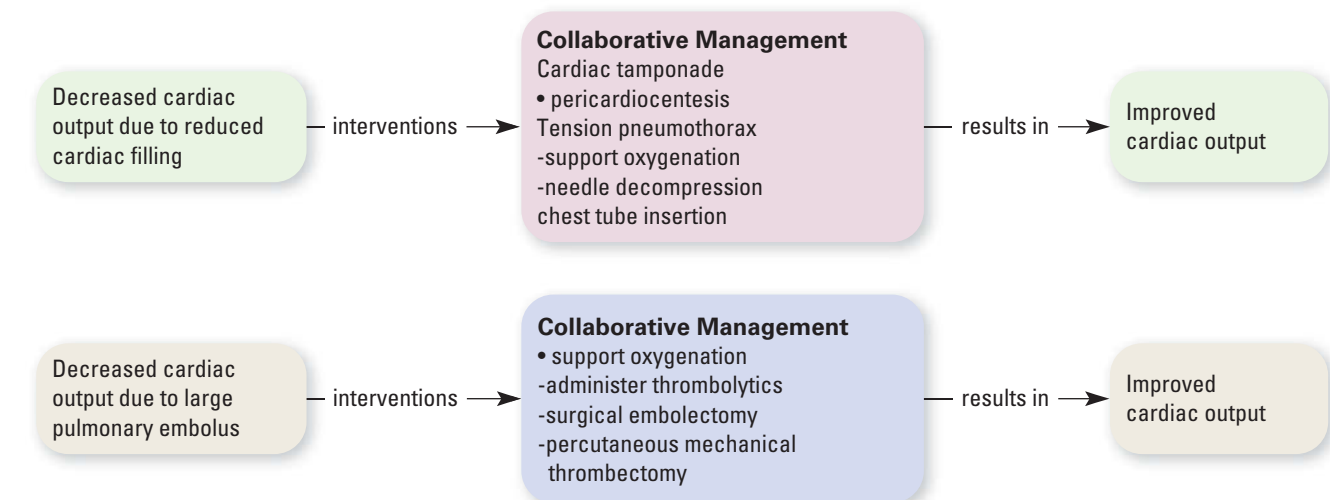
may be performed in high-risk patients with an absolute contraindication to thrombolytic treatment, and those in whom thrombolytic treatment has not improved the hemodynamic status (Ouellette, 2012). If tissue plasminogen activator is used the dose is 100 mg as an intravenous infusion over two hours. The most important side effect to observe for is bleeding.

NURSING ACTIONS

- Assist provider with preparation of equipment for pericardiocentesis or chest tube placement. Typically, it is the responsibility of the nurse to set up the chest drainage system prior to chest tube placement.
- Closely monitor heart rate, blood pressure, respiratory rate and pulse oximetry during procedures.
- Administer the thrombolytic without delay for patient with hemodynamically unstable pulmonary embolism. Closely monitor the patient for side effects such as external bleeding or signs and symptoms of cerebral bleeding, such as change in level of consciousness or change in neurological exam. Patients receiving thrombolytics must have a baseline neurological exam done prior to initiation of the medication. (Visual Map 8-10).

NURSING CARE

Enhancing comfort: The patient experiencing a cardiac tamponade, tension pneumothorax, or large pulmonary embolism will be markedly dyspneic and anxious. The nurse should stay with the patient to provide reassurance while facilitating appropriate definitive care. Allow the patient take a position of comfort. Provide oxygen support as necessary.



Visual Map 8-10 Obstructive Shock Collaborative Management

Maintaining Safety: The nurse must ensure that the universal protocol is followed when assisting the provider with procedures such as chest tube insertion or pericardiocentesis. Use of the universal protocol prevents a wrong patient, wrong procedure, wrong site procedure. This

pre-procedure verification includes a time out when all healthcare providers are present verifying that the patient and procedure are correct and marking the procedure site based on available diagnostic and physical exam information.

Gerontological Considerations

The elder patient experiencing shock may have many comorbidities and be on many medications that can alter the blood pressure, heart rate and respiratory rate from normal values. For example, in a hypertensive patient, the patient may have hypoperfusion well before the minimum values associated with shock. Patients on beta blockers have a difficult time mounting an increased heart rate in response to hypovolemia. When assessing the patient for shock signs and symptoms, it is the change from the patient's baseline that we are most interested in. Additionally, when the elderly are being fluid resuscitated, careful pulmonary assessment including respiratory rate, breath sounds, and pulse oximetry must be done to prevent fluid overload.

Bariatric Considerations

Obese patients have a reduced ability to withstand shock due to the significant impact of obesity on their cardiovascular system. Their greater needs for perfusion have already resulted in an increase in stroke volume, cardiac workload, and left ventricular workload. Although, fluid volume replacement is essential in hypovolemic shock, volume must be replaced cautiously to avoid heart failure.

Obesity may make the placement of an intravenous line extremely difficult. The obese patient may benefit from the placement of an intraosseous (IO) line access for fluid resuscitation. Following resuscitation from hemorrhagic shock, obese patients, especially those with "fatty livers", are at higher risk for hepatic ischemia and liver dysfunction than leaner individuals.

Commonly Used Medications

Dopamine

Introduction

Dopamine acts on beta 1, beta 2, and dopaminergic adrenergic receptors to increase contractility and afterload, thereby improving cardiac output.

Dose and Desired Effects: Dopamine is initiated at 2 to 5 mcg/kg/min and titrated to a maximum of 20 mcg/kg/min. Every patient responds differently to the dose ranges, however, a range of 5–15 mcg/kg/min is expected to have the beta 1 receptor effect of increasing contractility and increasing heart rate. Doses above 15 mcg/kg/min stimulate the alpha adrenergic receptors predominantly. Expected actions would be an increase in CO and SVR (Gahart & Nazareno, 2015).

Nursing Responsibilities:

- Dopamine should always be administered via an infusion pump.
- Prior to initiation, before each titration, and at the peak action of dopamine, the nurse must assess blood pressure, heart rate, respiratory rate, and oxygen saturation.

Frequent assessment of these parameters should continue throughout the infusion period.

- Infuse through a central line if available. If peripheral site is used monitor for the presence of extravasation.

Side and/or Toxic Effects: The nurse should pay particular attention to the heart rate. Stimulation of the beta 1 receptor can cause excessive increases in heart rate increasing myocardial oxygen demand. The patient should always be on a cardiac monitor as an increase in dysrhythmias can occur. At high doses, the nurse should assess extremities for decreased pulses, temperature, and paresthesias indicating significant peripheral vasoconstriction.

Epinephrine

Introduction

Epinephrine is used in the patient with shock as a vasopressor to increase blood pressure and cardiac output and also in anaphylactic shock to bronchodilate. Epinephrine stimulates alpha, beta 1, and beta 2 receptors strengthening cardiac contractility, increasing heart rate and vasoconstriction. It also

relaxes smooth muscle particularly in the bronchioles causing bronchodilation.

Dose and Desired Effect: As a vasopressor, Epinephrine is administered in a dose range of 2–10 mcg/min titrated to desired effect. Expected actions would be to increase blood pressure, heart rate, CO and SVR. As a bronchodilator for anaphylactic shock, epinephrine is administered IM 0.3 mg – 0.5 mg using a 1:1000 solution. Expected actions would be to produce bronchodilation indicated by a reduced work of breathing, lower respiratory rate, and less wheezing. (Gahart & Nazareno, 2015).

Nursing Responsibilities:

- Epinephrine should always be administered via an infusion pump.
- Prior to initiation, before each titration, and at the peak action of epinephrine, the nurse must assess blood pressure, heart rate, respiratory rate, and oxygen saturation. Frequent assessment of these parameters should continue throughout the infusion period.
 - Infuse through a central line if available. If peripheral site is used monitor for the presence of extravasation.

Side and/or Toxic Effects: The patient should always be on a cardiac monitor as an increase in dysrhythmias can occur. At high doses, the nurse should assess extremities for decreased pulses, temperature and paresthesias indicating significant peripheral vasoconstriction.

Norepinephrine

Introduction

Norepinephrine is used in shock states as a vasopressor. It stimulates alpha and beta 1 and 2 receptors. As compared to dopamine and epinephrine, the alpha effects are more pronounced with norepinephrine administration leading to vasoconstriction with less strengthening of cardiac contractility and increases in heart rate.

Dose and Desired Effect: Usual range is 2–12 mcg/min with initial infusion, and as low as 1 mcg/min and as high as 30 mcg/min in refractory shock. Expected effects are vasoconstriction and increase in blood pressure, CO, and SVR.

Nursing Responsibilities:

- Norepinephrine should always be administered via an infusion pump.
- Prior to initiation, before each titration, and at the peak action of norepinephrine, the nurse must assess blood pressure, heart rate, respiratory rate, and oxygen saturation. Frequent assessment of these parameters should continue throughout the infusion period.
 - Infuse through a central line if available. If peripheral site is used monitor for the presence of extravasation.

Side and/or Toxic Effects: The patient should always be on a cardiac monitor, as an increase in dysrhythmias can

occur. At high doses, the nurse should assess extremities for decreased pulses, temperature and paresthesias indicating significant peripheral vasoconstriction.

Phenylephrine

Introduction

Phenylephrine is used in shock states as a vasopressor. It is purely an alpha receptor drug leading to vasoconstriction without an increase in heart rate.

Dose and Desired Effect: Dose range is 40–180 mcg/min titrated to effect. Expected effect is to increase blood pressure, CO and SVR.

Nursing Responsibilities:

- Phenylephrine should always be administered via an infusion pump.
- Prior to initiation, before each titration, and at the peak action of phenylephrine, the nurse must assess blood pressure, heart rate, respiratory rate, and oxygen saturation. Frequent assessment of these parameters should continue throughout the infusion period.
 - Infuse through a central line if available. If peripheral site is used monitor for the presence of extravasation.

Side and/or Toxic Effects: The patient should always be on a cardiac monitor as bradycardia can occur. At high doses, the nurse should assess extremities for decreased pulses, temperature, and paresthesias, indicating significant peripheral vasoconstriction.

Vasopressin

Introduction

Vasopressin is used in vasodilatory shock states usually septic shock. It is used to potentiate the vasoconstrictor effects of other vasopressor agents such as norepinephrine.

Dose and Desired Effect: Dose is 0.03 units/min usually used to supplement other vasopressor infusions (Dellinger et al., 2013).

Nursing Responsibilities:

- Vasopressin should always be administered via an infusion pump.
- Vasopressin is usually not titrated but rather infused at a set dose of 0.03 units/min. Prior to initiation and at the peak action, the nurse must assess blood pressure, heart rate, respiratory rate, and oxygen saturation. Frequent assessment of these parameters should continue throughout the infusion period.
 - Infuse through a central line if available. If peripheral site is used monitor for the presence of extravasation.

Side and/or Toxic Effects: Since the addition of this medication enhances vasoconstriction, the nurse should assess extremities for decreased pulses, temperature, and paresthesias, indicating significant peripheral vasoconstriction.

Building Technology Skills

Technologies applied in the treatment of shock include the rapid infuser.

Rapid Infuser

Indications

A rapid infuser is used to deliver large amounts of warmed crystalloid or blood to a patient over a short period of time.

Principles of Treatment

Patients who are experiencing acute severe hypovolemia require large amounts of crystalloid or colloid solution over a short period of time to support their tissue perfusion. Common infusion pumps do not deliver volume at the rate necessary to achieve this goal. Additionally, the rapid infuser warms the solutions during infusion to prevent hypothermia.

Methods

The rapid infuser is best used with a large gauge intravenous access such as a 14-gauge angiocath or preferably a central line with an 8.5 or 9 French catheter. Manufacturer

specific tubing is attached to a bag (s) of crystalloid or packed red blood cells and loaded into the infuser. Do not use rapid infusers for infusing platelets or cryoprecipitates. The infuser delivers the volume under pressure (300 mmHg). The flow rate is dependent on the size of the tubing and size of the catheter. Some tubings are designed to deliver up to 950 ml/min. Actual delivery of that volume is dependent on the size of catheter through which it is infusing.

Nursing Responsibilities

Nursing responsibilities include:

- Setting up the fluid and priming the tubing paying particular attention to minimize air.
- Checking the packed red blood products.
- Replacing the fluid or blood on the infuser in a timely fashion when infusion complete.
- Keeping an accurate log of volume infused.

Complications

- Fluid volume overload
- Air embolus

Why/Why Not?

Mr. R is admitted to the intensive care unit for thrombolytic administration to treat his pulmonary embolism. Soon after the tissue plasminogen activator infusion is complete, his blood pressure drops from 124/80 (95) to 78/40(53). He is evaluated for signs and symptoms of bleeding which are negative. The healthcare provider orders dopamine 5 mcg/kg/min to keep his systolic blood pressure over 90 mmHg. Fifteen minutes after the Dopamine is started at

5 mcg/kg/min, his blood pressure is 82/48 (59) and his heart rate has increased from 88 beats per minute, sinus rhythm to 124 beats per minute, sinus tachycardia with frequent premature ventricular contractions. Should you titrate the dopamine up to reach the goal systolic blood pressure of 90 mmHg? Why or why not?

See answers to Why/Why Not? in the Answer Section.

Case Study 8.1

Hypovolemic Shock

An 84-year-old male was found at home unresponsive but breathing after vomiting large amounts of blood. He was transported by EMS to the emergency department (ED) with a massive GI bleed. Treatment by EMS en route included placement of two large-bore 16-gauge intravenous catheters, infusion of 0.9% normal saline, and 100% oxygen via a nonrebreather mask. His vital signs on admission were HR 120 regular, BP 50/30, RR 12, pulse oximetry 96%. He appears pale and slightly diaphoretic. He has

delayed capillary refill and absent peripheral pulses, but still has a carotid pulse. Lungs are clear to auscultation and his abdomen is distended. The patient has a remote history of excessive drinking, but according to his wife he has not had a drink in 25 years. Past medical history includes hypertension, depression, esophageal varices, cirrhosis, question of esophageal cancer, and abdominal aortic aneurysm repair performed 4 years ago. On admission, labs revealed: sodium 150, potassium 3.9, BUN 30, creatinine 1, glucose 178, hemoglobin 5.6, hematocrit 25, INR 2.28, PT 22, PTT 35.

1. Signs and symptoms indicating hypovolemia in this patient include:
2. What lab tests indicate hypovolemia?
3. Explain the relationship between the patient's hypotension, tachycardia, and peripheral vasoconstriction.
4. The patient's pulse oximetry is 96%. Further evaluate the saturation, considering his hemoglobin is 5.6.
5. What are immediate priorities in the collaborative management of this patient?
6. How would a nurse recognize that a hypovolemic patient had received adequate volume replacement?

Case Study 8.2

Cardiogenic Shock

A 56-year-old male presents to the ED complaining of substernal chest pain and dizziness. He is dyspneic, diaphoretic and pale. Vital signs are as follows: pulse 120 and irregular; BP 78/52; RR 28; pulse oximetry 88% on room air; afebrile. Crackles are heard bilaterally upon chest auscultation. A 12-lead electrocardiogram is performed and demonstrates ST elevation in the anterior leads. The ED is awaiting the cardiac catheterization team to transfer the patient for an acute angioplasty. The patient is placed on supplementary oxygen via nasal cannula, and a dopamine infusion is started at 5 mcg/kg/min to improve the blood pressure.

Furosemide 20 mg IVP is administered. Repeat BP is 100/74 and pulse oximetry is 94% on 4 L nasal cannula. Nitroglycerin infusion is started at 5 mcg/min and titrated to control chest pain while keeping systolic BP greater than 90. The patient is then transferred to the catheterization lab.

7. What signs and symptoms indicate cardiogenic shock in this patient?
8. Explain the pathophysiology for the patient's dyspnea and hypoxia.
9. What is the likely etiology of his cardiogenic shock?
10. What are immediate priorities in the collaborative management of this patient?

Case Study 8.3

Anaphylactic Shock

A 24-year-old female presents to the ED with a possible allergic reaction. The patient has a documented nut allergy and became ill during a wedding shower at a private residence. Normally she has an epinephrine pen with her, but for the event today she changed purses and forgot to switch over the epinephrine pen to the current purse. Her friends drove her to the ED as they believed it would be faster than calling an ambulance. She arrives

obviously dyspneic with audible wheezing. Her lips and eyes are swollen and she is flushed. She is complaining of abdominal cramping. Vital signs are as follows: BP 82/40; pulse 132 and regular; RR 42; pulse oximetry is 86% on room air; afebrile.

11. What signs and symptoms indicate that this is anaphylactic shock and not just a mild allergic reaction?
12. Identify the collaborative priorities of care as this patient enters the emergency department.

Case Study 8.4

Obstructive Shock

A 64-year-old female has been admitted to the medical-surgical unit for exacerbation of chronic obstructive pulmonary disease secondary to pneumonia. The patient has been receiving bronchodilator therapy via nebulizer and intravenous antibiotics for the last 48 hours. She puts her call light on and states she is having difficulty breathing. You arrive to find her obviously dyspneic, diaphoretic and more lethargic. Vital signs are as follows: BP 70/40; pulse 142 and regular; RR 36; pulse oximetry 88%; low grade fever. Upon auscultation of her lungs, you notice very diminished, possibly absent breath sounds on the left and a few crackles on the right. You notify the

provider who orders a stat portable chest x-ray and asks for chest tube insertion equipment to be brought to the patient's room.

13. What signs and symptoms indicate a tension pneumothorax and obstructive shock rather than a simple pneumothorax?
14. Explain the relationship between the tension pneumothorax and the hypotension.
15. In addition to chest tube equipment, what other interventions would you anticipate this patient might need?

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 8.1 A patient is admitted immediately post a motor vehicle collision with a head injury and a fractured femur bleeding profusely from the site. He has a history of hypertension being treated with hydrochlorothiazide and metoprolol. His blood pressure is 100/76, heart rate 68, respiratory rate 16, SpO₂ is 96% on a 100% non-rebreather mask. Within 15 minutes of arrival, his repeat vital signs are: BP 80/42, HR 56, respiratory rate 6, SpO₂ 90% on 100% non-rebreather mask. What is the priority for treatment? Why is his heart rate not tachycardic? What other parameters do you need to assess to determine if the patient has neurogenic shock? What is the treatment priority for the hypotension fluid administration or a vasopressor infusion and why?
- 8.2 You have just received an anaphylactic shock patient from the ED to your ICU. In the ED, the patient received epinephrine 0.3 mg IM repeated with

0.5 mg IM, 2 liters 0.9% normal saline IV bolus via 16-gauge peripheral catheter in antecubital space, and 50 mg diphenhydramine IV. The patient was intubated and placed on the mechanical ventilator. Just prior to transfer from the ED, an epinephrine infusion was started at 2mcg/min. It is infusing through the peripheral line. VS prior to transfer were: BP 90/50, HR 89, respiratory rate 14 assisted. Upon transfer the blood pressure was noted to drop to 78/42 with a heart rate of 94. You titrate the epinephrine up to 3 mcg/min. The blood pressure continues to drop despite titrating the epinephrine infusion upward to 5 mcg/min. Heart rate remained unchanged. What are all the possible reasons and for the epinephrine infusion failing to correct the blood pressure in this patient? Also reflect on what the corrective actions would be.

See answers to Chapter Review Questions in the Answer Section.

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Chapter 9

Care of the Patient Following a Traumatic Injury

Tricia L. Charise, MS, ACNP

Abbreviations

ABCs	Airway, Breathing, Circulation	EMS	Emergency Medical Services
ABGs	Arterial Blood Gases	FAST	Focused Assessment with Sonography
ALI	Acute Lung Injury	GCS	Glasgow Coma Scale
AVPU	Alert, Responds to Verbal Stimuli, Responds to Painful Stimuli, Unresponsive	MAP	Mean Arterial Pressure
BiPAP	Bilevel Positive Airway Pressure	MODS	Multi-organ Dysfunction Syndrome
BMI	Body Mass Index	MTP	Massive Transfusion Protocol
CDC	Centers for Disease Control and Prevention	MVC	Motor Vehicle Crash
DCR	Damage Control Resuscitation	PEA	Pulseless Electrical Activity
DPL	Diagnostic Peritoneal Lavage	RSI	Rapid Sequence Intubation
		RTS	Revised Trauma Score



Learning Outcomes

Upon completion of this chapter, the learner will be able to:

1. Explain factors prior to a trauma patient's arrival at a hospital that will impact the patient's survival.
2. Describe the use of the primary and secondary survey to assess and manage the care of a trauma patient immediately following arrival in the emergency department (ED).
3. Discuss commonly occurring traumatic injuries that impair a patient's airway and breathing and prioritize the immediate management of the injuries.
4. Describe assessment for and management of ineffective circulation in the trauma patient.
5. Explain assessment and prioritize management for disability in the trauma patient.
6. Prioritize environmental and abdominal assessment and management of the trauma patient.
7. Complete the secondary survey of a trauma patient and plan appropriate interventions.

Introduction

Trauma (unintentional injury) is the number one cause of death in the United States for people ranging in age from 1 to 44 (Heron, 2016). In addition, there are two cases of permanent disability for every death. As populations grow, the number of traumatic injuries increases while the aging population presents the challenge of treating more elderly and comorbid trauma patients. In order to provide competent care, the nurse needs to be knowledgeable about the standardized care of the trauma patient. In addition, the nurse is responsible for understanding the different types of traumatic injury and anticipating the associated body system injuries that can occur. Only by providing such evidence-based care can the nurse obtain the best outcomes for trauma patients.

Factors Impacting Trauma Patients' Survival

Trauma is defined as a physical injury or wound caused by external forces. Each year in the United States, trauma accounts for 41 million ED visits and 2.3 million hospital admissions (CDC, 2016). The National Trauma Data Bank reported that in 2014 the most common types of trauma were:

- Fall–42%
- Motor vehicle traffic–27%

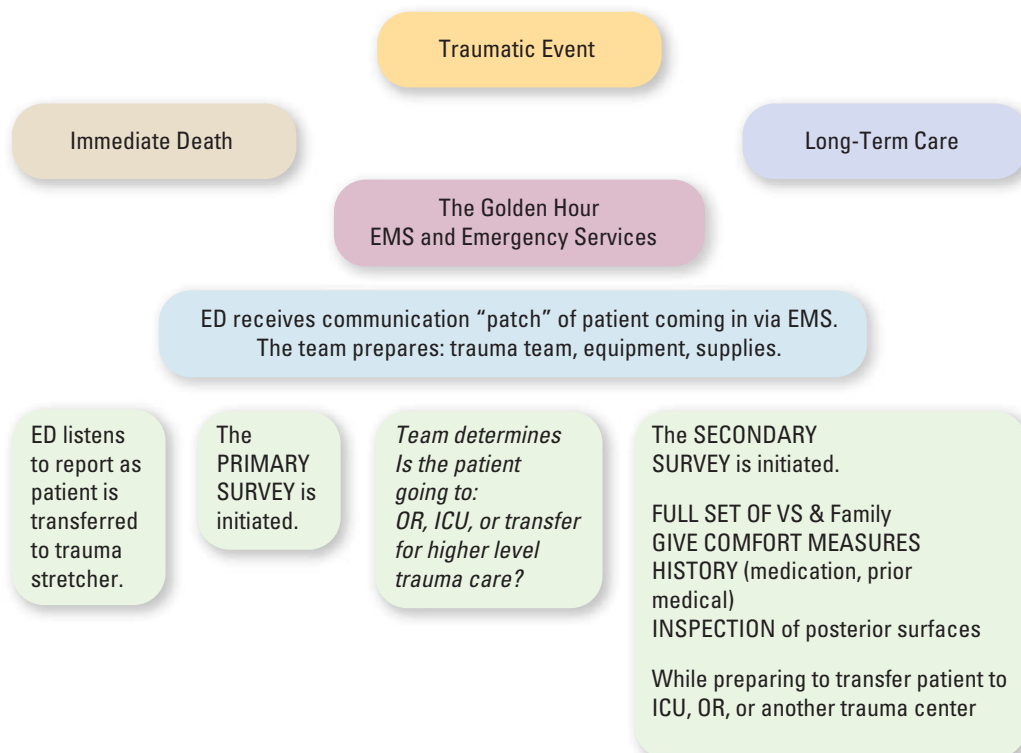
- Pedestrian struck–7%
- Firearm and cut/pierce–8.5%
- Other transport injuries–5%

Mechanisms of Injury

The preparation of care for the trauma patient includes a report on the mechanism of injury. The mechanism of injury determines the type of energy transferred from the environment to the person. There are several different types of energy sources responsible for trauma:

- Mechanical or kinetic energy
- Thermal energy
- Chemical energy
- Electrical energy
- Radiant energy
- Oxygen deprivation that is a cause, not an agent

Body tissue is injured when the energy source is beyond the body's resistance to tolerate that source. For example, a person striking his leg on a desk corner may develop a bruise. The body tissue is intact; however, the underlying vessels are ruptured. The skin tissue was able to resist or tolerate the strike to the leg, but the underlying vessels did not tolerate the energy source. The types of tissues injured in a trauma are important. The four different types of tissues that may be damaged are epithelial, connective, muscle, and nerve.



The energy sources violating the body can be further categorized as internal and external forces (mechanical energy). An external force equation that is important to remember is:

$$\begin{aligned} \text{amount of force} &= \text{mass of object or body} \\ &+ \text{velocity at which it is moving} \end{aligned}$$

The types of external force injuries are deceleration and acceleration. Deceleration occurs when a force stops or decreases the velocity of the moving victim, whereas acceleration is an increase in speed, for example, a pedestrian being struck by a car. Other types of forces include bullets, fists, stabbing, explosions, or blasts (may have blunt force or penetration injuries). Internal forces cause stress and strain on the body. Stress is the force applied to deform the body, and strain is tissue damage that results from stress. The different types of stress include tensile (tissue cells separated), compression (pressed together), and shearing (stress resulting from tangential force).

Types of Injuries

Injury to the different body systems is classified in two categories: blunt and penetrating trauma. In blunt trauma, the skin remains intact with widespread injury underlying the area of impact. Penetrating trauma is an open injury or disruption of the skin. The most common penetrating injuries are lacerations, punctures, and amputations.

The National Trauma Data Bank reported in 2014 that injuries to the lower extremities occurred in 37% of trauma patients, followed closely by injuries to the head at 36% and injuries to the upper extremities in 29% of patients. Injuries to the face and thorax followed at 24% and 21%, respectively. Spinal injuries occurred in 16.9% of trauma cases. Education, protective gear, and changes in the population's activities can all be considered contributing factors to these statistics.

Trimodal Distribution of Trauma Deaths

Trauma deaths occur in three peaks related to the onset of the trauma. In the first peak, deaths occur within minutes of the injury due to major neurological or vascular injury. Medical treatment rarely improves the outcome. The second peak occurs during the golden hour due to intracranial hematoma and major thoracic or abdominal injuries. **Golden hour** is a term used in trauma care that refers to the first hour following the initial traumatic injury. Patient survival rates increase with the proper care in the golden hour. The third peak occurs days to weeks after the trauma due to sepsis and multiple organ failure (Eachempati, Reed, St. Louis, & Fischer, 1998; Whitehorne, 1989). Nurses

are able to prevent death and disability by their systematic assessment and actions during the golden hour as well as by their ongoing evaluation of nursing care and vigilance in preventing sepsis and multiple-organ failure.

Trauma deaths that occur during the first 24 hours are often the result of the **lethal triad**. In trauma, the lethal triad refers to the spiraling effects of acute coagulopathy, acidosis, and hypothermia observed in exsanguinating patients. With hypovolemia, there is inadequate perfusion and decreased oxygenation resulting in anaerobic metabolism, lactate production, and metabolic acidosis. When this triad progresses to acute coagulopathy, there is a four-fold increase in patient mortality.

Nurses have a responsibility to address and intervene to prevent the lethal triad. Preventing hypothermia by providing a warm room and warm fluids is an intervention that is nurse controlled and is absolutely essential. The concept of permissive hypotension—maintaining the trauma patient's systolic blood pressure at 90—has been advocated by some clinicians but remains controversial. Less controversial are the protocols for volume replacement in the hypovolemic patient using rapid infusers, large-bore IVs, or intraosseous access to replace rapidly the volume of the exsanguinating trauma patient in a timely manner. Trauma centers have developed methodical protocols with lab services to ensure the timely delivery of isotonic fluids and blood products including plasma and platelets to prevent the lethal trauma triad. Nurses' close attention to their patients' temperature, intake and output, most recent labs, and arterial blood gases are critical to the use of the protocols and the prevention of deaths from the lethal triad (Jansen, Thomas, Loudon, & Brooks, 2009).

Special Populations

The elderly, pediatric patients, and pregnant women are considered special populations. According to the CDC (2016), injuries are the number one cause of death for all children over the age of 1. Although most pediatric injuries can be managed in the ED, severe trauma cases have a 25% decrease in mortality when treated in a Level I Pediatric Trauma Center. Motor vehicle collisions are the primary mechanism of traumatic injury for people between ages 14 and 29. However, for children under age 7 and adults over age 75, falls become a major contributor to traumatic injuries. Seventy percent of all trauma patients under the age of 70 are male, but after age 70 most patients are female (CDC, 2009).

Although the algorithm does not change for the age or sex of the patient, special consideration is given to certain populations. The hemodynamics of a 3-year-old are quite different from those of a healthy 25-year-old male.

Gerontological Considerations

In 2014, fatalities were most common in trauma patients age 70 and older, with falls being the leading cause of both injury and death (National Trauma Data Bank, 2014). The elderly (defined as older than 65) are the fastest growing trauma population (Ayoung-Chee et al., 2015).

Factors leading to poor outcomes in the geriatric trauma patient include comorbidities, age specific alterations in response to injury, anticoagulants, medication mismanagement, and delirium. The nurse should be hypervigilant in the assessment of these patients and take extra care with medication management. In addition, the nurse should be aware of the high risk for delirium and utilize measures to decrease the risk of delirium (Kozar et al., 2015, Calland et al., 2012). A complete medical history and medication list should be obtained

from elders who have sustained a traumatic injury as soon as possible.

Whereas a young, fit patient might easily manage one or two rib fractures with simple analgesia, the same injury in an older adult frequently leads to pneumonia and respiratory failure if not managed with aggressive turning, coughing, and deep breathing.

Older adults may not respond to hypovolemia with tachycardia and hypotension as readily as younger adults.

Failure rates for nonoperative management in older adults with splenic trauma are higher than for younger adults.

Spinal cord injury is more likely to occur in an older adult because of cervical stenosis.

Trauma Center Classifications

Hospitals are appointed a trauma designation from 1 to 5, according to state guidelines, based on available resources and annual admissions. On a national level, hospitals may voluntarily apply for trauma verification via the American College of Surgeons and are assigned a number between 1 and 4 based on stringent criteria. Hospitals may provide both adult and pediatric trauma care and must identify

their classifications in both adult and pediatric care. Following are some of the common criteria for both designation (state level) and verification (national level—voluntary):

- **Level 1**—Regional trauma center with 24-hour in-house specialists, referral resource
- **Level 2**—Able to initiate care with 24-hour coverage of ED staff and surgeons, but tertiary care may need to be referred to a Level 1

Bariatric Considerations

Obesity is a growing epidemic in our country. Most recent statistics suggest that 34.9% of the adult population is obese when defined as having a body mass index (BMI) greater than or equal to 30 (Ogden, Carroll, Kit, & Flegal, 2014).

Overweight patients in motor vehicle crashes may benefit from a “cushion effect” and be protected by their weight from dying during the crash. However, obese patients do not benefit from such an effect and have higher mortality rates than leaner people (Brown & Velmahos, 2006). Following crashes, obese patients have higher incidences of thoracic, pelvic, and lower extremity injuries, but a lower incidence of head injuries than non-obese patients. Obese patients have longer intensive care unit stays, more in hospital complications, and higher mortality rates (Ditillo et al., 2014).

One of the reasons obese patients may have more complications after they survive the initial trauma is

that accurate diagnoses may be delayed. This may occur because obesity makes the physical exam more difficult to perform (e.g., breath and heart sounds are difficult to auscultate because of the adipose tissue), ultrasound is less reliable, and/or they may not be able to fit into the available scanners.

Obese patients present challenges for intubation to the skilled healthcare provider at any time, and the obese patient who has experienced a traumatic injury should be considered a high risk for a difficult airway. Even with the best ventilator management, obese trauma patients usually are intubated for longer periods of time and have more problems following extubation than leaner individuals.

There is an increased incidence of infections of clean surgical wounds in overweight and obese trauma patients. Severely obese patients have a greater likelihood of wound dehiscence.

- **Level 3**—Able to resuscitate and stabilize patients with agreements in place for transfers to Level 1 or 2 facilities. Must have 24-hour ED coverage with prompt availability of surgeons
- **Level 4**—Basic EDs with ability to perform ACLS prior to transfer
- **Level 5**—Facility may not be open 24 hours a day, but after-hours activation protocols are in place

Emergency Medical Services (EMS) are required by law to assess trauma patients and transport them to the nearest hospital. The Emergency Services team at the receiving institution assesses the patient and rapidly determines the appropriate trauma level of care for that patient. If the hospital is unable to provide adequate care, the patient is assessed then prepared for transfer to an appropriate facility that will accept and agrees to manage the patient's injuries (Emergency Nurses Association [ENA], 2007).

Trauma Teams

Research and outcome-related studies favor trauma teams to decrease the mortality and morbidity related to trauma by improving and expediting the trauma process. The trauma team is horizontally organized to care for the trauma patient. Trauma teams have been demonstrated to significantly reduce resuscitation times due to the overlapping of care and allocating of tasks. This type of approach emphasizes a consistent effort in the care of trauma patients. Tasks are assigned prior to patient arrival. The members of the trauma team include the emergency department doctor, a registered nurse (RN) in charge of delegating tasks, an

EMT-P or RN, a critical care aide, an emergency department technician, department secretary, x-ray technician, case manager/social worker, laboratory technician, and a respiratory therapist.

Preparation for the Patient's Arrival

As soon as EMS calls the receiving facility with a report that a trauma patient is en route to that facility, the emergency services department begins preparing to receive the patient. Even though not all hospitals have trauma teams, all hospitals must be prepared to receive and assess a trauma patient. Teamwork among nurses and collaboration with the rest of the emergency services team are essential to successful implementation of the Trauma Assessment Protocol (Jansen et al., 2009).

The following individuals and departments should be notified of the anticipated arrival of a trauma patient:

- Primary and secondary nurses.
- Emergency physician identified as the provider for the patient *prior* to the patient's arrival.
- Surgery and anesthesia if surgical interventions are anticipated.
- Respiratory Therapy for assistance with airway management.
- Laboratory Technician for assistance with lab work.
- Emergency Room Tech for assistance with patient care tasks.
- Radiology Department for stat portable x-ray needs and CT scan.

Safety Initiatives

Improving Prehospital Intubation of Trauma Patients

PURPOSE: To ensure that trauma patients receive the best possible airway management during the golden hour.

RATIONALE: Endotracheal intubation (ET) has been used for airway management by paramedics for some time. Recently, questions have arisen as to the safety and effectiveness of both ET and rapid sequence intubation when performed outside the hospital (Wang & Yealy, 2006). In one study, 25% of ET tubes were misplaced, 66% were placed in the esophagus. More often, misplacement rates vary from 5.8% to 12%. In addition, ET has resulted in deleterious physiologic responses from hyperventilation. Because few studies have confirmed the current practice of out-of-hospital intubation, this initiative reviews whether out-of-hospital intubation is safe and which system level improvements might be endorsed, beginning with the question, "Should we intubate at all?" (Wang & Yealy, 2006).

HIGHLIGHTS OF RECOMMENDATIONS:

Consider increasing the number of successful intubations required for paramedics to graduate (the current standard for paramedics is 5, and for emergency physicians, 35) and limiting ETs to a well-prepared cadre of paramedics.

Consider the use of waveform capnography in the field to ensure correct placement of the ET tube.

Consider the addition of sedatives or neuromuscular blocking agents to the out-of-hospital protocol.

Consider the use of alternative airways such as Combitube and laryngeal mask airway instead of ET.

Source: Wang, H., & Yealy, D. (2006). Out of hospital endotracheal intubation: Where are we? *Annals of Emergency Medicine*, 47(6), 532–541.

- Laboratory department to prepare for transfusion protocols if needed.
- Pharmacy for support in the trauma room with appropriate dosing and administration of medication.
- Intensive Care Unit: an RN may be sent to the Trauma Room to assist with patient care.

The trauma team will prepare the room for patient's arrival by:

- Increasing the temperature in the patient room to 99° Fahrenheit.
- Raising the stretcher height for ease in transfer of the patient.
- Applying a trauma drape to the stretcher.
- Ensuring that the following supplies and equipment are present and ready:
 - Airway management and intubation equipment.
 - Supplies for placement of additional intravenous and intraosseous accesses with the tubings primed with warm fluids and a rapid infuser available.
 - Supplies for setting up and placing chest tubes, NG tubes, and Foley catheters.
 - Dressings for wound management.
 - Vials for laboratory specimens.

Essential for Safety

While preparing for the arrival of the trauma patient, each professional who will be caring for the patient should don personal protective equipment including trauma gowns (designed to prevent the passage of fluids), gloves, and protective face wear to cover mouths and noses and allow for visual assessment. Safety of the nurse and the team is essential in trauma care.

Organizing for Teamwork

While the team is preparing for the patient's arrival, specific consideration should be given to listen to the EMS report, which will provide critical information such as mechanism of injury, interventions, and patient's status. The emergency department team caring for the trauma patient works with the EMS team to ensure a patient-focused flow of care to the trauma room.

One nurse should be assigned to document patient care. This is a critical role. Documentation must be done in a timely manner, especially since trauma patients are often transferred to other centers, surgical care, intensive care, or specialized care, and the healthcare professionals who will be providing care in those sites need accurate and current information. Other tasks and assignments must be agreed on in advance. One trauma team method is to assign an RN to the right side of the stretcher and one to the left side

of the stretcher. The RN on the right side would have certain tasks and assessments to do, as would the RN on the left side. One example of how to assign these tasks and assessments is:

RN assigned to the right:

- Inserts an intravenous catheter and infuses fluid and/or blood through this (if a rapid infuser is used, one RN or paramedic is assigned to watch the rapid infusion and add more fluids as necessary due to the short time frame fluids infuse with this piece of equipment).
- Draws blood samples for laboratory testing.
- Assesses the patient's extremities and trunk for injuries.
- Assists with insertion of a chest tube.

RN assigned to the left:

- Assesses the patient's airway, breathing, circulation (ABCs).
- Applies electrodes for 12-lead ECG.
- Applies the blood pressure cuff, pulse oximeter, and takes a temperature.
- Inserts the Foley catheter, if needed, and after the medical doctor (MD) has performed a digital rectal exam.

Essential for Quality Assurance

The Documentation Challenge. The documentation of care of the trauma patient is very challenging. The documentation has to be accurate in terms of patient assessment and healthcare interventions and the times of both have to be noted precisely. This can be difficult because a trauma patient is being treated by many team members, with multiple interventions being implemented. Hospital Information Systems have templates for trauma documentation; however, in hospitals with fewer trauma patients, the nurses may not be as comfortable with the system. It is essential to ensure that there is *one* person assigned to document, but that additional help is available to obtain accurate times, vital signs, medications (including dosages), and interventions. The documentation challenge is a team effort.

Assessment and Management Upon Hospital Arrival

The nurse working in the emergency setting performs an initial assessment, known as the **primary survey**, of the trauma patient as soon as he arrives. The primary survey, along with the report from the emergency medical response team, helps the nurse to focus on injuries that are life threatening, and anticipate any acute conditions that may arise. The initial assessment identifies in a systematic

manner the extent and severity of the injuries. The nurse and members of the trauma team continue to reevaluate the elements in the primary survey (airway, breathing, circulation, disability, and exposure) throughout the patient's stay in the emergency department. It is important to remember that care of a trauma patient is based on assessment with treatment upon discovery. In other words, the trauma patient is assessed in an orderly fashion, and the treatment of each life-threatening injury is initiated upon discovery of that injury. The team does not address the next step in the trauma assessment process until that specific area is stabilized.

Beginning the Triage Process

Once the trauma patient has arrived, the primary RN and trauma team listen to the report from the EMS professionals as they begin the triage process. EMS professionals will include time of injury, mechanism of injury, vital patient assessment information, and interventions implemented. In addition, prior medical history, information about allergies, and medication lists may be obtained to improve individualized care.

The core steps in management of all trauma patients have two components: the primary and secondary surveys. The first step in the management of all trauma patients is the primary survey. During each assessment step, the appropriate intervention is implemented prior to continuing the assessment.

The primary survey consists of:

- A. Airway and cervical spine immobilization
- B. Breathing
- C. Circulation
- D. Disability
- E. Expose

This format of assessment is ongoing throughout patient care, ensuring that the priorities of assessment and continuous intervention are effective.

The **secondary survey** consists of:

- F. Full set of vital signs
- G. Give comfort measures
- H. History and head to toe assessment
- I. Inspect posterior surfaces

Essential for Quality Assurance

If the trauma patient is in need of resuscitation, this should be occurring concurrently with the initial assessment. Evaluation is an ongoing process, and the trauma patient is constantly assessed and reassessed.

After completion of the primary and secondary surveys, the trauma patient is stabilized then admitted to the hospital or transferred to another facility better equipped to manage the patient's specific traumatic injuries and needs.

Nursing Diagnoses

Patient Following a Traumatic Injury

- Ineffective airway clearance related to tracheobronchial obstruction
- Ineffective breathing pattern related to trauma
- Deficient fluid volume related to active fluid loss due to bleeding
- Acute pain related to trauma
- Risk for infection related to inadequate primary defenses
- Risk for ineffective tissue perfusion: peripheral, renal, gastrointestinal, cardiopulmonary, or cerebral related to hypovolemia, reduced arterial flow, and cerebral edema

Priorities of Care—Primary Assessment Overview

It is critical that the team follows the life-saving sequence of primary and secondary surveys and does not get distracted by obvious injuries.

In the primary survey:

- **A** stands for airway. Is the airway clear and patent? The tongue often obstructs the airway in unconscious patients. The cervical spine should be stabilized by being manually held until a hard cervical collar is placed (♦ Figure 9-1).
- **B** stands for breathing. If the patient is breathing on his own, at what rate, depth, and effectiveness? Ensure adequate bilateral ventilation.



Figure 9-1 Immobilization of the cervical spine at the scene of the accident is essential to prevent further injury to the spinal cord. The combined use of a hard cervical collar, head blocks, and tape best restricts flexion, extension, rotation, and lateral bending of the neck.

(Michal Heron/Pearson Education, Inc.)

- **C** stands for circulation. A systolic blood pressure (SBP) may be assessed when palpating for pulses. A radial pulse is palpable when the SBP is greater than 90, while a carotid or femoral pulse will be present if the SBP is greater than 60. In addition, the nurse should be checking the skin color, monitoring oxygen saturation as well as central and peripheral capillary refill time, and noting any obvious sources of bleeding.
- **D** is for disability and refers to the neurovascular status of the trauma patient. What is the level of consciousness of the patient? What are the size, shape, equality, and reactivity to light of the patient's pupils? Is the patient alert? Does the patient respond to verbal stimuli or painful stimuli? Is the patient unresponsive? The **Glasgow Coma Scale (GCS)** is quickly obtained by noting the best eye response, best verbal response, and best motor response. A total score between 3-15 is obtained. A general rule with the GCS is less than 8, intubate.
- **E** is for expose/environment, which consists of removing the clothing of the patient to check for other injuries while at the same time preventing heat loss with heated blankets, overhead warmers, a warm room, and/or warmed intravenous fluids.

Secondary Assessment: Overview

As the primary survey and reevaluation process are underway, the secondary assessment begins.

In the secondary survey:

- **F** is for a full set of vital signs, and family members of the patient need to be communicated with regarding the patient. The ENA supports family presence during invasive procedures and resuscitative efforts; however, the nursing team should ensure the family member is supported by a nurse or colleague to answer questions and provide a comforting presence.
- **G** refers to giving comfort to the patient and to the family. Comfort comes in the form of physical and emotional comfort. Physical comfort might consist of pain relief both in the form of pharmacological or alternative methods and, if possible, repositioning, distracting, and touching the patient. Research reflects that, although we are excellent at anesthetizing our trauma patients, we underestimate their need for pain control. Emotional comfort may be provided in the form of reassurance both physical and verbal, listening to the patient, and relaying information to the patient's family and friends.
- **H** represents head-to-toe assessment and medical history. The major part of the assessment has been done,

but a head-to-toe assessment is very important to identify any additional injuries. The head-to-toe inspection consists of a systematic inspection beginning with the patient's head and face, moving down the neck to the clavicles, shoulders, chest, abdomen, and flanks, then moving on to the pelvis and perineum and on to the extremities. The head-to-toe assessment comprises a visual as well as manual assessment and auscultation of the appropriate areas.

- **H** also stands for the patient's medical history. A patient's medical history is helpful, especially if the patient is an older adult. It is also important to obtain a list of the medications in the patient's medication regimen and any allergies.
- **I** stands for inspection of posterior surfaces. Most patients will arrive on a long backboard. It is important to log roll the patient and inspect the posterior surfaces, controlling any bleeding and documenting findings.

Trauma Scoring Systems

The **revised trauma score (RTS)** is a physiologic scoring system, with high interrater reliability and demonstrated accuracy in predicting death. It is scored from the first set of data obtained on the patient, and consists of the Glasgow Coma Scale (GCS), systolic blood pressure (SBP), and respiratory rate (RR).

Values for the RTS are 1 to 7.8.

The lower the number assigned to the patient, the higher the mortality rate. Patients who have a score of one or less are unlikely to survive. Patients who score less than 4 should be transported to Level 1–2 trauma centers for specialized care. Patients who have a score greater than 5 are very likely to survive if they receive excellent care at Level 3 and 4 providers (Trauma Care International, n.d.). The use of the trauma score, in conjunction with the patient's condition and mechanism of injury, aid in the determination of the plan of care. This may mean preparing the patient for immediate transfer to a higher level trauma center.

Once the Glasgow Coma Scale, systolic blood pressure, and respiratory rate have been determined, a trauma score can be assigned, and a decision about the level of care the patient needs can be made. If it is decided that the patient should be transferred, the work will focus on stabilizing any obvious injuries, securing any impaled objects, securing the airway, and establishing intravenous access for management of fluids and medications. Transfers may occur via a ground critical care team or via a helicopter to the receiving facility. The RN is required to provide a report about the patient to the RN at the receiving facility, and both names are documented on the appropriate forms.

Detailed Description of the Primary Survey

The trauma patient is assessed and treated accordingly as quickly as possible. The primary survey is implemented and the team moves steadily through each of the steps. What may seem like many, perhaps lengthy, time-consuming steps in reality moves very quickly. While the primary survey is in progress, a variety of interventions may be initiated and monitoring devices attached by the members of the team. However, the goal is to remain focused on a systemic method of assessment and care and not to get distracted by obvious deformities.

Assessment and Management of Airway and Breathing

The number one priority with all patients is the airway. To prevent hypoxemia, the patient requires an unobstructed, protected airway for provision of supplemental O₂ because the quickest killer of the injured is inadequate delivery of oxygenated blood to the brain and organs.

A: Airway

The airway is assessed first; simply asking a patient her name will indicate if she can speak. If the airway is not patent, it is opened while maintaining cervical precautions. Once the mouth is open, the nurse assesses it for loose teeth, foreign objects, swelling, vomitus, or blood. The team also assesses the trauma patient for the following conditions that indicate that the patient's airway may be in jeopardy: apnea, a Glasgow Score less than 8, or injuries near the mouth, face, neck, and thoracic cavity.

If the airway is compromised, or the injury will increase the difficulty of maintaining an open airway, the team may decide to intubate, placing an endotracheal tube in the airway to administer oxygen in a controlled manner. Until the patient is ready to be intubated, oxygen is administered via non-rebreather mask, or manual ventilation via a bag/valve mask may be utilized.

Rapid Sequence Intubation (RSI) is a method of preparing and administering medications in a specific order to prepare the patient for intubation. The professional administering these medications is determined by the scope of practice in each state. The RSI procedure is designed to promote patient comfort and enhance ease of intubation. Medications are administered in the following order: anesthetizing agents (short half-life),

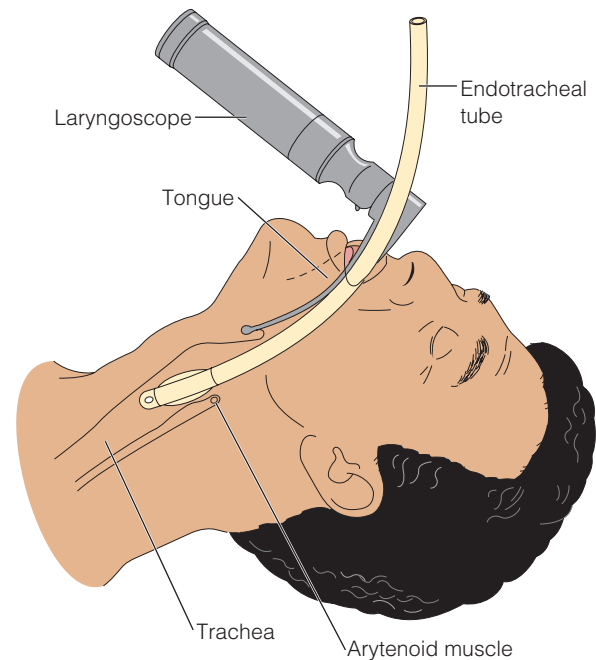


Figure 9-2 Placement of the oral endotracheal tube for intubation. When the ETT is in place, air or oxygen can be blown into the external opening of the tube and enter the trachea.

depolarizing or re-polarizing agents, and long-term anesthetic agents with pain management. The patient's level of consciousness and stability will affect the choice of medications ordered for intubation. RSI is frequently used, and nurses should maintain an excellent working knowledge of the medication administration sequence, pharmacokinetics, and pharmacodynamics of the agents. Placement of an endotracheal tube is shown in ♦ Figure 9-2.

The gold standard for confirming correct placement of the ET tube is the use of a CO₂ detector or an end tidal CO₂ monitor. However, auscultation of lung sounds, observation for bilateral chest rise, and chest x-ray are necessary supplemental measures. After the chest x-ray has been obtained, the ET tube may need to be inserted farther or pulled back to correct placement. To ensure continued patency and placement of the ET tube, the nurse ensures it has been secured after placement and documents its size and location. In addition, the patient may need to be suctioned to clear blood or mucus blocking the tube.

If an endotracheal tube cannot be placed due to facial trauma, placement of a surgical airway such as a cricothyroidotomy or emergent tracheostomy may occur. Once the airway is secured the team may move to the second step of the trauma survey, assessment of breathing. However, the nurse continually reassesses the patency of the airway.

Complex Airway: Assessment and Management of Midfacial Trauma

The patient with maxillofacial trauma is in need of aggressive management and is usually more comfortable sitting up. However, if the patient has a hard cervical collar in place the patient will remain flat until radiological testing confirms a stable cervical spine. Typically, maxillary trauma is associated with multisystem injury and the possibility of a compromised airway. Midface trauma is categorized as follows:

Le Fort I is a transverse fracture that involves a detachment of the entire maxilla above the teeth at the level of the nasal floor.

Le Fort II is a pyramidal fracture involving the triangular segment of the midface and nasal bones.

Le Fort III is a complete disjunction of the cranial attachments from the facial bones. These facial fractures are not always well defined and may occur as partial or complete fractures and in conjunction with one another.

The nursing assessment should take into consideration the mechanism of injury while examining the face and head. Manifestations of a facial fracture may include:

- Epistaxis
- Pain on the injury site
- Facial swelling, facial asymmetry or distortion
- Malocclusion, intra-oral ecchymosis, midface maxillary mobility
- Periorbital edema/ecchymosis
- Cerebrospinal rhinorrhea (LeFort II and III)
- Symptoms of intracranial and/or spinal injury
- Symptoms of multisystem injuries (Frakes & Evans, 2004)

Diagnostic tests would include dedicated maxillofacial CT scans.

Nursing interventions for the patient with maxillofacial trauma include:

- Positioning the patient for optimal airway clearance and constant assessment of airway patency
 - Continual reassessment of the patency of the airway
- Noting the degree of swelling to the face and the amount of blood loss
- Preparing the patient for definitive treatment such as open reduction, internal wiring for stabilization, and administering prophylactic antibiotics
- Administering analgesics as prescribed (Frakes & Evans, 2004)

Complex Airway: Assessment and Management of Neck Trauma

Neck trauma is recognized for the potential for **hemorrhage** and disruption of the larynx or trachea. Neck trauma may also involve spinal cord injury, which will be discussed later in this chapter. A trauma patient with laryngeal trauma typically exhibits:

- Hoarseness in speech.
- Laryngeal fracture palpable along with crepitus.
- Pain with swallowing, cough, or hemoptysis.

The nurse assesses the neck for:

- Ecchymosis.
- Abrasions.
- Loss of thyroid prominence.

The nurse must ensure ABCs. The patient should have a CT scan for diagnostics because soft tissue neck films are not sensitive to laryngeal injury. The nurse should monitor arterial blood gas (ABG) values, assist with cricothyrotomy or tracheotomy when the patient cannot be intubated, and administer high-humidity oxygen. The following may be indications for cricothyrotomy:

- Maxillofacial trauma
- Laryngeal fractures
- Facial or upper airway burns
- Severe oropharyngeal hemorrhage

B: Breathing

Assessment includes observation for trauma as well as auscultation of lung sounds. A rapid portable chest x-ray to confirm tube placement and evaluate the lung status may yield valuable information. Crepitus, diminished or absent lung sounds, and poor oxygen saturation levels are all cues that interventions should be implemented to improve the patient's respiratory status. The key to trauma management is to address the issue interfering with effective breathing prior to continuing the algorithm.

A variety of chest wall injuries may result in ineffective breathing for the patient with a traumatic injury. Thoracic trauma usually affects breathing due to the location of the lungs in the thoracic cavity. The thoracic cavity extends from the top of the sternum to the diaphragm. The structures contained within this cavity include the lungs, heart, esophagus, trachea, thoracic aorta, 12 thoracic vertebrae, inferior and superior venae cavae, vagus nerves, phrenic nerves, and other vascular structures. The diaphragm is included inferiorly in the thoracic cavity.

Chest wall injury is extremely common following blunt trauma. It varies in severity from minor bruising or an isolated rib fracture to severe crush injuries leading to respiratory compromise. Because trauma to the thoracic area is the second leading cause of traumatic death, it is essential that the nurse assess the trauma patient thoroughly for chest wall injuries.

Breathing assessment begins with determining if the patient is breathing spontaneously. If breathing is present, is it effective? The nurse should visually assess for symmetrical chest rise and fall as well as the rate and depth of ventilation. If breathing is ineffective, even in severe chest wall injuries, noninvasive ventilation has been shown to be effective and may be given a trial (Hernandez et al., 2010). However, if breathing is absent or noninvasive ventilation is ineffective, immediate intubation is indicated.

Continuing the assessment, the nurse auscultates lung sounds and notes the depth of respirations, presence of adventitious sounds, and symmetry of sounds. The nurse then assesses the work of breathing: Is the patient using accessory muscles and/or abdominal muscles? What position facilitates the patient's breathing? When the nurse observes the patient's neck, he should identify if the jugular veins are distended and the position of the patient's trachea. All of this information contributes to a thorough breathing assessment. The patient's oxygen saturation and ABGs should be monitored because thoracic trauma often results in hypoxia, hypercarbia, and acidosis.

Ineffective Breathing

It is extremely important to identify when chest injury is present because it has a mortality of up to 60% in trauma patients. Assessment findings that indicate potentially life-threatening problems include:

- The absence of spontaneous ventilation.
- Absent or asymmetric breath sounds (which may indicate either pneumothorax or ET malposition).
- Dyspnea (one of the most common presenting symptoms following thoracic trauma [Legere & Sawatzky, 2010]).
- Hyperresonance or dullness to chest percussion (suggesting tension pneumothorax or hemothorax).
- Gross chest wall instability or defects that compromise ventilation such as a flail chest or sucking chest wound.

In addition, the patient may have:

- Altered mental status due to lack of oxygenated blood to the brain.
- Cyanosis, especially circumoral cyanosis.

These findings should all be treated upon discovery.

NURSING ACTIONS

The goal of care is to restore normal breathing patterns. Interventions are directed toward the specific injury or the underlying cause of the respiratory distress.

Basic nursing interventions for the patient with ineffective breathing are to:

- Provide supplemental O₂: Give 100% O₂ via non-rebreather mask if the patient has a chest wall injury.
- Support the patient and continue assessment if noninvasive ventilation is attempted.
- Prepare the patient for intubation when necessary.
- Evaluate specific respiratory dysfunctions and provide appropriate interventions.

The treatment of patients with chest wall injuries can be extensive and prolonged. Patients with thoracic trauma are more likely to require longer periods of mechanical ventilation and hospitalization than other trauma patients. The initial injury may lead to the development of systemic inflammatory response syndrome, acute lung injury (ALI) or acute respiratory distress syndrome (ARDS), and nosocomial infections. Therefore, in an alert patient, a trial of noninvasive ventilation such as continuous positive airway pressure (CPAP) is tried first in conjunction with provision of adequate pain relief (Simon, Ebert, & Bokhari, 2012). When these methods are insufficient, the patient may be intubated and mechanically ventilated. If mechanical ventilation is necessary, the nurse is vigilant in implementing the ventilator bundle and using other measures to prevent sepsis.

Ineffective Breathing, Rib Fractures: Assessment and Management

Rib fractures may be identified when the nurse examines the patient and palpates the rib area; patients will complain of pain on inspiration and palpation. Bruising or seat belt signs may be visible, and palpation of these areas may reveal the crepitus associated with broken ribs.

Although simple rib fractures may require no specific therapy, the nurse should carefully assess for indications of underlying trauma. Fracture to one or more ribs is a strong indicator for pulmonary contusion below the level of injury. Fractures of the lower ribs may be associated with spleen or liver injuries. Injuries to upper ribs are infrequently associated with injuries to adjacent great vessels. When multiple rib fractures are present, the nurse should assess for a flail chest and/or pulmonary contusions.

Ineffective Breathing: Flail Chest: Assessment and Management

A flail chest occurs when there are at least two fractures of two or more adjoining ribs. This results in a segment of

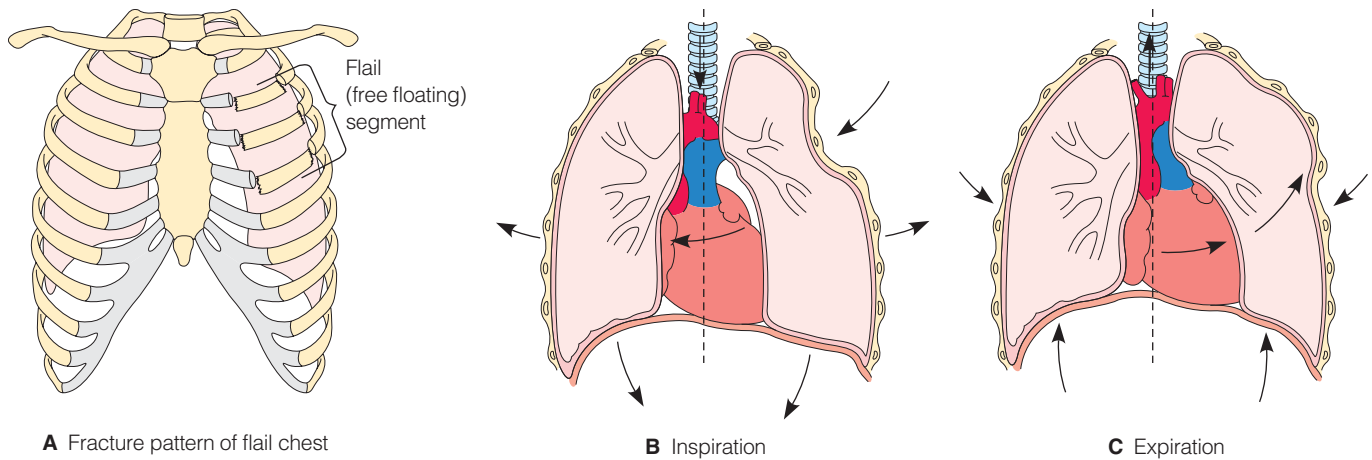


Figure 9-3 Flail chest with paradoxical movement.

the thoracic cage that is not attached to any of the other ribs. The flail segment moves paradoxically to the rest of the chest wall: inward during inspiration and outward during expiration (♦ Figure 9-3). Large flail segments involve a greater proportion of the chest wall and may extend bilaterally or involve the sternum. In these cases, the disruption of pulmonary mechanics may be enough to require mechanical ventilation. Signs and symptoms include:

- Dyspnea.
- Pain in the chest wall.
- Paradoxical movement of a section of the chest wall.

The presence of a flail chest indicates an underlying pulmonary contusion. Nonoperative management is the current standard of care and includes aggressive pain management, frequent pulmonary care, and, if necessary, intubation and mechanical ventilation. Fluid management is also very important in these patients. Once a patient is adequately resuscitated, care should be taken to avoid any unnecessary fluid. Surgical fixation of flail chest may be considered in cases of severe flail chest failing to wean from the ventilator, or when thoracotomy is required for other reasons (Simon et al., 2012). Therefore, the nurse needs to prepare the patient for the possibility of both mechanical ventilation and surgery. The nurse focuses on preventing the development of pneumonia, which is the most common complication resulting from a pulmonary contusion post–chest wall injury (Janus, Vaughan-Sarrazin, Baker, & Smith, 2012).

Pain relief is essential to the prevention of pneumonia in patients with rib fractures. The nurse provides the patient with sufficient pain relief so that he is able to cough and breathe deeply. Education and return demonstration of an incentive spirometer may be a helpful tool for the patient and improve lung capacity. After the emergent period, the recommended analgesia for a

severe chest wall injury is a continuous epidural infusion of a local anesthetic because it provides adequate pain relief without the risks of respiratory depression (Simon, et al., 2012).

Essential for Evidence-Based Practice

Pain relief, chest physical therapy, and early mobilization of the patient with thoracic trauma are essential components in the prevention of pneumonia.

Ineffective Breathing: Pneumothorax: Assessment and Management

A pneumothorax occurs when air enters the pleural space causing loss of negative intrapleural pressure and a total or partial collapse of the lung on the affected side. Open pneumothorax, or a sucking chest wound, refers to a traumatic injury in which the pleural cavity is exposed to the external environment through an open chest wall wound (♦ Figure 9-4).

Nursing assessment of a patient with a pneumothorax may reveal the following (Daley, 2015):.

- Respiratory distress (considered a universal finding) or respiratory arrest
- Tachypnea (or bradypnea as a preterminal event)
- Asymmetric lung expansion: A mediastinal and tracheal shift to the contralateral side can occur with a large tension pneumothorax.
- Distant or absent breath sounds: Unilaterally decreased or absent lung sounds is a common finding, but decreased air entry may be absent even in an advanced state of the disease.

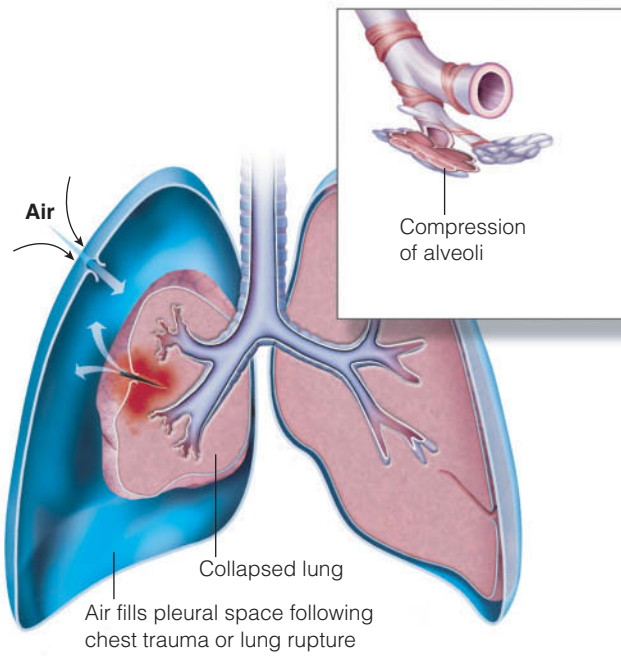


Figure 9-4 Open pneumothorax.

- Lung sounds transmitted from the unaffected hemithorax are minimal with auscultation at the midaxillary line.
- Hyperresonance on percussion: This is a rare finding and may be absent even in an advanced state of the disease.
- Decreased tactile fremitus
- Adventitious lung sounds (crackles, wheeze; an ipsilateral finding)

Immediate treatment depends on the extent of the chest wall injury and the size of the pneumothorax. It may be to administer oxygen via a non-rebreather mask or assist with bag/valve mask ventilations until endotracheal intubation is attempted. ABGs may be obtained along with a chest x-ray to further confirm the presence of a pneumothorax (Lettieri, 2006).

Interventions include:

- Maintaining ABCs.
- Administering supplemental oxygen.
- Placing a dressing that is taped on three sides over an open pneumothorax for temporary management.
- Preparing the patient with either a closed or an open pneumothorax for insertion of a chest tube.
- Continual monitoring of the patient and assessment of the vital signs, pulse oximetry, rhythm, rate, and depth of respirations.
- Administering pain medication as prescribed.

Ineffective Breathing: Tension Pneumothorax: Assessment and Management

Tension pneumothorax is a surgical emergency. An injury, often a lung laceration, allows air to enter the pleural space but not to exit, resulting in progressive accumulation of air in the pleural space. The pressure from the accumulating air may become so great that the patient may have a total lung collapse, and a mediastinal shift may occur (see ♦ Figure 9-5). Because the pressure in the chest is elevated, tension pneumothorax leads to impairment of venous return to the heart and a fall in cardiac output. The classic signs of a tension pneumothorax are:

- Deviation of the trachea away from the side with the tension.
- One hyperexpanded side of the chest that moves little with respiration.
- Absent breath sounds on the affected side.
- Hyperresonance to percussion on the affected side.
- Acute respiratory distress.
- Crepitus.
- An elevated central venous pressure.

The patient is almost always tachycardic and tachypneic and may be hypoxic. These signs are followed by hypotension and circulatory collapse with the possibility of traumatic arrest and pulseless electrical activity (PEA).

Chest tube insertion is the treatment for a tension pneumothorax. The trauma nurse will prepare the chest tube as the physician prepares for insertion. Premedication, if time allows, is focused on pain management and short-term anesthesia during the chest tube insertion. If there is no time to prepare for chest tube insertion, needle decompression is the emergency procedure utilized to reinflate the collapsing lung. When it is not possible for a chest tube

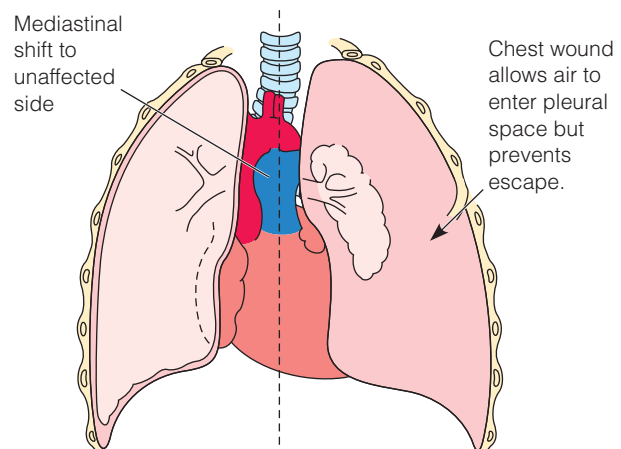


Figure 9-5 Tension pneumothorax.

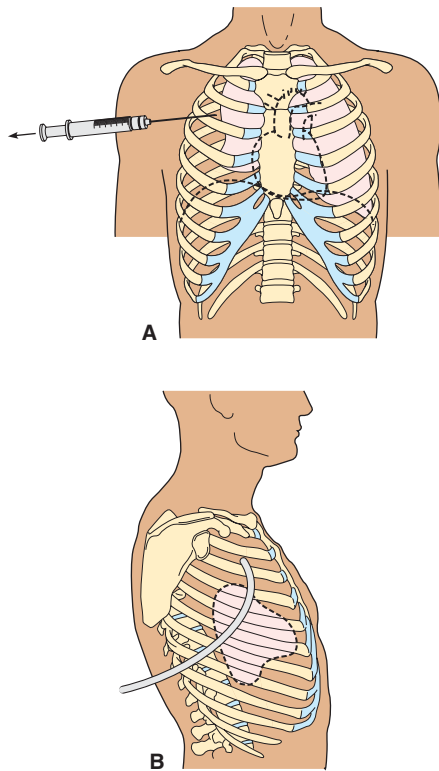


Figure 9-6 A needle thoracostomy may be used in the emergency treatment of a tension pneumothorax. A, A large-gauge needle is introduced, and air and fluid are aspirated. B, Alternatively, a chest tube may be inserted and connected to a chest drainage system.

to be inserted immediately, needle thoracotomy can be a life-saving option (Bascom, 2014) (♦ Figure 9-6).

Ineffective Breathing: Hemothorax—Assessment and Management

A hemothorax differs from a pneumothorax because it is blood rather than air that is filling the pleural space (♦ Figure 9-7), causing the area of lung to shrink and the work of breathing to become increasingly difficult and ineffective. This injury may be caused by blunt or penetrating trauma.

Because most hemothoraces are the result of rib fractures and lung parenchymal or venous injuries, the bleeding is usually self-limiting. The classic signs of a hemothorax are:

- decreased chest expansion,
- dullness to percussion, and
- reduced breath sounds on the affected side.

There is usually not a mediastinal or tracheal deviation unless there is a massive hemothorax. In fact, the signs may be subtle in the supine trauma patient in the ED, and many smaller hemothoraces will be diagnosed only after imaging studies. Not all hemothoraces need chest tube placement.

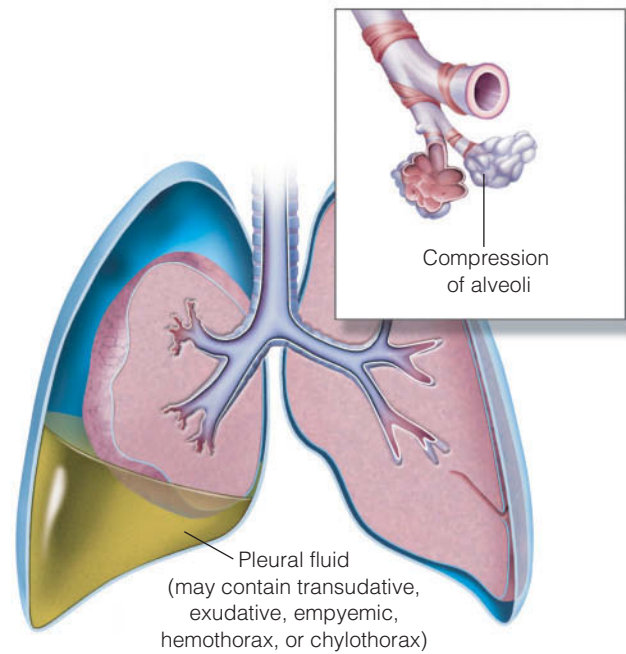


Figure 9-7 Hemothorax.

The determination is made by the team after reviewing radiological findings and patient status. Since the bleeding from the majority of hemothoraces is self-limiting, chest tube drainage is usually all that is required to treat the patient. However, if the initial output is over 1500 ml or the chest tube drains greater than 200 mL/hr for 4 hours, emergent operative intervention may be necessary. Therefore, the nurse must be vigilant in monitoring and reporting the drainage (Karmy-Jones et al., 2014).

Building Technology Skills

Chest tubes are drainage systems that allow air, as in a pneumothorax, or fluid or blood, as in a hemothorax, to drain out of the pleural space. The goal is to remove the air, blood, or fluid from the pleural space, thereby reestablishing subatmospheric intrapleural pressure. The lung will then reexpand.

Technological Requirements

A chest tube is a tube inserted in the chest cavity attached to a closed drainage system that is usually attached to suction. The suction allows material (the air, blood, or fluid) to be removed from the lung without allowing air to be sucked into the pleural space. There are two types of chest tubes: water seal and dry. In a water seal closed drainage system, there are three chambers: a collection chamber, a water seal chamber, and a suction control chamber (♦ Figure 9-8). A dry waterless chest drainage system has a valve in place of the water to allow blood/fluid and/or air to escape while preventing backflow. These chest drainage

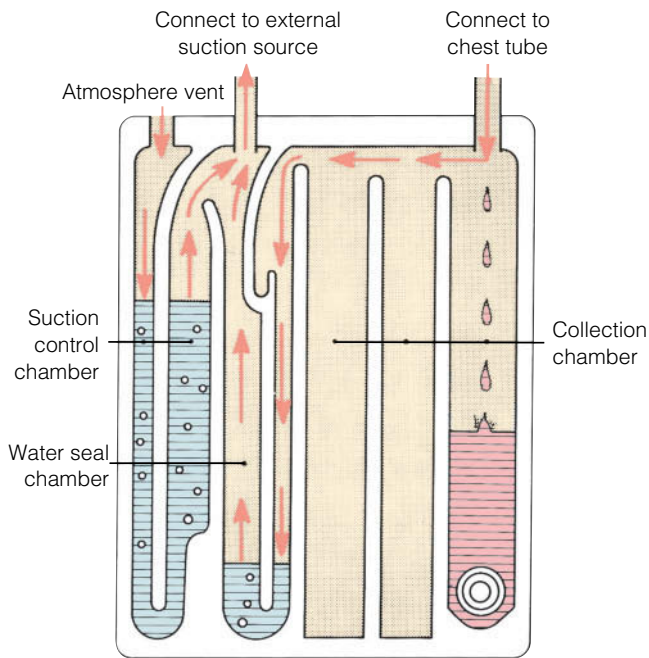


Figure 9-8 A closed-chest drainage system.

systems are less commonly used for trauma patients who are very likely to have both a pneumothorax and a hemothorax depending on the mechanisms of injury. However, a dry chest drainage system may be used for smaller spontaneous pneumothoraces or for transport of a patient. The nurse assists with setting up the system, while the MD will do the actual placement of the tube in the chest.

The insertion site of the chest tube depends on the type of drainage. Chest tubes from which air will be draining are placed high and anterior between the ribs, whereas fluid drainage chest tubes are placed low and posterior. The reason is that air tends to rise while fluid falls to the basal region of the pleural space. The most common complication of chest tubes is malposition. Therefore, chest tube placement should be confirmed with an x-ray.

Nursing Responsibilities

When caring for the patient with a chest tube, the nurse should:

- Secure the chest tube site and dressing. Palpate the site of chest tube insertion for crepitus, document the extent of crepitus, and reassess for changes.
- Maintain the level of the drainage system below the level of the chest and ensure that the tubes are free flowing (unkinked with no dependent loops). Maintain suction at the prescribed level.
- Monitor the amount of drainage at least hourly immediately after insertion—the drainage should decrease over each hour. The nurse should notify the physician if the expected decrease in drainage does not occur.

- Identify the presence of fluctuation in the drainage tubing (sometimes called *tidaling*) and gentle bubbling in the water seal chamber with respiration. When tidaling ceases, it may indicate that the lung has fully expanded or there is a blockage in the chest tube, so a chest x-ray may be indicated.
- Monitor the system for an air leak which is identified by continuous bubbling in the water seal and/or collection chamber. In the event of a leak, the nurse identifies the source (e.g., a cracked chamber, disconnected tubing, nonocclusive dressing at the insertion site) and fixes it (e.g., replaces the chest drainage system).

Assessment and Management of Circulation

The objective of the assessment is to determine if the patient's circulation is effective. If it is effective the team continues on with the systematic assessment.

The circulation assessment includes:

- Observing the patient's central and peripheral color.
- Palpating for carotid, brachial, radial, femoral, popliteal, and pedal pulses.
- The central pulses (carotid and femoral) are palpated first. If the patient exhibits these pulses, then his systolic blood pressure is usually at least 60 to 80 mmHg systolic. If the patient has more peripheral pulses, such as radial and pedal, the blood pressure is usually higher than 80.
- The nurse notes the quality and rate of the pulse, the skin color of the patient, and the temperature and degrees of diaphoresis.
- The patient is inspected for any external bleeding and whether or not it is controlled or needs external pressure.
- A blood pressure may be obtained manually, especially if pulses are weak or not palpable.
- The nurse inspects the neck veins for distention or collapse and auscultates for heart sounds.

NURSING ACTIONS

A lack of pulse should be confirmed then immediately responded to with CPR and resuscitation efforts.

Any bleeding that is not controlled should have pressure applied at the bleeding site. The extremity is elevated, if possible, and pressure is applied to the arterial pressure point medial to the injury.

Two large-bore intravenous catheters (size 14 gauge or 16 gauge) are started with infusions of an isotonic solution.

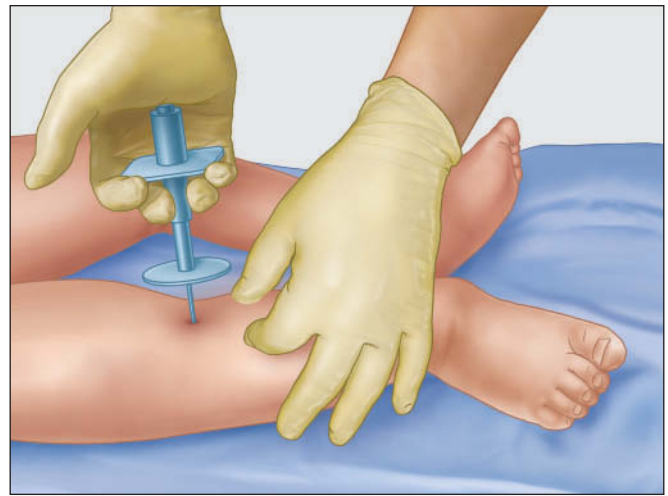
If intravenous access cannot be obtained rapidly, then an intraosseous needle may be inserted. **Intraosseous infusion (IO)** is the process of administering medications and other solutions into a catheter placed directly into the bone marrow (♦ Figure 9-9). An intraosseous needle allows for immediate volume replacement since it can usually be inserted in less than 1 minute (Daly, 2011). The technique is effective with both pediatric and adult patients who need immediate vascular access due to major trauma, cardiac arrest, severe shock, or airway compromise. The needle is injected through the bone's hard cortex and into the soft marrow interior, the stylet is removed, and the IO attached to intravenous tubing. Placement is verified by the rapid flow of fluid into the device without extravasation into the surrounding tissue. Once in place, the IO must be secured, then it can sustain rapid flow rates of up to 125 mL/minute when a pressure bag is used. The nurse must assess for extravasation, which may result in pain, or if large volumes of fluid are allowed to accumulate, compartment syndrome. If there is poor insertion technique or if the line is used for more than 24 hours, osteomyelitis can develop. Therefore, intraosseous needles are removed within 24 hours of placement (Daly, 2011).

Absent Circulation

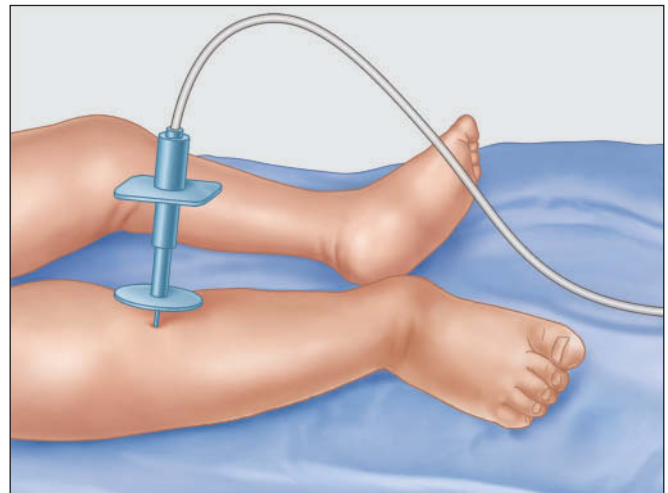
Any patient who becomes unresponsive with no pulse or spontaneous respirations is managed following the 2010 AHA BLS Guidelines of C-A-B (Travers et al., 2010). However, the guidelines are written for all patients. For the trauma patient, securing the airway remains the number one priority prior to implementing further assessment and actions. Advanced cardiac life support, which includes medication and intravenous fluids and/or blood, is initiated, along with cardiopulmonary resuscitation. The trauma patient may need definitive surgical care at this point.

Ineffective Circulation

A trauma patient may exhibit tachycardia or loss of consciousness as symptoms of ineffective circulation. The patient may have uncontrolled external bleeding that needs to be controlled with direct pressure. Other signs and symptoms of ineffective circulation include distended or abnormally flattened jugular veins; pale, cool, diaphoretic skin; and distant heart sounds. The cause of ineffective circulation is often fluid volume deficit. Important causes of ineffective circulation in the trauma patient include hypovolemia (possibly from hemorrhage) and cardiac tamponade.



A



B

Figure 9-9 A, Intraosseous needle being placed. B, Intraosseous needle in place.

COLLABORATIVE CARE

The first priority is to control any uncontrolled bleeding by placing pressure above the site, if possible. At least two large-bore intravenous catheters, preferably 14 to 16 gauge, are inserted, and infusion of lactated Ringer's solution is initiated. Lactated Ringer's solution is preferred for infusion in the trauma patient due to the close resemblance of the electrolyte composition of blood and plasma. Lactated Ringer's is an isotonic solution. The blood or intravenous (IV) fluids may be infused using a rapid infuser, which is a type of IV pump that simultaneously warms the fluid and can infuse a liter of fluid in approximately 2 minutes. A rapid infuser requires 1:1 attention of a nurse. After 2 liters of isotonic fluids have been administered, blood products may be ordered and they may also be infused via a rapid infuser.

While the IVs are being inserted, another team member should be drawing blood for the laboratory including blood for type and crossmatch. The trauma patient may need a blood transfusion; if there is no time to wait for the patient's blood type, type O negative blood, the universal red cell donor, is provided.

Shock

Shock is a clinical disorder evidenced by inadequate tissue and organ perfusion and oxygenation. For the trauma patient, it is imperative that the cause of the shock be identified and rectified. Shock in the trauma patient is most commonly caused by hemorrhage or hypovolemia. Shock does not result from isolated brain injuries. The patient who is cool to the touch and tachycardic is considered to be in shock until proven otherwise. It is important that the nurse recognize the signs and symptoms of shock in order to correct the problem as quickly as possible. Tachycardia is one of the first signs of shock; however, an elderly patient is less likely to exhibit tachycardia when in shock. Because the body may compensate for shock with vasoconstriction, the blood pressure may remain normal until 30% of the blood is lost.

Hemorrhagic Shock

This is the most common form of shock in trauma patients. The trauma patient who is tachycardic and cool to the touch is assumed to be in hypovolemic shock. The normal adult blood volume is 7% of the ideal body weight. Thus, a 70 kg adult would have a blood volume of approximately 5 liters. The healthcare professional is expected to recognize shock and manage the bleeding by assessing the patient's circulation, controlling or stopping the bleeding, and treating the hypovolemia with fluid and/or blood replacement while watching for fluid shifts (Levett, Vercueil, & Grocott, 2006). The fluid shifts may be caused by bleeding into the injury or by third spacing. Third spacing occurs when fluid shifts from the vascular space into an area where it is not readily accessible, as in extracellular fluid. The fluid remains in the body but is unavailable for use. This causes an isotonic fluid volume deficit. Because hemorrhage is the most common cause of shock, it is important to be aware of the different classifications of hemorrhage.

Pathophysiology and Blood Loss

The body responds to fluid volume deficit by progressively constricting the blood vessels in an attempt to preserve the vital organs. The heart rate speeds up in order to sustain cardiac output. The body releases catecholamines to increase the heart rate and the strength of cardiac contraction. Hormonal and cellular factors are released locally and systemically and represent a generalized body response to a scenario of danger or stress (Bonnano, 2012).

Classifications of Hemorrhage

- Class I hemorrhage is characterized by a >15% loss of total blood volume (up to 750 mL in an average-size adult), for which the patient is usually able to compensate.
 - Tachycardia may be the only symptom of a class I hemorrhage, although the patient may have a heart rate less than 100.
 - Blood pressure, pulse pressure, capillary refill, urine output, and mental status are usually all normal but the patient may be slightly anxious.
- Class II hemorrhage is marked by a 15% to 30% loss of blood volume (750 to 1,500 mL).
 - The signs and symptoms are tachycardia accompanied by a decrease in pulse pressure, anxiety on the part of the patient, and a mild decrease in urine output.
 - Systolic blood pressure is usually normal; mean arterial pressure (MAP) has usually dropped 10 to 15 mm Hg.
- Class III hemorrhage is marked by 30% to 40% blood loss (1500 to 2000 mL). It is classified as a moderate hemorrhage, and the patient is usually obviously ill.
 - The patient will exhibit: a heart rate greater than 120, tachypnea, mental status changes, a fall in the systolic blood pressure, and a drop of 20 mmHg in the MAP, which means the body is not compensating for the fluid volume deficit.
- Class IV hemorrhage is considered an emergent state with greater than 40% blood loss (more than 2000 mL).
 - The signs and symptoms are heart rate greater than 140, tachypnea, pronounced mental status changes, low systolic blood pressure with a MAP less than 60, and pale, cold skin with delayed capillary refill.

See Table 9-1 for levels of shock.

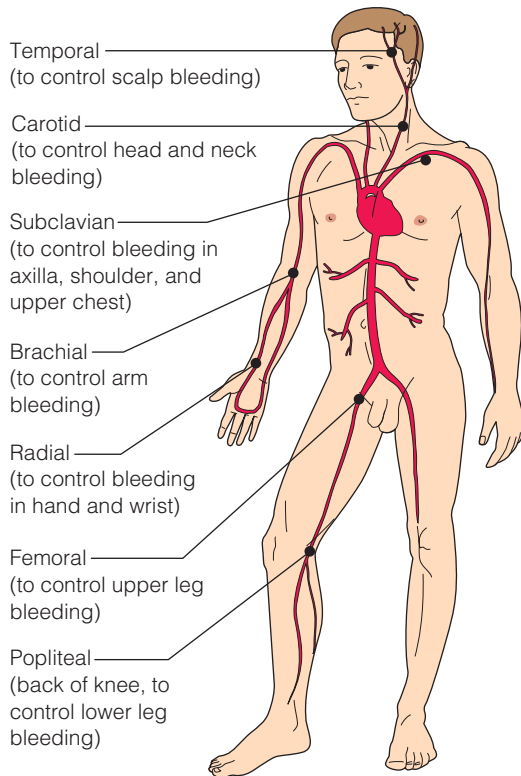
Collaborative Management of the Patient in Shock

At this point in trauma care, the airway should be secure and breathing effective. The patient is most likely intubated and receiving oxygen. The trauma patient may be tachypneic in order to maintain his body's acid-base balance. The nurse will need to carefully monitor the patient's arterial blood gases to determine that the patient is not becoming acidotic, a component of the trauma triad.

If the patient is actively bleeding, the trauma team will attempt to stop the bleeding either with external pressure or surgically. ♦ Figure 9-10 displays points where pressure may be applied in an attempt to control bleeding. In order

Table 9-1 Levels of Shock

Level of Shock	Est. Blood Loss	Patient S/S	Interventions
Class I	<15% up to 750 mL	SR to ST VSS Anxious	Isotonic solutions via IV
Class II	15–30% 750–1500 mL	Tachy, hypotensive	Isotonic solutions via IV
Class III	30–40% 1500–2,000 mL	HR > 120, tachypnea, mental status changes	Isotonic solutions via IV and probably blood products
Class IV	<40% blood loss	HR > 140, Low SBP	Prompt, fluids + blood

**Figure 9-10** The major pressure points used to control bleeding.

to treat hemorrhagic shock, the patient will be requiring fluids and blood replacement therapy. If the blood bank was notified of the trauma patient's impending arrival, blood product preparation should have started with the patient's arrival and products should be available when needed.

Damage Control Resuscitation

The concept of **damage control resuscitation (DCR)** has been widely accepted in the treatment of trauma patients. The principles of DCR have been developed based on lessons learned from damage control surgery and resuscitation of military conflict casualties. The main components of DCR are:

1. Rapid recognition of trauma-induced coagulopathy and shock.

2. Permissive hypotension.
3. Rapid surgical control of bleeding.
4. Prevention/treatment of hypothermia, acidosis, and hypocalcemia.
5. Avoidance of hemodilution by minimizing use of crystalloid intravenous fluid.
6. Transfusion of red blood cells (RBC): plasma:platelets in a high-unit ratio according to massive transfusion protocols.
7. Early and appropriate use of coagulation factor concentrates.
8. Use of fresh RBCs and whole blood when available (Camazine et al., 2015).

Access and Fluids

The standard for a trauma patient is to have two large-bore intravenous cannulas. If this cannot be obtained within 60–120 seconds an intraosseous needle or external jugular vein may be utilized (refer to Figure 9-10). Large-bore catheters are required because small peripheral lines are not capable of delivering the large amounts of fluids needed for the trauma patient. The preferred fluid for resuscitation is lactated Ringer's, which is classified as a crystalloid. Normal saline may be used; however, this fluid may increase the chloride levels of the patient and may lead to acidosis secondary to bicarbonate loss. D₅W is contraindicated because this fluid will dilute sodium and should be avoided in all trauma patients (Levet et al., 2006). Fluids will be administered rapidly using pressure bags or a rapid infuser. Once the patient's volume has been adequately replaced, the physician may consider administering vasopressor drugs, along with more fluids, for hypovolemic shock.

Blood

Patients with large blood losses due to trauma or who do not respond adequately to a crystalloid fluid bolus of 1–2 liters should be considered for an early blood transfusion versus the need for more crystalloid (ATLS Subcommittee, 2013). The decision to transfuse should not only be based on transfusion triggers, but should be coupled with adequate knowledge of the clinical symptoms, rate and

extent of ongoing blood loss, cardiac function, and the need for operative intervention. The end goal of transfusion is to restore volume and oxygen-carrying capacity (Kaur, Basu, Kaur, & Kaur, 2011).

When the nurse is preparing to administer blood to a patient with a traumatic injury:

- The patient should have a large-bore IV catheter in place (16- to 18-gauge).
- IV tubing specifically used for blood is obtained along with the doctor's order, the signed transfusion consent form, and 500 mL of normal saline.
- If time is of the essence, Type O negative packed cells are used because Type O is the universal donor. It is preferable to type, screen, and match the patient's blood. The patient is then given blood that is a match to his own blood type. In exsanguinating trauma patients, the rapid infuser may be used to administer blood products in 1–2 minutes; in addition, intraosseous or central line access may be used to expedite the administration of blood products.
- Before blood or blood products are transfused, two nurses check the blood bank number on the armband of the patient and on the blood, then both sign that this has been done.
- In non-emergent situations, the blood is infused slowly over the first 15 to 30 minutes because this is when a transfusion reaction is most likely to occur.
- The nurse begins the transfusion with a baseline set of vital signs (temperature included) and takes vital signs every 5 minutes for the first 15 minutes along with a patient assessment.
- If any of the reactions that are discussed next occur, the blood infusion is stopped immediately, and normal saline is infused. The nurse notes the reason for the cessation of the transfusion and the patient's vital signs, and the blood is placed in a biohazard bag along with the tubing and returned to the laboratory for further testing. Samples of the patient's blood and urine are obtained and sent to the laboratory. The doctor is alerted to the situation.

Massive Transfusion Protocols (MTPs)

Hemorrhage is the most common cause of death within the first hour of arrival at a trauma center. But the implementation of MTPs has been shown in numerous studies to significantly decrease mortality. In 2013, the American College of Surgeons Trauma Quality Improvement Program published guidelines to address the development of MTP protocols.

MTPs attempt to target and avoid the coagulopathy of trauma by identifying patients likely to require massive

transfusions by administering blood products in a 1:1:1 ratio of red blood cells:fresh frozen plasma:platelets. Specific protocols may differ slightly from institution to institution. But the nurse should be aware of the triggers to institute the protocol and the method for prompt communication and coordination with the blood bank to ensure timely administration of blood products.

Complications of Massive Transfusion

- Thrombocytopenia occurs because platelet function declines rapidly in stored blood. However, 1.5 times the patient's blood volume must be replaced before this becomes a clinical problem.
- Coagulopathy is observed in exsanguinating patients and part of the lethal trauma triad.
- Coagulation factor depletion often develops. It is unclear if this is a response to disseminated intravascular coagulopathy (DIC) or to deficiencies of coagulation factors in stored blood.
- O₂ delivery changes occur because the hemoglobin in stored blood has a high affinity for O₂, which limits O₂ delivery to the tissues.
- Hypocalcemia develops because each unit of blood contains citrate, which binds ionized calcium. With massive transfusions, patients may develop hypocalcemia and exhibit transient tetany and hypotension.
- Hyperkalemia may develop because the potassium concentration of blood increases during storage.
- Acid-base disturbances vary with the metabolism of the patient. Acidosis may develop from lactic acid in the stored blood and is another contributing factor to the lethal triad, so it must be managed quickly. Conversely, alkalosis may result when citrate is metabolized to bicarbonate.
- Hypothermia may develop if cool blood is administered quickly. Hypothermia causes additional metabolic problems because it leads to a reduction in citrate and lactate metabolism (resulting in hypocalcemia and metabolic acidosis); an increase in affinity of hemoglobin for O₂, resulting in a further reduction in O₂ delivery to the tissues; and platelet dysfunction.

Patients with penetrating trauma who are hemodynamically unstable (do not respond to volume replacement) require immediate surgery. They should be taken to the operating room as soon as it is possible. When the wounds may be penetrating multiple cavities (e.g., both the thorax and the abdomen) so the surgeon is unclear about the source of the bleeding, diagnostic peritoneal lavage (DPL) or focused assessment with sonography for trauma (FAST) scan may be used to determine the presence of free intraperitoneal fluid. For the DPL, an aspiration of

frank blood, or lavage fluid with a red cell count greater than 100,000/mL, confirms the presence of intraperitoneal hemorrhage.

Common Types of Transfusion Reactions

The most common types of transfusion reactions and the reasons for their development and their manifestations are:

- **Hemolytic transfusion reaction**—These reactions occur in 1 per 76,000 transfusions when mismatched blood has been transfused into the patient, resulting in destruction of the transfused red blood cells (RBCs) (Silvergleid, 2015). There is a grave risk of kidney damage or failure. Signs and symptoms include fever, chills, dyspnea, tachypnea, lumbar pain, hematuria, oliguria, and tightness in the chest.
- **Febrile reaction**—This reaction is thought to result from the recipient having preformed antibodies to the donor white blood cells (WBCs) or platelets. However, it may also result from the presence of cytokines in stored blood. The signs and symptoms include chills, fever, nausea, and lumbar pain.
- **Allergic**—Antibody reaction to allergens is one of the most common types of transfusion reactions, occurring in 3% to 4% of all transfusions. The patient has an allergic reaction to the blood that is manifested as itching, hives, wheezing, vertigo, bronchospasm, anaphylaxis, dyspnea, and/or generalized edema.
- **Circulatory overload**—This results from infusion of large amounts of blood, especially to patients with cardiac disease or extreme age. The signs and symptoms include pulmonary edema, shortness of breath (SOB), cough, rales, cyanosis, and increased central venous pressure (CVP).

Ineffective Circulation Due to Cardiac Tamponade

Another condition that can result in inadequate circulation following trauma is cardiac tamponade. Cardiac tamponade results from injury to the heart or great vessels with fluid accumulating in the pericardial space, causing compression of the cardiac chambers.

The signs and symptoms of cardiac tamponade in the trauma patient are classically presented as **Beck's triad**. Beck's triad is:

- Hypotension.
- Muffled heart tones (due to the accumulation of fluid around the heart).
- Increase in CVP (seen as jugular vein distension).

An additional sign is a paradoxical pulse. Paradoxical pulse is most clearly seen when the patient has an arterial

line but may also be palpated at any pulse point or auscultated with the blood pressure. In paradoxical pulse, blood pressure (and therefore the force of each heartbeat felt as a pulse) is at least 10 mm Hg higher on expiration than on inspiration.

When cardiac tamponade develops, intrapericardial pressure is elevated and impairs the filling of the heart during diastole (♦ Figure 9-11). This causes pronounced hypotension. The amount of fluid that results in tamponade depends on the rapidity of accumulation of the fluid. In the acutely ill patient, less than 100 mL of fluid in the pericardial space can produce an emergent life-threatening situation. The patient with chronic cardiac tamponade is usually ill but not emergently ill because the pericardium can stretch slowly and accommodate up to a liter of fluid. Chronic cardiac tamponade is generally caused by malignancies.

Treatment of Cardiac Tamponade

Treatment of cardiac tamponade is directed at relieving the pressure and compression by removing the accumulated fluid. This is done by pericardiocentesis (inserting a needle in the pericardial cavity and aspirating the fluid) or by surgically creating a window or opening in the pericardium. Pericardiocentesis is an emergent procedure. The patient is placed with the head and torso elevated at a 45-degree angle. A 16- or 18-gauge, 6-inch or longer, over-the-needle catheter is attached to a 60-mL syringe. Under either ECG or fluoroscopic guidance, the physician inserts a needle attached to an ECG electrode at a 45-degree angle, lateral and to the left side of the xiphoid, 1 to 2 cm inferior to the xiphochondral junction. Blood is aspirated during

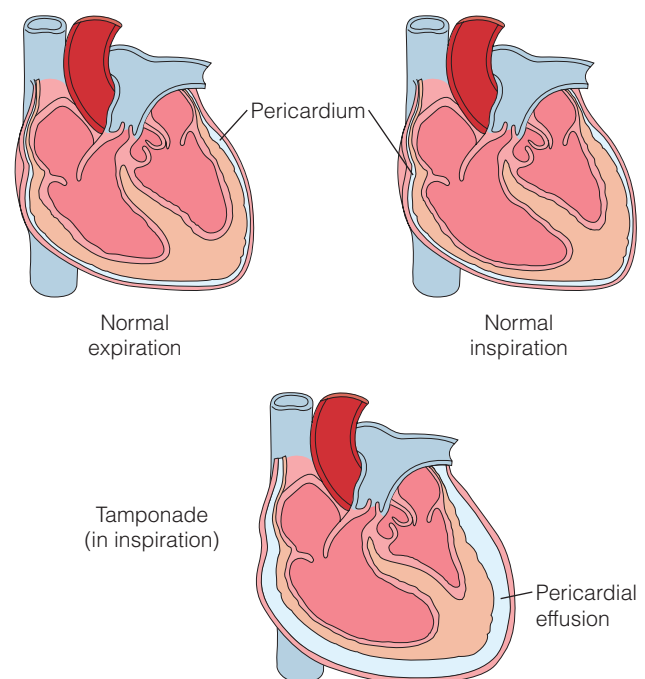


Figure 9-11 Cardiac tamponade.

introduction of the needle until as much nonclotted pericardial blood as possible is withdrawn. If the pericardial blood has clotted, the “open window” is necessary for treatment. Typically, pericardial blood does not clot and the aspiration of this blood will benefit the patient. The nurse observes the patient’s ECG closely during insertion of the needle to identify any ST or T-wave changes that would indicate ischemia from the needle touching the myocardium. Following the procedure, the nurse assesses the patient for redevelopment of symptoms because pericardiocentesis does not treat the underlying problem; it merely removes the accumulated fluid.

Assessment and Management of D: Disability

A brief neurological assessment is necessary to determine level of consciousness. In this assessment, it is important to assess the pupils for size, shape, equality, and reactivity to light. The AVPU scale is a mnemonic used to rapidly determine level of consciousness. The patient is assigned a value based on his response when the nurse speaks to him. He receives a(n):

- A—if he responds to verbal stimuli and is alert,
- V—if he responds to verbal stimuli,
- P—if he does not respond to a verbal stimulus but does respond to painful stimuli,
- U—if he is unresponsive and does not respond to any stimulus.

Once again, it is important to continue to monitor the patient’s ABCs. If the patient is unresponsive or has a decreased level of consciousness, the nurse should conduct a further investigation during the secondary assessment to attempt to find the cause for the decreased level of consciousness. If the patient exhibits active signs and symptoms of brain herniation or one pupil is suddenly dilated, the nurse might consider hyperventilating the patient while awaiting further treatment.

In addition, the nurse rapidly assesses the patient for spontaneous movement of the extremities. If the patient is awake and responsive, the presence of paraplegia or quadriplegia usually indicates spinal cord injury.

Spinal Cord Injuries

Motor vehicle crashes (MVCs) are the number one cause of **spinal cord injury (SCI)**, followed by falls. Most SCIs are produced in association with injury to the vertebral column. The mechanism of injury produces pathological flexion, extension, rotation, and/or compression of the

spine. When the vertebral column is deformed, there may be injury or secondary edema of the spinal cord, resulting in the loss of neurological function. Five percent of patients with spinal cord and/or vertebral column injury will worsen neurologically in the emergency department. Concurrent injuries associated with spinal cord trauma are closed head injuries, long bone fractures, and thoracic and abdominal injuries. The patient’s inability to feel pain makes it more likely that potentially serious injuries in other areas of the body may not be identified.

COLLABORATIVE CARE

The patient with suspected spinal injury is immobilized using a backboard and hard cervical collar until the injury is ruled out. It is imperative to immobilize and stabilize the head and neck to prevent further damage to the neck and spine. A suspected spinal injury is ruled out by a combination of conventional x-rays, computerized tomography (CT) scan, absence of a focal neurological deficit, and the patient’s statement of no midline cervical tenderness (Mraz & Hochstuhli, 2011). If the spinal injury is ruled out, the patient is “cleared” by the appropriate physician and the cervical collar is removed.

If the patient has a cervical spinal cord injury, he will require extra vigilance in the reassessment of his breathing. Some patients, such as those with C4 and above spinal cord injuries, may have required immediate intubation and ventilation. However, patients with lower level cervical injuries, especially those who are older than 40, smokers, and those who have associated chest trauma, may develop difficulty breathing as they tire and may need bilevel positive airway pressure (BiPAP) or intubation and ventilation for support of respiration. These patients will continue to need aggressive nursing care to prevent, identify, and treat atelectasis and pneumonia throughout their hospitalization.

Patients with spinal cord injury above the level of T4 often develop neurogenic shock because the sympathetic innervation to the vasculature is disrupted, resulting in pronounced vasodilation. Neurogenic shock is a distributive shock, resulting in venous pooling, shunting in microcirculation or decrease in venous resistance, and faulty distribution of blood flow. This vasodilation compounds the physiologic effects of hypovolemia. The classic presentation of neurogenic shock is:

- Hypotension with bradycardia.
- No cutaneous vasoconstriction.
- Normal pulse pressure (the difference between the diastolic and systolic pressures; normal pulse pressure is about a 40-point difference).

Treatment of hypotension presumes hypovolemia, and the patient is immediately provided with crystalloid fluids.

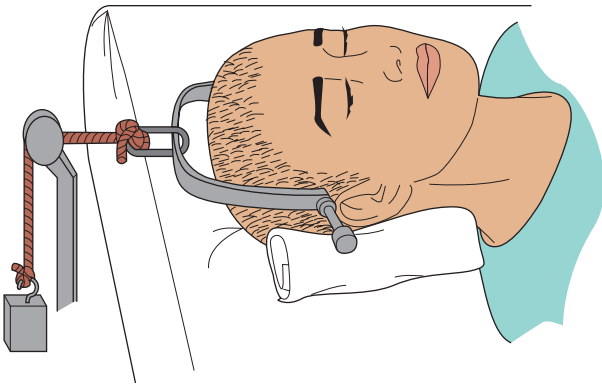


Figure 9-12 Cervical traction may be applied by several methods, including Gardner-Wells tongs.

If volume repletion does not restore an adequate blood pressure, vasopressors might be started, usually norepinephrine. If the patient is tachycardic, the nurse should assess carefully for concurrent injuries such as long bone fractures or abdominal injuries from which the patient may be losing blood. Treatment for the patient with a confirmed spinal cord injury is evolving. If there is a cervical spinal cord injury, skeletal tongs may be applied to maintain alignment of the spine and to relieve muscle spasm pain (♦ Figure 9-12).

Steroid therapy has been very controversial over the last couple of decades, and while occasionally still used in some institutions, as of 2013, the American Association of Neurosurgical Surgeons and Congress of Neurosurgical surgeons now recommend against steroid use in light of lack of evidence and potential for adverse outcomes (Hansebout & Kachur, 2015).

Musculoskeletal Trauma

Fractures account for more than half of all trauma admissions. Musculoskeletal injuries can involve bone, muscles, nerves, soft tissue, and/or blood vessels. Blunt trauma or penetrating injuries may be involved in trauma to the musculoskeletal system. Large amounts of blood may be lost in bone fractures depending on the bone involved. Femur fractures may lose up to 1500 mL of blood, whereas a fracture of the humerus can cause as much as 750 mL of blood to be lost. If the fracture is a closed fracture, this fluid and blood accumulates in the area of the fracture. These are the different types of fractures:

- Open fracture is when skin integrity at or near the site is disrupted.
- Closed fracture is which the skin remains intact.
- Complete fracture is a total interruption of the bone continuity.
- Incomplete fractures are incomplete interruptions in the continuity of the bone.

- Comminuted fractures are a splintering of the bone into fragments.
- Greenstick involves a buckling or bending of the bone.
- Impacted fracture is where the distal and proximal bone sites are wedged into each other.
- Displaced fractures involve the distal and proximal ends of bone fracture sites becoming misaligned.

NURSING ACTIONS

The first intervention with an open musculoskeletal fracture is to stop the bleeding by applying pressure or traction. The nurse assesses CSMs (circulation, sensation, and motor function distal to the injury) and marks on the patient the location of the distal pulse, before and after procedures. X-rays of the pelvis and chest are often obtained as part of a basic trauma work-up. Additional x-rays are obtained based on the patient's signs and symptoms. The nurse checks the joints above and below any fracture for dislocations. The nurse should always assess the suspected fracture for bleeding, deformity, color, movement, tenderness, temperature, sensation, capillary refill, and crepitus. If necessary, the nurse prepares the patient for joint reduction and/or bone realignment. Documentation should reflect the nursing assessment of pulse and CSM prior and post interventions for the fracture.

Compartment Syndrome

Compartment syndrome occurs as blood or fluid enters the fascia, causing an increase in pressure in the fascial compartment. The increased pressure has a tourniquet-like effect, decreasing circulation and resulting in ischemia. Compartment syndrome usually develops within 2 hours following fractures to the lower extremities. The signs and symptoms are:

- Unusually severe pain for the injury associated with motion.
- Decreased motion.
- Swelling and tension, with decreased capillary refill in the tissue.
- Paresthesia.
- Pulselessness—a very late sign and an emergency.

COLLABORATIVE CARE

The treatment is to release the pressure by removing the source of constriction on the extremity or performing a fasciotomy. A fasciotomy is a surgical procedure in which the surgeon makes an incision through the skin and subcutaneous tissues into the fascia of the affected compartment. This allows unrestricted swelling, and circulation is restored. The open wound is packed and dressed daily or negative

pressure wound vacuum therapy is used until secondary healing occurs, then the surgeon may debride the wound and close it or apply a skin graft.

Pelvic Fracture

The nurse can become suspicious of a pelvic fracture from the description of the injury. Common causes of pelvic fractures include motorcycle accidents, pedestrian accidents versus motor vehicle accidents, and falls greater than 12 feet. Pelvic fractures are classified into two categories: stable and unstable. A stable pelvic fracture reveals no deformities. An unstable pelvic fracture involves the pelvic ring being fractured and displaced in two places. Pelvic fractures usually involve large blood loss and can be life threatening. A patient with a 2-cm separation pelvic fracture can lose 1.3 liters of blood, whereas one with a 5-cm separation can lose up to 5 liters of blood (Smith, Scalea, & Ma, 2006). Therefore, the nurse carefully assesses the patient with a pelvic fracture for hypovolemia and shock.

Pelvic fractures may be open or closed, with open fractures having a significantly higher mortality rate. The nurse might stabilize a pelvic fracture with a sheet or pelvic sling to decrease bleeding. Continued unexplained blood loss despite fracture stabilization and aggressive resuscitation mandates angiographic exploration to look for continued arterial bleeding (Graf, 2014). Eventually, the patient with a pelvic fracture may need surgery to stabilize it or may need pins inserted externally to stabilize with traction. However, some pelvic fractures heal with bed rest.

E: Expose the Patient

Expose the patient to assess for any unseen injuries. E also stands for environment; keep the patient covered to prevent heat loss. The trauma patient is prone to hypothermia due to the clothing being removed, blood and body fluids on the skin, and the uncovering of the patient for assessment. Hypothermia must be prevented—or reversed, if present. The temperature of the trauma room should have been increased to 99°F upon notification from EMS of the trauma patient's arrival.

The nurse should consider using a Bair hugger, a device that forces warm air into an inflatable mattress placed over the patient to warm the body and that warms the fluids administered to the patient. The patient should be kept dry and breezes in the room avoided.

Abdominal Trauma

The abdominal cavity may be assessed during the secondary survey either as part of E, exposure, or H, head-to-toe assessment. The abdominal cavity lies between the thorax

and the pelvis. The liver, spleen, kidneys, and pancreas are the solid organs in the abdominal cavity. **Hollow organs** in this cavity include the stomach, small bowel, large bowel, and bladder. Assessment of the patient with abdominal injuries can be particularly difficult because the signs and symptoms of abdominal trauma can be difficult to distinguish, the abdominal cavity can hold large amounts of fluid before becoming distended, and abdominal assessment of the trauma patient is often hampered by alcohol intoxication, illicit drug use, brain injury, and/or other distracting injuries. However, abdominal assessment is essential because unrecognized abdominal injury can cause preventable death. Therefore, the nurse carefully assesses for blood loss, absent bowel sounds, abdominal tenderness, and specific patterns of pain that are linked to the various abdominal injuries.

Conservative, nonoperative management is becoming the norm for patients with grades I to IV injuries when the patient is initially stable following abdominal trauma. If such an approach is taken, the nurse is responsible for the ongoing assessment of the patient to detect any complications. This includes:

- Hourly assessment of vital signs including CVP with attention to any indication of ongoing bleeding.
- Assessment for the development of peritonitis every 1 to 4 hours depending on the status of the patient. Peritonitis may be identified by abdominal pain and tenderness with guarding, abdominal rigidity, tachycardia, fever, or decreased or absent bowel sounds.
- Ongoing assessment of specific abdominal organ functioning.

If the patient develops signs of hemodynamic instability or peritonitis during this period of observation, the surgeon should be notified and a laparotomy may be scheduled.

Traumatic Injury of the Liver

The liver is often injured in abdominal trauma due to its size and location in the abdomen. Liver injury is often manifested by:

- Upper right quadrant pain.
- Rebound tenderness.
- Hypoactive or absent bowel sounds.
- Abdominal wall muscle rigidity.
- Hemorrhage.
- Cullen's sign (bluish discoloration of the periumbilical skin caused by intraperitoneal hemorrhage).
- Grey Turner's sign (blue discoloration of the skin around the flanks or umbilicus in a patient with hemorrhagic pancreatitis).

COLLABORATIVE CARE

To identify the extent of bleeding and the likelihood of liver involvement, a hemoglobin and hematocrit, liver enzyme studies, and coagulation studies are required. Radiologically, a CT scan is ordered. Liver injuries are identifiable by CT scan and are graded from I to VI. A grade I injury is a nonexpanding subcapsular hematoma often resulting from a laceration of the liver. The surface area of the liver involved becomes greater as the grade gets higher, with a grade IV resulting in disruption involving 25% to 75% of the liver parenchyma or an entire hepatic lobe. Grade VI, the highest grade, represents hepatic avulsion.

The majority of patients who sustain liver trauma are currently treated conservatively because almost 90% of hepatic injuries have stopped bleeding by the time the patient reaches surgery. In addition, blood transfusion requirements and intra-abdominal complications are significantly lower in patients who do not receive surgery. The nurse is responsible for the ongoing monitoring of the patient. It is essential that the nurse carefully assess the patient for any worsening of the initial symptoms and indications of ongoing bleeding and alert the physician because these may indicate the need for surgical intervention.

Due to the vascularity of the liver and its ability to store blood, some injuries to the liver can result in hemorrhage and hemorrhagic shock. If a patient is hemodynamically stable, ongoing bleeding may be managed angiographically. However, an unstable patient requires surgical intervention. Surgical intervention is no longer dictated by grading of the injury but rather stability of the patient. Nonoperative management is the standard of care in the hemodynamically stable patient (Stassen et al., 2012). The postsurgical patient is closely monitored for the manifestations of bleeding and hypotension that include rapid, thready pulse and increase in abdominal girth along with diaphoresis.

Splenic Injury

The spleen is capable of storing 100 to 300 mL of blood, roughly 4% of total blood volume. An enlarged spleen increases the blood-storing capacity. Therefore, damage to the spleen can result in hemorrhage.

Signs and symptoms of splenic injury include:

- Signs of hemorrhage and/or hypovolemic shock.
- Kehr's sign, which is pain referred to the left shoulder.
- Upper left quadrant tenderness.
- Muscle wall rigidity in the abdomen.

COLLABORATIVE CARE

The current management of splenic injury also favors non-operative management. The nurse must observe the patient carefully during the first hours and days post trauma. If the patient has stable hemodynamic signs, stable hemoglobin levels, and requires 2 units or fewer of blood, she usually does not require surgery.

However, some patients continue to bleed and require surgical intervention. Efforts are usually made to preserve the spleen so it may be wrapped in a procedure called splenorrhaphy, or gel foam may be used for angio-embolization. Following these procedures, the nurse observes the patient carefully for any sign of re-bleeding. Surgical removal of the spleen is a lifesaving last resort. The patient usually recovers over a period of about 5 days, and a nasogastric tube is usually required for the first 2 days. One of the most important considerations for the patient whose spleen has been resected is that she is at risk for infections by encapsulated organisms such as pneumococcus. Therefore, she must receive at least a vaccination against pneumococcus, but possibly vaccinations against haemophilus and meningococcus as well. Immunizations may be administered anywhere from 24 hours to 2 weeks after injury.

Trauma to the Kidneys

Renal trauma is present in approximately 10% of all abdominal injuries, and 90% of renal trauma is due to blunt injury. However, major renal trauma is more often associated with penetrating trauma than with blunt trauma. When a patient has major blunt abdominal trauma or penetrating flank and back wounds, the nurse should suspect renal injury.

Contusion to the kidneys is the most common blunt trauma kidney injury. The nurse assesses such patients for ecchymosis over the flank, abdominal or flank tenderness on palpation, and gross hematuria. However, hematuria may be microscopic so the nurse reviews the hemoglobin and hematocrit for a drop in values as well as the urinalysis for evidence of blood in the urine. Current practice with blunt kidney trauma is the ongoing careful assessment described previously in conjunction with bed rest because most renal contusions resolve with time.

Penetrating renal trauma requires a CT scan of the abdomen to visualize injury, free air, and/or the path of penetrating objects. It can result in extrarenal and intrarenal bleeding along with urine extravasation causing hypovolemic shock, pain, and inflammation. If the renal blood vessels are damaged by the traumatic injury, surgical repair or angiographic embolization may be required to reestablish blood flow or stop bleeding.

Long-term complications of kidney trauma may include:

- Renal calculi.
- Abscess.
- Renal insufficiency.
- Chronic pyelonephrosis.

Bladder and Urethral Injury

Usually caused by blunt force trauma, the bladder may rupture in association with pelvic fracture. The patient may exhibit suprapubic pain, either gross or microscopic hematuria, urge to urinate but inability to do so, rebound tenderness, muscle rigidity of the abdominal wall, blood at the urethral meatus, blood in the scrotum, or displacement of the prostate gland. Male patients presenting with straddle injuries such as falling on the crossbar of a bicycle are highly suspected for urethral injuries. A Foley catheter is contraindicated for the suspected bladder or urethral injury.

Hollow Organ Injuries

Injuries to the hollow organs of the abdomen may be blunt injuries or penetrating injuries. The small bowel is the most frequently injured hollow organ. The patient injured in an MVC may have a rupture of the small bowel due to the compression caused by the lap belt. Evisceration of the small bowel or stomach, rebound tenderness of the abdominal wall, abdominal wall muscle rigidity, and pain are all manifestations of hollow organ injuries.

Detailed Description of the Secondary Assessment

The secondary assessment continues with the alphabet mnemonic F through I.

F: Full Set of Vital Signs

F represents ascertaining a record of a full set of vital signs including temperature (and if there is chest trauma, the blood pressure should be taken in both arms). Reicks and colleagues (2010) found that there was poor documentation of the temperatures of trauma patients. When nurses are unaware of their patients' temperatures, they are less likely to institute and document the rewarming methods necessary to prevent hypothermia.

Essential for Evidence-Based Practice

Utilization of a standardized trauma form with clearly visible spaces for the patients' temperatures and the use of rewarming techniques results in a reduction in patients experiencing hypothermia (Reicks, Thorson, Irwin, & Byrnes, 2010).

Family reassurance and presence also falls under the letter F. Despite the fact that most studies have found family presence to be beneficial to all involved, family presence during trauma care and resuscitation is an ongoing issue. Ethically, many practitioners feel that the family has a right to be at the side of a loved one during resuscitation. The other side of the argument is that it is too distracting to the trauma team and too graphic for the family to witness the resuscitative efforts.

No family plans for a traumatic event, and in a very short amount of time a large number of family and friends arrive at the emergency department. Florence Nightingale wrote about the impact of the environment on health. The environment of a trauma room is harsh at best. The room is hot, crowded, loud, and full of a lot of equipment, and body fluids may be on the floor mixed with discarded packaging from equipment needed STAT. The patient is likely partially covered. Although research and most facilities now fully support family presence during resuscitation efforts, there are families that do not want to see the patient until they are ready to transfer to ICU/Surgery/another facility or have died. Nursing, especially at the time of a traumatic injury and death, can play such an incredible role by simply being aware of the environment and being present for the family. In addition to contacting any spiritual adviser per family or patient request, nursing actions may include:

- Removing body fluids from the environment or if there is not enough time, covering them up.
- Making sure the patient's hand is out and secured so it does not drop suddenly and scare the family. Also, placing warm fluids near the lower arm to keep the patient warm is helpful.
- Ensuring the stretcher is low, with chairs around, and tissues and ice water.
- Being present—to answer questions, provide support, or simply to be there.
- Turning on one procedure light, facing up, turn off all the other lights, it instantly dims and calms the room. (Patients with head injuries and migraines appreciate this too.)
- Taking one's own pulse and breath—deeply caring for others requires self-care.

G: Stands for Give Comfort Measures

When a trauma patient arrives in the ED, analgesics are usually delayed until the patient has been assessed (pain management falls under G in the ABCs). Once the patient has been assessed and is stabilized, many institutions recommend the use of IV fentanyl in incremental doses to manage the pain. As pain medication is provided, the

nurse must closely assess the patient's RR and pattern as well as the patient's blood pressure. However, if the patient is unstable, pain management must be provided very carefully and often is delayed until the patient has stabilized.

Morse and Proctor (1998) have extensively researched how trauma nurses comfort patients who arrive in the ED at least partially conscious and aware that they are seriously ill. Although the nurses would indicate that they were too busy to comfort the patient, Morse and Proctor's research indicated that trauma nurses displayed distinctive patterns of touch, eye contact, and talking with their patients to encourage them to "hold on" through painful events. Patients who recalled their injury stated that these caring behaviors served as their lifeline.

In trauma situations, Morse and Proctor have found that nurses can effectively comfort their patients in three ways:

1. By retaining contact with the patient, usually a firm palmar touch that reassures the patient that someone is there
2. By assuming an "en face" position—looking directly into the patient's eyes and demanding the patient's attention
3. By using comfort talk—short rhythmic phrases that are addressed clearly to the patient; some are clearly comforting (such as "We're almost done") whereas others are clearly directive (such as "Lie still and we'll be done soon")

Reflect On

Consider how you relate to patients and families in crisis. Do you engage in the behaviors identified by Morse and Proctor? Which of them have you found to be useful? Are there specific other actions that you utilize in such situations?

H: History and Head to Toe Assessment

The healthcare team must obtain information about the nature of the traumatic injury as well as the patient's health history. In order to provide appropriate care, the nurse might consider the following questions: What is the mechanism of injury? What are the injuries, general condition of the patient, and the level of consciousness? What were the vital signs prior to arrival at the accepting facility? What initial treatments did the patient receive and how did the patient respond? What is the patient's past medical history?

Essential for Evidence-Based Practice

For patients with severe traumatic injury, the nurse should perform a tertiary survey after 48 hours to reassess the patient for additional injuries and verify the past medical history. During the first 48 hours, communication, especially of pain and past medical history, may have been impaired (Reicks et al., 2010).

Along with the history, a head-to-toe assessment comes under the letter H. Briefly, the head-to-toe assessment of the trauma patient includes general appearance and inspection of the head and face, neck, and chest. Systematically, the nurse moves down the body to inspect the abdomen and flanks and then the pelvis and perineum. The extremities are assessed, and at the completion of the head-to-toe assessment, the patient is log rolled for an inspection of posterior surfaces. If the patient is able, she will be asked to identify areas of pain along the spine and any paresthesias she is experiencing.

Trauma Summary

Trauma care is an ever-evolving area of nursing care. The number of traumas has increased due to the increase in population and the increase in the age of active adults. The number of patients surviving trauma is slowly increasing due to the research and development of standards of care. Predictors of long-term outcomes and survival of trauma are based on revised trauma scores. There are those who defy the odds and survive under the bleakest prognosis. This chapter provides an overview of current standards of trauma care practice. The care of a trauma patient is

focused, methodical, and with one single purpose, determining what are the life-threatening injuries, establishing if can they be managed at the current facility, and if not, providing for the rapid transition of the patient to the appropriate facility. However, trauma nurses must not only care for the physical health of the patient but must also maintain a therapeutic relationship with the patient and family. The work nurses do to comfort patients and families provides the emotional support they need to process the unexpected and unthinkable.

Why/Why Not?

A patient who was shot twice in his abdomen has just arrived in the ED. His BP in the ambulance was 90/48, HR 122, respiratory rate 26. He can say his name and his chest movement is symmetrical. However, he has no feeling or movement below his waist and his abdomen is rigid. He has two large bore IVs running, and a nurse is preparing the rapid infuser to begin administration of a liter of Ringer's

Lactate. The trauma surgeon arrives and tells the nurse not to begin the bolus infusion, but rather to run the Ringer's Lactate at 150 mL/hour and to prepare the patient immediately for scanning and the OR. Should the nurse infuse the Ringer's Lactate at the lower rate? Why or why not?

See answers to Why/Why Not? in the Answer Section.

Case Study

James Quinn, 22 years old, was found outside his car lying on the ground against a tree. EMS reports he was not wearing a seat belt, and there was no airbag deployment as it was an old car. EMS reports he is alert, disoriented, and able to maintain his airway. He is boarded and collared, with a large-bore IV in his left AC (antecubital space). His oxygen saturation is 99% with a nonrebreather mask in place. Vital signs include sinus tachycardia at 122, and blood pressure is 130/palp. James's right lower leg is deformed but there is no blood noted. He does smell of ETOH. Estimated time to arrival is 3 minutes. Upon arrival in the ED, James's airway is patent and he can speak his name. However, he has paradoxical movements of his left chest wall and a visibly deformed left leg. His breath smells of alcohol, and he is becoming increasingly confused and belligerent.

1. What are the priorities in his care?
2. What are the indications of a flail chest? Why is the pulmonary contusion of significance?
3. What are the possibilities for managing a flail chest? What would be the determining factor in how this will be managed?
4. James's blood pressure is 90/50. What injuries could be contributing to this? How should the healthcare team proceed?
5. What factors could be contributing to James's confusion?
6. What should be done about his visibly misshapen leg?
7. What factors might complicate his abdominal assessment? How should he be assessed?

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 9.1 A 19-year-old female presents to your ED having fallen off a trampoline; she is unable to move from the nipple line down. What should the nurse verify while assessing the airway?
- 9.2 A trauma patient arrives to your facility. As you assign tasks to the nursing staff helping you, a nurse begins to insert a Foley catheter in this patient. What assessment needs to be completed before the Foley catheter can be inserted?
- 9.3 What does the primary assessment of a trauma patient consist of? The secondary assessment?

- 9.4 The nurse on duty in the ICU accepts a trauma patient from the ED. The unweighted initial revised trauma score for this patient is 12; however, once the patient arrives in the ICU, the nurse assesses the patient and finds the revised trauma score to be 10. What factors might contribute to the trauma score dropping?
- 9.5 Name two indications for chest tube insertion.

See answers to Chapter Review Questions in the Answer Section.

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Chapter 10

Care of the Patient Experiencing an Intracranial Dysfunction

Kathleen Perrin, PhD, RN, CCRN

Abbreviations

ADH	Antidiuretic Hormone	GCS	Glasgow Coma Scale
ARDS	Adult Respiratory Distress Syndrome	ICP	Intracranial Pressure
CCTHR	Canadian CT Head Rule	IICP	Increased Intracranial Pressure
CDC	Centers for Disease Control and Prevention	MAP	Mean Arterial Pressure
CNS	Central Nervous System	MBI	Mild Brain Injuries
CPP	Cerebral Perfusion Pressure	PEEP	Positive End Expiratory Pressure
CSF	Cerebrospinal Fluid	SIADH	Syndrome of Inappropriate Antidiuretic Hormone
DI	Diabetes Insipidus	TBI	Traumatic Brain Injury
DIC	Disseminated Intravascular Coagulation		
DVT	Deep Vein Thrombosis		



Learning Outcomes

Upon completion of this chapter, the learner will be able to:

1. Describe normal intracranial pressure and explain causes of increased intracranial pressure.
2. Interpret focused assessment findings and intracranial pressure readings in patients with increased intracranial pressure.
3. Examine collaborative management and nursing responsibilities for patients with traumatic brain injuries.
4. Explain how meningitis and seizures can increase intracranial pressure and describe measures to limit brain injury.

Introduction

Nursing care of the patient with an intracranial dysfunction requires the nurse to have an understanding of anatomy and physiology and excellent neurological

assessment skills. The nurse also needs to recognize how changes in the volumes and pressures in the skull and primary and secondary brain injuries will impact patient outcomes.

Intracranial Pressure

In order to understand cerebral disorders, it is important to have an understanding of the anatomy and physiology of the brain. In this chapter, the metabolic requirements of the brain and Monro-Kellie hypothesis are reviewed.

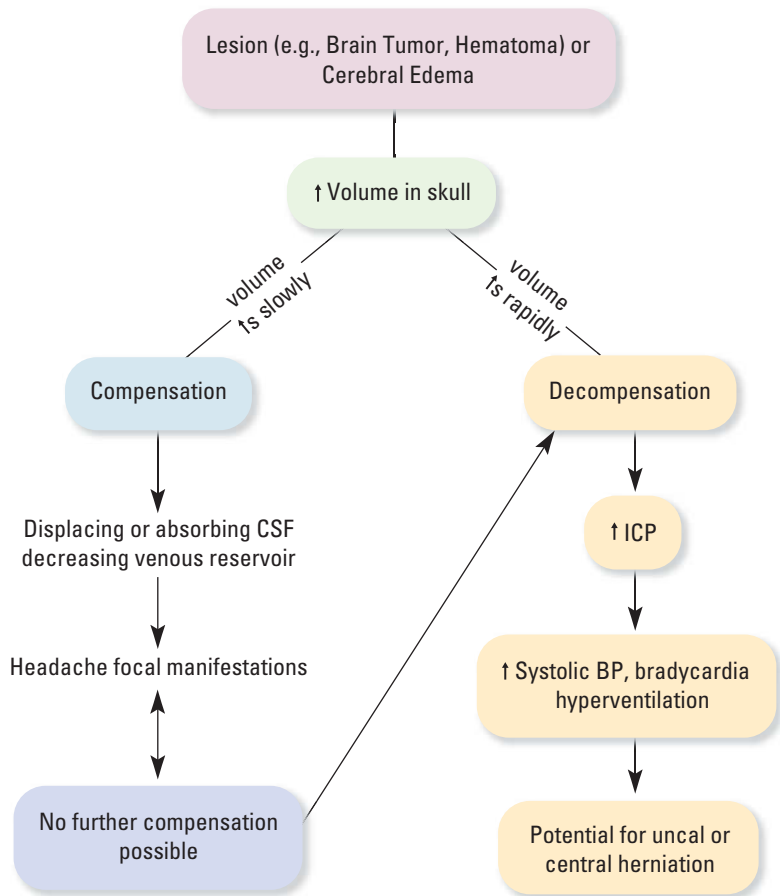
Metabolic Activity of the Brain

The brain is one of the most metabolically active organs in the body. It uses energy to maintain normal neuronal functioning and for biosynthesis. The brain requires oxygen and glucose, which it is unable to store, for energy metabolism. Therefore, the brain is dependent on a continuous blood flow to meet its energy requirements. Although brain tissue represents only 2% to 3% of body weight, it requires 20% of the cardiac output and 25% of total body glucose.

The brain depends on consistent blood flow to supply oxygen to its cells. Decreased and/or absent cerebral circulation causes neuronal cellular injury or death. The internal carotid arteries and the vertebral arteries supply blood to the brain. Cerebral blood flow drains through two sets of

veins into the venous plexus and dural sinuses, then into the internal jugular veins. The flow of venous blood is circulated by gravity because the veins do not have valves. Pressure differences between the extracranial veins and the venous sinuses may also influence the direction of flow. Autoregulation of the deep cerebral vessels manages the arterial blood flow, ensuring that the metabolic needs of the brain are met.

Autoregulation is the ability of the brain to maintain a constant perfusion despite wide variations in blood pressures. When the systemic blood pressure falls, cerebral vessels dilate, decreasing resistance and increasing cerebral blood flow. When systemic blood pressure is too high, cerebral vessels constrict and maintain normal cerebral blood flow. When systemic blood pressure is less than 50 mmHg or more than 160 mmHg, and when cerebral perfusion drops below a minimum level, autoregulation is not effective. Autoregulation also ensures that cerebral blood vessels dilate in response to a perceived increase in requirements for cerebral blood flow, such as when there is an increase in cerebral metabolism, a drop in cerebral oxygen levels, or an increase in cerebral carbon dioxide levels.



Visual Map 10-1 Intracranial Dysfunction Overview

Monro-Kellie Hypothesis

A rigid skull surrounds the brain, providing protection. However, it leaves no room for expansion to occur in any of the volumes in the skull without an increase in the intracranial pressure or a decrease in one of the other volumes. This is known as the **Monro-Kellie hypothesis**.

$$\text{Intracranial Volume (VIC)} = \text{Volume brain} + \text{Volume blood} + \text{Volume CSF} + \text{Volume lesion}$$

It states that changes in the volumes that are normally in the skull, the brain, the blood, and the cerebrospinal fluid (CSF) as well as the presence of any abnormal lesion such as a brain tumor, an aneurysm, or a hematoma have an effect on the intracranial pressure. Normally, the intracranial pressure is kept in a range between 5 and 15 mmHg by a mechanism known as compliance. When the volume of one of the components increases, the body may respond by:

- Displacing CSF into the lumbar cistern.
- Reabsorbing more CSF.
- Compressing veins and shunting blood out of the venous sinuses (Josephson, 2004).

These compensatory mechanisms allow for small increases in the volumes of components in the skull to occur without a significant increase in intracranial pressure. However, the mechanisms can only accommodate a certain amount of extra volume; when that is exceeded, intracranial pressure can rise rapidly. ♦ Figure 10-1 displays an example of a volume-pressure curve showing how the intracranial pressure remains within the normal range as long as compliance is normal, then increases rapidly once the compensatory mechanisms have been exhausted. When intracranial pressure rises, cerebral perfusion can be affected.

Cerebral Perfusion Pressure (CPP)

Cerebral perfusion is dependent on the blood pressure and the intracranial pressure (ICP). It is the difference between the pressure of the incoming blood (best measured as the mean arterial pressure [MAP]) and the force opposing perfusion of the brain, the intracranial pressure (ICP). The formula is:

$$\text{CPP} = \text{MAP} - \text{ICP}$$

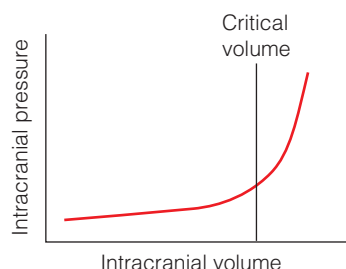


Figure 10-1 A volume-pressure curve.

Normally, values for CPP should be 60 to 65. A CPP less than 50 usually results in the loss of autoregulation and leads to hypoxia of cerebral tissue. From the formula, it can be seen that factors that decrease the MAP or increase the ICP can result in a reduction in perfusion of the brain.

Increased Intracranial Pressure

Increased intracranial pressure (IICP) is a higher than normal level of pressure within the cranial cavity. Because it may result in a decrease in CPP, prolonged or severe increases can result in tissue ischemia and neural tissue damage. As noted previously, normal ICP ranges between 5 and 15 mmHg.

Primary Causes of Increased Intracranial Pressure

Primary causes of IICP are underlying brain disorders. They may be associated with slow processes (such as a slow-growing brain tumor) where the brain initially compensates for the growth of the tumor and the ICP rises slowly. Or they may be sudden devastating increases in the volume within the cranial cavity, such as following trauma or a cerebral hemorrhage, where compensatory mechanisms are inadequate and ICP rises precipitously. The following is a list of common brain disorders that result in increases in ICP:

- Brain tumor or other space-occupying lesion
- Trauma
- Nontraumatic cerebral hemorrhage
- Ischemic stroke
- Hydrocephalus
- Postoperative cerebral edema
- Meningitis

Secondary Causes of Increased Intracranial Pressure

The secondary causes are extracranial or systemic processes that contribute to increases in ICP. If these conditions are allowed to exist, they often contribute to secondary injury, producing ongoing increases in ICP and additional damage to the patient who has sustained a brain injury. However, they are often remediable. How to manage them is discussed in detail throughout this chapter. These secondary causes include:

- Airway obstruction
- Hypoxia or hypercarbia from hypoventilation
- Hypertension or hypotension
- Position
- Hyperthermia
- Seizures
- Metabolic disorders including hyponatremia

Assessment of a Patient with a Potential for Increased Intracranial Pressure

One of the most important aspects of caring for a patient with a neurological disorder is ongoing and accurate neurological assessment. An accurate initial assessment establishes the patient’s baseline neurological status, whereas ongoing assessments are essential to determining how the patient is progressing. Critical care nurses often perform ongoing assessments on an hourly basis. Components of an hourly neurological assessment usually include at least the Glasgow Coma Scale (or another assessment of level of consciousness), pupillary response to light, motor function, and vital signs. Assessment of cranial nerves, reflexes, and sensation may be added if indicated. On occasion, the nurse might be involved with assessing brainstem functioning.

Essential for Safety

When the nurse is assuming care for an unstable patient with a neurological dysfunction, the nurse should complete a focused assessment of the patient with the outgoing nurse.

Glasgow Coma Scale

The **Glasgow Coma Scale** (GCS) assesses both level of consciousness and motor response to a stimulus (Table 10-1). After an extensive review of studies of the GCS, Chestnut and others (2000) concluded there is an increasing probability of poor outcome in a continuous, stepwise manner for patients as the GCS decreases. The GCS score was reliable as long as it had been obtained prior to intubation and either prior to the initiation of sedating medications or when medications had been paused for the neurological exam. When using the GCS, the nurse utilizes the patient’s best response to obtain the score for each section.

The first section is eye opening. The nurse notes if the patient opens her eyes when the nurse approaches (scored as 4).

- If the patient does not, the nurse might say “Good morning, Mrs. Jones” and wait for her to open her eyes (scored as 3).
- If the patient does not open her eyes to her name then the nurse applies noxious stimuli (such as pressure on the trapezius or a sternal rub) and waits to see if the patient opens her eyes (scored as 2).
- If the patient does not attempt to open her eyes to any stimulus, it is scored as a 1.

Table 10-1 Glasgow Coma Scale

Response	Score
Eye Opening	
Spontaneous	4
To speech	3
To pain	2
Never	1
Motor Response	
Obeys commands	6
Localizes pain	5
Normal flexion	4
Abnormal flexion	3
Extension	2
Nil	1
Verbal Response	
Oriented	5
Confused conversation	4
Inappropriate words	3
Incomprehensible sounds	2
None	1

The second section is motor response. The nurse asks the patient to obey a simple one-step command such as “Hold up your thumb” or “Hold up two fingers.” If the patient does not respond, the nurse might ask the patient to follow the command again while demonstrating the action. If the patient follows the command, it is scored as a 6.

- If the patient does not follow a command, then the nurse will need to determine how the patient responds to a noxious stimulus. The nurse might apply pressure on the trapezius; if the patient reaches toward the nurse’s hand to push it away or pulls her shoulder away, the nurse scores it as a 5. Scoring the motor response a 5 means that the patient has clearly identified where the nurse is applying the stimulus and attempted to remove it.
- If instead the patient squirms when the nurse applies the noxious stimuli, moving her entire body although not seeming to be able to identify where the stimulus is applied, it is scored as a 4.
- If the patient assumes a position of abnormal flexion, also called decorticate posturing (shown in ♦ Figure 10-2), either on one or both sides, it is scored as a 3. This is an abnormal finding, and if it is a new finding, the nurse should notify the physician after verifying it.

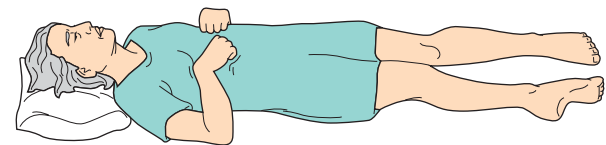


Figure 10-2 Decorticate posture is also known as abnormal flexion.

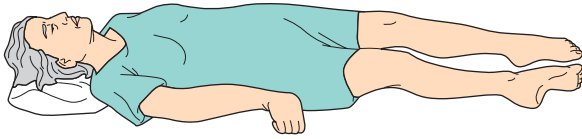


Figure 10-3 Decerebrate posture is also known as abnormal extension.

- With severe injuries, the patient might assume a position of abnormal extension known as decerebrate posturing either on one side or bilaterally (shown in ♦ Figure 10-3), which is scored as a 2. This is an ominous sign.
- If the patient does not respond at all to the stimulus, it is scored as a 1.

The last section is verbal response. Initially the patient is asked her name, the year, and her location. If she is able to respond accurately to these, she is scored a 5.

- If she responds to the question with inaccurate information (e.g., gives the incorrect year or says “I am in a department store”), she is scored a 4.
- If she replies with words unrelated to the question, she is scored a 3.
- If she only moans or groans in response, she is scored a 2.
- If she does not respond at all, she is scored a 1. According to Chestnut et al. (2000), assigning a score of 1 to patients who are intubated (and thus cannot speak) decreases the predictive accuracy of the score. However, some institutions continue that practice.

Finally, the score is totaled with a range from 3 to 15. In general, a score of 13 to 15 indicates mild or no brain injury, a score of 9 to 12 indicates moderate injury, and a score of 8 or less indicates severe injury.

Pupillary Function

Pupillary size and reaction to light are objective measurements that have high inter-rater reliability and are predictive of cognitive outcomes (Oh & Seo, 2009). The nurse begins by assessing the patient’s pupils for size and shape. The size of the pupil may be affected by factors other than the patient’s neurological status. A small pupil may be the result of a very bright room, medications for glaucoma, or administration of opiates. Neurologically, it may be the result of damage to the pons. A dilated pupil may result from fear, anxiety, or cocaine use. Neurologically, it may indicate brainstem compression and is sometimes referred to as a blown pupil when it is greater than 4 mm. There is early research that indicates that an ovoid pupil may be an indication of impending herniation. Pupils are called asymmetrical (also known as anisocoria) when one pupil is more than 1 mm larger than the other. Less than 1% of the

population has such a finding normally. When it occurs in a trauma patient, anisocoria is usually an indication that the ICP is higher than 20 mmHg (DeWall, 2010).

Next, the nurse checks each pupil separately for response to light. Normally, pupils react briskly and completely to light. A nonreactive (fixed) pupil that is dilated usually indicates damage to the oculomotor nerve (III) and impending herniation. A fixed but small pupil may indicate damage to the pons. Instead of manual examination, the nurse may use a pupillometer to determine the size and responsiveness of a patient’s pupils to light. Small significant changes in velocity and size can be detected with the pupillometer, and these changes can be indicative of changes in brain volume and increasing intracranial pressure. In addition, the automated pupillometer has been demonstrated to be more accurate and reliable than manual examination in measuring pupil size and reactivity (Cecil et al., 2011). (Refer to Figure 10-4, which displays how neurological signs, including pupillary changes, may change as ICP increases.)

Reflect On

A patient’s pupils are 2 mm in diameter and are not responding briskly to light. Before inferring the patient has a neurological injury, what other factors should the nurse consider?

Motor Assessment

If the nurse has utilized the GCS, then he has already completed the initial portion of a motor assessment, which is motor response to stimuli. The next section is presence and strength of movement in each of the extremities. If the patient is able to follow simple commands, the nurse asks the patient to lift and hold her arms extended in front of her with the palms up and her eyes closed. A weakness is often revealed when one arm drifts down. The nurse grades the strength of movement in the extremities on the scale utilized by his agency. One commonly used scale is:

- Patient lifts and holds extremity against resistance.
- Patient lifts and holds extremity against gravity.
- Patient lifts and extremity falls back.
- Patient moves extremity but cannot overcome gravity (moves on bed).
- Patient’s muscle shows a trace of movement.
- No movement is noted.

Although the nurse can assess the arms simultaneously, the legs are usually assessed separately. It is important that the nurse assess the movement in all four of the extremities and compare the strength of movement on both sides of the body.

If the patient is unable to move to command, the nurse notes if she is moving all her extremities spontaneously. If necessary, the nurse applies a noxious stimulus to one of

the patient's nail beds on each of her extremities. This allows the nurse to determine whether the patient can sense the noxious stimulus, whether the patient is able to move each extremity to withdraw from the stimulus, and the strength of the movement.

Although it is not voluntary movement, sometimes while the critical care nurse is assessing the ability of a patient to withdraw from a noxious stimulus, he will also check for the presence of a Babinski reflex. To elicit this reflex, the nurse strokes along the outer edge of the sole of the foot then across the ball of the foot with a dull object. The presence of the Babinski response, dorsiflexion of the big toe often accompanied by fanning of the other toes, is an abnormal finding.

A complete motor assessment would also include assessment of coordination. However, this is not ordinarily a part of an hourly neurological assessment. It would be included if the patient had had surgery on the cerebellum or experienced difficulty with coordination following posterior fossa surgery. Simple tests of coordination are the finger-to-nose test and the rapid alternating movement test.

Selected Cranial Nerve Assessment

A complete cranial nerve assessment is time consuming, so it is not always practical or necessary for nurses to perform one on every patient. However, nurses often test specific cranial nerves when caring for their patients. The following is a list of important cranial nerves to assess and ways to assess them:

- The II (optic) and III (oculomotor) nerves are assessed every time the nurse checks a patient's pupils. When the nurse checks for a direct response, he checks the third cranial nerve, and when he checks for a consensual response, he checks the second. It is also important to check the second cranial nerve by assessing the visual field of a patient who has a pituitary tumor, an aneurysm, or a stroke.
- Portions of the V (trigeminal) and VII (facial) cranial nerves can be assessed by checking for a corneal reflex. This is important to check in an unresponsive patient to determine if the patient is able to protect her corneas. It is also important to check following surgery on acoustic tumors.
- The nurse may need to check the VIII (acoustic) cranial nerve because it is one of the more common types of cranial nerve tumors. The nurse assesses the patient's hearing and asks the patient if she is experiencing tinnitus or dizziness.
- The nurse recognizes that the IX (glossopharyngeal) and X (vagus) nerves are intact when the patient exhibits cough and gag reflexes. When appropriate, the nurse may elicit these reflexes by touching the back of the tongue or throat with a tongue blade.

Evaluation of Brainstem Functioning

When a patient is comatose and damage to the brainstem is suspected, the nurse may be asked to assist with evaluation of the oculoccephalic and/or oculovestibular reflexes. The doll's eyes reflex, or oculoccephalic reflex, occurs only in comatose patients. Before attempting to elicit the doll's eyes reflex, the nurse must be certain that the patient's spinal cord has been cleared. The doll's eyes reflex is produced by turning the patient's head left or right. When the reflex is present, the eyes of the patient remain stationary while the head is turned (♦ Figure 10-4). If a comatose patient does not have a doll's eyes reflex, there is usually damage in the area of the pons and/or the medulla.

The oculovestibular reflex, or cold calorics, is performed only after determining that the tympanic membrane is intact. The head of the patient's bed is elevated 30 degrees, then 50 mL of cool saline is injected into an ear. The saline has the same effect as if the patient's head were turned to the opposite side of the injection. Thus, the patient's eyes will look toward the ear of injection for a sustained period. If the reflex is absent, there is most likely a lesion in the pons or the medulla. In a partially awake patient, the reflex will induce eye deviation and nystagmus in the direction of the noninjected ear.

Clinical Findings Associated with Increased Intracranial Pressure

When the volume of a lesion in the skull increases slowly, such as a slow-growing brain tumor, the body compensates for changes in the volume by shifting another volume (such as the CSF or blood). This controls and limits the rise in ICP. The patient usually demonstrates signs and symptoms of dysfunction of the area of the brain on which the tumor is encroaching. For example, depending on the location of the tumor, the patient might develop weakness in her arm and hand. However, the increase in pressure is limited so the patient experiences only mild signs and symptoms of IICP such as headache, nausea, or papilledema.

Nevertheless, once the expansion of the lesion has reached a certain point, or if the lesion is expanding rapidly, the body cannot compensate sufficiently and ICP increases precipitously as displayed in Figure 10-1. Rapidly increasing ICP is dangerous because of the likelihood of secondary injury to the brain, and because, if it continues unabated, it may result in herniation of the brain.

Downward herniation of the brain occurs when ICP is sufficiently elevated to push a portion of the brain downward through the tentorial notch, placing pressure on and distorting the midbrain. There are three types of downward herniation: uncal, central, and tonsillar. Clinical signs vary among the types of herniation. ♦ Figure 10-5

Head in neutral position



Eyes midline

Head rotated to patient's left

Doll's eyes present:
Eyes move right in
relation to head.Doll's eyes absent:
Eyes do not move
in relation to head.
Direction of vision follows
head to left.

Figure 10-4 Oculocephalic reflex: Also known as doll's eye movements, characteristic of altered level of consciousness.

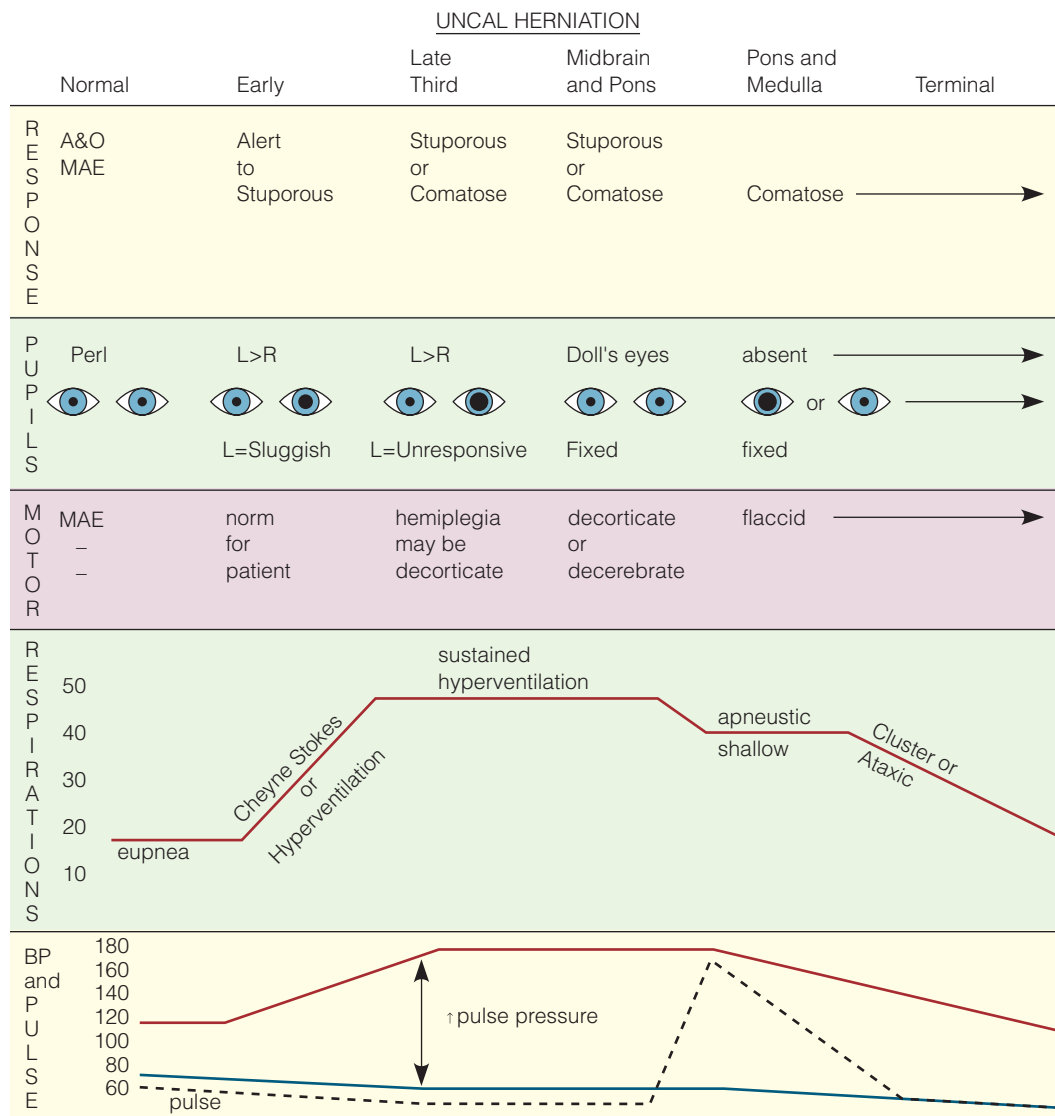


Figure 10-5 In central herniation, the changes begin with motor and level of consciousness. Pupillary changes occur later. The classic Cushing response is an increase in PP, slow pulse P, and respiratory variations. These are often very late signs.

displays a constellation of vital and neurological signs that may occur with uncal herniation and notes how it may differ from central. A triad of vital sign changes known as the Cushing response—bradycardia, hypertension (accompanied by a widened pulse pressure), and respiratory variations—may occur. It is extremely important to note that these changes in vital signs, dilation of the pupils, abnormal posturing, and absence of doll's eyes are very late signs indicating a significant amount of damage. Suspected herniation is an emergency and requires immediate response from the healthcare team. If intervention is not forthcoming, the patient will probably die.

Essential for Safety

If a nurse suspects herniation, he should immediately notify the appropriate neurological care provider.

Building Technology Skills

Intracranial Pressure Monitoring

Thus far, the assessment of the patient with IICP has been described purely in terms of subjective signs and symptoms. Because some of these signs may occur only after ICP is gravely elevated and the signs can be difficult to interpret, it is important to have objective data to correlate with the neurological signs. ICP monitoring obtains objective measurement of ICP to correlate with the neurological findings and to guide therapy. ICP monitoring is useful because it can be continually displayed, is more likely to detect IICP than a CT scan, and allows ICP to be assessed accurately while the patient remains sedated.

Indications

ICP monitoring may be indicated in severely ill but salvageable patients with:

- Severe head injury with a GCS between 3 and 8 and in patients with an abnormal CT who are over the age of 40, have a blood pressure less than 90 mmHg systolic, or motor posturing.
- Subarachnoid hemorrhage.
- Hydrocephalus.
- Brain tumors.
- Stroke.
- Meningitis.

Principles of Treatment

ICP monitoring is used to identify a potentially dangerous ICP (ICP greater than 20 for 5 to 10 minutes), to determine when to institute therapy, and to evaluate the effectiveness

of the therapy. According to the AANN (2011), “continuous monitoring of ICP is the only method to reliably monitor intracranial compliance, cerebrovascular status, and cerebral perfusion.” Yuan et al. (2015) determined that intracranial pressure monitoring in conjunction with targeted therapies is associated with significantly lower mortality in patients with traumatic brain injuries whose ICP is 3-5 on admission or drops to less than 8 within the first 24 hours after injury.

Methods

ICP monitoring devices may be inserted into a variety of spaces including the anterior horn of the lateral ventricle; the subdural, epidural, or subarachnoid spaces; and the brain parenchyma. There are three common types of sensing devices: fiber-optic, electronic sensor, and fluid-filled strain gauges. ♦ Figure 10-6 displays the location of various devices and the Brain Trauma Foundation (BTF) ranking by preference.

According to the Brain Trauma Foundation (2007), the ventricular catheter connected to an external strain gauge is the most accurate, low-cost, reliable method of monitoring ICP. In addition, unlike some of the other sensors, it can be used to drain CSF and can be recalibrated in place. Due to the emergent nature of the situation, ventricular catheters may be inserted in the intensive care unit (ICU), emergency department (ED), or operating room (OR) by a neurosurgeon utilizing aseptic technique. After the scalp is prepped, the neurosurgeon uses a twist drill to create a burr hole through the skull over the patient's nondominant hemisphere.

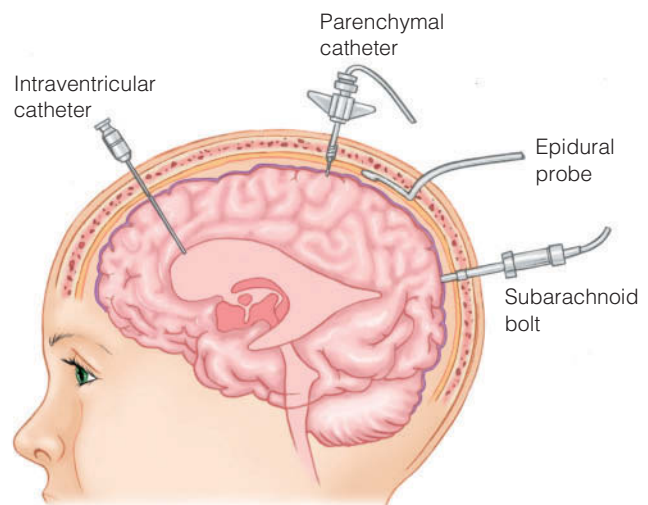


Figure 10-6 Location of types of intracranial pressure monitoring devices.

Numbers indicate location and BTF ranking by preference:

1. Intraventricular
2. Intraparenchymal
3. Subarachnoid
4. Epidural

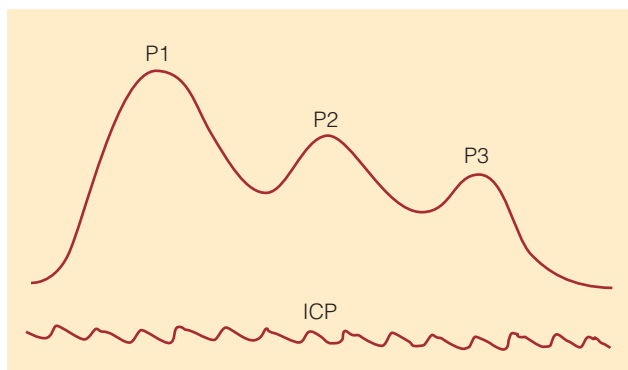


Figure 10-7 Contours of the normal ICP wave: P1, P2, P3.

The ventricular catheter is then inserted through the burr hole and the brain tissue into the lateral ventricle. Once in place and connected, the catheter transmits ICP waveforms to the transducer, which converts the impulses into visible tracings and/or digital values on a monitor screen. Normal ICP ranges between 5 and 15 mmHg. The normal ICP waveform has three peaks of decreasing height (P1, P2, and P3) that correlate with the arterial waveform (♦ Figure 10-7).

There are three common variations in ICP waveforms termed A, B, and C waves (♦ Figure 10-8). The first and most clinically significant are “A” waves, also known as plateau waves. “A” waves are elevations of ICP up to 50 to 100 mmHg that last for 5 to 20 minutes. They have a rapid onset and often occur from an already elevated baseline. “A” waves are significant because they are often accompanied by signs of neurological deterioration and may indicate impaired CSF flow, decreased compliance, or impending herniation.

Also clinically significant are “B” waves, which are rhythmic oscillations that occur once or twice a minute. They result in ICP increases of 20 to 50 mmHg and may occur on a normal or elevated baseline. They are significant because they tend to occur when cerebral compliance is decreased and often precede “A” waves. “C” waves are smaller rhythmic oscillations that occur four to eight times per minute. Although ICP may increase up to 20 mmHg, they tend to occur normally and are not indicative of pathology.

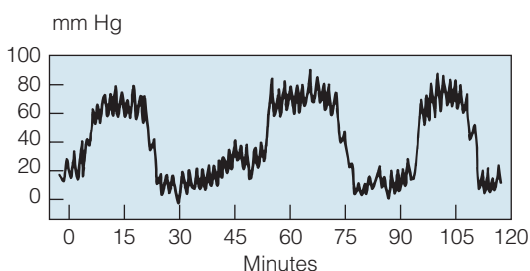


Figure 10-8 Common variations in ICP waveforms: A, B, and C waves.

Nursing Responsibilities

Although it is appropriate to obtain consent before placement of an intraventricular monitoring device, it may not be possible when the patient has experienced a severe brain injury and placement of the ICP monitoring device must be performed emergently. The nurse is usually responsible for setting up and calibrating the monitoring system. After the ICP monitoring system is established, the nurse assesses, reports, and records the patient’s initial pressures (MAP, ICP, and CPP), waveforms, and neurological status.

While an ICP monitor is in place, the nurse:

- Performs hourly neurological checks and compares them to the patient’s baseline.
- Monitors MAP, ICP, and CPP (MAP-ICP) at least hourly.
 - Utilizes the ICP and CPP to guide the provision of collaborative interventions and nursing care.
 - Monitors the effects of providing required nursing care such as suctioning and positioning on the ICP and CPP.
- Validates that ICP waveforms and values are accurate. Especially if the values have changed, the nurse verifies the readings, correlates them to neurological findings, and notifies the physician. Some of the reasons ICP readings may be inaccurate are if the stopcocks are turned incorrectly, the transducer is incorrectly positioned, there is air in the monitoring line, or the catheter has become obstructed. Problematic malfunctions of the ventricular strain gauge monitoring system occur in 6% to 16% of patients, and are one of the more common complications of ICP monitoring. One of the disadvantages of a fiber-optic catheter tip device is that obstruction may occur as often as 40% of the time.
- Maintains strict aseptic technique when working with the system.
- Ensures that the dressing at the insertion site is clean, dry, and occlusive. The dressing should cover only the insertion site and should be changed aseptically if it is damp.
- If the nurse suspects there is a CSF leak (identified by the presence of a damp dressing with a halo or glucose in the leaking fluid), she checks the system for leaks and notifies the physician.

Complications

The most common complication, occurring in 1% to 27% of patients with ICP monitoring devices, is infection. Infections develop with increasing frequency during the first 10 days following insertion but then decrease in frequency. They are more likely to occur if the system is opened,

irrigations are performed, or prophylactic bacitracin flushes are given. If infection develops, the patient may:

- Have a fever.
- Report a headache.
- Demonstrate signs of meningitis.
- Have cloudy CSF drainage.

The nurse anticipates that the CSF will be cultured, the patient will be treated with antibiotics, and the patient may undergo removal of the ICP monitoring system.

Another complication, occurring in 1% to 2% of patients with a ventriculostomy catheter, is hemorrhage during insertion of the catheter followed by hematoma formation. It is more likely to occur in patients with coagulation abnormalities. Usually the patient must be taken to the OR for repair.

Primary Cause of Increased Intracranial Pressure: Traumatic Brain Injury

In the United States in 2010, there were 2.5 million visits to an ED for traumatic brain injuries (TBIs) resulting in the deaths of more than 50,000 people and hospitalizations of 280,000 more (CDC, 2015). CDC reports that head injuries are a major cause of death and disability contributing to 30% of all injury deaths. Overall, men are about three times more likely than women to experience TBI, and adults over the age of 65 are more likely to die from TBI.

Predisposing or Risk Factors

Blunt trauma is the most common cause of TBI. Motor vehicle crashes account for about 15% of TBIs, whereas falls (40%), assaults (10%), unintentional blunt trauma and sports-related injuries account for the majority of the rest (CDC, 2015). Sports and recreation events are a major source of concussions and are thought to be severely underestimated in the national data sets because only between 8% and 20% of people sustaining such injuries lose consciousness and seek medical assistance (Langlois, Rutland-Brown, & Wald, 2006). These large numbers of injuries are especially problematic because TBI is one of the most disabling of injuries. Langlois et al. estimated that about 5.3 Americans are living with long-term or lifelong disability following hospitalization for a TBI. In addition, in the first 3 years post injury, patients recovering from a TBI are more likely to report binge drinking, develop epilepsy, and die. The remaining injuries are explained by penetrating injuries such as gunshot wounds and blast injuries. Although blast injuries are uncommon in the United States, they are the leading cause of TBI among active duty military in war zones.

There are clearly established ways to prevent TBI-related deaths. Wearing a lap belt reduces mortality in motor vehicle crashes by 50%. Wearing a motorcycle helmet reduces fatalities also by 50%. More impressively, wearing a bicycle helmet can reduce mortality from TBI in bicycle crashes by 85% (Nolan, 2005).

Gerontological Considerations

Because older adults have age-related brain atrophy, they are less likely to present with the generalized symptoms of IICP such as headache and papilledema and more likely to present with mental status changes.

In spite of having a significant intracranial injury, older adults may present with a normal level of consciousness, absence of neurological deficit, and no evidence of open or depressed skull fracture—especially if they are receiving anticoagulant therapy.

Adults over the age of 65 are more likely to be hospitalized and die from TBI.

Patients with acute subdural hematomas older than 65 who are comatose on admission have significantly higher mortality rates than other patients (Roger, Butler, & Benzel, 2006).

Chronic subdural hematomas are more common in older adults. This may be attributed to age-related brain

atrophy resulting in more subdural space so the older adult is less likely to be affected initially by a small subdural bleed.

If an older patient with a traumatic brain injury develops hyponatremia (serum sodium greater than 160 mmol/L), he is at increased risk for renal failure, heart failure, and pulmonary edema compared to a younger adult.

Depression occurs in as many as a third of elders following traumatic brain injury (Menzel, 2008).

The prevalence of epilepsy increases with age; thus, it is the third most common neurological condition in old age after dementia and stroke (Chappell, Bagshaw, & Crawford, 2010). Older adults need lower initial and maintenance doses of anticonvulsants due to slower hepatic metabolism and decreased renal clearance of the drugs.

Pathophysiology and Manifestations of Primary Brain Injuries

In TBI, the primary injury is a result of the initial impact to the head causing direct damage to the cerebral parenchyma and resulting in axonal injury. The severity of the injuries is often classified according to the GCS. A **mild traumatic brain injury** (GCS 13–15) is usually associated with a loss of consciousness or amnesia for less than an hour. A **moderate head injury** (GCS 10–12) may be associated with a loss of consciousness for up to a day, whereas a **severe head injury** (GCS less than or equal to 8) is usually associated with loss of consciousness for more than 24 hours. Guidelines have been developed for the assessment and management of both mild and severe head injuries. These specific injuries and the guidelines that have been developed to manage them are described next. At this time, no guidelines exist for the management of moderate head injuries (Bergman, Maltz, & Fletcher, 2010).

Concussion and Mild Brain Injury

Mild TBIs account for the majority of all diagnosed head injuries. MBIs often result from falls or blunt trauma, but may be especially common after sports-related injuries. Concussion, a type of MBI, is caused by the sudden deceleration of the brain against the fixed skull and is not associated with underlying parenchymal damage. In MBI, the exact neural mechanisms are unclear but most likely include release of neurotransmitters, mitochondrial dysfunction, diminished glucose metabolism, and transient hyperkalemia.

Despite the realization that the GCS lacks sensitivity, it is still commonly used for patients with TBI. A patient with a GCS score of 15 is considered to have mild injury. Amnesia is a hallmark of MBI, so patients should be asked to describe the circumstances surrounding the injury. The Canadian CT Head Rule (CCTHR depicted in Box 10-1) appears to be more sensitive than the New Orleans Criteria and may be used to identify the need for further evaluation and treatment (Bouida et al., 2013). According to Clement et al. (2006), less than 1% of patients with a GCS of 15 require surgical intervention, and all of these can be identified as being at high risk for neurological intervention on the CCTHR.

Problems experienced by patients with MBIs include headache, anxiety, dizziness, fatigue, and memory loss, which are not visible, are difficult to detect, and may result in functional loss (Bergman & Bay, 2010). For the majority of patients, these symptoms should resolve within several weeks to three months. Post-concussion syndrome, the persistence of memory or attention deficits accompanied by disordered sleep, headaches, irritability, dizziness, or personality changes beyond 6 months, occurs in 20% to

40% of people with MBI. Headache and dizziness can be difficult to manage and are associated with delayed return to work. There is evidence that repeated MBIs have a cumulative effect, especially if the repeat injury occurs before the patient has fully recovered from the first injury. This has implications for when patients should be allowed to return to their normal activities—especially participation in sports. A tool kit is available from the CDC to assist with assessment and management of the patient with a mild traumatic brain injury.

Cerebral Contusions and Diffuse Axonal Injuries

Contusions and axonal injuries often result from acceleration/deceleration injuries, such as a fall or a motor vehicle collision. Contusions develop as the brain accelerates against the fixed skull, causing disruption of the underlying cerebral parenchyma and blood vessels. This is known as a coup injury. After impacting the skull, the brain may recoil and impact the skull on the opposite side, causing additional damage to the cerebral parenchyma known as a countercoup injury. The severity of the contusion is related to the amount of energy that is transferred on impact. Cerebral edema develops 24 to 72 hours in response to the brain tissue injury and may result in IICP. The most common locations for cerebral contusions are the frontal and temporal lobes.

Diffuse axonal injury results from deceleration and shearing between the different densities of white and gray matter in the brain. Axonal injuries are graded from I to III with Grade I being found in patients with milder injuries, Grade II consisting of tear hemorrhages and axonal

Box 10-1 Canadian CT Head Rule

High-Risk for Neurological Intervention

- GCS score less than 15 at 2 hours after injury
- Suspected open or depressed skull fracture
- Any sign of basilar skull fracture
- Age older than 65 years

Medium Risk for Brain Injury on CT

- Amnesia before impact of greater than or equal to 30 minutes
- Dangerous mechanism (defined as pedestrian struck, an occupant ejected, or a fall from greater than three feet)
- The rule is not applicable for a nontrauma patient, a patient with a GCS less than 13, a patient less than 16 years of age, a patient with a bleeding disorder or taking an anticoagulant, or a patient with an open skull fracture.

SOURCE: Division of Emergency Medicine, University of Ottawa, Ottawa, Canada

injuries in the cerebral hemispheres, and Grade III including abnormalities of the brainstem in addition to the Grade II injuries. Axonal edema and degeneration may continue for the first 2 weeks following injury. Initial neuroimaging may be normal with edema, atrophy, and petechial hemorrhages becoming evident with time. The patient manifestations are dependent on the location and extent of the injury.

Skull Fractures

Skull fractures may occur in response to a blunt, penetrating, or blast injury. Common types of skull fractures include the following:

- Linear fractures are nondisplaced and occur from a low-velocity impact.
- Depressed skull fractures are fractures in which depression of the bone has developed at the point of impact. They may be closed (without direct penetration of the brain) or open (penetrating the dura). These patients usually require surgery for repair of the skull, debridement of bone fragments, and repair of the dura. Postoperatively, the nurse must assess the patient carefully for CSF leaks and the development of meningitis.
- Basilar fractures occur at the base of the skull. Patients may develop a dural tear and have CSF draining from their nose and/or ears. See the earlier discussion for management of a CSF leak. Eventually, the patient may develop “racoon eyes” and Battle’s sign, which are ecchymoses about the eyes and behind the ears.

Vascular Injuries

Vascular injuries may result from bleeding of the arteries and veins between the brain and the skull or in the brain tissue. Bleeding that occurs between the brain and the skull is a surgical emergency because the enlarging hematoma may compress the adjacent blood vessels causing cerebral ischemia, or may distort surrounding brain tissue.

Epidural Hematomas. **Epidural hematomas** occur in 1% to 2% of TBIs. They usually occur in conjunction with a skull fracture and result from a laceration of the middle meningeal artery, causing bleeding between the dura mater and the skull (♦ Figure 10-9). Approximately half of the patients who suffer this injury demonstrate the classic presentation of an initial loss of consciousness followed by a lucid interval then a sudden re-loss of consciousness with rapid deterioration in neurological status. Because the origin of the bleeding is arterial, the epidural hematomas usually accumulate rapidly and therefore must be evacuated as soon as possible. Patient outcomes are good if the condition is identified promptly and the patient is taken to the OR for immediate surgical evacuation of the hematoma.

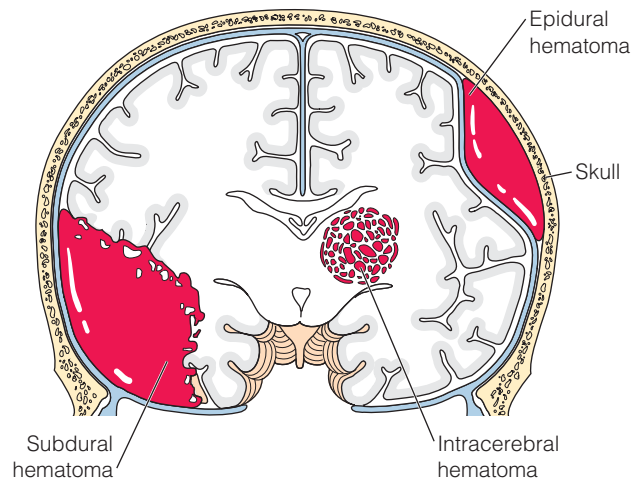


Figure 10-9 Hematomas: epidural, subdural, and intracerebral hematomas.

Subdural Hematomas. **Subdural hematomas** occur in 10% to 20% of TBIs. In blunt trauma, they usually are the result of countercoup injuries, occurring on the opposite side of the skull from the injury just below the dura (♦ Figure 10-9). In contrast to an epidural hematoma, this hematoma is usually the result of venous bleeding, often originating with the stretching of bridging veins. Subdural hematomas are classified as acute, subacute, and chronic.

Acute subdural hematomas are collections of thick, jellylike blood that accumulate within the first 24 to 48 hours after blunt trauma. Patients usually present with a loss of consciousness and deteriorating scores on the GCS. In addition, they may have focal signs such as hemiparesis or dysphagia. If allowed to continue to expand, the hematoma can have a mass effect, causing an increase in ICP, midline shift of the brain, and even herniation. Acute subdural hematomas can be rapidly identified on computerized tomography (CT) scan and require emergent craniotomy for evacuation of the thick, coagulated blood. Patients older than 65 with acute subdural hematomas who are comatose on admission have significantly higher mortality rates than other patients (Roger et al., 2006).

Subacute subdural hematomas usually develop over days to weeks following the injury. The clot usually has liquefied and surgical evacuation may be performed on an elective basis.

Chronic subdural hematomas are more common in older adults. This may be attributed to age-related brain atrophy resulting in more subdural space so the older adult is less likely to be affected initially by a small subdural bleed. However, during the 2 to 3 weeks following the injury, the hematoma liquefies, enlarges, and develops fibrous membranes. As the hematoma enlarges, the patient usually develops nonspecific symptoms such as headache, confusion, and speech deficits. The hematoma can be identified on CT scan. Treatment may

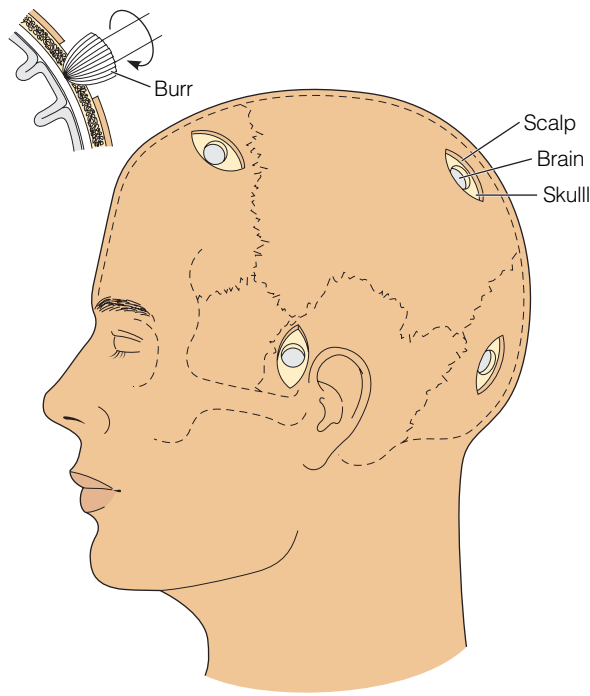


Figure 10-10 Possible location of burr holes.

involve drainage via burr holes (♦ Figure 10-10) or craniotomy, depending on how medically stable the patient is and the characteristics of the hematoma.

Postcraniotomy for subdural hematoma, the most common complication, occurring in 25% of patients, is a re-accumulation of the hematoma. This occurs more commonly in older adults who tend to have slow expansion of their brains after evacuation of the hematoma. Often, soft drains, such as Jackson Pratt drains, are placed for drainage. The nurse handles these aseptically and monitors the output. The nurse also assesses the patient's neurological signs and immediately identifies and reports any change from baseline.

Subarachnoid Hemorrhage. **Subarachnoid hemorrhage**, bleeding between the arachnoid and pia mater, may result from rupture of a preexisting or a traumatic cerebral aneurysm.

Intracranial Hemorrhage. Intracranial hemorrhages result in bleeding into the parenchyma or ventricles of the brain. Cerebral contusions and edema usually accompany them. Management will be described in the section Collaborative Care later in this chapter.

Severe Traumatic Brain Injury

For the patient with a TBI, the nurse completes the GCS and assesses the pupillary response to light, motor function, and vital signs. Assessment of cranial nerves, reflexes, and sensation may be added if indicated. On occasion, the nurse might also be involved in assessing brainstem functioning of the

patient with a severe TBI. Assessment of these elements was discussed earlier in this chapter in the section Assessment of a Patient with a Potential for Increased Intracranial Pressure. Any patient who has a severe TBI may also have injured his neck and spinal column: the patient will usually have some form of spinal stabilization in place and will need to be assessed for a spinal cord injury as well as a head injury.

There are several findings from the initial neurological assessment that predict death and disability for a patient with a severe TBI. The factors most predictive of outcome are:

- Patient age.
- GCS prior to intubation and administration of medications for sedation.
- Pupillary size and response to light.
- Presence of an extracranial injury.
- Findings on the CT scan.
- The presence of hypotension (Chestnut et al., 2000; MRC CRASH Trial Collaborators, 2008).

Patients who were older, had lower motor scores, had one or both nonreactive pupils, or had a blood pressure (BP) less than 90 mmHg systolic were more likely to have unfavorable outcomes. Hypotension was even more likely to result in a poor outcome if it was associated with hypoxia.

Findings from the initial assessment should be acted on with the patient's underlying condition in mind. Older adults may appear relatively normal, having a normal level of consciousness, no neurological deficit, and no skull fracture. Yet they may actually have a significant intracranial injury, especially if they are receiving anticoagulant therapy. Approximately 35% to 50% of patients who experience a TBI are under the influence of alcohol. Sperry et al. (2006) demonstrated that alcohol intoxication does not result in clinically significant changes in patient scores on the GCS, and recommends that appropriate monitoring and interventions be instituted immediately based on the patient's GCS and not delayed waiting for the patient to "sober up."

Essential for Evidence-Based Practice

The nurse should never assume that abnormal neurological findings found when performing the GCS are the result of alcohol intoxication.

According to the Brain Trauma Foundation (2007), ICP monitoring should be instituted in all salvageable patients with severe TBIs (GCS scores 3–8) who have abnormal CT scan findings such as hematoma, swelling, herniation, or compressed cisterns. In addition, it is indicated in patients with severe brain trauma with normal CT scan findings if they are over the age of 40, exhibiting posturing, or have a systolic BP less than 90 mmHg. The details of ICP

monitoring were described earlier in this chapter. The rationale for recommending ICP monitoring is that “only part of the damage occurs at the moment of impact and significant reductions in morbidity and mortality can be achieved in patients with severe TBI by using intensive management protocols” (p. S37).

Advanced monitoring techniques are available in some neurointensive care units. Collectively known as multimodal monitoring techniques, they provide direct evidence of the metabolic and energy state of the brain through measurements of brain metabolites as well as cerebral blood flow, brain oxygenation, and temperature (Cecil et al., 2011). Utilizing these measurements, neurocritical care management strategies can be focused on the prevention and limitation of secondary brain injury and can result in reduction in mortality and morbidity (Dagal & Lam, 2011). The detailed description of these multimodal strategies is beyond the scope of this book.

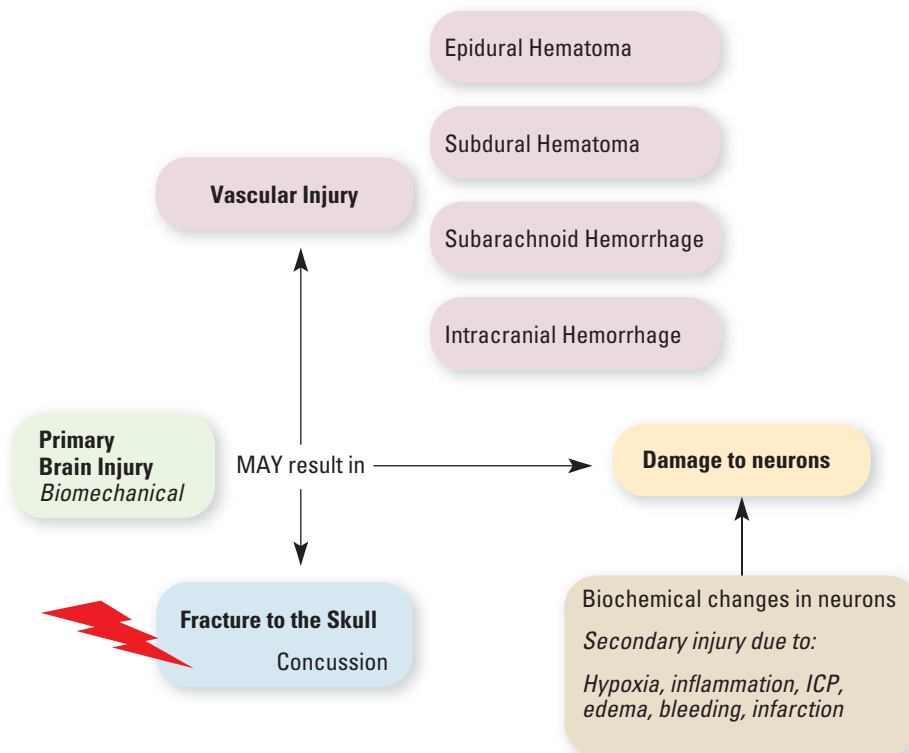
COLLABORATIVE CARE

In TBI, although the primary neurological injury can be life threatening, it is often management of the secondary injuries that determines the patient’s outcome. Secondary injuries are the physiological responses to the initial neurological insult. If the patient develops an uncontrolled increase in ICP, autoregulation may be impaired and viable cerebral tissue may not be adequately perfused. If the patient becomes

hypotensive, the CPP will fall and brain tissue may be in peril. The neurons’ response to this reduction in blood supply and ischemia is responsible for the secondary brain injury. When the cerebral cells are deprived of oxygen and they are forced to utilize anaerobic pathways of metabolism, normal processes fail. The sodium-potassium pump fails, allowing sodium and water to enter cells. At the same time the cell membrane breaks down. Both these factors result in edema formation (McIlvoy & Meyer, 2008). With insufficient energy available for cellular functioning, excitatory neurotransmitters are released, there is an influx of calcium into the neurons, oxygen-free radicals are generated, and inflammatory mediators are released. These processes contribute to a downward spiral, with cerebral edema increasing, ICP rising, perfusion of the brain decreasing, and cerebral ischemia worsening. Thus, the development of secondary injuries contributes significantly to disability and death post TBI.

The goal of collaborative management for the patient with a severe head injury is maintaining sufficient blood flow and oxygenation to meet the brain’s metabolic needs, thus avoiding secondary injury while the brain recovers from the traumatic event. According to the Brain Trauma Foundation (2007), protocols for immediate management of the severely head-injured patient call for:

- Early intubation.
- Transport.
- Prompt fluid resuscitation.



Visual Map 10-2 Primary Brain Injury

- CT scanning.
- Immediate evacuation of any intracranial mass lesion.
- Management in an ICU with ICP monitoring.

When Brain Trauma Foundation protocols are followed, intracranial pressure, two-week patient mortality, and overall mortality for patients with a severe TBI show significant improvement (Gerber, Chiu, Carney, Hartl, & Ghajar (2013). Thus, when a patient with a severe TBI arrives in the critical care unit, he may have been intubated, has usually undergone initial CT scanning, and may have had emergent surgery and/or an intraventricular catheter placed for monitoring and drainage of CSF.

Provision of Adequate Oxygenation

The critical care nurse recognizes that patients with severe TBIs will be at risk for respiratory compromise. Rangel-Castillo and Robertson (2006) state that 60% of patients with severe head injuries have breathing abnormalities such as periodic respirations. Almost 40% of comatose head-injured patients are hypoxic from respiratory dysfunction and require intubation and mechanical ventilation. It is imperative that the critical care nurse monitor the patient's pO_2 and oxygen saturation because hypoxia, defined by the Brain Trauma Foundation (2007) as a pO_2 less than 60 or an O_2 saturation less than 90%, is associated with increased morbidity and mortality. If the patient has not already been intubated, the nurse attempts to improve the patient's oxygenation through positioning and oxygen administration of 100% oxygen by mask or bag/valve mask. Should that not be feasible, she anticipates the likelihood of emergent intubation to maintain effective airway management and ventilation to ensure adequate oxygenation.

Essential for Safety

If a patient with a TBI has an oxygen saturation less than 90%, the nurse should immediately attempt to improve the saturation by repositioning the patient and supplying 100% oxygen.

Intubation and mechanical ventilation of the head-injured patient are not benign procedures and may be associated with problems for the severely head-injured person. Intubation can be a noxious procedure and may increase ICP. Although it has been postulated that premedication would be less noxious and blunt any rise in ICP, premedication has not been shown to have an effect on morbidity and mortality (DeWall, 2010). Nevertheless, rapid sequence intubation is utilized and might include:

- Supporting the patient's respirations with 100% O_2 via bag/valve mask.
- Administration of lidocaine to inhibit central responses that can increase ICP.

- Administration of a sedative hypnotic agent.
- Administration of a rapid-acting neuroblocking agent.
- Checking for jaw relaxation after 30 seconds; if it is present, intubation.
- Confirmation of tube placement.
- Sedation of patient with chosen agent.

Mechanical ventilation alone may have adverse effects on ICP. The critical care nurse will need to monitor the patient to determine that he is optimally ventilated. Davis et al. (2006) determined that mechanically ventilated head-injured patients who were either hypercarbic or hypocarbic had worse outcomes. It is postulated that a low pCO_2 results in vasoconstriction contributing to cerebral ischemia, whereas a high pCO_2 induces vasodilation and increases ICP. This finding highlights how essential it is that the nurse review the patient's blood gases and determine that the patient is adequately ventilated. The goals for blood gases for head-injured ventilated patients during the emergent and resuscitation phases are pCO_2 ranging between 35 and 40 and SpO_2 greater than 96% (Bader et al., 2014).

NURSING ACTIONS

The critical care nurse recognizes that suctioning the patient's endotracheal tube may result in transient reductions in oxygenation. In addition, suctioning is a noxious procedure, and for both these reasons may impact ICP. Therefore, the nurse:

- Pre-oxygenates the patient prior to suctioning.
- Medicates the patient prior to suctioning with sedatives or opiates to prevent coughing and an increase in ICP (AANN).
- Limits the duration of each suctioning pass to less than 10 seconds and the number of passes.
- Observes the effect of positive end expiratory pressure (PEEP) on the patient's blood pressure (BP) and ICP to be certain that they are not deleteriously affected. PEEP may have an adverse effect on the patient's ICP by either decreasing the venous return from the head or by decreasing the BP. The higher the ICP, the higher the PEEP must be to have an effect on venous return.

Provision of Adequate Perfusion

Because cerebral blood flow is dependent on the BP, the Brain Trauma Foundation (2007) states that the BP should be monitored and hypotension should be avoided. Although the Brain Trauma Foundation believes that it would be preferable to identify a target BP, preferably a target MAP, it states that there is not an adequate research base to support such a goal. Therefore, the Brain Trauma Foundation guidelines specify only that a systolic BP less than 90 mmHg should be

avoided while Bader et al. (2014) recommend keeping the MAP between 70 and 90. The Brain Trauma Foundation has also not been able to establish when the BP might be too high. Many patients with TBI and/or IICP will present with hypertension associated with sympathetic hyperactivity. In patients with intracranial lesions, this BP is usually not treated because the higher pressure is probably maintaining cerebral perfusion. In other hypertensive brain-injured patients, the BP may decrease in response to adequate sedation.

Although a target BP may not be identifiable, a systolic BP less than 90 mmHg or a MAP less than 70 in a patient with a TBI is clearly a predictor of a poor outcome. If the BP or MAP is decreasing, initial management involves ensuring that the patient is euvolemic. In general, hypotonic crystalloids are avoided in patients with TBIs because they have the potential for worsening cerebral edema. In addition, patients with TBI who receive large volumes of fluid are at much higher risks for the development of adult respiratory distress syndrome (ARDS). Therefore, isotonic crystalloids such as 0.9% saline is commonly used. Normal saline is preferred because it is inexpensive, iso-osmolar, and has no free water. The administration of free water is avoided because sodium disturbances are common in TBI patients and hyponatremia is associated with poor outcomes. Although a sufficient research base is not yet available for the Brain Trauma Foundation to include it as a recommendation, there have been successes reported utilizing 250 mL of hypertonic saline to expand intravascular volume and correct hypotension while limiting cerebral edema. One drawback of using hypertonic saline as a resuscitation fluid is that healthcare providers must know the serum sodium prior to beginning the infusion.

Essential for Evidence-Based Practice

If the patient with a TBI remains hypotensive after initial volume replacement, then the nurse should search for extracranial injuries such as femoral and pelvic fractures, since an adult cannot bleed enough into the brain to cause hemorrhagic shock (DeWall, 2010).

Maintenance of Adequate Cerebral Perfusion

Maintenance of a systolic pressure greater than 90 mmHg is only one of several factors necessary to maintain adequate perfusion of the brain. As noted previously, CPP is defined as the MAP minus the ICP.

$$\text{CPP} = \text{MAP} - \text{ICP}$$

Therefore, any factor that increases the ICP will have an impact on cerebral perfusion. At this point, it is clear that a low CPP (less than 60) should be avoided because it is associated with a loss of autoregulation, the brain's ability to call for an adequate BP to enhance cerebral perfusion, and with poor patient outcomes. The Brain Trauma Foundation states there is even clearer evidence that increasing

the CPP to values greater than 70 by providing fluids and vasopressors (which was once recommended) should be avoided due to the high risk of ARDS and the likelihood that a CPP greater than 70 has detrimental effects on the brain. Thus, it believes that an optimum value for a CPP lies about 60. Based on the equation, CPP can be increased by either increasing MAP or decreasing ICP.

Managing Intracranial Pressure

According to the Brain Trauma Foundation (2007), there is clear evidence that treatments to reduce ICP should be initiated when the ICP threshold rises above 20 mmHg. Sustained increases in ICP to greater than 20 mmHg for 10 minutes or longer are associated with poor patient outcomes. A variety of therapies may be attempted to reduce ICP.

NURSING ACTIONS

Sedation and Pain Relief

A patient who is agitated, coughing, or in pain may have a significant increase in his cerebral metabolic rate, oxygen consumption, BP, and ICP. The Brain Trauma Foundation (2007) notes that the use of sedation is a common management strategy even though it has not been shown to affect patient outcomes nor to consistently decrease ICP. Benzodiazepines may be utilized to control agitation associated with mechanical ventilation because they are generally well tolerated and can reduce cerebral metabolism. For ventilated patients, McIlvoy and Meyer (2008) recommend the use of propofol, a sedative-hypnotic anesthetic, which decreases ICP and allows less use of neuromuscular blockers and benzodiazepines. One advantage of propofol is that, in most patients, the dose may be lowered or discontinued, and within about 10 minutes an accurate neurological assessment can be obtained. Studies demonstrate that patients with TBI sedated with propofol show improvement in ICP control (Saiki, 2009). However, long-term and high-dose use of propofol are associated with significant morbidity, specifically the propofol infusion syndrome.

The patient with a head injury may experience significant pain either from the head injury or from other injuries incurred in the traumatic event. Morphine is widely used because it has a high level of efficacy and safety yet is minimally sedating. It can be given as a continuous infusion and titrated as needed for additional pain. If necessary for a neurological assessment, it can be reversed with Narcan. Fentanyl may be used cautiously because it results in a mild but definite increase in ICP, according to the Brain Trauma Foundation (2007).

Osmotherapy

The osmotically active agents currently being used to decrease ICP are mannitol and hypertonic saline. Class II

and III evidence suggest that both effectively lower ICP (Adamides et al., 2006; Rickard et al. 2014). However, Rickard mentions that there is a current trend favoring the use of hypertonic saline over mannitol, and Li, Chen, Chen,

Cai, and Hu (2015) determined that at 60 and 120 minutes post-intervention, hypertonic saline resulted in a significantly greater decrease in ICP than mannitol. See “Commonly Used Medications.”

Commonly Used Medications

Mannitol (Osmitol), an Osmotic Diuretic

Desired Effect: Mannitol decreases ICP while reducing cerebrovascular resistance and increasing cerebral blood flow. The reduction in ICP from mannitol begins almost immediately after administration, probably due to its plasma-expanding effect and a decrease in the rigidity and volume of the red blood cells. The result is an increased blood flow to cerebral tissue. After 15 to 30 minutes, a delayed osmotic effect develops with the direct removal of water from brain parenchyma. Onset of diuresis is in 1 to 3 hours with a duration of effect of up to 6 hours. The Brain Trauma Foundation (2007) states, “Mannitol is effective for control of raised ICP at doses of 0.25 to 1 g/kg body weight” (p. S14). Nonetheless, some centers utilize low-dose mannitol at scheduled 2-hour increments in an attempt to obtain a lower and smoother ICP. The use of continuous infusions or prolonged administration of mannitol is often avoided because it is theoretically possible that mannitol could accumulate in the cerebral tissues and result in a rebound edema.

Nursing Responsibilities:

- The nurse identifies when the patient’s ICP warrants treatment with mannitol (usually for a sustained ICP greater than 20).
- The nurse monitors the patient’s neurological signs and ICP following administration of mannitol.
- If the 15%, 20%, or 25% solutions are utilized, then the nurse administers mannitol through an in-line filter.
- Mannitol may cause hypotension after rapid administration, especially in volume-depleted patients. Therefore, it is important that the patient be euvolemic and the nurse administer a bolus dose no more rapidly than over 15 to 30 minutes.
- Mannitol increases the osmolality of the blood with optimal osmolality between 300 and 320 mOsm. If repeated doses of mannitol are given, the nurse monitors the serum osmolality every 4 to 6 hours and ensures that it remains less than 320 mOsm.
- The nurse also monitors the urine output to ensure that it is at least 30 to 50 mL/hr and reviews the lab results, looking specifically for hyponatremia and hypokalemia.

Potential Side and/or Toxic Effects: Even though side effects of mannitol are rare when administered as directed, the patient needs to be closely monitored for the adverse effects that can occur from its use. The most common

adverse effects are hypotension, congestive heart failure, pulmonary edema, and electrolyte imbalances (hyponatremia and hypokalemia).

Hypertonic Saline

Desired Effect: Theoretically, hypertonic saline decreases ICP while increasing CPP. The principal effect is probably mobilization of water and reduction in the cerebral water content. It also increases BP because of its plasma expansion effect and may have beneficial effects on the brain’s microcirculation. It has been associated with reductions in ICP and improved patient outcomes. The initial effects of the infusion on the ICP should be visible within 15 minutes of beginning the infusion.

Nursing Responsibilities:

- Hypertonic saline is available in 2%, 3%, 5%, 7.5%, and 23% solutions. In solutions up to 7.5%, it may be administered via continuous infusion at rates from 30 mL to 150 mL/hr, adjusted according to serum sodium levels. In acute elevations of ICP, a bolus dose of 30 mL of 23% might be given over 15 minutes.
- Any concentration greater than 2% should be administered by central line. The nurse monitors the patient’s neurological signs and ICP to determine if the dose has been effective.
- Serum sodium levels are monitored at least every 6 hours and levels are maintained within the 145 to 155 mmol/L range.
- The nurse should assess the patient’s volume status, including weight, central venous pressure, and intake and output (I&O) because patients are less likely to develop adverse side effects from hypertonic saline when they are euvolemic.
- Serum osmolality should be measured at least every 12 hours and maintained at less than 320 mOsm/L (Mortimer & Janick, 2006).
 - Hypertonic saline may be continued until 7 to 10 days after the acute injury or last episode of elevated ICP. It should be weaned rather than abruptly stopped, with the dose halved every six hours and the Serum Sodium measured every 12 hours.

Potential Side and/or Toxic Effects: The most potentially dangerous effect occurs in patients with chronic hyponatremia. Administration of hypertonic saline may result in

central pontine myelinolysis when serum sodium levels rise too quickly. There is rapid demyelination of the pons, resulting in a decreased level of consciousness and quadriplegia that may be difficult to identify in the TBI patient. Therefore, the serum sodium is monitored at least every 6 hours and not allowed to rise any more rapidly than 10 to 20 mEq/L per day. The patient with hyponatremia may be treated with 0.9% saline until the serum sodium is normal,

then therapy with hypertonic saline might be instituted (Mortimer & Janick, 2006).

If the patient develops hypernatremia (serum sodium greater than 160 mmol/L), he is at increased risk for renal failure, heart failure, and pulmonary edema. Older adults may develop these treatment-related adverse effects more readily than younger adults (Mortimer & Janick, 2006).

Cerebrospinal Fluid Drainage

CSF drainage can be accomplished through an external drainage system that is connected to an intraventricular catheter and a pressure transducer as shown in ♦ Figure 10-11. Information about the catheter and intracranial pressure monitoring is available earlier in the chapter. Unlike mannitol and hypertonic saline, CSF drainage has a negligible effect on cerebral blood flow. CSF drainage should lower intracranial pressure immediately by draining fluid from the ventricle. It also may decrease ICP on a more long-term basis by allowing edema fluid to drain into the system. A recent meta-analysis suggests that CSF drainage may not lower ICP unless the mean baseline ICP is 26 and at least 3 mL of CSF are drained (Olson, McNett, Lewis, Riemen, & Bautista, 2013). This leads to controversy about how, when, and how much drainage should occur. Some argue that CSF should be drained for a prescribed amount of time (such as 10 minutes) or a prescribed amount of volume such as 5 mL when the ICP exceeds 20 mmHg. Draining as little as 3 mL at that time may decrease the ICP 10% (McIlvoy & Meyer, 2008). Others argue that CSF should be drained almost

continuously, stopping drainage only to determine an accurate ICP or move the patient. However, when CSF is drained continuously, and the brain is diffusely swollen, the drainage of CSF may cause the ventricles to collapse.

NURSING ACTIONS

The nurse has specific responsibilities when caring for a patient whose CSF is being drained through an external ventricular drain (EVD).

- First, the transducer and stopcock must be leveled at the tragus of the ear or higher as determined by the neurosurgeon.
- If continuous drainage is being utilized, it is essential that the level be maintained as the patient moves or is repositioned.
- The amount of drainage as well as the color, clarity, and presence of sediment are noted at least hourly.
- Also hourly, the nurse accurately determines the ICP by temporarily turning off continuous drainage.
- To institute drainage in an intermittent system, the nurse turns the stopcock to allow drainage and is diligent about ensuring that CSF is drained only as prescribed. Too rapid draining of CSF can be life-threatening.
- Routine changing of CSF collection bags has been associated with an increase in infections, so collection bags are changed only when necessary.

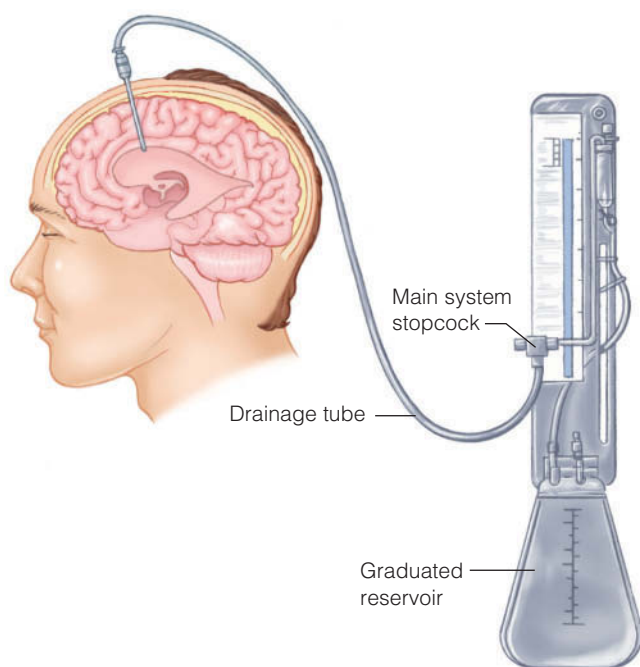


Figure 10-11 Cerebral spinal fluid drainage system.

Essential for Safety

An external ventricular drain is always maintained with scrupulous aseptic technique including the use of a mask and sterile gloves when changing the collection bag.

Hyperventilation

Hyperventilation, causing a purposeful reduction in the $p\text{CO}_2$ for the patient with increased ICP, has been associated in the long term with vasoconstriction, a decrease in cerebral perfusion, and poor patient outcomes. Thus, hyperventilation is not recommended for routine management of ICP and a low normal $p\text{CO}_2$ is preferred. While caring for a patient with increased ICP 24 hours after an

acute traumatic injury, the nurse monitors the patient's pCO₂ and attempts to maintain it between 30 and 40 (Bader, 2014). However, acute hyperventilation may be useful in neurological emergencies (AANN, 2012). Hyperventilation decreases the pCO₂, inducing vasoconstriction and resulting in a rapid reduction in ICP. Thus, when a patient has a sudden deterioration in neurological signs or acute severe elevations in ICP with herniation appearing to be imminent, short-term hyperventilation may be lifesaving. It may provide time for a CT to be performed, a pathology to be identified, and definitive therapy to be planned.

High-Dose Barbiturate Therapy

High-dose barbiturate therapy is controversial in the management of ICP but might be considered when the elevated ICP is refractory to other treatments, or when the patient has uncontrolled seizures. It most likely decreases ICP by decreasing cerebral metabolic rate, reducing cerebral blood flow, and inhibiting damage from oxygen free radicals. However, AANN (2012) states that the hypotensive effects of barbiturates most likely offset any decrease in the ICP and thus do not improve CPP. Furthermore, patients receiving high-dose barbiturate therapy are dependent on their nurses because their vital functions are depressed (Josephson, 2004).

NURSING ACTIONS

- Most patients who are placed in a barbiturate coma respond with a drop in BP sufficient to impact CPP. Therefore, hemodynamic stability is essential before high-dose barbiturate therapy is instituted. If necessary, vasopressors may be initiated to maintain an adequate BP.
- Once high-dose barbiturate therapy is instituted, it is no longer possible to perform conventional neurological assessments such as the GCS; therefore, the patient must have an ICP monitoring device in place. In addition, continuous electroencephalogram (EEG) monitoring or bispectral index monitor should be used to guide the therapy (Mcilvoy & Meyer, 2008).
- There is suppression of the ventilatory drive with high-dose barbiturate therapy. Therefore, the patient must be ventilated and carefully observed for the development of hypoventilation and hypoxia.
- Infection, specifically ventilator-associated pneumonia, is a common complication, so the nurse should institute the ventilator bundle.
- Patients often develop hypothermia; therefore, the nurse identifies the optimal temperature for the patient, monitors the patient's temperature, and initiates appropriate measures to maintain the temperature.
- Many patients develop a significant ileus that is not responsive to prokinetic agents (Bochicchio et al., 2006).

Therefore, the nurse assesses the patient carefully for the presence of bowel sounds and anticipates that it may be necessary to initiate parenteral nutrition.

- Patients often lack a corneal reflex. Therefore, the nurse must take measures to protect the patient's corneas.

Therapeutic Hypothermia or Targeted Temperature Management

Therapeutic hypothermia, also known as targeted temperature management, possibly reduces ICP by decreasing levels of excitatory neurotransmitters, free radical production, and cerebral edema. It also reduces the cerebral metabolic rate (Adamides et al., 2006). The use of hypothermia in the patient with a severe traumatic brain injury is controversial. Some believe that it should only be used in well-designed clinical trials (Georgiou & Manara, 2013), others state that it has been shown to be of benefit for patients with refractory increased ICP (Flynn, Rhodes, & Andrews, 2015). In the meantime, the critical care nurse may be providing care to a patient with therapeutic hypothermia, perhaps for a brain injury, perhaps for neuroprotection post cardiac arrest, or for intractable intracranial hypertension for some other reason such as severe liver disease.

NURSING ACTIONS

If the critical care nurse is caring for a patient with induced hypothermia:

- The goal is to attain target temperature (often 35° C) within 4 hours and to maintain it for 48 hours or longer for patients following TBI (AANN, 2012).
- The nurse should maintain two sources of core temperature and cool the patient according to an established protocol.
- Serum glucose and electrolytes must be monitored according to protocol (especially during cooling and rewarming) with special attention to potassium and magnesium.
- The patient will require sedation and analgesia and the nurse must be vigilant about preventing, identifying, and treating shivering because shivering can increase the patient's temperature and ICP.
- The nurse also assesses the patient for the following adverse effects: cardiac arrhythmias, coagulopathies, pneumonia, hypothermia-induced diuresis, electrolyte imbalances, skin breakdown, and hiccoughs (AANN, 2012).
- The nurse must rewarm the patient slowly (as per protocol) since rapid rewarming is associated with worse patient outcomes.

A more detailed discussion of managing the temperature of a patient with a TBI will be available later in the chapter.

Reflect On

Targeted temperature management has been demonstrated to improve outcomes in some post cardiac arrest patients. How does hypothermia protect the cardiac arrest survivor's brain? Why is good nursing care essential to the patient who is receiving targeted temperature management?

COLLABORATIVE CARE

Surgery to evacuate a mass lesion such as a brain tumor, an expanding hematoma, a depressed skull fracture, or an aneurysm is accepted and necessary practice for the patient with IICP. However, other surgeries are more controversial.

Decompression Craniectomy

When primary measures fail to control high ICP, second-line therapies are started. These second-line therapies include high-dose barbiturates, hyperventilation, and induction of moderate hypothermia. When these therapies have all failed, removal of a variable amount of skull bone (known as decompressive craniectomy) may be attempted (Sahuquillo & Arikan, 2006). Because opening the dura in addition to opening the skull results in further reductions in ICP, some physicians favor opening the dura and covering the dural defect with synthetic dura or pericranium (duroplasty) as well. The duroplasty leaves a space into which the intracranial volume can expand (Tazbir, Marthaler, Moredich, & Kereszles, 2005). Although such procedures decrease the ICP immediately in 85% of patients with intractably elevated ICP, there does not appear to be any long-term benefit. In fact, Cooper and associates (2011) found that patients who had craniectomies were more likely to be in a vegetative state or have a severe disability 6 months after surgery than comparable patients who had not had the surgery.

Indications for craniectomy are changing. The current indication is a continuing deterioration in neurological status despite treatment with signs of early herniation such as a unilateral fixed pupil or a dilated pupil. Once signs of late herniation such as bilateral pupil changes and extensor posturing are present, the patient is less likely to be a good candidate for craniectomy (Tazbir et al., 2005).

Postoperatively, the patient is critically ill and usually has an ICP monitoring device in place. Even with the presence of a duroplasty, CSF may still be drained if necessary. The nurse continues the methods to maintain adequate cerebral oxygenation and perfusion described previously. Placing the patient's head on the side of the craniectomy is contraindicated because it has the potential to push the cranial contents inward, increasing ICP. If there is a duroplasty, positioning the patient on that side may stress the suture line and allow a CSF leak (Tazbir et al., 2005). The nurse needs to assess the patient for development of

complications such as infection, hydrocephalus, subdural fluid collection, seizures, and CSF leak.

Prevention of Seizures

Seizure types and management will be described in more detail at the end of this chapter. However, because between 10% and 25% of patients experiencing a severe TBI develop early-onset seizures, specific concerns for these patients are highlighted now. Seizure activity in the first week following a traumatic injury may cause secondary brain damage as a result of increased metabolic demands, raised ICP, and excess neurotransmitter release. Therefore, about 60% of neurosurgeons initiate therapy with an anticonvulsant post TBI to prevent early seizures. Historically, phenytoin was the drug of choice. More recently, levetiracetam, which is equal in terms of effectiveness and adverse effects to phenytoin has gained favor (Kruer et al., 2013). (See "Commonly Used Medications" later in this chapter.) Antiseizure prophylaxis is associated with significant risks to the patient, including rashes, Stevens-Johnson syndrome, hematologic abnormalities, ataxia, and behavioral abnormalities. Some surgeons only initiate therapy if the patient is likely to experience an early seizure. Risk factors include:

- GCS less than 10
- Cortical contusion
- Depressed skull fracture
- Subdural, epidural, or intracerebral hematomas
- Penetrating head wounds
- A seizure within the first 24 hours post-injury

There is no indication that prophylactic anticonvulsants are effective in preventing late-onset seizures or in preventing disability (Schierhout & Roberts, 2012). If prophylactic therapy has been instituted, it is usually discontinued a week after the trauma if the patient has not had a seizure.

NURSING CARE

The way the critical care nurse provides care to the patient with IICP can have a pronounced effect on the ICP. The nurse intervenes to decrease ICP whenever possible, and avoids activities that have been demonstrated to increase it.

Promoting Safety: Avoiding Situations That Increase Intracranial Pressure

The patient's position has been shown to have an effect on ICP. Maintaining the head of the patient's bed at a 30-degree elevation allows adequate cerebral perfusion while promoting venous return from the head. Thus, it is the preferred position for the patient. In addition, it is important to keep the body and neck in alignment without knee elevation and to make certain that the cervical collar or endotracheal tube ties are not too tight to maintain good venous outflow (AANN, 2012).

Tume, Baines, and Lisboa (2011) reviewed the effects of bathing the patient, providing mouth and eye care as well as turning and endotracheal suctioning on ICP. They determined that, of these activities, only turning and suctioning resulted in increases in the patients' ICP. When nursing activities were delivered in a traditional cluster fashion, with one activity such as suctioning following another, the patient's ICP might rise with the first activity and continue to rise with each additional activity. However, when a patient's ICP was allowed to return to baseline before continuing with another activity (such as turning) or when a patient was adequately sedated prior to the activity, there was no clinically significant increase in ICP (Nyholm, Steffansson, Frojd, & Enblad, 2015).

When a patient engages in a Valsalva maneuver, such as when he strains when having a bowel movement or pushes himself up in bed, his ICP usually rises. Many neurosurgeons will provide orders for a variety of stool softeners or laxatives. The nurse then uses whichever is necessary to ensure that the patient has a daily soft bowel movement without straining.

Keeping external stimulation to a minimum has been demonstrated to limit the rise in ICP. This may include noxious stimuli like suctioning and turning that should be performed when necessary, but only as necessary. It also includes discussion around the patient by both the family and the healthcare team. Some studies have demonstrated a rise in a patient's ICP when discussions about the patient were conducted around him that did not include him. In contrast, family members or healthcare providers talking directly to the patient do not appear to increase ICP, and having a family member talk to the patient may actually decrease it (Olson et al., 2013).

Enhancing Comfort

The management of sedation and pain in the TBI patient is discussed in the Collaborative Care section (see page 250) of this chapter because pain management is an integral component of management of ICP.

Providing Nutrition

Patients with TBI have a metabolic rate that is increased to 120% to 240% of expected. Even if the patients are sedated or paralyzed, their metabolic needs are 100% to 120% of expected (Brain Trauma Foundation, 2007). There is evidence to suggest that patients have better outcomes when they receive full caloric replacement by the ninth day post-injury. Therefore, the Brain Trauma Foundation recommends that these patients should begin enteral feedings (either jejunal or gastric) by 72 hours post injury so that they can receive their full caloric needs by day 7. Enteral feedings are recommended because they are usually well tolerated and are less likely to be associated with infection than parenteral nutrition. Most patients with a TBI will tolerate continuous

gastric feedings if they are started at 25 mL/hr and increased by 25 mL every 12 hours until the dose that meets full caloric needs is reached. However, if a patient is unable to tolerate enteral feedings (as are most patients in barbiturate coma) then parenteral nutrition should be instituted.

Hyperglycemia, particularly glucose levels exceeding 170 mg/dL, is associated with longer hospital stays and increased mortality in the patient with TBI (McIlvoy & Meyer, 2008). A glucose level greater than 200 mg/dL that goes untreated has been related to IICP and impaired pupillary reaction. Therefore, the patient with a severe TBI injury should be closely monitored to maintain a blood glucose less than 180 mg/dL, and IV insulin might be initiated. However, there is disagreement about the most appropriate target for blood glucose. McIlvoy and Meyer state that patients who receive intensive insulin therapy to maintain their blood glucose less than 110 mg/dL have fewer seizures, less diabetes insipidus, and improved independent functioning 1 year post TBI. However, Cecil and associates (2011) believe that such intensive management can lead to dangerously low blood glucose levels and prefer a target blood glucose range of 110 to 180 mg/dL.

Maintaining Normothermia

Hyperthermia is common in patients with IICP or severe TBI. It is defined as a dysfunction of thermoregulatory mechanisms leading to an unregulated rise in temperature and is different from fever that is a response to an infection. The patient lacks the compensatory cooling mechanisms, and the elevated temperature may persist until physical cooling is instituted. Patients with increased ICP may develop hyperthermia but they may also develop fever. Unfortunately, an elevated temperature can further increase ICP by increasing cerebral metabolism, cerebral blood flow, and axonal swelling. It is associated with an increase in neurological deficits. Therefore, the critical care nurse must be vigilant in identifying and treating hyperthermia and fever in the TBI patient. It is advantageous to maintain a brain temperature between 36°C and 37°C (96.8°F and 98.6°F). Some centers measure brain temperature directly, but many rely on core temperatures through pulmonary artery (PA) catheters and bladder or rectal probes. A common conversion is that the PA temperature is usually 0.4°C less than the brain temperature, whereas rectal and bladder temperatures may underestimate the brain temperature by 0.1°C to 2°C.

There is no standard determination of when to initiate management of an elevated temperature. Thompson, Kirkness, and Mitchell (2006) noted that some authorities recommend initiating therapy at 37.5°C (99.5°F), yet the policies in most institutions do not call for treatment until the temperature exceeds 38.5°C (101.3°F). However, what Thompson and colleagues found to be most discouraging was that despite institutional policies and the known

hazard of an elevated temperature in patients with IICP, only 5% of patients in their study were normothermic. There were documented nursing interventions to decrease an elevated temperature identified in only 31% of the patients. In 2015, Rockett, Thompson, and Blissitt found that circumstances had barely improved, only 27% of hospitals had policies for maintaining normothermia in patients with neurological injuries and the temperatures at which nurses began to treat hyperthermia as well as the means they used to decrease temperature varied widely.

It is the responsibility of the critical care nurse to initiate management of an elevated temperature at the appropriate time. Treatment modalities include both antipyretics and physical means of cooling. Interventions to reduce fever should be assessed in terms of potential risks. Acetaminophen is the most commonly used antipyretic in patients with TBI (Rockett et al., 2015). It is inconsistently absorbed in critically ill patients with administration via nasogastric tube, often resulting in higher levels of the drug. Repeated therapy at recommended doses can result in drug accumulation, and hepatotoxicity has developed in some patients. Antipyretics are ineffective in those patients who have a disorder of the thermoregulatory system, and physical cooling may be needed.

Holtzclaw (2002) stated that physical cooling is justified when the patient's thermoregulatory mechanisms are impaired or when the patient has neurological damage. Treating hyperthermia preserves brain function because the metabolic needs of the brain are reduced by about 10% for each degree Celsius the patient's temperature is lowered. Patients may be cooled by convective airflow blankets, infusions of cool fluids, or indwelling cooling catheters such as Setpoint. However, external cooling (hypothermia) blankets are still commonly utilized to cool patients.

Of particular concern when instituting physical cooling is the potential for the patient to shiver. Shivering causes an increase in the metabolic rate of 100% to 200% and increases body temperature. Thus, it should be prevented. According to Holtzclaw (2002), in order to prevent shivering, the nurse might:

- Use higher temperatures on the cooling blanket (as high as 23.9°C [75°F]) so the patient's temperature does not drop as rapidly.
- Wrap the patient's arms and legs in cloth towels and position them so they are off the blanket.
- Place a bath blanket between the patient and the hypothermia blanket to prevent burning the patient's skin.
- Medicate the patient. The agents that have been shown to most effectively reduce shivering are clonidine, dexmedetomidine, and meperidine, which may be administered alone or in combination (Weant, Martin, Humphries, & Cook, 2010).

Essential for Evidence-Based Practice

The nurse must monitor the patient closely for the development of hypotension, especially when clonidine, dexmedetomidine, and/or meperidine are used in combination to prevent shivering.

Most patients are also treated with an antipyretic. According to Axelrod and Diringer (2006), if seriously ill febrile patients are externally cooled and sedated or paralyzed to suppress shivering, they may have a more rapid reduction of fever than if treated with antipyretic medications alone. The utilization of such an external cooling blanket requires continuous, accurate assessment of the patient's core or brain temperature. Usually the blanket is turned off when the patient's temperature is about 1°C above the desired temperature to allow the temperature to drift toward the goal.

Facilitating Communication

Family members and proxy decision-makers of patients with severe TBI define the situation as very stressful. It is so stressful for relatives serving as healthcare proxies that a study by Pielmaier and others (2011) found more than half of them experienced post-traumatic stress shortly after their relative's accident. Family members describe the experience as "waiting to wake up from a nightmare." During this time, families are unable to integrate the bad news and need to recount the story in great detail repeatedly before it seems to be real (Keenan and Joseph, 2010).

Lefebvre and Lever (2006) found that most family members believed the way they were provided the "bad news" had a significant impact on their adjustment. They reported that information was often insufficient, imprecise, or simply not provided. They believed they "endured long waits, were left to their own devices, and received only a trickle of information" (p. 714). Healthcare providers in the same study, although acknowledging the feelings of the family, stated they believed that part of the problem was related to the families' state of shock and inability to recall information. Moreover, the problem was worsened by the uncertainty of the prognosis and the healthcare providers not knowing what to say. They told Lefebvre and Lever (2006) about their "fear of being wrong; they were afraid of upsetting the family further or giving them false hope" (p. 715).

Nursing Diagnoses

Patient Experiencing an Intracranial Dysfunction

- Decreased intracranial adaptive capacity related to brain injury
- Ineffective tissue perfusion: cerebral related to increased intracranial pressure and decreased cerebral perfusion pressure
- Acute pain related to trauma

- Risk for aspiration related to reduced level of consciousness and depressed cough and gag reflexes
- Risk for injury related to seizure disorder

Reflect On

Have you cared for a patient with a brain injury whose prognosis was unclear? What types of questions did the family ask about the patient's prognosis? How did you respond? Would you respond in the same way in a similar situation? What would you do differently and why?

Marks and Daggett (2006) developed a critical pathway for meeting the needs of families of patients with severe TBI. It begins with health information and management. On the first day, the family meets with the physician and nurse to discuss the patient's injury and plan of care. At that time, a primary nurse contact and family contact person are identified. As the ICU stay progresses, the primary nursing contact is responsible for discussing new topics as the family expresses a need to know.

The next aspect of family care identified in the pathway is provision of emotional support. Throughout the patient's stay, all members of the staff are encouraged to answer questions, explain equipment, and assess for and reinforce positive coping behaviors. Contacts are made with the chaplain and social services as appropriate.

Another aspect of family care identified in the pathway is family involvement in patient care. As the patient stabilizes, the nurse demonstrates appropriate ways the family can touch and speak to the patient. After the patient has fully stabilized, the family, if willing, may be taught how to deliver oral hygiene, assist with turning, assist with physical therapy, or provide coma stimulation. Keenan and Joseph (2010) noted that parents of young adults and wives of patients were likely to be extremely involved in care of their loved one, saying that involvement allowed them to feel that they were able to help in their loved one's recovery.

Institution of Coma Stimulation

Coma stimulation is a treatment (such as an auditory, a tactile, an olfactory, or a gustatory stimulus) that is varied in intensity and frequency in an attempt to increase the patient's arousal and awareness (Gerber, 2005). In the past, it was not attempted until rehabilitation, but now it may be started in the critical care unit after 72 hours if the patient is stable. According to Gerber, it is important that the stimulation be provided when the patient has just completed a sleep/rest period, the room is uncluttered and quiet, and the patient is stable. The stimulation might be as simple as providing mouth care, changing position, or providing the patient with his favorite type of music. During the stimulation, only one person should be speaking at a time and

only one mode of stimulation provided at a time. The patient's family can become involved in coma stimulation by stroking the patient's hand, brushing his hair, and bringing in familiar objects from home. A Cochrane Review by Lombardi, Taricco, De Tanti, Telaro, and Liberati in 2002 determined that there is no reliable evidence to support, or rule out, the effectiveness of multisensory programs in patients in a coma or vegetative state.

Complications

Patients Develop a Wide Range of Complications Post TBI

Pulmonary Complications: Pneumonia and Adult Respiratory Distress Syndrome. Aspiration resulting in pneumonia occurs in as many as 40% of patients following TBI, usually between the second and fourth day post injury. Aspiration may occur immediately after the injury if the patient has been vomiting, or it may happen during intubation. This is an additional reason for the use of rapid sequence intubation in the head-injured patient. When the patient is ventilated, in order to prevent ventilator-associated pneumonia, the nurse institutes the ventilator bundle.

ARDS may develop following pneumonia or excessive fluid volume replacement. At one time, it was recommended that a patient with a TBI have a CPP greater than at least 70. To achieve this goal, patients received large amounts of volume to increase their MAP. Although this did not improve their cerebral function, it did increase their likelihood of developing ARDS and is therefore no longer an accepted mode of management.

Deep Vein Thrombosis. Patients with severe TBIs are at an elevated risk for deep vein thrombosis (DVT) because many of them have abnormalities in coagulation. Additional risks for DVT are common and include immobility in the acute care setting, being ventilated for longer than 3 days, paralysis, being obese, and concurrent orthopedic injuries. The result is that DVT occurs in 7% to 20% of patients during their rehabilitation (Carlile et al., 2006). One of the major concerns is that the patient who develops a DVT while immobile during acute care hospitalization will have a pulmonary embolus when mobilized in rehabilitation.

The Brain Injury Foundation (2007) recommends the use of intermittent pneumatic compression stockings until the patient is ambulatory unless prevented by lower extremity injury. Low molecular weight and fractionated heparin have been used in conjunction with stockings, but they increased the risk of bleeding. Therefore, there are no guidelines for their use in patients with TBI at this time. The critical care nurse should remember that the prevention of DVT is essential in the care of the TBI patient and institute the use of compression stockings or boots as soon as possible.

Essential for Evidence-Based Practice

Research has shown that even when patients have intermittent pneumatic compression stockings, they are often in place and in use for only 50% to 70% of the time. Given the potential for DVTs in the patient with increased intracranial pressure, this is not adequate.

Sodium Imbalances. Disturbances of the pituitary with either profound deficiencies of antidiuretic hormone (ADH), diabetes insipidus (DI), or excessive release of ADH—syndrome of inappropriate ADH (SIADH)—may develop. With the absence of ADH that occurs in DI, the patient excretes large volumes of dilute urine, and the serum sodium increases. It is more likely to develop following pituitary surgery or in patients with such severely elevated ICP that they are likely to suffer from brain death and be considered as transplant donors.

SIADH is excessive release of ADH from the pituitary that occurs following severe head trauma, a brain tumor, or meningitis. The patient excretes excessive amounts of ADH, too much water is reabsorbed, and the intravascular volume expands. The excessive intravascular water results in a relative deficiency of serum sodium. The nurse carefully

monitors the patient's hydration status and serum sodium. Treatment might be fluid restriction or careful sodium replacement.

Recovery

There are a variety of factors that may predict a patient's recovery during the first few weeks and months after a TBI. These include the duration and severity of the coma (as measured by the GCS), the duration of post-traumatic amnesia, the location and size of contusions and hemorrhages in the brain, and the severity of other injuries.

Recovery after brain injury results from several different mechanisms. Initially, the patient may show improvement as cerebral edema decreases. Next, over a period of several weeks to months, damaged brain cells may begin to function again. Finally, the patient may develop new pathways, and undamaged areas of the brain may take over some of the functions of permanently damaged areas. It is very difficult to predict any specific patient's precise recovery. In general, the more severe the patient's injury, the longer the recovery period, and the more likely the survivor will have a neurological impairment. Also, the longer a patient remains unresponsive, the longer the recovery time.

Safety Initiatives

Reducing Surgical Complications: Venous Thromboembolism (VTE) Prophylaxis

PURPOSE: To reduce the incidence of deep vein thrombosis (DVT) and subsequent pulmonary embolism in hospitalized patients

RATIONALE: If prophylaxis is not provided to surgical patients, between 10% and 40% of them will develop a DVT. Surgical patients are at increased risk for DVT because of immobility, pain, and the effects of pain-relieving medications. Nursing assessments for the common manifestations of DVT have been shown to be unreliable in the early stages. If a pulmonary embolism results from the DVT, there is the potential for significant patient morbidity and instantaneous death. In one study, 32% of patients who died suddenly during the first 30 days postoperative died from a pulmonary embolism. Therefore, prophylaxis for all surgical patients is a prudent course of action.

HIGHLIGHTS OF RECOMMENDATIONS:

- Develop standard order sets for DVT prophylaxis, which might include the following elements:
- A plan for active and passive lower extremity activity, including flexion and extension of the ankle, knees, and hips unless contraindicated.
- Early and aggressive ambulation unless contraindicated.
- Graduated compression stockings or intermittent pneumatic compression devices, which should be worn at all times when the patient is inactive except during skin care and bathing. They should be measured to ensure correct fit. Foot pumps might be used instead but these also should be worn whenever the patient is inactive (which includes when the patient is resting in a chair or away from the unit for a test).
- Anticoagulant therapy per specific protocol or physician order.
- Develop protocols for providing prophylaxis automatically, based on the surgical procedure.
- Provide education and training for staff on the importance of VTE prophylaxis.
- Educate patients preoperatively about the prophylaxis they will receive and steps they can take to reduce risk.

Source: Institute for Healthcare Improvement: Getting Started Kit: Reduce Surgical Complications (2008); and National Guideline Clearinghouse: Venous Thromboembolism Prophylaxis (2012).

Bariatric Considerations

DVT and pulmonary embolism are the most common complications in obese patients following any type of surgery, even when sequential compression devices and enforced ambulation are components of standard post-operative care.

When sequential compression devices are used for the obese patient, it may be necessary to combine two devices with a Velcro binder to surround the patient's extremity.

There is no consensus on optimal anticoagulant dosing using either low molecular weight heparin or unfractionated heparin in surgical patients who are morbidly obese (King & Velmahos, 2010). However, in general, higher doses are required in the obese patient to attain DVT prophylaxis.

Primary Causes of Increased Intracranial Pressure: Meningitis and Seizures

Meningitis

Meningitis is inflammation of the meninges and the underlying subarachnoid space that contains the CSF. Depending on the development and duration of symptoms, meningitis may be classified as acute, subacute, or chronic. Acute meningitis is particularly dangerous because in less than a day a patient may become severely ill with signs and symptoms of meningeal inflammation and systemic infection. The patient can decompensate very quickly and require emergency care, including antimicrobial therapy within minutes of ED arrival to survive. Fortunately, 75% of patients present subacutely. Although they still require urgent ED diagnosis and care, there is time for treatment. Chronic meningitis, the least common type, characteristically develops over several weeks. Although meningitis may be infectious or noninfectious, this section focuses on acute bacterial meningitis, the most common type.

Risk Factors

Common risk factors include:

- Circumstances that promote exposure to infectious organisms such as crowded living conditions and dormitories
- Presence of infections such as otitis media, sinusitis, or dental abscess
- Neurological injury, surgery, or invasive procedure but especially penetrating head trauma or a basal skull fracture with a dural tear
- IV drug use, especially if it results in endocarditis
- Immunocompromise and alcohol misuse

Pathophysiology

Bacteria bypass the usual protective barriers, gain access to the central nervous system (CNS), and cause disease in several ways. First, bacteria may colonize or establish a localized infection in the host, usually in the nasopharynx. From this site, the organisms gain access to the CNS via the bloodstream or by spreading from adjacent structures such as the sinuses. Bacteria may also enter through a hole in the dura following trauma, surgery, or an invasive procedure. Once in the CSF, infectious agents survive and flourish because host defenses such as antibodies and white blood cells are not abundant there. Replicating bacteria and increasing numbers of white cells account for the characteristic changes in CSF cell count, protein, and glucose. Exudates extend throughout the CSF, damaging cranial nerves such as VIII, resulting in hearing loss; obstructing CSF pathways, causing hydrocephalus; and inducing vasculitis, causing localized brain ischemia.

As ICP continues to rise and brain edema progresses, autoregulation of cerebral blood flow is impaired and blood flow begins to fall. Without medical intervention, a cycle of decreasing cerebral perfusion, cerebral edema, and increasing ICP continues unchecked (Razonable, 2011). Septic shock may develop, which also impairs cerebral blood flow, and the patient can die from systemic complications or from diffuse CNS ischemic injury. Morbidity and mortality depend on the infecting pathogen, the patient's age and condition, and how quickly the patient receives treatment. When an appropriate antibiotic is not provided within 48 hours of the onset of symptoms, the patient's outcome is more likely to be unfavorable (Dzupova, Rozsypal, Prochazka, & Benes, 2009). Before the widespread use of HiB vaccine, *H. influenzae* meningitis was the most common cause of bacterial meningitis. Now it represents less than 7% of all cases. The rate for *N. meningitidis* has remained constant at 14% to 25%, accounting for most of the cases among 2- to 18-year-old patients. It is carried in the nasopharynx of otherwise healthy individuals and initiates invasion by penetrating the airway

epithelial surface. *S. pneumoniae*, accounting for 47% of all cases, is now the most common cause of meningitis in all age groups. It colonizes the human nasopharynx in 5% to 10% of healthy adults and causes meningitis by escaping the local host defenses and seeding the CSF through bacteremia or through direct extension from sinusitis or otitis media. It is the most common cause of meningitis in patients with basilar skull fractures and CSF leaks. Pneumococcal meningitis has the highest rates of mortality (21%) and morbidity (15%).

Manifestations

The classic presentation of meningitis includes fever; headache; neck stiffness; photophobia; nausea; vomiting; and changes in mental status such as irritability, lethargy, confusion, and coma. However, this constellation of symptoms may not develop until 13 to 22 hours after the onset of the illness. Other indications of meningitis or patterns of symptoms may include:

- The development of limb pain and cold hands and feet with mottled skin which occurs in 72% of children 4 to 8 hours after the onset of the illness (Perrin & Glennie, 2008).
- The triad of fever, neck stiffness, and change in mental status is found in two thirds of patients.
- Signs of meningeal irritation are observed in approximately 50% of patients with bacterial meningitis. They include:
 - Nuchal rigidity: the patient experiences resistance and pain with passive flexion of the neck.
 - Kernig's and Brudzinski's signs, which have a sensitivity of only 5% but a specificity of 95%. That means that their absence does not rule out the occurrence of meningitis, but their presence indicates that the patient is highly likely to have meningitis (Ward, Greenwood, Kumar, Mazza, & Yale, 2010).
 - To elicit Kernig's sign: In a supine patient, the nurse begins with the patient's hip flexed to 90 degrees while the knee is flexed at 90 degrees. Extending the knee produces pain in the hamstrings and resistance to further extension.
 - To elicit Brudzinski's sign: With the patient supine and extremities extended, the nurse passively flexes the patient's neck. The patient responds with flexion of the hips when there is meningeal irritation.
- Cranial nerve palsies may be observed as exudates encase the nerve roots. The most common findings are deafness associated with cranial nerve VIII and defects of cranial nerves IV, VI, and VII.

- Focal neurological signs may develop as a result of ischemia from vascular inflammation and thrombosis.
- Seizures occur in approximately 30% of patients.
- Papilledema and other signs of increased ICP may be present but usually take several hours to develop.

Assessment

The development of acute bacterial meningitis is a medical emergency. Therefore, many EDs have triage protocols to identify patients at risk. When a patient complains of the symptoms and displays the classic signs of meningitis on focused neurological assessment, the healthcare team should ensure that the patient bypasses the usual history taking and proceeds immediately to definitive diagnosis and emergent management.

Diagnostic Criteria. If acute bacterial meningitis is suspected, the physician performs a lumbar puncture urgently. Examination of the CSF in patients with acute bacterial meningitis reveals the following:

- Cloudy or even purulent appearance
- White blood cell count of 1,000 to 10,000 with more than 80% polymorphonuclear cells
- Glucose less than 40 mg/dL in 60% of patients (normally 40 to 70 mg/dL)
- Elevated protein level (normally 20 to 50 mg/dL)

CSF is sent immediately for Gram stain, culture, and sensitivity. The Gram stain permits rapid identification of the bacterial cause in 60% to 90% of patients, allowing treatment to begin with an antibiotic that is likely to be effective. CSF bacterial cultures and sensitivities are also sent. They identify the bacterial cause in 70% to 85% of cases and ensure that the most appropriate antibiotic therapy is provided (Razonable, 2011).

Additionally, cultures of the blood, urine, nose, and throat may be sent to assist in identifying the appropriate pathogen. A complete blood count (CBC) is sent, which usually reveals leukocytosis. If a focal neurological deficit is present, a CT scan is often obtained.

COLLABORATIVE CARE

The goal of care is provision of an appropriate antibiotic, possibly in conjunction with steroids, within 30 minutes of identification of acute bacterial meningitis. When patients are treated aggressively, and CSF is sterilized rapidly, the signs and symptoms may resolve completely. Ideal antibiotic therapy is based on a clearly identified organism on CSF Gram stain. However, if the CSF obtained from the LP is bloody or turbid, empiric antibiotic therapy based on the patient's age and the known predisposing factors is

started as soon as possible (Caple & Walsh, 2015). This is necessary because delays in instituting antibiotic treatment in individuals with bacterial meningitis can lead to significant morbidity and mortality. Once the pathogen has been identified and susceptibilities determined, the antibiotic(s) may be modified for optimal treatment (Razonable, 2011).

Steroids are currently recommended as adjunctive treatment of bacterial meningitis. Dexamethasone is believed to interrupt the neurotoxic effects resulting from the lysis of bacteria during the first days of antibiotic use. Therefore, when steroids are given, they should be administered prior to or during the administration of antibiotics on the first 2 days of therapy.

NURSING CARE

Patients with *N. meningitidis* will require isolation for infection control. Prophylaxis is recommended for close contacts of a patient with *N. meningitidis* or *H. influenzae* type B meningitis. People who have been in contact with the person with bacterial meningitis should be instructed to notify a healthcare provider or ED immediately at the first sign of fever, sore throat, rash, or symptoms of meningitis. In addition, some forms of meningitis must be reported to state and federal health agencies.

Promoting Safety: Avoiding Situations That Increase Intracranial Pressure

The nurse performs a baseline neurological assessment, including at least GCS, pupillary response to light, motor activity, hearing, and the presence of seizure activity. The assessment is repeated hourly at first and compared to the baseline to detect any changes. If the patient is relatively stable, elevating the head of the bed to 30 degrees, controlling straining at stool, preventing coughing, and avoiding systemic hypotension by providing adequate volume will assist in preventing increases in ICP. For patients who are less stable, refer to the section on managing ICP in the trauma patient earlier in this chapter for additional management strategies.

Enhancing Comfort

Relief of pain is also important in preventing increases in ICP. Patients often describe the headache as initially severe and getting progressively worse before treatment. Elevating the head of the bed may help to decrease the headache. Short-acting, reversible analgesic agents are preferred for pain relief.

Photophobia may be pronounced and can be a major source of discomfort for these patients. Therefore, they

should be placed in a darkened room or a cloth is placed over their eyes.

Maintaining Normothermia

Fever develops in almost 90% of patients with acute bacterial meningitis, and the absence of fever predicts a poor outcome (Fernandes et al., 2014).

Therefore, the nurse assesses the temperature of a patient with meningitis at least every two hours. Antipyretics such as acetaminophen can be utilized initially for fever. If an appropriate antibiotic is provided and an antipyretic administered, the fever may respond quickly. Normothermia (37°C) is the goal, since patients with meningitis who present with, develop, or are treated with hypothermia have poorer outcomes (Fernandes et al., 2014; Caple & Walsh, 2015).

Common and Life-Threatening Complications

Complications from meningitis can be severe. Hearing loss is the most commonly reported complication, occurring in 10% of patients surviving meningococcal meningitis and 26% of people who survive pneumococcal meningitis (Blewitt, 2009). Hearing loss may be unilateral or bilateral and may range from mild to profound. During the acute phase of meningitis, approximately 30% of patients with bacterial meningitis develop seizures. If a seizure occurs, it is treated aggressively because seizure activity increases ICP. One recommended management strategy is lorazepam 0.1 mg/kg IV.

Two potentially life-threatening complications that are associated primarily with meningococcal meningitis are Waterhouse-Friderichsen syndrome and disseminated intravascular coagulation (DIC). Waterhouse-Friderichsen syndrome is an adrenal hemorrhage that results in adrenal insufficiency, hypotension, and circulatory collapse. It is treated with immediate replacement of adrenal corticosteroids and supportive therapy.

Seizures

Seizures are defined as “stereotyped behavior associated with electrographic abnormalities in the EEG” (Kohrman, 2007).

Overview

When a seizure occurs, the electrical system of the brain malfunctions. Instead of discharging electrical energy in an orderly manner, the brain cells fire repeatedly and haphazardly. If the entire brain is involved at once, the surge of energy may cause a loss of consciousness and massive contractions of the muscles. If instead only part of the brain is affected, the seizure may present as any activity that the brain can control. It may cloud awareness; block

normal communication; or produce a variety of undirected, uncontrolled, unorganized movements (Cavazos, 2011). Most seizures last for only a minute or two. However, the person may remain confused and drowsy for a period of time afterward (postictal phase). Epilepsy is the occurrence of at least two seizures without an identifiable cause. Epilepsy is defined as a “brain disorder characterized by an enduring predisposition to generate epileptic seizures and by the neurobiologic, cognitive, psychologic, and social consequences of this condition” (Cavazos, 2011).

Predisposing Conditions or Risk Factors

In the United States, approximately 300,000 people have a first convulsion each year. Many of these people will have a single, one-time seizure from an identifiable cause (also known as a provoked seizure). These people may require treatment of the specific cause at the time but usually do not require ongoing anticonvulsant medication. According to Cavazos (2011), it is not surprising that provoked seizures are common since the main function of the brain is the transmission of electrical impulses. Treatable causes of seizures include:

- Hyponatremia/hyponatremia
- Hyperglycemia/hypoglycemia
- Hypercalcemia/hypocalcemia
- Hypomagnesemia
- Hypophosphatemia
- Meningitis
- Toxins: pertussis, salmonella, shigella

Drug or alcohol intoxication or withdrawal (Kohrman, 2007)

There are a variety of other identifiable causes of seizures as well. Other known causes of seizures include:

- Hypoxia
- Brain tumors
- Fever
- Head injury

The risk for development of epilepsy following a single seizure varies between 15% and 80%. A patient who had a seizure with no focal characteristics, a normal EEG, and brain MRI post seizure has a likelihood of 15% while a patient who had a seizure with focal findings, an abnormal EEG, and abnormal MRI post seizure would have a likelihood of 80% (Cavazos, 2011). Currently there are about 2.7 million people in the United States with active epilepsy. The prevalence increases with age, and there are about 570,000 persons over the age of 65 with epilepsy.

Pathophysiology and Manifestations

Seizures result from a spontaneous electrical discharge initiated by highly excitable cortical neurons. This increased electrical activity may occur in response to a stimulus such as dehydration, fatigue, flashing lights, hypocarbia, emotional stress, or monthly hormonal fluctuations. The neuronal excitement may remain in one site (focal or partial), spread slowly (secondarily generalize), or generalize immediately at the onset. The clinical signs and symptoms of a specific seizure depend on the location and extent of the spread of the discharging neurons. Seizure classifications are displayed in Box 10-2.

Box 10-2 Classification of Seizures

- I. Partial seizures
 - A. Simple partial seizures: No change in the level of consciousness
 1. With motor signs
 - a. Focal (may occur with march [Jacksonian] or without march)
 - b. May be versive, postural, or phonatory
 2. With somatosensory or special-sensory symptoms such as visual, auditory, olfactory, gustatory, or vertiginous
 3. With autonomic symptoms or signs
 4. With psychic symptoms such as illusions, or structured hallucinations
 - B. Complex partial seizures: Impairment of consciousness may develop after the seizure begins or at the onset of the seizure
- C. Partial seizures evolving to secondarily generalized seizures
 1. Simple partial seizures evolving to generalized seizures
 2. Complex partial seizures evolving to generalized seizures
 3. Simple partial seizures evolving to complex evolving to generalized seizures
- II. Generalized seizures
 - A. Absence seizures
 1. Typical absence seizures
 2. Atypical absence seizures
 - B. Myoclonic seizures
 - C. Clonic seizures
 - D. Tonic seizures
 - E. Tonic-clonic seizures
 - F. Atonic seizures

Partial seizures are the most common type of seizures. Critical care nurses commonly see focal partial seizures after a patient has had a head injury, stroke, or brain surgery. The nurse might notice that the patient's hand was twitching rhythmically. If the seizure then progressed up the arm, along the side of the body, and finally encompassed the entire body, it would be a secondarily generalized seizure. Other types of complex partial seizures might be manifested by lip smacking or ritualized hand movements similar to knitting, which typically last for 60 to 90 seconds and are followed by postictal confusion. Consciousness is impaired during a complex partial seizure but it might be difficult for the critical care nurse to determine that. One way to determine this is if the nurse asks the patient if she remembers the event, the patient might respond that she remembers the aura but not the entire event. Cavazos (2011) states that when the patient does not recall an aura, the event is more likely to have been serious.

The most recognizable primary generalized seizure is the tonic-clonic seizure. Tonic-clonic seizures, also referred to as grand mal seizures, consist of several motor behaviors. The first is generalized tonic extension of the extremities lasting for 10 to 20 seconds, followed by clonic rhythmic movements lasting 20 to 30 seconds then prolonged postictal confusion (♦ Figure 10-12). Unlike secondarily generalized seizures, the patient experiencing a primarily generalized seizure does not recall experiencing an aura. However, the seizure and aura have characteristic findings on the EEG.

The critical care nurse is less likely to notice the other common type of generalized seizure, an absence seizure, which is a period of impaired consciousness that lasts for approximately 20 seconds but the EEG shows similar abnormalities to those shown in tonic seizures. Myoclonic seizures are less common, brief jerking motions that last less than one second and cluster together. They are similar

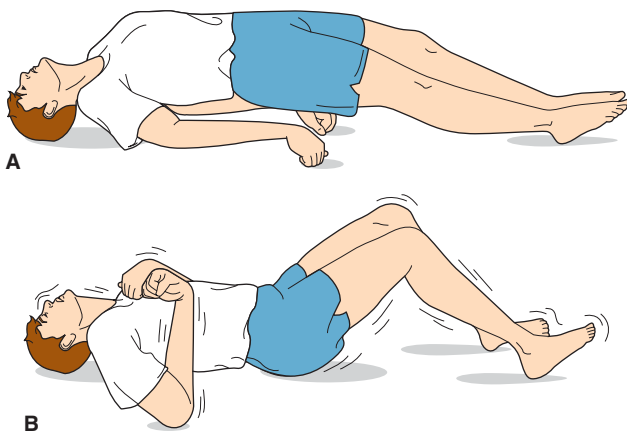


Figure 10-12 Tonic-clonic seizures in grand mal seizures. A, Tonic phase. B, Clonic phase.

to myoclonic jerks that occur as a person is falling asleep. Atonic seizures occur primarily in patients with significant neurological injuries, and are manifested as brief losses of postural tone that can result in falls and injuries.

Assessment with Intervention upon Discovery

If the nurse observes a seizure, then her first priority is to intervene to protect the patient and prevent aspiration. This usually involves:

- Lowering the patient to the floor or bed if he is not in a bed.
- Cushioning the patient's head.
- Loosening any tight clothing.
- Padding or removing objects that the patient might strike.

Essential for Safety

The nurse does not insert a tongue blade, restrain the patient, or interfere with the course of the seizure.

During the minute or two that the seizure lasts, the nurse observes the seizure. While the nurse is observing the seizure, or if the nurse is obtaining a history from a witness after the seizure, the nurse considers the following:

- Was there any warning that a seizure might occur? If so, what kind of warning occurred (e.g., Did the patient have a sensory aura?)?
- Was there any trigger (e.g., Was the patient looking at flashing lights?)?
- What did the patient do during the seizure? Was there a tonic phase? Were there rhythmic contractions of muscles? Where did they start? How did they progress?
- Was the patient able to relate to the environment during the seizure and/or does the patient remember the seizure?
- How long did each phase of the seizure last?

Once the seizure ends, the nurse may turn the patient on his side and make sure that the patient starts breathing. She checks for a return to consciousness and determines the patient's orientation. The patient's vital signs including temperature and oxygen saturation are obtained. The nurse should also determine:

- How the patient felt after the seizure.
- If the patient was incontinent or drooling.
- How long it took for the patient to get back to his baseline condition.

If the seizure does not end after a minute or two, or if it occurs in a patient post-head surgery or brain injury, it will

require aggressive management. Management will be described later in the chapter.

When the nurse is obtaining a history from the patient or family member, the nurse should ask:

- Has this ever happened before?
- If so, how frequently do these seizures occur?
- What has the patient been doing for the seizures and has he shown any response to therapy?

Diagnostic Criteria

A single generalized seizure that follows a pattern familiar to the patient and terminates without treatment may not require any diagnostic follow-up. When a patient with a known seizure disorder has seizures with increasing frequency, follow-up may involve determining the blood level of the patient's antiseizure medication. However, when a patient has a first seizure or develops status epilepticus, there is usually a diagnostic workup to determine the cause of the seizure.

- Laboratory studies may be performed to check for:
 - Any of the electrolyte imbalances described previously that can cause treatable seizures (particularly sodium imbalances).
 - Hypoglycemia (a blood glucose is obtained as soon as possible).
 - Anticonvulsant levels in a patient with a history of seizures.
 - Levels of alcohol and drugs of abuse.
- For patients with new-onset seizures, noncontrast CT is indicated because it quickly detects any brain lesion or the presence of acute hemorrhagic stroke.
- An electroencephalogram (EEG) is obtained to evaluate the brain's electrical activity. When the number of discharging neurons exceeds several million, then the seizure can usually be detected with electrodes placed on the patient's scalp (Cavazos, 2011).
- A lumbar puncture (LP) is indicated if CNS infection is suspected.

COLLABORATIVE CARE

Recurrent unprovoked seizures are usually managed with an anticonvulsant. However, if the patient has had only a single unprovoked seizure, the likelihood of him having another is small. Therefore, anticonvulsants are not recommended for a single seizure and the patient is told to avoid triggers such as sleep deprivation, flashing lights, and dehydration.

Seizure control is the key to quality of life for a patient with recurrent seizures (Kohrman, 2007). Approximately 70%

of patients with epilepsy will become seizure-free with an appropriate anticonvulsant, and will remain seizure-free even after they are weaned from the medication. Weaning usually begins if the patient has been taking medication for 2 to 5 years and is seizure-free. An additional 10% to 15% of patients with epilepsy will remain seizure-free while continuing to take medication. However, between 15% and 20% of people will continue to have seizures despite appropriate, even aggressive, anticonvulsant therapy.

The goal of anticonvulsant therapy is to attain seizure control with a minimum of side effects and optimum quality of life (Rosenberg, 2007). General principles of seizure control include:

- Use of a single anticonvulsant (also known as monotherapy) to minimize side effects and avoid drug interactions
- Once a day dosing (if possible) to enhance patient compliance
- Slow increases in the dose of the medication until control of seizures is achieved or the patient experiences intolerable side effects (Kohrman, 2007)

Anticonvulsants

The type of seizure and the specific epileptic syndrome play a role in the selection of the initial anticonvulsant. Antiseizure medication recommendations for long-term therapy of specific types of seizures include the following:

- Valproic acid is considered first-line therapy, with topiramate or lamotrigine as second-line therapy for primary generalized tonic-clonic seizures; levetiracetam may be adjunctive therapy.
- Carbamazepine and lamotrigine are first-line therapy for partial-onset seizures
- Ethosuccimide is first-line therapy if the patient is only having absence seizures, valproic acid is utilized if other types of seizures are also present.

However, the anticonvulsant phenytoin is often utilized in critical care because it is effective against both generalized and partial-onset seizures and is one of the most effective drugs for treating acute seizures and status epilepticus. Levetiracetam is gaining favor in critical care for use in complex partial seizures, prevention of seizures post TBI and SAH, and as adjunctive therapy in status epilepticus.

Ketogenic Diet

A ketogenic diet or modified Atkins diet may be utilized to prevent seizures. They are high-fat diets consisting of four parts fat to one part protein and carbohydrate. The diets may work because ketogenesis results in the production of

an endogenous anticonvulsant and is about as effective as standard anticonvulsant therapy. They are primarily used in children and young adults. The anticonvulsant effect occurs within days to weeks with about 30% of patients having at least a 50% reduction in seizures (Klein, Tyrlikova, & Mathews, 2014). However, half of patients stop the diet before its completion due to the complexity, restrictiveness, and adverse effects. Adverse effects include hyperlipidemia, constipation, kidney stones, fractures, and slowed growth in children.

Invasive and Surgical Management

A patient has medically refractory epilepsy when three different anticonvulsants have failed to control the patient's seizures despite attaining adequate serum concentrations. If another anticonvulsant is attempted in the patient with medically refractory epilepsy, the likelihood of success is less than 5%. Therefore, such a patient may be referred for possible invasive or surgical management.

Invasive management might be implantation of a programmable vagus nerve stimulator. It intermittently stimulates the vagus nerve, resulting in altered brain excitability

and decreasing the frequency and severity of seizures. It is similar to anticonvulsants in its ability to prevent seizures, and the stimulator may be activated by a magnet if the patient senses a seizure is about to occur. Problems are infections at the implantation site, voice changes when the device is activated, and, in rare circumstances, bradycardia or asystole.

Current surgical procedures are safe and effective (Kohrman, 2007). Preoperatively, the epileptogenic zone is usually mapped using video-EEG monitoring with intracranial electrodes. Mapping of motor, sensory, and speech areas is also performed. Several curative surgeries are possible, including lobectomy, lesionectomy, hemispherectomy, and callosotomy. Outcomes of temporal-lobe surgeries are better than those for surgeries in other areas. If a patient has unilateral temporal-lobe seizures, the likelihood of no seizures at 2 years is 85% (Cavazos, 2011). After surgery, the nurse performs regular neurological assessments, paying particular attention to visual field defects and mild memory deficits in the patient who has had a temporal lobectomy and paresis or urinary incontinence in the patient who has had a callosotomy.

Commonly Used Medications

Phenytoin (Dilantin)

Phenytoin is an anticonvulsant that works by blocking the repetitive action of the sodium channel.

Desired Effect: Phenytoin is used to control and prevent tonic-clonic and psychomotor seizures. It has a long half-life, so a loading dose is utilized when beginning therapy. The oral loading dose is 15 to 18 mg/kg usually followed by a 300 mg/day maintenance dose. When phenytoin is administered in emergent situations, the loading dose is 10 to 20 mg/kg IV (maximum dose 1.5 gm). In an emergency situation, it may take 15 to 20 minutes for a seizure to be controlled by IV phenytoin, so an IV benzodiazepine is usually administered concomitantly.

Nursing Responsibilities:

- IV phenytoin must be administered carefully. It is very alkaline so it is essential to verify the patency of the IV before administering the drug. It must be given into an IV of normal saline because it will precipitate in dextrose, and it is appropriate to flush the line before and after with NS. Phenytoin is administered no faster than 25 to 50 mg/min, because faster administration may result in bradycardia, hypotension, heart block, and ventricular fibrillation.
- The nurse monitors the ECG and BP during administration and for at least half an hour afterward.

Potential Side and/or Toxic Effects: Adverse effects are common with long-term use and may result in patients, especially adolescent females, discontinuing the medication. The effects include hirsutism, acne, and gingival hyperplasia. Patients may also develop hematologic abnormalities such as agranulocytosis and aplastic anemia. A potentially severe but rare adverse effect is the development of Stevens-Johnson syndrome. Phenytoin may therefore be discontinued if the patient develops a rash.

There is a narrow therapeutic window, and toxic effects may begin just above the therapeutic level (10 to 20 mcg/mL). Initially the patient may present with nystagmus then develop ataxia, tremors, and drowsiness.

Because IV administration of phenytoin is associated with so many problems, fosphenytoin (Cerebyx) might be given instead. The dose of fosphenytoin is always expressed as phenytoin equivalents (PE). If fosphenytoin is being administered for status epilepticus, the loading dose is 15 to 20 mg PE/kg. It may be given into an IV of D₅W or NS and administered at a rate of 100 to 150 mg PE/min. The patient may become hypotensive if this rate is exceeded. The patient is transferred to oral phenytoin as soon as possible.

Commonly Used Medications

Levetiracetam (Keppra) Levetiracetam is an anticonvulsant medication for which the precise mechanism by which it prevents seizures is unknown. Indications for its use have been expanding over the past several years and it is currently used to prevent seizures post TBI, post SAH, as monotherapy for some types of seizures, and as adjunctive therapy in status epilepticus.

Desired Effect: Levetiracetam is intended to prevent, stop, or decrease the incidence of seizure activity. It is rapidly absorbed when given orally and equivalent doses of IV and oral levetiracetam result in equal systemic exposure when IV levetiracetam is administered over 15 minutes.

Nursing Responsibilities:

Initial doses are usually 500 mg twice daily whether administered orally or intravenously. IV doses should be diluted in 100mL of NS, LR, or D5W and administered over 15 minutes.

Oral doses may be administered with or without food. Doses may be increased at two week intervals for seizure control. Supplemental doses may be needed for patients receiving dialysis.

The nurse monitors the patient with a previous history of psychiatric illness for depression and suicidal ideation and all patients for the development of infection.

A CBC with differential and LFTs should be monitored since less than 1% of the time neutropenia, leukopenia, bone marrow suppression and hepatitis occur.

Potential Side and/or Toxic Effects:

The most common adverse effects are asthenia (11–15%), headache (14–19%), infection (11–15%), increased blood pressure, and fatigue.

Hostility develops in about 10% of patients, and suicidal tendencies develop in 1% of patients.

NURSING CARE

Nursing care is related to the patient's underlying illness and likelihood of having a recurrent seizure. The nurse may need to help some patients prepare for additional testing to determine the cause of their seizures. These patients may be quite anxious and may need the opportunity to verbalize their anxiety. The nurse will be able to reassure other patients that recurrence of a seizure is unlikely as long as they follow their medical regimes carefully. This is especially true for the diabetic with hypoglycemia or the patient in congestive heart failure (CHF) with hyponatremia. Those patients with recurrent unprovoked seizures will need referral to a neurologist and thoughtful education.

Education of the Patient and Family

The information provided to patients and their families needs to be specific to the patients' epileptic syndrome and medication. However, there are some general categories of information that patients and families need. These include:

- Information about epilepsy
- How to care for the patient before, during, and after a seizure
- When to contact a healthcare provider for a seizure and when to go to the ED
- Information about the patient's anticonvulsant medication, such as how often drug levels should be monitored, the potential for status epilepticus if the medication is stopped abruptly, and the adverse and toxic effects of the specific anticonvulsant
- Explanation that weaning from the anticonvulsant, if it is to occur, will likely be gradual over about 10 weeks.

The relapse rate is about 40% to 50% after anticonvulsant weaning with most relapses occurring within the first year.

- Safety considerations such as wearing a Medic Alert bracelet, and restrictions on driving, swimming, climbing in high places, and the use of power tools or fire.

Life-Threatening Complication: Status Epilepticus

Status epilepticus has been defined as 20 to 30 minutes of continuous seizure activity or seizures occurring in succession for 20 to 30 minutes without a return to consciousness. However, because it increasingly appears that brain injury and further potential for further seizures result from status epilepticus, there has been a movement to define it as any seizure that lasts for longer than 5 minutes (Huff & Fountain, 2011). Any type of seizure may occur continuously, but is much easier to recognize when a major motor seizure does not terminate. Approximately 150,000 identified cases of status epilepticus occur each year resulting in over 40,000 deaths. The mortality rate is highest in elderly patients who develop status epilepticus following a stroke. People who survive following an episode frequently have cognitive declines.

There are three general categories of causes of status epilepticus. In roughly one-third of cases, the patient has a known seizure disorder. According to Roth and Berman (2011), in this circumstance it is likely that a change in medication by the physician or an abrupt cessation of the medication by the patient has triggered the event. In another third of cases, the episode of status epilepticus is the first seizure the patient has experienced. The final set of causes

includes a variety of toxic and metabolic conditions as well as events such as stroke, head trauma, or tumors that cause damage to the cerebral cortex (Roth & Berman, 2011).

A variety of physiologic events occur during status epilepticus. There is a surge of catecholamines so the patient often develops hypertension, tachycardia, cardiac arrhythmias, and hyperglycemia. The patient's body temperature may increase when there is vigorous muscle activity. Lactic acidosis, which occurs even after a single generalized motor seizure, is common in status epilepticus. In the early phases of status epilepticus, neuronal loss has been linked to rapid and chaotic firing. Later, the cerebral metabolic needs are increased and often exceed available oxygen and glucose delivery, resulting in potentially irreversible neuronal destruction. These events progress until the seizure is terminated. Thus, termination is necessary to preserve brain functioning as well as to resolve the hypertension, tachycardia, hyperthermia, and lactic acidosis. Most seizures terminate spontaneously. Why the seizure(s) continue in status epilepticus is unknown.

COLLABORATIVE CARE

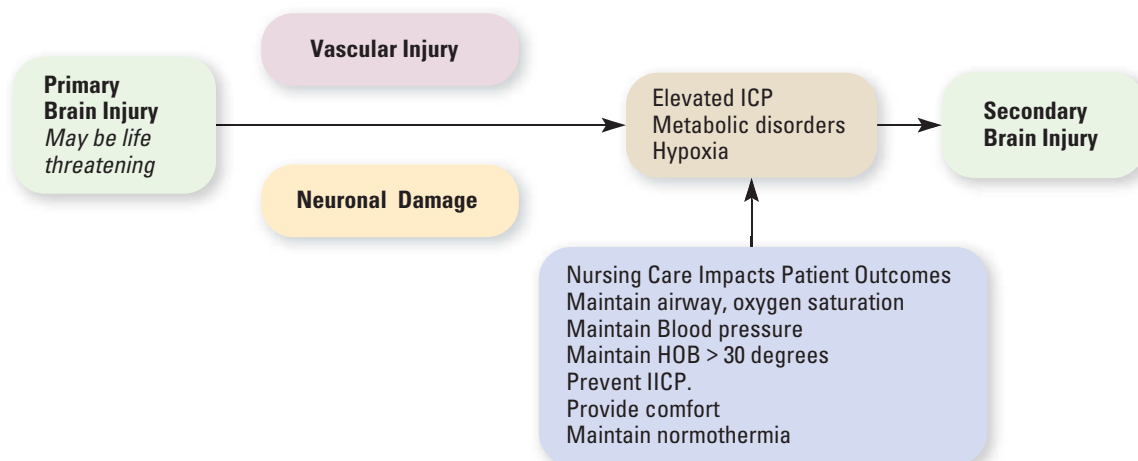
Aggressive supportive care and prompt termination of seizure activity are the goals of treatment. Specific protocols vary according to the institution. However, some general guidelines for management exist.

Essential for Safety

The nurse should stay with the patient, observe the seizure, and recognize if it does not terminate after the usual minute or two. When a seizure does not terminate after 4 to 5 minutes, the nurse must take action.

NURSING ACTIONS

- The first priority in status epilepticus, as always, is airway and oxygenation; for some patients, a nasopharyngeal airway is sufficient with provision of oxygen by nasal cannula. For other patients, endotracheal intubation is necessary.
 - ECG, BP, and O₂ saturation monitoring are necessary and are initiated (if not already in place). An IV of normal saline is started. Labs are drawn to check the likely causes of seizure activity, including glucose, electrolytes, levels of the patient's prescribed anticonvulsant, and toxicologies. A blood glucose is obtained. If it is not possible to obtain a blood glucose or hypoglycemia is suspected, 50 mL of D50 are given. Thiamine may be given to the patient suspected of alcohol misuse (Shearer & Riviello, 2011).
- "Seizures beget seizures" is an accepted clinical axiom (Huff, 2005). Therefore, early treatment of status epilepticus is the rule. Anticonvulsant medications are administered if seizure activity does not terminate after 5 to 10 minutes. IV administration has been the preferred route because it allows therapeutic levels to be attained more rapidly. Unfortunately, at this time there are few randomized trials so the treatment of status epilepticus is based primarily on clinical experience and consensus (Shearer & Riviello, 2011).
 - The initial drug of choice is a benzodiazepine, usually lorazepam (2 to 4 mg IV over 1 minute) because it terminates seizures 60% to 80% of the time. The dose may be repeated after 5 to 10 minutes if the seizure has not stopped. The nurse monitors the patient's BP, respirations, and O₂ saturation closely because the major adverse effects are respiratory depression, hypotension, and sedation.



Visual Map 10-3 Life-Threatening Brain Injury

(Pearson Education, Inc.)

- A phenytoin, phenytoin sodium or fosphenytoin (Cerebyx), is usually administered concomitantly to maintain seizure control since it is very effective in the termination of seizures. (See earlier discussion.)
- If the seizure continues beyond 20 to 30 minutes, the patient is intubated, a Foley catheter is inserted, an EEG is obtained, and the patient's temperature is monitored.
 - Failure to respond to optimal doses of a benzodiazepine and phenytoin is defined as refractory status epilepticus. There is no consensus on the best third-line drug, and protocols vary greatly. The alternatives include levetiracetam, valproate,

phenobarbital, midazolam, propofol, pentobarbital, and lidocaine.

Once control of the seizure is attained, the nurse will need to continue to monitor the patient's respiratory rate, O₂ saturation, and monitor pattern. The elevated heart rate, BP, and temperature should return to baseline, usually without treatment. In fact, the patient may become hypotensive following administration of the anticonvulsant medications. As the patient awakens, the nurse will need to orient him to his surroundings and explain what has occurred. An EEG may be obtained to verify there is no ongoing seizure activity.

Intracranial Dysfunction Summary

The nurse has important roles in the care of the patient with IICP. It is usually the nurse, while performing hourly nursing assessments, who is responsible for evaluating the patient's ongoing neurological status. It is also the nurse who is responsible for providing care in a fashion

that prevents secondary brain injury. Once the patient has survived the initial injury, the patient's outcome is often determined by the way that care is delivered and secondary brain injury is avoided.

Why/Why Not?

A 19-year-old, 165-pound male with a severe TBI is ventilated and receiving 3% saline at 75 mL/hr through a central line. The patient is 36 hours post-TBI, his ICP is currently 19, his BP by arterial line is 132/78, serum sodium 152, serum osmolarity 320, and urine output

40 mL/hr. Determine if it would be appropriate to continue the infusion now that the ICP is 19. Why? Why not?

See answers to Why/Why Not? in the Answer Section.

Case Study

Adam Simmons, 25 years old, was in a snowmobile accident. He was found lying against a tree unresponsive. In the field his blood pressure was 132/72, pulse 74, and respirations 14. When he arrived at the hospital, his pupils were unequal and reacted slowly. He moved his left side only to noxious stimuli and did not move his right side at all. His BP was 170/70, pulse 60, and respirations 22.

1. What do these findings most likely indicate and why did the vital sign changes occur?
2. Mannitol 50 g was ordered immediately. What was its purpose? What are the nursing implications of administering mannitol emergently?
3. Adam was taken to CT scan and then immediately to the OR for evacuation of an epidural hematoma. Why

does discovery of an epidural hematoma require emergent surgery?

4. On transfer to the ICU following surgery, Adam was receiving Diprivan (propofol) for sedation. Why might sedation have been required? What are the advantages and problems of using propofol for sedation in the patient with IICP?
5. In the ICU, Adam had an ICP monitor and an external ventricular drain that was to be maintained 5 cm above the tragus and drained continuously. What are the nursing responsibilities associated with maintaining such a system?

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 10.1 How does the brain maintain a relatively normal intracranial pressure when a patient has a slowing expanding subdural hematoma?
- 10.2 Why do secondary insults (secondary causes of increased intracranial pressure) have such an impact on patient outcome?
- 10.3 Which of the components of the Glasgow Coma Scale is most predictive of patient outcome in traumatic brain injury?
- 10.4 What is the clinical significance of a patient developing an intracranial pressure of 34 and a cerebral perfusion pressure of 50? How should the nurse respond to such findings?
- 10.5 Why is traumatic brain injury a significant public health concern?
- 10.6 What is the difference in patient presentation and collaborative management between subdural and epidural hematomas?
- 10.7 What are the initial priorities in the collaborative management of a patient with a traumatic brain injury?
- 10.8 What measures can a nurse utilize to prevent shivering when using external cooling to decrease a patient's temperature?
- 10.9 What are the early manifestations of meningitis? What should a nurse do if she suspects a patient has meningitis?
- 10.10 How should a nurse respond when a patient with a traumatic brain injury has a seizure?

See answers to Chapter Review Questions in the Answer Section.

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Chapter 11

Care of the Patient with a Cerebral or Cerebrovascular Disorder

Susan Barnard, MS, APRN, CCRN

Abbreviations

ADLS	Activities of Daily Living	NIH	National Institutes of Health
ASA	American Stroke Association	NINDS	National Institute for Disorders and Stroke
BAC	Brain Attack Coalition	PSC	Primary Stroke Center
HPA	Hypothalamic-Pituitary-Adrenocortical	TPP	Thrombotic Thrombocytopenic Purpura



Learning Outcomes

Upon completion of this chapter, the learner will be able to:

1. List the common manifestations of brain tumors and explain their causation.
2. Compare and contrast the care of patients with supratentorial, posterior fossa, and pituitary tumors.
3. Explain the mechanisms and manifestations of hemorrhagic and ischemic strokes.
4. Describe emergent management of the patient with an ischemic stroke.
5. Compare and contrast care of patients following intracerebral hemorrhage and subarachnoid hemorrhage.
6. Prioritize nursing care of the stroke patient.

Cerebral Disorders

Nursing care of the patient with cerebral and cerebrovascular disorders requires nurses to understand the anatomy and physiology of the brain and have excellent neurological assessment skills. Initial symptoms of dysfunction can be subtle, so patients may not recognize the earliest signs of brain tumors or stroke and may wait to present for treatment until more dramatic symptoms have developed.

Nurses need to educate patients about the risk factors and clinical manifestations of these disorders to promote prevention and prompt treatment as improved outcomes, especially for stroke, are dependent on immediate interventions.

Anatomy and Physiology Review

In order to understand cerebral and cerebrovascular disorders, it is important to have an understanding of the

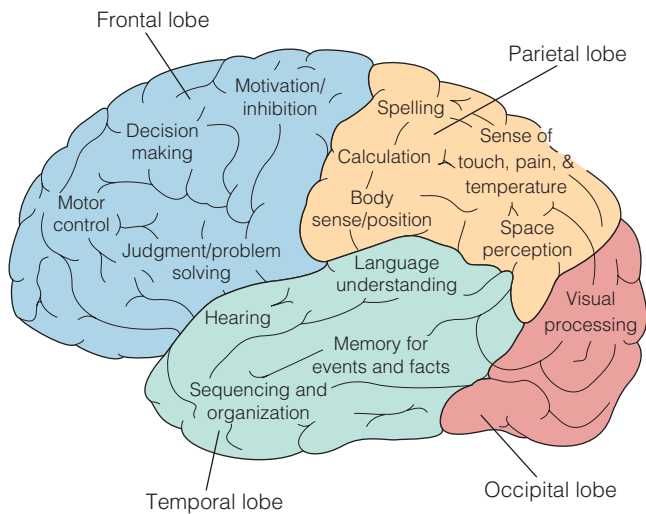


Figure 11-1 Functions of the lobes of the brain.

anatomy and physiology of the brain. This chapter includes a brief review of the functions of various parts of the brain, its vascular supply, and the blood-brain barrier. Review the metabolic requirements of the brain, the Monro-Kellie hypothesis, and cerebral perfusion pressure, as well as the primary and secondary causes of increased intracranial pressure.

The brain can be divided into three main parts; the cerebrum, the cerebellum, and the brain stem. The cerebrum is then broken down into lobes. The locations and functions of the lobes of the cerebrum are displayed in ♦ Figure 11-1. In order to understand the neurological deficits that a patient may develop from cerebral blood vessel disorders, the nurse must understand the functions of the different areas of the brain.

The brain is one of the most metabolically active organs in the body and is dependent on a consistent blood

flow to meet its oxygen and energy requirements. When there is a disruption in cerebral blood flow, neuronal cellular injury or death may occur. ♦ Figure 11-2 displays cerebral blood flow, indicating which vessels perfuse which portions of the brain. Understanding which blood vessels perfuse which area of the brain helps the nurse understand what deficits will be manifested from blood vessel disruptions caused by brain tumors, strokes, or aneurysms.

The brain also requires a very stable chemical environment in order for the nerve cells to communicate with one another. Glial cells form a layer around blood vessels called the blood-brain barrier to provide just such protection for the brain. The blood-brain barrier protects the brain from foreign substances in the blood that may injure the brain, as well as from hormones and neurotransmitters in the rest of the body. Large molecules and molecules with high electrical charge do not pass through the blood-brain barrier easily. However, lipid-soluble molecules cross rapidly into the brain. Thus, the blood-brain barrier helps to maintain the stable chemical environment required for the brain to function.

Brain Tumors

In 2015, approximately 77,670 new cases of primary non-malignant and malignant brain tumors are expected to be diagnosed in the United States (CBTRUS, 2015). In the United States, gliomas account for 30% of all brain tumors and 80% of malignant tumors, while meningiomas, a type of benign tumor, are the most common type of tumor. The majority of primary tumors (34%) are located within the meninges, followed by those located within the frontal, temporal, parietal and occipital lobes of the brain (22%; American Brain Tumor Association, 2014a). An estimated 16,616 deaths will be attributed to primary malignant brain and CNS tumors in the United States in 2016 (CBTRUS, 2015).

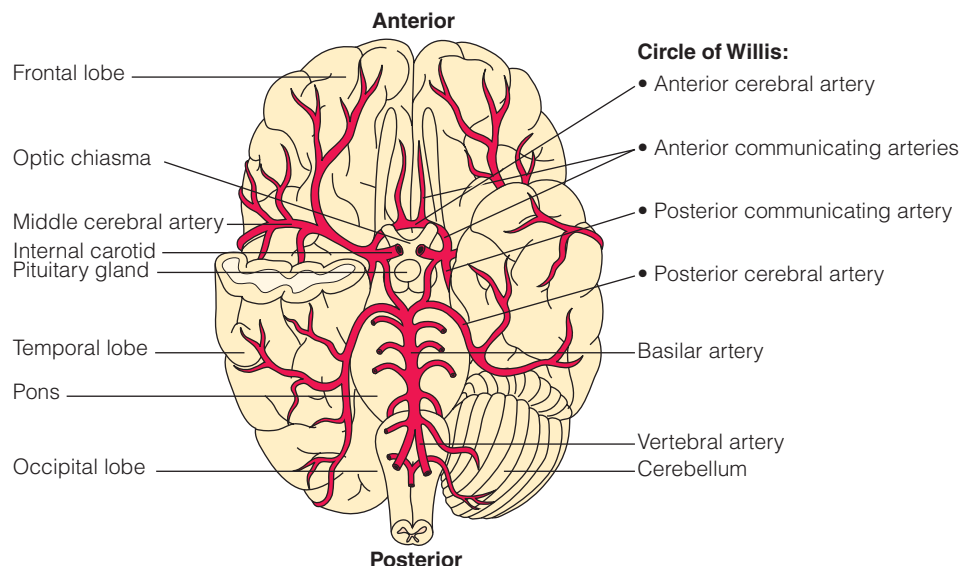


Figure 11-2 Cerebral blood flow.

Types and Characteristics of Brain Tumors

Brain tumors are classified as primary or secondary. Primary brain tumors originate from the cells within the brain and are classified by the type of cell from which they develop. Secondary, or metastatic brain tumors, occur when cancer cells from other parts of the body invade brain tissue. Primary brain tumors may be either benign or malignant.

Primary brain tumors are named after the cells involved in the tumor. Astrocytomas, a type of malignant tumor, develop from astrocytes that provide structure for the brain and spinal cord. Normal astrocytes become tumor cells through gene mutation, which allows the cells to divide abnormally. The World Health Organization (WHO) has developed a grading system for astrocytomas based on cell type, described numerically as I through IV. Grade I and Grade II tumors are considered to be low-grade tumors and have the most favorable survival rates. The tumor cells are well defined, have a slow growth rate, and a low incidence of brain infiltration. Grades III and IV are considered higher-grade tumor cells which are abnormally shaped and have a pronounced ability to infiltrate normal brain tissue (Hinkle, 2010).

Meningiomas, the most common benign brain tumors, develop from the meninges and may occur intracranially or within the spinal canal. They are usually well circumscribed, may be attached to the dura, and are associated with an excellent prognosis when gross-total resection is possible. Other common benign brain tumors arise from nerve sheaths (acoustic neuromas) or the pituitary (pituitary adenomas). Noncancerous primary brain tumors may be life threatening if their growth puts pressure on vital brain structures or undergoes malignant transformation.

Pathophysiology and Risk Factors

Genetic mutations and deletions of tumor suppressor genes increase the risk for some types of brain cancer. In rare cases, inherited diseases such as neurofibromatosis and retinoblastoma are also associated with brain tumors. Exposure to vinyl chloride is an environmental risk factor for brain cancer. Vinyl chloride is a carcinogen used in manufacturing plastic products such as pipes, wire coatings, furniture, car parts, and housewares. It is also present in tobacco smoke. People who work or live in close proximity to manufacturing and chemical plants that produce vinyl chloride have an increased risk for brain cancer. Radiation and radiotherapy for childhood cancers and leukemia have also been associated with the development of brain tumors (Ney & Lassman, 2010). The cumulative radiation received over a lifetime can increase the risk of cancer, which is why best practice dictates caution in irradiating pediatric patients.

Brain tumors appear to cause symptoms by several different mechanisms. The first two mechanisms, invasion of

the brain parenchyma and compression of brain tissue, result in dysfunction of the area of the brain where the tumor is located, and the appearance of focal neurological symptoms. The next mechanism is the development of cerebral edema. Although not fully understood, cerebral edema appears to develop once tumors have increased in size beyond 1 mm. The new blood vessels that feed the tumor lack the normal blood-brain barrier and are more permeable to macromolecules, proteins, and ions being released into the brain tissue, resulting in vasogenic cerebral edema. Simultaneously, macrophages and inflammatory mediators are released that increase vascular permeability and edema. The final two mechanisms by which brain tumors cause symptoms are obstruction of the flow of cerebrospinal fluid, resulting in hydrocephalus, and the compression and resulting shift of brain contents resulting in herniation. These mechanisms cause symptoms related to the increased mass within the cranial cavity and the increased intracranial pressure (IICP) (Hinkle, 2010).

Patient History and Assessment

Increased intracranial pressure from any cause is a serious medical condition, so identification is key. Patients with brain tumors commonly have the following signs and symptoms:

- Headache, as an initial symptom, typically worse in the morning with improvement after the patient arises, may worsen with coughing, exercise, or with a change in position such as bending or kneeling and often does not respond to the usual headache remedies.
- Nausea and vomiting
- Drowsiness
- Visual problems such as blurred vision, double vision and loss of peripheral vision. A swollen optic nerve (papilledema), a clear sign of IICP, is common in young children, in persons with slow-growing tumors, and in patients with tumors in the posterior fossa. Older adults who have age-related brain atrophy are less likely to present with generalized symptoms of IICP such as headache and papilledema and more likely to present with mental status changes.
- Mental status and/or personality changes can be caused by the tumor itself, by IICP, or by involvement of the parts of the brain that control personality. These changes can range from problems with memory (especially short-term memory), speech, communication, and/or concentration changes to severe intellectual problems and confusion. Changes in behavior, temperament, and personality may also occur, depending on where the tumor is located (Bohan & Glass-Macenska, 2009).
- **Seizures** are a common presenting symptom of brain tumors resulting from tumor irritation of brain tissue. The likelihood of a patient developing a seizure from a

brain tumor is dependent on the location and type of the tumor as well as the age of the patient. Slow-growing tumors near the cerebral cortex (such as meningiomas) are more likely to cause seizures, and older adults with brain tumors are less likely to develop seizures than younger adults (Bohan & Glass-Macenska, 2009). Seizures in patients with brain tumors may present as generalized convulsions with loss of consciousness. However, focal seizures, such as muscle twitching or jerking of an arm or leg, abnormal smells or tastes, problems with speech, or numbness and tingling, may also occur.

In addition to the general symptoms listed, other more specific symptoms, known as focal symptoms, occur in approximately one-third of patients with brain tumors. Focal symptoms include hearing problems such as ringing or buzzing sounds or hearing loss, decreased muscle control, lack of coordination, decreased sensation, weakness or paralysis, difficulty with walking or speech, or double vision. Because the symptoms are usually caused by invasion or compression from the tumor, these focal symptoms can help identify the location of the tumor. ♦ Figure 11-3 displays the focal symptoms that often develop from brain tumors originating in specific structures of the brain. The tracts of the central nervous system cross near the base of the skull, so a tumor on one side of the brain causes symptoms on the opposite side of the body.

Focused Assessment

History taking is an important initial step in the nursing assessment of a patient with a brain tumor. History taking questions should focus on any signs and symptoms the patient may be experiencing, with special attention to the time of day when they occurred and what exacerbated them. For example: Was the headache severe on awakening? Did it improve as the day progressed? What makes it feel better or worse? Depending on the symptoms the patient describes, the nurse will need to direct the nursing assessment in order to determine the patient's ability to perform activities of daily living (ADL) while experiencing these symptoms. Finally, the nurse completes a baseline neurological assessment with particularly careful emphasis on the areas affected by the tumor.

Magnetic resonance imaging (MRI) and computerized tomography (CT) scanning are standard imaging tools for diagnosis and guided treatment of brain tumors. Functional MRIs may be used to assess the potential clinical outcomes of the tumor. Positron emission tomography (PET) scans have a role in grading a tumor for prognosis, localizing a tumor for biopsy, and mapping brain areas prior to surgery. A computer-assisted stereotactic biopsy may be used to detect a deep-seated brain tumor and determine prognosis and treatment options (Weber, Lovblad, & Rogers, 2010).

Common Manifestations of Brain Tumors Located in Specific Areas of the Brain

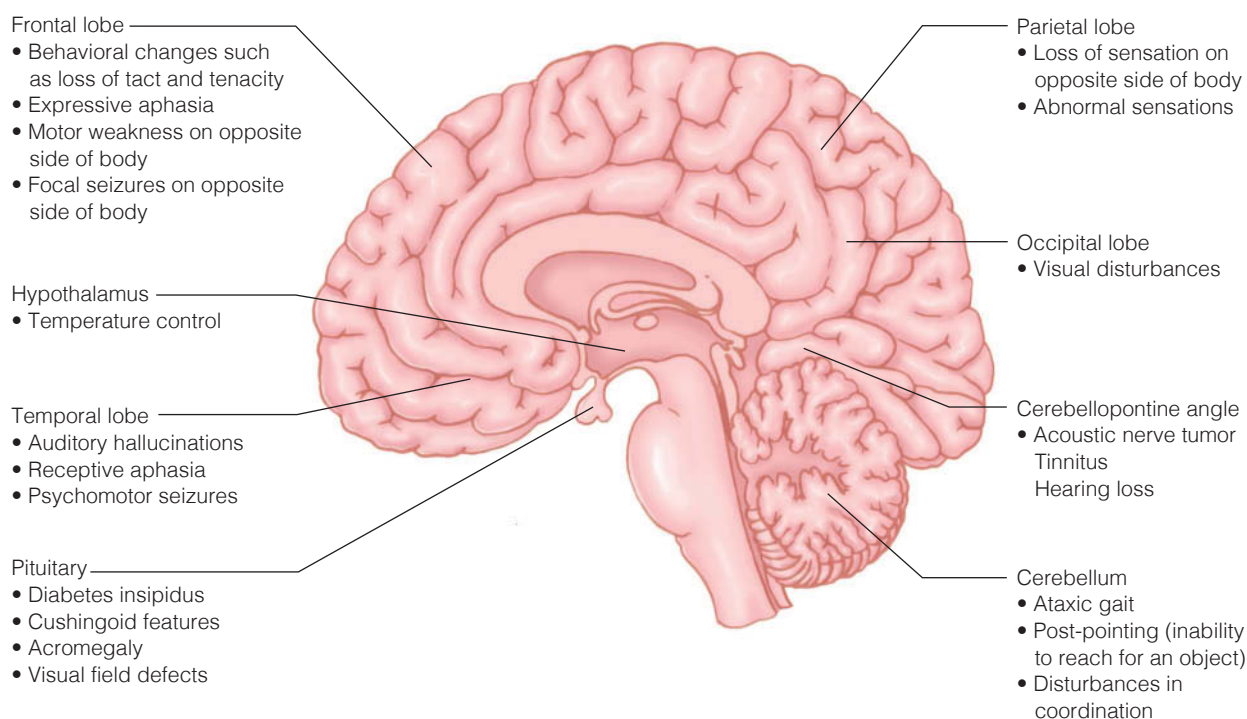


Figure 11-3 Focal symptoms that develop with damage to specific areas of the brain.

COLLABORATIVE CARE OF THE PATIENT WITH A BRAIN TUMOR

The collaborative management of the patient with a brain tumor is aimed at reducing the edema surrounding the tumor, decreasing the tumor mass, dealing with any possible spread of the cancer and symptom management including pain control. Management may involve steroids, biopsy or resection of the tumor, radiation therapy, and chemotherapy.

Medical Management

Medical management may include a combination of modalities such as chemotherapy, radiation therapy or targeted cancer therapies, affecting the growth and spread of cancer cells on a molecular level. Pharmacological therapy includes glucocorticoids that are the mainstay of treatment for vasogenic cerebral edema. These agents decrease the tissue swelling associated with brain tumors and manage some of the troublesome signs and symptoms that patients experience, including headaches, seizures, motor deficits, and altered mental status. Glucocorticoids directly affect vascular endothelial cell function and restore normal capillary permeability. Dexamethasone may also cause cerebral vasoconstriction.

Essential for Evidence-Based Practice

Dexamethasone has been the corticosteroid of choice because of its high-potency, low-mineralocorticoid effect that makes it less likely to result in sodium retention, and 48-hour half-life (Kotsarini, Griffiths, Wilkinson, & Hoggard, 2010).

Surgical Management

Surgery is the initial treatment for most benign and many malignant tumors. It is often the preferred treatment when a tumor can be removed without any unnecessary risk of neurological damage. The most common types of brain surgery performed are biopsy, craniotomy and neuroendoscopy. The extent of tumor resection is predictive of patient outcome for most types of brain tumors, and resection is usually first-line treatment. The amount of tissue resected ranges from gross total to partial to biopsy only with the best outcomes resulting when gross total resection is possible. The amount of tissue that can be resected is dependent on the invasiveness of the tumor, its location in the brain, and the patient's health status. Surgery may also be utilized to provide direct access for chemotherapy, radiation implants, or genetic treatment of malignant tumors as well as to treat uncontrolled seizures caused by brain tumors (American Brain Tumor Association, 2014b).

Once in the operating room (OR), the hair on the scalp is clipped and an incision is made. Then a circular piece of the skull bone is removed and the dura is opened to expose the brain. After the brain is exposed, the surgeon may do some mapping to identify precisely what function is performed by parts of the brain near the surgical site. There are several ways to "map" the brain. Some are used during surgery; others may have been performed preoperatively. Mapping may involve:

- Stimulating brain tissue with tiny electrical currents
- Measuring brain waves as they are stimulated
- Using ultrasound probes inside or near brain structures
- Probing the brain with special computerized "wands"
- Using PET or single photon emission computed tomography (SPECT)

Cortical mapping allows the surgeon to identify and avoid "eloquent" areas of the brain, such as the motor strip, sensory areas and speech area, and allows the medical team to interact with the patient during surgery.

Most tumors have been precisely located by MRI and CT preoperatively. When surgery is performed, it is usually computer-assisted with specialized equipment to aid in the positioning of the patient and the delivery of the treatment. Called surgical navigation, this approach allows for precise, limited craniotomy with minimal disruption of normal tissue.

Essential for Safety

An awake craniotomy technique is being studied to allow for intraoperative sensory, motor, and speech testing during surgery with a goal of decreasing neurological deficits (Wolff, Naruse, & Gold, 2010). Similarly, intraoperative MRI is being used to aid in the resection of lesions (Parney et al., 2010).

In addition to surgery, alternative methods of tumor destruction may be utilized. These treatment methods can be directed at the tumors so exactly that little or no nearby normal tissue is damaged. Thermal destruction instruments, such as lasers, can be placed in the exact spot needed to destroy tumor tissue. Ultrasonic aspiration may be used to break up tumor tissue and evacuate it from the brain.

If tumor removal is possible, then portions of the resected tumor are sent to pathology for a definitive diagnosis. When the tumor may not be resected, the biopsy is usually performed stereotactically using either a CT- or MRI-based frame. These systems allow precise placement of the biopsy probe within the tumor mass.

Alternatives to surgery are also available. The following procedures are considered ablative procedures because they cause cell death and necrosis of the tumor over time. They are most appropriate for people with smaller tumors

in areas inaccessible to surgical intervention. Multiple small doses of external radiation can be aimed at the tumor (stereotactic radiosurgery or “gamma knife”). The blood supply to the tumors can be identified by angiography, then a variety of plugs can be introduced to block the artery, causing the tumor to die from lack of blood flow (Dea, Borduas, Kenny, Fortin, & Mathieu, 2010).

NURSING ACTIONS

Prior to surgery, the nurse ensures that the consent form has been signed and that all pre-operative procedures and orders have been completed. Providing thorough patient and family education including the amount of hair to be clipped, the length of the surgery, and what to expect post-operatively will decrease the patient’s and family’s anxiety and promote patient-centered care. The nurse also clearly documents the patient’s neurological assessment so it can be used as a baseline for the postoperative assessment.

Post-operatively, the nursing care of the craniotomy patient is centered on assessment of the patient’s neurological status and prevention of complications such as increased intracranial pressure or infection. Close observation and monitoring of the post-operative patient for neurological deficits and vital sign changes and comparing to baseline findings are important for identifying complications.

Essential for Safety

Most patients are transferred to an intensive care unit (ICU) for at least 24 hours following cranial surgery so their neurological status can be assessed frequently until they have stabilized. A study by Rhondali et al. (2011) concluded that elective craniotomy patients who had immediate successful extubation and a surgery lasting less than 4 hours could safely be transferred to a neurosurgical floor rather than an ICU.

Initially the patient is monitored for recovery from anesthesia and return to baseline neurological status.

Commonly Used Medications

Dexamethasone

Introduction

Dexamethasone (Decadron), a glucocorticoid, is indicated for use in patients who have symptomatic cerebral edema from brain tumors, particularly because of its long half-life and decreased tendency to cause psychosis (Dietrich, Krithika, Pastorino, & Kesari, 2011). It is often administered during the perioperative period and while the patient is undergoing radiation. However, it may be continued for as long as it alleviates patient symptoms of cerebral edema. The usual initial dose is 10–20 mg of IV dexamethasone followed by maintenance dosing of 4 mg IV every 4–6 hours. Post-operative dosing is dependent on the type and location of the tumor. Some tumors, such as metastatic tumors, have more glucocorticoid receptors and are more responsive to dexamethasone treatment, whereas others, such as meningiomas, are not.

Desired Effect: Administration of dexamethasone can produce a reduction in cerebral edema and an improvement in neurological symptoms within 8 to 48 hours of the initial dosing, most commonly in 12 to 24 hours. The decline in cerebral edema and ICP can persist for as long as 72 hours.

Nursing Actions:

- Side effects from glucocorticoid therapy are common, so it is important for the healthcare team to determine whether or not the patient is among the 70% to 80% of patients who could benefit from the therapy if it is to be continued.
- To avoid adverse effects, the dose is adjusted to the minimum that will control the patient’s symptoms. Even at the lowest appropriate dose, typical doses given to patients with brain tumors have the potential to suppress the hypothalamic-pituitary-adrenocortical (HPA) axis.

- If the dose is to be discontinued, it should be tapered to allow the HPA axis time to recover. In addition, if the steroids are abruptly discontinued, rebound edema may occur and the patient may have an abrupt return of neurological symptoms. Tapering schedules for dexamethasone vary depending on the length of time the patient has been on the steroid and the patient’s symptoms.

Potential Side and/or Toxic Effects: Adverse effects are dose and time dependent with as many as 50% of patients experiencing at least one toxic symptom. Common neurological effects include insomnia, visual blurring, tremor, behavioral changes and decreased taste and smell with more systemic effects including increased appetite, weight gain, hyperglycemia, hypertension, muscle weakness in the legs (the patient may complain of inability to climb stairs or arise from chairs); stomach ulcer; and increased risk of infection.

Patient and family education concerning muscle weakness is important because they may fear it is an indication of worsening neurological function. Dietary counseling is important to prevent excessive weight gain and high blood sugars. Nurses should encourage the patients to take steroids with food and avoid aspirin and nonsteroidal anti-inflammatory agents to prevent gastric ulceration and bleeding. When patients with brain tumors receive steroids for a prolonged period, their CD4 count may drop enough to predispose them to opportunistic infections. Nurses should inspect the mouths of these patients to detect the presence of oral and esophageal candidiasis. To prevent the development of *Pneumocystis pneumonia*, Bactrim might be administered.

Once the patient has recovered from anesthesia, changes in neurological status are identified and reported to the surgical team. A change in neurological status could be indicative of increasing edema around the tumor site; this usually responds to an increase in the dose of dexamethasone. Deterioration in neurological status might also be an indication of a generalized increase in ICP. Rarely, deterioration in neurological status postoperatively is associated with postoperative hemorrhage, and the patient must be returned to the OR. Nursing assessments and interventions specific to patients with common types of cranial surgeries are displayed in Table 11-1.

Post-operatively, patients are usually positioned with the head of their bed elevated 30 degrees. This facilitates venous drainage from the head and neck, preventing increases in ICP and increasing patient comfort. If a large tumor was resected, the patient is usually instructed to keep head and neck in a neutral, midline position. If the patient has had posterior fossa surgery, a stiff dressing or cervical collar may be applied to prevent hyperflexion or hyperextension. Patients are often ordered laxative, antitussive and antiemetic medications to prevent straining with a bowel movement, coughing or vomiting which could increase intrathoracic pressure and decrease venous return from the brain (Elsevier Health, 2012).

NURSING CARE

Enhancing Comfort

There is disagreement concerning the amount of discomfort experienced by patients post-craniotomy. A review of the literature by Flexman, Ng, and Gelb, (2010) indicated that acute and chronic pain post-craniotomy is

frequent and under-recognized. The incidence and severity of the pain may be based on surgical and patient factors. Although the brain itself does not have any nociceptors, stretching of the dura and increasing pressure in the skull can cause pain. Codeine is frequently used post-operatively and is usually sufficient in the relief of headache.

Essential for Patient-Centered Care

Research suggests that morphine is safe and extremely effective in treating post-craniotomy pain (Flexman et al., 2010). The major concern of using morphine for pain with this patient population is that it may alter the patient's respiratory rate and neurological signs. With these factors in mind, critical care nurses need to actively assess their post-craniotomy patients for pain and consider the pharmacological properties of the analgesics being used.

Providing Nutrition

The role of the critical care nurse in nutrition therapy is to monitor central and peripheral IV lines and/or feeding tubes for patency and insertion sites for infection. An accurate intake and output should be monitored and documented. Before oral feedings are initiated, the patient's cough and gag reflexes need to be evaluated. If there is a risk of a swallow deficit due to a neuromuscular dysfunction, a more thorough swallow evaluation with videofluoroscopy may be ordered. Symptoms prior to the surgery that may influence nutrition may persist post-operatively such as nausea, vomiting, and diarrhea. If the patient is receiving other treatments such as chemotherapy or radiation, the nurse needs to understand the possible side effects and how they may affect the patient's nutritional status.

Table 11-1 Common Types of Cranial Surgeries

Type Of Surgery	Supratentorial	Posterior Fossa	Transphenoidal
Type of Tumor	May be either a metastatic or a primary brain tumor.	Primary brain tumors often of the cerebellum, perhaps of the acoustic nerve.	Pituitary tumors.
Age of Patient	Majority of adult tumors.	55%–70% of pediatric tumors. 15%–20% of adult tumors.	10% of adult tumors.
Surgical Considerations	Mapping may be used to avoid “eloquent” areas of the brain.	Small enclosed space near critical brain structures, including the brainstem, the cerebellum, and cranial nerves.	Approach is through the nose and sinuses. Pituitary sits on the optic chiasma.
Nursing Considerations	Patients are usually positioned with their head of the bed elevated 30 degrees postoperatively. Patients are not turned to the side of the tumor if a large tumor has been removed. At a minimum the Glasgow Coma Scale, pupillary response, and strength, movement, and sensation in extremities should be assessed.	Level of the head of patient's bed varies by institution from flat to 60-degree elevation. Prevent patient from pronounced flexion or extension of the neck post-op. Assess function of cranial nerves (V, VII, VIII, IX, and X). Evaluate coordination.	Maintain head of the bed elevated 30–45 degrees. Provide a moustache dressing. Assess for CSF leakage on dressing. Discourage patient from sneezing and blowing the nose. Assess for visual field defect. Assess pituitary function and identify presence of diabetes insipidus.

An understanding of the patient's tumor etiology, outcome of the surgical procedure, medications, and other treatment modalities will guide the nurse in meeting the patient's nutritional needs. Collaboration with a registered dietician will ensure that an optimal nutritional plan of care is in place (Scanlon, 2006–2007).

Facilitating Communication

Communication can be a particularly frustrating experience for the post-craniotomy patient, possibly due to sensory deprivation. Periorbital edema is common and may obstruct vision to one or both eyes. In addition, the patient may have a bulky head dressing that may decrease the ability to hear clearly. If the patient is intubated, the nurse must consider how it increases the challenges of communicating. It is also essential that the nurse recognize the patient's fears, self-image issues, and coping capabilities. Some ways that the nurse can enhance communication with a post-craniotomy patient include the following:

- Announce your presence when entering the room (avoids surprising the patient whose vision is impaired)
- Face the patient directly when speaking and raise voice if necessary (dressing may inhibit sound)
- Use picture cards, dry erase board/marker, and/or communication system of blinking eyes/squeezing hand for yes or no (with intubated patients who cannot speak)
- Encourage the patient to verbalize feelings and frustrations
- Assist with grooming using patient's own clothes and head cover (turban and later wig)
- Involve family and friends and, if needed, social workers, spiritual advisors, and psychological counselors

Listening carefully and providing reassurance are therapeutic strategies to facilitate communication. Support groups may offer the survivor and caretakers support and strength through listening and telling of their lived experience, which then validates that of the survivor's experience.

Fostering Patient-Centered Care

It is essential to empower patients to be actively involved in their plan of care. There are ways that critical care nurses can advocate for patient centered care and provide support to families of critically ill patients. Education is key, and specific educational needs must be met so the patient is able to make informed decisions. For example, if the patient has a diagnosis of cancer, then treatment options must be explained. Effective patient education can improve clinical outcomes through compliance of treatments and recognition of complications. It is especially important

when working with post-craniotomy patients that the nurse begins discharge planning on the day of admission. Early determination of the patients' probable home care needs will enhance the likelihood of their successful return to home.

Maintaining Safety

Self-care deficits of the neurosurgical patient depend on the extent of the surgery and outcomes. They may be at risk for falls due to paralysis, muscle weakness, and lack of coordination. They may also have cognitive impairments resulting in poor judgment. The nurse needs to assess and document the patient's limitations and strengths. A fall risk assessment should be performed (see "Safety Initiatives") to determine the patient's level of risk. In order to meet the patient's safety needs, the nurse may collaborate with physical therapists, occupational therapists, and/or speech therapists.

Complications

Four of the potentially serious complications following cranial surgery are systemic venous thromboembolism, cerebrospinal fluid leaks, meningitis, and seizures.

Symptomatic Venous Thromboembolism

Symptomatic **venous thromboembolism (VTE)** is a serious post-operative complication that may occur up to 6 weeks after surgery. Hypercoagulopathy is a potential complication of glioma increasing the risk of deep vein thrombosis (DVT) and pulmonary embolism (PE). Ney and Lassman (2010) note that a biologic factor secreted by some types of malignant brain tumors may be one of the causes of the coagulation abnormality. Risk factors for development of VTE include age older than 60, leg weakness, large tumor size, surgery lasting longer than 4 hours, bedrest, and tumor histology.

Prophylaxis for VTE is recommended for most patients following surgery for malignant primary brain tumors. Sequential pneumatic compression boots and graduated compression stockings have been shown to decrease the occurrence of VTEs without increasing ICP. An alternative is the use of compression boots prior to, during, and for 24 hours after the surgery followed by low-dose low molecular weight heparin (LMWH) 5,000 units twice a day or enoxaparin 40 mg/day. Passive range of motion and early ambulation are also preventative measures. Evidence-based VTE prophylaxis is especially important to manage because although these patients usually experience a hypercoagulable state, treatment with antithrombotics can cause bleeding into the tumor and neurological complications so other strategies must be used (Wen, Lee, Leung, & Eichler, 2015).

The critical care nurse needs to maintain the compression boots or stockings during the immediate postoperative period. Studies have shown that compression devices are often removed for longer than necessary for bathing, moving, or transport. Although the nurse assesses the patient for such evidence of deep vein thrombosis as leg discomfort, swelling, warmth, and a positive Homan's sign, patients with VTE are asymptomatic 80% of the time.

Cerebrospinal Fluid Leaks

Cerebrospinal fluid (CSF) leakages occur when there is a tear in the dura, allowing an opening to develop between the subarachnoid space and the outside environment. The critical care nurse may identify a CSF leak by clear fluid containing glucose leaking from the patient's ear or nose and forming a halo as it settles on a pillowcase or filter paper. Experts do remind us, though, that the presence of a halo sign is not exclusive to CSF leaks and can lead to false positive results. CSF leaks may be problematic because they may result in CSF depletion. If so, the patient will complain of a headache, which is usually more severe when the patient is in the upright position, and is alleviated when the patient is supine. Most CSF leaks heal spontaneously within a week. However, on occasion, surgery is required to seal the dura. If the leak continues, the nurse must be vigilant in monitoring the patient:

- If the CSF leak is from the nose, position the patient with the head of the bed elevated slightly, with a moustache dressing applied to catch the leaking CSF.
- If the CSF is leaking from the ear or surgical wound, position the patient on the affected side (or as per surgeon's order) and apply a sterile dressing.
- Do not suction nasally, and packing should not be inserted into the nose or ears.

The major concern with a CSF leak is that there is an open pathway to the subarachnoid space with the potential for developing meningitis (Kaptain, Kanter, Hamilton, & Laws, 2010).

Meningitis

Meningitis is caused by inflammation of the meninges, which are protective membranes that cover the brain and spinal cord. The three primary causes are bacterial, viral and fungal. There is an increased risk of meningitis post-craniotomy due to manipulation of brain tissues, incision site and shunts. High risk for infection has been associated with endonasal endoscopic skull base surgery (ESBS) due to the approach through the sinuses, which are not sterile. Risk factors for developing meningitis include postoperative external ventricular shunt, remote site

infection, CSF leaks and repeat operation. Meningitis post-craniotomy results in a high mortality rate and a longer hospital stay for the patients who survive (Kono et al., 2011). In order to promote the prevention of infection, the critical care nurse must:

- Use aseptic technique when caring for external ventricular shunts, wound drains, and surgical sites.
- Observe diligently for the manifestations of meningitis, which include fever, chills, increasing headache, neck stiffness, and photophobia. The patient may also develop a petechial rash on the trunk and lower extremities, hemiparesis, altered mental status or seizures (American Association of Neuroscience Nurses, 2006).

Seizures

Review seizure types and management described earlier in your reading. The risk of seizures after craniotomy is extremely common, but the incidence varies greatly based on the primary diagnosis, severity of surgical insult, and pre-existing seizure activity. The risk of seizure development is partially dependent on the type and location of the brain tumor. Patients with low-grade, slow-growing gliomas are most likely to develop seizures. Of patients who develop seizures, approximately 50% have tonic-clonic seizures, including status epilepticus. Even if they receive optimal treatment, the majority of patients with brain tumors continue with some type of seizure, most commonly a focal seizure. Unfortunately, side effects of treatment with antiepilepsy drugs (AED) occur more frequently in patients with brain tumors than in other patients with seizures. A recent study suggests the use of newer (second generation) non-enzyme inducing antiepileptic drugs (non-EIAED) as they seem to have less interactions with other medications (e.g., chemotherapy) (Kurzweil, Herringer, & Simon, 2010). The study concluded that recurrent seizures post-craniotomy continue to cause significant clinical problems.

Recovery

Recovery depends on a variety of factors, including patient age, the location of the tumor, the histology of the tumor, the amount of tumor resected, the patient's neurological status, and the radiation dose. Radiation is one of the most effective treatments for gliomas and has been found to increase survival rates as compared to surgery alone for malignant brain tumors.

Standard treatment for malignant gliomas has included primary resection, followed by radiation therapy and temozolomide, which has shown improved prognosis. Recent studies have looked at the addition of bevacizumab (Avastin), which was approved by the FDA in 2009, for

Gerontological Considerations

- Older adults with brain tumors are less likely to develop seizures than children or younger adults.
- Older patients with brain tumors are at higher risk for venous thromboembolism.
- If an older patient with a cerebral injury develops hypernatremia (serum sodium greater than 160 mmol/L), he is at increased risk for renal failure, heart failure, and pulmonary edema than a younger adult.
- Headaches and seizures are the most common symptoms at presentation in the elderly.
- Diagnosis of primary brain tumors in the elderly is more difficult and often delayed due to non-specific symptoms that mimic the physical and cognitive changes seen in the normal aging process.

single agent use in recurrent glioblastoma. Avastin has several known side effects, and more research is needed to determine the risk versus benefit of this drug in malignant gliomas (Vargo et al., 2011).

For older patients with glioblastoma multiforme, important questions have arisen about the timing of chemotherapy. Specifically, should chemotherapy for older individuals be delayed until there is evidence that the tumor is growing again after radiation therapy, thus allowing the patient time to recover strength? Or should chemotherapy begin immediately after radiation therapy with different therapy provided when the tumor starts to grow again? Long-term results of randomized trials in high-risk, low-grade gliomas and anaplastic oligodendroglial tumors have shown that the addition of certain chemotherapeutic agents to radiation therapy after surgery have extended survival (National Cancer Institute, 2015).

Nursing Diagnoses

Patient with a Cerebral or Cerebrovascular Disorder

- Decreased intracranial adaptive capacity related to cerebral lesion
- Disturbed body image related to alteration in body function
- Disturbed sensory perception related to neurological deficit
- Ineffective tissue perfusion: cerebral related to increased intracranial pressure
- Self-care deficit (need to specify) related to paralysis
- Risk for impaired swallowing related to neuromuscular dysfunction
- Risk for aspiration related to reduced level of consciousness and depressed cough and gag reflexes
- Risk for injury related to seizure disorder and disturbed sensory perception

Cerebrovascular Disorders

Cerebral Vascular Accident

Stroke, also referred to as **cerebral vascular accident (CVA)**, or **brain attack**, is a decrease in blood flow and oxygen to brain cells with the subsequent loss of neurological functioning. Causes of stroke are classified as ischemic (disruption of blood flow to part of the brain due to a thrombus or embolus), which accounts for 80% of strokes; and hemorrhagic (loss of blood flow due to rupture of cerebral vessels), which accounts for 20%. The extent of damage to the brain cells varies according to the length of time blood flow is disrupted, the area of the brain affected, and the size of the area involved. An extensive disruption of blood flow to the brain can result in severe disability or death. Stroke is the fifth leading cause of death (130,000/year) and a leading cause of long-term disability (costing an estimated \$34 billion) in the United States (CDC, 2015). Complications related to stroke resulting in morbidity and mortality are very common and often include recurrent stroke.

The American Heart Association (AHA)'s 2020 goal is to increase the cardiovascular health of all Americans by 20% and to decrease deaths by cardiac disease and stroke by 20% (AHA, 2013). To meet these outcomes, it recommends the establishment of acute stroke centers. Certified Comprehensive and Primary Stroke Centers offer an advanced level of specialized stroke care, including specially trained stroke teams, clinical practice guidelines for ischemic and hemorrhagic stroke and TIA and stroke resources focusing on inpatient and rehabilitative care. Get with the Guidelines Stroke (GWTG-S), sponsored by the AHA/ASA, is a quality improvement program that uses performance measures to ensure evidence-based care of patients hospitalized with stroke. The program offers hospitals web-based tools and resources as a means to enhance patient outcomes.

Risk Factors

The single most important controllable risk factor for stroke is high blood pressure. Many providers believe that effective treatment of high blood pressure is an important reason for the declining death rates for stroke. Age is one of the strongest uncontrollable risk factors for stroke with chances of having a stroke doubling for each decade of life after age 55. Atrial fibrillation (AF) is the most common dysrhythmia associated with ischemic stroke. Blood clots can develop in the quivering of the atria. Because these clots are a common cause of embolic strokes, most patients in AF now receive an anticoagulant (Ver Hage, 2011).

Increased total cholesterol and decreased high-density lipoprotein can increase the risk of an ischemic stroke due to plaque buildup in the artery walls. Hyperinsulinemia or increased insulin resistance may result in arterial stiffening, which can increase the risk of ischemic stroke. Patients who smoke or are physically inactive and/or obese should be counseled on how these risk factors can be controlled to decrease the chances of having a stroke. Other modifiable risk factors for stroke include high-fat diet, excessive alcohol intake, and drug abuse. Strokes caused by drug abuse are often seen in a younger population.

Pathophysiology

Because the brain is so metabolically active, pathophysiologic changes begin seconds after a reduction in blood flow and oxygen supply to the cerebral neurons. Cellular metabolism stops as glucose, glycogen, and adenosine triphosphate (ATP) are depleted, resulting in failure of the sodium-potassium pump. Cerebral blood vessels swell, resulting in further decreased blood flow. Vasospasm and increased blood viscosity can result in obstruction to blood flow, even after circulation is restored. When brain cells are

damaged, function of the body parts they control is impaired or lost, causing paralysis, speech and sensory problems, memory and reasoning deficits, coma, and possibly death. The degree of damage to the brain cells depends on the size of the perfusion deficit, the amount of brain tissue that is infarcted, and the type of stroke.

The two major categories of stroke, hemorrhagic and ischemic, cause decreased perfusion to the cerebral tissue in different ways. Hemorrhagic stroke is defined as rupture of a weakened blood vessel causing bleeding into the surrounding brain. This blood accumulates and compresses the affected brain tissue, causing decreased perfusion to those brain cells. Ischemic stroke occurs as a result of an obstruction to a blood vessel supplying nutrients and oxygen to the brain. This focal ischemia is seen with reduced blood flow to a particular brain region or regions. Ischemic stroke can also be caused by hypoperfusion, a diffuse decrease or cessation of blood flow to the brain, but is referred to as a global cerebral ischemia because it affects a large area of the brain (Columbia University Medical Center, 2015). Hemorrhagic stroke can further be broken down into intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH). Thrombotic and embolic are the two main types of ischemic stroke with hypoperfusion also causing ischemia of cerebral tissue.

Hemorrhagic Stroke

Intracerebral Hemorrhage (ICH)

ICH is usually derived from bleeding of small arteries or arterioles directly into the brain, forming a localized hematoma that spreads along white matter tracts. Blood accumulates over minutes to hours as the hematoma enlarges, resulting in the progressive development of neurological symptoms. In contrast to brain embolism and subarachnoid hemorrhage, the neurological symptoms do not begin

Bariatric Considerations

The higher a person's degree of obesity, the higher their risk of stroke—regardless of race, gender, and how obesity is measured (Yatsuya et al., 2010).

Incidence rates for stroke differ substantially between whites and blacks by BMI. The stroke rate in the lowest BMI category was 1.2 per 1,000 person-years for overweight white women and 4.3 per 1,000 person-years for overweight black women. The rate in the highest BMI category was 2.2 for severely obese white women, but 8.0 for severely obese black men.

When waist-to-hip ratio was used as the measure of obesity instead of BMI, those risk ratios ranged from 1.65 to 3.19 and 1.69 to 2.55, respectively. Thus, by any

measure of obesity, individuals in the highest category had approximately twice the risk of stroke compared to their overweight counterparts.

Individuals with higher degrees of obesity tend to have higher blood pressure levels and higher diabetes prevalence. The coexistence of these major risk factors explains much of the obesity-stroke association (Yatsuya et al., 2010). The obesity epidemic may be the cause of a sharp increase in stroke incidences in younger Americans. Stroke, once a disease of older adults, appears to be becoming a disease of the young as well. Almost one fourth of strokes occur in people under age 65 and 10% to 15% affect individuals 45 and younger (CDC, 2015).

immediately and are not maximal at onset. The most common causes of ICH are hypertension, illicit drug use (particularly amphetamines and cocaine), vascular malformations, and bleeding diathesis. ICH results from hypertension when the arteries in the brain become brittle, susceptible to cracking, and rupture.

Subarachnoid Hemorrhage (SAH)

SAH is most commonly a rupture of an aneurysm that releases blood directly into the subarachnoid space just outside the brain. The blood spreads rapidly within the CSF, immediately increasing ICP. If bleeding continues, deep coma or death may result. Typically, the bleeding lasts only a few seconds, but there is risk of re-bleeding. The classic symptom is a sudden, severe headache that begins abruptly and is described as “the worst headache of my life”

Ischemic Stroke

Ischemic stroke includes the following subtypes: thrombotic and embolic, and hypoperfusion.

Thrombotic Stroke

Thrombotic stroke occurs when the pathologic process promotes thrombus formation in a cerebral artery, causing infarction and stroke due to decreased blood flow. Disease of the arterial wall, dissection, or fibromuscular dysplasia may cause the obstruction. Atherosclerosis is the most common cause of occlusion within the large extracranial and intracranial arteries that supply the brain.

Embolic Stroke

An **embolic stroke** is caused by particles that arise from another part of the body, resulting in blockage of arterial blood flow to a particular area of the brain. Embolic strokes commonly originate from a source in the heart, aorta, or large vessels. The onset of symptoms is abrupt and maximal, because the embolus suddenly blocks the involved area of the brain. Embolic strokes are divided into four categories related to the cause:

- Known source is cardiac
- An arterial source
- Possible cardiac or aortic source based on transthoracic and/or transesophageal echocardiographic findings
- Unknown source in which these tests are negative or inconclusive

Systemic Hypoperfusion

Systemic hypoperfusion is a general circulatory problem that can occur in the brain and possibly other organs.

Decreased perfusion can be due to cardiac arrest, arrhythmia, pulmonary embolism, pericardial effusion, or bleeding. Hypoxemia may further decrease the amount of oxygen carried to the brain. Symptoms of brain dysfunction are diffuse and less specific as compared to strokes that are thrombotic or embolic in nature.

Manifestations

Strokes typically manifest with the sudden onset of focal neurological deficits resulting from damage to the injured portion of the brain. Classic signs and symptoms include:

- Sudden confusion
- Sudden difficulty understanding or speaking
- Sudden loss of vision out of one eye
- Sudden severe headache
- Sudden weakness of the face, arm, or leg, especially affecting one side of the body

The public should be encouraged to respond to these symptoms as a brain attack, just as they would to the development of chest pain and a heart attack, and immediately activate the Emergency Medical Services (EMS) system. The Cincinnati Prehospital Stroke Scale (Box 11-1) is a valid tool that can be performed by emergency medical personnel and is highly predictive of stroke. Facial droop, arm drift, and speech are the three categories assessed, with abnormal findings used to help identify an acute stroke.

Manifestations of a stroke are usually specific to the area of the brain that has been affected (refer to Figure 11-3). Each of the four major neuroanatomic stroke syndromes results from disruption of the vascular distribution to specific areas of the brain.

Middle cerebral artery (MCA) occlusions commonly produce:

- Hemiplegia (paralysis) of the contralateral side, affecting the lower part of the face, arm and hand
- Hypesthesia (sensory loss) of the contralateral side
- Homonymous hemianopsia (blindness in one half of the visual field) affecting the same half of the visual field in both eyes
- Gaze preference toward the side of the lesion
 - If the left brain is affected, there may be aphasia.
 - If the right brain is affected, unilateral neglect (decreased awareness of one side) may be identified (Tocco, 2011).

Anterior cerebral artery occlusions primarily affect frontal lobe function and can result in:

- amnesia
- speech perseveration

Box 11-1 The Cincinnati Prehospital Stroke Scale

The Cincinnati Prehospital Stroke Scale is a system used to help identify a suspected stroke. If any one of the three tests shows abnormal findings, the patient may be having a stroke and should be transported to a hospital as soon as possible.

1. **Facial Droop:** Ask the person to smile or show his teeth.
 - *Normal:* Both sides of face move equally
 - *Abnormal:* One side of face does not move as well as the other (or at all)
2. **Arm Drift:** Ask the person to close his eyes and hold his arms straight out in front for about 10 seconds.
 - *Normal:* Both arms move equally or not at all
 - *Abnormal:* One arm does not move, or one arm drifts down compared with the other side
3. **Speech:** Ask the person to say, “You can’t teach an old dog new tricks,” or some other simple, familiar saying.
 - *Normal:* Patient uses correct words with no slurring
 - *Abnormal:* Slurred or inappropriate words or mute

- confusion
- impaired judgment
- contralateral leg weakness
- urinary incontinence

Posterior cerebral artery occlusions affect vision and thought, producing:

- homonymous hemianopsia
- unilateral cortical blindness
- visual agnosia
- motor dysfunction
- impaired memory

The last type of stroke syndrome, vertebrobasilar artery occlusion, is difficult to detect because it results in a wide variety of cranial nerve, cerebellar, and brainstem deficits (Ver Hage, 2011). Vertigo, dizziness, nausea, vomiting and head or neck pain are the most common initial symptoms reported.

Patient History and Assessment

Time is of the essence in the assessment of stroke patients. A focused history and neurological exam along with diagnostic tests should detect the stroke mechanism and guide therapy. The single most important point in the

patient’s history is the time that the patient was last seen well (not displaying symptoms) and should be established as soon as possible. Additional important information to obtain is any recent medical, surgical, traumatic, or transient ischemic attack (TIA) events. The nurse reviews the patient’s history to identify any risk factors for strokes and obtains a complete list of medications the patient is taking, especially anticoagulant and antiplatelet agents.

The neurological examination should corroborate findings from the event history and provide a quantifiable, objective way to evaluate neurological changes. The National Institutes of Health (NIH) Stroke Scale (Table 11-2) assessment should be performed by a certified healthcare provider as soon as a stroke is suspected. It is a valid, reliable tool that measures the severity of neurological dysfunction in stroke patients and trends the assessment of changes in neurological deficits over time. An experienced provider can score all of the items in 7 minutes. The NIH Stroke Scale examines visual, motor, sensory, cerebellar, inattention, language, and level of consciousness (LOC) functioning. A maximum score of 42 signifies a severe stroke, whereas a score of 0 indicates a normal exam.

Diagnostic Criteria

All patients with a suspected stroke should have the following tests as soon as possible following admission to the emergency department (ED):

- Noncontrast brain CT or brain MRI (see neuroimaging)
- Serum glucose (Hypoglycemia can present with neurological deficits mimicking stroke, and severe hypoglycemia can cause neuronal damage. Hyperglycemia is common in patients with acute ischemic stroke and is associated with a poorer prognosis.)
- Prothrombin time (PT) and international normalized ratio (INR)—anticoagulant use is a common cause of intracerebral hemorrhage
- Electrocardiogram (ECG) used to diagnose any cardiac dysrhythmias or myocardial infarction (MI)
- Complete blood count (CBC) including platelets (Platelet count is used to rule out thrombotic thrombocytopenic purpura [TPP].)
- Cardiac enzymes and troponin
- Electrolytes, urea nitrogen, creatinine (Hyponatremia [Na less than 135 mEq/L] is found in 10% to 40% of patients with subarachnoid hemorrhage.)
- Partial thromboplastin time (PTT)
- Oxygen saturation

Table 11-2 National Institutes of Health Stroke Scale

Category	Description	Score	
1a	Level of consciousness (LOC) A 3 is scored only if the patient makes no movement (other than reflexive) in response to noxious stimulation.	Alert	0
		Drowsy	1
		Stuporous	2
		Coma	3
1b	LOC questions (month, age) The answer must be correct—there is no partial credit for being close.	Answers both correctly	0
		Answers 1 correctly	1
		Incorrect on both	2
1c	LOC commands (open–close eyes, grip and release hand) Substitute another one step command if the hands cannot be used.	Obeys both correctly	0
		Obeys 1 correctly	1
		Incorrect on both	2
2	Best gaze (follow finger) Only horizontal eye movements will be tested.	Normal	0
		Partial gaze palsy	1
		Forced deviation	2
3	Best visual (visual fields) Visual fields (upper and lower) quadrants) are tested by confrontation, using finger counting or visual threat.	No visual loss	0
		Partial hemianopsia	1
		Complete hemianopsia	2
		Bilateral hemianopsia	3
4	Facial palsy (show teeth, raise brows, squeeze eyes shut) Score symmetry of grimace in response to noxious stimuli in the poorly responsive/non-comprehending patient.	Normal	0
		Minor	1
		Partial	2
		Complete	3
5	Motor arm left* (raise 90°, hold 10 seconds) The limb is placed in the appropriate position; extend the arms (palms down) 90 degrees (if sitting) or 45 degrees (if supine). Each limb is tested in turn beginning with the non-paretic are.	No drift	0
		Drift	1
		Cannot resist gravity	2
		No effort against gravity	3
		No movement	4
6	Motor leg left* (raise 30°, hold 5 seconds) The limb is placed in the appropriate position: hold the leg at 30 degrees (always tested supine). Each leg is tested in turn beginning with the non-paretic leg.	No drift	0
		Drift	1
		Cannot resist gravity	2
		No effort against gravity	3
		No movement	4
7	Limb ataxia (finger–nose, heel–shin) Test with eyes open. In case of visual defect, test in intact visual field.	Absent	0
		Present in 1 limb	1
		Present in 2 limbs	2
8	Sensory (pinprick to face, arm, leg) Only sensory loss attributed to stroke is scored as abnormal.	Normal	0
		Partial loss	1
		Severe loss	2
9	Best language** (name items, describe pictures) For this scale item, the patient is asked to describe what is happening in the picture provided, to name the items on the naming sheet and read sentences.	No aphasia	0
		Mild to moderate aphasia	1
		Severe aphasia	2
		Mute	3
10	Dysarthria (speech clarity to “mama, baseball, huckleberry, tip-top, fifty-fifty”) As adequate sample of speech must be obtained by asking patient to read or repeat words from the list provided.	Normal articulation	0
		Mild to moderate dysarthria	1
		Near to unintelligible or worse	2
11	Extinction/neglect (double simultaneous testing) Sufficient information to identify neglect may occur during prior testing.	No neglect	0
		Partial neglect	1
		Complete neglect	2
	Total	—	0–42

* For limbs with amputation, joint fusion, etc., score 9 and explain.

** For intubation or other physical barriers to speech, score 9 and explain. Do not add 9 to the total score.

Neuroimaging

In the evaluation of the acute stroke patient, imaging studies are necessary to rule out hemorrhage as a cause of the presenting symptoms. They are also beneficial in determining the degree of brain injury and to identify the vascular lesion accountable for the ischemic deficit. Brain imaging studies are imperative to differentiate ischemic stroke from hemorrhage and to determine vascular distribution of the ischemic lesion.

Computerized Tomography. CT is the current minimal standard imaging study to rule out hemorrhagic events and to identify patients who are eligible for rtPA therapy. It should be performed within 25 minutes and interpreted within 20 minutes of the patient's arrival to the hospital ED. If the CT scan is positive for a hemorrhagic stroke, an immediate neurosurgical consult should be ordered. In the case of ischemic stroke, intravenous thrombolysis (rtPA) should be administered if the time since the patient was last seen well (without symptoms) is less than 3 hours (or 4.5 hours in some facilities) and the patient is eligible based on criteria.

Magnetic Resonance Imagery. MRI can immediately provide information in regard to blood flow in vascular territories and specific brain regions. More specifically, it can determine the size of a perfusion deficit and identify brain tissue that may be ischemic but not infarcted and potentially viable tissue.

Magnetic Resonance Angiography Magnetic resonance angiography (MRA) is a noninvasive and effective test to visualize abnormalities of the intracranial and the extracranial cerebral circulation.

Computed Tomography Angiography

Computed Tomography Angiography (CTA) is considered the "gold standard" for detecting cerebral aneurysms, arteriovenous malformations (AVMs), and arteriovenous fistulae (AVFs). It can also measure the exact degree of stenosis in extracranial and intracranial arteries and guide decisions regarding the use of recannulization therapy. It can be performed more rapidly than MRI and is often better tolerated by patients.

Carotid Ultrasound. Carotid ultrasound is a noninvasive and inexpensive test used to detect occlusions of the extracranial carotid and vertebral arteries. It can be used in patients in whom an MRA is contraindicated (pacemaker, metal implants, etc.) or who are unable to receive contrast material.

Transcranial Doppler. Transcranial doppler (TCD) is a noninvasive and low-cost technique for imaging the large

intracranial vessels at the base of the skull. It is used in patients with acute cerebral ischemia to detect intracranial stenosis and occlusions. It may also be used to detect vasospasms in patients with neurological deterioration from a subarachnoid hemorrhage.

Transthoracic and Transesophageal Echocardiography. Transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) use may be indicated based on the high percentage of strokes that are of cardioembolic origin. TTE is a routine test used to view the heart for the presence of clots, valvular abnormalities, and left ventricular function. TEE is a highly sensitive test for detecting cardiac and aortic lesions that may cause ischemic strokes.

COLLABORATIVE CARE DURING EMERGENT PHASE

In recent years, stroke has changed from being called a cerebrovascular accident to an infarct of specific causality. By understanding the underlying mechanism, appropriate measures can be instituted to improve acute care and foster positive long-term outcomes. The immediate goals of collaborative management for the patient with a stroke include minimizing brain injury and preventing medical complications.

Emergent Care

Treatment is ideally initiated in the pre-hospital area with rapid recognition of stroke symptoms and immediate transport to a stroke center. This collaboration in stroke care continues in the ED where clinical identification, testing, and treatment is immediately initiated. Inpatient stroke care is best administered in an ICU where the multidisciplinary team is able to foresee and respond rapidly to complications related to airway patency and breathing pattern, cardiovascular status (cardiac rhythm and rate, blood pressure), and serious neurological deficits.

NURSING ACTIONS

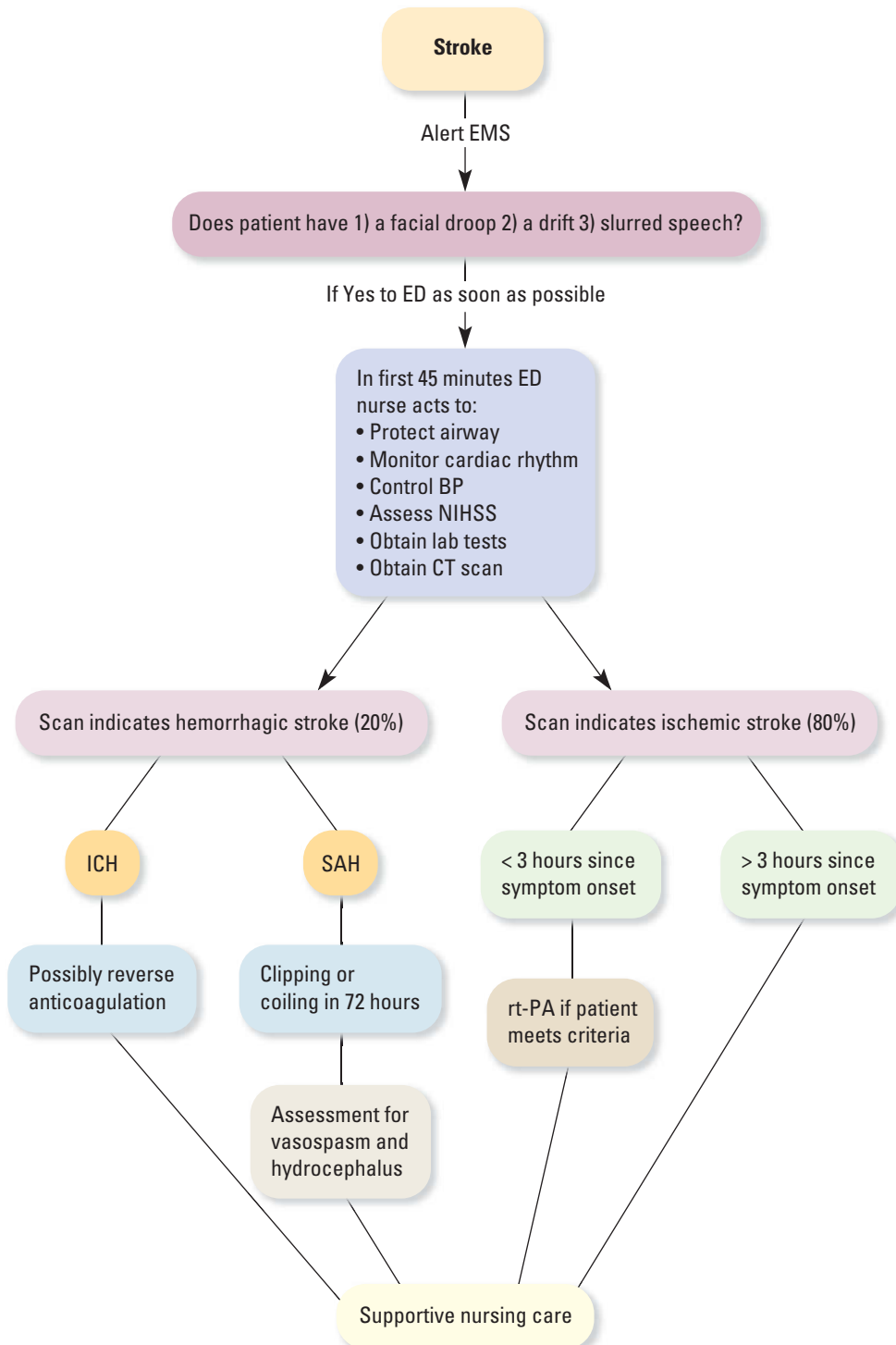
Airway and Breathing Management

The goal of airway management is to prevent hypoxia, hypoventilation, and worsening cerebral injury. Impaired consciousness may result in partial airway obstruction or aspiration of saliva and other secretions. Positioning the patient on his side may help to open the airway. However, intubation may be required in some patients to reestablish adequate ventilation.

Essential for Collaboration

Stroke patients who require intubation are usually sicker, and 50% die in the first 30 days following stroke. The nurse collaborates with the pulmonologist and respiratory therapist to determine the goals of care for the patient.

The nurse should monitor all stroke patients' oxygen saturations. Oxygen (O₂) therapy is indicated when an arterial blood gas (ABG) or O₂ saturation less than 92% (or per medical provider) suggests hypoxia. It is prudent to use the least amount of oxygen required to maintain an adequate O₂ saturation.



Cardiac Monitoring

Ongoing cardiac monitoring is imperative for detecting signs of related acute cardiac ischemia. The ECG can detect acute or chronic arrhythmias such as AF that may have precipitated an embolic event. The stroke itself can cause arrhythmias when the sympathetic response results in demand-induced myocardial ischemia. Lastly, there may be centrally mediated changes in the ECG when large strokes occur, particularly with a subarachnoid hemorrhage.

Monitoring for Hyperthermia

Fever may promote further brain injury in patients with an acute stroke. One study showed that body temperature was independently related to the severity and size of the infarct of the initial stroke, and that for each 1°C increase in body temperature the relative risk of a poor outcome increased by 2.2. Treatment includes finding the source of fever and immediately treating with antipyretic agents. Patients who have sustained a stroke are often provided acetaminophen as soon as their temperature reaches 38°C (100.4°F).

Blood Pressure Management

Acute management of blood pressure (BP) will vary depending on the type of stroke. A neuroimaging study with CT or MRI is vital to help determine how to manage the BP of stroke patients.

Ischemic Stroke

Hypertension occurs frequently with acute ischemic stroke and needs to be closely monitored. In patients experiencing an ischemic stroke, the perfusion pressure distal to the occluded vessel is reduced and the cerebral vessels are dilated. The mean arterial pressure (MAP) is commonly elevated in patients with an acute stroke due to an immediate sympathetic response or chronic hypertension. The elevated BP may be necessary to maintain brain perfusion, and rapidly lowering it could cause neurological deterioration.

Many patients have a spontaneous decline in BP within the first 24 hours, so antihypertensive agents may not be needed. When the BP remains elevated, the nurse should first investigate if there are other factors such as pain or discomfort from bladder distention that are contributing to the patient's hypertension. Specific goals for BP management are individualized for each patient and type of stroke. When treatment is determined to be necessary, cautious lowering of the BP by about 15% during the first 24 hours after the onset of stroke is recommended with continual reassessment of neurological function.

Special recommendations apply to BP control in patients with ischemic stroke who are eligible for thrombolytic therapy. Before lytic therapy is started, it is suggested that SBP be less than or equal to 185 mmHg and DBP be less than or equal

to 110 mmHg (Ottwell, Phillippe, & Dixon, 2010). To accomplish this, the nurse might administer labetalol (Normodyne) 10 to 20 mg IV over 1 to 2 minutes. The dose may be repeated once. The goal is to maintain the BP below 180/105 for a minimum of 24 hours after intravenous tPA treatment. If the blood pressure does not drop to an appropriate level, the patient will usually be excluded as a candidate for thrombolytic therapy.

Hemorrhagic Stroke

An elevation in BP may increase the bleeding in patients with a hemorrhagic stroke. The benefits and risks of BP management are examined to determine the appropriate treatment. Decreasing the BP in patients with ICH or SAH may produce benefits by preventing further bleeding and additional vascular damage. When ICP monitoring is in place, the BP may be reduced to lower levels because the cerebral perfusion pressure may be calculated (see Chapter 10 ). In patients with ICH and normal cerebral perfusion, the BP can be lowered by at least 15% without causing ischemia to the tissue surrounding the affected area.

There is not sufficient evidence to support a specific therapy for hypertension in the patient with a SAH. An elevated BP can aggravate a SAH because the direct force across the plugged bleeding area is related to the pressure difference between the CSF and the systemic blood pressure. In contrast, lowering the BP may reduce the risk of re-bleeding from an aneurysm; however, ischemia may result due to inadequate cerebral perfusion. The recommendation of BP control for hemorrhagic stroke is to maintain the SBP between 140 and 160 mmHg and to carefully monitor the patient for signs of cerebral hypoperfusion, which is caused by a fall in BP. An SBP of greater than 170 mmHg should be treated with intravenous medications such as labetalol (Normodyne), nicardipine (Cardene), or nitroprusside (Nitropress).

Determining Diagnosis

Time is crucial in the evaluation of the stroke mechanism because it will determine therapy choices. In most cases, a patient history, physical examination with NIH stroke scale, and noncontrast CT scan are adequate. Differential diagnosis is necessary to rule out conditions that mimic stroke, such as drug overdose, migraine, head trauma, brain tumor, systemic infection, hypoglycemia, hyponatremia, and thrombotic thrombocytopenic purpura. A thorough history will determine if the patient is on anticoagulant therapy, a common contributor to intracerebral hemorrhage.

Collaborative Care after Diagnosis Is Determined

The goal of acute stroke management is rapid and efficient care. After assessment of the patient's airway, breathing, and circulation (ABCs) and a stroke evaluation, it should

be determined if the patient is suffering an ischemic stroke and is a candidate for thrombolytic therapy. If the patient is eligible, therapy should be administered within 1 hour from the patient's presentation to the ED. The National Institute for Neurological Disorders and Stroke (NINDS) has recommended the following time benchmarks for the potential thrombolysis candidate:

Door to doctor	10 minutes
Access to neurological expertise	15 minutes
Door to CT scan completion	25 minutes
Door to CT scan interpretation	45 minutes
Door to treatment	60 minutes
Admission to monitored bed	3 hours

Ischemic Stroke

In the United States, the vast majority of strokes, more than 80%, are ischemic. Revascularization (reestablishment of blood flow through the artery) is the most critical aspect of treatment for a patient with an ischemic stroke.

Medical Management. When patients arrive for medical assistance within the first hours following the onset of symptoms, it may be possible to utilize a thrombolytic to reestablish blood flow through the involved cerebral artery. *Thrombolysis*, the administration of recombinant tissue-type plasminogen activator (rt-PA or alteplase), ideally dissolves the clot in the cerebral artery, restores blood flow, and improves neurological functioning. Alteplase should be given as soon as possible after the onset of symptoms, but usually within 3 hours of when the patient was last seen without symptoms.

Essential for Safety

The AHA/ASA has recommended expanding the administration time to 4½ hours for qualified patients (Keefe, 2009) and many stroke centers in the United States are doing this. Because tPA administration in the 3- to 4½-hour time frame is not approved by the FDA, it is important to be aware of individual hospital policies, protocols, consents, and additional exclusion criteria.

In addition, a patient might be considered for either intravenous or intra-arterial thrombolysis beyond the 4.5-hour window, depending on where the cerebral occlusion is after the development of symptoms, if imaging tests show a substantial at-risk brain that is underperfused but not yet infarcted, and there is an occlusive thromboembolus.

Essential for Quality Assurance

Stroke is the AHA/ASA's newest quality improvement initiative, which provides information and tools to improve door-to-needle time of administering tPA to qualified patients.

Inclusion criteria for patients who are candidates for thrombolysis may include:

- Age 18 years or more
- Diagnosis of ischemic stroke with onset of symptoms within previous 3 hours
- CT or MRI show no evidence of ICH
- Blood pressure in acceptable range (less than 185/110)
- Serum glucose between 50 to 400 mg/dL, platelets < 100,000/mm³, INR > 1.7
- No head trauma, MI, or stroke within the previous 3 months
- No history of ICH, AVM, or aneurysm
- No urinary or GI bleed within the previous 3 weeks
- No major surgery or trauma in the past 2 weeks
- No arterial puncture or lumbar puncture within previous week
- Not received heparin within 48 hours with elevated PTT
- Not currently pregnant
- Currently, any patient on a direct thrombin inhibitor or Factor Xa inhibitor is not a candidate for tPA per guidelines of the AHA/ASA.

Invasive and Surgical Management

Blood flow to ischemic cerebral tissue may also be reestablished by interventional radiology, a specialty that uses an endovascular approach and views the blood vessels that supply the nervous system from inside the vessel. Two endovascular approaches to help restore normal blood flow are thrombolysis and embolectomy. In an arterial thrombolysis, alteplase may be administered directly to the site of obstruction for up to 6 hours after onset of symptoms. Embolectomy restores blood flow in stroke patients by removing the clot. This is accomplished using FDA-approved devices that are minimally invasive and are performed under fluoroscopy to extract clots in interventional radiology units. The procedures can be performed within 8 hours of symptom onset. The MERCI (Mechanical Embolus Removal in Cerebral Ischemia) uses a balloon-guided catheter that is guided up through the femoral artery into the cerebral vasculature to the site of the clot, where a retriever is used to capture the clot. The Penumbra System differs from the MERCI in that it breaks up the clots and then aspirates the fragments. The Trevo Stent Retriever and Covidien Solitaire Stent Retriever System enable entrapment of the thrombus between the stent and the blood vessel wall, resulting in rapid recanalization and re-establishment of blood flow and oxygen supply before the clot is retrieved. They also allow thrombectomy to be performed.

Other surgical options for preventing and treating an ischemic stroke include carotid endarterectomy, angioplasty,

and carotid stenting. Carotid endarterectomy is a procedure that cleans out and opens up the narrowed artery. The surgeon scrapes away the plaque from the wall of the artery, allowing for increased blood flow to the brain. Angioplasty is a procedure performed in an interventional radiology unit, and the approach is as described for embolectomy. Using fluoroscopy and a contrast agent, the physician obtains angiograms of the lesion to determine the baseline cerebral circulation. A tiny balloon at the end of a catheter is advanced through the artery to the blockage then inflated to open the artery. Once the vessel is dilated, an intracranial stent may be placed inside the artery to hold it open and to maintain increased blood flow. Multimodal reperfusion therapy (MMRT) is a combination of angioplasty, surgical treatments, stent placement, and intra-arterial infusion of thrombolytics and/or antiplatelet agents. Recent studies show that MMRT increases recanalization and reperfusion rates (Cohen et al., 2011).

After the approximately 1-hour procedure, the nurse needs to carefully monitor the patient's BP because unstable BP is a common occurrence. The patient is usually monitored because bradycardia develops frequently. At regular

intervals beginning with every 15 minutes, the nurse assesses the patient's neurological status (see earlier) and hemodynamic status.

Hemorrhagic Stroke

In the United States, about 20% of all strokes are hemorrhagic and account for the most common cause for individuals 18 to 45 years of age. There are two primary types of hemorrhagic strokes: ICH and SAH.

Intracerebral Hemorrhages

Of the hemorrhagic strokes, up to 41% may be ICH. It is recommended that patients with ICH be cared for in ICU because patients experience frequent increases in ICP and usually have additional medical issues. Immediate complications include cerebral hypoxia, decreased cerebral blood flow, and increased risk of further bleeding (Taft, 2009). Hydrocephalus is also a common complication of ICH. A ventriculostomy may need to be performed and an external ventricular drain inserted to drain CSF. Review

Commonly Used Medications

Thrombolytic Therapy rt-PA (Tissue Plasminogen Activator, Recombinant) Alteplase, Activase

Desired Effect: rt-PA is an enzyme that is used to restore blood flow, minimize the ischemic penumbra, and limit infarction volume. It binds to the fibrin in a thrombus and converts the trapped plasminogen to plasmin, which triggers local fibrinolysis and clears the blocked artery, restoring circulation. The usual dose of rt-PA is 0.9 mg/kg (maximum dose 90 mg) administered over 60 minutes with the first 10% given over 1 minute as a bolus. Onset of action is prompt with patency of the vessel usually occurring within 1 to 2 hours (Keefe, 2009).

Nursing Actions:

- The nurse reviews the criteria with the medical team and determines that the patient is a candidate for thrombolysis.
- Patients may not be able to consent to thrombolytic therapy due to their neurological impairment. This should not prevent them from receiving the therapy, and both patients and their families need extensive education on the possible risks and benefits.
- It is best to establish a separate IV for administration. It is recommended to have at least 2 large bore IVs established before the administration of rtPA.
- The patient receiving rtPA should be admitted to ICU. Focused neurological assessments and measurements of BP are required. The usual protocol for vital signs and neurological (neuro) signs following the administration of rtPA is:

- Vital signs and Neuro signs Q 15 minutes x 2 hours, Q 30 minutes x 6 hours and Q 1 hour x 16 hours.
- The infusion should be discontinued if the patient develops severe headache, hypertension, or nausea and vomiting because these may indicate intracranial bleeding.
- The nurse should delay placing a nasogastric (NG) tube, Foley catheter, or arterial line during administration of the thrombolytic. These should be considered prior to the start of rtPA.
- A foley should only be inserted if the patient is in need of close fluid status monitoring because of critical illness, develops urinary retention or bladder flow obstruction, or will require prolonged immobilization per CAUTI guidelines.
- A bedside nursing dysphagia screen or Speech Therapy dysphagia evaluation must be completed before anything can be given by mouth.
- A follow-up CT scan is indicated 24 hours after administration.

Potential Side and/or Toxic Effects: Bleeding is the most common side effect. It may be internal such as GI or GU, or may be external such as at intravenous (IV) sites or from the gums and nose. Approximately 6% of patients receiving thrombolytic therapy develop intracranial hemorrhage usually during the first 36 hours.

management of the ventilated patient with increased ICP and external ventricular drain, as discussed in Chapter 10.

Therapy targeted to management of the intracerebral hematoma is limited. If the patient has been receiving an anticoagulant, the appropriate drug to reverse its effects might be administered. Protamine sulfate is used to reverse heparin-associated ICH with the dose dependent on the time since the cessation of heparin, whereas intravenous vitamin K in combination with fresh frozen plasma is used to reverse the effects of warfarin. New, faster acting medications are currently in various stages of development and approval for the reversal of anticoagulants. A craniotomy for hematoma removal might be performed, especially if the bleeding occurred near the surface of the brain. Other therapies may include injecting a thrombolytic inside the hematoma to facilitate endoscopic removal of the blood, and using recombinant factor VIIa to reduce hematoma size.

Subarachnoid Hemorrhages

The most common cause of bleeding in the subarachnoid space is rupture of an aneurysm or an AVM. Major contributing factors in the development and eventual rupture of cerebral aneurysms are prolonged hemodynamic stress and local arterial degeneration at vessel bifurcations. Nearly 90% of intracranial aneurysms arise on the anterior (carotid) circulation. Common locations include the anterior communicating artery, the internal carotid artery at the posterior communicating artery origin, and the MCA bifurcation (Taft, 2009).

In most instances, the presence of an aneurysm is not known until the aneurysm ruptures and the patient presents with a SAH. The focused assessment of the patient with a ruptured cerebral aneurysm includes a careful neurological assessment with grading of severity by the Hunt and Hess scale. The scale uses clinical findings to measure the severity of the hemorrhage on admission and has been shown to correlate with patient outcome. The grades of the Hunt and Hess scale are:

- Grade 0—Unruptured aneurysm
- Grade 1—Asymptomatic or minimal headache and slight nuchal rigidity
- Grade 1A—No acute meningeal or brain reaction but with fixed neurological deficit
- Grade 2—Moderate-to-severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy
- Grade 3—Drowsiness, confusion, or mild focal deficit
- Grade 4—Stupor, moderate-to-severe hemiparesis, possible early decerebrate rigidity, and vegetative disturbances
- Grade 5—Deep coma, decerebrate rigidity, and moribund appearance

Signs and symptoms of aneurysms other than those associated with SAH are relatively uncommon. However, a posterior communicating aneurysm may result in a third nerve palsy, and giant aneurysms may result in focal symptoms because of their mass effect (Taft, 2009).

Diagnostic Studies

Possible diagnostic studies include a noncontrast CT scan, an MRA, angiography, and TCD. These tests are described earlier in the chapter.

COLLABORATIVE CARE

Mortality and morbidity are high following rupture of a cerebral aneurysm. After the diagnosis of stroke is determined, there is a brief period of time in which viable brain tissue can be saved. The trend for surgical treatment of a SAH is toward performing surgery early versus previous practices of delaying surgery.

Essential for Safety

Research demonstrates that surgery in Grades 3 and 4 patients within 48 hours of SAH, is associated with better outcomes than surgery performed between 3 and 6 days after SAH. Ruptured aneurysms are most likely to rebleed within the first day, and the risk remains very high for 2 weeks if the aneurysm is not repaired (Mahaney, Todd, Bayman, & Torner, 2011).

In the past, most patients were treated with craniotomy and surgical clipping of the aneurysm. In this procedure, after the patient is anesthetized, the skull is opened and the aneurysm is located. The neurosurgeon isolates the affected blood vessel and places a small, metal clip on the neck of the aneurysm, restricting its blood supply. The clip remains in place, preventing future bleeding (♦ Figure 11-4). When this is not anatomically feasible, the aneurysm may be reinforced by wrapping it to provide support and induce scarring.

Less invasive procedures include endovascular embolization. Once the patient has been anesthetized, the doctor inserts a catheter into an artery (usually the femoral) and threads it to the site of the aneurysm. Using a guide wire,

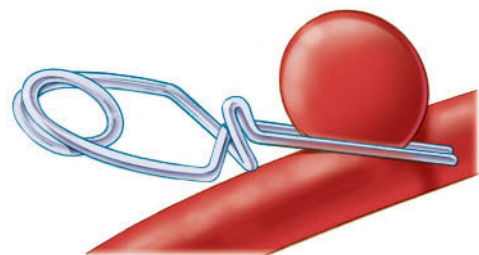


Figure 11.4 Aneurysm with clip.

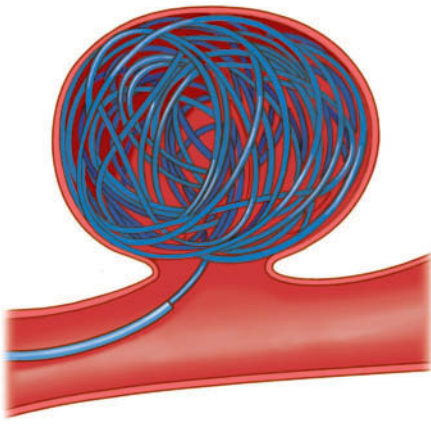


Figure 11.5 Aneurysm with Guglielmi coils.

detachable coils of platinum wire are passed through the catheter and released into the aneurysm. The process is continued until angiography demonstrates that the coils have obliterated the aneurysm (♦ Figure 11-5). The most common coils used in endovascular procedures are platinum Guglielmi detachable coils. The purpose of the coils is to induce thrombosis in the aneurysm via electrothrombosis. Electrothrombosis occurs because white and red blood cells, platelets, and fibrinogen are negatively charged. When the positively charged platinum coils are inserted in the aneurysm, they attract the negatively charged blood components and a clot is formed. Studies comparing the two techniques have found coiling to be safer but slightly less durable than clipping. However, if necessary, coiling may be performed more than once during a patient's lifetime (Sherif et al., 2009).

Nursing care after obliteration of a ruptured aneurysm is focused on accurate neurological assessments and prevention of complications. Following treatment, the patient remains at high risk for two of the major complications of aneurysm rupture: **vasospasm** and **hydrocephalus**.

Vasospasm

Now that early intervention limits the amount of re-bleeding, vasospasm is the most feared complication. In the case of ruptured aneurysms, cerebral vasospasm accounts for about 20% of patients with severe disability or death (Agrawal et al., 2009). Vasospasm is defined as an angiographic narrowing of cerebral blood vessel(s) that can lead to delayed ischemia. The Fisher grade, which describes the amount of blood seen on a noncontrast head CT, is useful for identifying the likelihood the patient will develop vasospasm. Fisher grades are:

1. No blood detected
2. Diffuse or vertical layers less than 1 mm thick
3. Localized clot or vertical layer greater than or equal to 1 mm
4. Intracerebral or intraventricular clot with diffuse or no SAH

Vasospasm is most likely to occur in patients with a Fisher grade 3 and a high grade on the Hunt and Hess scale. Most patients who have survived rupture of a cerebral aneurysm receive a calcium channel blocker, usually nimodipine (Nimotop) for 21 days. Initially nimodipine was administered because it was thought to prevent vasospasm. Although this mode of action has not been confirmed, nimodipine has been shown to have a neuroprotective effect after subarachnoid hemorrhage and is still administered routinely to patients.

The nurse assesses for the presence of vasospasm when doing routine neurological signs. Signs and symptoms may include:

1. Deterioration in mental status (restlessness or lethargy)
2. Development of focal neurological deficits (hemiparesis, dysphasia)
3. Fever
4. Neck stiffness

Symptoms may wax and wane, changing from minute to minute. They tend to become more apparent when the patient's BP drops and less obvious when the BP increases. TCD and CA studies might be utilized to confirm the diagnosis.

Aggressively Managed Vasospasm

Vasospasm must be aggressively managed once it is detected to prevent permanent disability and death. Current management is triple-H (HHH): hypertension, hypervolemia, and hemodilution therapy. The theory behind the treatment is that the only way to increase blood flow to cerebral tissue during vasospasm is to increase the BP. The simplest way to achieve all three aims of therapy simultaneously is through volume expansion. Precise parameters for volume expansion have not been defined so treatment varies between institutions. Most authorities recommend placement of a pulmonary artery (PA) line to guide fluid administration but target values for PA pressures and the fluid used to achieve volume expansion vary. Hemodilution usually results from the hypervolemia. If the patient does not achieve a BP of 10 to 60 mmHg above baseline or 150 to 200 mmHg systolic (depending on institutional policy) by volume expansion alone, then hypertension might be attained by the use of a vasoactive drug such as dopamine (Intropin) or phenylephrine (Neo-Synephrine). When HHH therapy fails, transluminal balloon angioplasty is the method of choice, but intra-arterial papaverine may be used for vasospasm in the distal vasculature, where balloons may not be able to access. The instillation of sodium nitroprusside (SNP) into the ventricles is currently being tested in the treatment of cerebral ischemia and delayed vasospasm refractory to conventional treatment (Agrawal et al., 2009).

Hydrocephalus

The third common major neurological complication following rupture of a cerebral aneurysm is hydrocephalus. It develops when blood in the subarachnoid space obliterates the arachnoidal villi, preventing absorption of CSF, or blood within the ventricles blocks the foramen of Monro, preventing drainage of CSF. If hydrocephalus leads to an increase in ICP and deterioration of the patient's neurological status, an external ventricular drain might be emergently placed for CSF diversion. The nurse anticipates that the patient's neurological examination will improve dramatically after the drain has been placed and hydrocephalus has been treated. In some cases, the hydrocephalus is persistent and the patient requires a ventriculoperitoneal shunt (Rincon et al., 2010).

Essential for Quality Assurance

Research has identified predictors of long-term shunt-dependent hydrocephalus. They include admission CT findings of Fisher Grade 4, fourth ventricle hemorrhage, hyperglycemic at admission, and development of hospital-acquired meningitis (Rincon et al., 2010).

NURSING CARE OF THE STROKE PATIENT

Ongoing neurological assessments and seizure precautions are necessary components of nursing care of the stroke patient. A nursing priority is to monitor for complications such as cerebral edema or intracerebral hemorrhage that can result in increased intracranial pressure (IICP). During the first few days, the nurse carefully assesses the patient's neurological status because cerebral edema usually peaks within 3 to 5 days post-stroke (Ver Hage, 2011). If more aggressive therapies such as osmotic diuretics or CSF drainage are required, the patient usually requires ICP monitoring.

Small-vessel hemorrhage or hemorrhagic transformation is a grave concern for patients with an embolic stroke. Patients who develop hemorrhagic transformation, like patients with progressive cerebral edema, will demonstrate acute clinical decline. Therefore, patients need to be thoroughly monitored for a decline in neurological status during the first week post-stroke to identify and treat hemorrhagic conversion and diminish its neurological effects. The American Heart Association stroke guidelines recommend appropriate antiepileptic therapy for the treatment of seizures in patients with intracranial hemorrhage (Taft, 2009). The nurse also needs to be vigilant in identifying seizures and aware of treatment modalities.

Essential for Quality Assurance

Admission to a stroke center is effective in reducing complications, stroke reoccurrence, and disability in the stroke population. When stroke centers are not accessible, telestroke communication methods may enhance care through consultations. Telestroke methods may be via the telephone, the Internet, and video conferencing (Demaerschalk, 2011).

Management of patient care may be established through standing admission orders and a critical path care plan. Standardized admission orders prompt the physician to consider all facets of care and acts as a guide to determine the appropriate treatment plan. Patient care plans should include neurological assessments, monitoring vital signs and lab values, medications, nutrients and fluids, and positioning/mobility. Additional important components of nursing care include screening for dysphagia, enhancing comfort, providing adequate nutrition, facilitating communication, fostering communication, assessing for signs of depression, and maintaining safety.

Screening for Dysphagia

Dysphagia, difficulty swallowing, is very common post-stroke and is a major risk factor for developing aspiration pneumonia. Dysphagia can occur when there is damage to the areas of the brain that control speech and/or swallowing and results in muscle weakness of the mouth and throat. When dysphagia is present, there is an increased risk of aspirating saliva, medications or food, which may result in pneumonia.

Essential for Safety

To determine if a patient has a swallowing deficit, the nurse might use the following swallow screening criteria. Prior to swallow screening, the nurse should:

- Evaluate lung sounds and obtain the patient's most recent vital signs, including temperature.
- Evaluate the ability of the patient to follow directions.

If the patient demonstrates any of the following problems at any time during the assessment, the nurse should cease the evaluation, keep the patient NPO, and ask the MD for a speech therapy order for a swallowing evaluation:

- Coughing before, during, or after swallowing
 - Gurgly/wet vocal quality or any voice changes
 - Need to swallow two or more times to clear
 - Excessive length of time to move food to the back of the throat to swallow
 - Pocketing of food
 - Excessive secretions
-

The nurse should consider each of the following when doing a swallow screening:

1. Does the patient have facial weakness or a droop?
2. Does the patient have difficulty with arousal?
3. Does the patient have an absent gag reflex?
4. Given one bite of applesauce, does the patient cough or clear her throat?
5. Given one sip of water, does the patient cough or clear her throat?
6. Given consecutive sips of water, does the patient cough or clear her throat?
7. Given a graham cracker or saltine, does the patient have difficulty chewing, have oral residue after swallowing, or cough and clear her throat?
8. Does the patient need to swallow more than one time per bite/sip?
9. Does the patient need more time to chew and/or initiate swallowing?

If the answer is yes to any of the questions, the nurse should keep the patient NPO, alert the physician, and obtain an order for a swallowing evaluation by speech therapy.

Swallowing evaluations may include videofluoroscopy or barium swallow. Videofluoroscopy allows for accurate visualization of the sequence of events that comprise a swallow. Test analysis identifies abnormal movement of fluid/food such as pooling or aspiration. It may also detect any abnormal movement of anatomic structures and inability of muscle activities. It is essential to test the effects of various consistencies of food and positions to determine the patient's swallowing potential and ensure safety when feeding. A barium swallow may identify the presence of an aspiration and subtler anatomic abnormalities. This test is especially useful if more than one abnormality is discovered (Kiphuth et al., 2011).

Enhancing Comfort

Initially, simple measures such as elevating the head of the patient's bed and providing sedation or analgesia are attempted. Areas that may need to be addressed are pain, incontinence, and constipation. When one is assessing pain, an appropriate scale should be used to determine the patient's level of pain.

Bladder distention may cause sufficient discomfort in a stroke patient to result in hypertension. Urinary catheterization might be initiated if monitoring output is necessary or if the patient is retaining urine and is uncomfortable. If possible, bladder programs should be used to control incontinence. During a bladder training program or when a catheter is discontinued, a bladder scan may be used to monitor post-void residual (PVR).

If the PVR is greater than 400 cc, a straight catheterization is usually recommended. When the PVR is less than 100 cc for three consecutive times, then the bladder scan protocol may be discontinued. Frequent toileting will also help prevent incontinence and assist with increasing bladder tone.

Constipation and fecal impaction can be significant sources of discomfort for the patient post-stroke. A GI assessment including history of bowel habits prior to the stroke should be obtained. Aside from monitoring bowel movements, interventions may include stool softeners, fiber, increased fluids, and frequent toileting.

Providing Nutrition

Undernourished stroke survivors have a higher mortality rate six months post-stroke than survivors who are well nourished. Undernourished survivors are also more likely to develop complications such as pneumonia, other infections, and GI bleeding during their hospital stay. During recovery, when anabolism exceeds catabolism, the goal is to replenish nutritional deficits by maintaining a positive nitrogen balance and replacing protein stores. A thorough nutritional assessment should include the patient's past and present weight, eating habits, blood testing, and a physical exam of the eyes, hair, skin, mouth, and muscles. An accurate intake and output (I & O) and calorie count should be monitored to determine if the patient's needs are being met. The American Heart Association/American Stroke Association (AHA/ASA) currently recommend that patients with acute stroke who cannot take fluids and food orally should receive hydration and nutrition via an NG tube or percutaneous endoscopic gastrostomy (PEG) tube while waiting for swallowing to resume (Alshekhlee et al., 2010). They believe that patients should be provided sufficient calories to meet their nutritional needs, but should not require additional supplementation.

Monitoring Lab Values for Alterations in Blood Glucose

Alterations in blood glucose are common during the acute phase of stroke and are associated with adverse outcomes. All patients should be monitored for hyperglycemia and hypoglycemia because patients whose glucose is more closely maintained in an optimal range have better outcomes.

Hyperglycemia, defined as a serum glucose level greater than 126 mg/dL, is common (in up to 50% of patients) and may be prolonged in patients with acute ischemic stroke regardless of diabetes status.

Hyperglycemia may intensify brain injury by increasing tissue acidosis and increasing blood-brain barrier permeability. The AHA/ASA guidelines recommend treatment with insulin for patients who have blood glucose levels greater than 140 to 185 mg/dL (Radermecker & Scheen, 2010).

Hypoglycemia, defined as a serum glucose level less than 70 mg/dL, can cause focal neurological deficits that mimic stroke. It is essential to manage low blood glucose levels as they alone can cause neuronal damage. It may be necessary to administer glucose with stroke patients who take oral hypoglycemic agents or insulin.

Facilitating Communication

Communication can be a particularly frustrating experience for the stroke patient. The patient may be experiencing expressive or receptive aphasia and realize that she is unable to retrieve the words that she needs. Some ways that the nurse can enhance communication with a patient who has aphasia include:

- Facing the patient when speaking so the patient can see the nurse's face
- Keeping environmental distractions to a minimum
- Using visual cues when able including pictures or word boards
- Speaking slowly and clearly rather than loudly
- Using simple language and only making one statement at a time
- Engaging the patient in conversation
- Listening carefully and patiently
- Complementing the patient on any noticeable progress

Music therapy may be recommended so patients can learn to sing their thoughts as they may be able to express themselves more fluently in song. Gradually, they move from singing to speaking in a sing-song voice to more fluent normal speech (Tamplin, 2008). If a patient does not wish to sing, the nurse needs to be patient, listening to the patient and encouraging her to express herself but not allowing her to become overly frustrated when her speech is not fluent. It is only through practice the fluency will begin to return. Speech therapy will be key in helping the patient re-establish their speech and ways of communication.

Reflect On

Have you ever experienced a time when it was difficult to communicate with a patient? What worked with this patient?

Fostering Patient-Centered Care

The nurse may need to advocate for the stroke patient and her family by providing resources and by arranging for appropriate referrals. Resources may include educational materials (written, online sites, videos) and support groups for the survivor and caregivers through the National Stroke Association or local organizations. Just as for the patient with a brain tumor, the nurse must begin discharge planning on the day of admission to enhance the likelihood of the patient's successful return home.

Maintaining Safety

Falls have been documented as one of the most common incidents after an acute stroke. Hospitalized patients on bed rest may develop diminished bone density, which increases their risk of fractures. Stroke survivors may be at risk for falls due to paralysis, muscle weakness, and lack of coordination. They may also have poor judgment and be impulsive due to cognitive impairments. A fall risk assessment should be performed (see "Safety Initiatives") to determine the patient's level of risk. Physical therapy and occupational therapy consults will determine the individual's needs and physical abilities related to activities of daily living.

Prevention and Management of Complications

Approximately half of the deaths after stroke are due to complications. The prevention of medical complications of stroke is an essential goal of the nursing management of the stroke patient. Common acute and subacute complications that may occur are:

- Cerebral edema
- Hemorrhagic conversion of an ischemic infarct
- Progression of penumbra to infarction
- Seizures
- Deep vein thrombosis
- Pulmonary embolism
- Urinary tract infection
- Aspiration pneumonia
- Decubitus ulcers

Knowledge of potential stroke complications is important to early diagnosis, appropriate preventative strategies, and management. Several of these complications are preventable and are measured as a quality indicator in institutions with stroke center designation (Freeman, Dawson, & Flemming, 2010).

Monitor for Urinary Tract Complications

UTI is a common complication of patients during the first 3 months post-stroke. Following a stroke, patients have difficulty emptying their bladder and a catheter may need to be inserted. Indwelling catheters increase the risk of infection; therefore, they should only be used when necessary and, if used, discontinued as soon as possible. Nurses should monitor and report any signs or symptoms of infection.

Monitor for Altered Tissue Perfusion

Deep vein thrombosis is a common complication in acute stroke and a precursor of a pulmonary embolus (PE). A DVT is a blood clot that develops in the deep veins of the legs. When the clot breaks off and travels to the lung, it is called a PE. Blood clots occur most often between the second and seventh day post stroke. A post-stroke patient has an increased risk of developing a blood clot due to decreased mobility or paralysis. The major causes of DVT are venous stasis, hypercoagulability, and vessel injury. Anticoagulative therapy is the most common treatment for DVT; however, due to the risk of bleeding, hemorrhagic

stroke survivors should be treated initially with compression boots or stockings. Aspirin is recommended early in post stroke course to prevent a recurrent ischemic stroke. Dosages recommended by the American Heart Association ranging from 50 mg to 325 mg daily, depending on the patient's situation, may be started 24 to 48 hours post stroke but only in patients with no allergic or bleeding complications. Early range of motion and ambulation should be established as soon as the patient is stable to decrease the risk of DVT (Freeman et al., 2010).

Recovery

Recovery time and plans of care are specific to each individual. Early aggressive rehabilitation therapies maximize functional recovery. A recent study examined patient preference for their initial therapy setting and determined that it was their home. The research concluded that the providers should offer treatment options to allow for informed decision-making, while taking into consideration the patient's preference (Gregory, Edwards, Faurot, Williams, & Felix, 2010).

Safety Initiatives

Prevention of Falls

PURPOSE: To update the issues, strategies, and tools to prevent falls among patients in acute care settings

RATIONALE: Falls are a major health problem around the world, occurring in all types of healthcare institutions, in all patient populations, and are the most common reason for completion of an incident report on a hospitalized patient. Between 3% and 20% of patients experience these “never events” at least once during their hospitalization, with between 6% and 44% of patients being injured by the fall. Consequences for the patient may include, at a minimum, fractures, soft tissue or head injury, anxiety, and depression. The morbidity, mortality, and financial burdens from patient falls make it one of the most serious risk management issues for healthcare institutions.

HIGHLIGHTS OF RECOMMENDATIONS:

- Measure and track falls using a “fall rate” (the fall rate is the number of patient falls multiplied by 1000 and divided by the number of patient days) or other rate used to track falls such as the number of patients at risk, the number of patients who fell, and the number of falls/bed.
- Trends and rates in falls should take into account the fall risk of the patient population.
- Identify and classify the causes of falls (accidental, such as when a patient falls because of environmental factors;

unanticipated physiologic falls, such as when a patient has an unanticipated seizure and falls; or anticipated physiologic falls, such as when a patient falls who has had a prior fall, has a weak gait, or has been identified as at risk for falling).

- Institute general safety interventions such as:
 - Conduct a standardized risk assessment on admission and when the patient's status changes, using a validated tool such as the Morse Fall Scale.
 - Screen for fall related injury risk factors and history of fall
- Provide risk fall assessments of the patient to all the patient's healthcare providers.
- Standardize interventions for patients at risk for falling (e.g., instruct the patient to request assistance; consider peak effects of medication that might affect the patient's level of consciousness, gait, or elimination when planning nursing care; and provide appropriate footwear, environmental surfaces, and lighting)
- Develop an individualized plan for falls prevention for patients who are at the highest risk for falling.

Sources: Institute for Healthcare Improvement (IHI): Falls Prevention. Boushon, B., Nielsen, G., Quigley, P., Rutherford, P., Taylor, J., . . . Rita, S. *How-to Guide: Reducing patient injuries from falls*. Cambridge, MA: Institute for Healthcare Improvement; 2012. Available at www.IHI.org.

Cerebral or Cerebrovascular Disorder Summary

Nurses play an important role in preventing complications and improving outcomes for patients with cerebral dysfunction. Nurses caring for patients with cerebral or cerebrovascular disorders must be able to identify subtle changes in neurological function, notify the appropriate colleague(s) and provide evidenced-based best practices in order to preserve neurological function and optimize

patient abilities. Nurses must also be skilled in identifying those functions (such as swallowing or airway protection) that patients are no longer able to safely perform and prepared to arrange for appropriate consults and therapy. Finally, the nurse must be able to educate the patient and family, assist in the restoration of neurological functioning, and prevent the development of complications.

Why/Why Not?

The daughter of a stroke patient has rushed her mother to the Emergency Department after returning from work and finding her mother with a right sided facial droop. The daughter tells the nurse, "I've seen the warning signs on TV, I knew that I needed to get her to the

hospital as soon as possible to get that medication that will fix the stroke." Is the woman a candidate for rt-PA? Why or Why not?

See answers to Why/Why Not? in the Answer Section.

Case Study

Mr. Williams is a 75-year-old man admitted to the ED after his wife noted that his left leg and arm were weak and his speech was slurred. His wife also noticed that the left side of his face was "flat." She called 911, and he was transported via ambulance. The paramedics reported that his initial vital signs were BP 180/100, pulse 60, and respirations 16. On admission, his temperature was 100°F (37.8°C) and NIHSS was 12. He was able to speak and denied headache, chest pain, or shortness of breath. The admission assessment revealed a history of hypertension, carotid stenosis, and TIAs.

1. Prioritize a list of immediate nursing strategies.

2. What other information needs to be obtained as part of the admission assessment?
3. What are the likely risk factors contributing to his admission?
4. What diagnostic tests should be ordered?

Mr. Williams's test results show a thrombotic stroke affecting the right side of his brain.

5. What are some possible treatment options?
6. Identify some post-stroke complications and teaching needs.

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 11.1 What are the most common generalized symptoms of a brain tumor? What is a focal symptom?
- 11.2 How does dexamethasone decrease cerebral edema for the patient with a brain tumor? What are the nursing implications associated with dexamethasone administration?
- 11.3 What are the nursing responsibilities in the post-op care of the patient with a pituitary tumor?
- 11.4 How should the nurse prevent a DVT in the patient who has a cerebrovascular disease?

- 11.5 What is the difference between a hemorrhagic and an ischemic stroke?
- 11.6 What are the four common stroke syndromes?
- 11.7 How would a nurse identify if a patient was developing vasospasm following a subarachnoid hemorrhage?
- 11.8 Why is screening for dysphagia essential in a stroke survivor? What are the steps in dysphagia screening?

See answers to Chapter Review Questions in the Answer Section.

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Chapter 12

Care of the Critically Ill Patient Experiencing Alcohol Withdrawal and/or Liver Failure

June Kasper, MSN, RN, CGRN

Michele Bettinelli, BS, RN, CCRN

Abbreviations

ALF	Acute Liver Failure	HE	Hepatic Encephalopathy
AUD	Alcohol Use Disorder	HRS	Hepatorenal Syndrome
AWS	Alcohol Withdrawal Syndrome	NAC	N-Acetylcysteine
CIWA-Ar	Clinical Institute Withdrawal Assessment for Alcohol Scale Revised	NIAAA	National Institute for Alcohol Abuse and Addiction
DSM-IV-TR	Diagnostic and Statistical Manual of Mental Disorders Text Revision	TIPS	Transjugular Intrahepatic Portosystemic Shunt
DSM-V	Diagnostic and Statistical Manual of Mental Disorders (5th Edition)		



Learning Outcomes

Upon completion of this chapter, the learner will be able to:

1. Discuss the essential components of a focused assessment to detect alcohol use disorders (AUD) and alcohol withdrawal.
2. Discuss collaborative and nursing management of a patient experiencing alcohol withdrawal syndrome.
3. Differentiate between acute liver failure and chronic liver failure.
4. Explain the relationship between portal hypertension and the development and manifestations of decompensated liver disease and describe the collaborative care and nursing responsibilities for the patient with decompensated liver disease.

Introduction

Alcohol is the most commonly abused substance in the United States. In 2013, data from the National Survey on Drug Use and Health showed that 135.5 million Americans ages 12 and older use alcohol, and an estimated 18 million have an alcohol use disorder (AUD). Alcohol withdrawal syndrome (AWS), the constellation of symptoms that may develop in individuals with habitual alcohol intake who stop or significantly decrease their alcohol consumption, occurs in as many as 20% of critical care patients (Riddle, Bush, Tittle, & Dilkush, 2010). AWS is associated with an increase in complications and prolonged length of stay in intensive care and contributes to 100,000 deaths annually. Estimated cost is 200 billion annually.

Acute liver failure (ALF) is a rare syndrome characterized by a rapid decline in liver function that occurs in a person without preexisting liver disease. Acetaminophen (Tylenol) overdosing is the leading cause of ALF in the United States. Acetaminophen overdoses are potentiated by alcohol use.

Cirrhosis, in contrast, is the end stage of chronic liver disease. It results from disorders such as hepatitis and alcohol misuse that damage liver cells over time. In the early stages, cirrhosis can be silent, causing few, if any, signs or symptoms. In the later stages, it is characterized by progressive deterioration in liver function and development of portal hypertension. Management involves treating the underlying disease in hopes of halting the process while preventing or treating complications and evaluating the patient for transplantation.

Anatomy and Physiology Review

The following key terms related to anatomy and physiology are foundational to understanding the chapter.

Neurotransmitters: Chemical substances that allow neurons to communicate with each other. In the brain, neurotransmitters either excite (accelerate) or inhibit (brake) impulses along the neurons. Normally, there is a balance between excitatory and inhibitory neurotransmission.

Neuroreceptor: A site on or inside a neuron to which a hormone or neurotransmitter can initiate a chemical or electrical reaction.

Gamma-aminobutyric acid (GABA)—A CNS inhibitory neurotransmitter.

Gamma-aminobutyric acid A (GABA A)—A neuroreceptor for GABA.

Glutamate: A CNS excitatory neurotransmitter that can interact with several receptors.

N-methyl-D-aspartate (NMDA)—One of the neuroreceptors for glutamate.

Depressant: A substance that slows down the nervous system and its transmission of messages.

Alcohol (ethanol): A CNS depressant.

Liver Anatomy

The largest internal organ, weighing about 3 to 4 pounds, the liver is located in the right upper quadrant of the abdomen, below the diaphragm. It is divided into a right and left lobe with the right lobe approximately six times larger than the left. Each lobe is divided into lobules. Approximately 1500 mL of blood enters the liver each minute, making it the most vascular organ in the body (♦ Figure 12-1).

The hepatic lobule is the functional unit of the liver, and an estimated one million lobules form the mass of the liver. They are 2 mm high and 1 mm in circumference (Figure 12-1). Each lobule consists of:

- **Hepatocytes:** The functional cells of the liver that secrete bile and perform a number of metabolic functions.
- **Sinusoids:** Specialized vascular beds located between each row of hepatocytes that are lined with Kupffer cells and highly permeable endothelium.
- **Kupffer cells:** Phagocytic cells that remove amino acids, nutrients, old red blood cells, bacteria, and debris from the blood. They detoxify toxins and other substances. They also play a role in maintaining vascular homeostasis through production of vasoactive mediators.

The liver receives blood from both venous and arterial sources and has a vast vascular network capable of storing a large volume of blood. The amount of blood stored at any one time depends on the pressure relationships between the arteries and the veins. Unlike other organs, the majority of the blood supply is venous.

- **Portal vein:** Supplies 75% of the blood flow and 50% of the oxygen. This blood originates from the capillaries of the entire gastrointestinal (GI) tract, and though it carries some oxygen, it is full of nutrients that have been absorbed from the digestive tract. Oxygen and nutrients diffuse through the capillaries into the liver cells.
- **Hepatic artery:** Supplies 25% of the blood flow and 50% of the oxygen. It branches from the abdominal aorta, providing oxygenated blood.
- **Hepatic vein:** Blood from the sinusoids drain into a central vein and then into the hepatic vein, which drains blood from the liver.
- **Inferior vena cava:** Blood from the hepatic vein flows to the vena cava to return to the right side of the heart.

Each lobule contains a hepatic artery, a portal vein, and a bile duct, known as the portal triad. The hepatic duct is responsible for the transport of bile produced by the liver cells to the gallbladder and duodenum. The bile ducts in each lobule connect to small ducts that connect to larger ducts that eventually connect to form the main hepatic ducts (♦ Figure 12-2).

Normal liver

The liver contains multiple lobules made up of plates of hepatocytes, the functional cells of the liver, surrounded by small capillaries called sinusoids. These sinusoids receive a mixture of venous and arterial blood from branches of the portal vein and hepatic artery. Blood from the sinusoids drains into the central vein of the lobule. Hepatocytes produce bile, which drains outward to bile ducts.

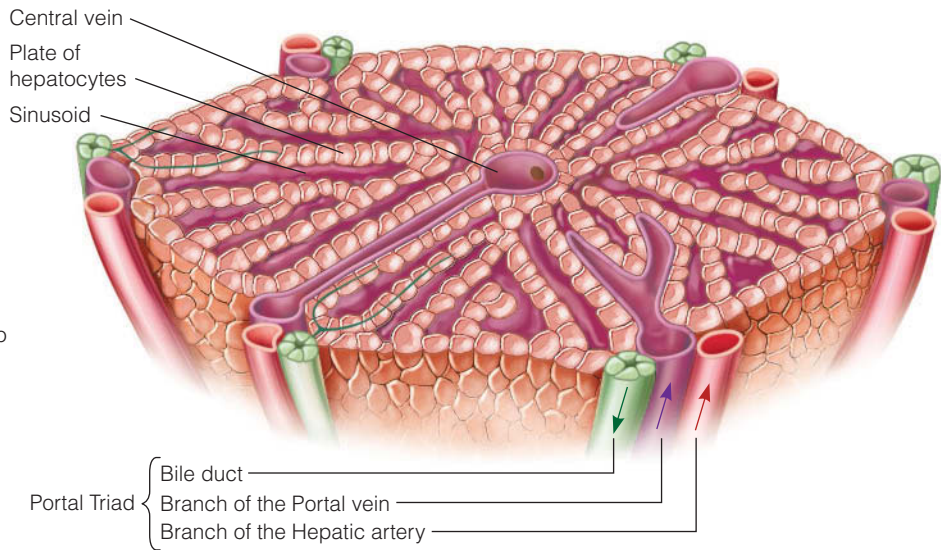


Figure 12-1 Normal liver.

Liver Physiology

The liver has more than 500 specific functions, including metabolic and hematologic regulation and the production of bile as summarized in the following list:

- Bile formation and secretion—help to carry waste away and break down fats in the small intestine.
- Metabolism of bilirubin, a by-product of the destruction of aged red blood cells
- Production of proteins for blood plasma (produces albumin, a protein responsible for maintenance of normal fluid balance in the body)
- Metabolism of carbohydrates, proteins, and fats

Distribution of Vessels and Ducts

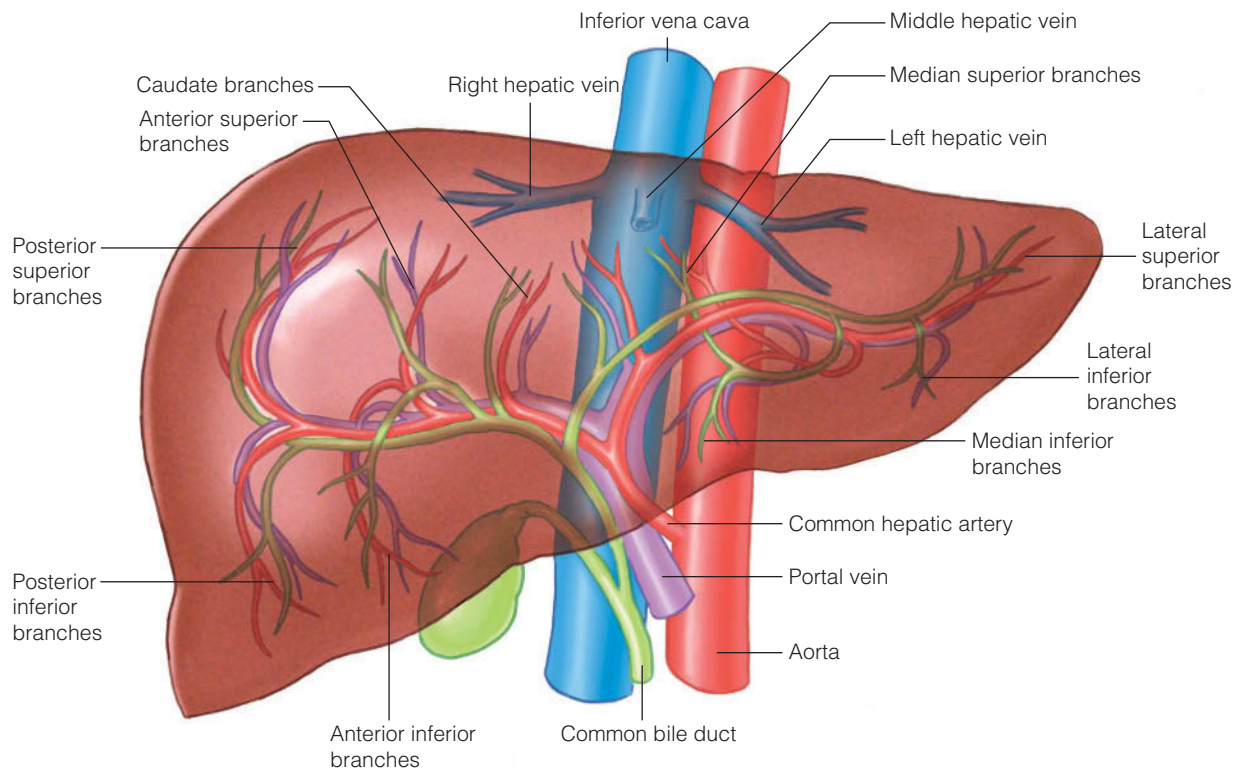


Figure 12-2 Distribution of vessels and ducts in the liver.

- Conversion of ammonia to urea, which is then excreted in the urine
- Coagulation and anticoagulation—producing blood proteins necessary for normal clotting
- Metabolic detoxification
- Metabolizing medications
- Storage of minerals and vitamins

Liver cells have the ability to regenerate themselves within three weeks.

Alcohol Withdrawal Syndrome

When exposed to repeated doses of alcohol, the central nervous system (CNS) becomes accustomed to the depressant effects of the alcohol and produces adaptive changes in an attempt to function normally. In the absence of alcohol or with a significant decrease in the amount of alcohol, chaos erupts within the CNS. When alcohol is no longer acting as a depressant, the compensatory actions cause excessive CNS excitability. It is analogous to having an accelerator without a brake. The time course of withdrawal is determined by the time it takes to restore balance.

Alcohol Use Disorders

Prior to 2013, the *Diagnostic and Statistical Manual of Mental Disorders Text Revision* (DSM-IV-TR; American Psychiatric Association, 2000) defined two distinct disorders: alcohol

abuse and alcohol dependency (alcoholism). The *Diagnostic and Statistical Manual of Mental Disorders* (DSM-V; American Psychiatric Association, 2013) integrated the two disorders into a single disorder/medical diagnosis called alcohol use disorder defined by meeting at least 2 of the 11 identified criteria (behavioral and physical symptoms) during a 12-month period. AUD is subcategorized into mild, moderate and severe based upon the number of criteria met (Table 12-1).

Patients who meet the criteria for AUDs are at risk for alcohol withdrawal syndrome (AWS), which is defined by the DSM-V as having two components:

- Cessation or significant reduction in prolonged and heavy alcohol use
- The presence of two or more of the characteristic alcohol withdrawal symptoms:
 - Psychomotor agitation
 - Anxiety
 - Autonomic hyperactivity (tachycardia or sweating)
 - Increased hand tremor
 - Insomnia
 - Nausea or vomiting
 - Transient tactile, visual, or auditory hallucinations
 - Tonic-clonic seizure

Additionally, significant distress or impairment in social, occupational, or other important areas of functioning must also be present. These symptoms must be directly caused by stopping or reducing alcohol intake and not attributable to other medical conditions,

Table 12-1 Alcohol Use Disorder Criteria

1. Alcohol taken in larger amounts or over a longer period than was intended.
2. There is a persistent desire or unsuccessful efforts to cut down or control alcohol use.
3. A great deal of time is spent in activities necessary to obtain alcohol, use alcohol or recover from its effects.
4. Craving, or strong desire or urge to use alcohol.
5. Recurrent alcohol use resulting in failure to fulfill major role obligations at work, school or home.
6. Continued alcohol use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of alcohol.
7. Important social, occupational or recreational activities are given up or reduced because of alcohol use.
8. Recurrent alcohol use in situations in which it is physically hazardous.
9. Alcohol use is continued despite knowledge of having a persistent or recurrent problem that is likely to have been caused or exacerbated by alcohol.
10. Tolerance, as defined by either one of the following:
 1. A need for markedly increased amounts of alcohol to achieve intoxication or desired effect
 2. A markedly diminished effect with continued use of the same amount of alcohol
11. Withdrawal, as manifested by either of the following:
 1. The characteristic withdrawal syndrome for alcohol
 2. Alcohol (or closely related substance, such as a benzodiazepine) is taken to relieve or avoid withdrawal symptoms.

Mild—presence of 2-3 symptoms

Moderate—presence of 4-5 symptoms

Severe—presence of 6 or more symptoms

a primary mental disorder, or the influence of another substance (Kupfer, 2013). Literature has linked alcohol abuse to a mild AUD, and alcohol dependency to a moderate or severe AUD.

The nurse should consider the clinical manifestations of AWS on a progressive continuum, where time frames and durations are not exact and often overlap. The pattern, severity, and exact manifestations may be inconsistent from patient to patient. Stages of withdrawal are described chronologically as early and late, or according to severity in terms of minor and major or mild, moderate, or severe.

Severity is evaluated by the degree of autonomic hyperactivity and neuropsychiatric behavior and the occurrence of complications. Severity is believed to be dose dependent with more severe withdrawal associated with higher blood alcohol concentrations, longer duration of heavy alcohol consumption, and intensified by the stress of concurrent illnesses. Having a history of previous withdrawals also suggests that subsequent withdrawals can be progressively more severe. This is known as a *kindling effect*. Other risk factors include liver dysfunction and use of other addictive drugs.

As the patient progresses through the continuum, the withdrawal manifestations become more intense. The majority of the patients experience minor withdrawal and have an uneventful recovery within a few days. Others may progress to major withdrawal and experience the most severe manifestations, including withdrawal seizures, alcoholic hallucinosis, withdrawal delirium, or delirium tremens (DTs).

Assessment and Management of the Patient with Alcohol Withdrawal Syndrome

Most important is the assessment and recognition of all patients who are at risk for AWS. Critical to treatment is an accurate history, which can be a challenge in patients with an impaired cognitive status and an unknown past. The goals of management are to:

- Identify patients at risk for AWS
- Establish severity
- Decrease agitation and prevent withdrawal progression
- Provide supportive care
- Maintain fluid and electrolyte balance
- Provide a safe and dignified environment
- Minimize effects on acute and chronic comorbid illnesses
- Prevent complications
- Initiate case management services for future rehabilitation treatment

Risk Assessment for Alcohol Withdrawal Syndrome

Ideally a standardized tool/questionnaire should be utilized to detect patients with an AUD who are at risk for withdrawal. Early recognition and prevention management decrease both the risk for severe AWS and mortality rates. There are several valid and reliable tools available. Some are designed for specific populations and healthcare settings. Such tools identify patients highly suspicious for an AUD who warrant further evaluation with an AWS scale. These tools are of limited utility if the patient is unable to respond to questions. Also consider that emergent admissions may not have had an accurate assessment at the point of entry (trauma, surgery). Patients with a known AUD diagnosis should be automatically assessed with an AWS scale. Biochemical markers have been investigated but not validated. The most critical information to clinicians is described below. The nurse obtains a complete history in a nonthreatening manner from the patient and/or family. While observing the patient's nonverbal responses and presence or absence of eye contact, the nurse questions the patient and/or family related to:

- Current and past alcohol use and family history of alcohol problems. AUD's are more prevalent in families where first-degree relatives have been afflicted.
- Quantity and frequency or pattern of alcohol use. Patients and families may underreport consumption and abuse or deny it. It is helpful to educate the patient and family as to why this information is so important, as well as to explain what at-risk drinking is and what a "standard" drink is so more accurate information may be obtained. The likelihood of AWS occurring is less likely with episodic, intermittent drinking behavior.
 - The National Institute for Alcohol Abuse and Alcoholism (NIAAA) defines *low-risk* drinking and at-risk/heavy drinking (Table 12-1).
 - It is important to obtain specific information related to the type and amount of alcohol consumed. The NIAAA defines a standard drink as containing 14 grams of alcohol (Table 12-2).

Table 12-2 What's Low-Risk Drinking? What's Heavy or At-Risk Drinking?

Risk for Alcohol Problems	Number of Drinks for Men	Number of Drinks for Women and Anyone Over 65
Low-risk drinking	No more than 4 drinks daily	No more than 3 drinks daily
At-risk/heavy drinking	No more than 14 drinks per week or more than 4 per occasion	No more than 7 drinks per week or more than 3 per occasion

Source: National Institute on Alcohol Abuse and Alcoholism/National Institute of Health Publication (2010). *Rethinking drinking: Alcohol and your health*. Retrieved April 2, 2011, from http://pubs.niaaa.nih.gov/publications/RethinkingDrinking/Rethinking_Drinking.pdf

Table 12-3 National Institute for Alcohol Abuse and Alcoholism Standard Drink Equivalents

Standard Drink Equivalents	Approximate Number of Standard Drinks in
Beer or Cooler	
12 oz.	• 12 oz. = 1 • 16 oz. = 1.3 • 22 oz. = 2 • 40 oz. = 3.3
	
~5% alcohol	
Malt Liquor	
8–9 oz.	• 12 oz. = 1.5 • 16 oz. = 2 • 22 oz. = 2.5 • 40 oz. = 4.5
	
~7% alcohol	
Table Wine	
5 oz.	• a 750 mL (25 oz.) bottle = 5
	
~12% alcohol	
80-Proof Spirits (Hard Liquor)	
1.5 oz.	• a mixed drink = 1 or more* • a pint (16 oz.) = 11 • a fifth (25 oz.) = 17 • 1.75 L (59 oz.) = 39
	
~40% alcohol	

Source: From National Institute on Alcohol Abuse and Alcoholism (NIAAA) Publication/ National Institute of Health Publication (2010). *Rethinking drinking: Alcohol and your health*. Retrieved April 2, 2011, from http://pubs.niaaa.nih.gov/publications/RethinkingDrinking/Rethinking_Drinking.pdf

*Note: Depending on factors such as the type of spirits and the recipe, one mixed drink can contain from one to three or more standard drinks.

depression, and other substance use disorders in individuals with alcohol use disorders. Alcohol and tobacco are strongly associated with each other.

The CAGE Questionnaire

Additionally, a standardized questionnaire should be utilized to detect dependency. There are several reliable and valid questionnaires available. One such questionnaire is the CAGE. It is a simple, fast, short, reliable, and valid questionnaire. The acronym helps the clinician to recall the following questions:

- Have you ever felt the need to CUT down on drinking?
- Have you ever felt ANNOYED by criticism of your drinking?
- Have you ever had GUILTY feelings about your drinking?
- Have you ever had an EYE opener drink first thing in the morning to steady your nerves or get rid of a hangover? (Ewing, 1984)

Scoring consists of 1 point for each positive answer. A score of 1 to 3 is highly suspicious for alcohol dependency and warrants further evaluation with an AWS withdrawal scale. The questionnaire will be of limited utility if the patient is unable to respond to the questions.

Clinical Institute Withdrawal Assessment of Alcohol Scale, Revised

To objectively assess and manage AWS according to severity, the most frequently used scale is the Clinical Institute Withdrawal Assessment of Alcohol Scale Revised (CIWA-Ar) (Table 12-4).

The nurse assesses and scores 10 specific symptoms: nausea and vomiting, tremor, sweating, anxiety, agitation, headache, disorientation, tactile disturbances, visual disturbances, and auditory disturbances. Concurrently, vital signs including temperature and pulse oximetry are evaluated. Patients are medicated when they cross a designated threshold of severity. The frequency of assessments are determined by the severity, treatment, response to treatment, and overall acuity. As the patient's score increases, so does the patient's risk for severe withdrawal symptoms (Table 12-5). Best practice utilizes the CIWA-Ar to guide pharmacological therapy and direct the level of care provided as shown in Table 12-6.

Reflect On

Statistics show that 40% of patients admitted to the hospital have some degree of alcohol use disorder. Think about how often you utilize the CIWA-Ar scale. Do you think that screening is consistent, appropriate, and accurate? Could the screening system be improved?

- Time of last alcohol ingestion.
- Characteristics of an AUD as previously described.
- History of liver disease or other alcohol-related illnesses, previous withdrawals, psychiatric or behavioral issues, and alcohol-related injuries (trauma, falls, and collisions). There is a high incidence of anxiety,

Table 12-4 Clinical Institute Withdrawal Assessment of Alcohol, Revised (CIWA-Ar)

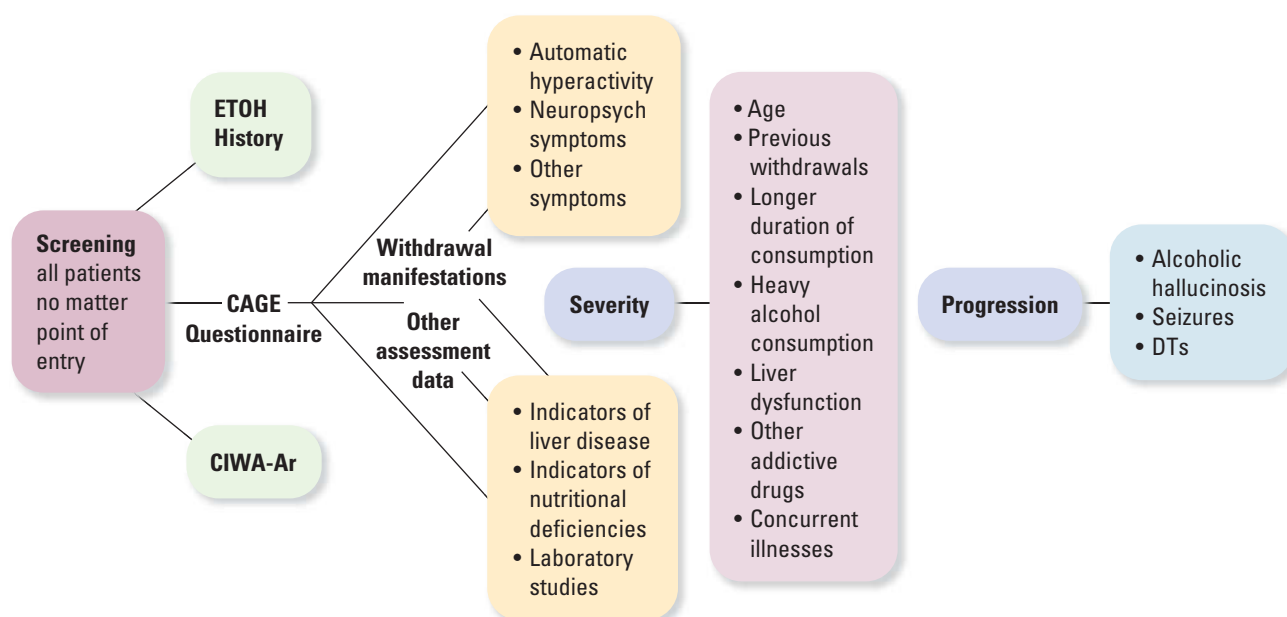
Patient: _____ Date: (yy/mm/dd) ____/____/____ Time: (24 hr) _____	
Pulse or heart rate: _____ Blood Pressure: _____	
Nausea and Vomiting —Ask “Do you feel sick to your stomach?” “Have you vomited?” Observation. • 0—no nausea and no vomiting • 1—mild nausea with no vomiting • 2 • 3 • 4—intermittent nausea with dry heaves • 5 • 6 • 7—constant nausea, frequent dry heaves and vomiting	Tactile Disturbances —Ask “Have you any itching, pins and needles sensations, any burning, any numbness, or do you feel bugs crawling on or under your skin?” Observation. • 0—none • 1—very mild itching, pins and needles, burning, or numbness • 2—mild itching, pins and needles, burning, or numbness • 3—moderate itching, pins and needles, burning, or numbness • 4—moderately severe hallucinations • 5—severe hallucinations • 6—extremely severe hallucinations • 7—continuous hallucinations
Tremor —Arms extended and fingers spread apart. Observation. • 0—no tremor • 1—not visible, but can be felt fingertip to fingertip • 2 • 3 • 4—moderate, with patient’s arms extended • 5 • 6 • 7—severe, even with arms not extended	Auditory Disturbances —Ask “Are you more aware of sounds around you? Are they harsh? Do they frighten you? Are you hearing anything that is disturbing to you? Are you hearing things that you know aren’t there?” Observation. • 0—not present • 1—very mild harshness or ability to frighten • 2—mild harshness or ability to frighten • 3—moderate harshness or ability to frighten • 4—moderately severe hallucinations • 5—severe hallucinations • 6—extremely severe hallucinations • 7—continuous hallucinations
Paroxysmal Sweats —Observation. • 0—no sweat visible • 1—barely perceptible sweating, palms moist • 2 • 3 • 4—beads of sweat obvious on forehead • 5 • 6 • 7—drenching sweats	Visual Disturbances —Ask “Does the light appear to be too bright? Is its color different? Does it hurt your eyes? Are you seeing anything that is disturbing you? Are you seeing things that you know aren’t there?” Observation. • 0—not present • 1—very mild sensitivity • 2—mild sensitivity • 3—moderate sensitivity • 4—moderately severe hallucinations • 5—severe hallucinations • 6—extremely severe hallucinations • 7—continuous hallucinations
Anxiety —Ask “Do you feel nervous?” Observation. • 0—no anxiety, at ease • 1—mildly anxious • 2 • 3 • 4—moderately anxious, or guarded, so anxiety is inferred • 5 • 6 • 7—equivalent to acute panic states as seen in severe delirium or acute schizophrenic reactions	Headache, Fullness in Head —Ask “Does your head feel different? Does it feel like there is a band around your head?” Do not rate dizziness or lightheadedness. Otherwise, rate severity. • 0—not present • 1—very mild • 2—mild • 3—moderate • 4—moderately severe • 5—severe • 6—very severe • 7—extremely severe
Agitation —Observation. • 0—normal activity • 1—somewhat more than normal activity • 2 • 3 • 4—moderately fidgety and restless • 5 • 6 • 7—paces back and forth during most of the interview, or constantly thrashes about	Orientation and Clouding of Sensorium —Ask “What day is this? Where are you? Who am I?” • 0—oriented and can do serial additions • 1—cannot do serial additions or is uncertain about date • 2—disoriented for date by no more than two calendar days • 3—disoriented for date by more than two calendar days • 4—disoriented for place and/or person
Note: Table 12-5 correlates the CIWA-Ar score with the severity of withdrawal.	
Total CIWA-Ar Score _____	
Rater’s Initials _____	
Maximum Possible Score = 67	

Source: Sullivan, J. T., Sykora, K., Schneiderman, J., Naranjo, C. A., & Sellers, E. M. (1989). Assessment of alcohol withdrawal: The revised Clinical Institute Withdrawal Assessment for Alcohol scale (CIWA-Ar). *British Journal of Addiction*, 84, 1353–1357.

Because alcohol is short acting, the nurse anticipates that signs and symptoms of minor withdrawal commonly appear within 6 to 12 hours of the last ingestion, peak in 24 to 36 hours, and resolve after 48 hours. In the patient whose liver is already compromised, AWS can last much longer secondary to poor metabolic function not allowing the liver to excrete toxins effectively. In addition to the

CIWA-Ar scale, the nurse’s history and physical assessment should also consider the following indicators associated with withdrawal:

- Autonomic manifestations: hyperventilation, tachycardia, palpitations, hypertension, increased body temperature, hyperreflexia, insomnia, restlessness, diaphoresis, tremors, mydriasis, and seizures



Visual Map 12-1 AWS Assessment

Table 12-5 Clinical Institute Withdrawal Assessment for Alcohol Scale, Revised (CIWA-Ar)

Scores	Severity of Withdrawal
Less than 8–10	Minimal to mild withdrawal
8–15	Moderate symptoms
>15	Severe symptoms

Source: Sullivan, J. T., Sykora, K., Schneiderman, J., Naranjo, C. A., & Sellers, E. M. (1989). Assessment of alcohol withdrawal: The revised Clinical Institute Withdrawal Assessment for Alcohol scale (CIWA-Ar). *British Journal of Addiction*, 84, 1353–1357.

- Neuropsychiatric manifestations: anxiety, fear, anger, argumentative, belligerent, paranoid, impaired attention span, disorientation, tactile, auditory-visual hallucinations, global confusion
- Other manifestations: nausea, vomiting, diarrhea, anorexia, abdominal cramps, and recurrent infections

NURSING ACTIONS

The nurse also assesses for indications of liver disease and nutritional deficiencies. Repeated excessive consumption of alcohol frequently results in deterioration of liver function. If chronic alcohol use is suspected the nurse should assess the patient for the following indications of liver disease; these are described later in the chapter:

- Skin vascularization: spider angioma, palmar erythema
- Jaundice
- Ascites/peripheral edema
- Encephalopathy/impaired mental status
- Hepatomegaly

Repeated excessive consumption of alcohol often provides empty calories, suppresses the appetite, and causes early satiety (fullness), resulting in nutritional deficiencies and malnutrition. The nurse assesses for the following manifestations of nutritional deficiencies:

- Underweight
- Fatigue (folic acid/folate deficiency)
- Anemia (folic acid/folate deficiency)
- Ataxia (thiamine deficiency/Wernicke's encephalopathy)
- Nystagmus (thiamine deficiency/Wernicke's encephalopathy)
- Peripheral neuropathy (thiamine deficiency)
- Ophthalmoplegia (thiamine deficiency/Wernicke's encephalopathy)
- Muscle weakness and wasting

Laboratory Studies

In addition, the nurse should review the following laboratory studies:

- Toxicology screen (blood and urine) to include a blood alcohol concentration—will confirm recent alcohol ingestion and the presence of other substance use.
- Complete blood count (CBC)—often reveals anemia but macrocytosis (elevated MCV between 100 and 110 fL) develops prior to the anemia in the majority of alcoholics. Also seen with chronic abuse is leukocytosis and thrombocytopenia.
- Serum glucose (normal 70 to 110 mg/dL)—hypoglycemia may accompany AWS and symptoms of AWS can mimic that of hypoglycemia. Less than 50 mg/dL is a critical value.

- Serum electrolytes including magnesium and phosphorus—electrolyte abnormalities are commonly related to fluid balance issues and nutritional deficiencies.
- Blood urea nitrogen (BUN) and creatinine—are evaluated for hydration status and renal function.
- Prothrombin time/international normalized ratio (INR)—can be increased with excessive alcohol consumption and liver dysfunction.
- Liver function tests—a variety of studies to assess liver function and the presence of liver disease are performed. Alcohol use is a common cause of abnormal liver function tests.

Because alcohol can affect all systems, the nurse anticipates tests may be ordered relative to nutritional deficiencies (e.g., serum iron, folate, protein, albumin) and other alcohol-related disorders. Diagnostic tests and physical characteristics help to support the diagnosis of alcohol use disorders; however, normal tests and a physical exam do not rule out the diagnosis.

Assessment of the Patient with Severe Alcohol Withdrawal

Early withdrawal is manifested by anxiety, tremulousness, palpitations, nausea, and anorexia. As withdrawal severity increases, the critical care nurse expects that manifestations will be progressive, exaggerated, and potentially life threatening. Focused attention is on cardiac and respiratory status and neuropsychiatric indicators of the **delirium tremens (DTs)**. In severe withdrawal, the patient may need to be intubated and mechanically ventilated in order to adequately control symptoms.

Alcohol Withdrawal Seizures. Alcohol withdrawal seizures commonly occur and can manifest anytime during withdrawal. They often appear early, within 6 to 48 hours of the last alcohol ingestion. Though they can occur in the absence of other signs and symptoms, they are usually part of the progression. Patients with a chronic heavy drinking history who have experienced previous episodes of withdrawal have an increased incidence of seizures. The nurse will identify high-risk individuals, assess characteristics of seizure activity, and implement seizure precautions and one-to-one observations as necessary.

Withdrawal seizures are likely to be brief, single, generalized, and tonic-clonic. Occasionally they present in small clusters. The majority of the seizures terminate spontaneously. Patients experiencing seizures, especially status epilepticus, which is rare, often have a neurological workup and medical evaluation for a differential diagnosis. The nurse monitors for fluid and electrolyte abnormalities and hypoglycemia, which may be associated with seizures. Patients who experience seizures predictably have a

more complicated withdrawal and an increased incidence of developing DTs.

Alcoholic Hallucinoses. Alcoholic hallucinosis can manifest from 12 to 24 hours after the last alcohol ingestion. With alcohol hallucinosis, the patient experiences perceptual disturbances, usually visual, auditory, or tactile phenomena, without sensorial alterations. The patient is fully conscious, aware of the environment, and acknowledging that the hallucinations are related to the substance dependence and withdrawal. Though the duration can be variable, lasting for days, they usually resolve in 24 to 48 hours. The nurse assesses the characteristics of the hallucinations along with orientation status to distinguish hallucinosis from the hallucinations characteristic of the DTs. DTs usually do not appear for 48 hours after the last alcohol ingestion.

Delirium Tremens. DTs, also called alcohol withdrawal delirium, is the most severe complication of withdrawal. Often it is the result of under treatment or lack of treatment. It is seen in approximately 5% of the cases. Mortality varies depending on the comorbidities, but the average is up to 1-4% and death is often caused by arrhythmias and associated critical illnesses (Schuckit, 2014). Risk factors include older age, preexisting lung disease, coexisting liver disease, concurrent illnesses, a previous history of DTs, and/or a history of sustained drinking.

The autonomic and neuropsychological manifestations are profoundly exaggerated. The nurse must recognize that the most characteristic distinction of the DTs is the disorientation and global confusion. The nurse must assess for marked agitation, hallucinations, and distractibility with accentuated response to external stimuli, and increases in heart rate, blood pressure, respiratory rate, and temperature. The hallucinations can be horrific, thus increasing agitation, confusion, and aggressiveness. Often patients become violent in an attempt to escape. Autonomic hyperactivity and instability evidenced by pronounced tachycardia greater than 120 beats per minute, temperature elevation of greater than 100°F (37.5°C), hypertension (often labile), and tachypnea can cause increased cardiac workload, oxygen delivery, and consumption. The nurse needs to assess for cardiac dysrhythmias, respiratory insufficiency, severe dehydration, and hyperthermia. If autonomic instability is not recognized and treated properly death may occur from respiratory and/or cardiovascular collapse. Patients with a core body temperature greater than 104°F (40°C) have an increased risk of mortality (Hoffman & Weinhouse, 2015). The nurse also considers that other etiologies can mimic the physical symptoms and delirium (sleep deprivation, anesthesia, pain, sepsis, organ dysfunction).

Essential for Safety

The nurse must be able to recognize the symptoms of DTs and obtain assistance immediately since DTs are a medical emergency.

The timing and duration of the DTs on the continuum is variable. DTs typically occur within 48 to 96 hours after the patient's last alcoholic beverage, and they can last 1 to 5 days (Hoffman & Weinhouse, 2015). Reports have indicated that DTs have lasted up to 14 days. Lingering cognitive dysfunction may last from 4 to 12 weeks. Knowing the approximate time frames enables the nurse to be more vigilant.

COLLABORATIVE CARE OF THE PATIENT EXPERIENCING ALCOHOL WITHDRAWAL

The nurse works collaboratively with the healthcare team (physician and appropriate healthcare disciplines) to establish risk for AWS, control autonomic hyperactivity, and identify neuropsychiatric behaviors. The goal is to prevent, recognize, and treat symptoms; halt progression; provide a safe and dignified withdrawal; and prevent and treat complications. Supportive care and pharmacological therapy are instrumental in accomplishing management goals.

Symptom-Triggered Therapy

Symptom-triggered therapy is the preferred method of medication administration for AWS. The nurse monitors the signs and symptoms, utilizing the CIWA-Ar assessment tool. Medication administration and dosing protocols are based on the total score. The goal is to achieve the therapeutic effect of the medication evident by light somnolence, no breakthrough or progression of manifestations, and hemodynamic stability. The nurses' responsibilities include:

- Assessment of vital signs, including temperature and pulse oximetry. Actual frequency will depend on acuity, CIWA-Ar score, and medication administration and therapeutic response.
- Assessment of AWS symptoms (while the patient is awake) using the CIWA-Ar scale, the frequency as described above.
- Administer prescribed medications for clinically significant symptoms as established by the total CIWA-Ar score:
 - Best practice for the management of AWS supports development of specific order sets and treatment plans that include assessment parameters and appropriate interventions and outcome evaluation criteria based on the total score. Separate order sets for prophylaxis, actual AWS, and different levels of care may be developed.

- Depending on the risk, prophylaxis may be ordered for a CIWA-Ar score of less than 8.
- The CIWA-Ar may have limited use in a critically ill patient who is unable to participate secondary to severe agitation and/or mechanical ventilation. Some critical care protocols may require a sedation-agitation scale assessment in conjunction with the clinical picture and CIWA-Ar. An example would be the Riker Sedation-Agitation Scale (SAS).
- Order sets establish the severity threshold or total score at which sedation is initiated. The nurse will see some variation as to what total score receives sedation. Table 12-6 shows an example of a combined prophylaxis and treatment order set paralleling the SAS and CIWA-Ar as criteria for administering medication.

Multiple studies demonstrate that symptom-triggered therapy is preferred over fixed dosing schedules, and studies have shown that it requires considerably less medication and shorter duration of treatment. It also limits both undersedation and oversedation. Low-dose benzodiazepines may be considered prophylactically for a patient who has a history of severe withdrawals. A low-dose, fixed-schedule regimen may be ordered with routine CIWA-Ar assessments. If necessary, the fixed schedule can be transitioned to symptom-triggered therapy according to the total CIWA-Ar score.

Essential for Safety

At any transition of care (change of shift, transfer), it is recommended that two nurses assess and complete the CIWA-Ar/SAS score together to ensure agreement and consistency of care.

Refractory DTs

There are multiple management approaches to patients with DTs resistant to benzodiazepines. The following options can be instituted in combination. The ultimate goal is to control agitation and avoid intubation and mechanical ventilation.

- Progressively larger bolus doses of benzodiazepines
- Addition of a barbiturate (phenobarbital)
- Continuous IV infusion of:
 - Midazolam/Versed—a short acting benzodiazepine
 - Lorazepam/Ativan—an intermediate acting benzodiazepine (Chapter 2)
 - Propofol/Diprivan—a short acting general anesthetic (Chapter 2)
 - Dexmedetomidine/Precedex—an alpha 2 agonist (Chapter 2)

Table 12-6 Example of Alcohol Withdrawal Prophylaxis and Treatment Protocol

Allergies: _____			
Weight: _____			
CIWA-Ar	SAS	Benzodiazepine Dosing	Reassess CIWA-Ar/SAS and Vital Signs
< 8	4	Prevention and High Risk for Withdrawal	☒ One hour after any dose
		• 10 mg diazepam po q 8 hours x 24 hours	☒ At least every 4 hours
		OR	☒ PRN worsening agitation
		• 1 mg lorazepam po q 6 hours x 24 hours then 1 mg po q 6 hours x 24 hours	
		OR	
8-15	5	• 2 mg lorazepam po q 6 hours x 24 hours then 1 mg po q 6 hours x 24 hours	
		OR	
		• 3mg lorazepam po q 6 hours x 24 hours then 1 mg po q 6 hours x 24 hours	
		(Avoid diazepam in liver disease and consider lower doses in elderly patients)	
			☒ 15 minutes after any dose
> 15	> 5	Mild—Moderate Withdrawal	☒ At least every 4 hours
		☒ 2 mg IV lorazepam every 15 minutes PRN CIWA-Ar 8-15 or SAS 5	☒ PRN worsening agitation
		Severe Withdrawal	
		☒ 4 mg IV lorazepam every 15 minutes PRN CIWA - Ar > 15 SAS > 5	
Escalation of Treatment of Alcohol Withdrawal			
If total lorazepam equals 10 mg or more in 1 hour and CIWA–Ar remains >15 (30 minutes after last dose) contact MD to reassess patient for adjunctive treatment. Consider continuous IV infusion of a benzodiazepine, and /or dexmedetomidine phenobarbital, propofol, other)			
Call M.D. for:			
• Heart rate less than 55 beats per minute or greater than 110 beats per minute			
• Diastolic blood pressure greater than 95 mmHg			
• Systolic blood pressure greater than 180 mmHg			
• Respiratory rate less than 12 breaths per minute or greater than 28 breaths per minute			
• Oxygen saturation less than 90%			
• Seizures			
• Expressed or suspected suicidal tendencies			
• Serum magnesium level is less than 1.6 mEq/L			
• Serum potassium level is less than 3.1 mEq/L			
CONSULTS			
• Social Service • Nutrition • Case Management			
• Psychiatry • Other _____			

Phenobarbital

Phenobarbital for AWS is currently an off label use. Literature supports the use of phenobarbital as an adjunct to benzodiazepines when high doses of benzodiazepines fail to control agitation. Phenobarbital is a long acting barbiturate with a narrow therapeutic window, making dosage titration seemingly difficult and potentially causes respiratory failure. Its mechanism of action not only facilitates GABA-A activity potentiating benzodiazepines (synergistic effect) but also inhibits stimulating glutamate receptors (additional effect).

Currently, there is no standard dosing protocol. Hoffman and Weinhouse (2015) suggest phenobarbital 130-260 mg IV repeated every 20 minutes until symptoms are controlled. In a case study by Hayner, Wuestefeld, and Bolton (2009), a patient with benzodiazepine resistant AWS was given symptom-triggered doses of phenobarbital to control symptoms. Additionally, when the patient failed benzodiazepine weaning, scheduled doses of phenobarbital were given 3 to 4 times a day. This created a steady serum concentration state. They concluded that phenobarbital improved symptom control, minimized propylene glycol toxicity, facilitated weaning of benzodiazepines and was not associated with respiratory depression. A 2013

study by Rosenson and colleagues looked at the benefit of administering a single dose of phenobarbital in the emergency department (ED). It was concluded that an early single dose with symptom-triggered benzodiazepine regimen decreased ICU admissions and did not result in adverse reactions. Elderly, patients with hepatic disease and patients with pulmonary disease have an increased risk of over sedation and may not be a candidate for phenobarbital.

Clearly additional prospective data is required for the development of protocols for phenobarbital use in AWS. Research should focus on both benzodiazepine resistant AWS as well as early more prophylactic use.

Alternative Agents

Evidence clearly supports the use of benzodiazepines as the gold standard of practice for AWS. However, other agents may be considered in the treatment of AWS, though there are limited data and evidence is inconsistent. Neuroleptic agents (e.g., haloperidol [Haldol]) are not recommended as monotherapy for AWS. In comparison to benzodiazepines, they have been implicated in higher mortality, longer delirium, and more complications. They lower the seizure threshold, may interfere with heat dissipation, prolong the QT interval, and are not cross-tolerant with alcohol.

Commonly Used Medications

Sedative-Hypnotics: Benzodiazepines

Introduction

Benzodiazepines are the foundation of pharmacological therapy for AWS. These agents serve as a substitute for alcohol by acting on inhibitory GABA mediators, replacing the depressant effects on the CNS. As a substitute for alcohol, they counteract hyperactivity; thus, they are known to have “cross tolerance.” In addition to their sedative-hypnotic effect, they also have anticonvulsant properties and less adverse effects than other drugs in this classification. Benzodiazepines have been proven to be safe and effective in preventing and reducing withdrawal severity, including seizures and delirium.

Desired Effect: A variety of benzodiazepines are available with similar efficacies and no one drug has proven superiority; therefore, the nurse may see some variation in clinical practice. The choice of drug is based on the individual's clinical picture, comorbidities, and pharmacokinetic factors, including:

- Prophylaxis versus progressive AWS
- Age
- Route of administration
- Onset of action
- Duration (elimination, half-life)
- The presence of active metabolites
- History of previous withdrawals with seizures or DTs
- Impaired liver function

Benzodiazepines can be long acting, intermediate acting, or short acting. Diazepam (Valium) and chlordiazepoxide (Librium) are long-acting agents. Lorazepam (Ativan) and oxazepam (Serax) are intermediate-acting agents, and midazolam (Versed) is a short-acting agent.

Generally, long-acting agents allow for a smoother course with less chance of breakthrough symptoms and a decreased risk of seizures or delirium. They are preferred in moderate to severe withdrawals.

Intermediate-acting agents, preferred for older adults and individuals with liver and severe lung disease, have a higher incidence of breakthrough symptoms and seizures.

Nursing Responsibilities:

- The nurse anticipates that the route of administration and dosing will be guided by the clinical picture and the kinetic properties of the benzodiazepine chosen. The therapeutic goal is to achieve light somnolence. Evidence of light somnolence is that the patient sleeps when not stimulated, yet is easily arousable when sleeping. Intravenous administration has the quickest onset. Continuous intravenous administration is acceptable but is usually reserved for severe, complicated withdrawals requiring intubation and ventilation.
- Dosing requirements are affected by age, severity of signs and symptoms, comorbidities, and tolerance. It must be noted that these drugs are replacing alcohol. General principles are to start early based on symptoms and history rather than wait for withdrawal to advance, and anticipate higher doses in order to counteract the tolerance. Once withdrawal is prevented or under control and the patient's overall condition stabilizes, the doses and dosing intervals will be adjusted to wean patients off the original substance.
- For severe withdrawal, the nurse can expect to be administering larger doses of intravenous benzodiazepines. In fact, massive doses may be required in some severe cases. Apprehension in administering the necessary massive doses often results in undertreatment of AWS. The patient receiving massive doses is monitored in an ICU setting for hemodynamic stability and airway protection.
- Patients with a critical concurrent illness and/or severe withdrawal may require mechanical ventilation. Continuous intravenous sedation (midazolam/lorazepam/propofol/dexmedetomidine) may be acceptable for comfort and AWS symptom management.

Literature cautiously considers neuroleptic agents as an adjunct to benzodiazepine therapy when a patient has a known coexisting decompensated thought disorder like schizophrenia (Hoffman and Weinhouse, 2015). Perry (2014) advocates consideration when agitation, sensorial impairment, psychosis, and perceptual disorders are not well controlled with benzodiazepines.

Essential for Patient Centered Care

If neuroleptic agents are used in AWS, the patient must be monitored for the possible development of tardive dyskinesia and neuroleptic malignant syndrome.

It is important for the nurse to be aware of the problems associated with the use of haloperidol, especially if a facility does not have an attentive AWS screening protocol. Too often, especially if a patient has a psychiatric history, agitation is not properly evaluated and is quickly treated with neuroleptic agents. The nurse must remember that patients with alcohol use disorders have a high incidence of psychiatric and behavioral disorders.

Other medications that can be considered as adjuncts to benzodiazepines, but are not routinely used are:

- Beta blockers—(e.g., propranolol [Inderal]) for symptomatic treatment of autonomic activity. May worsen the delirium and mask symptoms of DT's. Patients

with known cardiac disease should receive their maintenance dose after sedation and fluid resuscitation.

- Centrally acting alpha-2 agonists (e.g., clonidine [Catapres])—can also reduce autonomic activity.

Caution must be taken with these agents as they can mask signs and symptoms of hemodynamic instability.

Interventions for the Treatment of Wernicke-Korsakoff Syndrome

Thiamine (vitamin B₁) deficiency is frequently seen in alcohol-dependent individuals because of insufficient dietary intake, impaired gastrointestinal absorption, and impaired utilization. Thiamine is an essential for normal metabolism and utilization of glucose. As a cofactor for several enzyme systems, it plays a role in cerebral energy utilization and maintenance of nerve impulses. Alcohol also depletes liver glycogen stores and impairs glucogenesis (the formation of glucose from glycogen); thus, hypoglycemia may accompany AWS. Administration of intravenous (IV) dextrose without thiamine administration increases the patient's risk for developing an acute neurological complication called **Wernicke's encephalopathy** (confusion, abnormal gait, and paralysis of certain eye muscles). This is a complication of a nutritional deficiency, not AWS. **Korsakoff's syndrome** (selective memory disturbances, amnesia) occurs in the majority of the patients recovering from both Wernicke's encephalopathy and AWS. When the two occur together, it is referred to as Wernicke-Korsakoff syndrome.

AWS treatment protocols initiate the administration of thiamine immediately and prior to any IV dextrose infusion to prevent Wernicke-Korsakoff syndrome. Patient history and risk factors for severe withdrawal will influence the route of administration. The parenteral route is preferred because the oral route can have erratic absorption. The nurse can anticipate administering thiamine 100 mg IM/IV as soon as possible. Generally, thiamine 100 mg is ordered daily in a liter of IV fluids for at least 3 days. Once a normal diet is tolerated, IV thiamine may be transitioned to dosages of 50 to 100 mg as tolerated orally. IV administration is safe with rare adverse effects. Larger doses of thiamine are administered to patients with Wernicke-Korsakoff syndrome. The nurse monitors the patient for any adverse effects of the medication and for signs and symptoms of Wernicke-Korsakoff syndrome.

Interventions for the Treatment of Alcohol Withdrawal Seizures

The nurse should implement seizure precautions for all high-risk patients. Alcohol withdrawal seizures do not require treatment with an anticonvulsant (Hoffman & Weinhouse, 2015). Benzodiazepines have anticonvulsant properties and

are usually sufficient for both primary and secondary prevention and status epilepticus. Patients with a history of withdrawal seizures or a predicted severe withdrawal may have higher initial doses ordered and a slower tapering schedule. As described, alcohol withdrawal seizures are often self-limited and can terminate spontaneously.

Interventions to Maintain Fluid and Electrolyte Balance

The nurse works collaboratively with the healthcare team to promote fluid and electrolyte balance. Signs and symptoms of dehydration and overhydration have to be accurately monitored. Severe withdrawal, including the DTs, typically causes dehydration. The goal of management is to maintain fluid and electrolyte balance.

Assessment of Fluid Balance

Early stages of withdrawal are often marked by fluid retention secondary to the effect of alcohol on antidiuretic hormone (ADH). Alcohol inhibits the secretion of ADH, causing an increase in urine output. In withdrawal, ADH is no longer inhibited, causing fluid retention. In severe AWS, especially complicated by the DTs, dehydration is a concern secondary to the additional fluid losses from vomiting, diarrhea, hyperventilation, diaphoresis, and fever. The nurse realizes that vital signs may not be an accurate reflection of fluid status secondary to the increased adrenergic stimulation from the withdrawal syndrome causing tachycardia and hypertension, and the massive doses of sedatives causing hypotension. Therefore, the nurse evaluates the following for evidence of dehydration or overhydration:

- Weight
- Vital signs/other hemodynamic values (see Chapter 5 )
- Intake and output
- BUN, creatinine, and electrolytes
- Skin and mucous membrane characteristics (warm, dry, moist, cool)
- Presence/absence of edema
- Lung sounds

Fluid Administration

Depending on the history and severity of the AWS, hydration is provided orally or intravenously. Typically, a glucose-based solution (e.g., D5¹/₂ NS) is initiated to administer thiamine. Protocols may also provide for multivitamin and folic acid administration either orally or intravenously. The rate of the IV will depend on:

- Ability to take oral supplements
- Other comorbidities, acute illnesses

- Duration of severe withdrawal symptoms
- Presence of autonomic/hemodynamic stability
- Diagnostic evidence from serum electrolytes, BUN, creatinine, and urinalysis

Occasionally, severe cases may need aggressive fluid resuscitation. Patients admitted with the DTs already in progress may already have significant fluid losses. Boluses of an isotonic solution, typically normal saline, may be required in addition to the glucose/vitamin solution. Critically ill patients may have fluid administration guided by hemodynamic monitoring (central venous pressure or pulmonary artery pressures).

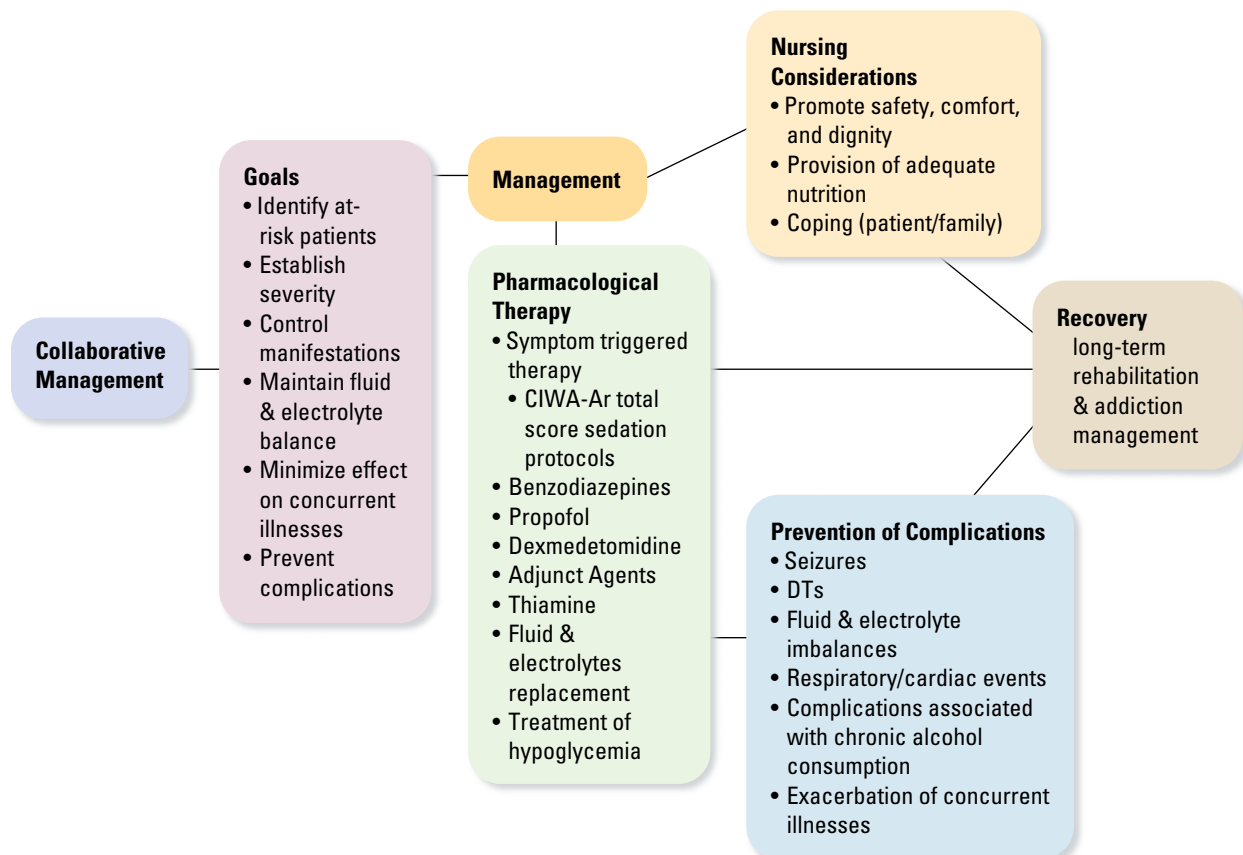
Assessment of Potassium Balance

A low serum potassium (hypokalemia less than 3.5 mEq/L) is a frequent finding in AWS related to inadequate intake, excessive diuresis, vomiting, and diarrhea. Manifestations of AWS can potentially mask the manifestations of hypokalemia. The nurse reviews the patient's serum potassium level and assesses for the cardiac signs of severe hypokalemia (less than 2.5 mEq/L), ventricular dysrhythmias, and electrocardiogram changes: flattened or inverted T wave, prominent U waves, prolonged QT, and ST depression.

Potassium Administration

Typically, the nurse administers potassium chloride (KCl) as ordered for replacement. Route of administration depends on the patient's serum level, the need for prompt replacement, and the patient's ability to take and tolerate supplements orally. Potassium administration is guided by serum potassium levels, renal function, hydration status, and protocols for specific concentrations and rate of administration. (Commonly, potassium sulfate is added to a liter of maintenance fluid one or more times a day.)

Because alcohol-dependent patients may also have associated low serum phosphorus (hypophosphatemia less than 1.7 mEq/L), potassium phosphate may be ordered based on the degree of phosphate deficiency. If low serum magnesium exists with hypokalemia, administration of potassium will not completely resolve the deficit without magnesium replacement. If the patient's potassium level is between 3.0 and 3.5 mEq/L, the nurse can expect that it will take between 100 and 200 mEq of KCl to elevate the potassium level 1 mEq/L (Kee, 2015). IV potassium can cause pain at the IV site irritating the vein, causing phlebitis, and infiltration can irritate the tissue, causing sloughing. Serum levels and resolution of signs and symptoms will serve as evaluative criteria.



Visual Map 12-2 AWS Management

Assessment of Magnesium Imbalance

A low serum magnesium (hypomagnesemia less than 1.5 mEq/L) is associated with chronic alcohol ingestion related to poor nutritional status, malabsorption, and increased magnesium excretion. During withdrawal, magnesium shifts into the cells secondary to metabolic changes, including alcohol ketoacidosis, lactic acidosis, and hypoglycemia, thus decreasing serum levels. Magnesium's membrane-stabilizing properties promote potassium and calcium homeostasis and may offer protection against seizures and arrhythmias. The nurse evaluates the serum potassium and serum calcium levels concurrently because hypokalemia and a low calcium level (hypocalcemia total calcium less than 4.5 mEq/L) can cause hypomagnesemia. Similar to hypokalemia, hypomagnesemia manifestations can mimic AWS manifestations. The nurse reviews the serum magnesium level and assesses for the following manifestations of severe hypomagnesemia (less than 1 mEq/L):

- Neuromuscular irritability/tetany symptoms (tremors, spasticity, spasms, twitching)
- Anorexia, nausea, or vomiting
- Behavioral changes (mood changes, confusion, insomnia)
- Tachycardia, hypertension, and cardiac dysrhythmias
- Electrocardiogram changes: inverted or flat T wave
- Positive Chvostek's sign (observes spasm of the cheek and twitching of the corner of the lip in response to tapping the facial nerve just in front of the ear)

Magnesium Administration

The nurse administers magnesium sulfate as ordered for a documented magnesium deficiency. A 10% magnesium sulfate (1 g/10 mL) solution is available for IV use. Currently, there is no concrete evidence that magnesium specifically benefits alcoholic delirium. Magnesium administration is guided by serum magnesium levels, renal function, hydration status, and protocols for specific concentrations and rate of administration.

The nurse anticipates that the patient may experience a hot flushed feeling with IV administration. Calcium gluconate should be available in case of magnesium toxicity. Serum levels and resolution of signs and symptoms will serve as evaluative criteria.

Assessment of Hypoglycemia (Decreased Blood Sugar Level)

Hypoglycemia is encountered in AWS because alcohol exhausts liver glycogen stores and impairs gluconeogenesis (the synthesis of glucose from protein or lipids).

Long-term metabolism of alcohol alters the metabolism of glucose, amino acids, and fats, increasing the susceptibility to hypoglycemia. Alcoholic hypoglycemia has both adrenergic and CNS manifestations (see Chapter 14 for manifestations). Management involves the administration of thiamine prior to IV glucose administration to prevent Wernicke-Korsakoff syndrome as discussed. The nurse can anticipate administering a dextrose-based IV solution (e.g., D5¹/₂ NS) to prevent hypoglycemia. Acute hypoglycemic episodes are treated with 50% dextrose as any episode of hypoglycemia (see Chapter 14 ∞).

NURSING CARE

The nursing care of the patient with alcohol withdrawal is centered on providing a safe environment and assisting the patient and family to cope with the immediate situation.

Maintaining Safety

The nurse focuses on maintaining patient safety while ensuring patient privacy and promoting patient dignity. The signs and symptoms of AWS can be uncomfortable, embarrassing, and often put the patient at significant risk for harm. Both the patient and the family will need support and understanding. The following interventions are implemented to decrease environmental stimulation and promote orientation and comfort:

- Quiet room; decreased lighting, sound, and activity
- Evaluation of visitation on an individual basis (see Chapter 2 ∞)
- Avoidance of caffeine
- Environmental adjuncts (clock, calendar), frequent reorientation
- Education related to what to expect from the withdrawal to both patient and family
- Reassurance and positive encouragement
- Consistent caregivers whenever possible

The nurse will also implement interventions to promote safety and dignity:

- Accurate assessment and appropriate medication administration per protocols (vital signs and level of consciousness [LOC])
- One-to-one continuous observation and monitoring
- Institute fall protocols relative to disorientation and sedation
- Seizure precautions
- Aspiration precautions
- Physical restraints to prevent injury to self and staff (see Chapter 2 ∞)
- Be safe; provide care as a team and dress appropriately with no dangling articles that can be pulled by a violent patient

Providing Nutrition

One of the health dangers of alcohol use disorders is malnutrition. Appetite is often suppressed by the euphoria and the empty calories of alcohol. Because alcohol affects all body systems, it can cause damage to the lining of the stomach and impair digestion. Because alcohol impairs absorption and metabolism, disrupts liver function, and increases the excretion of many nutrients, malnutrition can be present even when intake is adequate. In addition to protein energy malnutrition, there are significant

deficiencies in vitamins, minerals, and electrolytes causing bone loss, bleeding tendencies, anemia, and neurological changes.

All patients diagnosed with an AUD should have a nutritional consult. Damage to other organs must be a consideration in terms of requiring a special diet (cardiac, liver, renal dysfunction). As discussed, electrolyte replacement is guided by blood studies. Thiamine, multivitamins (MVI), and folate are routinely administered. Depending on the severity of the withdrawal, patients may be NPO

Safety Initiatives

Restraint Reduction

PURPOSE: To reduce restraint use in all settings in all hospitals with all patient populations

RATIONALE: The Final Rule is a regulation that “focuses on patient safety and the protection of patients from abuse.” It supports “patients’ rights in the hospital setting specifically the right to be free from the inappropriate use of restraints” with regulations that protect the patient when restraint is necessary. The use of restraint is not prohibited but “using restraints as a substitute for adequate staffing, monitoring, assessment, or investigation of the reason behind a patient’s behavior . . . which may be indicative of unmet needs” is.

Harmful consequences can occur either directly or indirectly from the use of restraints. Psychosocial consequences can develop, including anger, agitation, and depression. Some of the short-term physical consequences from the use of restraints are hyperthermia, new onset of bowel and bladder incontinence, new pressure ulcers, and an increased rate of nosocomial infections. Permanent injuries of the brachial plexus can occur from wrist restraints. At least a hundred deaths occurred in the nation’s hospitals between 1999 and 2004 from the use of restraints, more than 40% of which were not reported to the appropriate governmental agency as required.

HIGHLIGHTS OF RECOMMENDATIONS: A restraint is defined by the Final Rule as “any manual method, physical or mechanical device, material, or equipment that immobilizes or reduces the ability of a patient to move his or her arms, legs, body, or head freely. However, restraints are not devices such as orthopedically prescribed devices, surgical dressings or bandages, or devices to protect the patient from falling out of bed, or to permit the patient to participate in activities without the risk of physical harm.” Thus, the Final Rule does not apply to measures (such as side rails) that might keep a patient who is recovering from anesthesia, sedated, or on a therapeutic bed from falling out of the bed. However, it does apply to patients who might be trying to leave their bed, because this might cause entrapment. The Final Rule states that clinical

judgment determines whether side rails are considered restraints and governed by the Final Rule or not.

According to the Final Rule restraint may only be used when:

- Less restrictive interventions have been determined to be ineffective to protect the patient or others from harm.
- The type or techniques used are the least restrictive intervention that will be effective.
- It is in accordance with a written modification to the patient’s plan of care and implemented in accordance with safe and appropriate techniques as determined by hospital policy and state law.

In addition:

- Restraint must be in accordance with the order of a physician or other licensed independent practitioner who is responsible for the care of the patient and is authorized to order restraint by hospital policy in accordance with state law.
- The order may **never** be written as a standing order or on an as-needed basis.
- The use of restraint must be discontinued at the earliest possible time, regardless of the length of time identified in the order.

Required documentation includes:

- The patient’s condition or symptoms that warranted the use of the restraint
- The patient’s response to the intervention, including monitoring the physical and psychological well-being of the patient who is restrained for respiratory and circulatory status, skin integrity, vital signs, and any special requirements specified by hospital policy associated with the 1-hour face-to-face clinical identification of specific behavioral changes that indicate that restraint is no longer necessary.

Sources: Federal Regulation, Vol. 71, No. 236, December 8, 2006; National Government Clearinghouse.

because they are at high risk for aspiration pneumonia related to agitation, seizures, sedation, and impaired immune system secondary to chronic alcohol use.

In the critical care units, nutritional status is monitored closely and short-term nutritional requirements are usually easy to meet by enteral or parenteral means. Long-term maintenance of nutritional status may be a challenge and will depend on the success of the patient's efforts to abstain from alcohol. A collaborative team effort is initiated with the patient and responsible support systems to develop a nutritional/diet teaching plan.

Fostering Patient and Family Centered Care

Coping may be very difficult for the patient and the family. They are often dealing with withdrawal symptoms that involve violent and embarrassing behavior. Depending on its severity, AWS may be life threatening. Chances are the patient and family have been dealing with the social, legal, occupational, and interpersonal implications of an AUD for some time. Additionally, AWS is often coupled with an acute illness. The nurse needs to be sensitive to the needs of the patient and family. When the patient is adequately sedated, the family may be the priority. Often families experience guilt in response to the situation. The nurse should:

- Evaluate the patient and family's coping strategies.
- Provide reassurance regarding current and anticipated signs and symptoms, especially behavior.
- Encourage questions and discussion of negative feelings.
- Promote forgiveness.
- Provide education (consider level of learning and willingness and readiness to learn):
 - Related to duration and resolution of AWS
 - Related to other comorbidities influencing the patient's condition
- Collaborate with other members of the health team to evaluate for and initiate necessary referrals.

Prevention of Complications

Prevention of complications is crucial to patient recovery and positive patient outcomes. Mortality from AWS in the general population is related to development of the DTs. If the DTs are unrecognized or untreated, mortality is as high as 35%. With early recognition and treatment, mortality is less than 5% (Hoffman and Weinhouse, 2015). Other factors influencing mortality are older age, core temperature greater than 104°F (40°C), and preexisting pulmonary and liver disease. Additionally, the critical care patient may have the added burden of a critical illness coupled with AWS.

The nurse needs to consider the effect of chronic alcohol use on all body systems and anticipate cardiac, respiratory, hepatic, neurological, renal, and gastrointestinal complications.

Nursing Diagnoses

Critically Ill Patient Experiencing Alcohol Withdrawal and Liver Failure

- Anxiety related to situational crisis: alcohol withdrawal
 - Acute confusion related to the effects of alcohol
 - Dysfunctional family processes: alcoholism related to abuse of alcohol
 - Excess fluid volume related to portal hypertension
 - Imbalanced nutrition less than body requirements related to suppression of appetite and inability to absorb nutrients
 - Risk for injury related to alcohol intoxication or withdrawal and abnormal blood profile (increased serum ammonia)
 - Risk for infection related to chronic liver disease
 - Risk for deficient fluid volume related to loss of fluid from hemorrhage, vomiting, severe fluid shift, or diuresis
-

The nurses' role in prevention of complications for patients experiencing AWS is to limit the progression of AWS and ultimately to prevent the DTs. Concerns with severe AWS and the treatment of the DTs are the potential for massive doses of sedation, self-harm, and the physiologic effects of autonomic hyperactivity. The nurse:

- Monitors airway and breathing status
- Monitors cardiac status for dysrhythmias, decreased myocardial perfusion, cardiovascular collapse
- Supports fluid and electrolyte status
- Takes prompt action for signs and symptoms refractory to pharmacological therapy

Chronic alcohol use affects all body systems. The nurse anticipates and assesses for the development of the following complications:

- Respiratory infections
- Acute respiratory distress syndrome (ARDS)
- Metabolic dysfunction (lactic acidosis, ketoacidosis, hypoglycemia, electrolyte abnormalities)
- Cardiovascular complications (cardiomyopathy, atrial and ventricular dysrhythmias, variant angina)

The nurse recognizes that long-term alcohol misuse is one of the leading causes of chronic liver disease and liver failure in the United States. Therefore, the nurse assesses the patient undergoing AWS for the development of any of the following manifestations of liver failure.

Gerontological Considerations

Alcohol Withdrawal and Misuse

Age-related changes can cause older adults to have a greater sensitivity to the effects of alcohol. A decrease in total body fluid results in higher blood alcohol concentrations.

Decreased hepatic blood flow, slower metabolism, and increased permeability of the blood-brain barrier cause an increase in the duration of exposure and significant effects from smaller amounts of alcohol in older adults.

It is estimated that interactions between alcohol and prescription medications result in problems for 17% of older adults.

Most at-risk older drinkers are Caucasian men aged 65 to 74, 25% of whom had a history of heavy drinking when they were younger.

The acute care alcohol-related admission rate for older adults is equal to the admission rate for myocardial infarction (MI) in older adults.

Although older men are more likely to be admitted with problems from alcohol misuse, older women are more likely to have been drinking alone so that significant others are not aware of their pattern of alcohol consumption.

The likelihood of serious complications developing from liver disease is greater in older adults because, as a person ages, the liver is less able to recover from severe physiologic stressors or to tolerate toxic accumulations.

It is likely that the liver cells of older adults are less able to regenerate following an insult.

It can be easy to miss hepatic encephalopathy when it develops in an older person because it may be mistaken for dementia or delirium.

These manifestations will be described further elsewhere in the chapter:

- Hepatic encephalopathy
- Wernicke-Korsakoff syndrome
- Gastrointestinal bleeding
- Renal insufficiency/failure

Acute Liver Failure

In the United States, the most common reasons for the development of liver failure are alcohol misuse, acetaminophen overdose, and hepatitis. These factors may precipitate liver failure singly or in conjunction with one another. Liver failure can be acute, chronic, or acute superimposed on chronic. It can be caused by an acute injury where the patient will rapidly develop massive liver cell death, or it can be the result of a chronic disease. Patients with chronic liver disease may have a precipitating event that causes them to experience an acute deterioration in liver function; this is known as acute on chronic disease.

Etiology of Acute Liver Failure

Acute liver failure is an uncommon syndrome, estimated at 3000 cases per year in the United States. It is defined by the onset of coagulopathy (international normalized ratio [INR]1.5) and mental status changes within 26 weeks of presentation (O'Grady, 2016). There is a rapid development of acute liver injury, loss of hepatic cellular function, and the liver can no longer meet the metabolic demands of

the body. ALF is likely underreported and the low incidence makes it difficult to attain reliable data and evidence-based interventions.

The most common causes of acute liver failure (ALF) include:

- Toxins/drugs especially acetaminophen
- Viral hepatitis (inflammation of the liver)—though hepatitis A is the most common form of acute hepatitis, hepatitis B is the most common hepatitis that causes ALF
- Vascular—hypoperfusion/shock states/ischemic injury
- Metabolic—Wilson's disease, acute fatty liver of pregnancy
- Unknown—unidentified toxins and autoimmune processes are proposed underlying mechanisms (O'Grady, 2016)

In ALF, the specific etiology guides management decisions and is one of the best prognostic markers. Early identification of the cause is a critical component of care. Diagnosis is clinical, based upon coagulopathy and encephalopathy. Determinants of outcomes/prognosis are age and grade of encephalopathy. Poorer outcomes are associated with a history of jaundice for more than 7 days before the onset of encephalopathy. Other variables influencing the prognosis are the development of complications including cerebral edema, renal failure, acute lung injury (ARDS), and infections. In view of the significance of acetaminophen overdose as a cause of ALF, assessment and management of ALF are described.

COLLABORATIVE CARE

ALF is a medical emergency with the potential for a high mortality rate resulting from the consequences of cerebral edema and the development of multisystem organ failure. The assessment and management of ALF involve recognition, prompt care in a critical care unit to support all systems and prevent complications, aggressive search for the precise etiology to guide treatments, and early evaluation of transplant criteria.

Patient History

Review of the patient's history should focus on exposure to viral infections, drugs or other toxins, or other recent injuries or illnesses. The patient and family should be questioned specifically about acetaminophen intake.

Clinical Manifestations

Clinical manifestations may result from impaired liver function and the effects of liver dysfunction on other organs. The nurse assesses for nonspecific complaints commonly seen with early liver dysfunction such as nausea, vomiting, and malaise. Assessment for signs and symptoms of liver failure must also be considered.

The nurse assesses for characteristic manifestations and for potential complications of ALF. The nurse assesses the following indicators of ALF:

- Neurological status—requires vigilant assessment because **hepatic encephalopathy (HE)**, cerebral edema, and increased intracranial pressure can rapidly progress to brainstem herniation and death.
 - Altered mental status, level of consciousness
 - Presence and stage of HE (HE is a condition characterized by a wide range of potentially reversible neuropsychiatric manifestations. See the discussion earlier in the chapter.)
 - Evidence of cerebral edema, increased intracranial pressure (see Chapter 10 )
- Coagulopathy
 - Bruising or acute bleeding
 - Increased prothrombin time/INR > 1.5
 - Thrombocytopenia
 - Signs and symptoms of disseminated intravascular coagulation (DIC)
- Jaundice—severe acute liver injury can lead to impaired elimination of bilirubin; thus, jaundice may appear immediately before or soon after presentation.

The nurse also assesses for potential complications of ALF:

- Respiratory insufficiency/failure—increased pulmonary vascular permeability and structural alterations in pulmonary vasculature increase risk for ARDS

- Hemodynamic instability—related to hyperdynamic circulation (decreased systemic vascular resistance, decreased pulmonary vascular resistance, increased cardiac output, increased metabolic rate, low mean arterial pressure) and increased risk of bleeding (coagulopathy, stress ulceration)
- Infection/sepsis—related to decreased immune function and invasive procedures. May trigger a systemic inflammatory response syndrome (SIRS).
- Hypoglycemia—related to decreased glucose synthesis
- Acid-base and electrolyte disturbances—metabolic acidosis, respiratory alkalosis, hypokalemia, hyponatremia, hypophosphatemia
- Renal injury—related to hypovolemia, increased risk for acute kidney injury
- Malnutrition—preexisting, prevention of catabolism related to increased metabolic rate

The nurse anticipates an extensive diagnostic workup in an effort to evaluate cause and severity.

The nurse works collaboratively with the healthcare team to treat the specific cause and to provide intensive supportive care to prevent, recognize, and treat complications.

Critical Care Monitoring

Management includes critical care monitoring and support of the following systems because the patient's condition can deteriorate rapidly:

- Respiratory support—possible mechanical ventilation for management of cerebral edema and ARDS. Ventilation strategies for ARDS can be problematic in ALF related to potentially worsening cerebral edema.
- Circulatory support—hemodynamic monitoring, fluid management, vasopressor support
- Neurological support—HE is a major complication of ALF and a prognostic marker because it is progressive unless the liver failure is resolved. Treatment focuses on resolution of liver failure, decreasing ammonia levels, and monitoring intracranial pressure with interventions to decrease cerebral edema. The goal is to maintain intracranial pressure below 20 mmHg and the cerebral perfusion pressure above 50–60 mmHg (Goldberg and Chopra, 2015a). During certain stages of HE, the patient may experience agitation requiring judicious administration of sedation and analgesia.
- Coagulopathy—another major complication and prognostic marker. Replacement therapy for thrombocytopenia or increased prothrombin time/INR is recommended in active bleeding and prior to invasive procedures. Serial measurements of uncorrected coagulation values are needed for prognosticating and timing for liver transplant.

- Gastrointestinal support—prevention of stress ulcers with a proton pump inhibitor, provision of nutrition (enteral preferred) to prevent malnutrition
- Renal support—volume replacement, avoidance of nephrotoxic drugs, continuous renal replacement therapy (CRRT), vasopressor support
- Infection—aseptic care, monitor for infection/sepsis, appropriate surveillance cultures, antibiotic prophylaxis may be considered
- Metabolic support—treat electrolyte and acid-base disorders, monitor blood glucose level, and treat hypoglycemia

Liver transplantation has greatly improved survival rates and remains the most definitive treatment. Early consideration must be given to the likelihood of recovery without a transplant and the contraindications to transplantation. Prompt evaluation, referral, and transport to a transplant facility are imperative when prognostic markers show a high probability of death.

Acetaminophen Toxicity

Acetaminophen, a readily available, commonly used analgesic, is one of the most common contributors to ALF. As a component of many over-the-counter medications and prescription combinations, patients unknowingly may be taking several drugs that contain acetaminophen. In addition, because of easy access, it is a popular drug for intentional overdoses. In therapeutic doses, 1 to 4 grams/day for an adult, it has an excellent safety profile. However, it is a dose-dependent drug that can result in both fatal and nonfatal hepatotoxicity when overdosed, misused, or combined with large amounts of alcohol.

Age, malnutrition, fasting state, dose, blood level of acetaminophen, certain medications (antitubercular and antiepileptic drugs), time of presentation, and chronic alcohol intake influence the metabolism of acetaminophen and increase the risk of toxicity. There is controversy related to the optimal dose of acetaminophen in individuals who regularly consume alcohol. Generally, the recommended dose is 2 grams per 24 hours for individuals consuming two to three drinks a day.

Acetaminophen is readily absorbed from the GI tract, primarily metabolized by the liver, and has a half-life of 2 to 4 hours (Burns, Friedman, & Larson, 2015). Peak levels may not occur until 4 hours after an overdose as the half-life is prolonged after toxic doses. When acetaminophen is taken in therapeutic doses, 90% is metabolized to a conjugate, which is excreted in the urine. A portion of acetaminophen that remains is excreted unchanged in the urine, and the remaining portion is metabolized into a toxic metabolite. Normally, this toxic metabolite is conjugated by hepatic glutathione to form a nontoxic substance that is excreted in the urine.

When there is an acute overdose or the maximum daily dose is repeatedly exceeded, the normal pathways of metabolism become saturated and more of the toxic metabolite is produced. When the hepatic glutathione stores are depleted, the toxic substances accumulate, causing hepatic injury. The minimal dose that can produce liver injury varies between 4 to 10 grams. Hepatotoxicity usually occurs when the dose is greater than 10 grams in a 24-hour period, but should be considered whenever the dose exceeds 4 grams/day.

N-acetylcysteine (NAC) (Mucomyst) is an antidote that counteracts the effects of acetaminophen toxicity. It should be administered immediately in patients at risk for hepatotoxicity. When administered within 8–12 hours of ingestion of a single dose it can eliminate significant hepatic injury. NAC has several mechanisms of action:

- Limits the formation and accumulation of the toxic metabolite
- Increases glutathione stores
- Directly detoxifies the toxic metabolite to a nontoxic substance
- Has both anti-inflammatory and antioxidant effects that may limit tissue injury
- Has vasodilating and positive inotropic effects to improve microcirculatory blood flow and oxygen delivery (Farrell, 2015)

There is continual debate related to the optimal route of administration and the duration of administration. Common dosing protocols include an intravenous 20-hour protocol and an oral 72-hour protocol as described. However, most experts agree that NAC therapy should be tailored to the individual patient using clinical data for endpoints rather than time to determine the duration of treatment (Heard & Dart, 2015).

The predicted risk of toxicity from a single acute overdose depends on the time of ingestion and serum acetaminophen level. Whether a level is toxic or nontoxic can only be interpreted if the time of ingestion is known. The Rumack-Matthew nomogram can be used to predict hepatic toxicity between 4 and 24 hours after an acute ingestion.

The diagnosis and risk assessment for repeated high therapeutic doses is less concrete. The clinical manifestations are often subtle and nonspecific, and do not predict delayed hepatotoxicity. Diagnosis depends on an insightful history, clinical manifestations, and laboratory studies but not the Rumack-Matthew nomogram. Instead, the acetaminophen level, INR, and the aminotransferase (AST/ALT) level are used to guide treatment. The prognosis is favorable for a single overdose if the antidote is administered promptly. However, prognosis for repeated chronic overdose is less clear because the onset of toxicity is not always apparent.

Assessment and management of acetaminophen toxicity require prompt recognition or suspicion, supportive care, prevention of absorption, and antidotal treatment to promote drug elimination. It is also important to recognize the differences between a single acute overdose and repeated chronic overdoses. The goal is to administer the antidote within 8–12 hours of acute ingestion.

Assessment: Acetaminophen Toxicity / Acute Liver Failure

The nurse must review and investigate historical data related to acetaminophen intake. It is important to establish:

- **Intent**—Is it an intentional ingestion of a large amount of acetaminophen in an effort to commit suicide or an unintentional ingestion of high quantities therapeutically for pain over a period of time? If the patient's intent was to commit suicide, the patient needs to be placed on suicide precautions and have a psychiatric consult.
- **Dose.**
- **Pattern**—Single/repeated.
- **Time of ingestion.**
- **Coingestants**—It is important to establish the particular form of the drug or drugs (extended release, other drugs, or combination drugs).

The clinical manifestations of an acute acetaminophen overdose are generally delayed and seen in four phases (Burns et al., 2015).

- **Phase I - 0 to 24 hours**—Anorexia, nausea, vomiting, malaise, pallor, lethargy, diaphoresis, and dehydration may be present or the patient may be asymptomatic. Laboratory studies are usually normal.
- **Phase II - 24 to 72 hours**—Nausea and vomiting usually resolve temporarily, and the patient may improve clinically, or right upper quadrant abdominal pain/tenderness on palpation, tachycardia, hypotension, and hepatomegaly may develop. AST and ALT begin to elevate by 24 hours with progressive elevation by 36 hours. In a massive overdose, these values can rise as early as 8–12 hours. Prothrombin time (PT)/INR, total bilirubin, and renal function tests may elevate.
- **Phase III - 72 to 96 hours**—Decreased alveolar compliance, atelectasis, and fluid-filled alveoli interfere with gas exchange across the alveolar-capillary membrane. Blood oxygen (PaO_2) levels fall. Because carbon dioxide diffuses more readily than oxygen, however, blood carbon dioxide (PaCO_2) levels also fall initially as tachypnea causes more CO_2 to be expired.

- **Phase IV - 4 to 14 days or longer**—Recovery phase typically begins by day 4, and is often complete by day 7. Severely ill patients may take several weeks to recover. Chronic liver impairment does not ensue.

Therefore, the nurse should assess for the following phases and symptoms

The nurse reviews the following studies for patients with acetaminophen toxicity:

- **Arterial blood gases**—A pH less than 7.3 that does not respond to fluid administration indicates a poor prognosis and a high mortality rate without a transplant.
- **Serum creatinine**—Renal injury often occurs with hepatic failure. The severity of the ingestion influences the incidence of renal dysfunction.
- **Prothrombin time/INR**—Elevates 24 to 72 hours after an acute ingestion due to liver cell damage and is an early prognostic indicator.
- **Serum total bilirubin**—Elevates with hepatocellular injury. It starts in stage II and continues to elevate. A bilirubin higher than 10 mg/dL may lead to a false positive acetaminophen level.
- **Serum acetaminophen level**—This value can help predict the risk of liver injury following a single ingestion. Acute single overdose patients need to wait to have this drawn until at least 4 hours after ingestion. A level drawn before 4 hours may not represent the peak value. Therapeutic level 10–30 mcg/mL.
- **Aminotransferases**—Elevate 24 to 72 hours after acute ingestion. Levels greater than 1000 IU/L define toxicity.

Essential for Evidence-Based Practice

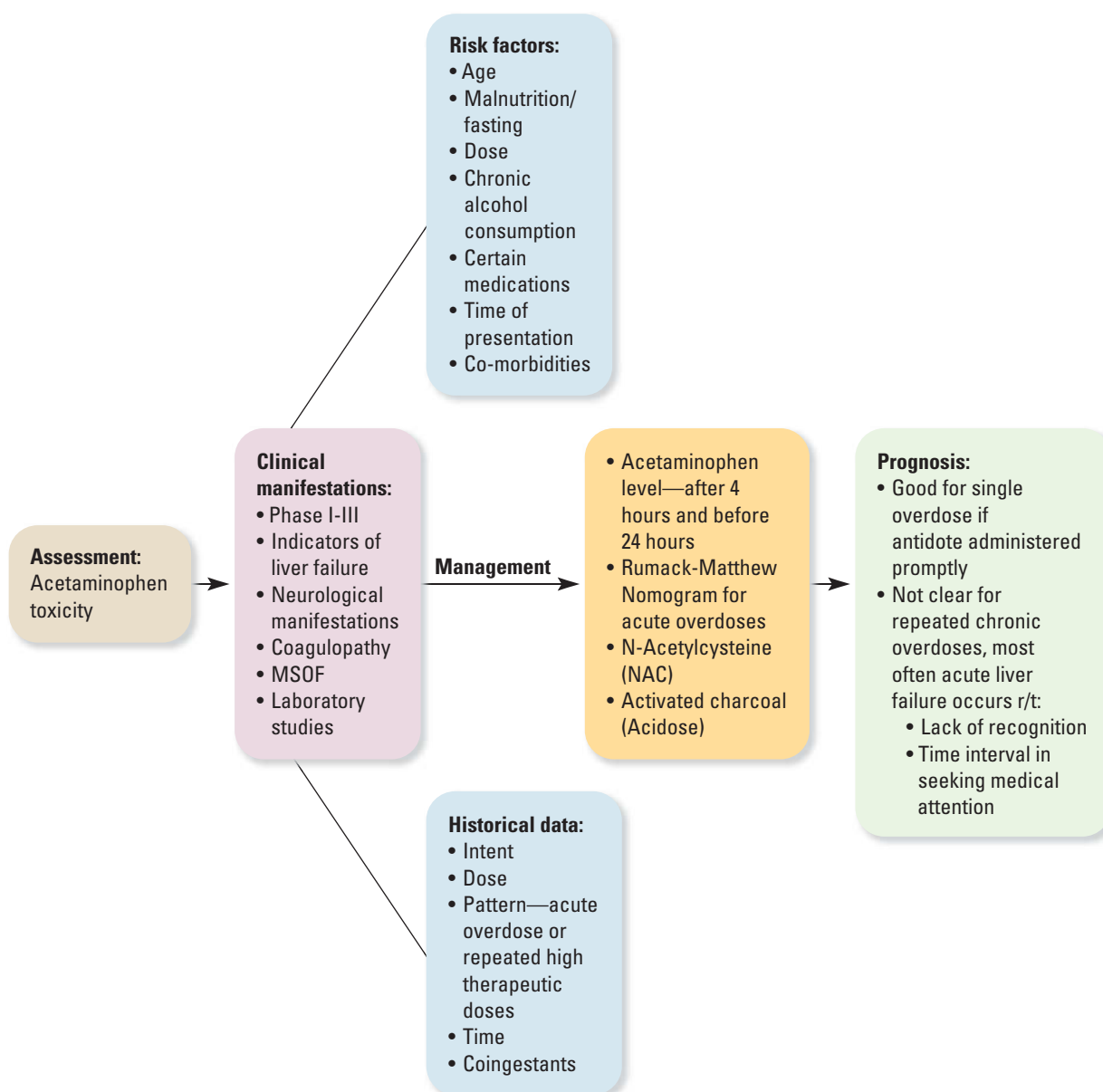
Aminotransferases are the hallmark of acetaminophen hepatotoxicity and levels can be between 2000 and 10,000 U/L (O'Grady, 2016). Acetaminophen overdose should be considered in all patients with levels higher than 1000 IU/mL.

COLLABORATIVE CARE

The nurse works collaboratively with the healthcare team and the regional poison control center to diagnose and treat acetaminophen toxicity. The goal of management is to promptly administer the antidote to prevent extensive hepatotoxicity and acute liver failure.

Interventions for Acetaminophen Toxicity

Establishing the exact time of ingestion is crucial to using the Rumack-Matthew nomogram because it plots the



Visual Map 12-3 Acetaminophen Toxicity Assessment and Management

hours after ingestion against the acetaminophen level. A level drawn before 4 hours does not represent the peak value and should not be used. If the patient presents within 4 hours of a massive ingestion, gastric lavage of pill fragments and administration of activated charcoal to reduce absorption may be considered (Heard and Dart, 2015).

If the ingestion was more than 4 hours but less than 24 hours prior to presentation, a serum acetaminophen level is drawn immediately. A serum acetaminophen level drawn during that time interval can be plotted on the Rumack-Matthew nomogram (an interactive version of the nomogram is available from ARS Informatica) to estimate whether the patient is at “no risk,” “possible risk,” or “probable risk” of hepatotoxicity. This information determines whether antidotal treatment is indicated. The upper line on

the nomogram is the probable risk line and the lower line is the possible- risk line. The standard of care is to start antidotal treatment with N-acetylcysteine (NAC) (Mucomyst) on all patients who fall above the possible risk line.

Chronic Liver Failure

Cirrhosis is a consequence of chronic liver disease. The most common causes of cirrhosis are excessive alcohol consumption, viral hepatitis, and fatty liver disease. In excessive alcohol consumption (alcoholic cirrhosis/Laennec’s cirrhosis), alcohol is absorbed from the small intestine and brought directly to the liver by the blood where it is converted to a toxic chemical. Hepatitis C is the most

Commonly Used Medications

N-Acetylcysteine (NAC) (Mucomyst)

Desired Effect: N-acetylcysteine (NAC) (Mucomyst) is the antidote for acetaminophen toxicity and is administered if the serum acetaminophen level drawn between 4 and 24 hours or more after a single ingestion falls above the “possible risk” line on the Rumack-Matthew nomogram. NAC (Mucomyst) is also recommended based on less concrete criteria and historical data. Overall, it is administered if the patient has risk factors for toxicity, suspected ingestion either single or repeated, and indications of hepatotoxicity or liver failure.

Nursing Responsibilities:

- Oral dosing is initiated on nonpregnant patients with a functioning GI system and no indications of hepatotoxicity. The loading dose is 140 mg/kg followed by 17 doses of 70 mg/kg every 4 hours. It is typically available in a 20% solution (200 mg/mL) and diluted to a 5% solution with fruit juice, a carbonated beverage, or water.
- The nurse assesses the patient’s GI system to evaluate tolerance of the medication. The solution has a foul “rotten-egg” odor, and patients best tolerate the smaller volume of solution in chilled orange juice. Antiemetic therapy is considered for the patient with nausea and vomiting. Ondansetron (Zofran) and metoclopramide (Reglan, [Chapter 13 ]) are frequently used. If the patient vomits within an hour of the dose, it should be repeated as rapidly as possible.
- IV dosing of NAC is acceptable in all cases of acetaminophen toxicity. It is recommended instead of the oral dosing:
 - When the patient is unable to tolerate oral NAC
 - When the patient’s condition contraindicates the administration of oral NAC (e.g., GI bleeding/obstruction)
 - In patients with significant hepatotoxicity
 - In pregnant patients
 - With coingestants requiring ongoing GI contamination
- The IV loading dose is 150 mg/kg infused through a peripheral IV over 1 hour using an in-line 0.2-micron filter. Some sources concur with administering the loading dose over 15 minutes (Heard & Dart, 2015). The maintenance dose is a continuous IV infusion over the next 20 hours. Usually different doses are infused over the first 4 hours and the remaining 16 hours. Depending on the presence or absence of biochemical evidence of hepatic failure (INR < 2) and whether it was a single or repeated chronic ingestion, several different maintenance protocols exist. The nurse should follow facility policy for specific dosing protocols and dilution guidelines.
- The nurse assesses for the response to NAC by monitoring patients for any signs or symptoms of hepatotoxicity and following the laboratory studies (PT/INR, creatinine,

liver function tests). It must be noted that laboratory evidence of hepatic damage may not be seen for 36 hours. Lastly, if the patient is progressing or is likely to progress to ALF, the patient should be transferred to a liver transplant center.

- **Side and/or Toxic Effects:** The nurse assesses for adverse effects of the IV preparation, including pruritus, flushing, nausea, fever, chill, urticaria, bronchospasms, and anaphylaxis. Non-life-threatening effects are treated by holding the infusion, administering antihistamines, and restarting the infusion at a slower rate. IV NAC should be used with caution in patients with a history of bronchospasms or asthma.

Activated Charcoal (Actidose)

Desired Effect: Activated charcoal (Actidose) 50 grams is administered to all patients if ingestion has been within 4 hours. Activated charcoal is a decontamination agent that prevents absorption of acetaminophen by absorbing the drug in the intestine. For maximum effect, it needs to be administered within 30 minutes after ingestion. There has been some concern with administering the charcoal concurrently with the antidote. The administration of activated charcoal or the antidote should not be delayed. Activated charcoal (Actidose) will absorb some of the antidote but not sufficiently to affect its detoxifying effects or to warrant increased doses of the antidote.

Nursing Responsibilities:

- The nurse should administer activated charcoal (Actidose) as ordered. The nurse must consider the patient’s mental status, LOC, and ability to cooperate. Altered mental status, decreased LOC, and uncooperative patients are at risk for aspiration and should have the activated charcoal (Actidose) administered through a nasogastric tube.
- Activated charcoal (Actidose) is a black tasteless gritty solution. Some preparations are available in a cherry flavor. The nurse should follow the manufacturer’s instructions and the facility’s policy regarding administering the activated charcoal. Some preparations need to be mixed with water and others are available in a ready-to-use suspension. All preparations need to be shaken well. The black solution is not very appealing, so to increase compliance, the nurse can put it in an opaque container with an opaque cover and offer a straw.

Side and/or Toxic Effects: The nurse anticipates that the patient may vomit, especially if the solution is administered rapidly. The nurse should be prepared to protect the airway if vomiting occurs. The frequency, quantity, color, and consistency of the stools should be assessed. The activated charcoal (Actidose) will color the stools black.

Bariatric Considerations

Nonalcoholic steatohepatitis (NASH) resembles alcoholic liver disease, but occurs primarily in overweight and obese patients. It is not associated with alcohol intake. Approximately 20% of Americans have “fatty liver,” the earliest stage of both NASH and alcoholic liver disease, but only about 2% to 5% eventually develop the inflammation and scarring that lead to permanent dysfunction in NASH. Currently, NASH is the third most common reason for the development of cirrhosis in the United States.

During the initial stage, the overweight person is usually asymptomatic, and NASH is suspected because of elevations in routine tests of liver function. A liver biopsy is required to confirm the diagnosis of NASH.

As the disease progresses and fibrosis develops, the patient may begin to experience fatigue, weight loss, and weakness. The disease may progress slowly over years until the patient develops cirrhosis, usually between the ages of 50 and 60. Most of these patients also have insulin dependent diabetes.

There are no specific therapies for the management of NASH. Therefore, patients are advised to reduce their weight, eat a balanced diet, increase their physical activity, and avoid both alcohol and unnecessary medications. Experimental therapies include antioxidants and antidiabetes medications. With the obesity epidemic, it is predicted that in a decade this will be one of the most common causes of cirrhosis in the United States.

dangerous form of chronic viral hepatitis, with a significant number of people with chronic disease developing cirrhosis after 20 years. Recent manifestations used to treat hepatitis C have been effective in preventing chronic liver disease. Because of the increasing prevalence of alcohol use, hepatitis C, and obesity in the United States, the incidence of cirrhosis is expected to rise.

Alterations in Structure and Function Occurring in Cirrhosis

Cirrhosis is the result of many years of inflammation and injury to the liver resulting in severe scarring. The gradual destruction, death, and regeneration of functional liver tissue have significant consequences for the liver and other organs. The regenerative response to cell injury and death involves nodule formation and replacing dead liver cells with fibrous tissue, which leads to fibrosis (scarring).

Anatomically, such an abnormal pattern of regeneration alters the functional component and the architecture of the sinusoids including the vasculature and bile ducts. Over time, liver function deteriorates secondary to altered bile duct flow and stasis, impaired hepatic venous outflow, and decreased blood flow to and through the liver because of increased resistance.

Portal Hypertension

The normal pressure in the portal system is about 5 to 10 mmHg, and higher values are defined as portal hypertension. Portal pressure is the result of two dynamic factors:

- The amount of blood draining into the portal venous system is controlled by:
 - Vascular tone (vasoconstriction/vasodilation) of the mesenteric arterioles.

- Hyperdynamic circulation: an increase in portal venous flow, which is marked by peripheral and splanchnic vasodilation, reduced mean arterial pressure, and increased cardiac output, is responsible for increasing the inflow of systemic blood into the portal circulation.
- The amount of resistance to the blood flowing out of the portal venous system caused by:
 - Distorted hepatic sinusoids.
 - Intrahepatic vasoconstriction.

Portal hypertension (♦ Figure 12-3) develops over time, is initially asymptomatic, and is responsible for an array of complications that can markedly reduce patients' life expectancy. The following complications are directly attributable to portal hypertension:

- Ascites
- Variceal hemorrhage
- HE, portosystemic encephalopathy (PSE)
- Hepatorenal syndrome

In the early stages, treatment of cirrhosis and portal hypertension can be directed toward the underlying cause in an effort to halt the cirrhosis and preserve whatever function may remain. In alcoholic cirrhosis, abstinence from alcohol may decrease liver cell injury and improve portal hypertension. Prevention and treatment of complications may enhance the quality and lengthen the life of patients in the early stages. In the later stages, cirrhosis is progressive and irreversible, so liver transplantation is the only feasible treatment.

The modified Child-Pugh classification system is used to classify the severity of the disease, predict the development of complications, and anticipate 1-year survival rates.

Portal hypertension

Bands of fibrotic scar tissue obstruct the sinusoids and blood flow from the portal vein to the hepatic vein. Pressure in the portal venous system, which drains the gastrointestinal tract, pancreas, and spleen, increases. This increased pressure opens collateral vessels in the esophagus, anterior abdominal wall, and rectum, allowing blood to bypass the obstructed portal vessels. Prolonged portal hypertension leads to the development of (1) varices (fragile, distended veins) in the lower esophagus, stomach, and rectum; (2) splenomegaly (an enlarged spleen); (3) ascites (accumulation of fluid in the abdomen); and (4) hepatic encephalopathy (disrupted CNS function with altered consciousness).

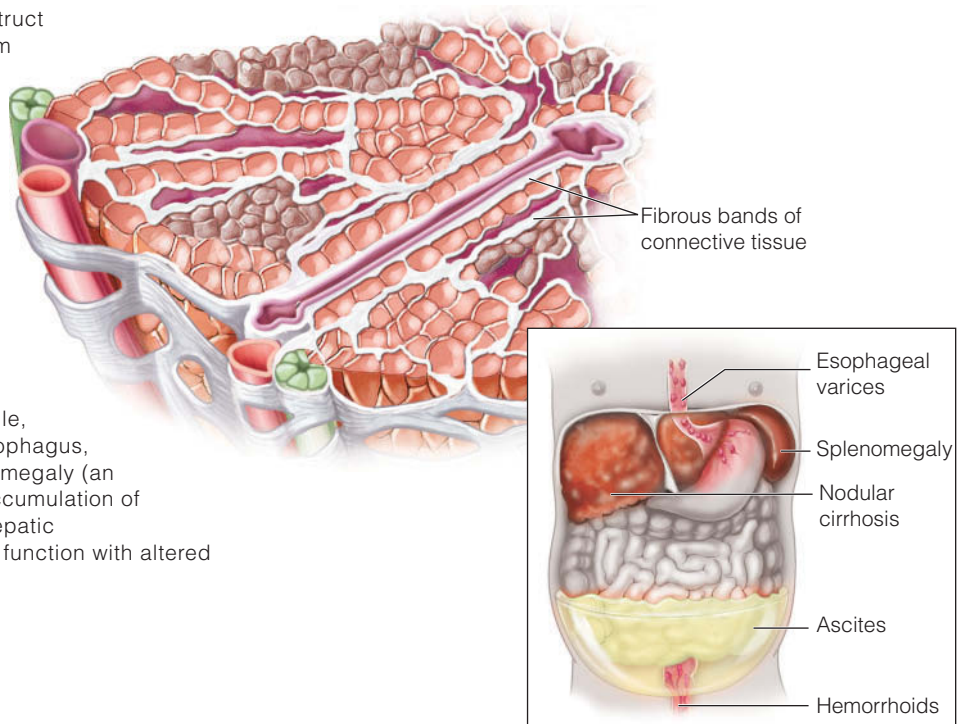


Figure 12-3 Portal hypertension.

Based on blood tests and presence of symptoms, the system grades five parameters:

- Degree of ascites
- Bilirubin mg/dL
- Albumin g/dL
- Prothrombin time (seconds over control and INR)
- Degree of encephalopathy

COLLABORATIVE CARE

The assessment and management of chronic liver failure involves recognizing chronic liver disease, establishing the cause, and treating it. Cirrhosis is typically marked by a prolonged asymptomatic period known as compensated disease. Decompensated disease occurs when the liver is so severely damaged it no longer has the capacity to carry out its normal functions and the metabolic functions of the body are affected. The ultimate goal is to prevent complications, but because cirrhosis is most often progressive and irreversible despite the most appropriate care, many of the patients will still develop complications or decompensated liver disease. This chapter focuses on decompensated disease and how the health-care team should monitor closely for and prevent and treat complications.

Laboratory Studies

The nurse should review the following laboratory studies for an indication of decompensated disease:

- **Liver enzymes**—In particular, aminotransferases (alanine [ALT] and aspartate [AST]) are elevated as the damaged liver releases enzymes
 - **ALT**—Found predominantly in the liver; sensitive and specific for hepatocellular disease.
 - **AST**—Found in the heart, liver, and skeletal muscle. A lesser concentration is found in the kidney and pancreas. Disorders that affect hepatocytes will increase this enzyme.
 - **Total bilirubin**—Reflects the balance between bilirubin production and hepatic clearance. Normally it is metabolized in the liver and excreted in the urine. Total bilirubin is the sum of conjugated (direct) bilirubin and unconjugated (indirect) bilirubin. Elevations cause jaundice. Levels may be normal in well-compensated disease. They rise as liver cell and bile duct damage progress and the bilirubin can no longer be processed. Elevated values reflect a poorer prognosis.
- **Serum albumin**—Albumin is synthesized in the liver and is a measure of hepatocyte function. Reflecting the synthetic function of the liver, the level will fall as the severity of liver disease progresses. It is also the main

determinant of plasma oncotic pressure and is decreased in cirrhosis and ascites.

- **Prothrombin time (PT)/INR**—Reflects the liver's ability to synthesize blood-clotting factors. A prolonged value will be evident when an estimated 80% of the hepatic synthetic function is impaired. It is a prognostic indicator for declining liver function.
- **Complete blood count**—The patient with cirrhosis typically has anemia from a variety of sources, including acute and chronic blood loss, impaired red blood cell formation, and excess destruction.
- **Thrombocytopenia**—Common in patients with portal hypertension and splenomegaly.

Additional Tests

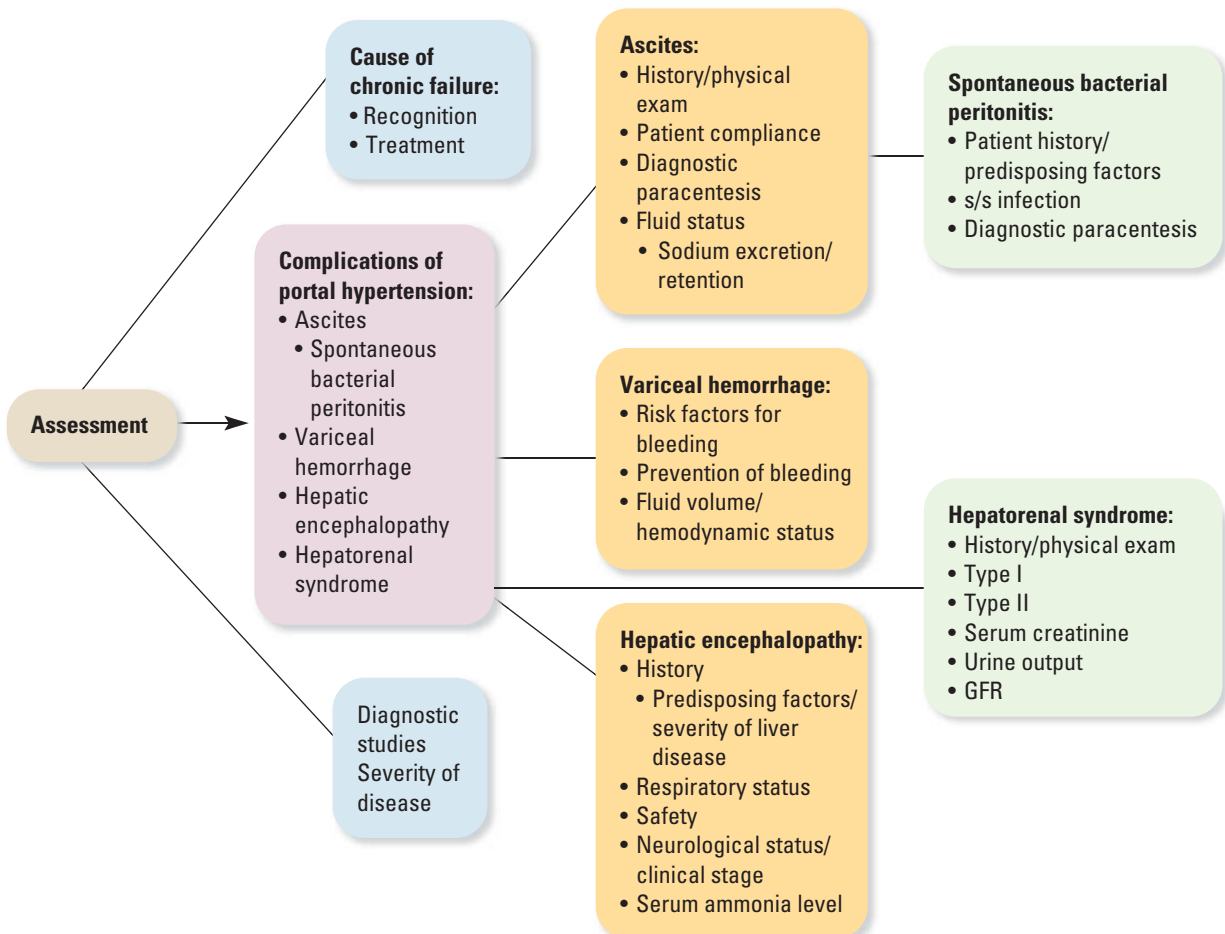
Additional tests to be reviewed may include abdominal ultrasound and liver biopsy. An abdominal ultrasound shows the size and texture of the liver as blood flows through hepatic and portal veins. Nodularity, irregularity, and atrophy are typical findings in cirrhosis (Figure 12-3). It can also identify ascites that is less than 100 mL. A liver biopsy is the gold standard to evaluate liver disease and diagnose

cirrhosis. It can assist in determining the cause, extent of damage, treatment possibilities, and long-term prognosis.

Complications of Portal Hypertension: Assessment and Management: Ascites

Ascites is the accumulation of a large amount of protein-rich fluid in the peritoneal cavity. Ascites is a marker for severe progression of liver disease. It is the most common complication of cirrhosis and has a 2-year survival rate of 50%. Understanding the complex pathogenesis of ascites is critical to understanding management. The following principles and mechanisms are involved:

- Changes in capillary permeability and the hydrostatic and oncotic pressure gradients due to hypoalbuminemia and low oncotic pressure increase the leakage of plasma from the lymph.
- The release of nitric oxide and other mediators results in splanchnic arterial vasodilation.
- Vasodilation and hyperdynamic circulation cause a marked decrease in systemic vascular resistance (SVR),



Visual Map 12-4 Chronic Liver Failure Assessment

mean arterial pressure (MAP), and an increase in heart rate and cardiac output.

- Vasodilation activates compensatory mechanisms (such as renin-angiotensin-aldosterone, sympathetic nervous system, and ADH) to restore perfusion pressure and results in sodium and water retention.
- Water retention is related to the increased secretion of ADH. Being unable to excrete water promotes dilutional hyponatremia and hypo-osmolality. The degree of hyponatremia parallels the degree of liver disease.
- Renal blood flow is reduced because of the vasoconstrictors. Initially, protective mechanisms maintain perfusion through renal vasodilation but these mechanisms eventually become overpowered. The result is progressive hypoperfusion of the kidneys and a gradual decrease in the glomerular filtration rate.

Assessment: Fluid Status

Ascites is usually one of the early indicators of decompensated disease. Diagnostic assessment goals include establishing the presence of ascites, determining the severity, determining the cause and detecting the presence of complications (spontaneous bacterial peritonitis and renal injury). The history (chronic liver disease) and physical appearance (large, distended abdomen) are often indicative of the presence of ascites (♦ Figure 12-4).

The nurse questions the patient related to:

- Increased abdominal girth (pants getting tighter)
- Unexplained weight gain
- Alcohol consumption—alcohol-induced injury is the most reversible with abstinence
- Daily dietary pattern and knowledge related to dietary restrictions

The nurse assesses:

- For shifting dullness by abdominal percussion (Approximately 1500 mL of fluid must be present before flank dullness is detected, and obese patients often require an ultrasound to detect ascites.)
- For the presence of peripheral edema (often pitting in the legs and feet)
- Daily weight (fluid loss and weight gain are directly related to sodium balance)
- Abdominal girths (typically inaccurate evaluation of ascites)
- Difficulty breathing (fluid accumulation and possibly displaced intestines may increase pressure on the diaphragm and decrease lung expansion)
- Lung sounds (decreased especially in the bases; pleural effusions may develop and threaten respiratory function)
- Abdominal discomfort/pain using a standardized pain assessment tool
- Skin breakdown or potential risk using a standardized assessment tool (e.g., Braden scale)
- Orthostatic vital signs
- Intake and output

The nurse anticipates the presence of other manifestations of chronic liver disease including:

- Nausea, loss of appetite, malnutrition
- Fatigue
- Spider angiomas—dilated blood vessels with a red center and spiderlike branches (number and size often correlated with the severity of the disease)
- Palmar erythema—red area on the palms that blanches
- Gynecomastia—benign enlargement of male breasts secondary to endocrine changes related to decreased testosterone in males

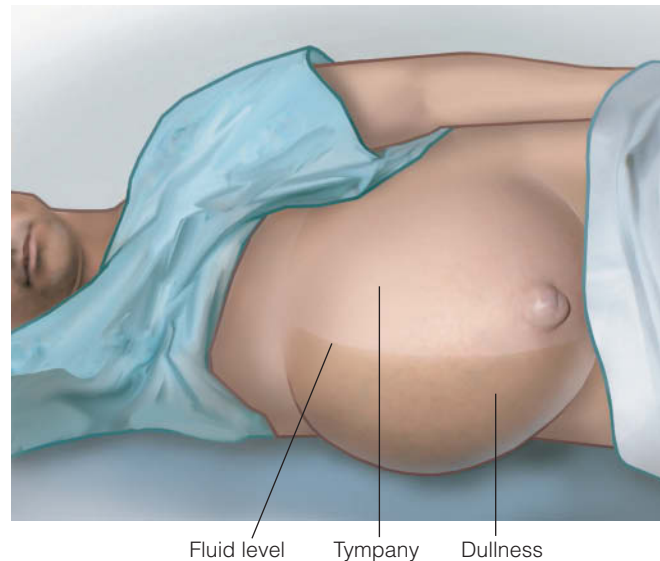
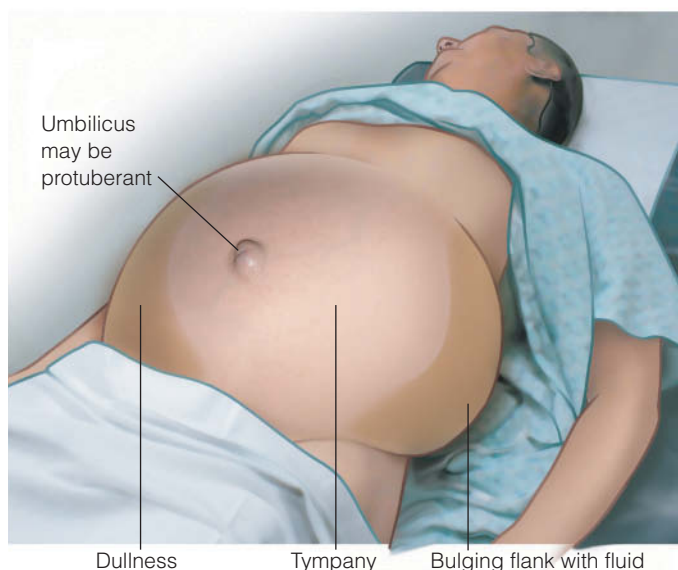


Figure 12-4 Ascites.

- Caput medusae—dilated superficial abdominal blood vessels
- Splenomegaly—enlarged spleen related to the backup of blood from the portal vein
- Hepatomegaly—size may be decreased in more advanced disease
- Jaundice—results from increased serum bilirubin; usually not apparent until the bilirubin is greater than 2 to 3 mg/dL; often accompanied by dark-colored urine
- Fetor hepaticus (sweet musty breath odor)
- Asterixis (liver flap, flapping tremor)
- Bleeding tendencies, bruising

Additionally, the nurse should review the patient's CBC, clotting studies, and liver functions tests for indications related to the severity of the liver disease. Of particular importance for patients with ascites is:

- Serum electrolytes—Electrolyte abnormalities are common in chronic liver disease with ascites.
 - Sodium is monitored because diuretic therapy is the main treatment modality for ascites. The degree of sodium retention and hyponatremia are prognostic predictors.
 - Potassium is monitored because of diuretic therapy.
- Blood urea nitrogen/creatinine are evaluated to assess hydration status and renal function.
- 24-hour urine for sodium excretion: The sodium content in the urine reflects the balance between dietary sodium and renal excretion of sodium. It is helpful in ascites when weight loss is less than desired. The results can be compared to the patients' dietary sodium restriction and weight loss to determine if patients are compliant with their dietary restrictions.
- Random urine sodium/potassium: A concentration ratio greater than 1 predicts that the patient should be losing weight if a sodium-restricted diet is followed. It is more convenient than a 24-hour urine.
- Abdominal ultrasound can evaluate and confirm ascites in morbidly obese patients, and may be used to locate an appropriate paracentesis site.
- Abdominal paracentesis may be diagnostic or therapeutic. It can confirm the diagnosis or the presence of infection. Fluid relative to portal hypertension can be differentiated from other causes. A therapeutic paracentesis removes larger volumes of fluid in refractory ascites (see "Building Technology Skills").
- Ascitic fluid analysis: Initial screening tests for uncomplicated ascites include cell count with differential, total protein, and albumin concentration. Serial outpatient therapeutic paracentesis should be tested only for cell count and differential. If the polymorphonuclear leukocyte count (PMN) is greater than 250 cells/mm³, a specimen for culture should be sent.

COLLABORATIVE CARE

The nurse works collaboratively with the healthcare team to promote mobilization of the ascitic fluid. The goal is that the level of sodium excreted is more than the sodium intake, causing a decrease in ascites and peripheral edema because fluid passively follows sodium. Proper management improves quality of life and decreases risk of complications.

Alcohol Abstinence

If there is an alcohol component to the liver injury/disease, it is essential that the patient abstain from all alcohol. The nurse should screen all patients for alcohol use disorders and the risk for AWS. The nurse should provide education to the patient and family related to the serious implications of continued alcohol use. If the patient is unable to abstain from alcohol consumption, members of the healthcare team are consulted to evaluate the patient for a treatment program that would best benefit the needs of the individual and family. The 2012 American Association for the Study of Liver Diseases (AASLD) guidelines suggest the administration of baclofen to reduce alcohol craving and consumption (Runyon, 2013).

Sodium Restriction

In patients with portal hypertension, weight changes and fluid loss are directly related to sodium balance. A daily dietary sodium restriction of 88 mmol/day (2000 mg) is often attempted first. The goal of therapy is to increase the patient's urinary sodium excretion to greater than 78 mmol/L. A patient on an 88 mmol/L restriction of sodium who is excreting 78 mmol/L should be losing weight. If this is not happening, it is likely that the patient has not been compliant with their diet. In fact, only 10% to 15% of patients can attain this excretion goal and be managed by dietary sodium restriction alone (Runyon, 2013). Most patients prefer to have a more generous sodium intake since a diet with less than 2000 mg of sodium a day is usually perceived as tasteless. These patients are prescribed a diuretic medication to assist with sodium excretion.

Essential for Patient-Centered Care

Since the consequences of noncompliance can be severe, it is imperative that the nurse ensure the patient and family understand the dietary sodium restriction.

Diuretic Therapy

Previously, an oral dose of spironolactone (Aldactone) 100 mg daily was the indicated diuretic therapy. Spironolactone blocks the action of aldosterone and slowly promotes sodium loss and potassium retention. However, now, because

patients may develop hyperkalemia or gynecomastia and have only a mild diuresis, only those patients with minimal fluid overload are managed with spironolactone alone.

The preferred dosing regimen now is a single oral dose of furosemide (Lasix) 40 mg daily, a diuretic that promotes potassium loss as well as fluid loss, used in combination with spironolactone to maintain normal potassium and increase fluid loss. Dual drug usage accomplishes rapid diuresis and maintains normokalemia. If diuresis, as evidenced by weight loss, urine output, and decreased abdominal girth, is insufficient, the doses of the two medications are typically increased simultaneously to help maintain normokalemia. Maximum doses are 160 mg/day for furosemide (Lasix) and 400 mg/day for spironolactone. Dietary sodium restriction and dual diuretic therapy has been successful in achieving acceptable fluid reduction in approximately 90% of patients (Runyon, 2013).

Because the long half-life of spironolactone is typically prolonged to 24 hours in patients with cirrhosis, the nurse should be aware that it takes several days for the onset of action to be apparent. It may take up to 2 weeks to a month for a steady state to be achieved. The physician will likely titrate the doses every 3 to 5 days to determine the optimal dose for the patient. If the patient has concurrent renal disease, patient tolerance of spironolactone may be decreased secondary to hyperkalemia. If the patient is managed with dual therapy, furosemide (Lasix) can be held temporally if hypokalemia should occur.

The nurse expects that when ascites is associated with peripheral edema there is no limit to the daily weight loss. In the absence of peripheral edema, the nurse should expect no more than a 0.3 to 0.5 kg/day weight loss. If the desired weight loss is not accomplished, a measurement of the urine sodium excretion may be ordered.

Essential for Evidence-Based Practice

The nurse monitors the patient's serum potassium, daily weight, and possibly the urine sodium excretion to determine the effectiveness of diuretic therapy for ascites.

Failure of Diuretic Therapy

The nurse recognizes that diuretic therapy has failed if the patient has minimal to no weight loss with urine sodium excretion less than 78 mmol/day and/or the occurrence of clinically important complications, such as encephalopathy, hyperkalemia, elevated creatinine, or hyponatremia. The nurse anticipates that diuretic(s) may be discontinued if the following occur:

- Encephalopathy
- Serum sodium less than 120 mmol/L regardless of fluid restriction compliance

- Hyperkalemia (potassium greater than 5.3 mEq/L)
- Metabolic acidosis
- Increased creatinine greater than 2.0 mg/dL

IV administration of a diuretic in patients with cirrhosis and ascites should be avoided because it can cause an acute decrease in glomerular filtration rate.

Fluid Restriction

Because there are no data supporting a specific threshold for initiating a fluid restriction, practice guidelines advocate a sodium less than 120–125 mmol/L, as a reasonable threshold (Runyon, 2013). As previously noted, unless the decline in serum sodium is very rapid, cirrhotic patients do not usually develop symptoms unless sodium is less than 110 mmol/L. Additionally, fluid restrictions tend to alienate patients from their healthcare team and cause hyponatremia (sodium greater than 145 mEq/L).

Interventions for the Management of Tense Ascites

A large volume paracentesis may be performed on patients presenting with tense ascites and clinically significant symptoms such as shortness of breath, difficulty breathing, and marked decrease in mobility. This does not rectify the underlying problem, and it is not recommended as first-line therapy.

In most patients, 4 to 6 liters can be removed without causing hemodynamic problems. Larger volumes can be safely removed with the administration of albumin to prevent or decrease intravascular hypovolemia and promote renal perfusion. However, the benefit of the practice is questioned and not all sources advocate this practice. Albumin is generally administered 8 to 10 grams per liter removed; usually 50 to 75 grams of a 20% albumin is administered per session. Diuretic therapy and dietary sodium restriction should be initiated and/or adjusted. In patients who have not sought medical attention until their breathing was compromised, large-volume paracentesis can be urgent and greater than 20 liters can be removed safely (Runyon, 2013).

Interventions for Refractory Ascites

Refractory ascites occurs when fluid mobilization is resistant to sodium restriction and maximal diuretic therapy or if the patient cannot tolerate diuretic therapy. For patients who have truly failed diuretic therapy, it is managed with routine large-volume paracentesis (Runyon, 2009). Because removing the fluid does not alter the underlying pathology, the fluid quickly reorganizes and the patient usually requires a large-volume paracentesis every 2 weeks.

Hypotension

Independently, arterial blood pressure predicts survival in patients with cirrhosis. Patients with a mean arterial pressure greater than 82 mmHg have a 1-year survival of 70% whereas a mean arterial pressure less than or equal to 82 mmHg indicates a 40% chance. Because of the mechanisms previously described, angiotensin converting enzyme inhibitors and angiotensin blockers are cautiously considered in patients with cirrhosis and ascites and avoided in patients with refractory ascites. Additionally, the benefits and the risks of beta-blockers must be carefully evaluated in each patient because systemic hypotension complicates their use. Oral midodrine (an alpha agonist) can be considered in patients with refractory ascites. It has been shown to improve clinical outcomes and survival by increasing urine volume, urine sodium and mean arterial pressure. Typical dose is 7.5 mg three times a day. The addition of midodrine may allow diuretics to be restarted in patients with poor renal perfusion (Runyon, 2013).

Building Technology Skills

Paracentesis

Paracentesis is the removal of fluid from the peritoneal space using a large-bore needle or catheter system. The fluid can be removed by syringe, vacuum bottles, or bags depending on the amount of fluid being removed. A peristaltic pump may be used for therapeutic paracentesis to expedite the fluid removal. The procedure is performed at the bedside as shown in ♦ Figure 12-5.

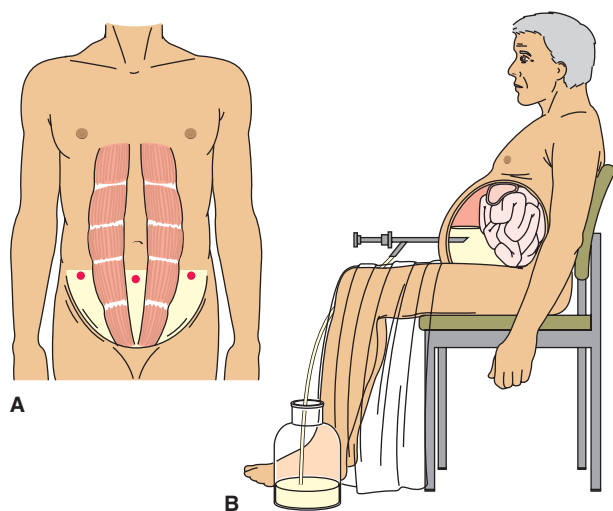


Figure 12-5 Sites and position for paracentesis. A, Potential sites of needle or trocar insertion to avoid abdominal organ damage. B, The patient sits comfortably; in this position, the intestines float back and away from the insertion site.

The position of the patient will depend on practitioner preference, acuity of the patient, volume to be removed, and thickness of the abdominal wall. The patient may be positioned sitting on the side of the bed, but typically the patient is placed in a semi-Fowler's position. Mild ascites may require a left lateral decubitus position. The midline is the preferred site but tends to have a thicker abdominal wall. Typically, the lower abdominal quadrants are used with the left lower quadrant being preferred. The right lower quadrant is more likely to have scars and an increased chance that the cecum is distended. Surgical scar areas are avoided because of the likelihood that the needle may enter the bowel. Patients with obesity and/or multiple scars may require ultrasound guidance.

The procedure is performed utilizing sterile technique and a topical anesthetic. Commercial paracentesis kits are available with the necessary equipment for the insertion of the needle/catheter. Once the needle/catheter is inserted, specimens are obtained and the needle/catheter will be stabilized if more fluid is to be removed.

Purpose

The purpose of a paracentesis can be diagnostic or therapeutic. Diagnostic procedures withdraw the appropriate amount of fluid for analysis. Therapeutic procedures commonly withdraw large volumes of fluid to relieve pressure to promote patient comfort, increase mobility, decrease pressure on the bladder, and improve respiratory status.

Indications and Expected Outcomes

Indications for a diagnostic paracentesis and fluid analysis are to:

- Confirm the diagnosis of ascites.
- Diagnose the cause of ascites (Approximately 15% of the patients may have a cause other than liver disease and some patients have a dual cause).
- Diagnose infection (Patients with clinical signs or symptoms of deterioration and or laboratory abnormalities suggesting infection).
- It may be performed in patients without ascites to evaluate for bleeding following trauma or a perforated viscus.

Indications for a therapeutic paracentesis are:

- Initial treatment of tense ascites for symptom relief
- Refractory ascites

The results of a diagnostic paracentesis will determine the patient's overall management. By confirming the diagnosis and establishing the cause, treatment can be specific to the cause. Identification and treatment of infection are critical to survival. After a therapeutic paracentesis that

removes larger volumes of fluid, the patient will be temporarily more comfortable, have greater mobility, less urinary frequency, and improved respiratory status.

Technological Requirements

This procedure is generally performed by a physician or a specifically trained physician's assistant or advanced practice nurse. Ultrasonography should be available for patients with difficult site access.

Nursing Responsibilities

The critical care nurse will have pre-, intra-, and post-procedure responsibilities. The critical care patient requiring a paracentesis will require more intense monitoring depending on the acuity, overall hemodynamic status, and volume to be removed.

Pre-procedure responsibilities:

- Verifies informed consent
- Provides patient/family teaching
- Reviews the patient's coagulation studies (abnormal studies are common but are not typically a contraindication)
- Reviews allergy history (especially topical anesthetics, novocaine, lidocaine)
- If no indwelling catheter, ensures that the patient voids to avoid puncturing a distended bladder
- Measures baseline vital signs, weight
- Ensures proper positioning and appropriate draping

Intraprocedure responsibilities:

- Universal protocol: participates in a "time out" to verify patient identification, procedure, and site marking immediately before the procedure
- Reassures the patient
- Maintains a sterile field and assists the practitioner with obtaining specimens, repositioning, fluid withdrawal, site dressing
- Monitors hemodynamic status for evidence of hypovolemia (drainage can be slowed or stopped for hemodynamic instability)
- Infuses albumin as ordered

Post-procedure responsibilities:

- Monitors hemodynamic status
- Assesses abdominal pain
- Maintains accurate intake and output (amount of fluid removed, colloid/crystalloid infusion)
- Assesses characteristics of fluid withdrawn (color, viscosity, odor)

- Processes specimens per facility protocols
- Monitors dressing for persistent leakage
- Repositions the patient for comfort
- Monitors for signs and symptoms of the following complications:
 - Hemorrhage
 - Perforation of bowel
 - Hypovolemia/shock
 - Infection

Ascites unresponsive to medical therapy is associated with a mortality rate of 21% within 6 months, and mortality rates are higher for patients with alcoholic liver disease. A transjugular intrahepatic portosystemic shunt (TIPS) may be useful in preventing recurrent ascites in appropriately selected patients (Runyon, 2013). However, if a liver transplant is the appropriate option for a patient with unresponsive ascites, referral to a transplant center should occur as soon as possible because of the high mortality rate.

Surgical Portosystemic Shunts

Placement of a transjugular intrahepatic portosystemic shunt (TIPS) can significantly decrease portal pressure and ascites. Surgical shunt placement has a high morbidity and mortality rate, and thus has limited use in patients with ascites. One such shunt, the peritoneovenous (Le Veen) shunt, which drains ascitic fluid into the vascular system, is limited to unusual circumstances since it has an increased incidence of complications coupled with no demonstrated survival advantage. It may be considered in the rare patient who is not a candidate for paracentesis, TIPS, or transplant.

Spontaneous Bacterial Peritonitis

Spontaneous bacterial peritonitis (SBP) is an infection in the ascitic fluid without indication of another intra-abdominal source such as a perforated viscus. It is caused by bacterial translocation resulting from intestinal bacterial overgrowth, increased intestinal permeability, altered intestinal motility and impaired immune mechanisms. SBP is a frequent occurrence in end-stage liver disease. Without early recognition and treatment, mortality is high.

The nurse should review the patient's history for the following predisposing factors:

- Elevated serum bilirubin level (above 2.5 mg/dl)
- Ascitic fluid total protein concentration less than 1g/dl
- Use of proton pump inhibitors
- Abnormal PT/INR
- Child-Pugh classification B or C
- GI hemorrhage
- Urinary tract infection
- Previous history of SBP

Because it is very common for patients with ascites to have SBP, the nurse assesses all patients with ascites for signs and symptoms of SBP. Although the majority of the patients with SBP have some manifestations, the signs and symptoms are often very subtle or patients may be asymptomatic with only minor abnormalities in their laboratory studies. The nurse assesses for abdominal pain and tenderness, fever, mental status change (an infection can exacerbate HE/PSE), and the appearance of the ascitic fluid.

The nurse would review the following studies for evidence of infection:

- **Complete blood count**—Elevated white blood cells (WBCs).
- **Ascitic fluid PMN leukocyte count**—Greater than or equal to 250 cells/mm³. It is rapidly available and it is often sent with culture on hold.
- **Ascitic fluid bacterial culture**—If PMN is elevated, the culture is processed. Typically, gram-negative organisms are isolated.

COLLABORATIVE CARE

The nurse works collaboratively with the healthcare team to recognize signs and symptoms of SBP, confirm the diagnosis, treat the patient, and prevent future episodes of SBP. The goal is to recognize, successfully treat, and prevent future episodes. Survivors of SBP are evaluated for transplant because they have a poor long-term prognosis.

- **Diagnostic Paracentesis:** A diagnostic paracentesis should be done on all patients with ascites admitted to the hospital regardless of the admission diagnosis. An ascitic fluid analysis should include a cell count with differential, total protein, and albumin concentration and a culture and sensitivity. The diagnosis is confirmed by a positive ascitic fluid bacterial culture and an elevated ascitic fluid absolute polymorphonuclear leukocyte count. A clinical diagnosis of SBP without a paracentesis and fluid analysis is not recommended.
- **Empiric Antibiotic Treatment:** Patients with signs and symptoms of SBP, a strong suspicion of SBP, or a PMN count greater than or equal to 250 cells/mm³ are treated aggressively with empiric therapy. Broad-spectrum antibiotics are initially ordered but a narrow-spectrum antibiotic can usually be substituted once sensitivity results are available. Albumin (25%) may be given intravenously at 1.5 grams/kg on day 1 and 1 gram/kg on day 3 to reduce the risk of renal failure and improve survival (Runyon, 2013). Once a patient has had an episode of SBP, the patient should receive long-term antibiotic prophylaxis.

The nurse needs to review allergy history and administer the first dose of antibiotic immediately. A response to

the antibiotic treatment should be evident by the reduction of signs and symptoms, fever, pain, and tenderness within 48 hours. In addition, laboratory studies indicative of infection will return to normal. The nurse also monitors for hypersensitivity reactions and renal function studies. A repeat paracentesis and fluid analysis is only recommended if there is not a typical response to treatment within 48 hours.

- **Proton Pump Inhibitor (PPI) Therapy:** Careful evaluation of the absolute indication for PPI therapy needs to be determined. Because gastric acid is a defense mechanism against ingested microorganisms, literature has associated PPI use in cirrhotic patients with increased incidence of SBP. In a large meta-analysis by Deshpande et al. (2013) cirrhotic patients receiving a PPI had a three-time risk of developing SBP compared to those not receiving a PPI.

Complications of Portal Hypertension: Variceal Bleeding

Variceal bleeding is a life-threatening emergency and one of the most significant complications resulting from portal hypertension and cirrhosis. In this setting, the mortality rate is approximately 15% (Garcia-Tsao & Bosch, 2015). Gastroesophageal varices are present in approximately 50% of the patients with cirrhosis, and their presence correlates with the severity of liver disease. Each episode of active bleeding has a 33% chance of mortality, and survivors have a 60% chance of rebleeding in the first year (Garcia-Tsao & Bosch, 2015). More than 50% of the patients awaiting liver transplant will have a variceal bleed prior to transplant (Gordon, 2007).

Variceal bleeding results from collateral circulation that develops to bypass abnormally high pressure in the portal system. The increased pressure in the portal system causes a backup of blood throughout the digestive vasculature, spleen, and mesentery. Additionally, the hyperdynamic circulation resulting from vasodilation increases portal flow. Blood seeks alternate routes and the path of least resistance, which results in the formation of collateral blood vessels in the GI system. The most common locations for these collateral vessels are the esophagus, stomach, and rectum. These vessels, which are intended for low volume and low-pressure flow, become distended, tortuous, and fragile as shown in

♦ Figure 12-6.

The high pressure frequently causes the esophageal and/or gastric varices to rupture and bleed. The most common site for a variceal bleed is the submucosa of the distal end of the esophagus. Gastric varices are less common yet more problematic to manage. It is imperative that the source of bleeding be confirmed. Esophageal variceal

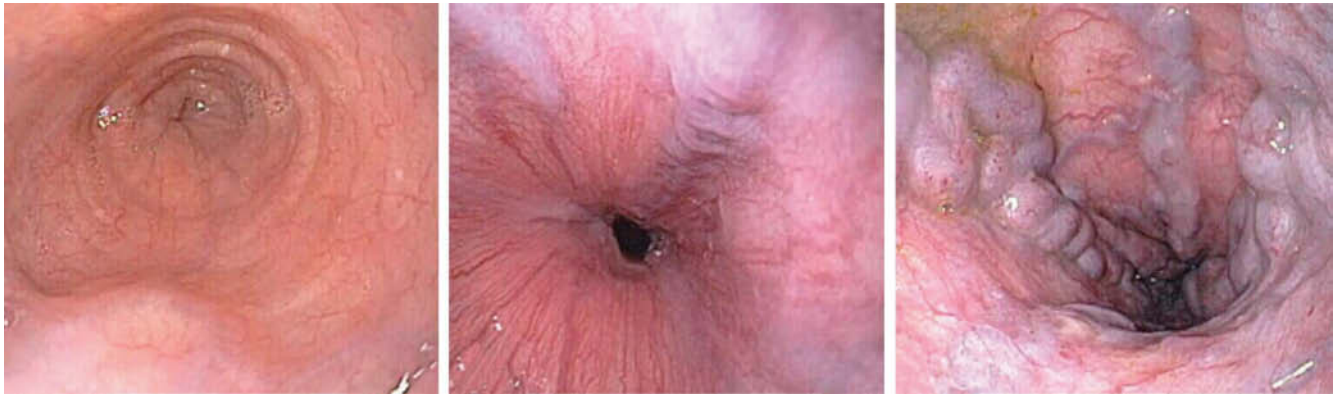


Figure 12-6 Varices increase in diameter progressively.

(Lahey Clinic)

bleeding is usually massive related to the high pressure and high volume. Risk factors for bleeding include:

- Decreased clotting factor synthesis, increased platelet destruction by an enlarged spleen, and impaired vitamin K absorption and storage
- The severity of liver disease; the higher the modified Child-Pugh score, the greater the risk of bleeding
- Previous bleed
- Increasing varix size and diameter—the most important predictor of hemorrhage is the size of the varix
- Continued alcohol consumption

COLLABORATIVE CARE

A patient presenting with a suspected variceal bleed is potentially critically ill and may suffer many consequences as a result of the bleed. Variceal bleeding can be massive, and management can be complicated by the degree of liver dysfunction and the presence or absence of coagulopathy, ascites, encephalopathy, and renal involvement. Other complications include aspiration pneumonia, SBP, and sepsis.

Initial assessment and management is the same as for nonvariceal bleeds. The nurse focuses on hemodynamic status, which will direct resuscitation and stabilization efforts. A concurrent history and physical exam needs to focus on the source and severity of the bleed. A team of specialists, including a gastroenterologist/endoscopist, hepatologist, interventional radiologist, and surgeon, must be available for consultation. Differential diagnosis (see nonvariceal bleeding) must be considered, because peptic ulcer disease is more frequent in cirrhotic patients.

The goal is to maintain hemodynamic stability, identify the source, implement interventions to stop the bleeding, prevent recurrent bleeding, and prevent and treat complications. In the event that varices are discovered prior to bleeding, the goal is to evaluate them and the

degree of liver disease and implement prophylaxis to prevent an initial bleeding event. Additional interventions specific to the management of a patient with a variceal bleed are described next.

Assessment of Fluid Volume

Assessment of fluid volume status in the patient with liver disease is discussed earlier in this chapter. The nurse works with the healthcare team to identify and treat hypovolemia. The goal of fluid resuscitation is to restore intravascular volume, maintain the oxygen-carrying capacity of the blood, maintain cardiac output, restore red blood cells, and prevent complications of red blood cell loss such as pulmonary, neurological, cardiac, or renal injury.

Interventions for Maximizing the Oxygen-Carrying Capacity of the Blood

The nurse protects the patient's airway and supports breathing with supplemental oxygen to maximize the oxygen-carrying capacity of the blood. The oxygen-carrying capacity is decreased by an increased cardiac workload and decreased cardiac output. The nurse will continuously evaluate pulse oximetry and report values less than 92%. With esophageal variceal bleeding, special attention must be directed toward maintaining airway integrity. There is an increased risk of aspiration because bleeding is often brisk with hematemesis, and neurological status may be impaired related to the potential for HE/PSE. Respiratory status is continually assessed, and best practice is to electively intubate and ventilate, avoiding an emergent situation.

Interventions for Restoration of Normovolemia

Interventions for restoration of normovolemia parallel that which is outlined in the discussion of GI bleeding. Half of the patients with a variceal bleed spontaneously stop

bleeding, resulting from hypovolemia triggering splenic vasoconstriction and a decrease in portal pressure. If the patient is hemodynamically stable, caution is taken to avoid fluid overload, which can trigger a rebound increase in portal pressure, which may contribute to failure to control acute bleeding and the development of early rebleeding regardless of the initial treatment. According to practice guidelines, the goal is to keep the hemoglobin 7-8 g/dL (American Society for Gastrointestinal Endoscopy Standards of Practice Committee, 2014). A restrictive transfusion strategy (hemoglobin less than 7g/dL) is preferred over a more liberal transfusion strategy (hemoglobin less than 9 g/dL) due to the increase in portal pressure and mortality with the liberal transfusion strategy. Factors such as age, cardiovascular disorders, ongoing hemorrhage and hemodynamic status must be considered for each patient (Garcia-Tsao & Bosch, 2015).

Essential for Patient-Centered Care

Because coagulopathy is commonly present in patients with cirrhosis, patients frequently require aggressive treatment with platelets, fresh frozen plasma (FFP), and vitamin K (Mephyton).

Pharmacological Therapy

Somatostatin is a hormone that has an inhibitory effect on the secretion of several vasodilator hormones (glucagon and vasoactive intestine peptide) that are responsible for splanchnic vasodilation stimulated by blood in the intestines. This, in turn, causes splanchnic vasoconstriction and decreases portal pressure. Octreotide (Sandostatin) is a longer-acting (half-life 80 to 120 minutes) synthetic somatostatin analogue with similar properties. Octreotide (Sandostatin) has an excellent safety profile, even for cardiac patients, and its administration is not limited to ICUs. It may be started when a variceal bleed is highly suspected.

Octreotide is effective in temporarily stopping the bleeding in approximately 80% of the patients. The nurse can expect to administer octreotide 50 micrograms IV bolus over 3 minutes, followed by 50 mcg/hour continuous infusion for 3 to 5 days. Higher doses of octreotide may increase systemic venous pressure and do not increase the portal hypotensive effects. To evaluate response to the octreotide infusion, the nurse would continue to monitor the patient's hemodynamic status and expect to see the patient's vital signs return to normal, urine output increase, and a decrease in overt bleeding: hematemesis, melena, and hematochezia. Serial hematocrit and hemoglobin levels should be evaluated.

Vasopressin is a potent vasoconstrictor that can decrease portal pressure by directly constricting the mesenteric arterioles. This drug also causes significant

systemic vasoconstriction, resulting in myocardial, cerebral, bowel, and limb ischemia. Octreotide is the preferred vasoactive agent because of its efficacy and safety profile.

Prophylactic Antibiotic Therapy

Variceal bleeding is a common risk factor in the development of bacteremia and SBP. The risk peaks at 48 hours after the onset of the bleed. It is probable that a shock state increases the translocation of bacteria from the intestines to extra-intestinal sites. The presence of infection in this population also increases mortality, and studies have shown that the administration of antibiotics may significantly reduce mortality and recurrent bleeding (Sanyal, 2015b). The benefit of antibiotics is likely greater in patients with advanced liver disease. Standard practice is to treat all patients with an active bleed with short-term prophylactic antibiotics for 7 days. Oral quinolones or intravenous ceftriaxone (Rocephin) are typically chosen.

Endoscopic Therapy

Endoscopic therapy, the hallmark of variceal bleeding management, is necessary to identify the source of the bleeding and to provide therapeutic interventions. Ideally the endoscopy is performed as soon as the patient receives fluid resuscitation and is hemodynamically stable (within 12 hours). Urgency is dictated by the severity of the bleed and the clinical setting. Exsanguinating patients may need immediate interventions to stop bleeding. Endoscopic sclerotherapy involves injecting a sclerosing (hardening) solution into the varix to stop the bleeding by causing a thrombosis and obliteration of the vein. The nurse should monitor for complications including chest discomfort, sclerosant-induced esophageal ulcers, strictures, and perforations. Esophageal variceal ligation (EVL) involves suctioning the varix into the scope cylinder and deploying a band around the varix. The band strangulates the varix, causing thrombosis and obliteration. Both are effective therapeutic modalities in stopping the bleeding in the majority of the patients. Esophageal band ligation is considered the primary endoscopic therapy and is shown in ♦ Figure 12-7. It has fewer complications than sclerotherapy. However, in situations where there is severe active bleeding and poor visibility, banding may be more difficult. The use of endoscopic therapy in combination with a vasoactive agent (octreotide) is more successful than either therapy alone. EVL may be performed every 2 to 4 weeks until the varices have been eliminated. Repeated surveillance after eradication is recommended every 6 to 12 months.

Ulcer development after banding is common and a small percentage of patients (2.6% to 7.3%) will experience bleeding from these ulcers (Tierney, Toriz, Mian, & Brown, 2013). To decrease bleeding from both the ulcers

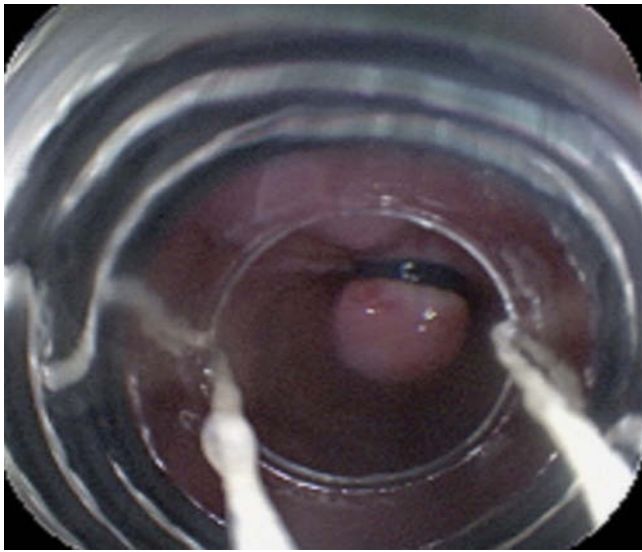


Figure 12-7 Endoscopic banding of esophageal varices.

(D Martin/Lahey Clinic)

and the varix, it is also reasonable to administer a daily proton pump inhibitor to prevent ulcer formation and/or promote healing. Proton pump inhibitors have a high safety profile, are simple to administer, and are very well tolerated.

Transjugular Intrahepatic Portosystemic Shunt (TIPS) is indicated in active variceal bleeding when the patient is refractory to medical therapy.

Building Technology Skills

Transjugular Intrahepatic Portosystemic Shunt (TIPS)

TIPS is a minimally invasive radiological procedure performed with moderate sedation. General anesthesia may be appropriate for hemodynamically unstable patients. It is performed under fluoroscopy to monitor the location of the guidewires and catheters. The jugular vein is

accessed with an IV-like catheter called an introducer sheath. All of the work will take place through this sheath.

A guidewire is introduced and advanced through the heart to the hepatic vein. A catheter with a needle is directed by the guidewire and is passed through the wall of the hepatic vein through the body of the liver into the portal vein. This creates a narrow pathway between the branch of the portal vein and the hepatic vein. A catheter with a deflated balloon is advanced over the guidewire to the tunneled area. Once in position, the balloon is inflated to dilate the area and widen the track between the branch of the portal vein and the hepatic vein.

An expandable metal mesh stent (tube), loaded onto another specialized catheter, is advanced over the guidewire to the tunneled area connecting the portal venous system with the hepatic venous system. The stent is then deployed (left in place).

Purpose

The purpose is to manage complications of portal hypertension. The stent creates a passage between the high-pressure portal vein and the low-pressure hepatic vein. It decompresses the portal venous system and reduces elevated portal pressure to decrease re-bleeding from varices and stop or reduce the formation of ascites. It allows blood to flow from the portal system to the hepatic vein (bypassing the liver) to the vena cava as shown in ♦ Figure 12-8. It does not have any effect on liver function, and will not improve survival (Boyer & Haskal, 2010). Overall, TIPS has gained extensive acceptance as a treatment for severe or refractory complications of portal hypertension.

Indications and Expected Outcomes

The primary indications for TIPS are:

- Acute variceal bleeding uncontrolled by pharmacological and endoscopic therapy

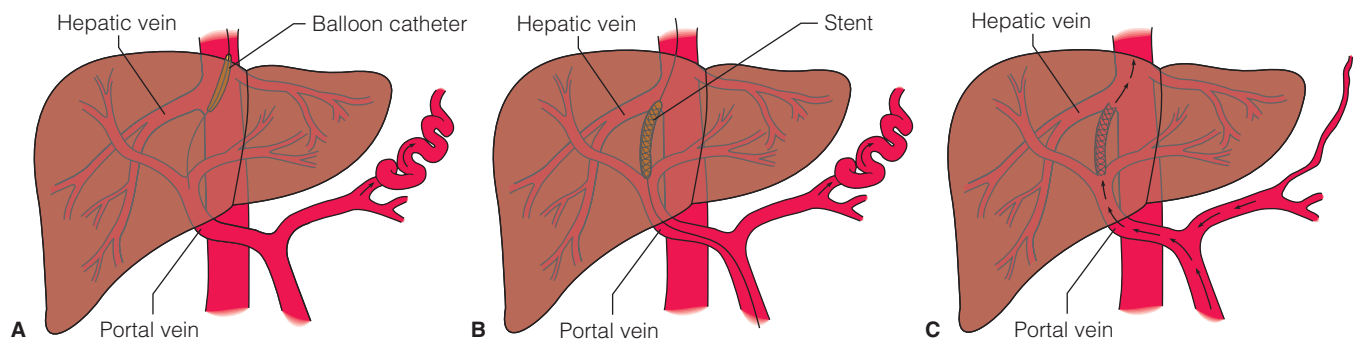


Figure 12-8 Transjugular intrahepatic portosystemic shunt (TIPS). A, Guided by angiography, a balloon catheter inserted via the jugular vein is advanced to the hepatic veins and through the substance of the liver to create a portacaval (portal vein-to-vena cava) channel. B, A metal stent is positioned into the channel and expanded by inflating the balloon. C, The stent remains in place in the hepatic vein.

- Current studies are looking at possible improved outcomes of performing “early” TIPs (within 24-48 hours of admission) on a selected group of patients who are high risk for pharmacological and endoscopic treatment failure (Siramolpiwat, 2014).
- Recurrent variceal bleeding that is refractory or intolerant to secondary prophylaxis treatments
- Refractory ascites that is intolerant to repeated large-volume paracentesis that may require a transplant

Prior to a TIPS procedure, the patient undergoes an extensive evaluation including liver function tests, coagulation studies, kidney function tests, and imaging to access portal venous system patency, and to exclude any liver masses. If the patient has a cardiac history, additional tests may be ordered to rule out heart failure. Absolute contraindications include:

- Severe pulmonary hypertension (mean pressure greater than 45 mmHg)
- Congestive heart failure
- Systemic infection sepsis (uncontrolled)
- Unrelieved biliary obstruction
- Primary prophylaxis for variceal bleed
- Multiple hepatic cysts

A multidisciplinary approach including the transplant team must look at the risks of the procedure in high-risk patients and address the probability of whether the patient will survive long enough after the procedure to receive a liver transplant.

The expected outcome is that portal pressure will be reduced. In the case of variceal bleeds, the hope is to lower the portal pressure, therefore decreasing the pressure in the varices to control the acute bleeding and prevent a recurrent bleed. In refractory ascites, the goals are to decrease production of ascites, eliminate the need for serial paracentesis, and increase the efficacy of other interventions. Because more blood is now bypassing the liver and not being exposed to liver cells that detoxify toxins, more toxins are going directly to the systemic circulation. Hepatic encephalopathy, an anticipated complication of portal hypertension, often appears or worsens after TIPS.

Technological Requirements

Technological requirements include a medical facility that has the radiology equipment, an interventional radiologist, and a support team with expertise in performing such a complex advanced procedure. TIPS avoids the risks of major surgery and, in most cases, that of general anesthesia. However, there are complications associated with the insertion of TIPS:

- Potential reaction to sedation medication/anesthesia

- Cardiac arrhythmias as the catheter is being passed through the heart
- Complications related to creation of the intrahepatic shunt, portal vein manipulation, and stent placement

Nursing Responsibilities

The critical care nurse has both pre- and post-procedural responsibilities.

Pre-procedural responsibilities:

- Maintains NPO status for 6 to 8 hours prior to the procedure
- Monitors baseline vital signs including temperature and oxygen saturation
- Evaluates neurological status, LOC, presence of HE/PSE
- Ensures that informed consent has been obtained
- Provides patient/family education
- Determines allergies, especially to contrast solution/topical anesthetics
- Reviews the patient's platelet count and PT/INR. The recommendation is to have a platelet count greater than 60,000 and an INR less than 1.4; however, this is not absolute. The nurse can anticipate the administration of clotting factors or platelets to patients with significant coagulopathy.
- Inquires about antibiotic administration
- Provides Situational Briefing Assessment Record (SBAR) report to interventional radiology nurse/anesthesiologist on transfer

Post-procedural responsibilities:

- Monitors vital signs including temperature and oxygen saturation and compares them to baseline
- Reports hemodynamic changes, which may indicate bleeding
- Assesses and treats pain (patients often complain of muscle stiffness)
- Monitors the insertion site for bruising
- Monitors for fever or other signs and symptoms of infection
- Monitors for potential complications related to the portosystemic shunting

To detect HE/PSE (the incidence of HE/PSE is 30% to 35% [Sanyal & Bajaj, 2014]), the nurse reviews the patient's history for risk factors, including advanced disease, older age, and prior history of HE/PSE and evaluates the patient's neurological status paying attention to the new onset or worsening of symptoms of HE/PSE. The nurse can anticipate initiating standard therapy to those patients who are symptomatic.

The nurse provides education to patient/family because HE/PSE may not appear until 2 to 3 weeks after the procedure. If HE/PSE appears after 6 weeks, patients should notify the physician because the cause is often GI bleeding.

The nurse assesses for the development of additional complications such as hemolytic anemia (may present in 7 to 10 days after the TIPS procedure), severe hyperbilirubinemia, and vegetative infections.

The nurse monitors for the development of stent occlusion since the likelihood of stent stenosis has been high (especially in the first year). The best indicator of stenosis is the reoccurrence of the problem that required the TIPS (Shah & Kamath, 2016). AASLD guidelines (2010) prefer the use of expanded polytetrafluoroethylene-covered stents is now preferred because of the lower risk of shunt dysfunction (Boyer & Haskal, 2010).

The nurse educates the patient and family about the signs and symptoms of recurring portal hypertension evident by the reoccurrence of the initial problem(s) (GI bleeding, returning ascites) and the importance of scheduled follow-up visits for evaluation of stent patency (usually at 3- to 6-month intervals).

Surgery

Advances in endoscopic therapy and improvements in TIPS have markedly decreased the number of surgical portosystemic shunts surgeries. Consequently, the number of facilities and surgeons with this expertise is limited. This surgery is reserved for patients who fail endoscopic and pharmacological therapy and who are not a candidate for TIPS. In general, patients with preexisting liver disease are at a greater risk for surgical and anesthetic complications that can lead to significant mortality after surgery. The severity of the disease as well as the type of surgery and urgency will all influence the outcome. Identification of the surgical risk is imperative. There are several surgical options available; the distal spleno-renal shunt is usually the operative procedure of choice because it is associated with a low risk of operative morbidity, and a reduced shunt dysfunction rate. In the 2009 guidelines by the AASLD, TIPS and a distal spleno-renal shunt were similarly effective in the prevention of re-bleeding in patients that failed medical therapy. Individual clinical circumstances and available surgical and interventional radiological expertise must be considered in the choice of therapy. The combination of increased efficacy of endoscopy and pharmacological management and TIPS, and the decrease in the number of surgical shunts, has resulted in limited recent comparative data. Liver transplantation is the only surgical treatment that corrects liver failure and portal hypertension.

Balloon Tamponade

Balloon tamponade involves the insertion of a specialized multilumen tube either orally or nasally. The tube may

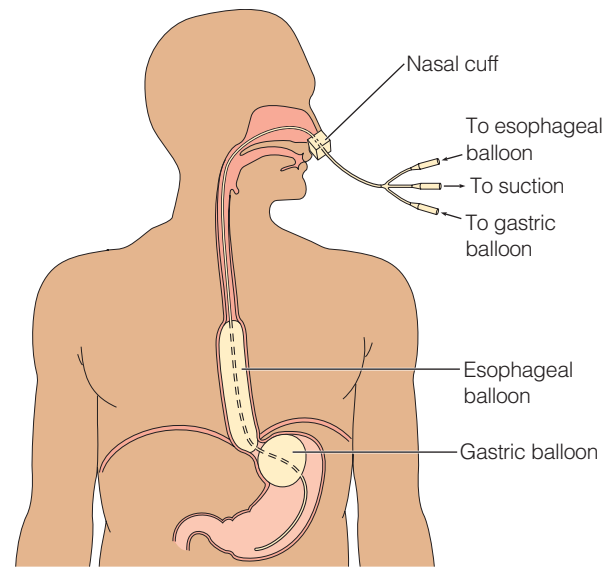


Figure 12-9 Triple-lumen nasogastric tube (Sengstaken-Blakemore) used to control bleeding esophageal varices.

have a gastric balloon or, more commonly, a gastric and esophageal balloon. Once inflated, the balloons exert direct pressure (a tamponade effect) on the gastric esophageal junction and the bleeding areas of the esophagus to reduce blood flow through the varix and control the bleeding, as shown in ♦ Figure 12-9.

With increased use and success of endoscopic therapeutic interventions and TIPS, this procedure is used infrequently. It is a rescue procedure to achieve stabilization when the patient has been unresponsive to medical therapy and serves as a bridge to more definitive treatment. It can be successful in accomplishing short-term hemostasis; however, serious complications can occur and there is a high rate of re-bleeding once the balloon is deflated. The most common tubes are the Sengstaken-Blakemore tube (three lumens, gastric and esophageal balloon), the Minnesota tube (four lumens, gastric and esophageal balloon), and Linton-Nachlas tube (three lumens, gastric balloon only).

The tube is inserted by a physician according to the manufacturer's instructions. Placement is confirmed by chest radiography. The patient is likely to be critically ill with hemodynamic instability requiring resuscitation. Caring for a patient with one of these tubes requires very specific nursing interventions. The nurse will continuously monitor the patient's respiratory and hemodynamic status. It is recommended that the patient be intubated and sedated prior to insertion to secure airway integrity and prevent aspiration. If the patient is not intubated, the biggest concern is the migration of the esophageal balloon over the airway. Because of this potential complication, scissors are taped to the bedside and travel with the patient for emergency removal of the tamponade tube. The tube should not be in place for longer than 24 hours.

Migration is commonly caused by inadvertent deflation of the gastric balloon. Balloon pressures and traction are monitored at regular intervals. There may be an order to routinely deflate the esophageal balloon for a short period (e.g., 5 minutes every 4 to 6 hours) to avoid pressure injury to the mucosa.

Essential for Safety

If a patient requiring esophageal tamponade is not intubated and develops respiratory distress, the nurse takes immediate action by deflating the esophageal balloon, usually by cutting all of the ports, thus ensuring deflation of both the esophageal and gastric balloons.

Esophageal and gastric aspirate are assessed for color and amount. Gastric lavage may be ordered. Evidence that the balloon tamponade has been successful in causing hemostasis would be confirmed by clear gastric and esophageal aspirate, stabilization of vital signs, and an increase in urine output. The patient requires frequent mouth care and skin assessments around the insertion site. The patient may require continued sedation. Chest pain is a frequent complaint that must be thoroughly investigated. Potential complications include:

- Aspiration
- Tissue injury: ulceration/necrosis
- Perforated esophagus
- Airway compromise
- Tracheal rupture/fistula

Though bleeding stops in 90% of the patients, the majority of the patients re-bleed. Once the balloons are deflated, the patients need to be monitored for re-bleeding and scheduled for additional treatment modalities.

Prevention of Initial and Recurrent Bleeding from Esophageal Varices

Assessment and management of esophageal varices involves identifying patients with probable progressive liver disease. Once the diagnosis of progressive liver disease is suspected or confirmed, the main goal is to identify the presence of varices, assess risk of hemorrhage, and to prevent the first variceal bleeding.

Patients with diagnosed cirrhosis should have an esophagogastroduodenoscopy (EGD) to screen for the presence of varices. Based on the presence or absence of varices, the size of the varices, and whether the patient has compensated or decompensated cirrhosis, further EGD surveillance will be scheduled accordingly.

Nonselective beta-blocker therapy is the gold standard for prevention of the first variceal bleed (primary prophylaxis). Patients with cirrhosis may also benefit from statins.

It is thought that they decrease fibrogenesis, improve liver microcirculation and decrease portal pressure, though further evaluation is necessary for efficacy evidence (Garcia-Tsao & Bosch, 2015). Endoscopic variceal ligation therapy can be an acceptable alternative to beta-blockers for primary prophylaxis for patients who are at high risk for bleeding and unable to have medical therapy. Appropriate sized varices can be eradicated. Sessions can be scheduled every two weeks until all varices have been obliterated.

Once the patient has bled, secondary prophylaxis is initiated to eradicate the varices and prevent a re-bleed. The nurse should be aware that the greatest risk for re-bleeding is within 48 to 72 hours after the acute bleed. Because of this high risk, secondary prophylactic treatment is typically started shortly after the first bleeding episode.

Beta-blockers are also indicated for secondary prophylaxis following a bleed. If the patient who has a variceal bleed was not receiving beta-blocker therapy as discussed, it may be instituted after a bleeding episode. If the patient has bled despite the use of beta-blockers, the addition of a long-acting nitrate, specifically isosorbide mononitrate (Imdur), to the beta-blocker regime can be tried. Nitrates cause dilation in the venous bed, decreasing portal venous blood flow, thus portal pressure. After a bleed or when beta-blocker therapy is not getting a therapeutic response, isosorbide mononitrate can be added. Because of severe hypotension and headaches, patients usually do not tolerate this course of therapy. Recent research data has disproved that this combination treatment has any advantage over non-selective beta-blockers alone. The AASLD practice guidelines (Garcia-Tsao, Sanyal, Grace, & Carey, 2007) and the Baveno Consensus Workshop (Garcia-Tsao & Bosch, 2015) recommend the combination of a beta-blocker plus endoscopic ligation as the best option for secondary prophylaxis.

The most recent Baveno 6 Consensus Conference/Workshop in April 2015 focused on varices and variceal hemorrhage in cirrhosis (Garcia-Pagan, 2015). The workshop stressed individualized care for portal hypertension based upon the clinical stages and sub-stages of cirrhosis, and complications influencing the risk of variceal hemorrhage. This workshop typically triggers guideline revision. It is anticipated that the AASLD 2007 guidelines will be revised by this publication.

Complication of Portal Hypertension: Hepatic Encephalopathy (HE)

HE, also known as portosystemic encephalopathy (PSE), is a condition characterized by a wide range of potentially reversible neuropsychiatric manifestations that occurs in patients with advanced liver disease and portal hypertension. It may be a single event or a frequent occurrence. These manifestations result from a disturbance in the CNS because of impaired liver function. The manifestations can be subtle

and intermittent, or progressive with overt confusion and coma. As the encephalopathy progresses, the manifestations become more noticeable. The degree of neurological and intellectual dysfunction of HE is often referred to in clinical stages ranging from I to IV. When coma appears imminent, the Glasgow Coma Scale may be utilized. The diagnosis is through exclusion of other causes of brain dysfunction.

The pathogenesis is complex, is not clearly defined, and is beyond the scope of this chapter. However, ammonia is a significant factor in the pathogenesis of HE because elevated ammonia levels are present in HE, and interventions to decrease ammonia improve HE (Ferenci, 2015).

- Ammonia, produced primarily by the GI tract, enters the circulation via the portal vein to the liver.
- The functioning liver detoxifies ammonia before it can reach the systemic circulation. Through a series of enzymatic reactions, ammonia is converted to glutamine, a nontoxic substance, and urea for secretion by the kidneys.

- In advanced liver disease, detoxification is inefficient, resulting in ammonia entering the systemic circulation and consequently the CNS.
- Ammonia is neurotoxic and can interfere with cerebral function and neurotransmission, resulting in changes in consciousness and behavior.
- Portal systemic shunting from the development of collaterals is also responsible for substances absorbed by the gut, bypassing the liver into the systemic circulation without being detoxified.
- Muscle is also a site for ammonia clearance; many patients with advanced liver disease have severe muscle atrophy that may also contribute to an increase in ammonia.

Precipitating factors for HE include:

- GI hemorrhage (metabolism of blood protein in the gut)
- Azotemia/uremia (retention of blood nitrogenous substances by the kidney)

Commonly Used Medications

Beta Blockers

Action: Beta-blockers decrease cardiac output and block mesenteric arteriole vasodilation, promoting vasoconstriction and a subsequent decrease in portal venous flow. The goal of treatment is to decrease portal and variceal pressure.

Indications: Recently, carvedilol (Coreg) was accepted as an alternative to nadolol and propranolol for management of moderate to large varices that have not bled. It is a potent non-selected beta-blocker with a mild intrinsic anti-alpha-adrenergic action that reduces hepatic vascular tone and resistance. It may increase plasma volume without a decrease in glomerular filtration rate. Patients may require the addition of a diuretic or an increase in dose (Garcia-Tsao & Bosch, 2015).

Nursing Responsibilities: Either propranolol (Inderal) or nadolol (Corgard) are recommended for primary prophylaxis. The dosage should be titrated to reduce the patient's heart rate by 25%. Some practitioners prefer nadolol because it can be administered once a day, it is excreted in the kidneys, and it has a lower risk of CNS side effects.

- Typical starting dose of nadolol is 40 mg daily (40 to 160 mg orally daily).
- Typical starting dose of propranolol (Inderal) is 40 mg two times a day (40 to 80 mg two times a day).
- Typical starting dose of carvedilol is 6.25 mg orally daily. After 3 days, dose can be increased to 12.5 mg orally daily. The maximum dose is 12.5 mg daily unless patient has arterial hypertension. Therapy goal is that the systolic arterial blood pressure should not decrease less than 90 mmHg.

- Response to the beta blockade is determined by monitoring the patient's heart rate. The goal is to achieve a heart rate of 50 to 60 beats/min.
- If patients receive high doses without a decrease in pulse rate, individual noncompliance should be considered. Higher doses cause more side effects.
- Nadolol is excreted by the kidneys; thus, renal function must be considered and monitored. Doses may need to be adjusted according to renal insufficiency.

Side and/or Toxic Effects: Because there are numerous side effects associated with beta-blockers, they may be contraindicated or not tolerated. Often intolerance is related to headaches, and noncompliance is related to sexual dysfunction and impotence. Clinically significant adverse effects for a patient with cirrhosis include:

- Heart failure
- Bronchoconstriction
- Hypotension—if the patient has a significant systemic hypotension to begin with (MAP less than 85 mmHg), a lower dose (nadolol 20 mg daily) can be tried

The nurse must monitor the patients for adverse effects of the medication such as heart rate less than targeted rate, hypotension, orthostatic symptoms, respiratory distress, and headache. In an acute bleed, the nurse must be aware that beta blockers can mask the compensatory tachycardia needed to maintain cardiac output. The nurse also needs to educate the patient related to the importance of the medication, the significance of noncompliance, and stopping the medication abruptly.

- Electrolyte abnormalities (especially hypokalemia, which increases ammonia production by the kidneys, and hypokalemia and alkalosis, which promote cellular uptake of ammonia)
- Constipation (increases ammonia related to bacterial action on the feces)
- Excessive dietary protein
- Dehydration/hypovolemia (vomiting, diarrhea, hemorrhage, diuretics, large-volume paracentesis)
- Hypoxia
- Hypoglycemia
- Blood bypassing the liver and entering the systemic circulation following surgical or radiological shunting procedures
- Infections
- Medications such as benzodiazepines, narcotics, other sedatives

The majority of the patients with chronic HE have a precipitating cause rather than a progression of hepatocyte dysfunction.

Assessment of the Patient with Hepatic Encephalopathy

Critical care monitoring of a patient with HE involves reviewing the patient's history for severity of liver disease, previous episodes of HE, and precipitating factors. Medications should also be evaluated as a causative agent. The nurse should routinely assess:

- Respiratory status: airway maintenance and oxygenation
- Safety related to decreased LOC and confusion: fall and aspiration precautions
- Neurological status: for signs and symptoms of impaired personality/behavior, consciousness, and intellectual and neuromuscular functioning. Manifestations are on a progressive continuum and referenced in four stages from mild impairment to coma. Variations in the literature exist related to the exact manifestations in each stage.
 - **Stage I**—Impaired/shortened attention, lack of awareness, euphoria or depression, inversion of sleep pattern, slight tremor
 - **Stage II**—Lethargy, memory problems, behavioral changes, slurred speech, tremor progresses to asterixis, apraxia, ataxia
 - **Stage III**—Somnolence, marked confusion, disorientation, amnesia, asterixis, abusiveness, violence
 - **Stage IV**—Coma
 - Glasgow Coma Scale

Essential for Patient Safety

It is imperative that the nurse identify rapid deterioration promptly and report changes immediately. Rapid changes could be resulting from an acute event.

The nurse anticipates that an array of routine laboratory and imaging tests may be performed to screen for precipitating factors and differential diagnoses or causes of cerebral dysfunction. Tests may include, but are not limited to, CBC, electrolytes, BUN, creatinine, arterial blood gases, glucose, toxic screen, head CT, and EEG. Of particular interest is the blood ammonia level because an elevated ammonia is associated with HE. Because of other neurotoxic substances, a normal ammonia does not exclude the diagnosis but warrants reevaluation. Increased ammonia levels alone do not add diagnostic, staging or prognostic value (Vilstrup et al., 2014).

COLLABORATIVE CARE

The goal of management is to provide supportive care (airway maintenance, safety [aspiration precautions/fall prevention], adequate nutrition), identify and treat precipitating factors, reduce nitrogenous waste from the gut, and evaluate the need for long-term treatment interventions.

Identification and Correction of Precipitating Factors

All precipitating factors should be aggressively investigated. Determination of specific causes should direct immediate corrective actions. For example, if the patient is hypokalemic, potassium supplementation should be provided. If corrective actions are taken, the nurse anticipates an improvement in neurological status. The time frames for improvement depend on the cause and treatment.

Evidence-Based Interventions for Reduction of Nitrogenous Waste from the Gut

The goal of treatment is to reduce ammonia production and/or increase its removal, thereby lowering elevated ammonia levels. This may occur by a variety of methods. One of the simplest is bowel cleansing. Because nitrogenous wastes are produced primarily in the gut, bowel cleansing can reduce colonic bacteria responsible for synthesizing ammonia and thus decrease the amount of ammonia reaching the systemic circulation.

The AASLD recommends lactulose (Cephulac), a non-absorbable disaccharide, as the first choice for initial treatment of overt HE and for the prevention of recurrent

episodes. In addition to its laxative effect, multiple mechanisms work to decrease the absorption of ammonia from the gastrointestinal tract. Lactulose is also cost effective (Vilstrup et al, 2014).

Oral antibiotics work primarily by modifying the intestinal flora and decreasing the stool pH to facilitate the excretion of ammonia resulting in decreased ammonia production. Rifaximin, an oral antibiotic, is recommended as an add-on therapy to lactulose for reducing the risk of HE recurrence. Typically, rifaximin 550 mg is administered orally twice a day. Currently no solid data exists to support the use of rifaximin alone (Vilstrup et al., 2014). Some practitioners may support its solitary use for patients intolerant to lactulose. The potential to cause diarrhea, malabsorption, and bacterial overgrowth syndromes must be considered. Chronic use requires meticulous monitoring including renal and neurological status. Antibiotics, such as neomycin, metronidazole, and vancomycin, are alternative short-term choices because of their potential adverse effects (nephrotoxicity and ototoxicity).

Other agents with limited proven efficacy that can be safely used and recommended as an alternative or additional agent to treat HE unresponsive to conventional therapy are oral branched-chained amino acids (BACCs)

Prophylactic lactulose and rifaximin are not recommended for the prevention of HE after TIPS, however it is used to treat recurrent HE post-TIPS (Vilstrup et al., 2014).

COLLABORATIVE CARE

Hepatorenal syndrome (HRS) is a progressive reversible form of renal failure in a patient who has advanced liver disease and ascites due to cirrhosis. It represents the end stage of a progressive sequence of vasoactive circulatory derangements that occur in advanced liver disease, causing a decrease in renal perfusion and glomerular filtration rate. The pathophysiology is complex, but, ultimately, there is profound renal vasoconstriction and prevailing peripheral vasodilation. In this syndrome, the kidneys have no intrinsic renal disease and are histologically normal. A nephrology consult is mandatory.

The diagnosis is one of exclusion, and it is important to distinguish HRS from prerenal azotemia and acute tubular necrosis. Manifestations include a decrease in urine output and difficulty managing ascites. The patient may also experience nausea, drowsiness, and thirst that is often indistinguishable from the way the patient typically feels. Eventually, manifestations include hemodynamic instability, oliguria, and coma.

There are two types of HRS. Type I is more serious with acute deterioration (twofold increase in serum creatinine reflecting decreased creatinine clearance) during a period of less than 2 weeks (Runyon, 2015). Often there is an acute precipitating event such as GI bleeding, bacterial infection, or large-volume paracentesis. Type II is less

Commonly Used Medications

Lactulose

Action: The result of lactulose (Cephulac) and its catabolism by the bacteria flora is a lowering of the colon pH to about 5.0. This causes the formation of a nonabsorbable form of ammonia and traps it in the colon. Because it is a cathartic, it speeds up transit through the colon, increasing the excretion of nitrogen. Most studies have found that lactulose has successfully decreased ammonia levels and improved encephalopathy in 80% of the cases. The nurse continues to monitor neurological status and expects to see an improvement in severe HE. It can take up to 48 to 72 hours to see a response.

Nursing Responsibilities:

- Lactulose may be administered in a variety of routes and schedules. In an acute situation to induce a rapid drop in serum ammonia, lactulose 45 mL may be ordered orally every hour until a bowel movement occurs.
- A sweet, syrupy liquid, it may be administered orally or through a nasogastric tube (NGT) for comatose patients or for patients with a decreased level of consciousness.
- If the patient cannot tolerate oral or gastric feeding, it may be administered as a retention enema. Then,

lactulose 200 g (300 mL) is diluted in 700 mL to 1 liter of water and ideally retained for an hour. It may be administered with a balloon (Foley) catheter to facilitate retention.

- It may be repeated every 4 to 6 hours as necessary. As a maintenance dose to prevent HE, lactulose 20 to 30 g (30 to 45 mL) may be administered three or four times a day, titrated to achieve two to three soft, controllable bowel movements per day. AASLD guidelines clearly stress that it is a misconception that no effect from small amounts of lactulose will be improved by administering larger doses (Vilstrup et al., 2014).

Side and/or Toxic Effects: The nurse anticipates that the patient may experience mild side effects such as bloating, flatulence, and cramps. The nurse should evaluate the patient for the development of tolerance to the side effects. Severe side effects such as diarrhea, dehydration, and acidosis need to be addressed. Diarrhea may indicate over-dosage and usually responds to dose reduction. The nurse anticipates a dose adjustment or stopping the medication temporarily or permanently if tolerance does not improve.

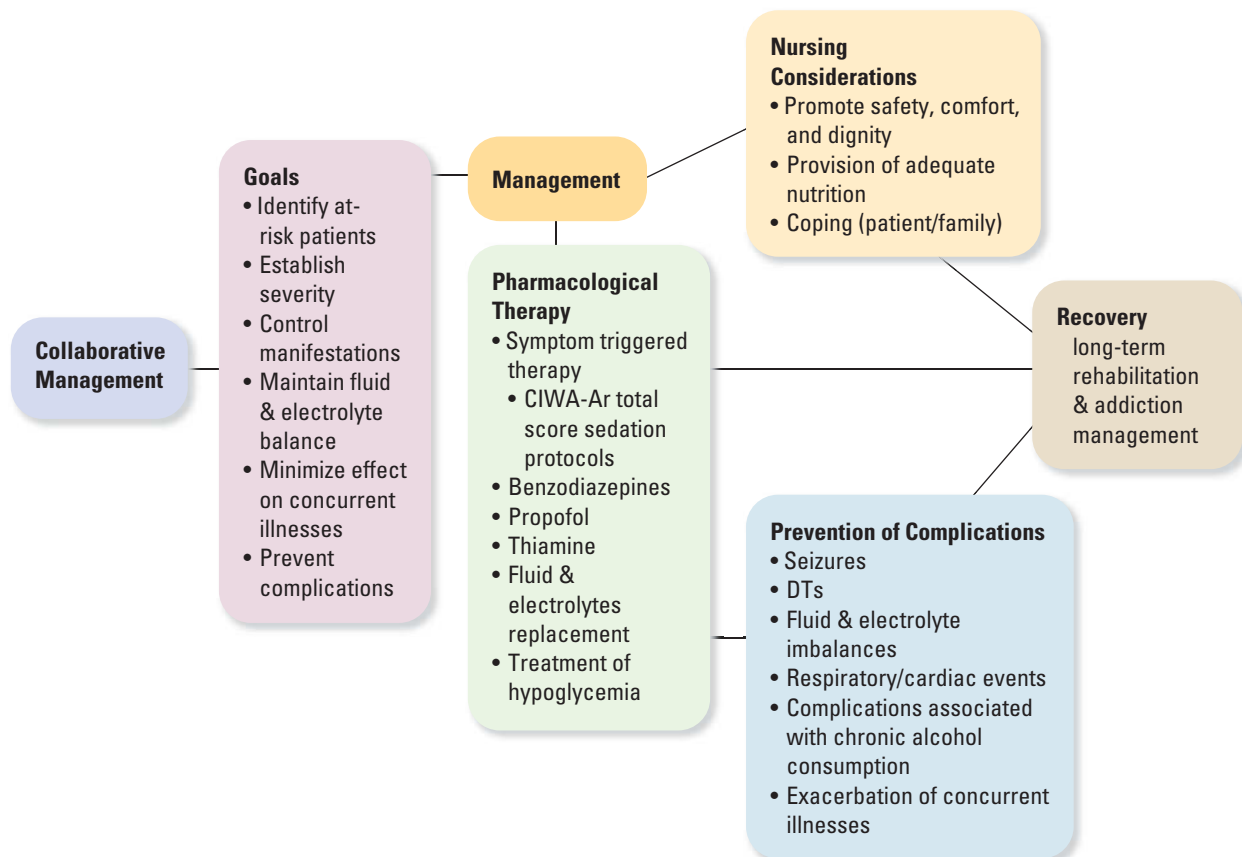
severe with a gradual progression, subtle onset, and refractory ascites is the main clinical feature. The mortality of patients with ALF who develop HRS is significantly worse. Outcomes of patients with HRS depend on improving or reversing hepatic failure. In the setting of end-stage liver disease, transplantation is the only cure for appropriate candidates.

Assessment and management focuses on prevention, prompt recognition, and treatments to improve glomerular filtration rate (GFR). Prevention stresses meticulous management of ascites and portal hypertension (Lenz, Buder, Kapun, & Voglmayr, 2015):

- Careful management of diuretics
- Immediate resuscitation in hypovolemic situations
- Albumin administration for SBP and large volume paracentesis
- Antibiotic prophylaxis for patients with high risk for SBP
- Withdrawal of beta blockers with recurrent and refractory cirrhosis
- Cautious use of nephrotoxic agents

Pharmacological therapy and albumin infusion can be considered in an attempt to manipulate renal and systemic hemodynamics to prolong survival until the patient can undergo a transplant. Intensive care patients typically require vasopressor infusion(s) in combination with albumin. Patients that are not critically ill may receive a combination of octreotide (infusion or subcutaneously) and midodrine (orally) in combination with albumin.

Certain patients may benefit from a TIPS procedure that may also prolong survival to receive a transplant. TIPS has been associated with a gradual improvement in GFR. Renal replacement therapy is also a consideration but often controversial in the setting of end-stage liver disease. Appropriate patients may be selected for dialysis as a bridge to transplantation. The risks of dialysis in this patient population are significant. Liver transplantation early in the course of HRS will most likely reestablish kidney function. Later in the course of the disease, patients may need a liver and kidney transplant. Recognizing that the patients/families are facing a grave situation, the nurse needs to evaluate their understanding and assess the need for the appropriate support services.



Visual Map 12-5 Chronic Liver Failure Management

NURSING CARE

Nursing care of the patient with chronic liver disease can be challenging. The nurse must provide for the patient's comfort while ensuring the patient's safety. It is also the responsibility of the nurse to ensure that the patient is adequately nourished to prevent the problems associated with protein calorie malnutrition.

Enhancing Comfort

Pain should be assessed and treated using a standardized scale, and the facility's pain management protocols should be followed. Patients with liver disease can have both acute and chronic pain from a variety of causes unrelated to their liver disease. Patients with ascites often complain of abdominal pain and low back pain, and patients with gynecomastia often have breast pain.

Patients with mild disease usually have no analgesic medication restrictions. As liver disease worsens, consideration must be given to both the choice of drug as well as the dose. However, there are no exact criteria or cutoff for modifying drugs and dosages. Generally, patients with cirrhosis, especially those with evidence of portal hypertension, should have their analgesic medications modified. Patients requiring long-term analgesia should be referred to a pain management program. General considerations related to analgesic medications and cirrhosis include the following:

- Nonsteroidal anti-inflammatory drugs should be avoided. They are associated with an increase in the risk of variceal bleeding, impaired renal function, and the development of refractory ascites.
- Opioids should be used with caution, and are typically prescribed in reduced doses with prolonged intervals between doses related to decreased clearance and increased bioavailability.
- For patients who are not drinking alcohol, acetaminophen is a safe analgesic when used in low doses. Between 2 and 4 grams per day is suggested. It should not be used in any patients with cirrhosis who continue to consume alcohol.

Pruritus

Itching can be a major source of discomfort for patients. It is caused by the accumulation of bile acids under the skin. It can be treated with medications that bind with the bile acids and prevent them from accumulating under the skin. Common medications are cholestyramine, colestipol, and colesevelam. Additionally, diphenhydramine may be prescribed at night for relief of symptoms and sedative effect. The nurse should caution the patient if a sedative effect is an issue. It is also helpful to keep the patient's nails short and to provide distractions.

Muscle Cramps

Patients with advanced liver disease may also experience severe muscle cramps. The cause is not totally understood, but is believed to be related to the reduction in effective circulating plasma volume. Such patients will have electrolytes, complete blood count, and INR closely monitored. In the past, quinine sulfate was prescribed; however, it is no longer available for the treatment of cramps. Though there are no evidence-based data, the small amount of quinine found in tonic water may be clinically helpful. Patients may be asked to drink 3 to 4 ounces of tonic water, evaluate effectiveness, and repeat as necessary for relief (Goldberg & Chopra, 2015b). Other options include branched chained amino acids, taurine, and vitamin E.

Maintaining Safety

All patients need to be assessed for risk of falls. Patients with advanced liver disease often have ascites and peripheral edema, which restrict their mobility and make ambulation very difficult. Patients experiencing HE may have varying degrees of confusion and disorientation. Fall assessments and prevention protocols should be appropriately implemented.

Patients with altered mental status and decreased level of consciousness are also at risk for aspiration, especially during a variceal bleeding with hematemesis. The nurse should assess the patient's risk and implement aspiration precautions as necessary.

Skin integrity is a concern for critically ill patients who are likely confined to bed related to ascites, peripheral edema, and immobility. Additionally, the patient with advanced liver disease is often malnourished, which is also a major risk factor for skin breakdown. The nurse should assess the patient's risk for skin breakdown by using an objective numerical assessment tool (e.g., Braden Scale). Depending on the risk, prevention measures according to hospital protocol or the guidelines published by the Agency for Healthcare Policy and Research (AHCPR) should be implemented.

Reflect On

Complications associated with cirrhosis and decompensated liver disease can affect the patient's quality and extent of life. What key interventions influence compliance regarding preventive measures and health maintenance? What is the nurse's role?

Providing Nutrition

In cirrhosis, protein calorie malnutrition (PCM) is prevalent and often considered a complication with poor outcomes. It can increase the rate of variceal bleeding, infection, renal and pulmonary complications, and HE, influencing both morbidity and mortality. PCM is a diet deficient in calories and proteins that results in body wasting. Additionally,

PCM has considerable consequences for patients who may be awaiting transplantation since it may lead to increased complications and postoperative mortality.

Patients with HE should be managed with medications and should not have a protein restriction. They actually have an increased need for protein, and limiting it will accelerate PCM. Limiting protein can result in a negative nitrogen balance, weight loss, weakness, malnutrition, and an increased chance of infection. If a patient is awaiting transplant, these factors can significantly influence post-transplant outcomes. Patients awaiting transplantation require aggressive nutritional support to prevent complications.

In an acute HE episode, a temporary protein restriction can be implemented for a day or so, but not be prolonged. A nutritional consult should be completed to design an individual diet for the patient, evaluate adequate nitrogen intake, and discuss the feasibility of substituting vegetable protein for other sources of protein. Vegetable proteins are preferred over animal sources because they have a higher calorie-to-nitrogen ratio, and are less likely to cause HE.

An increase in protein intake and tolerance may also be accomplished by combining other therapies. It is recommended that the restriction of dietary protein be reserved for patients with HE who cannot be controlled in any other way. In these cases, a daily energy intake should be 35–40 kcal/kg/day, and a protein intake of 1.2–1.5 g/kg/day. As above a diet rich in vegetables and dairy protein should be encouraged. The AASLD practice guidelines recommend branched chained amino acid (BACC) supplements for HE especially for patients intolerant of dietary protein. Other suggested benefits are the relief of muscle cramps, improvement of immune function, higher serum albumin levels, overall better nutritional status, and improved quality of life (Vilstrup et al., 2014).

Overall, it is recommended that chronic liver disease patient's optimal daily energy intake is 30–50 cal/kg/day, protein 1.0 to 1.8 g/kg/day, carbohydrates 45–75% of caloric intake, fats 20–30% of caloric intake and vitamins and minerals to correct specific deficiencies (Bemeur & Butterworth, 2014). Patients should be advised to consume small frequent meals throughout the day including a bedtime snack of complex carbohydrates (adjusted for obese patients) to maintain positive nitrogen balance.

The nurse may be required to document protein calorie intake and monitor weight. Continued monitoring of neurological status is necessary to evaluate therapeutic effect.

Factors That Interfere with Adequate Nutrition

The nurse needs to be aware of the following factors that interfere with eating, causing insufficient intake in the patient with cirrhosis:

- Anorexia, nausea, and vomiting (often induced by medications)
- Early satiety (fullness) (increased pressure and compression from ascites)
- Bloating, abdominal distention, constipation, or diarrhea
- Alterations in taste and smell (possibly related to vitamin A and/or zinc deficiency)
- Unpleasant, tasteless diets (low sodium)
- Fatigue, lack of activity
- Encephalopathy (Altered mental status may alter eating behavior.)
- Alcohol (Patients with alcoholic cirrhosis often have pancreatic insufficiency which can cause a decrease in fat digestion and malabsorption.)

Nutritional Assessment and Chronic Liver Disease

All patients with chronic liver disease should have a nutritional assessment and interventional plan for nursing to promote. Most likely, patients with compensated disease will be prescribed a normal, balanced, healthy diet with adequate calories, proteins, fats, and carbohydrates. If oral intake is inadequate, additional oral supplements or tube feedings should be initiated. Supplemental nocturnal nutrition has improved overall nutritional status. Depending on the cause of the liver disease, vitamins and minerals may be prescribed, but megadoses should be avoided because these may also cause hepatotoxicity. Osteoporosis is a common finding in chronic liver disease. Calcium and vitamin D will be prescribed accordingly. All herbal supplements and nontraditional herbal remedies should be discussed with the physician and dietician. Raw seafood should be avoided because of the risk of septicemia with a species of halophilic gram-negative bacilli found in marine organisms and seawater. The goal is to follow these patients closely to recognize disease progression and implement therapy to prevent PCM.

As the liver disease progresses, the patient's nutritional status is often a challenge and needs close intervention and monitoring. Many of the markers that are typically used for assessment are no longer effective. With ascites and edema, the nurse recognizes that weight is not an accurate measure and that laboratory studies, especially albumin, are also unreliable. The dietician can employ several other assessment techniques to evaluate the patient's nutritional status. The goals for nutritional interventions are to prevent PCM, improve existing PCM, and correct nutrient deficiencies.

Prevention of Infection

Twenty percent of the patients with cirrhosis who are admitted with an active bleed have a bacterial infection present on admission, and another 50% of the patients are likely to develop an infection while in the hospital (Sanyal, 2015a). Urinary tract infections, SBP, and respiratory and primary bacteremia are the most common

sources of infection. Acute bleeds are often associated with gut translocation and motility issues causing bacterial infections. Infected cirrhotic patients also have a higher re-bleeding rate. Therefore, it is imperative that the nurse utilize all appropriate methods to prevent infection. This includes hand washing, implementing the central line bundle, preventing catheter-associated urinary tract infections (CAUTI), providing adequate nutrition by the enteral route when possible, and instituting the ventilator bundle (if appropriate).

The presence of infection in this population also increases mortality, and studies have shown that the administration of antibiotics may significantly reduce mortality and recurrent bleeding (Sanyal, 2015b). The nurse anticipates the administration of prophylactic antibiotics. If an infection is suspected on admission, appropriate cultures may be ordered. However, in the

presence of a massive hemorrhage, cultures will not be a priority and antibiotics will be administered. The nurse must review the patient's allergy history and administer according to facility protocol. Patients need to be monitored for hypersensitivity reactions, evidence of infection/sepsis, and renal function throughout the course of therapy. Antibiotics known to cause nephrotoxicity should be avoided.

Essential for Evidence-Based Practice

It is imperative that the nurse is compliant with all infection control measures especially hand washing in order to prevent infections. This patient population is considered to be immunosuppressed, and infection can have a horrific effect on clinical status and survival.

Alcohol Withdrawal/Liver Failure Summary

Alcohol use disorders can affect an individual's personal, social, and vocational relationships as well as have acute and chronic effects on all physiologic systems. Prompt identification and treatment of patients at risk for or who are experiencing AWS, prevention and treatment of complications, and provision of the appropriate level of care directed by clinical assessments are fundamental to positive patient outcomes.

Liver failure is very complex and presents many challenges to the healthcare team. Though time frames and durations differ, both acute and chronic liver failure have serious life-threatening complications and implications for transplantation. Acute liver failure is a medical

emergency requiring prompt diagnosis, identification, and treatment of the cause and determination of candidacy for liver transplantation. The prognosis of end-stage liver disease or cirrhosis is variable and does not always have end-of-life implications. The healthcare team may encounter patients experiencing either compensated or decompensated disease. No matter what stage, the nurse's role as an educator and advocate is instrumental in engaging the patient and family to actively participate in their care. It is imperative that patients and families understand the effects of cirrhosis on all body systems and the importance of vigilant compliance with treatment.

Why/Why Not?

A 62-year-old patient with bleeding esophageal varices has been typed and crossed matched for 3 units of packed red blood cells. His blood pressure is currently 98/54 (68), heart rate 98, and hemoglobin 8.2 g/dL. Should the nurse

prepare for an immediate transfusion of a unit of blood? Why or why not?

See answers to Why/Why Not? in the Answer Section.

Case Study

Barbara Ramsey is a 39-year-old woman admitted to the ICU from the emergency department (ED). On arrival in the ED, her blood pressure was 60 systolic. She says she vomited bright red blood with large clots three times at home

then fainted. She just vomited approximately 250 mL on admission to the ICU. On admission to the ED, her hematocrit was 24, her hemoglobin was 8, her systolic blood pressure was 62 mmHg, and her pulse rate was 146 beats/min.

She has received a liter of normal saline and two units of blood already. Her blood pressure is now 82 systolic.

Barbara is known to the facility. She was discharged a week earlier after being admitted for bleeding esophageal varices and ascites. In the past, she stated that she drank a pint of vodka with crème de menthe chasers daily. She is scheduled for an immediate endoscopy with possible banding of esophageal varices.

1. What should be the first priority in Barbara's care?
2. What additional information would the nurse want to obtain?

The nurse will need additional assessment information, especially a rapid full physical assessment,

3. What factors are contributing to the severity of Barbara's bleeding?
4. What factors contributed to the likelihood that Barbara would re-bleed from her varices?

5. What should the nurse do to prepare Barbara for endoscopy and possible banding?
6. What alternatives are there to banding to control bleeding from esophageal varices?
7. Should the nurse be concerned that Barbara might undergo AWS during this hospitalization?

A day later, Barbara no longer responds to her name. She is disoriented but arousable in the afternoon, but her speech is incomprehensible. By the evening, she is no longer arousable.

8. How should the nurse respond to the change in Barbara's mental status?
9. What is probably happening to Barbara? What is the most likely collaborative management?
10. Would Barbara be a good candidate for TIPS? Why or why not?

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 12.1 What is the relationship between the pharmacological effects of alcohol and the cause of withdrawal?
- 12.2 What are the essential components of a focused assessment to screen for alcohol abuse and dependency and the risk for AWS?
- 12.3 What is the role of the CIWA-Ar scale in the collaborative management of AWS?
- 12.4 What is the clinical significance of a patient developing the DTs?
- 12.5 What is the clinical distinction between alcohol hallucinosis and the DTs?
- 12.6 What are the safety concerns in the nursing management of AWS?
- 12.7 Why are benzodiazepines the drug of choice for the management of AWS? What are the nursing considerations in the administration of benzodiazepines?
- 12.8 What is the clinical significance of AWS developing in a critical care patient?
- 12.9 Explain the differences between acute liver failure and chronic liver failure
- 12.10 Why is acetaminophen overdose dangerous?
- 12.11 When and how is acetylcysteine utilized as an antidote for acetaminophen overdose?
- 12.12 What is the relationship between portal hypertension and the development of decompensated liver disease?
- 12.13 What are the essential assessments that the nurse should perform for the patient with decompensated liver disease?
- 12.14 What are the nursing responsibilities for the patient with ascites or hepatic encephalopathy?

See answers to Chapter Review Questions in the Answer Section.

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Chapter 13

Care of the Patient with an Acute Gastrointestinal Bleed or Pancreatitis

June Kasper, MSN, RN, CGRN,
Michele Bettinelli, BS, RN, CCRN

Abbreviations

AP	Acute Pancreatitis	MSOF	Multisystem Organ Failure
EGD	Esophagogastroduodenoscopy	PPI	Proton Pump Inhibitor
FFP	Fresh Frozen Plasma	SIRS	Systemic Inflammatory Response Syndrome
FNA	Fine Needle Aspiration	UGI	Upper Gastrointestinal
LGI	Lower Gastrointestinal		



Learning Outcomes

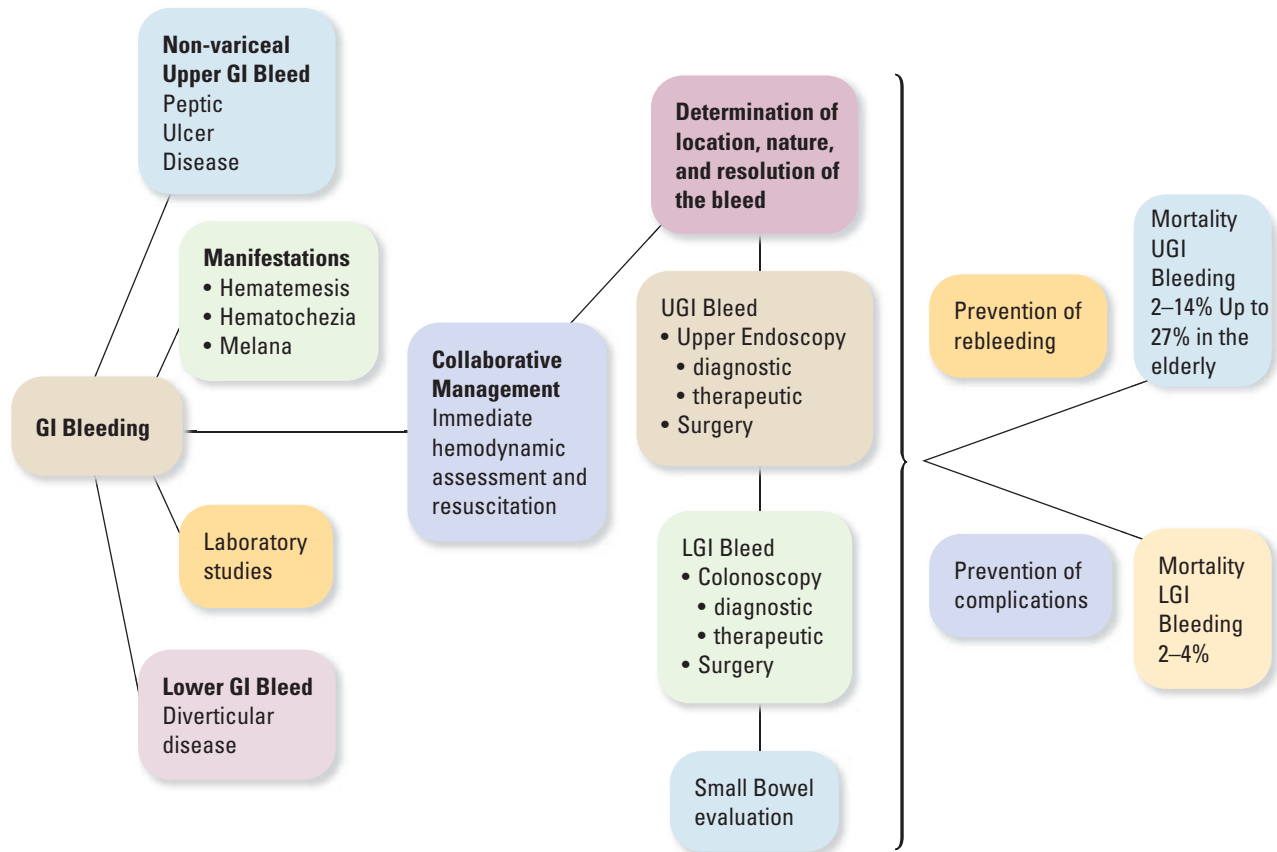
Upon completion of this chapter, the learner will be able to:

1. Compare and contrast risk factors, clinical manifestations, and collaborative management of the person with upper and lower gastrointestinal bleeding.
2. Explain why pancreatitis may develop and differentiate between the manifestations of mild and severe pancreatitis.
3. Describe collaborative management and nursing responsibilities when caring for the patient with severe pancreatitis.

Introduction

Gastrointestinal (GI) bleeding is a common, costly, and potentially life-threatening medical condition. Bleeding can occur from numerous types of lesions anywhere in the GI tract. Upper gastrointestinal (UGI) bleeding originates proximal to the ligament of Treitz. UGI bleeding is classified as variceal or nonvariceal (see Chapter 12). The annual incidence of hospitalization for acute upper GI bleeding is approximately 100 per 100,000 adults. UGI bleeding is twice as

common in males as in females, and increases with age (Rockey, 2016). Although there have been improvements in medical management, the overall mortality rate is 2% to 14% and as high as 27% in elderly patients (Simon, Travis, & Saltzman, 2015). Lower gastrointestinal (LGI) bleeding refers to bleeding originating distal to the ligament of Treitz, and is differentiated into bleeding from the small bowel or mid-GI bleeding, and bleeding from the colon, or lower GI bleeding (Strate, 2015a). Recent studies indicate that the incidence of hospitalizations for LGI bleeding is now close to that of UGI



Visual Map 13-1 Overview of Gastrointestinal Bleeding

bleeding (Strate, 2015a). Approximately 35.7% per 100,000 adults are hospitalized for lower GI bleeding annually, and the mortality rate ranges from 2% to 4% (ASGE, 2014). Best practice is a multidisciplinary team approach adhering to clinical guidelines. Management focuses on immediate assessment, hemodynamic stabilization, identification of the source, treatment of the bleeding, and prevention of re-bleeding. Responsibilities should be clearly defined to avoid lapses in resuscitation (Rajala & Ginsberg, 2015).

Anatomy and Physiology Review

The GI tract begins at the oral cavity and ends at the anus (♦ Figure 13-1). The structures of the GI tract have overlapping function yet each has an area of specialization and distinct histological characteristics. The major functions of the GI tract include:

- Ingestion
- Mechanical processing
- Digestion
- Secretion

- Absorption
- Excretion

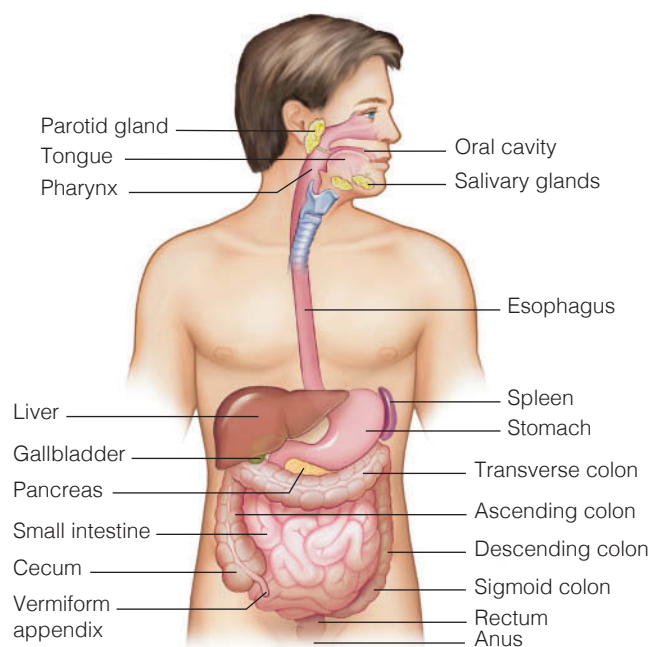


Figure 13-1 Organs of the alimentary canal and related accessory organs.

The Gastrointestinal Tract

The structures of the GI tract include the esophagus, stomach, small intestine, ileocecal valve, and large intestine.

The esophagus is a hollow muscular tube, approximately 10 inches long and 1-inch-wide, that carries solids and liquids from the pharynx to the stomach. The stomach is a J-shaped organ located below the diaphragm between the esophagus and the small intestine. Functions include storage of food; mechanical breakdown of food; and production of gastric secretions, hydrochloric acid (pH 1.0–4.0), and intrinsic factor. The small intestine is a tubular structure, approximately 20 to 25 feet long and 1½ inches wide, responsible for most of the important digestive and absorptive functions. It consists of three sections: The duodenum is 10 inches long with the terminal landmark, the ligament of Treitz; the jejunum is 8 feet long; and the ileum is 12 feet long. The distal end of the ileum connects to the large intestine. The ileocecal valve marks the transition between the small and the large intestines. The large intestine is a tubular structure, approximately 5 to 6 feet long and 2 to 4 inches wide. It consists of five sections: cecum/ascending colon, transverse colon, descending colon, sigmoid colon, and rectum. It functions to eliminate wastes and absorb water and electrolytes.

The Patient With Gastrointestinal Bleeding

GI bleeding is a common reason for a patient to be admitted to an intensive care unit (ICU). Studies have also determined that 74–100% of critically ill patients develop stress-related GI mucosal erosions within 24 hours of admission that can lead to serious GI bleeding in 0.6 to 4% of patients (Plummer, Blaser, & Deane, 2014).

Predisposing Factors and Causes of Gastrointestinal Hemorrhage

Peptic ulcer disease accounts for approximately 31% to 67% (Simon et al., 2015) of nonvariceal UGI bleeds, and diverticular bleeding accounts for 20% to 65% (ASGE, 2014) of acute LGI bleeds. Individuals with LGI bleeding often present with less severity than those with UGI bleeds, and are less likely to manifest orthostasis or shock or require blood transfusions. Mortality from UGI bleeding has decreased in the past two decades but still ranges from 2% to 15% (Fortinsky, Bardou, & Barkun, 2015).

Peptic Ulcer Disease

Peptic ulcer disease (PUD) typically refers to ulcers in the stomach and the first part of the duodenum, also called

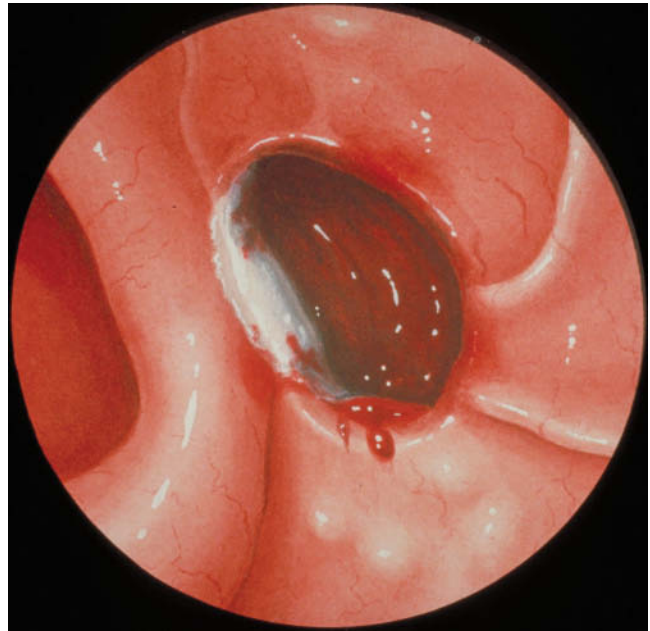


Figure 13-2 Peptic ulcer disease.

(St Bartholomew's Hospital/Science Source)

the duodenal bulb. Ulcers occur less commonly in the esophagus. In addition to ulcers, erosions can form in the GI tract. The difference between ulcers and erosions is the depth of penetration. Erosions are superficial and do not involve the smooth muscle layer. ♦ Figure 13-2 displays a peptic ulcer with an adherent clot.

The major risk factors for peptic ulcer disease are:

- Age—more common in older adults (60% > 60 years and 20% > 80 years [Wong & Sung, 2015])
- *Helicobacter pylori*—a highly mobile bacterium that avoids acid by burrowing underneath the mucosa. It causes the mucosa to be more susceptible to peptic acid damage and provokes an inflammatory response, which results in further epithelial injury.
- Nonsteroidal anti-inflammatory drugs (NSAIDs) and aspirin (including low dose)—cause local and systemic effects that promote mucosal damage. Many studies have compared GI toxicity of different NSAIDs. One study looked at asymptomatic individuals taking aspirin and found that 4% had ulcers and 34% had erosions (Feldman & Das, 2015). NSAIDs have also been associated factor in non-healing ulcers.
- Stress-related mucosal damage (including stress ulcers)—develop in hospitalized patients with serious illnesses. Patients with GI bleeding secondary to these lesions have a higher mortality than those admitted with primary UGI bleeding (Rockey, 2016). The cause of stress-related mucosal damage is thought to be related to a disproportion in the production of gastric

acid and mechanisms to protect the gastric mucosa. Mucosal ischemia resulting from hypoxia, systemic hypotension, and splanchnic hypoperfusion is thought to contribute to mucosal injury. The risk is increased in patients with respiratory failure and those with a coagulopathy. Other associations include sepsis, hepatic failure, renal failure, burns and major trauma (Plummer et al., 2014).

- Gastric acid and pepsin are co-contributors to the above etiologies. Because of the mucosal impairment, there is increased permeability of acid. In rare occasions, hyperactivity of these secretions can cause ulcers.

Other less frequent causes include:

- Erosive esophagitis, gastritis, or duodenitis
- Mallory-Weiss tear—mucosal lacerations at the gastroesophageal junctions or the cardia of the stomach associated with wrenching and vomiting
- Benign or malignant tumors
- Vascular abnormalities

Contributing factors include:

- Smoking—thought to increase the risk of ulcer development and impair healing
- Alcohol use disorder—thought to interfere with the healing process

Clinical risk assessment and prediction of patient outcomes is based on clinical, laboratory, and endoscopic data. Many scoring systems have been developed to assess severity of bleeding and determine appropriate interventions to reduce mortality and morbidity. The immediate priority in managing an UGI bleed is to secure the airway and initiate resuscitation before any other procedure (Fortinsky et al., 2015). The goal is to restore circulating blood volume and prevention of hypovolemic shock. Endoscopy within 12 to 24 hours of admission is essential for those unstable on admission or who continue to actively bleed after resuscitation. Urgent endoscopy within 12 hours is recommended for patients with a history concerning for variceal bleeding and within 24 hours for suspected non-variceal bleeding (Rajala & Ginsberg, 2015). Clinical factors linked to poorer outcomes include:

- Hemodynamic instability
- Low initial hemoglobin level
- Multiple blood transfusions
- Presence of bright red blood in emesis or stool
- Age greater than 60
- Concurrent hospitalization/illnesses

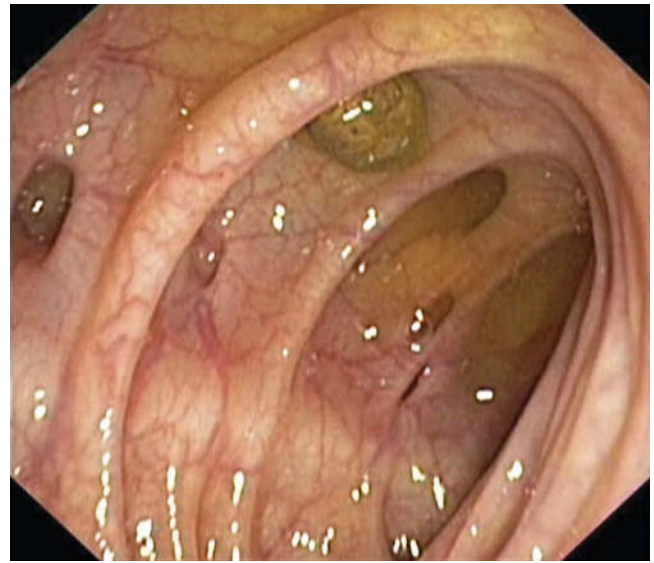


Figure 13-3 Diverticular disease.

(Gastrolab/Science Source)

- Coagulopathy
- Continued bleeding or re-bleeding (especially within 72 hours of initial bleed)
- Emergent surgical intervention

Diverticular Disease

Diverticular disease results from weak points on the intestinal wall that herniate to form a saclike projection called diverticula (♦ Figure 13-3). They are most often found in the descending and sigmoid colon, but they can be throughout the colon. Diverticula are present in approximately 30% of individuals ≥ 50 years old and prevalence increases to 60% in individuals > 80 years old (ASGE, 2014). Bleeding results from rupture of submucosal arterial vessels at the neck or the dome of the diverticulum. NSAID use increases the risk for diverticular bleeding. Bleeding usually resolves spontaneously in 75% to 80% of patients, but recurs in 25% to 40% within 4 years (ASGE, 2014). Clinical presentation is usually acute, painless passage of bright red blood. Such bleeding has not been correlated with inflammation (diverticulitis). As the incidence of LGI bleeds increases with age, comorbidities have a significant impact on mortality.

Other less common causes include:

- Ischemic colitis
- Vascular ectasis (angiodysplasia, angioectasis)
- Anorectal disease (hemorrhoids, anal fissures)
- Neoplasms (small intestine/large intestine)
- Post-polypectomy bleed
- NSAID usage

Gerontological Considerations

Forty-five percent of all admissions for GI bleeding are in older adults. There is an increased risk of UGI and LGI bleeding associated with antithrombotic medications and NSAIDs, which many older adults require to treat cardiovascular and/or rheumatologic conditions. In addition, the incidence of diverticular disease, vascular ectasia, neoplasms, and ischemic colitis increases with age (Strate, 2015b). Colonic arteriosclerosis is universal in the elderly, which results in changes to the mesenteric vasculature and predisposition for ischemic colitis (ASGE, 2014).

Older adults are more likely to have complex medical problems/comorbidities, complicated medication regimens, and more diverse causes of PUD.

GI bleeding in the older adult may present with signs of dehydration and abdominal cramping.

More than 50% of adults over the age of 50 have radiological evidence of diverticulitis, which is believed to contribute to the incidence of lower GI bleeding in the older adult.

In older adults, pancreatitis is rarely due to alcohol use and often presents with subtle symptoms rather than acute pain, making the diagnosis more difficult.

Acute pancreatitis is responsible for between 5% and 7% of abdominal pain in older adults.

Manifestations of Gastrointestinal Bleeding

The most common manifestations of GI bleeding are hematemesis, melena, and hematochezia. The appearance and presentation of the blood facilitates identification of the site as well as determination of the acuity and severity of the bleeding. **Hematemesis** is vomiting of blood that is either bright red or has a coffee grounds appearance, indicating UGI bleeding. Coffee grounds appearance is caused when blood is mixed with digestive juices, and it usually indicates the bleeding has slowed or stopped. **Melena** is the passage of black tarry colored stool with a very characteristic foul odor. It reflects the action of intestinal contents on the blood. Melena can result from as little as 50 mL of blood in the stomach and usually suggests an UGI bleed, however the source can also arise from the small bowel or ascending (right) colon. **Hematochezia** is usually red or maroon blood in the stool and is most often due to lower GI bleeding however it can also occur with massive UGI bleeding which is usually associated with orthostatic hypotension (Saltzman, 2015a). Though helpful in evaluating the source of bleeding, stool color is not absolute. Both UGI and LGI bleeders can have melena and/or hematochezia.

past medical history and identifying comorbidities and medications that may cause GI bleeding helps to develop the appropriate care.

Immediate management is directed by hemodynamics and may involve resuscitation and stabilization of vital signs. The goal is to identify the source, stop the bleeding, prevent recurrent bleeding, and prevent and treat complications. A prompt and accurate diagnosis and the severity of the bleeding will direct therapeutic interventions that should decrease complications and mortality. Severe GI bleeding usually requires ICU monitoring, which has been linked with positive outcomes and decreased mortality.

Assessment of Fluid Volume Status

The critical care nurse will evaluate the extent of blood loss by immediately assessing the patient's hemodynamic status. Assessment of vital signs is the best way to evaluate stability, amount of blood loss, and suddenness of the loss and the extent of cardiac and vascular compensation. Vital signs can also provide prognostic information. The nurse

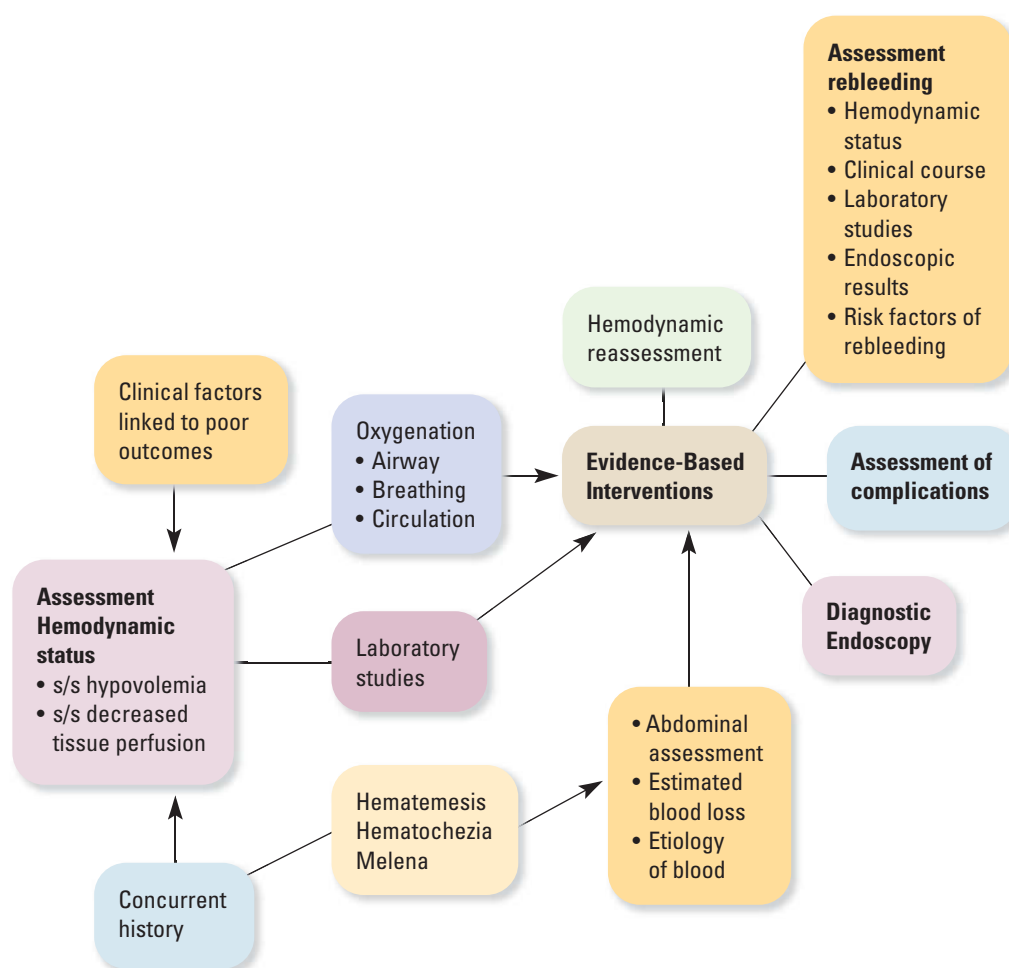
COLLABORATIVE CARE

Initial assessment focuses on hemodynamic status with a concurrent history and physical focusing on the source and the severity of the bleeding (Table 13-1). Patients should be asked about prior episodes of UGI bleeding because up to 60% of patients with a history of UGI bleeding will be bleeding from the same lesion (Saltzman, 2015a). Reviewing the

Table 13-1 Sources Of Bleeding

Hematemesis	Esophagus, Stomach, Duodenum
Coffee-ground emesis	Esophagus, Stomach, Duodenum
Melena	Esophagus, Stomach, Duodenum Right Colon
Hematochezia	Right Colon, Left Colon (if severe bleeding, Esophagus, Stomach, Duodenum)
Guaiac-positive stool	Esophagus, Stomach, Duodenum, Right Colon, Left Colon

Source: Simon et al., 2015.



Visual Map 13-2 Assessment of Gastrointestinal Bleeding

assesses for manifestations of hypovolemia and poor tissue perfusion, including:

- Hypotension (systolic blood pressure less than 90 and mean arterial pressure less than 60 mmHg). Associated with a blood loss of approximately 40%.
- Narrowed pulse pressure.
- Orthostatic hypotension—if the patient is not hypotensive, orthostatic signs can identify smaller volume depletion. A decrease of greater than 20 mmHg in the blood pressure (BP) or an increase greater than 20 beats/minute in heart rate from recumbence to standing indicates a blood loss of at least 15%.
- Tachycardia (heart rate greater than 100). Focus on resting heart rate and consider whether rate is influenced by beta blocker medication.
- Electrocardiogram (ECG) changes, dysrhythmias, and ST changes in response to ischemia. Advanced age and a history of cardiac disease can increase risk of ischemia.
- Chest pain. Not uncommon is the simultaneous presentation of an acute myocardial infarction secondary to massive bleeding causing hypovolemia, hypoperfusion, and hemodynamic compromise. Often in

these circumstances the patient does not present with typical chest pain, and/or the chest pain can be misinterpreted for epigastric pain.

- Capillary refill greater than 3 seconds with cold clammy skin and weak peripheral pulses.
- Dry mucous membranes, poor skin turgor, and flat jugular veins.
- Decreased urine output (less than 30 cc/hour).
- Mental status changes.

Significant comorbidities or hemodynamic instability may necessitate more invasive monitoring; thus, the patient may have an indwelling urinary catheter and a central venous pressure (CVP) line. The nurse will assess the characteristics of overt blood loss from hematemesis, melena, and/or hematochezia.

Calculation of blood loss relative to the amount of measured melena or hematochezia is difficult and inaccurate related to being mixed with stool. The nurse should remember that the sickest patients are the easiest to diagnose. It is the patients with less than 30% blood loss who are more difficult to recognize. Although it is important not to underestimate blood loss, the rate of blood loss is often more critical than the volume.

Laboratory Studies

Additionally, the nurse reviews the following laboratory studies:

- **Hemoglobin**—The initial hemoglobin may not reflect the degree of blood loss, because there is a delay in intravascular equilibrium that will not immediately reflect blood loss. Patients with chronic slow bleeding may have a very low baseline hemoglobin but are generally hemodynamically stable. The nurse should also recognize that resuscitation efforts causing overhydration can lead to falsely low hemoglobin values.
- **Platelets**—A low platelet count may indicate a preexisting thrombocytopenia that needs to be treated. It may also suggest the presence of portal hypertension.
- **Electrolytes**—May be abnormal related to vomiting and diarrhea.
- **Blood urea nitrogen (BUN)/creatinine**—These are evaluated to assess hydration status and renal function and to help differentiate between a UGI and LGI bleed. Patients with acute UGI bleeding have decreased renal perfusion and typically have an elevated BUN to creatinine ratio (20:1). The higher the ratio, the more likely bleeding is from an upper source (Saltzman, 2015a).
- **Prothrombin time (PT)/international normalization ratio (INR)**—May indicate preexisting coagulopathy that needs to be treated. It may also be a marker for liver disease.
- **Cardiac enzymes**—Should be considered in all patients with massive bleeding and elderly patients with hypotension to exclude myocardial infarction.
- **Liver function tests**—Liver function is evaluated to assess for the presence of liver disease.
- **Type and cross-match**—Needs to be procured and processed immediately on all patients suspected of a GI bleed to determine blood type.

NURSING ACTIONS

The nurse works collaboratively with the healthcare team to identify and treat hypovolemia. The goal of resuscitation is to restore intravascular volume, maintain cardiac output, restore blood cells, and prevent complications of red blood cell loss such as pulmonary, neurological, cardiac, and renal injury.

Interventions to Maximize Oxygen-Carrying Capacity of the Blood

The nurse protects the patient's airway and supports breathing with supplemental oxygen to maximize oxygen-carrying capacity of the blood. The nurse will continuously evaluate pulse oximetry and report values

less than 92%. In the event of a massive UGI bleed and/or hemodynamic instability, the need for intubation and ventilation should be evaluated. NPO status should be maintained.

Interventions for Restoration of Normovolemia

Early aggressive resuscitation has been shown to improve patient outcomes. The patient should have two large-bore intravenous catheters placed immediately. Studies have indicated that short large-bore peripheral IV catheters are capable of faster flow rates than longer central line catheters of the same gauge (Simon et al., 2015). The initial resuscitation protocol for hemodynamic instability is the rapid administration of a crystalloid solution, generally normal saline (lactated Ringer's may be preferred by some clinicians), to replace intravascular volume and prevent shock. The crystalloid solution provides adequate circulation for the remaining erythrocytes until the type and cross-matched blood is obtained.

Total fluid deficit cannot be accurately predicted, and fluid resuscitation should remain at a rapid rate as long as the BP remains low. Other clinical parameters guiding resuscitation are vital signs, urine output, mental status, and peripheral perfusion. The nurse anticipates that the patient's vital signs shall begin to normalize and the urine output will increase within 15 to 20 minutes of the fluid challenge. However, if severe bleeding continues, the vital signs may continue to deteriorate and the patient may experience shock.

The nurse closely monitors the patient's cardiovascular and respiratory status for tolerance of rapid infusion rates. Patients with a history of heart failure, valvular disease, pulmonary disease, and other comorbidities may need a PA line for a more accurate indication of volume status and to minimize risk for fluid overload. The nurse looks for clinical indications of fluid overload, including:

- Abnormal lung sounds
- Tachypnea/shortness of breath
- Peripheral edema and neck vein distention
- Weight gain
- Chest pain
- Tachycardia
- Oxygen desaturation

Blood Transfusions

The decision to transfuse the patient with packed red blood cells is individualized based on the patient's clinical picture. The optimal hemoglobin transfusion strategy remains controversial. Guidelines recommend a restrictive strategy

(less than or equal to 7g/dl hemoglobin threshold) for initiation of blood transfusions in patients with acute gastrointestinal bleeding. This restrictive strategy as compared to a more liberal strategy (less than or equal to 9-10g/dl) significantly improves outcomes. For acute UGI bleeding, a study by Villanueva et al., (2013) concluded that a restrictive strategy reduced the risk of further bleeding, the need for rescue therapy, the rate of complications, and increased the rate of survival. The liberal approach has been associated with adverse clinical outcomes (Simon et al., 2015). A patient with chronic anemia and low hemoglobin may receive alternate therapies. Generally, young, previously healthy individuals tolerate a lower hemoglobin than elderly patients or patients with severe comorbid illnesses. The restrictive strategy may not be ideal for certain populations including massive bleeding, hypovolemia, shock, and tissue hypoxia/ischemia. Patients simultaneously experiencing an acute coronary syndrome have increased mortality rates and require a higher hemoglobin strategy to avoid decompensation (Fortinsky et al., 2015).

The following variables must be considered:

- Hemodynamic status/unstable vital signs
- Evidence of tissue hypoxia
- Age
- Estimated blood loss/severity
- Evidence of active bleeding/rate of bleeding/risk for re-bleeding
- Comorbidities, especially cardiovascular/pulmonary disease
- Hemoglobin

Packed Red Blood Cells

The nurse anticipates the infusion of packed red blood cells (RBCs) primarily to prevent or alleviate signs and symptoms of inadequate oxygen tissue delivery, maintain adequate tissue perfusion, and prevent end organ damage. The number of units infused and the rate of infusion will again depend on multiple individual variables as discussed. Typically, patients with hemodynamic instability have had considerable blood loss and will require a blood transfusion. Patients with evidence of continued bleeding, poor tissue oxygenation, unresolved hemodynamic instability, and repeatedly low hemoglobin will require aggressive transfusion therapy. In rare emergent situations, whole blood may be ordered while awaiting cross-matched blood.

The nurse follows facility protocols for administering blood and blood products, and will monitor the patient closely for transfusion reactions. The nurse anticipates that serial hemoglobins levels will be monitored after initial crystalloid resuscitation, the plasma volume may be overexpanded, and an immediate post transfusion

hemoglobin may underestimate the final value. Given adequate time to equilibrate, absence of an increased or decreased hemoglobin should cause concern. To review information on blood transfusions, see Chapter 9 . Laboratory values should not be a substitute for ongoing clinical assessment.

If the patient receives multiple transfusions, blood is delivered through a blood warmer to prevent a decrease in body temperature (hypothermia).

Platelets/Fresh Frozen Plasma (FFP)/Cryoprecipitate (Factor VIII)

The optimal platelet transfusion threshold has not been established. Expert consensus recommends platelet transfusion for platelets less than 50,000/ μ L, or if altered platelet function is suspected. Preexisting coagulopathy (INR greater than 1.5) may necessitate transfusing FFP. Studies have suggested a relationship between an INR greater than 1.5 and increased mortality (Simon et al., 2015). Treatment of dilutional coagulopathy that accompanies multiple red blood cell transfusions, must also be evaluated. When possible anticoagulant and antiplatelet medications should be held. Management of these agents, including the numerous novel oral anticoagulants, must consider the urgent setting vs. the non-urgent setting. Evaluating the individual benefits and risks involved with discontinuing or reversing coagulation is critical. Additionally, other reversal agents should be considered for specific coagulation disorders as well as the specific anticoagulant/antiplatelet agents. For example, vitamin K may be ordered to correct warfarin-related coagulopathy. Correction of coagulopathy should not delay endoscopy. Patients being considered for surgery need to be treated aggressively.

Patient Positioning

If a patient's BP is less than 90 mmHg systolic, the nurse should consider positioning the patient supine with legs elevated to facilitate venous blood return to the right atrium. As the BP returns to normal, the patient's legs can be gradually lowered while the nurse monitors carefully for BP changes.

Gathering of Additional Assessment Data

Soon after the emergent assessment and resuscitation efforts, the nurse needs to review the patient's clinical history and perform a more focused physical exam. Patient's history must be reviewed for significant risk factors and clues to the etiology and severity of the bleed. The physical examination, including clinical presentation and emesis/stool characteristics, are essential for evaluation of site and acuity.

Key historical elements include:

- Demographics—common disorders according to age
- Previous or current GI disease/GI bleeding
- Prior surgery
- Aspirin/NSAID ingestion
- Alcohol use disorders
- Liver disease
- Anticoagulants/antiplatelet agents/clotting disorders
- Pain
- Vomiting/retching, weight loss, anorexia, change in bowel habits
- Non-GI disorders (pulmonary/nasopharyngeal)

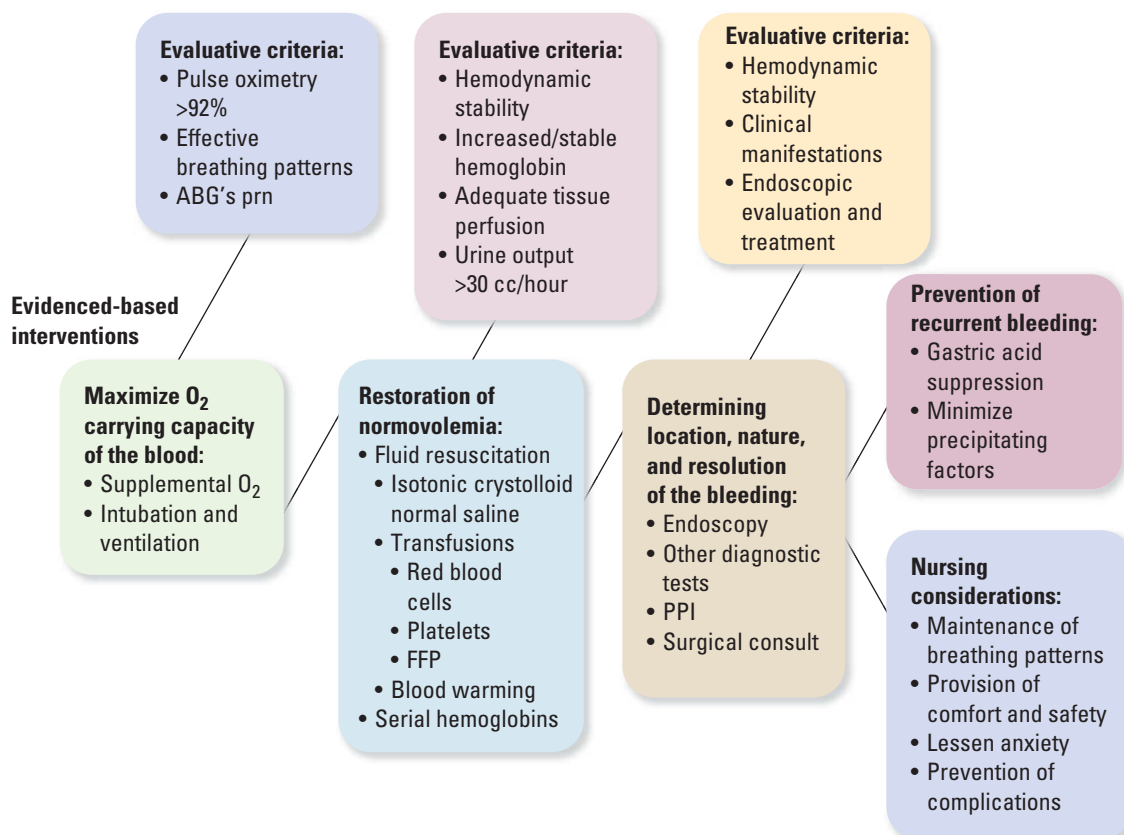
The physical exam should focus on:

- Abdominal assessment:
 - Bowel sounds—Because blood acts as an irritant stimulating peristalsis, patients with a UGI bleed often present with hyperactive bowel sounds. A patient with LGI bleeding is more likely to have normoactive bowel sounds.
 - Abdominal tenderness/pain—May suggest bowel ischemia, perforation, or obstruction

- Evidence of liver disease:
 - Ascites
- Quantity, frequency, and characteristics of emesis/stool:
 - Hematochezia or melena is indicative of active bleeding.
 - Brown stool is unlikely to have active bleeding.
 - Infrequent stools are not likely to have active bleeding.
 - Coffee grounds emesis may indicate slowed or stopped bleeding.

COLLABORATIVE CARE

The nurse works collaboratively with the healthcare team to identify the etiology of the bleeding and patients at high risk for continued or re-bleeding and to resolve continued active bleeding. The etiology of the bleed, risk for re-bleeding, and the presence of continued active bleeding will direct further decisions and interventions. The goal is to identify the source, stop the bleeding, and predict risk for re-bleeding. The nurse will assist with modalities to identify the source of the GI bleeding so that it can be appropriately managed.



Visual Map 13-3 Collaborative Management of GI Bleeding

Nasogastric Tube Placement

There is controversy related to the value of nasogastric tube (NGT) placement. Traditionally, NGT insertion and lavage have been utilized to confirm GI bleeding and distinguish upper from lower bleeding. Studies have failed to demonstrate a benefit to NGT placement and lavage regarding clinical outcomes. A study of 625 patients with GI bleeding noted that those who received NGT and lavage were associated with a shorter time to endoscopy than those who did not receive NGT/lavage; however, there was no difference in mortality, length of stay, surgery or transfusion requirements (Huang, Karsan, & Kanwal, 2011). Nasogastric aspirate can help to validate UGI bleeding, but it does not provide information about the specific cause of the bleeding. It is important to note that the absence of blood in the nasogastric drainage does not eliminate active GI bleeding. The American College of Gastroenterology states that nasogastric lavage is not required in patients with an UGI bleed for diagnosis, prognosis, visualization, or therapeutic effect (Laine & Jensen, 2012).

Erythromycin Administration

Patients with severe bleeding are likely to have a stomach full of blood, which can be problematic for accurate endoscopic evaluation and intervention related to poor visualization. In this situation, erythromycin can be beneficial because erythromycin promotes gastric emptying (Saltzman, 2015b). The nurse can anticipate a single dose of intravenous erythromycin 3 mg/kg may be ordered over 20 to 30 minutes approximately 30 to 90 minutes before endoscopy. In a large meta-analysis conducted by Theivanayagam and colleagues, the results demonstrated statistically significant improvement in gastric visualization, decrease in the need

for repeat endoscopy and a shorter duration of hospitalization in patients who received erythromycin compared to those that did not.

Acid Suppression: Proton Pump Inhibitors

Proton pump inhibitors (PPIs) cross the parietal cell membrane, resulting in irreversible inhibition of gastric secretion of hydrochloric acid by the proton pump. This raises and maintains a high gastric pH. Because gastric acid antagonizes hemostasis by inhibiting clot formation and promoting clot lysis, aggressive PPI treatment to maintain the gastric pH between 6.0 and 6.5 is recommended to promote clot stability and reduce the effect of gastric acid (Saltzman, 2015b). PPI gastric suppression is superior to that of histamine H₂-receptor blockers, therefore, histamine H₂-receptor blockers are not recommended in acute bleeding. Pre-endoscopic administration of a PPI is recommended until the cause of bleeding is established. This practice is thought to decrease the number of patients having high-risk endoscopic lesions (non-bleeding visible vessel, active bleeding, adherent clot). The nurse should anticipate a high dose intravenous bolus (commonly, pantoprazole) followed by a continuous intravenous infusion. PPI therapy should never delay resuscitation efforts. Once the cause has been established, further treatment with PPIs will be determined. After successful therapeutic endoscopy of a high-risk lesion, it is recommended that the high-dose PPI therapy be continued for at least three days. Evidence has shown that high-dose PPIs administered intravenously in patients with high-risk ulcers receiving therapeutic endoscopy results in a decrease in hospital

Commonly Used Medications

Pantoprazole (Protonix)—A Proton Pump Inhibitor (PPI)

Desired Effect: PPI treatment causes an elevation of gastric pH levels which stabilizes blood clots and improves clinical outcomes and thus PPI treatment is recommended for all patients with peptic ulcer bleeding (Saltzman, 2015b).

Nursing Responsibilities:

- Pantoprazole is administered in a high-dose continuous infusion starting with an 80 mg bolus dose followed by 8mg/hour. Current studies are comparing outcomes of continuous infusions versus intermittent dosing and the incidence of re-bleeding. No revised dosing recommendations have been made at the time of this publication. Administration through a dedicated line is preferred, but

a Y site may be used. The line should be flushed before and after administration of intermittent or bolus doses of pantoprazole.

- Protonix is used with caution in patients with severe hepatic disease.
- The nurse reviews evaluative criteria: a stable hemoglobin, absence of signs and symptoms indicating a rebleed, and evidence of a healed ulcer if follow-up endoscopy is performed.

Side and/or Toxic Effects: Protonix has a high safety profile. The nurse monitors the patient for hypersensitivity reactions, headache, diarrhea, abdominal pain, and nausea.

Commonly Used Medications

Polyethylene Glycol Solutions

Desired Effect: It is an osmotically balanced electrolyte solution that passes through the bowel without significant absorption or secretion, thus avoiding significant fluid and electrolyte shifts. Bowel preparation is key to ensure effective visualization and successful colonoscopy.

Nursing Responsibilities:

- It can be administered orally or, if the patient's condition necessitates, it can be administered through an NGT or nasal jejunum tube.
- The nurse can anticipate administering the preparation at a rate of approximately 1 liter every 30 to 45 minutes. The nurse should administer cautiously in patients who are at high risk for aspiration and/or fluid overload.
- It has been safely used in patients with liver disease, renal failure, heart failure, and electrolyte imbalances.

Side and/or Toxic Effects: The disadvantage is that relatively large volumes must be given to get a cathartic effect. Patient intolerance evidenced by nausea and vomiting are common side effects.

Metoclopramide (Reglan)

Desired Effect: Metoclopramide is a dopamine antagonist gastroprokinetic. It increases the amplitude of gastric contraction and peristalsis of the duodenum and jejunum. Metoclopramide administration has also been used in conjunction with erythromycin to improve gastric emptying and improve visualization during endoscopy. This can be an adjunct to a polyethylene glycol solution to help reduce nausea, vomiting, and bloating and promote overall tolerance of the preparation. However, it does not improve colonic cleansing and some studies indicate no improvement in patient tolerance.

Nursing Responsibilities: Metoclopramide (Reglan) 10 mg IV may be administered at the start of the prep. Monitor vital signs and promptly report an irregular heart rate or increase in blood pressure.

Side and/or Toxic Effects: Mild sedation, fatigue, restlessness, diarrhea, extrapyramidal symptoms (acute dystonic reaction), neuroleptic malignant syndrome. Tardive dyskinesia has been associated with long-term use.

length of stay, rebleeding rate, and need for blood transfusions (Saltzman, 2015b).

Overall, PPIs have minimal side effects and few significant drug interactions. Conflicting evidence exists linking PPI use with *clostridium difficile* infection, pneumonia, and osteoporosis-related factors (Johnson & Oldfield, 2013). These problems correlate to the long-term safety profile of PPIs and current recommendations state that the benefits of PPIs outweigh the risks for those patients experiencing an acute UGI bleeding episode. The decision to continue the PPI after the three days as well as after discharge will be individualized. This will be based upon endoscopic findings and treatment, precipitating factors, risk for re-bleeding, and current medication regime. Research continues to compare the efficacy of different PPIs, dosing options, routes of administration, and treatment duration.

Bowel Preparation

A bowel preparation is recommended prior to a colonoscopy. Although the procedure may be performed without a preparation, because blood is considered a cathartic, a cleansed colon allows for a safer procedure and better chance at visualization. There is an increased chance of perforation with an unprepared colon secondary to decreased visibility. If the source of bleeding is strongly

suspected to be from the rectal-sigmoid area, a sigmoidoscopy without a preparation is reasonable.

The nurse anticipates that a rapid high-volume (4–6 liters) preparation with a balanced electrolyte solution will be administered over 3–4 hours to cleanse the colon. After administration of the preparation, the nurse must frequently assess the characteristics of the stool for clearing. Ideal timing for an urgent colonoscopy is within 1–2 hours of stool clearing. Promotility medications such as metoclopramide (Reglan) may be ordered to decrease nausea and improve tolerance of the preparation.

COLLABORATIVE CARE

Endoscopy is a procedure that uses a flexible fiber-optic endoscope to directly visualize the inside of a hollow organ or cavity. An upper endoscopy, sigmoidoscopy, colonoscopy, and push enteroscopy are all examples of endoscopic procedures that inspect the lumens of the GI tract. The difference is in the specific area to be examined and the intubation orifice. Endoscopy can provide real-time images that are viewed as a video on the monitor in the procedure room. Still photos of areas can be taken. These procedures can be diagnostic, prognostic, and therapeutic.

Ideally, patients should be resuscitated and stabilized prior to endoscopy. However, urgent endoscopy may be considered for patients experiencing ongoing bleeding and hemodynamic compromise. Such patients will need cardiovascular and respiratory support. Patients triaged to intensive care will likely have the procedure performed in the ICU.

Upper Gastrointestinal Endoscopy

An upper GI endoscopy (**esophagogastroduodenoscopy [EGD]**) involves oral intubation with a flexible endoscope to visualize the esophagus, stomach, and proximal duodenum. A push enteroscopy is similar to an upper endoscopy except that a longer scope is introduced and pushed into the small intestine so the distal duodenum and proximal portion of the jejunum are visualized. Early upper endoscopy, within 24 hours for high-risk patients, is associated with improved outcomes, reduced resource utilization, and best practice in terms of allowing for safe and timely discharge. A routine second-look EGD is not recommended, however, when blood obscures the source of bleeding a second endoscopy may be necessary (Saltzman, 2015a).

Colonoscopy

A colonoscopy involves the insertion of the endoscope into the anus to examine the colon or large intestine from the rectum to the ileocecal valve. It is also possible to view the distal portion of the ileum. The bowel wall can be observed for bleeding sites, tumors, and lesions during the insertion and withdrawal. During the procedure, air is introduced into the colon through the endoscope to expand the lumen to facilitate visualization and navigation.

Sigmoidoscopy

A sigmoidoscopy involves the inspection and visualization of only the rectal-sigmoid area of the colon. If bleeding is highly suspected to be coming from the rectum or sigmoid area, an unprepped sigmoidoscopy can be performed. If the source is not revealed, the patient will commonly be prepped for a colonoscopy.

Purpose

The purpose of an endoscopy in patients with an acute GI bleed is to establish the site and etiology of the bleed, to allow for endoscopic assessment to determine whether the site has high-risk characteristics for re-bleeding (risk stratification/endoscopic stigmata), and to perform therapeutic interventions to resolve active bleeding and prevent re-bleeding.

Once the site and source have been determined, there are multiple therapeutic interventions that can be

implemented to achieve hemostasis (stoppage of bleeding). The choice is influenced by the endoscopist's preference and experience and the location and characteristics of the bleeding lesion. The interventions, which are performed by either an endoscopy nurse or technician, can be employed as monotherapy or in combination depending on the nature of the bleed.

Categories of Therapeutic Interventions

There are three main categories of therapeutic interventions: injection therapy, thermal coagulation, and mechanical techniques.

Injection Therapy

While directly visualizing the site with an endoscope, an injection needle is used to inject a solution into the desired location. The site may be injected with a variety of agents. The agents may sclerose (harden), vasoconstrict, or cause a tamponade effect. The most commonly used agent is epinephrine. Dilute epinephrine (1:10,000) is injected to create a combination of vasoconstriction and vascular tamponade. Because re-bleeding rates are high with injection therapy as a single intervention, this method is typically combined with thermal or mechanical therapy.

Thermal Coagulation

There are multiple types of thermal therapy. Some work by coming in physical contact with the bleeder and applying pressure followed by thermal coagulation of the vessel (bipolar electrocoagulation and heater probes). These are typically used in acute bleeds. No one single method has been found to be superior.

Others work by noncontact thermal energy. Examples would be argon plasma coagulation (APC) and laser therapy. These noncontact methods are not usually used in an acute bleed situation.

Mechanical Techniques

Endoclips (hemoclips) involve the application and deployment of a clip that achieves hemostasis by compressing the tissue together. Its action is similar to that of a surgical stitch. Depending on the situation, multiple clips may be applied. Frequently, this method is combined with an epinephrine injection. Another technique that may be used for variceal bleeding is band ligation.

New Endoscopic Technologies

Several new endoscopic therapies have emerged to assist with establishing hemostasis for UGI and LGI bleeding. Such therapies include application/injection of tissue

adhesive or fibrin glue, over the scope clips, endoscopic suturing, radio frequency ablation, cryotherapy, and endoscopic ultrasound-guided angiotherapy. These can be used for both initial treatment and rescue therapy. Informed consent must disclose the experimental nature of some of these treatments, which are in various stages of research and use. Success is influenced by appropriate lesion selection and the expertise of the endoscopist. Long-term clinical efficacy and safety have not yet been established for many of these therapies.

Indications and Expected Outcomes

An EGD is indicated for any patient with the diagnosis or suspected diagnosis of UGI bleed. The timing of the procedure is critical, depending on the significance of the bleed and the patient's hemodynamic status. Patients considered high risk based on clinical criteria should have an endoscopy within 24 hours. Low-risk patients may have an endoscopy scheduled as an outpatient.

An urgent colonoscopy is usually the initial diagnostic study of choice for locating and treating an LGI bleed. *Urgent* generally means within 8 to 24 hours after admission following an adequate preparation as discussed. A reduced length of hospital stay is correlated with early colonoscopy. The detection rate for finding an active or recently bleeding diverticular lesion is low. A colonoscopy is contraindicated in patients in shock or if there is suspicion of an obstruction or perforation. Abdominal x-rays are obtained to rule out these complications prior to the colonoscopy.

The expected outcome of endoscopy is that the bleeding site will be located and treated appropriately to prevent re-bleeding. If blood obscures the lining, it may be impossible to determine the site and etiology.

The nurse should expect to see no signs or symptoms of re-bleeding evidenced by:

- Stable vital signs
- Adequate urine output (at least 30 mL/hour)
- Negative orthostatic signs
- Stabilization of hemoglobin
- Baseline mental status

NURSING ACTIONS

If the patient is managed in the ICU, the endoscopy team will likely perform the procedure in the unit. The critical care nurse will have pre-, intra-, and post-procedural responsibilities. The majority of the procedures are performed with moderate/procedural sedation administered by the endoscopy nurse. In the critical care setting, especially if the patient is intubated on a ventilator, the patient may already be receiving sedation. In this situation, the critical care nurse may be accountable for continued sedation. It is essential

that the endoscopy nurse and the critical care nurse have ongoing communication related to the patient's condition and their roles and responsibilities.

Pre-procedural nursing responsibilities of the critical care nurse:

- Maintains NPO status ideally for 6–8 hours prior to the procedure (depending on urgency, this may not be possible)
- Ensures informed consent
- Removes dentures for EGD patients
- Monitors baseline vital signs including temperature and oxygen saturation.
- Provides patient/family education
- Provides interventions for better visualization such as administration of erythromycin and/or metoclopramide, and assists patient with bowel preparation
- Consults the endoscopist and respiratory therapist if the patient is mechanically ventilated for temporary adjustment of the ventilator settings to maximize oxygenation during sedation

Intraprocedural nursing responsibilities of the critical care nurse:

- May be responsible for administering continued sedation
- Continues to monitor vital signs, oxygen saturation and some facilities require capnography or end tidal CO₂ monitoring during sedation
- Continues interventions to maintain hemodynamic stability
- Participates in a universal protocol “time-out,” which will verify the correct patient and procedure immediately prior to the procedure
- Assists the endoscopy team as needed

Post-procedural nursing responsibilities of the critical care nurse:

- If the patient is not intubated, maintains recovery position (left lateral decubitus) to protect the airway until the patient is fully awake:
 - EGD patients may have a topical anesthetic sprayed to the throat area, which may impair swallowing. Once a common practice, many endoscopists are no longer routinely administering a topical anesthetic.
 - In addition to airway protection, colonoscopy patients benefit from the left lateral decubitus position because it facilitates the passage of air that was inserted during the procedure.
- Monitors vital signs, including temperature and oxygen saturation and level of pain and consciousness, until the patient returns to baseline (typically every 10 to 15 minutes for 30 minutes to an hour, then per ICU protocol or more frequently depending on acuity).

- Colonoscopy patients require abdominal assessments for distention. The air that is purposely introduced commonly causes distention. In most cases, this distention is resolved by having the patient bear down and push in an effort to pass gas.
- Monitors for potential complications:
 - Gastrointestinal blood loss (rebleeding)
 - Perforation of the GI tract and subsequent release of GI contents create a full-thickness injury to the wall. Endoscopy increases the risk for perforation. The incidence increases with the complexity of the procedure thus is more common during therapeutic procedures. Other causes include trauma, obstruction, ingestion (caustic, foreign bodies), violent retching/vomiting, IBD, appendicitis, PUD, diverticular disease, ischemia (CVD), infectious disease, neoplasms, and connective tissue diseases. Pain is the most significant clinical manifestation. The location and severity depends upon the organ(s) affected. Diagnosis includes a thorough history, physical exam but relies on imaging that demonstrates the presence of free air outside the GI tract. Management includes IV therapy, maintaining NPO status, broad spectrum antibiotics and evaluation for surgical intervention versus conservative management. Clear indications for surgical management include abdominal sepsis/peritonitis, bowel ischemia, and complete bowel obstruction.
 - Aspiration
 - Adverse reaction to procedural sedation medications (allergy, drug interactions, respiratory depression and hypotension). Patients are commonly given an intravenous (IV) narcotic and a short-acting benzodiazepine, typically midazolam (Versed) and sublimaze (Fentanyl).

Other Diagnostic Tests

In the event that colonoscopy cannot be performed or does not yield a bleeding site, other diagnostic options can assist in locating the LGI site, including angiography, radionuclide imaging, and a helical computer tomography scan. The use of radiographic technologies can be limited because they require active bleeding at the time of the exam. A colonoscopy is recommended prior to radionuclide imaging and angiography because of its ability to more accurately diagnose and its ability to treat. Angiography is reserved for patients who cannot have a colonoscopy because of massive ongoing bleeding, or when colonoscopy has failed to reveal a source. If both the colonoscopy and upper endoscopy are negative, patients who are hemodynamically stable should have a

capsule endoscopy to evaluate the small bowel. If resources are available, percutaneous embolization (various agents can be used to embolize the vessels feeding the bleeding lesion) can be considered as an alternative to surgery especially in patients who are a high surgical risk.

Surgical Consult

The role of surgery in the emergent management of UGI bleeding has diminished; however, it is the most definitive and may be the final option for some bleeding lesions. The majority of patients with UGI bleeding related to peptic ulcer disease (PUD) are managed with resuscitation, medical, and endoscopic interventions. Patients must be evaluated very carefully for surgical management, especially those with severe comorbidities that will affect surgical outcomes. Because surgery has a high morbidity and mortality, it is generally reserved for patients whose bleeding is not controlled by endoscopic treatment, or where massive bleeding prevents visualization and treatment. A second attempt at endoscopic therapy is usually recommended prior to surgical consideration. Surgical consults should be considered early for critical patients at high risk for a failed endoscopic therapy.

In extreme cases where the source of bleeding cannot be established, a laparotomy may be indicated. Surgical intervention for a bleeding peptic ulcer may involve ligation of the vessel and closure of the ulcer.

Surgery for LGI bleeding is associated with increased complications and mortality. Patients with continued life-threatening hemorrhage requiring more than 4 units of RBCs in a 24-hour period, or 6 units of blood during the same hospitalization, failed endoscopic and other management, or experience multiple recurrent bleeding episodes should be considered for surgery (Cagir, 2014). Depending on the acuity of the patient, every effort should be made to accurately localize the bleeding site prior to surgery to avoid increased mortality and morbidity. If the bleeding is localized, a segmental resection can be performed as opposed to a subtotal colectomy for patients with continued bleeding and undiagnosed site. A subtotal colectomy is associated with higher morbidity than a segmental colectomy. Whenever possible, it is better to stabilize the patient and perform surgery on an elective basis. Emergent surgery is associated with more complications.

Identification and Prevention of Recurrent Bleeding

The majority of patients with UGI bleeds caused by PUD and LGI bleeds secondary to colonic diverticula will not re-bleed during the hospitalization. However, certain patients with an initial severe bleed are at high risk for re-bleeding. Determining risk for re-bleeding can guide triaging the

patient to the appropriate level of care. The absence of these high-risk characteristics can identify patients who can be safely discharged as opposed to those who need further assessment and management.

Risks for Recurrent Gastrointestinal Bleeding

The nurse reviews the patient's clinical course, laboratory studies, and endoscopic results to identify risk factors for re-bleeding:

- Older age
- Comorbid disease states
- Hemodynamic instability
- Coagulopathy/anticoagulants
- Endoscopic diagnosis/stigmata (active arterial bleeding, visible vessel, adherent clot)

The patients who are identified as high risk for re-bleeding will require continued close nursing assessment for re-bleeding, including:

- Hemodynamic/fluid volume status
- Mental status/restlessness
- Pain/discomfort
- Presence of hematemesis, melena, hematochezia
- Serial hemoglobin, renal function tests

COLLABORATIVE CARE

The nurse works collaboratively with the healthcare team to prevent re-bleeding. Management efforts should be directed at healing the ulcer/lesion and eliminating precipitating factors.

Acid Suppression: Proton Pump Inhibitors

As mentioned earlier, high dose intravenous PPIs are administered in the acute phase of UGI bleeding. Once the cause, the presence of active bleeding, and other high-risk stigmata have been established, further treatment with PPIs will be determined. Patients may be transitioned to intermittent IV doses or an oral PPI and discharged on a PPI for a duration dictated by the underlying etiology.

Elimination of Precipitating Factors

To prevent recurrent bleeding, it is essential that patients be assisted to eliminate the precipitating factors. Patients whose GI bleeding has been attributed to the use of NSAIDs should be advised to discontinue these medications. Counseling related to safer alternative medications

should be provided. If the NSAIDs need to be continued, a maintenance dose of PPI should be continued to decrease risk.

The gastrointestinal risk of aspirin and the cardiovascular benefit of aspirin often present a dilemma. For patients at risk for cardiovascular complications taking low-dose aspirin, aspirin therapy should be restarted once the cardiovascular risk outweighs the risk for bleeding. Generally, the higher the dose of aspirin, the higher the risk for ulcers and re-bleeding. Aspirin plus a PPI is recommended for preventing ulcer reoccurrence and re-bleeding. Patients receiving clopidogrel (Plavix) are also at a high risk for re-bleeding. Earlier studies investigating the concomitant use of clopidogrel with a PPI, notably omeprazole, indicated a decrease in the antiplatelet effect. Evidence currently does not support significant interactions between antiplatelet agents and PPI's (Mourkarbel & Bhatt, 2012). With the increasing number of patients on some type of antithrombotic therapy, questions related to management during and after bleeding are complicated and may require a cardiology consult for high-risk individuals.

All patients presenting with a UGI bleed should be tested for *Helicobacter pylori* (*H. pylori*), and if positive should receive eradication therapy and confirmed eradication. Additionally, *H. pylori* infection and NSAID ingestion act synergistically to cause ulcers. There is evidence that eliminating *H. pylori* is associated with higher healing rates and decreased ulcer reoccurrence and bleeding complications. There are a variety of eradication treatment regimens. Antimicrobial agent(s) are typically administered with a PPI. It is not clear if any one treatment regimen is superior. Cost, side effects, and simplicity of administration are considered. Patient compliance significantly influences outcome. Therapy can be started immediately or during a follow-up visit. The goal is to promote compliance by educating the patient related to the medication action and administration regimen, the importance of the treatment, and to prevent further complications.

NURSING CARE

The nurse is responsible for ensuring the patency of the patient's airway, providing adequate nutritional intake when appropriate, maintaining patient comfort and safety, and relieving patient and family anxiety.

Maintaining Safety

Maintenance of airway, breathing, and circulation are the immediate nursing priorities during the initial assessment and resuscitation management. Respiratory compromise can occur in patients with both a UGI and LGI bleed related to significant blood loss, potential or actual hemorrhagic

shock resulting in decreased oxygen delivery to all vital organs, and changes in mental status. In addition, hemorrhage can worsen preexisting pulmonary disease.

Patients presenting with a UGI bleed with significant blood loss, potential mental status changes, and hematemesis may have difficulty protecting their airway and are at an increased risk for aspiration. Aspiration is an avoidable complication that significantly affects morbidity and patient outcomes. With vigilant nursing assessment and monitoring, these high-risk patients should be readily recognized and electively, rather than emergently, intubated in a controlled setting. The nurse will frequently monitor the patient's:

- Respiratory status: ability to protect the airway, continuous oxygen saturation, breathing patterns, arterial blood gases, adventitious lung sound associated with pulmonary edema
- Hemodynamic status: stability of vital signs
- Level of consciousness: mental status, ability to follow commands, lethargy
- Hematemesis: presence, frequency, amount

The nurse will:

- Implement aspiration precautions (head of bed elevated; if unable, left lateral decubitus position)
- Confirm the presence of necessary resuscitation/suctioning equipment
- Administer oxygen using the appropriate delivery system and flow rate to maintain pulse oximetry 95% or greater

The expected outcomes for all patients presenting with a GI bleed is that the airway will be protected and adequate breathing patterns will be maintained.

Patients should be routinely assessed for fall risk, utilizing a standardized risk assessment tool. Certain medications, unstable vital signs, positive orthostatic signs, syncope, and altered mental status commonly occur with GI bleeding and will increase a patient's fall risk potential. Fall risk protocols should be implemented.

Providing Nutrition

The patient experiencing an UGI bleed will typically remain NPO until undergoing the necessary diagnostic tests to identify and treat the bleeding, most often an upper endoscopy. NPO status is maintained for visualization and safety. The patient will receive procedural sedation and possibly a topical anesthetic to the pharynx. After endoscopic evaluation, patients considered low risk for re-bleeding may eat immediately. Studies have shown that the timing of feeding for this low-risk group does not influence the hospital course. Patients scheduled for a colonoscopy will be NPO with the exception of the laxative that must be ingested to purge the colon for visualization.

The decision to feed patients with LGI bleeding or patients not in the low risk for UGI re-bleeding category will be made according to the individual patient's clinical status, evidence that bleeding has subsided, and the potential for repeated endoscopy or other procedures or surgery. Patients likely to remain unable to take or tolerate oral feeding for an extended period should have a nutritional consult, and be evaluated for enteral or parenteral nutrition.

Enhancing Comfort

The majority of upper and lower GI bleeding is painless. The nurse will routinely assess and treat pain per pain management protocol. However, patients are likely to experience discomfort related to invasive monitoring devices, NGT insertion, and diagnostic tests. The discomfort associated with tests and invasive monitoring can often be lessened by basic nursing care through assisting with patient positioning, hygiene (mouth, nares care, bathing, back care), and patient education. It is important for patients to be aware of the rationale for invasive monitoring devices and any discomfort associated with the procedures. Often the discomfort is short term, and preparing the patient can help the patient better deal with the discomfort.

If nursing care interventions are not successful in easing the discomfort, the nurse should collaborate with the physician related to the nature of the discomfort and the possibility of medications or other interventions that may help with management.

Fostering Patient and Family-Centered Care

The patient/family often experience different degrees of anxiety related to the stress of experiencing an acute crisis that may be life threatening; has totally disrupted their roles and responsibilities; has put them in an unfamiliar environment with a loss of control; has potentially caused pain and discomfort related to invasive procedures and therapeutic treatments; and has created fear, especially of the unknown: diagnosis, prognosis, treatments.

The critical care nurse must be sensitive to the patient/family's fears, needs, and coping skills and implement interventions to decrease anxiety. The nurse should empower the patient/family to take an active role in the patient's care. In addition to compassionate care and realistic hopefulness, the nurse should encourage questions and participation in decision-making.

All members of the healthcare team should provide education and consistent information related to the diagnosis, treatments, and overall plan of care based on the patient/family's readiness and willingness to learn. Barriers to learning (cognitive development, language, culture, etc.) should

be addressed appropriately. The patient/family should be evaluated for appropriate referrals: pastoral care, social services, case management, tobacco cessations programs and alcohol use disorder counseling. The nurse should routinely assess for evidence of decreased anxiety by verbal and non-verbal communication and evaluating physical, behavioral, emotional, and cognitive signs and symptoms.

Essential for Patient-Centered Care

Being a patient in an ICU is very stressful for both the patient and the family. It is imperative that nurses take the initiative to make sure that the patient and/or family are adequately informed about the patient's condition and treatment plans. No matter what the prognosis, interaction with the multidisciplinary team can really make a difference in the patient's and family's coping skills. Too often, the patient has no continuity in nursing staff; therefore, this important line of communication is fragmented (see "Safety Initiative").

Prevention of Complications

Prevention of complications is essential for promoting positive patient outcomes. Acute GI bleeding needs to be systematically assessed and treated. Aggressive resuscitation and stabilization, identifying the source, stopping and treating the bleeding, and preventing recurrent bleeding are the fundamentals of care. Depending on the rate of bleeding and the patient's response to treatment, hypotension, and potential shock states can result from acute blood loss. Vigilant assessment, interventions, and evaluation of the patient's response to the interventions are critical in

preventing shock and the consequences of shock. The nurse must monitor for evidence of:

- Myocardial ischemia/infarction
- Cerebral ischemia/thrombosis
- Respiratory insufficiency/failure
- Acute renal injury
- Hepatic failure
- Disseminated intravascular coagulation
- Sepsis
- Multisystem organ failure (MSOF)

Invasive monitoring devices and procedures, the hospital environment, surgery, and immobility increase the patient's risk for developing infections. Essential nursing interventions to minimize the development of infections are implemented (see Chapter 18  for the discussion on sepsis).

Patients who have had surgery need meticulous postoperative care and monitoring. Patients with lower GI surgery are especially prone to complications. During the recovery period the nurse must closely observe for the following common early postoperative complications:

- Bleeding
- Mechanical small bowel obstruction
- Ileus
- Intra-abdominal sepsis
- Localized/generalized peritonitis
- Wound infection or disruption
- Thrombophlebitis

Bariatric Considerations

Obese patients are more likely to have UGI symptoms and an increased prevalence of UGI pathology. *Helicobacter pylori* infection is present in 23% to 70% of patients scheduled for bariatric surgery (ASGE, 2015).

Obese patients are more likely to have inadequate bowel preparations, and often require a more aggressive bowel preparation regime.

Administering sedation to obese patients undergoing endoscopic procedures requires extreme caution. Obese patients have an increased risk of airway difficulties and obstructive sleep apnea. Positioning the patient in a left lateral decubitus position may further compromise airway and respiratory status. Additionally, patients with sleep apnea have an increased risk of developing cardiovascular complications, oxygen desaturation, and hypercapnic episodes related to sedation.

One complication of bariatric surgery is the development of a marginal or stomal ulceration at the gastrojejunal anastomosis site, most often on the intestinal side of the anastomosis. This is typically seen 1 to 6 months after surgery.

Endoscopy plays an important role in the diagnosis and treatment of the complications from bariatric surgery. However, depending on the specific bariatric surgery, postsurgical anatomy can be very challenging for the endoscopist.

Obesity is a risk factor and a constant parameter in many of the severity scales for acute pancreatitis. The growing obesity epidemic has likely influenced the increase in the number of cases of pancreatitis.

Nursing Diagnoses

Gastrointestinal Bleeding

- Deficient fluid volume related to active blood loss
- Acute pain related to mucosal irritation
- Nausea related to gastrointestinal bleeding
- Anxiety related to health status

Pancreatitis

- Acute pain related to edema of the pancreas
- Deficient fluid volume related to severe fluid shift
- Imbalanced nutrition less than body requirements related to inability to ingest and digest foods

Pancreatitis

Acute Pancreatitis

Acute **pancreatitis** (AP) is a sudden nonbacterial inflammatory process of the pancreas that occurs from the activation of digestive enzymes found inside the acinar cells that compromise the pancreatic gland, nearby tissues, and other organs (Cruz-Santamaria, Taxonera, & Giner, 2012). It is responsible for 280,000 hospital admissions per year and inpatient cost exceeding 2.5 billion dollars in the United States (Peery, Dellon, & Lund, 2012). The incidence has been increasing globally. Recent studies indicate that between 4.9 and 73.4 cases per 100,000 occur worldwide (Tenner, Baillie, DeWitt, & Vege, 2013). The clinical course ranges from a mild interstitial self-limiting illness to a severe life-threatening disorder. Initial treatment is directed at identifying and correcting the cause. Gallstone disease and excessive alcohol use are the most common causes of

acute pancreatitis, accounting for an estimated 80%. Management is supportive and emphasizes aggressive fluid management to prevent complications, most notably hypovolemia and hypoxemia. Pancreatic necrosis and the presence, timing, and duration of organ failure influence morbidity and mortality. The majority of the patients with severe pancreatitis without organ failure survive, whereas those with multisystem organ failure (MSOF) have an increased risk of death. The risk of death doubles when patients have both infected necrosis and persistent organ failure (Wu and Banks, 2013). Overall mortality has decreased because of improved intensive care management and supportive care.

Anatomy and Physiology Review

The Pancreas

The pancreas is an elongated, lobulated gland that lies behind the stomach in the retroperitoneal space and extends from the duodenum to the spleen (♦ Figure 13-4). It is an accessory organ to the GI tract with both exocrine and endocrine functions. In addition, the pancreas has both a cellular and a ductal system.

The pancreas is divided into three segments: the head, which lies over the vena cava in the C-shaped curve of the duodenum; the body, which lies behind the duodenum and extends across the abdomen behind the stomach and across the spine; and the tail, which is situated under the spleen.

Cellular Systems of the Pancreas. Exocrine cells make up 98% to 99% of the pancreatic tissue, and are responsible for the production of pancreatic juices and digestive enzymes

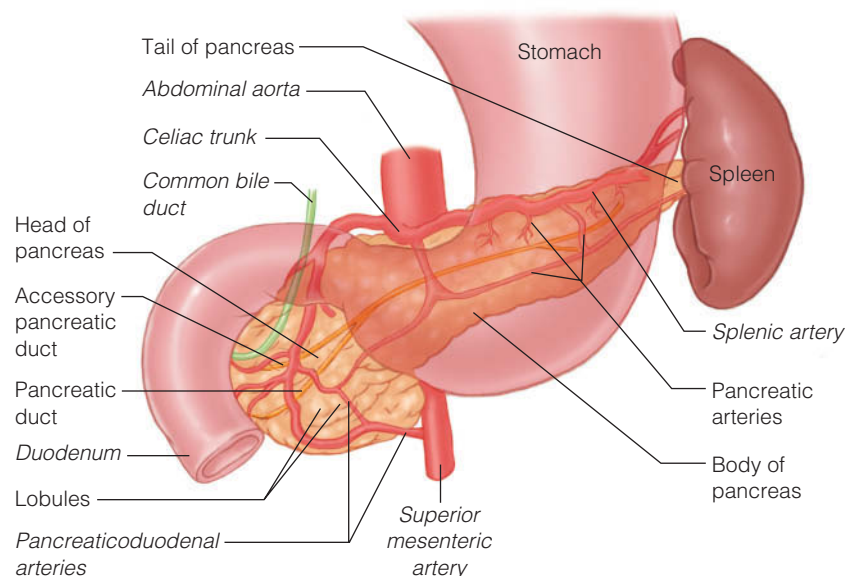


Figure 13-4 The pancreas. The gross anatomy of the pancreas. The head of the pancreas is tucked into a C-shaped curve of the duodenum that begins at the pylorus of the stomach.

that assist with the breakdown of nutrients in the intestines to facilitate absorption.

Acini cells—The functional cells of the exocrine pancreas responsible for producing pancreatic enzymes

Pancreatic juices—Made up of water and bicarbonate, which neutralizes the acidic chyme, thus protecting the intestines from acid damage

Pancreatic enzymes

The major types of pancreatic enzymes are:

Amylase—Responsible for breaking down certain starches

Lipase—Responsible for breaking down certain complex fats

Proteases—Responsible for breaking down proteins

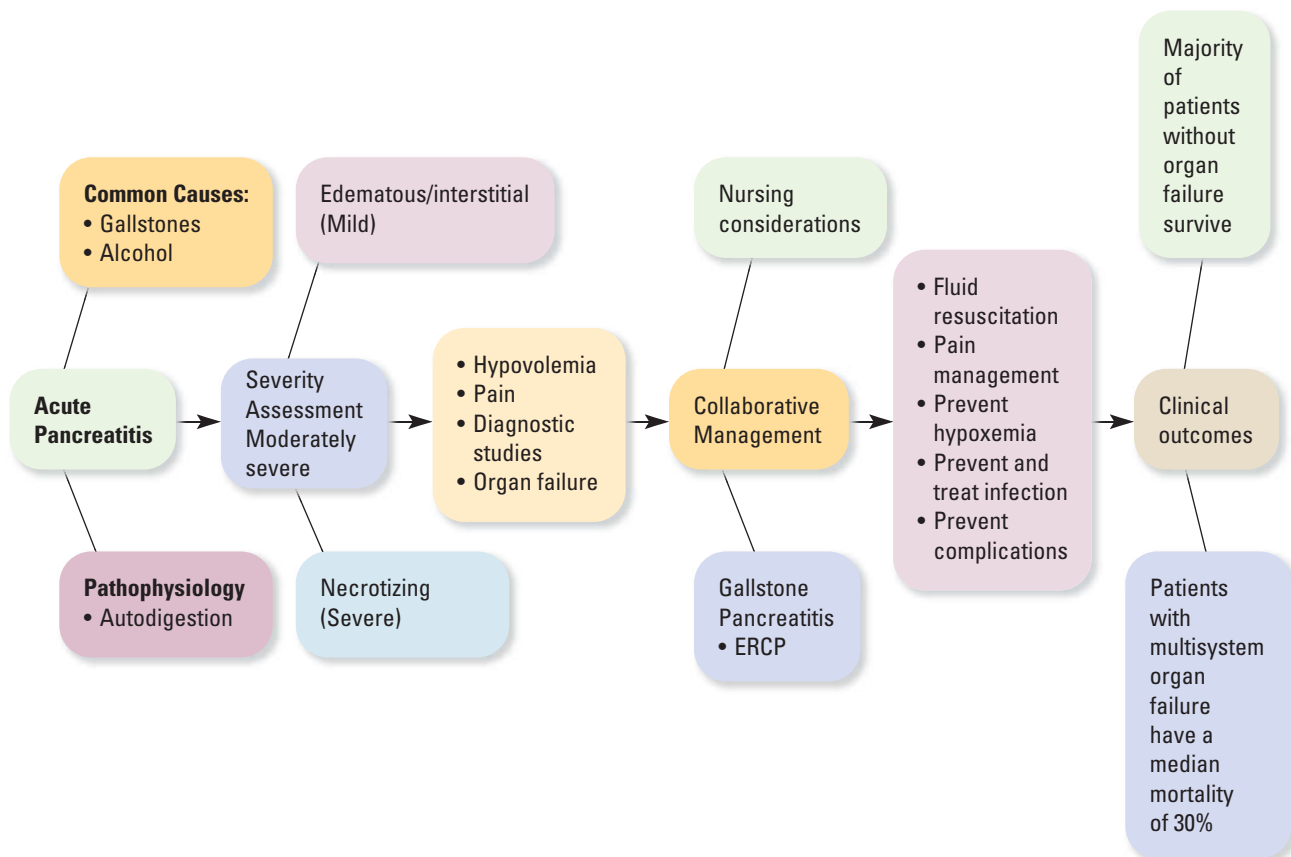
These enzymes are stored within the pancreas as proenzymes (inactive enzymes). Proenzymes serve to protect the pancreatic cells and tissues from the destructive effects of their own products. Trypsinogen, a protease and precursor of trypsin, is activated in the duodenum and is a catalyst for activating other enzymes. Trypsin inhibitor is produced by the pancreas to prevent enzymes from being activated before they reach the duodenum. Should these enzymes become activated prior to reaching the duodenum, autodigestion of the pancreatic tissue can occur.

Endocrine cells make up 1% to 2% of the pancreatic tissue. They are found mostly in the tail of the pancreas in an area known as the islets of Langerhans. They secrete hormones, primarily insulin, directly into the bloodstream.

Pancreatic Ductal System. The main pancreatic duct, the duct of Wirsung, runs the whole length of the pancreas from left to right. It joins the common bile duct at an area known as the ampulla of Vater. The papilla of Vater is a nipple-like protrusion where the common bile duct and the pancreatic duct penetrate the duodenum. At the point where the common bile duct opens into the duodenum is the sphincter of Oddi, a circular muscle constricting the opening. This sphincter relaxes to promote drainage and constricts to prevent reflux.

The Patient with Pancreatitis

Patients with AP are usually critically ill. For optimum patient outcomes, they require care by a multidisciplinary team familiar with the essential elements of care for the patient with pancreatitis as well as the potential complications of the disease.



Visual Map 13-4 Overview of Acute Pancreatitis

Predisposing Factors and Causes of Acute Pancreatitis

The most common risk factors for AP include gallstone disease and excessive alcohol use. Other less common causes include infections, medications, toxins, developmental abnormalities, autoimmune disorders, mechanical obstruction of the ducts, hypertriglyceridemia, hypercalcemia, trauma, heredity, vascular abnormalities, and idiopathic causes. Multiple studies have concluded that smoking is an independent risk factor in the development of AP and effects may synergize with alcohol (Yadav and Lowenfels, 2013). Determination of the cause is critical for decision making and directing immediate interventions. An example of a causal event needing prompt intervention is the presence of common bile duct stones. Such patients are evaluated for stone removal within the first 24 hours of admission, and a possible gallbladder removal in the near future.

The exact mechanism as to how the different predisposing factors induce pancreatitis is uncertain. General consensus regarding pathogenesis is autodigestion. The acinar cells are damaged, causing inappropriate activation of trypsinogen to trypsin. Trypsin activates a cascade of other enzymes that begin digestive functions in the pancreas, resulting in inflammation and tissue damage. The normal defense and inhibitory mechanisms are overwhelmed by the large amounts of activated enzymes.

The release of inflammatory mediators causes increased vascular permeability with varying degrees of edema, hemorrhage, and necrosis. For the majority of the patients, the inflammatory response is self-limiting. However, 10% to 20% of the patients will experience increased local inflammation, resulting in toxic enzymes and mediators entering the systemic circulation. The inflammatory response goes beyond the pancreas to a systemic inflammatory response syndrome (SIRS), which can contribute to pancreatic necrosis and result in multisystem organ dysfunction.

Determination of the Severity of Pancreatitis

The diagnosis of AP is based on careful consideration of medical history, differential diagnoses, clinical assessment, and laboratory and radiographic imaging studies. Once the diagnosis is made, further studies may be conducted to find a cause and assess for complications. Early diagnosis is essential for halting the disease progression and preventing complications. Accepted criteria for the clinical diagnosis of AP require the presence of two out of three features:

- Characteristic epigastric or left upper quadrant pain that may radiate to the back, chest, or flank and is often severe
- Serum amylase and/or lipase greater than or equal to three times the upper limit of normal. The magnitude

or duration of elevation of amylase and lipase levels do not correlate with the severity of the attack or patient outcomes.

- Serum amylase: rises within 12 hours and is typically elevated for 3 to 5 days. It is a nonspecific test because other disorders can cause an elevation, though they typically do not elevate to such levels. It has no correlation with severity or patient outcomes.
- Serum lipase: rises within 24 to 48 hours and is typically elevated for 14 days. It is a more specific and sensitive test than amylase because it will stay normal in some nonpancreatic disorders that would increase the amylase. Because it rises later and stays elevated longer, it is a good marker for those who linger at home and present later.
- If the enzyme limits are below three times the upper limit and doubt exists about the diagnosis, advanced abdominal imaging may be performed to look for characteristic findings of AP (Wu and Banks, 2013).

Once the diagnosis of AP is made, establishing the severity and identifying those patients likely to develop severe disease is critical to positive patient outcomes. The 2013 American College of Gastroenterology guidelines for the clinical management of AP revised the definitions of AP and clearly identified two phases. The new definitions reflect a more detailed system emphasizing severity and distinct time frames. Early phase AP is within 1 week and is characterized by the systemic inflammatory response system (SIRS) and/or organ failure. Late phase AP is greater than 1 week and is characterized by local complications such as peripancreatic fluid collections, pancreatic and peripancreatic necrosis, and pseudocysts as well as persistent organ failure and infected necrosis. They have also improved upon the severity of illness grading system and included a new category (Tenner et al., 2013).

Mild AP—characterized by the absence of organ failure and local or systemic complications.

- Most do not require pancreatic imaging.
- Are usually discharged within 3 to 5 days

Moderately severe AP—characterized by no organ failure or transient organ failure (< 48 hours) and/or local complications.

- Frequently have extended length of stay
- May exacerbate comorbidities
- Have lower mortality rates than the severe form

Severe AP—characterized by persistent organ failure (>48 hours) that may involve one or multiple organs.

- Most have pancreatic necrosis

- 30% mortality rate
- Risk of in hospital death doubles with both organ failure and necrosis (Tenner et al., 2013)

It is crucial to recognize organ failure when determining the severity of AP so the patient is triaged to the appropriate level of care. Patient management in the initial 12 to 24 hours is of paramount importance because the highest incidence of organ dysfunction occurs during this time (Wu & Banks, 2013). In the most severe cases, the pancreas becomes hemorrhagic.

Multiple scoring systems, each with its advantages and disadvantages, are available and recommended in predicting the severity. These scoring systems have varying degrees of complexity and inaccuracy and often are not consistently used. On admission, risk factors of severity include:

- Older age (mortality increases in patients 60 years or greater)
- Obesity (BMI > 30)
- Hypovolemia
- Altered mental status
- Comorbidities (cancer, heart failure, chronic kidney and liver disease)
- Pulmonary infiltrates or pleural effusions
- SIRS
- Organ failure

In severe acute AP, decreased circulating volume presents as hemoconcentration—a hematocrit higher than 44% on admission (hemoconcentrated)—or if it rises in the first 24–48 hours has been suggested to be an early marker for predicting complications. Studies evaluating this have had variable results. A low or normal hematocrit on admission is associated with a milder disease (Vege, 2015). A persistent increase in hematocrit has been associated with an increased risk of necrosis and organ failure (Wu & Banks, 2013). In a study by Wu and colleagues (2011), there was a strong association between a blood urea nitrogen (BUN) level of 20mg/dl or greater, and an increased risk of death compared to a BUN of less than 20mg/ml. Any increase in the BUN within the first 24 hours was also associated with an increased risk of death; therefore, careful monitoring of serial BUN values in the first 24 hours could help identify patients at increased risk for mortality. A creatinine level > 1.8 within the first 24 hours is associated with an increased risk of pancreatic necrosis. C-reactive protein is a widely studied inflammatory marker; but it takes 72 hours to become accurate thus it is not practical for early prediction (Tenner et al., 2013). Overall, the most critical markers of severity are organ failure (especially MSOF) and pancreatic necrosis.

During the first 24 to 72 hours, serial bedside clinical assessments are crucial for reassessing severity and evaluating a patient's response to treatment. Assessments should focus on:

- Patient's symptoms
- Intravascular volume status
- Systemic inflammatory response (respirations, temperature, leukocytosis)

Collaborative and Nursing Care of the Patient with Severe Pancreatitis

The critical care patient is likely to be experiencing moderate to severe acute disease. Assessments focus on hemodynamic stability, pain, electrolyte balances, safety, and preventing and recognizing complications. Management involves correcting the underlying cause and aggressive supportive care, including fluid resuscitation, pain relief, restoring electrolyte balance, and nutritional support. Local and/or systemic complications create many clinical challenges that will determine further priorities and interventions. Additionally, if alcohol is thought to be a predisposing factor, the risks and complications of alcohol withdrawal must be considered concurrently with acute treatment.

Assessment of Hypovolemia

The nurse anticipates that the patient with acute pancreatitis will manifest pronounced volume depletion associated with external fluid losses, internal fluid shifts, and third spacing secondary to vomiting, fever, diaphoresis, and both local and systemic inflammatory responses.

Local inflammation may cause:

- Interstitial edema within the pancreas
- Vascular damage causing increased permeability
- Loss of albumin
- Hypovolemia, which affects the microcirculation of the pancreas, promoting pancreatic necrosis
- Tissue damage and fat necrosis possibly eating through the pancreas
 - Fluid leakage (albumin, activated enzymes, toxins, inflammatory mediators) into the retroperitoneal cavity and peritoneum, causing damage outside the pancreas
 - Hemorrhage from necrosis or ruptured blood vessels

SIRS results from the inflammatory mediators going beyond the pancreas and into the systemic circulation and may cause:

- Damage to structures and organs in the retroperitoneal and peritoneal cavity
- Peripheral (systemic) vasodilatation and vascular permeability
- Vital organ dysfunction and failure
- Massive fluid sequestrations (third spacing) into the:
 - Retroperitoneal space
 - Intraperitoneal cavity
 - Gut
 - Pleural space
- Hypovolemic, septic, and cardiogenic shock

The nurse reviews the patient's past and current history for predisposing factors, risk factors associated with severity, and evidence of systemic involvement and pancreatic necrosis. The nurse assesses for the signs and symptoms of hypovolemia described earlier in the chapter, including: thirst; poor skin turgor with dry mucous membranes; cool, clammy skin; flat jugular veins; hypotension or orthostatic hypotension with dizziness; narrowed pulse pressure; tachycardia; decreased capillary refill; decreased urine output; and mental status changes. Significant comorbidities or hemodynamic instability may necessitate more invasive monitoring. Thus, the patient may have an indwelling urinary catheter and advanced hemodynamic monitoring.

In addition, the nurse observes for two infrequent but classic signs associated with severe necrotizing pancreatitis: Cullen's sign and Grey-Turner's sign. Cullen's sign is a bluish discoloration around the umbilicus from the escape of blood into the peritoneum. Grey-Turner's sign is a bluish brown discoloration around the flanks from blood in the retroperitoneal space.

The nurse reviews the following laboratory tests to identify the patient's volume status and the severity of the disease:

- Hematocrit—A hematocrit greater than 44 on admission is consistent with hemoconcentration and evidence of volume depletion. An excessively low hematocrit would be indicative of bleeding. A normal hematocrit on admission suggests an uneventful clinical course. Serial hematocrits will be surrogate markers to assess adequacy of fluid resuscitation. They will likely be followed at 6, 12, and 24 hours to guide resuscitation efforts. The goal is to see a decrease in the hematocrit indicating hemodilution.
- Leukocytes—The degree of elevation is an indicator of severity.
- Serum electrolytes—Sodium, chloride, potassium, and magnesium are evaluated and replaced as needed. The values can influence the type of crystalloid solutions infused.
- Blood urea nitrogen (BUN)—BUN can be useful in evaluating severity and fluid volume status. It is also a surrogate marker for successful fluid resuscitation and will be followed serially with the hematocrit to guide resuscitation efforts. The goal is to see a decrease in the BUN, thus increasing renal perfusion. It is best evaluated relative to the serum creatinine. With volume depletion, BUN will increase out of proportion to the serum creatinine. However, if hypovolemia causes the mean arterial pressure to fall significantly for an extended duration, the glomerular filtration rate of the kidneys cannot be maintained, and change in renal function can occur. Patients must be evaluated for the development of acute kidney injury.
- Creatinine—Provides the most accurate estimate of glomerular filtration rate. It may rise with significant intravascular fluid loss, but a value greater than 2 mg/dL after hydration may be suggestive of renal involvement and has been associated with greater mortality. As with the BUN and hematocrit the creatinine will be followed serially. The goal is to maintain a normal creatinine the first 24 hours.
- Liver enzymes—Used to evaluate for gallstone pancreatitis (especially total bilirubin and alanine aminotransferase).
- Calcium—Hypercalcemia (total calcium greater than 11 mg/dL) may be a causal factor. Hypocalcemia (total serum Ca^{++} less than 8.5 mg/dL) can be caused by multiple factors in acute pancreatitis, including hypoalbuminemia (as most calcium is bound to albumin), hypomagnesemia (especially where excessive alcohol use is implicated), and fat necrosis. Routine monitoring and replacement is necessary to prevent neuromuscular and cardiac dysfunction. It may be evaluated concurrently with magnesium. The value can also influence the type of crystalloid solution as lactated Ringers is contraindicated in patients with hypercalcemia.
- Glucose—Hyperglycemia on admission is a consideration in predicting severity and may indicate pancreatic endocrine dysfunction.
- Abdominal ultrasound—This may be performed on admission to assess for gallstone etiology.
- Abdominal imaging—CT scan and/or MRI is indicated as mentioned for differential diagnosis situations on admission, or after admission when the patient's clinical status fails to improve within the first 48-72 hours. It can confirm the presence and extent of necrosis. Later imaging may be performed for suspected

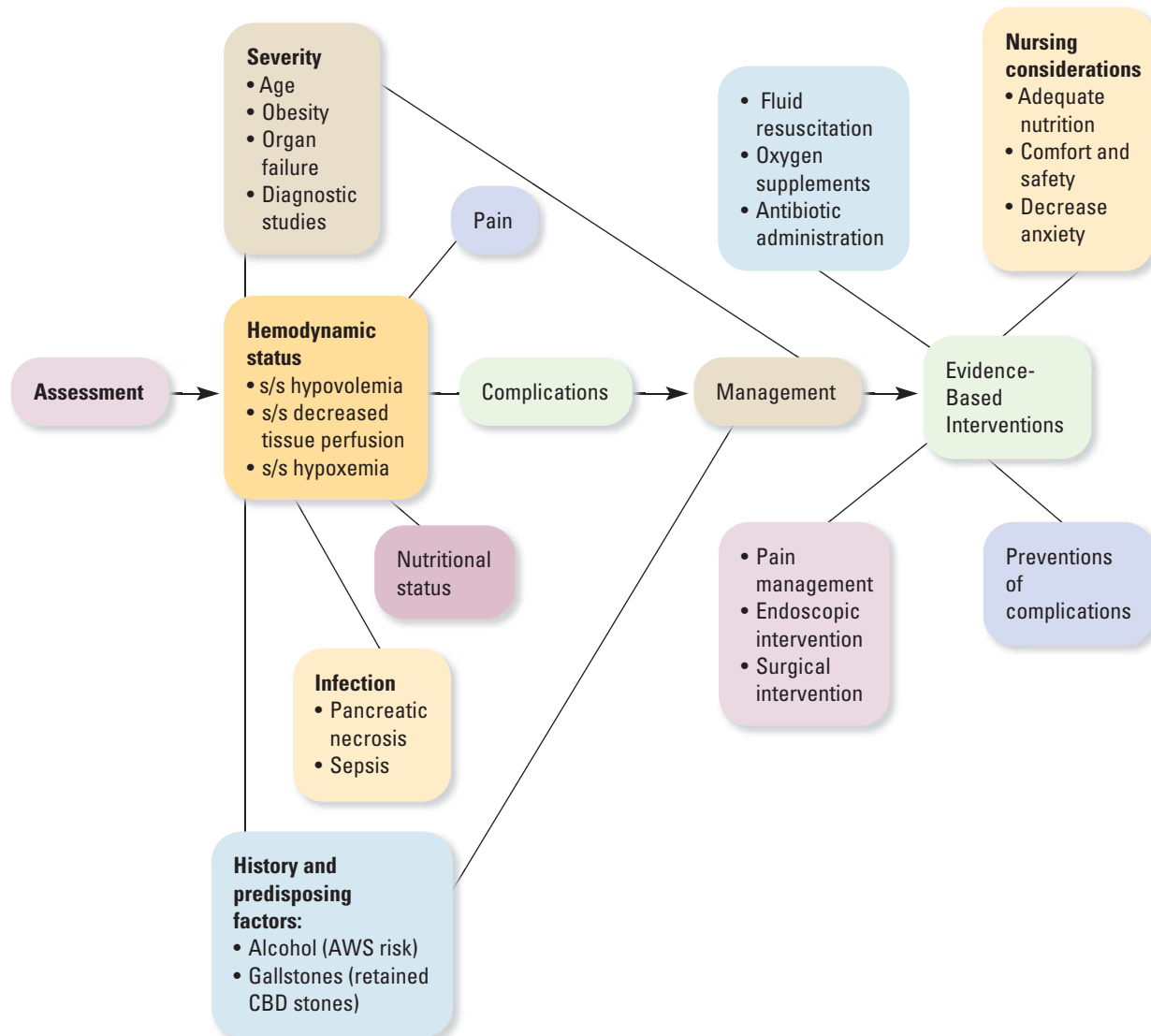
infected necrosis to guide fine needle aspiration for staining and culture or to identify other complications. Patient's renal and hydration status must be considered prior to IV contrast. If performed too early (less than 48 hours) after onset, tissue damage may not have reached its maximum and may be underestimated.

Maintain Hemodynamic Stability and Normovolemia

The nurse works collaboratively with the healthcare team to maintain hemodynamic stability, restore and maintain normovolemia, and, ultimately, maintain tissue perfusion. The goal of therapy is to avoid hypovolemia not only to prevent systemic complications, but because early restoration is instrumental in resolving existing organ involvement. In addition, aggressive fluid replacement can prevent or limit pancreatic necrosis.

Fluid Resuscitation

Aggressive fluid resuscitation for both mild and severe disease is the most critical therapeutic intervention. It needs to be initiated immediately on admission to the emergency department (ED). The 2013 ACG guidelines for management of acute pancreatitis emphasize the role of aggressive early hydration within the first 6-12 hours (Tenner et al., 2013). Aggressive hydration is defined as 250-500 mL per hour of isotonic crystalloid solution. In the past, guidelines recommended normal saline or Lactated Ringer's solution per practitioner preference. In a prospective randomized trial by Wu and colleagues (2011), resuscitation with Lactated Ringer's solution decreased the incidence of SIRS compared to saline resuscitation. ACG guidelines (2013) state that Lactated Ringer's may be the preferred solution. Large amounts of Lactated Ringer's is contraindicated in patients with hypercalcemia because it contains calcium. Patients with existing cardiopulmonary disease or other



Visual Map 13-5 Assessment and Management of Acute Pancreatitis

existing pertinent comorbidities need scrupulous management of their infusion rates and may require invasive hemodynamic monitoring. Careful attention must be given to clinical manifestations of fluid overload, especially respiratory compromise and/or pulmonary edema.

ACG guidelines indicate that the first 24 hours of aggressive hydration is the most beneficial. Fluid volume requirements need to be reevaluated frequently (Tenner et al., 2013). Guidelines suggest reassessment within 6 hours after admission and for the next 24 to 48 hours. Under resuscitation has been linked to increased pancreatic necrosis and mortality (Wu & Banks, 2013).

Severe hemorrhagic pancreatitis may require transfusions of blood or clotting factors, depending on hematocrit and clotting studies. Electrolyte solutions may be ordered after the initial resuscitation based on electrolyte values. Successful fluid resuscitation will be evident by the following changes:

- Normalization of BP
- Decreased/normal resting heart rate
- Increased urine output greater than 0.5 cc/kg/hour, approximately 30 cc per hour
- Normalization of advance hemodynamic monitoring (pulmonary artery pressures, cardiac output and index)
- If the hematocrit was initially hemoconcentrated, adequate fluid resuscitation would result in decreasing the hematocrit.

- Decreased BUN and normal creatinine
- Normal capillary refill with warm, dry skin

Strict cardiovascular and respiratory monitoring is warranted and subtle changes must not be overlooked. It is imperative to recognize either deterioration or improvement. If the patient's fluid status does not improve or worsens, consider hypovolemic shock or other complications.

Essential for Evidence-Based Practice

During the first 24 hours, the nurse must be vigilant in assessing the patient for signs of hypovolemia and assisting with the provision of aggressive fluid replacement to ensure optimal patient outcomes.

Assessment and Management of Pain

Pain is the characteristic diagnostic sign of acute pancreatitis. It results from irritation and edema of the inflamed pancreas and, in severe cases, the release of pancreatic enzymes into the surrounding tissues.

The nurse needs to conduct a thorough GI evaluation and pain assessment, using an appropriate standardized pain scale. Because manifestations may vary depending on severity, the nurse can expect that the patient will display many of the following characteristics:

- Sudden, severe epigastrium pain often peaking within 30 to 60 minutes and lasting hours to days.

Safety Initiatives

Implement Multidisciplinary Rounds

PURPOSE: Enhance the communication among members of the health care team, improve the flow of patients through the ICU, increase satisfaction among members of the health care team, and improve patient outcomes

RATIONALE: Multidisciplinary rounds (MDR) enable all members of the team caring for critically ill patients to come together and offer expertise in patient care (IHI, 2015). When nursing, pharmacy, nutrition, respiratory therapy, social work, patients, families, and physicians from other specialties caring for a patient contribute their expertise to identify patient goals and develop a plan of care, patient outcomes are better.

HIGHLIGHTS OF RECOMMENDATIONS:

1. Convene a multidisciplinary rounds conference preferably at the patient's bedside daily if possible but hopefullly at least 2 or 3 times a week.
2. Invite intensivists, generalists, ICU nurses, pharmacists, respiratory therapists, nutritionists, social workers, case managers, and family members.

3. Discuss:

- Patient's goals and preferences
- Patient's care needs
- Acuity assessment and discharge planning

4. Tips:

- Agree on one person to complete orders based on the recommendations made by the group.
- Agree that team members will present issues to the person in charge during multidisciplinary rounds.
- Expect the person in charge to request and rely on information from other team members.
- Carefully monitor patient progress by assessing whether goals are met.
- Adhere to protocols.
- Prepare for patient discharge and ensure follow-up services are available.

Source: Institute for Healthcare Improvement (2015)

- Deep, visceral, steady pain.
- Poorly localized pain often radiating to the back, chest, lower abdomen, and flanks.
- Abdominal guarding, rebound tenderness.
- Increased intensity supine, somewhat decreased with sitting, trunk flexed forward.
- Frequently accompanied by nausea and vomiting. Vomiting does not relieve the pain and might make it worse.
- Associated diminished or absent bowel sounds, paralytic ileus.
- May display physiologic signs: tachycardia, hypertension, tachypnea, splinting.
- Appears acutely distressed, anxious, inconsolable.

The nurse works collaboratively with the healthcare team to provide the patient with adequate pain relief. Adequate pain relief is a critical goal because the pain is severe, it increases the patient's anxiety level, and unrelieved pain may indicate disease progression.

Because the pain of acute pancreatitis is generally severe and the patient is unable to take anything by mouth, adequate pain control requires IV administration of medications related to rapid onset and ease of titration. Depending on the severity of the pain, medications may be ordered on a set schedule, as necessary, as a continuous infusion, or as patient-controlled analgesia (PCA). If the patient is cognitively and physically able, PCA is preferred because it maintains patient control and provides more effective pain management. If the pain is unrelieved with IV opioid administration, epidural analgesic may be considered. Epidural analgesia requires expert consultation and not all patients qualify.

Generally, severe pain requires opiates; milder cases may be managed with nonsteroidal anti-inflammatory medications such as ketorolac tramethamine (Toradol). There is no evidence to indicate which specific opiate is best. Current recommendations discourage the routine use of meperidine (Demerol) related to the accumulation of a toxic metabolite and adverse effects profile. Morphine and other opiates such as hydromorphone (Dilaudid) and fentanyl (Sublimaze) may be used.

NURSING ACTIONS

The nurse should anticipate that the patient may require high doses of analgesia. It is imperative that the patient is monitored for pain and hemodynamic responses. The nurse should assess the patient's pain response frequently, using the initial assessment tool. Expected outcomes are that the patient verbalizes decreased pain less than 4 on a scale of 0 to 10 and visible behavioral clues such as body

movement and facial expressions of pain. If the patient has received the maximal dose and the pain is still severe, the nurse must realize that the analgesia treatment is not working and therefore confer with the physician. The nurse must consider that unsuccessful analgesia may indicate that the disease is progressing. A pain management consult may also be considered. Further, vital signs including oxygen saturation must be monitored frequently by the nurse and evaluated concurrently with the patient's overall hemodynamic status.

The nurse would expect to see the following:

- BP would decrease because of decreased pain and the vasodilator effects of the opioids. If a decrease in pain is not attained, the BP may stay the same or increase.
- Pulse would decrease related to decreased pain, anxiety, and release of catecholamines or stay elevated if pain intensity continues.
- Respirations may increase or decrease. Pain relief may allow the patient to breathe easier; however, the depressant effects of the opioids may cause respiratory depression.
- Oxygen saturation may increase related to comfort or decrease related to respiratory depression.

Naloxone (Narcan) in small doses may reverse the respiratory depressant effects without affecting the analgesia. However, the decision to support the patient's respiratory status with intubation and ventilation must be considered.

Essential for Safety

Evaluating the patient's pain relief is a crucial component of nursing care. In acute pancreatitis, escalating pain despite the appropriate administration of opiates should be seen as an indication of worsening clinical status and must be communicated to medical personnel immediately.

Often, impaired respiratory function is the first sign of organ failure. Therefore, the nurse is especially attentive to the assessment and management of hypoxemia in the patient with pancreatitis.

Assessment

The nurse must frequently assess the patient's respiratory status. Factors influencing the risk of developing hypoxemia include:

- Potential/actual hypovolemia and decreased tissue perfusion
- Depressant effects of opioid administration
- Abdominal pain resulting in:
 - Hypoventilation, decreased lung expansion, vital capacity, and alveolar ventilation

- Increased catecholamine release, tachycardia, hypertension, increased metabolic rate, increased cardiac workload, and oxygen demand/consumption
- Fluid overload related to massive fluid resuscitation, pulmonary edema
- Release of toxins from the pancreas resulting in increased incidence of pleural effusion (especially on the left), pneumonia, and adult respiratory distress syndrome (ARDS). ARDS can develop rapidly.

Indications of Hypoxemia

To identify hypoxemia, the nurse assesses for the following clinical indicators:

- Decreased oxygen saturation/pulse oximetry (less than 92%)
- Increased or decreased respiratory rate
- Labored breathing, shortness of breath
- Abnormal lung sounds: crackles, wheezes, or decreased lung sounds (may indicate a pleural effusion)
- Restlessness, anxiety, or decreased level of consciousness
- Cardiac dysrhythmias
- Respiratory insufficiency/failure necessitating intubation ventilation (PaO_2 less than 60 despite increased oxygen concentrations is a criterion for intubation/ventilation)

The nurse should review the arterial blood gases, which are ordered initially as part of the severity evaluation criteria and as necessary when the patient exhibits decreased oxygen saturations with or without other symptoms. Particular attention should be on the PaO_2 and the pH. The nurse reviews the chest X-ray, which is routinely part of the initial evaluation. Particular attention is given to the presence of pleural effusions or infiltrates on admission or within 24 hours. Such findings are risk factors correlated with increased severity of pancreatitis.

COLLABORATIVE CARE

The nurse works collaboratively with the healthcare team to maintain oxygenation and prevent hypoxemia. The goal of therapy is to maintain oxygen tissue delivery, which can minimize pancreatic necrosis and prevent and sometimes resolve organ dysfunction. As discussed, aggressive fluid resuscitation is necessary to prevent hypovolemia and maintain tissue perfusion.

Supplemental Oxygen

Supplemental oxygen is administered usually for the first 24 to 48 hours or until there is no threat of hypoxemia. Oxygen percentage and delivery system should be

appropriate to maintain the patient's oxygen saturation at 92% or greater. Mild self-limiting cases are managed with nasal cannula at 1 to 4 liters. Progressive desaturation should be treated aggressively as respiratory insufficiency/failure, and the patient should be intubated and ventilated (see Chapter 3 .

The nurse continues to monitor the patient's respiratory status, assessing for signs and symptoms of hypoxemia concurrently with the patient's hemodynamic status. Pain relief interventions, as discussed, may help or hinder the patient's respiratory efforts. The nurse positions the patient for maximal comfort.

Essential for Safety

As the patient's pain often escalates requiring large doses of opiates, the nurse must be vigilant in assessing the patient's mental status and ability to protect the airway.

Assessment and Management of Infection

Pancreatic necrosis is defined as "diffuse or focal areas of non-viable pancreatic parenchyma >3 cm in size or >30% of the pancreas" (Tenner et al., 2013). Pancreatic necrosis can be sterile (absence of bacteria) or infected (presence of bacteria). Infection is a constant threat to the patient with severe necrotizing pancreatitis related to both pancreatic (infected necrosis) and extrapancreatic (pneumonia, bacteremia, cholangitis, urinary tract, etc.) causes. Infectious complications have a major impact on morbidity and mortality. Infected necrosis typically occurs between 7 and 14 days after the onset of the illness. Patients admitted with gallstone pancreatitis with cholangitis (infection of the common bile duct) or who develop other extrapancreatic infections will be treated with the appropriate antibiotics.

Assessment of Infection

The nurse assesses for the signs and symptoms of infection and sepsis. However, because the patient with pancreatitis without infected necrosis is likely to have a systemic inflammatory response, he may have fever and leukocytosis from SIRS. This can make it difficult to distinguish when sepsis has developed in the necrotic tissue. Infected necrosis is suspected if the patient fails to improve or deteriorates rapidly and unexpectedly, and/or presents with new progressive or persistent signs and symptoms of infection.

The nurse works collaboratively with the healthcare team to prevent, recognize, and appropriately treat infected pancreatic necrosis. It is crucial to distinguish between sterile and infected necrosis. Sterile necrosis is usually less extensive and can be managed conservatively, whereas infected necrosis needs aggressive management and is associated with poorer outcomes.

Evidence-Based Interventions

Evidence-based interventions for the prevention and treatment of infected pancreatic necrosis include:

- Fluid resuscitation, avoidance of hypovolemia and hypoxemia, and the maintenance of tissue perfusion, (as previously discussed) can prevent or limit pancreatic necrosis.
- Early enteral nutritional support helps to preserve the functioning of the gut's mucosal barrier and decrease bacterial translocations (movement of bacteria across the intestinal wall), resulting in decreased infectious complications. Organisms from the GI tract are typically the cause of infected necrosis.
- There is consensus that prophylactic antibiotics are not justified in interstitial pancreatitis secondary to an increased rate of secondary, superimposed bacterial and fungal infections. The role of prophylactic antibiotics in severe necrotizing pancreatitis has been debated. The current American College of Gastroenterology (Tenner et al., 2013) practice guidelines do not recommend the use of prophylactic antibiotics in severe acute pancreatitis (Tenner et al., 2013). The use of antibiotics in patients with sterile necrosis to prevent infection is also not recommended even if the patient has severe disease. Deteriorating conditions suggesting infection or sepsis and the presence of extensive necrosis with organ failure are clinical indicators justifying antibiotics while the source is actively being investigated.

The nurse anticipates that the patient will have blood, urine, sputum, and other appropriate cultures as ordered. In some cases, a CT scan with a fine needle aspiration (FNA) and culture of the necrotic tissue will be performed by the interventional radiologist. If appropriate cultures and the FNA are negative, antibiotics should be discontinued. Pending positive cultures and sensitivities, the choice of antibiotics will vary. Initially a broad-spectrum antibiotic that can penetrate the pancreatic tissue will be ordered. Not all antibiotics have the ability to penetrate pancreatic tissue and achieve therapeutic levels. High-dose cephalosporins, carbapenems, quinolones, and metronidazole are commonly used because they have a high degree of pancreatic penetration. Routine administration of antifungal agents is not recommended (Tenner et al., 2013).

The nurse would continue to monitor for signs and symptoms of infection and improvement of overall clinical status for indications that treatment is working, including decreasing white blood cell count, stable vital signs with normal temperature, decreasing pain, and overall clinical improvement.

Debridement of Necrosis (Necrosectomy)/Surgery

Necrosectomy is the resection of the necrotic tissue. Urgent debridement of infected or sterile necrosis is only recommended when the patient is unstable with symptoms clearly attributed to the necrosis/pseudocyst (persistent abdominal pain, nausea, vomiting, anorexia due to mechanical obstruction [common bile duct or gastric outlet] or a secondary infection). The standard of care for asymptomatic pseudocysts, and pancreatic and/or extra-pancreatic necrosis is no intervention regardless of size, location and/or extension (Tenner et al., 2013). Timing is critical, and survival rates increase when surgical, radiologic and/or endoscopic debridement is delayed preferably for more than 30 days. Delayed treatment enables the patient's clinical status to optimize and allows for liquification of the contents and the development of a fibrous wall (demarcation) to occur between the viable tissue and the unviable tissue. This is often referred to as walled-off necrosis. Ultimately, the patient's clinical condition makes the determination about timing and extent of the necrosectomy. The goal is to remove all of the necrotic tissue and preserve the viable tissue. Current recommendations prefer minimally invasive methods of necrosectomy to open (surgical) necrosectomy (Tenner et al., 2013). Less-invasive procedures include percutaneous retroperitoneal necrosectomy, laparoscopic necrosectomy, percutaneous catheter drainage, and endoscopic debridement and drainage.

Post-procedure or surgery, the nurse will monitor the patient's vital signs and pain level and be alert for evidence of bleeding. Typically, the patient will return with an array of drains and irrigating tubes. The nurse is responsible for monitoring, promoting drainage, and maintaining patency of the tubes as prescribed. On rare occasions, patients may develop peritonitis related to surgical injury of intra-abdominal organs and/or damage related to the leakage of pancreatic enzymes into the abdominal cavity.

Building Technology Skills

Endoscopic Retrograde Cholangiopancreatography (ERCP)

Endoscopic retrograde cholangiopancreatography (ERCP) is an invasive, endoscopic, and radiological procedure that involves oral intubation with a flexible fiber-optic endoscope and advancing it into the duodenum to directly view the ampulla/papilla of Vater. Once the papilla is identified, a small cannula is advanced into the orifice of the ampulla. It can then be manipulated to enter either the common bile

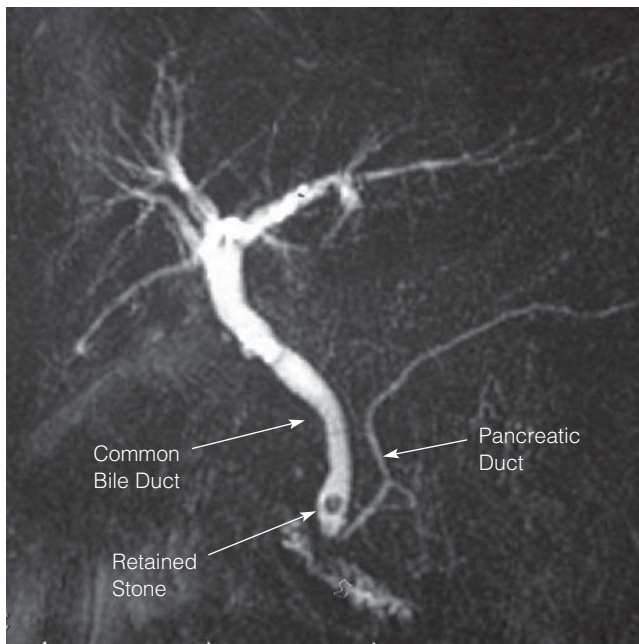


Figure 13-5 MRCP stone.

(Lahey Clinic)

duct or the pancreatic duct. Direct visualization of the ducts is accomplished by injecting radiographic contrast medium and taking a series of x-ray films. Often an endoscopic ultrasound or a magnetic resonance cholangiopancreatography (MRCP) may be performed to verify a common bile duct stone prior to an ERCP. An example of directly visualizing a stone in the biliary tract is shown in ♦ Figure 13-5.

Purpose

The goal of a prompt ERCP is to remove the obstruction, create a passageway for sludge and other stones, improve pancreatitis, and prevent cholangitis. ERCP is a therapeutic intervention in this setting and has no diagnostic role. Most gallstones causing acute pancreatitis pass spontaneously through the ampulla of Vater to the duodenum. Because the gallbladder shares a duct, gallstones that become lodged in the duct can prevent the flow of pancreatic enzymes, thus triggering acute pancreatitis. Lodged stones can cause cholangitis, a rare bacterial infection of the common bile duct that may progress to sepsis, further complicating the patient's condition. Both persistent obstruction and cholangitis contribute to severity and mortality.

The X-ray films can show the configuration of the ducts and identify strictures and obstructions. To remove an impacted stone, the endoscopist performs a sphincterotomy (also called papillotomy) that involves electrosurgical cutting of the papilla of Vater and the muscle fibers of the sphincter of Oddi. The goal of the sphincterotomy is to split tissue and fiber that may be hindering the passage of

stones and bile. The endoscopist then manipulates a variety of instruments to remove the stone, thus relieving the obstruction. If there is a stricture, a stent can be placed to facilitate drainage through the stricture. If an impacted stone is not found, a sphincterotomy will most likely be performed to prevent further impactions until a cholecystectomy can be performed.

Indications and Expected Outcomes

Urgent ERCP within 24 to 72 hours (preferably 24 hours from admission) should be considered in any patient with acute gallstone pancreatitis complicated by:

- Obstructive jaundice
- Concomitant cholangitis/biliary sepsis
- Severe pancreatitis (organ failure) with high suspicion of retained common bile duct stones

There is a higher mortality associated with gallstone-induced pancreatitis compared to alcohol-induced pancreatitis. The expected outcome after removing the biliary obstruction is that the patient's clinical picture will improve. Indications of improvement include:

- Decreased pain
- Resolution of the signs and symptoms of infection/sepsis
- Resolution of jaundice
- Decreased nausea, vomiting, gastric distention

The most common complication of an ERCP is acute pancreatitis. To decrease this risk, the endoscopist has technical interventions that can be employed during the procedure. A study by Elmunzer et al. (2012) found that the rectal administration of indomethacin (two 50 mg suppositories) immediately after the ERCP significantly reduced the incidence of pancreatitis especially in patients at high risk for this complication. The American College of Gastroenterology guidelines (Tenner et al., 2013) recommend this practice.

Technological Requirements

Technological requirements include a medical facility that has the endoscopic and radiological equipment to perform an ERCP. It is of utmost importance to have an endoscopist and supportive staff with the expertise to perform such an advanced procedure.

Nursing Responsibilities

The critical care nurse has both pre- and post-procedure responsibilities. Current standard of care is the administration of sedation (propofol) or general anesthetics by anesthesia staff.

Pre-procedural Responsibilities

- Maintains NPO status for 6 to 8 hours prior to the procedure
- Removes dentures
- Monitors baseline vital signs, including temperature and oxygen saturation
- Determines any allergies to contrast material
- Ensures that informed consent has been obtained
- Situation-background-assessment-recommendation (SBAR) communication

Post-procedural Responsibilities

- Maintains recovery position until the patient is fully awake
- Monitors vital signs including temperature, oxygen saturation, and level of pain and consciousness until the patient returns to baseline
- Monitors for an adverse response to medications
- Monitors for potential complications: worsening pancreatitis, perforation, and bleeding
 - Worsening of pain
 - Nausea and vomiting
 - Fever and chills
 - Unstable vital signs

Once the patient has stabilized, a cholecystectomy should be considered because delayed surgery could put the patient at risk for a recurrent attack.

NURSING CARE

The nursing care of the patient with acute pancreatitis is focused on maintaining adequate nutrition for healing, promoting comfort and safety, and relieving anxiety.

Providing Nutrition

Because of pain and associated GI symptoms (nausea, vomiting, distention, ileus), all patients with pancreatitis are initially kept NPO. In mild pancreatitis, a low-fat soft diet can be started if there is no nausea or vomiting and the abdominal pain has resolved. Initiating feeding with a low-fat soft diet has been shown to decrease length of stay, infectious complications, morbidity and mortality. In the past, patients were kept NPO for extended periods in order to “rest” the pancreas. This theory is no longer supported by laboratory and clinical data (Tenner et al., 2013).

The patient with moderate to severe pancreatitis experiences an increased metabolic demand and increased resting

energy expenditure related to severe pain, hemodynamic status, and inflammation. They may also have a negative nitrogen balance up to 40 grams/day. In severe pancreatitis, enteral nutrition is preferred over total parenteral nutrition (TPN). The advantages of enteral nutrition include:

- Maintenance of the intestinal barrier, thus preventing translocation of intestinal bacteria and reducing septic complications.
- Elimination of the many complications encountered with parenteral nutrition (including sepsis and metabolic abnormalities).
- Decreased rates of infection, resulting in decreased need for surgical intervention.
- May modify the stress response and reduce duration of the disease process and length of nutritional therapy
- Fewer overall complications
- Decreased hospital lengths of stay

As with all critically ill patients, early nutritional support positively affects outcomes and should be started within the first 2 to 3 days. Multiple studies report positive outcomes initiating enteral feeding for severe AP within 48 to 72 hours of admission (Olah & Romics, 2014). Discussion continues related to the optimal level or route of enteral nutrition (gastric vs. postpyloric jejunal feedings). The National Institutes of Health is currently sponsoring a large study to determine the optimal route in severe pancreatitis. Multiple studies have found that nasogastric feeding is safe and well tolerated in this population. Compared to nasojejunal, nasogastric tube placement is easier especially in ICU. However, there is an increased risk of aspiration, and the patient should be in an upright position and placed on aspiration precautions (Tenner et al., 2013).

The likelihood of gastric feedings being tolerated by a specific patient is unknown because individual patients will have a wide range of tolerance based on the different degrees of severity of the disease. In addition, tolerance can be influenced by the level of infusion, the specific formula, the stress response, the mode of infusion, and intestinal motility influenced by the effects of opiates. If gastric feedings are initiated and not tolerated, the jejunal route should be tried. Changing the level of the feeding further down the GI tract should improve tolerance.

The choice of enteral feeding level will depend on the individual practitioner and patient’s clinical picture. Postpyloric nasojejunal feedings seem to be the most commonly used route. The chance of aspiration is minimized when proper tube positioning is maintained. The tube should be in the distal duodenum or jejunum beyond the ligament of Treitz. In severe cases, enteral feeding may be required for weeks while inflammation decreases, and necrotic areas have been debrided or drained. The disadvantages of jejunal feedings are the difficulty with placement of the tubes, maintaining proper position, and patency. The tube’s small

diameter often causes clogging. If the patient requires surgery, it is often advantageous to have a jejunum tube inserted. This provides that patient with more comfort especially if enteral feeding is long term.

The patient should have an early nutritional consult to evaluate enteral caloric requirements based on energy expenditure and formula selection. The patient's pancreatic endocrine function related to glucose metabolism, acid-base balance, and electrolyte balance must be considered. Recommended energy requirements can fluctuate between 25 and 30 kcal/kg/day. Nutritional support should integrate vitamins and trace elements, especially vitamins A, C, and E and zinc (Seron-Arbeloa, Zamora-Elson, Labarta-Monzon, & Mallor-Bonet, 2013). TPN is appropriate only when enteral feedings cannot be tolerated and may be used in combination with enteral feedings when caloric requirements cannot be met. In patients intolerant of enteral feeding, it is also recommended that a minimal enteral amount be maintained (Seron-Arbeloa et al., 2013).

The nurse assesses the patient for indications of adequate nutrition:

- Intake and output
- Weight—because of the potential for massive fluid sequestration and the aggressive fluid resuscitation, weight may not be initially reliable
- Tolerance of the feedings
- Development of an ileus
- Laboratory studies: electrolytes and glucose.

The nurse also assesses the patient for potential complications of feeding, most importantly, the increase or return of pain; nausea, vomiting, cramping; aspiration; tube obstruction; hyperglycemia; or diarrhea/constipation.

Reflect On

The benefits of early nutrition in the critically ill patients are well known. There is much discussion related to the optimal level or route of enteral feedings in patients with severe pancreatitis. If a patient with severe pancreatitis is receiving TPN, what clinical data would support this intervention? What data would support transition to enteral feedings?

Promoting Comfort

The patient will often experience nausea and vomiting. If the nausea and vomiting are persistent, the patient may be treated with an antiemetic medication. Once administered the patient must be reevaluated for relief. The patient may have an NGT inserted to relieve persistent nausea, vomiting, and gastric distention. This is not a routine intervention and is performed only to promote comfort when nausea and vomiting continue despite medication. Such decompression has not been shown to affect outcomes.

While the patient may get relief, the NGT is uncomfortable and the patient must receive meticulous nasal and oral care. The patient should be positioned for optimal comfort, often sitting with the trunk flexed forward.

Reflect On

Have you ever discharged a patient who had an extensive hospitalization (e.g., 50 out of 60 days in the hospital, including 15 days in an ICU, 11 of those days intubated and sedated)? What were some of the issues that confronted this patient/family upon discharge? What nursing actions did you take to facilitate this transition?

Fostering Patient and Family-Centered Care

Whether patients have mild or severe forms of the disease, they are usually extremely anxious related to the severe pain, sudden onset of the illness, and the lack of knowledge regarding the disease. As discussed, pain management is a necessity. Patients and families should be instructed about the disease, the rationale for the interventions, and expected outcomes. Simple, consistent, and repetitive information needs to be given by all members of the healthcare team because anxiety often impairs patients' and families' readiness and ability to learn. Comprehension of the information should be consistently evaluated. Consistent staff and family visitation often promotes comfort and trust while decreasing anxiety.

Prevention of Complications

The development of complications clearly affects the patient's clinical course and patient outcomes. The nurse recognizes that the severity of acute pancreatitis often cannot be predicted early in the course of the disease. One of the common practice errors is incorrectly labeling a patient with mild disease within the first 48 hours. Crucial to preventing complications is prompt diagnosis, identification and treatment of the cause, and management according to evidence-based guidelines. All patients should receive aggressive fluid resuscitation and supplemental oxygen until the severity of the disease is established. In general, the mortality rate is 3% for interstitial pancreatitis and 15% for necrotizing pancreatitis (Wu & Banks, 2013). Early deaths, usually within two weeks, are caused by organ failure; later deaths are attributed to infections and sepsis related to pancreatic necrosis. As mentioned, the majority of the patients with severe pancreatitis without organ failure survive, whereas those with persistent organ failure have a 30% mortality rate and the risk of in-hospital death doubles with both infected necrosis and organ failure (Wu & Banks, 2013).

All routine evidence-based interventions for protecting the patient from injury must be implemented, including hand washing and strict aseptic technique, fall, and immobility protocols. The two most important clinical parameters that precipitate both systemic and local complications are hypovolemia and tissue hypoxia. Third spacing of massive amounts of fluid is easily underestimated. Nurses need to be vigilant in monitoring the patient's hemodynamic and respiratory status. It is imperative that the nurse evaluates the adequacy of fluid resuscitation, oxygenation efforts, and overall tissue perfusion. The nurse realizes the significance of assessment and management in preventing and recognizing complications. Effective communication with the healthcare team is essential. The nurse must assess for and implement measures to prevent the following:

- Respiratory failure, ARDS
- Cardiovascular collapse: hypovolemic shock
- Renal failure, acute tubular necrosis
- Decreased cerebral perfusion/neurological dysfunction
- Metabolic abnormalities (electrolyte and acid-base imbalances, hyperglycemia, diabetes)
- GI bleeding
- Disseminated intravascular coagulation
- Sepsis/septic shock

Other local complications include pancreatic pseudocysts and abscesses. A pancreatic pseudocyst is a collection

of debris, tissue, fluid, and pancreatic juices that is enclosed by a nonepithelialized wall. Pseudocysts are generally sterile, and they can take up to 4 weeks after a severe episode of acute pancreatitis to develop. The majority resolve on their own, whereas other four may become infected or rupture. An infected pseudocyst is a pancreatic abscess. Treatment depends on the size, location, and the presence of symptoms. Pseudocysts can produce persistent pain, the inability to eat, and, depending on the size, an abdominal mass may be palpated. Drainage of a pseudocyst can be performed surgically, endoscopically, or percutaneously. Often, large pseudocysts can compress the stomach or duodenum, causing the inability to eat. Additionally, compression of the common bile duct can block drainage from the liver, resulting in a rise in liver function tests, an increase in bilirubin, and jaundice.

Patients with severe pancreatitis are at risk for developing intra-abdominal hypertension and possibly abdominal compartment syndrome secondary to a plethora of mechanisms related to the inflammatory processes, large-volume resuscitation, and the development of MSOF and sepsis. Capillary leakage, pancreatic and visceral edema, and the development of fluid collections and ascites all contribute to intra-abdominal hypertension. Additionally, an elevated intra-abdominal pressure may contribute to the development of pancreatic hypoperfusion and necrosis.

Acute Gastrointestinal Bleed and Pancreatitis Summary

Gastrointestinal bleeding remains a common clinical problem with cost, morbidity, and mortality implications. The clinical situations are very diverse, depending on the source of bleeding, type of lesion, and rate of bleeding. Positive patient outcomes rely on the nurses' thorough monitoring, comprehension of the patient's hemodynamic status, and effective communication with the healthcare team. Advances in gastrointestinal endoscopy have become instrumental for diagnosing and treating the bleeding lesions as well as predicting the risk for re-bleeding. The success of interventional endoscopy has decreased the need for surgical intervention, which continues to have a high morbidity and mortality. Diagnosis of an initial bleed affords the opportunity for the healthcare team not only to treat the bleed but also to provide secondary prevention. The nurse must provide patients and families the necessary knowledge to understand the risk factors

and causes associated with the bleeding and the importance of medication compliance. Decreasing or eliminating alcohol consumption and NSAID usage are often two common lifestyle changes that will decrease the risk of future bleeding.

Patients who are critically ill with pancreatitis require comprehensive medical and, possibly, surgical care coupled with extensive nursing care. Fluid resuscitation is key to preventing continuing pancreatic destruction as well as to limiting organ failure. Initial treatment is directed at identifying and correcting the cause—for example, relieving a gallstone obstruction. The nurse must be vigilant in monitoring the patient's fluid balance, managing the patient's pain, and assessing for the early signs of complications, most notably hypovolemia and hypoxemia. Pancreatic necrosis and the presence, timing, and duration of organ failure influence morbidity and mortality.

Why/Why Not?

A ventilated patient with severe pancreatitis has been receiving a continuous infusion of 75 mg of fentanyl per hour with good pain relief and a CPOT score of 1. When the nurse assesses the patient a noon, the patient's blood pressure has decreased while the pulse has increased and the CPOT score

of 5. The nurse can provide a 25 mg IV bolus of fentanyl and increase the infusion to 100 mg/hour. Should the nurse provide the bolus and increase the infusion? Why/Why not?

See answers to Why/Why Not? in the Answer Section.

Case Study

Georg Jensen is a 57-year-old male admitted to the ICU for acute pancreatitis. His mother died 4 months ago, and shortly after that he lost his job. Recently, he has been drinking a gallon of vodka a day. In addition, he has a history of diabetes mellitus. He was brought to the ED by ambulance when his brother found him unresponsive at home.

He was intubated in the ambulance on the way to the ED and remains intubated and ventilated due to hypoxia. In the ED, his serum hematocrit was 45%, his WBC 21,000 μL (mm^3), BUN 35 mg/dL, creatinine 1.4 mg/dL, blood glucose 425, and serum amylase 1,250 SI units. If he awakens, he writhes in pain. He is currently receiving 5 mg of midazolam an hour, 200 mcg of fentanyl an hour, and 8 units of insulin per hour. Since arrival in the ED 2 hours ago, he has received 4 liters of normal saline and is currently receiving 500 mL NS/hour. His blood pressure

is currently 92/65 mmHg, heart rate 124 beats/min, and CVP 8 mmHg.

1. What do his laboratory studies tell you about his hydration status?
2. Is that information confirmed by his vital signs? Why or why not?
3. What is the most likely reason why he developed pancreatitis?
4. How severe is Mr. Jensen's pancreatitis? Why do you believe that?
5. Why is he experiencing such pain?
6. What are the two most essential collaborative interventions for Mr. Jensen?
7. What is the most important nursing diagnosis for him?

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 13.1 What is the most frequent cause of gastrointestinal bleeding in the older adult? Why is it a common cause of GI bleeding?
- 13.2 What are the differences between the manifestations of upper GI bleeding in the middle-aged and older adult?
- 13.3 How do the manifestations of upper GI bleeding differ from the signs and symptoms of a lower GI bleed?
- 13.4 How can a nurse identify when a patient who is GI bleeding has received adequate fluid and blood replacement?
- 13.5 What are the important components of discharge teaching for the patient with a GI bleed?
- 13.6 What are the essential nursing interventions when caring for a patient who has just had an endoscopy for upper gastrointestinal bleeding?
- 13.7 How do alcohol misuse and gallstones result in pancreatitis?
- 13.8 What manifestations should suggest that the patient was developing severe pancreatitis?
- 13.9 What are the two priorities of care for the patient with severe pancreatitis?

See answers to Chapter Review Questions in the Answer Section.

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Chapter 14

Care of the Patient with Problems in Glucose Metabolism

Betsy Swinny, MSN, RN, CCRN

Abbreviations

ADA	American Diabetes Association	HHNS	Hyperglycemic Hyperosmolar Nonketotic Syndrome
CDE	Certified Diabetic Educator	JDRF	Juvenile Diabetes Research Foundation International
CSS	Corrected Serum Sodium		
DKA	Diabetic Ketoacidosis		



Learning Outcomes

Upon completion of this chapter, the learner will be able to:

1. Compare and contrast the difference between the physiology of normal metabolism and the pathophysiology of type 1 and type 2 diabetes and metabolic syndrome.
2. Articulate the pathophysiology, complications, and patient education associated with hyperglycemia of critical illness.
3. Compare and contrast the pathophysiology, precipitating factors, assessment and complications of diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar nonketotic syndrome (HHNS).
4. Articulate essential considerations related to the safe administration of insulin.

Introduction

Diabetes is a very common condition with increasing prevalence in the United States. The National Diabetes Information Clearinghouse (NDIC), a branch of the National Institutes of Health (NIH), and the Centers for Disease Control and Prevention (CDC) report statistics about diabetes and its increasing prevalence in the United States. Diabetes has become a national epidemic that costs the nation billions of dollars annually. Direct medical costs were estimated to be \$176 billion in 2012. In addition, it is estimated that \$69 billion was spent on indirect costs such

as lost time from work (CDC, 2014; NDIC, 2014). Both the CDC and the NDIC report that 9.3% of the total U.S. population, or 29.1 million people of all ages, have diabetes. Another estimated 8.1 million people have diabetes, but have not yet been diagnosed with the disease. In addition, an estimated 86 million Americans have prediabetes. These people are at very high risk to develop diabetes. Diabetes continues to be prevalent in older people, though the disease has exploded in prevalence in the last 10 years. The increased prevalence of diabetes in the United States is related to the epidemic of overweight and obese Americans, poor dietary choices, and lack of exercise.

The large majority of diabetics have type 2 diabetes. Because of its cost and prevalence, it is important to fully understand diabetes and its management.

Physiology and Pathophysiologies of Glucose Metabolism

In order to understand the pathophysiology of diabetic ketoacidosis and hyperglycemic hyperosmolar nonketotic syndrome (HHNS), it is important to first review the normal process of food metabolism. Foods are composed of carbohydrates, fats, and proteins. Each of these components is metabolized into a different set of end products during the process of digestion and absorption.

Carbohydrate Metabolism

Carbohydrates are composed of carbon, hydrogen, and oxygen. They are categorized as monosaccharides, disaccharides, or polysaccharides, depending on the number of sugars they contain. Carbohydrates are broken down into simple sugars such as glucose, galactose, and fructose during the process of digestion. Once digested, these simple sugars enter the bloodstream through the intestinal mucosa. These simple sugars are taken into the cell with insulin, and they are a direct source of fuel for the body. Sugars are used by cells during the process of metabolism to produce energy. Carbohydrates produce 4 kilocalories per gram (kcal/g). Extra hydrogen ions are excreted through the kidneys in the urine. Carbon and oxygen combine to form carbon dioxide, which is excreted through the lungs.

If more carbohydrates are consumed than are needed for immediate metabolism, the body stores the excess digested sugars as glycogen in the liver and muscle. **Glycogenesis** is the conversion of excess glucose to glycogen. In addition to storing sugars as glycogen, the body may convert excess sugars to fat and store it in adipose tissues. Conversely, when the body needs glucose for metabolism and none is available, glycogen (stored in the liver) is broken down into glucose. **Glycogenolysis** is the conversion of glycogen to glucose. If glycogen stores are used up or carbohydrate intake is limited, the body begins to create glucose from fats and amino acids.

Fat Metabolism

Fats or lipid particles are also composed of carbon, hydrogen, and oxygen. Fats come from the diet in the forms of saturated fats and unsaturated fats. Saturated fats include animal products such as butter, milk, and cheese. Unsaturated fats include monounsaturated fats such as olive oil

and polyunsaturated fats such as vegetable oils (e.g., corn oil) and fish.

Fats are digested and absorbed into the bloodstream as glycerides (such as triglycerides), phospholipids, and sterols (such as cholesterol). Unlike sugars, glycerides, phospholipids, and sterols are not taken up directly by any cell. Instead, these fats must be broken down into fatty acids and glycerol, which can then enter the cell for metabolism. Fatty acids and glycerol are produced when lipid particles are hydrolyzed. **Hydrolysis** is a process that splits the bonds and adds water to fats. When fatty acids and glycerol enter the cell, lipolysis (fat breakdown) occurs in the mitochondria. Fats produce 9 kcal/g, compared with 4 kcal/g for carbohydrates.

When carbohydrate (glucose) is not available at the cellular level, the body begins to break down fats for cellular metabolism to produce energy. This process occurs for three main reasons: if glucose cannot reach the cell due to lack of insulin; prolonged starvation; and when fats are eaten in the absence of carbohydrates, for example, in carbohydrate-free diets. In all of these situations, glucose is not available at the cellular level for metabolism, so fatty acids and glycerol are used to produce energy instead of glucose.

The breakdown of fatty acids and glycerol result in the formation of ketone bodies. There are three types of ketone bodies: acetoacetate, β -hydroxybutyrate, and acetone. Acetoacetate and β -hydroxybutyrate can be used for fuel by most body tissues. Acetone cannot be used by the body in cellular metabolism and is excreted in the urine and through exhalation. When acetone is exhaled, a “fruity breath” or “ketone breath” odor is produced and is a recognized symptom of ketosis. Ketosis occurs when more ketone bodies are being formed than used for cellular metabolism, resulting in extra ketones in the bloodstream.

Protein Metabolism

Proteins contain carbon, hydrogen, oxygen, and nitrogen. Nitrogen is an essential component of protein because it is important to the formation of amino acids. Both animal and vegetable proteins form amino acids when they are digested. Animal proteins include various meats. Vegetable proteins include beans and legumes. Once digested, most amino acids are converted to glucose in the liver. The glucose derived from amino acids can then be transported to the cellular level with insulin and used for cellular metabolism. The end products of protein metabolism include adenosine triphosphate (ATP), carbon dioxide, water, urea, and ammonia. ATP is used at the cellular level to produce energy. Proteins, like carbohydrates, produce 4 kcal/g. Water and carbon dioxide are excreted through the lungs and kidneys. Urea and ammonia are also excreted in the urine through the kidneys.

If more proteins are eaten than are needed, they are converted to glycogen and stored in the liver, or converted to fat and stored in adipose tissue. Conversely, proteins are broken down to be used in cellular metabolism to produce energy when dietary intake of carbohydrates is inadequate. Use of proteins for energy occurs when glucose cannot reach the cell due to lack of insulin, during prolonged starvation, and when the diet is solely protein. The proteins in the liver are used in preference to those of other tissues such as the brain. Gluconeogenesis is the formation of glucose from amino acids and fats when carbohydrate intake is limited and there are no glycogen stores.

Function of Insulin

The pancreas is the organ that makes the hormone insulin. The pancreas is located in the abdominal cavity behind and to the medial side of the stomach. A small portion of the pancreas contains the islets of Langerhans. The islets of Langerhans secrete four hormones: Alpha cells make and secrete glucagon. Beta cells make and secrete insulin. Delta cells make and secrete somatostatin, and F cells secrete pancreatic polypeptide. In addition to producing these hormones, the pancreas also produces digestive enzymes.

Insulin is an **anabolic hormone**, which means that it becomes more complex as glucose binds onto it. One function of insulin is to move glucose from the bloodstream into muscle, fat, and liver cells, where it can be metabolized into fuel. A second function of insulin is to stimulate the liver to store excess glucose in the form of glycogen. When carbohydrate (glucose) is absorbed from the intestines into the bloodstream, the pancreas detects the increase in blood glucose and releases insulin into the bloodstream. Insulin travels into the various body tissues (in the muscle, fat, and liver) and binds to insulin receptor sites within the cell. Insulin then facilitates the transport of glucose from the bloodstream into the cell. Once glucose is inside the cell, it can be used by the cell to create energy or can be stored by the liver. If glucose cannot be transported into the cell because there is a lack of insulin, the cells will starve. Even though there may be too much glucose in the bloodstream,

without insulin the glucose cannot reach the cellular level to be converted into energy, and the cells cannot produce the energy needed for normal cellular metabolism.

Type 1 Diabetes

The defining characteristic of type 1 diabetics is that the beta cells produce negligible or no insulin. Without insulin, glucose builds up in the bloodstream but cannot enter the cells. The type 1 diabetic needs to take insulin in order to maintain normal cellular glucose uptake and metabolism.

Type 1 diabetes is usually diagnosed in childhood. Adults diagnosed with the disease are usually less than age 30. Type 1 diabetes is caused by a hereditary autoimmune process where the beta cells of the islets of Langerhans are targeted and destroyed. The trigger for the destruction of the beta cells is still under investigation. Some feel that the trigger is a virus, others think it is environmental such as a food trigger, and others feel that the autoimmune trigger occurs as a result of an internal trigger based on genetics (Li, Song, & Qin, 2014). The insulin-producing beta cells of the pancreas are destroyed, and little to no insulin is produced. The onset of symptoms in type 1 diabetes is severe and usually occurs rapidly. Because of the rapid onset of type 1 diabetes, people are often in diabetic ketoacidosis at the time of diagnosis.

Type 2 Diabetes

The prevalence of type 2 diabetes is far more common than the prevalence of type 1 diabetes, making up 90% or more of all cases of diabetes. In the past, most people with type 2 diabetes were diagnosed in adulthood, though the disease is now much more common in younger adults and children as more people have common risk factors associated with type 2 diabetes. Common risk factors associated with the disease include genetic predisposition or family history plus obesity, failure to exercise, and poor dietary habits. In addition, older people are at risk for type 2 diabetes, and the prevalence of diabetes is increasing as the population of older Americans increases (Box 14-1).

Box 14-1 Risk Factors for Type 2 Diabetes

Risk Factors for Type 2 Diabetes

Family history of diabetes

Race/ethnicity (the following all have high rates of diabetes):

- African Americans
- Hispanic Americans
- Native Americans

Obesity

Identified impaired glucose tolerance

High blood pressure

High cholesterol

- HDL cholesterol of less than 35 mg/dL
- Triglyceride level of greater than 250 mg/dL

Gestational diabetes or delivering a baby weighing more than 9 pounds

Age greater than 45 years

Bariatric Considerations

Insulin resistance increases with obesity. Insulin resistance is a cause of type 2 diabetes and makes the control of blood sugar more difficult. Obese patients will require more medication to control hyperglycemia.

Those who are obese and have diabetes are at great increased risk of vascular complications such as peripheral vascular disease, stroke, and acute myocardial infarction. Both hyperglycemia and insulin resistance

increase the amount of circulating triglycerides in the bloodstream. The result is increased plaque formation in the vascular tree.

The latest recommendations suggest that bariatric surgery should be considered for obese patients with diabetes. When obese patients lose weight, control of blood glucose improves (Schauer et al., 2012).

Type 2 diabetes is characterized by insulin resistance. The insulin produced by the pancreas is unable to bind to the insulin receptor sites inside the cell. Therefore, glucose is unable to move into the cell, and cells are unable to produce energy. Because the cells are not getting the glucose they need, the pancreas produces more and more insulin. The insulin resistance, lack of functioning insulin, causes abnormally high levels of glucose to build up in the blood (hyperglycemia). People with type 2 diabetes have hyperglycemia and high blood insulin levels (hyperinsulinemia) at the same time.

The onset of type 2 diabetes usually occurs over a period of years as insulin resistance gradually increases. Most people with the disease are overweight. People who are overweight have a higher risk of insulin resistance, because fat interferes with the body's ability to use insulin. Because of its gradual onset, people with type 2 diabetes are often not aware that they have the disease at the time of diagnosis. The chronic complications of diabetes such as heart disease, stroke, vascular disease, renal disease, retinopathy, and neuropathy may have already occurred prior

to diagnosis. For this reason, the American Diabetes Association (ADA) recommends that all adults be screened for diabetes at a minimum of every 3 years. Those at high risk should be screened more often.

Hyperglycemia in Critical Illness

In addition to recognizing and treating those with diabetes, it is also important to treat patients who are prediabetic with metabolic syndrome. Metabolic syndrome coupled with physiologic stress leads to hyperglycemia, and glucose monitoring should be implemented in order to detect hyperglycemia in this population. Hyperglycemia causes endovascular inflammation and results in increased complications, increased length of stay in the critical care unit, and increased mortality. Research has shown that recognition and treatment of hyperglycemia improves quality of care as well as decreasing the cost of treatment.

Gerontological Considerations

The prevalence of type 2 diabetes increases with age and affects 25.9% of people over age 65 in the United States (CDC, 2014).

The diagnosis of diabetes in the older person is problematic because the older patient with diabetes may not present with the classic symptoms. Typical symptoms of hyperglycemia such as polyuria, polydipsia, and polyphagia may be masked. Because increased thirst can be absent, the initial presentation among older patients may be dehydration with dry eyes, dry mouth, and confusion.

Both diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar nonketotic syndrome (HHNS) occur in older adults. However, DKA is rare and its features

and management do not differ from those in younger diabetics, but its mortality is greater primarily because of associated cardiovascular disease.

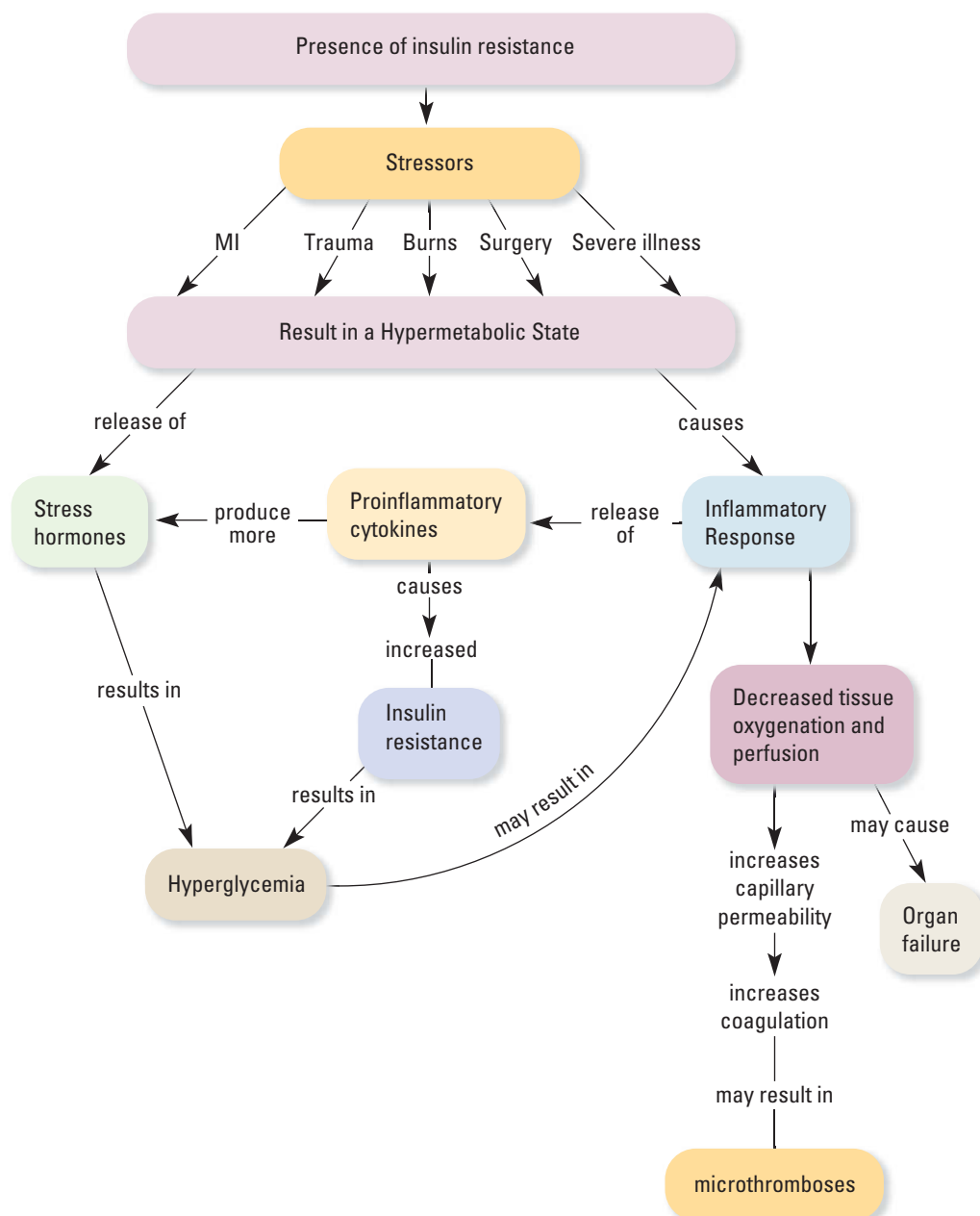
HHNS almost always occurs in older people, and half the time it is the initial manifestation of diabetes. The ready development of HHNS in the older adult can be explained by a combination of factors, including reduced thirst perception. Acute infection is the most frequent predisposing factor of HHNS, with pneumonia being the most common infection.

The risk of hypoglycemia associated with insulin increases with age. In older adults, symptoms of hypoglycemia might not appear until the blood sugar is less than 50 mg/dL.

Critical illness causes a physiologic stress response, that in turn causes the body to enter a hypermetabolic state in an attempt to heal. The physiologic stress response can be caused by a variety of critical illnesses including acute myocardial infarction, trauma, severe infections, postsurgical healing, burns, or other conditions in which the body has a stress response. Two main processes cause hyperglycemia. First, the reaction to counter-regulatory hormones has a direct hormonal effect to produce hyperglycemia. Second, pro-inflammatory factors cause insulin resistance, which also leads to hyperglycemia. It is very important to recognize and treat hyperglycemia in critically ill patients because research findings show that the inflammatory results of hyperglycemia cause decreased vascular flow to cellular tissues and to organs.

Stress Hormones

During the hypermetabolic state, the body releases a variety of counterregulatory hormones including catecholamines (epinephrine, norepinephrine, and dopamine), cortisol, growth hormones, and glucagon. These counterregulatory hormones contribute to hyperglycemia through the following processes. Catecholamines specifically inhibit insulin secretion from the beta cells, and also have an anti-insulin effect. Glycogenesis is inhibited; the liver does not convert excess glucose to be stored as glycogen. In addition, glycogenolysis (the conversion of glycogen to glucose) and gluconeogenesis (the formation of glucose from amino acids and fats) are both accelerated. Glycogenolysis also occurs in



Visual Map 14-1 Hyperglycemia During Critical Illness

muscle tissue. Lipolysis, the conversion of stored fat to glucose, takes place in the peripheral tissues. Later, as metabolic needs continue to be high, muscle protein is broken down.

Alterations in Glucose Metabolism and Insulin Resistance

Pro-inflammatory cytokines cause insulin resistance in the skeletal muscle, fat, and liver by altering biochemical markers that insulin binds to at the receptor sites. This insulin resistance has a direct effect on hyperglycemia by preventing glucose from entering cells. Pro-inflammatory cytokines also stimulate the release of the counterregulatory hormones described earlier, which further contributes to hyperglycemia. The nervous system and the blood cells are affected less by insulin resistance because these tissues do not rely on insulin to transport glucose into cells.

Inflammatory Effect of Hyperglycemia

Research over the last decade has proven the link between hyperglycemia and the endothelial inflammatory response that results in vascular damage. This response causes both the short-term and long-term complications of diabetes. Research has also shown that prevention of hyperglycemia, even short-term episodes, can have very positive results on health and healing, and the prevention of the long-term complications of diabetes.

Hyperglycemia has been found to cause an inflammatory response that causes leukocytes to invade endothelial tissue (Langouche et al. 2005; Daniele et al., 2014). The endothelial tissue lines the blood and lymph vessels, the heart, and the serous cavities of the body. It is very vascular, and provides essential nutrients and oxygen to cellular tissues. As systemic endothelial tissue damage occurs and perfusion of the endothelial tissues is compromised, the result is decreased transport of oxygen and nutrients, including glucose, to the cells. In addition, vascular permeability increases and capillaries leak fluids, leading to increased coagulation. Microthromboses impede perfusion, and cell damage eventually proceeds to organ failure.

Prevention of hyperglycemia in the hospital, even short-term episodes, can have very positive results on health and healing. Van den Berghe (2003) reports findings from a large randomized study in Leuven, Belgium, of 1,548 surgical intensive care unit (ICU) patients. When blood glucose levels were kept within normal ranges, between 80 and 110 mg/dL, there were 30% to 40% improvements in mortality, 46% reduced risk of severe infections, and reduced use of antibiotics. Many other studies have shown similar results. More recent studies such as Ding and colleagues (2014) continue to find

increased mortality due to organ failure as a result of the vascular damage related to hyperglycemia.

Glucose Target Range

More recent studies have suggested that maintaining the blood glucose slightly higher than normal in the critically ill patient is appropriate. Blood glucose should never be higher than 180 mg/dL or as low as 40 mg/dL. Blood glucose levels that are too high or too low both cause complications and increased mortality. Further discussion on glucose management is found in the Collaborative Care section.

Metabolic Syndrome and Impaired Glucose Tolerance

Metabolic syndrome and impaired glucose tolerance both predispose patients to develop hyperglycemia. Symptoms of these conditions cannot be separated because they cross over in many areas, and both often lead to the development of type 2 diabetes.

Impaired glucose tolerance is also known as prediabetes. This condition is characterized by hyperglycemia. Pro-inflammatory cytokines cause insulin resistance and stimulate the release of the counterregulatory hormones. Random blood glucose readings are sometimes normal, though often a glucose tolerance test will show impaired glucose tolerance. This condition may or may not be present with metabolic syndrome.

Metabolic syndrome, also known as insulin resistance syndrome, includes a group of abnormalities. First, insulin levels are elevated and fasting blood glucose is greater than 110 mg/dL. Insulin receptor sites in the skeletal muscle, fat, and liver become unresponsive to the action of insulin. As this occurs, glucose cannot enter the cells in sufficient quantities. Initially, the pancreas responds by producing larger amounts of insulin. People with this condition are often asymptomatic though they have both hyperglycemia and hyperinsulinemia. Other abnormalities that are present in metabolic syndrome include central obesity, defined as a waist circumference larger than 40 inches or 102 cm in men or larger than 35 inches or 89 cm in women; high triglyceride levels, over 150 mg/dL; high LDL; and low HDL, less than 40 mg/dL in men and less than 50 mg/dL in women. These factors greatly increase the risk of heart disease.

Those at risk of developing metabolic syndrome or impaired glucose tolerance are the same population as those predisposed to type 2 diabetes (Box 14-1). In fact, those who have metabolic syndrome or impaired glucose tolerance usually do develop type 2 diabetes, though the development can be delayed through weight loss and exercise. Metabolic syndrome affects a huge percentage of the population. The CDC (2014) estimates that 37% of U.S.

Table 14-1 Factors That Increase or Decrease Serum Blood Glucose

Factors That May Increase Serum Glucose	Factors That May Decrease Serum Glucose
- Commonly prescribed medications - Calcium channel blockers - Propranolol (Inderal) - Thiazide diuretics (hydrochlorothiazide/HCTZ) - Sympathomimetics (dopamine, dobutamine, epinephrine, norepinephrine, albuterol, and phenylephrine) - Phenytoin (Dilantin) - Glucocorticoids (hydrocortisone, prednisone, dexamethasone) - Feedings - Total parenteral nutrition (TPN) - Enteral/tube feedings - Oral intake of foods and fluids - Change in dialysis or continuous renal replacement therapy (CRRT) schedule - Change in clinical condition	- Advanced age - Malnutrition - Change in dialysis or CRRT schedule - Gastrointestinal malabsorption syndromes - Inflammatory bowel disease - Septic shock - Burns - Alcoholism

adults over the age of 20 and 51% of adults over the age of 65 have pre-diabetes based on lab data of glucose levels. Diabetes in the United States is truly an epidemic!

Focused Assessment of a Patient with the Disorder

An assessment of risk factors related to metabolic syndrome should be accomplished. Box 14-2 summarizes risk factors related to metabolic syndrome.

An assessment should also include identification of other factors that frequently increase or decrease serum blood glucose. Table 14-1 summarizes a list of factors to take into account that may increase or decrease serum blood glucose.

Patients may become hyperglycemic due to the physiologic stress of critical illness. Many of the patients with hyperglycemia resulting from critical illness initially have no signs or symptoms. The typical and easy-to-recognize

clinical signs of thirst and frequent urination that accompany hyperosmolarity have not yet occurred. When blood glucose is less than 200 mg/dL, it does not cause osmotic diuresis. An important nursing consideration includes testing for hyperglycemia and recognizing that hyperglycemia causes vascular disease as well as short term complications such as infection. Hyperglycemic episodes need to be measured and reported to the physician so that treatment can be implemented to prevent complications.

Diagnostic Criteria

It is recommended that all patients in the ICU be screened for hyperglycemia. Normal blood glucose ranges from 70 to 110 mg/dL. Though any result over 110 mg/dL meets the definition of hyperglycemia, most protocols recommend waiting until two or three consecutive readings are over 150 mg/dL before initiating therapy. Many health systems now have protocols in place for the routine assessment of hyperglycemia to prevent complications in surgical patients as shown in Box 14-3.

Both the hemoglobin A_{1C} and the glucose tolerance test help diagnose diabetes. The hemoglobin A_{1C} determines

Box 14-2 Risk Factors Related to Metabolic Syndrome

- Central obesity
 - Waist circumference larger than 40 inches or 102 cm in men
 - Waist circumference larger than 35 inches or 89 cm in women
- Abnormal lipid panel
 - Triglyceride levels over 150 mg/dL
 - High LDL
 - HDL less than 40 mg/dL in men
 - HDL less than 50 mg/dL in women
- Fasting blood glucose is greater than 100 mg/dL
- Hyperinsulinemia

Box 14-3 Assessment of Hyperglycemia

- Identify patients at high risk; those who have three or more signs of metabolic syndrome (Box 14-2)
- Assess blood glucose on admission
- Assess blood glucose postoperatively:
 - In post-anesthesia care area (within an hour of the end of the procedure)
 - 2 hours post-op
 - 4 hours post-op
- Notify physician of two consecutive blood glucose readings higher than 150 mg/dL

Table 14-2 Normal Results of a Four-Hour Glucose Tolerance Test

Time	Serum Glucose mg/dL	Serum Glucose mmol/L
First glucose value—Fasting	70–100 mg/dL	Less than 6.4 mmol/L
30 minutes	Less than 200 mg/dL	Less than 11.1 mmol/L
1 hour	Less than 200 mg/dL	Less than 11.1 mmol/L
2 hours	Less than 140 mg/dL	Less than 7.8 mmol/L
3 hours	70–110 mg/dL	Less than 6.4 mmol/L
4 hours	70–110 mg/dL	Less than 6.4 mmol/L

the amount of glucose in the bloodstream over the life span of the red blood cell, the preceding 100 to 120 days. In other words, it is an accurate measure of blood glucose over the preceding 3 to 4 months. The normal hemoglobin A_{1C} in the nondiabetic adult is 2% to 5%. The glucose tolerance test measures serum blood glucose levels at prescribed intervals following a large dose of oral glucose solution. Normally, blood glucose should not exceed 200 mg/dL during the test. The time of the test varies from 2 to 6 hours. Table 14-2 gives an example of normal results of a 4-hour glucose tolerance test (Kee, 2014).

COLLABORATIVE CARE

Controlling blood glucose can prevent complications, decrease the cost of care, and shorten the length of stay in the intensive care area. In the intensive care area, the nurse is often caring for patients with multiple disorders. There are numerous priorities, assessments and treatments occurring all at the same time. Nurses and physicians who are competent and develop professional relationships including professional communication and collaboration will provide better care. Patient outcomes improve with interprofessional collaboration (Manski-Nankervis et al., 2014)

Insulin Protocols

Blood glucose levels are best controlled and hypoglycemic episodes are avoided when insulin protocols are used. Protocols should be developed through collaboration of a multidisciplinary team. Physicians, nurses, and pharmacists all need to provide input into new insulin protocols. Protocols should be thought out so that they are easy to use, and decisions to change insulin infusion rates can be made quickly and accurately. Rapid decisions regarding titration are important because these decisions will be made each hour. Tools that are user friendly will help to reduce errors in medication calculations and ensure that hyperglycemia is precisely controlled. Outcomes of the use of insulin protocols should be studied to ensure that the intent of providing an easy, rapid, and accurate tool has occurred.

Prevention and Detection of Common or Life-Threatening Complications

Both short- and long-term complications may result from hyperglycemia. Short-term complications of hyperglycemia include higher mortality, increased risk for infection such as sternal wound infections and sepsis, increased risk of acute organ failure, and longer lengths of stay in the ICU. One of the first large studies, The Diabetes and Insulin Glucose Infusion in Acute Myocardial Infarction (DIGAMI) study (Malmberg, Norhammar, Wedel, & Ryden, 1999) found that mortality was significantly lower in patients receiving intensive IV insulin infusions to keep glucose close to normal ranges. Since the late 1990s, many studies have also reported decreased mortality, and decreased complications such as sepsis and acute renal failure, when intensive insulin therapy is used. Other studies list decreased length of stay in ICU and decreased length of mechanical ventilation related to prevention of hyperglycemia with intensive insulin therapy.

Research shows that even short-term episodes of hyperglycemia cause undesirable cellular changes and vascular damage. These changes lead to long-term complications, such as peripheral and autonomic neuropathies, retinopathy, renal dysfunction, and vascular changes, over a period of years. Because it is not yet known how long it takes to cause permanent damage related to hyperglycemia, it is important to maintain normal blood glucose levels during critical illness.

Following an episode of hyperglycemia during a critical illness, follow-up and teaching are important. Follow-up should focus on aggressive management of risk factors associated with metabolic syndrome. Tests to detect the presence of type 2 diabetes should be performed, and if diabetes is present, early management is important. Patient teaching should focus on risk factors for the development of diabetes and how to prevent or delay its onset.

Pathophysiology of Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar Nonketotic Syndrome

Diabetic Ketoacidosis

DKA is caused primarily by insulin deficiency. It was traditionally thought that DKA occurred in type 1 diabetics, though DKA can also occur in type 2 diabetics who produce very little endogenous insulin. In DKA, no insulin is present

in the bloodstream to facilitate the transport of glucose from the bloodstream into the cells. Hyperglycemia is present (usually higher than 300 mg/dL) because glucose continues to be absorbed from the intestines into the bloodstream, and the liver continues to produce glucose through the processes of gluconeogenesis and glycogenolysis. However, the glucose is not transported into the cells because of the lack of insulin. The lack of cellular glucose causes the cells to “starve.” Because the cells are starving, stress hormones are released to induce more glucose production. Stress hormones include glucagon, cortisol, catecholamines such as epinephrine, and growth hormone. These stress hormones induce even more hyperglycemia. Catecholamines also reduce glucose absorption in skeletal muscles.

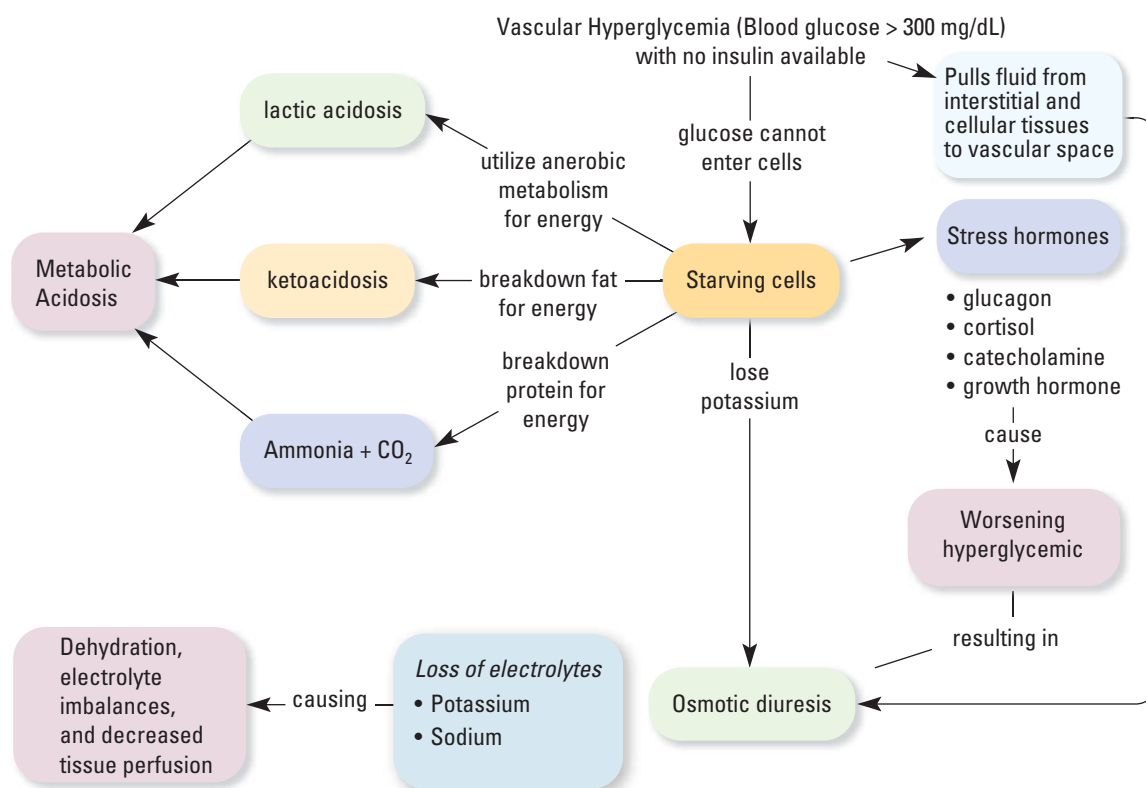
In the presence of hyperglycemia, osmotic diuresis occurs. The increased levels of glucose result in increased osmolarity of the blood. As a result, water is absorbed into the vascular space from the interstitial spaces and even from cellular tissues (such as the brain and other organs) to decrease serum osmolarity (dilute the glucose). Glucose and water are diuresed from the vascular space through the kidneys. Severe diuresis in hyperglycemia results in loss of water, sodium, potassium, magnesium, and phosphates. Osmotic diuresis causes severe dehydration, including cellular dehydration, as well as loss of electrolytes (Visual Map 14-2).

Severe dehydration also causes several other complications. The viscosity of the blood increases as blood volume

decreases, and results in increased probability of platelet aggregation and thromboembolism. Decreased organ perfusion results from dehydration and can rapidly progress to hypovolemic shock with accompanied organ dysfunction. Perfusion to all vital organs and tissues is decreased, causing organ dysfunction and potential acute organ failure. As a result of decreased perfusion, cells will begin anaerobic metabolism. This, in turn, results in the production and buildup of lactic acid, and contributes to metabolic acidosis.

Because there is a lack of glucose at the cellular level, the body begins to break down fats and proteins for use as cellular fuels. The breakdown of fatty acids results in the formation of three ketone bodies: acetoacetate, β -hydroxybutyrate, and acetone. Brain cells cannot use the by-products of fatty acids. Brain cells use only glucose and oxygen for metabolism. Acetoacetate and β -hydroxybutyrate are used for fuel within the cells of organs (other than the brain). Acetone cannot be used in cellular metabolism and is excreted through the kidneys and lungs. When acetone is exhaled a “fruity breath” or “ketone breath” odor is produced, and is a recognized symptom of ketosis. **Ketoacidosis** occurs as a result of buildup of fatty acids. Ketoacidosis causes metabolic acidosis. Both lactic acid from anaerobic cellular metabolism as well as ketoacidosis contribute to metabolic acidosis.

Proteins are also metabolized to create cellular energy during cellular starvation. High levels of circulating cortisol increase protein lysis. The end products of protein metabolism include ATP, carbon dioxide, water, urea, and



Visual Map 14-2 Diabetic Ketoacidosis

ammonia. ATP is used for fuel at the cellular level. Water and carbon dioxide are excreted through the lungs and kidneys. Urea and ammonia are also excreted through the kidneys in the urine.

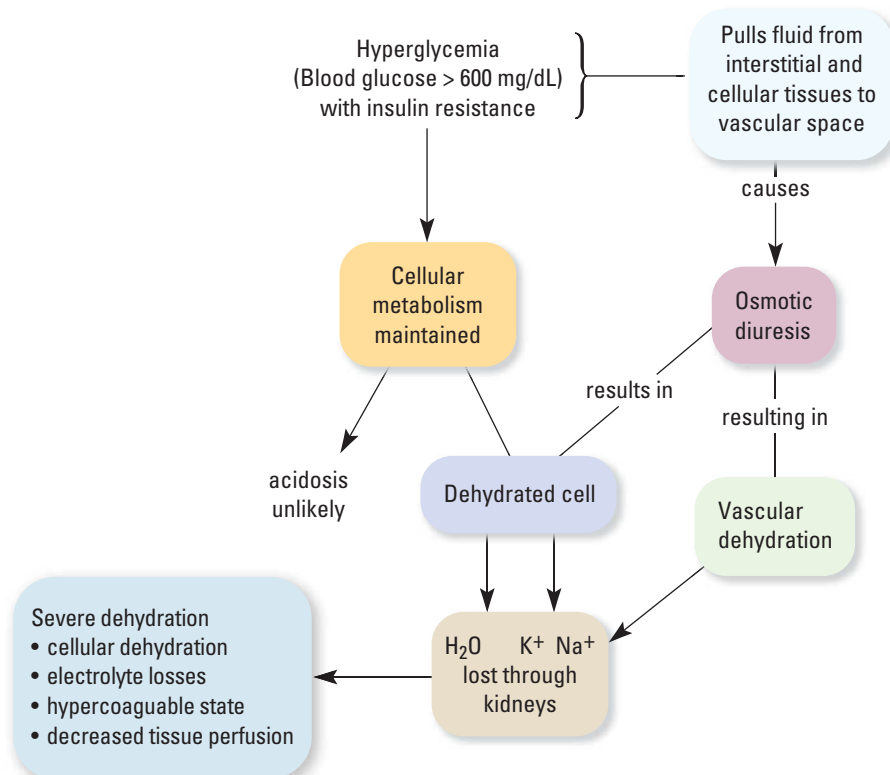
Because organ function is decreased due to decreased perfusion, by-products of glucose, fat, and protein metabolism often build up in the bloodstream. Decreased kidney function results in additional acidosis caused by the buildup of hydrogen ions and ketones, which are normally excreted through the kidneys. Brain function is also impaired. The severe dehydration caused by osmotic diuresis contributes to the loss of cellular fluid in the brain cells. Glucose and oxygen, both vital to brain function, cannot reach brain cells due to lack of insulin and decreased perfusion. All organs are affected by cellular dehydration, lack of perfusion, and lack of glucose. Overall mortality from DKA is 2% to 10%.

Hyperglycemic Hyperosmolar Nonketotic Syndrome (HHNS)

HHNS is common in the patient with type 2 diabetes. It is similar to DKA in that hyperglycemia is present. However, hyperglycemia is often more severe (usually greater than 600 mg/dL). The severe hyperglycemia results in the same physiologic results described in DKA. The increased levels of glucose result in increased osmolarity of the blood. In

the presence of hyperglycemia osmotic diuresis occurs. As a result, water is absorbed into the vascular space from the interstitial spaces and even from cellular tissues (such as the brain and other organs) to decrease vascular serum osmolarity (dilute the glucose). Glucose and water are diuresed from the vascular space through the kidneys. Severe diuresis in hyperglycemia results in loss of water, sodium, potassium, magnesium, and phosphates. Osmotic diuresis causes severe dehydration, including cellular dehydration, as well as loss of electrolytes.

Severe dehydration causes the same complications as seen in DKA. The viscosity of the blood increases as blood volume decreases. Cellular, interstitial, and vascular dehydration, as well as loss of electrolytes, is often more severe in HHNS than in DKA, and is proportional to the levels of vascular glucose. Increased blood viscosity results in increased probability of platelet aggregation and thromboembolism. Decreased organ perfusion results from dehydration and can rapidly progress to hypovolemic shock with accompanied organ dysfunction (see the section “Focused Assessment of a Patient with Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar Nonketotic Syndrome”). Perfusion to all vital organs and tissues is decreased. As a result of decreased perfusion, cells will begin anaerobic metabolism. This, in turn, results in the production and buildup of lactic acid, and contributes to metabolic acidosis. (Visual Map 14-3)



Visual Map 14-3 Hyperglycemic Hyperosmolar Nonketotic Syndrome

A main difference between DKA and HHNS is in HHNS, insulin is present. Insulin facilitates the transport of enough glucose into the cells that the breakdown of fatty acids for cellular energy is minimal. As a result the cells do not starve and often there is no ketosis, or if present, ketosis is mild. Metabolic acidosis, if present, is often mild. However, the overall mortality of HHNS is higher (12% to 46%) and is related to the severe dehydration and associated complications that result from osmotic diuresis.

Precipitating Factors of Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar Nonketotic Syndrome

In a study that spanned over six years, it is not surprising that Lohiya, Kreisberg, and Lohiya (2013) report that the most common cause of admission for DKA is lack of insulin. Almost half of the patients had an infection at the time of admission. The other half omitted insulin for a variety of reasons related to psychosocial and socioeconomic factors. An infection causes a physiologic stress reaction (and resulting stress hormones), that precipitate hyperglycemia. Failure to take additional insulin during the physiologic stress of illness often starts the cascade to DKA or HHNS. The patient who is taking insulin needs to understand the importance of taking extra insulin, and closely monitoring the blood glucose levels. The patient with type 2 diabetes may not even have insulin at home, and therefore may not be able to effectively treat rising blood glucose levels. Because infection is a common cause of DKA and HHNS, it is important for the nurse to evaluate for infection when a patient with DKA or HHNS presents to the hospital. Other common causes of DKA and HHNS include: Initial diagnosis of type 1 diabetes, initial presentation of type 2 diabetes, and poor self-management of the disease.

Nursing Diagnoses

Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar Nonketotic Syndrome

- Deficient fluid volume related to active fluid loss (increased urine output)
- Imbalanced nutrition less than body requirements related to inability to utilize glucose
- Ineffective tissue perfusion related to hypovolemia and interruption in peripheral blood flow
- Lack of knowledge related to the management of diabetes during periods of stress and illness
- Risk for ineffective management of the therapeutic regimen related to complexity of the medical regimen
- Risk for injury related to abnormal blood glucose
- Risk for infection related to abnormal blood glucose and interruption in peripheral blood flow

Conditions that induce stress such as heart attack, cerebral vascular accident, trauma, or surgery also can lead to DKA and HHNS. Pregnancy causes metabolic strain and can induce DKA or HHNS. Medications such as glucocorticoids, thiazide diuretics, calcium channel blockers, propranolol, phenytoin, sympathomimetics, and total parenteral nutrition (TPN) increase the blood glucose and can complicate or precipitate DKA and HHNS. Alcohol and cocaine use are also associated with the development of DKA or HHNS. In addition to the effects of cocaine or alcohol, patients who use these substances are at risk for poor nutrition and poor compliance with the treatment of their diabetes (Box 14-4).

Focused Assessment of a Patient with Diabetic Ketoacidosis and Hyperglycemic Hyperosmolar Nonketotic Syndrome

Some of the symptoms with which the patient with DKA or HHNS presents are similar. All patients will present with some degree of dehydration. The following assessment findings indicate dehydration: poor skin turgor, sunken appearance of the eyes, dry mouth and mucous membranes, tachycardia, orthostatic drop in blood pressures, and low urine output. Urine output may be an inaccurate method to measure the degree of dehydration because the patient may continue to have high urine output even in the presence of dehydration as a result of osmotic diuresis. All patients also present with hyperglycemia. The hallmark symptoms that indicate hyperglycemia include thirst and frequent urination. Patients with both DKA and HHNS should be assessed for presence of infection. Increased temperature and increased white blood cell (WBC) count are signs of infection. The source of infection should be identified. Common sources of infection include urinary tract infections, pneumonia, and even tooth abscesses. The skin should also be inspected for signs of a wound or skin infection. Culture and sensitivity of the source of infection (e.g., sputum cultures, urine cultures, or wound cultures) will help identify the type of infection and aid in identifying effective antibiotics. Blood cultures may also be completed to assess for sepsis. Patients with high blood glucose are at increased risk for sepsis, or sepsis may be a precipitating factor of DKA and HHNS. Change in level of consciousness and changes in mental status are important assessments that correlate with the degree of hyperosmolality. Assessment for deep vein thrombosis and other thromboses is important because of the increased blood viscosity. Patients with fluid and electrolyte imbalances may exhibit leg cramps and ECG changes.

The presentation of the patient with DKA may differ slightly from the presentation of the patient with HHNS.

Box 14-4 Precipitating Factors for DKA and HHNS

Infection	Commonly prescribed medications
• May be complicated by the patient failing to take adequate insulin	• Calcium channel blockers
New onset of diabetes	• Propranolol (Inderal)
• DKA results from new onset of type 1 diabetes • HHNS results from new onset of type 2 diabetes	• Thiazide diuretics (hydrochlorothiazide [HCTZ]) • Sympathomimetics (dopamine, dobutamine, epinephrine, norepinephrine, albuterol, and phenylephrine)
Conditions that induce a stress reaction	• Phenytoin (Dilantin) • Glucocorticoids (hydrocortisone, prednisone, dexamethasone) • Total parenteral nutrition (TPN)
• Heart attack • Stroke • Trauma • Surgery • Pregnancy	Other toxins
	• Alcohol • Cocaine

The patient with type 1 diabetes often presents in metabolic acidosis. Signs and symptoms that need to be assessed for include gastrointestinal symptoms such as loss of appetite, abdominal pain, and nausea and vomiting. These symptoms sometimes make the patient appear to have an acutely inflamed abdomen. The cause of these symptoms is actually related to decreased perfusion of the mesentery. **Kussmaul respirations** are also present in metabolic acidosis. These deep and rapid respirations decrease carbon dioxide in order to compensate for metabolic acidosis. Ketosis is also a sign of DKA. Positive assessment findings for ketosis include acetone or fruity odor of the breath, and positive urine ketones.

The patient in HHNS should be assessed for visual disturbances, blurred vision, and weight loss. These are signs of severe dehydration complicated by high blood osmolality and high blood glucose.

The patient with DKA or HHNS may present with mild, moderate, or severe symptoms. Some patients will be ambulatory and able to give an accurate history. Others will be comatose, in shock, and close to death. The patient may present in either hypovolemic or septic shock. General

signs of shock include tachycardia and low blood pressure. Shock also includes decreased perfusion of organs and is reflected through organ failure. Assessments include function of each organ and laboratory findings indicating failure. For instance, renal failure is evidenced through urine output less than 0.5 cc/kg/hr, decreased glomerular filtration rate, and increased creatinine. Table 14-3 differentiates the symptoms of DKA and HHNS.

Diagnostic Criteria

Laboratory criteria that are important in the diagnosis of DKA include blood glucose higher than 300 mg/dL, but often over 500 mg/dL. In HHNS, the blood glucose is often higher than 600 mg/dL, and can be 1000 mg/dL or higher. The serum osmolality will be increased and is proportional to the amount of blood glucose.

Serum osmolality is equal to $(2 \times \text{sodium}) + (\text{serum glucose}/18) + (\text{BUN}/2.8)$. Normal serum osmolality is 280 to 300 mmol/L. In DKA and HHNS, patients typically have a serum osmolality over 300 mmol/L. In HHNS, serum osmolality is often higher than in DKA as a result of

Table 14-3 Differentiating the Symptoms of DKA and HHNS

Assessments Indicating DKA	Assessments Indicating HHNS
• Dehydration: poor skin turgor, sunken appearance of the eyes, dry mouth and mucous membranes, tachycardia, orthostatic drop in blood pressures, and low urine output • Hyperglycemia: thirst and frequent urination • Presence of infection: fever, increased WBC • Ketosis: fruity odor to the breath, positive urine ketones • Metabolic acidosis: loss of appetite, abdominal pain, nausea and vomiting, and rapid deep respirations (Kussmaul respirations) • High osmolality and dehydration: mental status changes, loss of consciousness, increased blood viscosity • Fluid and electrolyte abnormalities: abnormal labs, leg cramps, and ECG changes • Shock: tachycardia, low blood pressure, and signs of organ failure	• Dehydration: poor skin turgor, sunken appearance of the eyes, dry mouth and mucous membranes, tachycardia, orthostatic drop in blood pressures, and low urine output • Hyperglycemia: thirst and frequent urination • Presence of infection: fever, increased WBC • High osmolality and dehydration: visual disturbances, blurred vision, weight loss, mental status changes, loss of consciousness, and increased blood viscosity • Fluid and electrolyte abnormalities: abnormal labs, leg cramps, and ECG changes • Shock: tachycardia, low blood pressure, and signs of organ failure

Table 14-4 Calculating Serum Osmolarity

1. Na ⁺ 140 K ⁺ 3.8 Glucose 90 BUN 15	$(2 \times 140) + (90/18) + (15/2.8) = (280) + (5) + (5.36) = 290.36$ mmol/liter (normal)
2. Na ⁺ 130 K ⁺ 6.2 Glucose 604 BUN 36	$(2 \times 130) + (604/18) + (36/2.8) = (260) + (33.5) + (12.8) = 306$ mmol/liter (elevated)
3. Na ⁺ 128 K ⁺ 4.5 Glucose 990 BUN 40	$(2 \times 128) + (990/18) + (40/2.8) = (256) + (55) + (14.29) = 325$ mmol/liter (very elevated)

Table 14-5 Calculating Anion Gap

1. Na ⁺ 140 K ⁺ 3.8 CL 102 Bicarb 30	$(140 + 3.8) - (102 + 30) = (143.8) - (132) = 11.8$ mEq/L (normal)
2. Na ⁺ 132 K ⁺ 4.4 CL 100 Bicarb 17	$(132 + 4.4) - (100 + 17) = (136.4) - (117) = 19.4$ mEq/L (metabolic acidosis)

increased serum glucose. Table 14-4 illustrates how serum osmolarity becomes more critical as serum glucose rises. The first calculation is based on normal laboratory values, and the serum osmolarity is normal. The second calculation represents laboratory values typically seen in DKA. The third example, typical of a patient with HHNS, illustrates how critical osmolarity can become.

Both the anion gap and arterial blood gases will show the presence of acidosis. Metabolic acidosis is profound in DKA and can be life threatening, though there is often little to no acidosis in HHNS. The anion gap is calculated to show the presence of metabolic acidosis in DKA. The normal anion gap is 10 to 17 mEq/L and is found using the following equation: (sodium + potassium) – (chloride + bicarbonate). An anion gap that is higher than normal is evidence of metabolic acidosis (Kee, 2014). Arterial blood gases will also show the presence of metabolic acidosis. Serum pH will be lower than 7.35, showing acidity. The pO₂ may be normal or low if the patient is comatose and breathing shallowly. The pCO₂ will be low as the patient attempts to blow off carbon dioxide (CO₂) to compensate for acidosis. Bicarbonate is also often low because the patient has used up bicarbonate stores in an attempt to correct acidosis (Table 14-5).

Reflect On

Calculate the anion gap with the following data: Na⁺ 134, K⁺ 5.0, CL 91, Bicarb 16. Evaluate the result and consider what treatments will help to correct it.

Other laboratory findings that are significant include electrolytes, a complete blood count (CBC), cultures, urine studies, and clotting studies. Initial electrolyte findings often reveal high potassium even though intracellular potassium is often depleted. Once the patient is treated with insulin, potassium moves rapidly toward the intracellular space, and serum potassium is often very low (Kee, 2014). The sodium, magnesium, phosphorus, and calcium are also often low due to osmotic diuresis. Electrolytes are evaluated often until they are within normal limits. A CBC will show high WBCs if infection is present and also can be

used to determine the extent of dehydration. Blood cultures should be anticipated if the patient is febrile to rule out sepsis. Urine is collected and tested for **glycosuria** and **ketonuria**. Urine cultures will detect the presence of urinary tract infection. Sputum cultures may be done if the patient has signs of pneumonia. If pulmonary congestion is heard on auscultation, a chest x-ray should be anticipated to rule out pneumonia or other signs of infection. If wounds are present, wound cultures should also be completed prior to the initiation of antibiotics. Clotting studies such as a prothrombin time (PT), partial thromboplastin time (PTT), and a D-Dimer are accomplished to detect increased potential of blood clots.

Kidney function is evaluated through the findings of glomerular filtration, serum creatinine, and BUN. In renal failure, both BUN and creatinine will be elevated. If the glomerular filtration rate is decreased, the findings indicate a renal insult or renal failure. Liver function is also analyzed through analysis of liver enzymes. Aspartate aminotransferase (AST), and alanine aminotransferase (ALT), will be elevated in liver failure (Kee, 2014). Lactate dehydrogenase (LDH) will be more elevated in DKA, not only as a result of liver dysfunction but also as a result of lactic acid production seen in anaerobic metabolism.

Additional laboratory studies are aimed specifically at identifying the precipitating cause of DKA or HHNS. Cardiac enzymes are completed to rule out acute myocardial infarction. A computerized tomography (CT) scan of the head may be done to rule out a stroke. A pregnancy test should be completed on any premenopausal female. As previously mentioned, cultures should be completed to identify infections. A toxic screen should be anticipated to identify presence of illegal drugs or alcohol. Levels of prescribed medications, especially those that precipitate DKA and HHNS, are also analyzed.

Monitoring the heart rhythm using ECG is also an essential diagnostic tool. Findings from ECG monitoring can help to identify cardiovascular dysfunction as a result of electrolyte imbalances. Sodium, potassium, calcium, and magnesium all participate in cardiovascular function. A sign of elevated serum potassium is high, peaked T waves. A sign of hypokalemia is U waves. If the patient has

been prescribed calcium channel blockers or beta blockers, they will be unable to clear the circulation due to decreased perfusion to the liver and kidney, resulting in toxicity. A low heart rate and potential heart blocks may result.

COLLABORATIVE CARE

Both DKA and HHNS are life-threatening emergencies. Initial therapies are aimed at correcting conditions that are a threat to life. Collaborative management includes fluid therapy, insulin therapy, correction of electrolytes, correction of acidosis, and treatment of the precipitating cause. When the patient is stable, collaborative management changes to focus on maintaining a stable state, preventing another episode, and teaching the patient how to manage diabetes as well as complications that arise. Although DKA is complicated by metabolic acidosis, the treatments for both DKA and HHNS are similar.

Resuscitation

An important first step in the care of the patient experiencing DKA or HHNS is to evaluate and manage the airway and breathing. If oxygenation is inadequate, then oxygen would be supplemented. External oxygen equipment such as a cannula or mask could be used if ventilation is adequate. Intubation with ventilation will be necessary if ventilation is inadequate. Continuous assessment of vital signs, pulse oximetry and EKG monitoring would be anticipated.

Fluid Therapy

Fluid therapy is vital to correct vascular volume as well as interstitial and cellular fluid deficits. Perfusion to vital organs is increased as fluid volume increases. The kidneys need to function well to clear metabolites such as ketone bodies. Hydration also improves the effectiveness of insulin and decreases blood viscosity.

In severe dehydration or hypovolemic shock, large volumes of IV fluid will be necessary to correct volume deficits. Two IV sites are recommended for fluid resuscitation. A common calculation used to estimate fluid loss is 100 mL/kg of weight. Table 14-6 presents an example of

Table 14-6 Example of Fluid Replacement in Severe Dehydration

Patient weight = 165 pounds (75 kg)	Fluid replacement – 100 mL/kg, equals 7500 mL in the first 24 hours
First hour: IV rate = 1,000 mL/hr	Total: 1000 mL
Hours 2–8 (7 hours): IV rate = 500 mL/hr	+ 3500 mL = total: 4500 mL
Hours 9–24 (16 hours): IV rate = 200 mL/hr	+ 3200 mL = total 7700 mL

Table 14-7 Calculating Corrected Serum Sodium

1. Na ⁺ 128 Glucose 400	$128 + \{[(400 - 100)/100] \times 1.6\}$ $\times 1.6\} = 128 + \{[300/100] \times 1.6\}$ $= 128 + 4.8 = 132.8$	IV Fluid Choice: Normal saline (0.9% NS)
2. Na ⁺ 128 Glucose 990	$128 + \{[(990 - 100)/100] \times 1.6\}$ $\times 1.6\} = 128 + \{[890/100] \times 1.6\}$ $= 128 + 14.24 = 142.24$	IV Fluid Choice: Half- normal saline (0.45% NS)

fluid replacement based on estimated losses in severe dehydration. It should be noted that following 8 hours of therapy, the patient has received approximately one half of the total fluid replacement. The second half of the volume is given in the remaining 16 hours.

Normal saline (0.9%) is the intravenous (IV) fluid used most often when a patient is in shock because it is retained in the intravascular space, and it replaces severe sodium losses. Once the blood pressure returns to normal, and minimum urine output is 0.5 mL/kg/hr, the IV fluid is often changed to half-normal saline (0.45% NS). Another method to determine whether to use normal saline (0.9% NS) or half-normal saline (0.45% NS) is to calculate the corrected serum sodium (CSS). The CSS = Serum Na⁺ + $\{[(\text{Serum glucose (mg/dL)} - 100)/100] \times 1.6\}$. The determination of which IV fluid to use is based on the corrected serum sodium result. Normal serum sodium is 135 to 145 mEq/L. If the CSS is high or normal, half-normal saline (0.45% NS) is used. If the CSS is low, normal saline (0.9% NS) will help to correct sodium losses. Table 14-7 presents two examples of the calculation of CSS.

Essential for Safety

Renal function, serum creatinine, and glomerular filtration rate (GFR) needs to be assessed prior to instituting large amounts of IV fluids. Rapid administration of IV fluid can cause rapid fluid shifts and can result in edema in vital organs such as the brain and the lungs.

In addition to replacing total body water deficits, hourly fluid losses due to osmotic diuresis need to be considered. If hourly urine output is extremely high, the patient will continue to lose fluid even though IV fluid is being administered. The volume lost each hour in urine may be measured, and the hourly IV fluid rate is calculated to add volume lost in urine output. Osmotic diuresis will decrease as the serum glucose decreases. Dextrose (5%) is added to the IV fluid to help prevent hypoglycemia when the serum glucose drops below 250 mg/dL to 300 mg/dL.

The example of fluid volume replacement (above) may be more volume than some patients need or could tolerate. It should be noted that the example in Table 14-6 illustrates

that the patient weighing 165 pounds (75 kg) would receive 7500 mL of fluid in the first 24 hours of therapy. Patients present with varying degrees of dehydration, and fluid therapy needs to be tailored to the level of dehydration. Rapid administration of IV fluid can cause rapid fluid shifts and can result in edema in vital organs such as the brain and the lungs. In addition, preexisting medical conditions such as renal failure, heart failure, and pulmonary disease also need to be considered prior to instituting large amounts of IV fluids. Hemodynamic monitoring may be indicated to measure central venous pressure (CVP), pulmonary artery pressure (PAP), pulmonary artery wedge pressure (PAWP), cardiac output (CO), cardiac index (CI), mixed venous oxygen saturation (SVO₂), and systemic vascular resistance (SVR). The data collected can help determine the rate of fluid volume tolerated for the patient with preexisting renal, heart, or pulmonary disease. The nurse should be vigilant to the signs of fluid volume overload such as pulmonary congestion, neck vein distention, increasing CVP and PAWP, and generalized edema.

In addition to hemodynamic monitoring, assessment of the outcome of fluid replacement includes frequent vital signs. Vital signs are done every hour at the minimum, though they may be much more frequent until the patient is initially stabilized. Strict intake and output is also essential. Daily weights will accurately measure fluid volume increases or losses over a 24-hour period. Neurological assessments are also important. Decreasing level of consciousness and headache may be signs of cerebral edema.

Reflect On

A 77-year-old woman was admitted with HHNS related to her diabetes. She also has a history of heart failure. Consider how you would manage her fluid correction in the presence of heart failure.

Insulin Therapy

Insulin therapy facilitates the transport of glucose in the cells. The glucose promotes normal cellular metabolism. When glucose is present, fat and protein breakdown for the production of cellular energy will stop. Toxic metabolites such as ketoacids and lactic acids will also stop being produced.

The standard of care is to administer regular insulin IV until blood glucose returns close to normal limits. Subcutaneous (SQ) insulin therapies, including insulin pumps, are stopped until the blood glucose is stabilized. Prior to administering insulin, it is important to identify and correct hypokalemia to prevent an unsafe serum potassium level. It is also essential to know renal function to manage fluid and electrolyte replacement. A bolus of IV insulin is common prior to starting an insulin infusion. A common bolus dose is equal to 0.15 unit/kg. Insulin infusions are often started at 0.1 unit/kg/hr. Using these calculations, an adult weighing 176 pounds, or 80 kg, would require a bolus dose of 12 units of insulin, and an initial infusion starting at 8 units per hour.

Regular insulin is most commonly used for IV infusions. After the first hour and each hour thereafter, the rate

Commonly Used Medications

Insulin

Regular Insulin

Regular insulin, also known as Humulin R, Novolin R, Velosulin, Velosulin BR, and Velosulin Human, is a hormone. It is a short-acting insulin that travels into the various body tissues (in the muscle, fat, and liver) binding to insulin receptor sites within the cell.

It lowers blood glucose by transporting glucose from the bloodstream into the cell, and is metabolized primarily in the liver.

Dose and Desired Effect: Given by IV route in critically ill patients, usually as a bolus infusion of 0.15 unit/kg followed by an infusion of 0.1 unit/kg/hour, the insulin should have an onset of effect within 15 to 30 minutes and a peak of 2 to 3 hours. It should decrease the serum glucose by 70 to 100 mg/dL/hour.

Nursing Responsibilities: Always use an insulin syringe to measure insulin doses.

- Bolus may be administered undiluted over 1 minute.
- Note the insulin infusion should be diluted in 0.45% NS or 0.9% NS.
- Prior to initiating the IV infusion, waste 25 to 50 mL through the new IV tubing because insulin adheres to the IV tubing.
- Measure the blood glucose hourly during IV insulin infusions.
- When the blood glucose reaches 250 to 300 mg/dL, the rate of administration of insulin is either stopped and transitioned to SQ insulin or decreased by half to 0.05 unit/kg/hr until signs of acidosis have resolved.

Potential Side and/or Toxic Effects: Adverse effects include hypoglycemia, which is common, and anaphylaxis, which is rare (Wilson, Shannon & Shields, 2016).

Safety Initiatives

Prevent Harm from High-Alert Medication: Insulin

PURPOSE: To prevent harm from high-alert medications by implementing the changes in care recommended in the Institute for Healthcare Improvement (IHI) guide. High-alert (or high-hazard) medications are medications that are likely to cause significant harm to the patient, even when used as intended.

RATIONALE: Insulin has resulted in improved quality and length of life for diabetics. However, mismanagement can result in hyperglycemia or hypoglycemia, both of which are harmful to the patient. Hypoglycemia is the most common complication of any insulin therapy in a hospital; one study reported 56 occurrences of hypoglycemia in 100 treatment days.

HIGHLIGHTS OF RECOMMENDATIONS: Changes designed to ensure standardization, such as eliminating the use of sliding insulin dosage scales or standardizing the scale through the use of one preprinted protocol that clearly

designates the specific increments of insulin coverage, and using a single concentration of IV infusion insulin that is prepared in the pharmacy. Requires an independent double-check of the drug, concentration, dose, pump settings, route of administration, and patient identity before administering all IV insulin.

Changes designed to ensure better partnering with patients and families, such as encouraging patient self-management and patient questions about doses and timing of insulin administration. It is even better to have patients manage their own insulin and coordinate meal and insulin times during hospitalization if they are capable.

Source: Institute for Healthcare Improvement. (2012). *How-to guide: Prevent harm from high-alert medications*. Retrieved July 30, 2015, from <http://www.ihl.org/resources/Pages/Tools/HowtoGuidePreventHarmfromHighAlertMedications.aspx>

of infusion is adjusted based on hourly bedside blood glucose measurements. The goal is to decrease serum glucose by 70–100 mg/dL/hr. Complications result if insulin is infused too rapidly. Quick drops in blood glucose may result in hypoglycemia as well as a rapid shift in potassium from the serum into the intracellular compartment. This rapid electrolyte shift can cause hypokalemia and result in life-threatening cardiac arrhythmias. Conversely, if serum glucose does not decrease by 70–100 mg/dL in the first hour, insulin doses are doubled or additional bolus doses of IV insulin may be administered.

Once the serum glucose reaches 250 to 300 mg/dL, the insulin infusion is either transitioned to the SQ route or decreased by half to 0.05 unit/kg/hr. If there is little to no acidosis present, IV insulin may be transitioned to SQ insulin. IV insulin is normally discontinued 1 to 2 hours after the initial dose of SQ insulin. This transition prevents hypoglycemia or hyperglycemia. If acidosis is present, IV insulin is continued until acidosis is beginning to resolve, which is defined by bicarbonate greater than 18 mEq/L, venous pH greater than 7.3, normal anion gap, and no ketones in the urine.

Management of Glucose

Blood glucose levels in the intensive care setting are managed at levels slightly higher than normal. Numerous research studies have analyzed glucose control in various patient populations. Original studies such as the DIGAMI study (Malmberg et al., 1999) in the 1990s proved that high glucose levels increased mortality. Since that time, many research studies have shown increased mortality with

blood glucose levels higher than 140 mg/dL. The systemic inflammatory changes that occur with even short term hyperglycemia cause damage in the endothelial tissues and lead to cell and organ damage. At the same time, glucose levels that dip below 40 mg/dL due to intensive insulin therapy also increase mortality. In 2009, results of the NICE-SUGAR study found that maintaining glucose levels between 140 and 180 mg/dL decreased mortality in critically ill patients, and found a higher incidence of mortality with intensive glucose control where glucose was maintained between 80 and 108 mg/dL (NICE-SUGAR Study Investigators et al., 2009). The 2015 clinical practice guidelines of the American Association of Clinical Endocrinologists (AACE) published by Handelsman et al. (2015) recommend to maintain glucose levels of critically ill diabetic patients between 140 and 180 mg/dL. The guidelines further recommend to keep serum glucose levels closer to 140 mg/dL, the lower end of 140 and 180 mg/dL.

Correction of Electrolytes and Acidosis

Fluid therapy corrects dehydration and increases perfusion to vital organs. Insulin therapy corrects hyperglycemia and ketosis, and then acidosis begins to resolve. The next step is correction of abnormal electrolyte levels to prevent life-threatening cardiac arrhythmias, promote cellular metabolism, improve smooth and skeletal muscle function, and maintain endocrine function. Although fluid therapy, insulin therapy, and correction of electrolytes are discussed separately and in detail, it is important to mention that treatment is rapid and often concurrent in the clinical setting.

Essential for Safety

Electrolyte abnormalities can cause life-threatening cardiac arrhythmias. It is therefore essential to monitor the ECG rhythm during treatment of hyperglycemic emergencies.

Serum potassium may be high, normal, or low in the patient presenting with DKA or HHNS. High serum osmolarity pulls potassium into the vascular space, making serum potassium levels high. Once in the vascular space potassium is lost through the urine due to osmotic diuresis, making serum potassium levels normal or low. It is important to remember that patients who present with normal or even slightly elevated serum potassium have intracellular potassium deficits.

Essential for Safety

The nurse must pay strict attention to the serum potassium level since the level may fluctuate dramatically. Initially, serum potassium may be high causing cardiac arrhythmias and a heightened T wave on the ECG waveform. However, serum potassium often drops rapidly with fluid administration and insulin therapy. The nurse will expect to replace potassium as potassium shifts back into the intracellular compartment. See "Commonly Used Medications: Potassium" for details.

In DKA, the additional problems of anaerobic cellular metabolism and acidosis cause potassium to shift from intracellular to extracellular (vascular and interstitial) spaces. Replacement of potassium will depend on initial

potassium levels. In addition, renal function must be adequate prior to administering potassium. Serum potassium often drops rapidly with fluid administration and insulin therapy as potassium shifts back into the intracellular compartment. The goal is to keep serum potassium levels between 4 and 5 mEq/L. It is common to give as much as 100 to 200 mEq of potassium over the first 24 hours to treat intracellular deficits.

In addition to potassium, serum sodium, calcium, and magnesium are also low in the patient presenting with DKA or HHNS as a result of losses due to osmotic diuresis. Sodium losses are replaced using the calculated corrected sodium as discussed previously. It is also important to replace calcium and magnesium if levels are very low. All of these electrolytes (sodium, potassium, calcium, and magnesium) are important to cellular function and uptake of glucose, skeletal and smooth muscle function, CNS function, and endocrine function.

Low phosphate can contribute to respiratory depression, muscle weakness, and cardiac arrhythmias. Research has not demonstrated a clinical benefit to the routine administration of phosphate, however it is replaced often with potassium (potassium phosphate) when levels are low. Giving too much phosphate can cause low serum calcium.

Bicarbonate therapy is reserved for those patients with severe acidosis (those patients with DKA). Giving too much bicarbonate can cause rebound alkalosis, hypokalemia, hypernatremia, elevated lactate, paradoxical cellular acidosis, and slowed improvement of ketosis. In addition, there has not been a researched improvement in morbidity

Commonly Used Medications

Potassium

Potassium chloride and potassium phosphate are electrolyte replacements. Potassium, an intracellular electrolyte, is essential for nerve conduction, muscle contraction (cardiac, skeletal, and smooth), maintenance of normal kidney function, and maintenance of normal enzyme action. Potassium also plays a role in acid-base balance.

Dose and Desired Effect: Given intravenously as a usual dose of 10 mEq/hr, or a high dose of 20–40 mEq/hr and diluted to 10–20 mEq per 100 mL of solution. The desired effect is a slow increase in serum potassium. The goal is to keep serum potassium levels between 4 and 5 mEq/L.

Nursing Responsibilities:

- NEVER give potassium IV push or in any concentrated form. Rapid infusion may cause fatal arrhythmias.
- Always use an IV infusion pump to administer potassium.

- Never add potassium to an IV that is infusing. After adding potassium to an IV bag, invert it several times to ensure even distribution of potassium in solution.
- If possible, infuse into a central vein to prevent pain at the IV site and phlebitis.
- Assess renal function prior to administration of potassium because impaired renal function may cause severe hyperkalemia.
- Monitor the ECG of patients receiving infusions exceeding 10 mEq/hr.
- Monitor serum electrolytes frequently, usually 6 hours post-infusion.

Potential Side and/or Toxic Effects: Adverse effects include hyperkalemia, respiratory distress, cardiac depression, arrhythmias, cardiac arrest, and flaccid paralysis.

and mortality by administering bicarbonate to patients with pH as low as 6.9 to 7.1. On the other hand, treating severe acidosis is helpful because it promotes insulin absorption, improves hemodynamic functioning, raises the threshold for ventricular fibrillation, improves CNS functioning, and improves organ function. Therefore, treatment of acidosis with bicarbonate is typically reserved for those patients with a pH less than 7.0. It is recommended that bicarbonate be mixed in an IV solution of water or hypotonic saline (0.45% NS) so that it is isotonic. Bicarbonate is then administered slowly, over an hour.

Electrolytes and arterial or venous blood gases are typically measured every hour or two initially. Neurological assessments, strength, orientation, and mentation should all be assessed hourly. If sodium levels are low, seizure precautions are appropriate. Frequent monitoring is essential because levels change rapidly as fluids and insulin are administered. Acidosis will resolve as metabolism returns to normal. Maintaining adequate electrolyte levels is important to cellular metabolism, organ function, neuromuscular function, and endocrine function.

Treatment of the Precipitating Cause

Treatment of the initial precipitating cause is essential. Underlying medical conditions such as myocardial infarction, drug toxicity, or sepsis can be life threatening, and are often the precipitating factor for DKA and HHNS. Other precipitating factors can cause serious complications. A careful admission history is paramount to determine if the patient has signs of infection or is not aware of having diabetes, such as a newly diagnosed diabetic. Because infection is a common precipitating factor to the development of DKA or HHNS, it is important to start appropriate treatment immediately. If infection is suspected, broad-spectrum antibiotics including antibacterials and antifungal agents are often started. The culture and sensitivity results will be available 24 to 72 hours after the initial culture.

Essential for Evidence-Based Practice

Infection is the most common precipitating cause of DKA and HHNS. An important nursing role is to culture potential sites of infection using proper technique, and to collaborate with the laboratory to ensure that cultures are in the best environment to grow (deliver cultures to the laboratory rapidly). Cultures need to be obtained prior to the administration of antimicrobial medications.

Cardiovascular disease (myocardial infarction and heart failure) is the leading cause of death in the United States, and diabetics who have experienced hyperglycemia are at increased risk for cardiovascular diseases. In addition to myocardial infarction and heart failure, this includes

stroke, deep vein thromboses, and other thromboembolisms, as well as decreased arterial perfusion to organs. One common result of decreased arterial perfusion to the kidneys is partial or total renal failure. The admission history should be focused to assess for potential cardiovascular disease and any history of organ failure. All precipitating causes of DKA and HHNS are treated in the usual ways. However, as a result of the presence of DKA or HHNS plus other comorbidities, morbidity and mortality rates are increased.

Commonly prescribed medications such as calcium channel blockers, propranolol, thiazide diuretics, sympathomimetics, phenytoin, and steroids may be used during the treatment or may have been used prior to admission for DKA or HHNS. Calcium channel blockers and propranolol are used to treat hypertension. Thiazide diuretics are used to treat preexisting heart failure. Sympathomimetics such as dopamine, dobutamine, epinephrine, or norepinephrine may be used to improve blood pressure and cardiac output in the critically ill. Albuterol is often used as an inhalant in patients with asthma, and phenylephrine is an ingredient in some over-the-counter cold medications. Phenytoin is a medication used to prevent seizures. Glucocorticoids such as hydrocortisone, prednisone, or dexamethasone may be topical for skin disorders, inhaled for treatment of inflammatory pulmonary conditions, or taken orally to decrease a variety of inflammatory responses. All of the medications listed complicate the treatment of DKA and HHNS because they raise the blood glucose. If they are given concurrently during treatment more insulin may be needed to achieve expected decreases in serum glucose levels. It is important to have an accurate list of preadmission medications, including prescribed medications as well as over-the-counter medications, vitamins, and herbals.

NURSING ACTIONS

When the patient is no longer critically ill, collaborative management changes focus to maintaining stability, preventing another episode, and teaching the patient how to manage diabetes as well as complications that arise. Although it is important to begin teaching as soon as the patient is ready, it is even more important to wait until the patient is no longer critically ill. An assessment of knowledge needs to be completed because each patient has different levels of knowledge and different teaching needs. In order to individualize the teaching plan, knowledge of the following topics needs to be assessed:

- Basic knowledge about diabetes
- Monitoring blood glucose
- Recognition and treatment of hypoglycemia and hyperglycemia

- Diet, including adjustments during high and low blood sugars
- Exercise, including adjustments during high and low blood sugars
- Awareness of all aspects of medications used to treat diabetes
- Proficiency in insulin administration
- Insight into diabetes complications and how to prevent them
- Knowledge of how to manage diabetes during illnesses
- Awareness of the specific precipitating factor that caused the present DKA or HHNS episode and a plan to prevent future occurrences

Because infection is the most common precipitating cause of DKA and HHNS (20% to 25%), it is important to understand diabetes care during acute illness. Box 14-5 presents guidelines recommended by the ADA (2015a) for diabetes management at home during acute illness.

In addition to assessing the patient's knowledge, it is important to positively reinforce or strengthen desirable behaviors, while coaching to improve undesirable habits. In order to determine past compliance, the hemoglobin A_{1C} is used. This blood test determines the amount of glucose in the bloodstream over the life span of the red blood cell, which is the preceding 100 to 120 days. In other words, the hemoglobin A_{1C} is an accurate measure of blood glucose over the preceding 3 to 4 months! Normal hemoglobin A_{1C} is 2% to 5%. The most recent recommendations from the ADA (2015a) advise that patients with diabetes keep their hemoglobin A_{1C} less than 7%. Additionally, diabetics

Table 14-8 Correlation of Hemoglobin A_{1C} to Blood Glucose

Hemoglobin A _{1C}	Blood Glucose mg/dL	Blood Glucose mmol/L
6%	135	7.5
7%	170	9.5
8%	205	11.5
9%	240	13.5
10%	275	15.5
11%	310	17.5
12%	345	19.5

should keep their blood glucose levels as close to normal as possible, within 90 to 130 mg/dL, and peak blood glucose results should not exceed 180 mg/dL. These recommendations are based on many well-conducted research studies. Research has shown that patients with lower blood glucose and hemoglobin A_{1C} levels are healthier and have fewer complications. Even the cost of diabetes care is less for those with better glucose control. Table 14-8 correlates the results of hemoglobin A_{1C} with serum blood glucose results.

Essential for Patient-Centered Care

It is essential that the patient understands how to prevent severe hyperglycemic emergencies. The nurse can use various resources to teach the patient how to manage diabetes during acute illness. It is essential for the type 1 diabetic to continue to take insulin and frequently monitor blood glucose during periods of illness.

Box 14-5 Diabetes Management During Acute Illness

1. Increase frequency of blood glucose checks.
2. Take your medications.
 - Continue to take oral medications if blood sugar is high.
 - If you take insulin, continue taking it. The dose or type of insulin may be adjusted. For instance, some individuals may switch to using only short-acting insulin on a sliding scale. This scale is prescribed by the doctor.
3. Check urine for ketones.
4. Eat!
 - If you are vomiting, you still need to eat 15 grams of carbohydrates every hour. There are many examples of foods that contain 15 grams of carbohydrates available from the ADA.
 - If blood glucose is high, drink fluids with no calories.
5. Watch over-the-counter medications. Many contain sugar or other ingredients that raise your blood sugar. Others, such as aspirin, may lower your blood sugar. Recommendations

regarding over-the-counter medications should come from your doctor.

6. Make a "sick day" bag. Include a blood glucose chart, a urine ketone chart, sliding scale insulin recommendations, a list of foods that contain 15 grams of carbohydrates, and the name and phone number of your physician(s).

When to call the doctor:

1. High blood glucose readings, over 240
2. Vomiting or diarrhea for more than 6 hours
3. High fever that keeps rising, or that lasts for more than 1 day
4. Moderate or high urine ketones or ketones in the urine for more than 12 hours
5. Signs of dehydration, abdominal pains, chest pains, or any other symptom that is new or bothersome

Source: American Diabetes Association. (2015b).

The certified diabetic educator (CDE) is a valuable resource to assess knowledge, provide in-depth information based on individual needs, and coach diabetic patients. There are many outpatient resources available to diabetics, such as classes, community support groups, and even camps for children. Many companies offer low-cost or free glucose meters to those who need them. Medicare, as well as many private insurance providers, cover the cost of diabetes supplies (meter strips, syringes, insulin, etc.). The ADA and the Juvenile Diabetes Research Foundation International (JDRF) offer information, are a resource for current research studies and research findings, and encourage communities to develop support and fundraising groups. Contact information for these organizations and others is found in the Pearson Nursing Student Resources.

Prevention and Detection of Common or Life-Threatening Complications

Complications that can result from DKA or HHNS or their treatment include hypokalemia, hypoglycemia, fluid volume overload, heart failure, cerebral edema, adult respiratory distress syndrome (ARDS), hyperchloremic metabolic acidosis, and thrombosis. Prevention of these complications is an important aspect of care. However, sometimes prevention is not possible. In those cases, early detection and management will improve healthcare outcomes.

Hypokalemia

Hypokalemia is caused by rapid shifts of potassium into the cell as fluid and insulin are administered. Careful monitoring of potassium is important to prevent life-threatening deficits. Continuous ECG monitoring is helpful. Subtle rhythm changes, such as U waves, indicate potassium deficits. Often potassium supplementation is started when levels are normal because intracellular deficits and rapid fluid shifts are anticipated.

Hypoglycemia

Hypoglycemia is defined as a blood glucose level less than 70 mg/dL. During treatment of hyperglycemia, the goal is to decrease serum glucose by 70–100 mg/dL/hr. In order to prevent hypoglycemia the following strategies are used. Bedside blood glucose monitoring is performed every hour during IV insulin administration. Once the serum glucose reaches 250 to 300 mg/dL, 5% dextrose is added to the IV solution in order to prevent hypoglycemia. In addition, the rate of IV insulin is either decreased by half or changed to

SQ administration depending on the presence of metabolic acidosis.

The signs and symptoms of hypoglycemia vary based on the severity of the episode (Box 14-6). In addition, those with hypoglycemic unawareness may have a very severe hypoglycemic episode with few warning signs. People with diabetes lose the ability to release glucagon in response to hypoglycemia. As a result, those with diabetes rely on the release of epinephrine to raise the blood sugar. Early signs of hypoglycemia include shakiness, dizziness, sweating, hunger, and tachycardia. These signs are caused by the release of epinephrine. As hypoglycemia progresses, signs include headache, pale skin color, tingling sensations around the mouth, sudden moodiness or behavioral changes, loss of coordination, and difficulty paying attention. Neurological signs are related to the lack of glucose in the brain. Late signs or signs of severe hypoglycemia include confusion, loss of consciousness, and seizures. When a diabetic has hypoglycemic unawareness, he or she does not have an epinephrine surge, and therefore does not have the typical early warning signs of hypoglycemia. The first warning signs of hypoglycemia in those with hypoglycemic unawareness are often lethargy and confusion, followed quickly by loss of consciousness and seizures. Hypoglycemic unawareness occurs more in the following populations: type 1 diabetics, those who have frequent hypoglycemic episodes, those who are on beta blockers (which block the effects of epinephrine), older diabetics, and those with autonomic neuropathy.

If the patient is alert, hypoglycemia is treated by giving oral carbohydrates. If the patient is unable to safely take oral fluids, glucagon may be given via the SQ or intramuscular (IM) routes. Glucagon is a hormone that promotes rapid conversion of glycogen to glucose. Though this injectable hormone is often prescribed to diabetics at home, it is rarely used in the critical care setting. In the critical care setting, 50% dextrose administered via the IV route is a more common therapy. This concentrated sugar rapidly raises the blood sugar.

Box 14-6 Signs of Hypoglycemia

- Early signs: shakiness, dizziness, sweating, hunger, and tachycardia
- As hypoglycemia progresses: headache, pale skin color, tingling sensations around the mouth, sudden moodiness or behavioral changes, loss of coordination, and difficulty paying attention
- Late signs: confusion, loss of consciousness, and seizures

Fluid Volume Overload and Heart Failure

Fluid volume overload and heart failure are complications that are caused by administering fluids more rapidly than the patient can tolerate. Those with comorbidities such as heart failure or renal insufficiency are at very high risk for fluid volume overload and should be monitored very closely. Hemodynamic monitoring gives the healthcare team additional information that helps to prevent fluid overload in patients with existing heart disease, pulmonary disease, or renal failure. Signs of fluid volume overload include pulmonary congestion evidenced by new or worsening rales or rhonchi on auscultation; neck vein distention evidenced by full neck veins with the head of bed elevated 30 degrees; increasing CVP, PAP, and PCWP; and generalized edema. Although fluid volume overload and heart failure cannot always be prevented, prevention strategies include precise management of intake and output. This includes accurate measurements of intake and output and accurate administration of fluids. Management of fluid volume overload includes maintaining normal oxygenation, diuretics (if indicated), and/or decreasing the rate of fluid administration. Medications to improve heart contractility and CO are also commonly used in the presence of heart failure.

Cerebral Edema and Adult Respiratory Distress Syndrome

Cerebral edema and ARDS are both caused by rapid intracellular fluid shifts during administration of fluids. Those with higher serum osmolality are at highest risk for both of these complications.

Early signs of cerebral edema include headache, lethargy, confusion, and irritability. A CT scan can help to identify cerebral edema. Prevention of rapid fluid shifts by decreasing serum glucose slowly and slow replacement of sodium are indicated to prevent cerebral edema. The serum osmolality should be decreased gradually. Treatment of this complication includes the use of mannitol and may include short-term hyperventilation to decrease intracranial pressure. A neurosurgeon should be consulted. Mortality of those with cerebral edema is high.

Symptoms of ARDS include scattered rhonchi, decreased oxygenation, and decreased lung compliance. Chest x-ray will show scattered infiltrates throughout lung fields. Treatment includes slow rehydration with gradual decrease in serum osmolality. Mechanical ventilation is used to improve oxygenation. Medications to improve lung compliance are also commonly prescribed. Both of these complications contribute to the high mortality rates seen in patients with DKA and HHNS.

Circulatory System Complications

Patients experiencing DKA and HHNS are at higher risk for a variety of acute thromboembolic complications such as myocardial infarction, stroke, deep vein thrombosis, and pulmonary embolism. Dehydration causes increased viscosity of the blood. Therapy aimed at preventing thromboses includes early hydration to decrease the viscosity of the blood. A focused assessment to detect these complications is important.

Cardiovascular disease (myocardial infarction and heart failure) is the leading cause of death in the United States, and diabetics who do not meet recommended glycemic targets are at increased risk for cardiovascular complications. In addition to myocardial infarction and heart failure, cardiovascular complications include stroke, peripheral arterial disease, deep vein thromboses, and decreased arterial perfusion to all organs.

Prevention of circulatory system complications is a key healthcare initiative. Numerous research studies have shown reduced morbidity and mortality as well as decreased cost of providing healthcare in the United States when these complications are prevented. Additionally, the quality of life for a diabetic is improved drastically when these complications are not present. See Table 14-9 for complications, symptoms, and recommended therapies.

The ADA (2015a) recommends aggressive treatment of hypertension and hyperlipidemia in order to prevent cardiovascular complications. The recommended blood pressure should be no greater than 130 mm/Hg systolic, and no greater than 80 mm/Hg diastolic. Control of blood pressure in the diabetic includes medications such as diuretics, beta blockers, and angiotensin-converting enzyme inhibitors (ACE-I) or angiotensin receptor blockers (ARBs). Both ACE-I and ARBs also improve renal circulation. Additional therapies are aimed at lifestyle modifications such as weight loss, decreased sodium in the diet, better dietary choices (such as increased fruits and vegetables), moderation of alcohol intake, and increased physical activity.

Lipid management is focused on decreasing lipids to acceptable limits: low density lipids (LDL) should be less than 100 mg/dL, high density lipids (HDL) should be greater than 50 mg/dL, and triglycerides should be less than 150 mg/dL. Medications such as statin therapy is recommended for all diabetics. Lifestyle modifications mentioned above are also crucial to decreasing lipids and will also help the diabetic to meet glycemic goals (ADA, 2015a).

Antiplatelet medications such as aspirin and/or clopidogrel have been found to be helpful in reducing cardiovascular events in diabetics. Cessation of smoking is also recommended, as this is a major factor in the prevention of myocardial infarction and stroke. Risk factors for cardiovascular disease should be assessed annually. Treatment,

Table 14-9 Circulatory System Complications of Diabetes

Complication	Signs and Symptoms	Recommended Therapy
Coronary artery disease Acute myocardial infarction	Chest pressure or chest pain; pain in the upper body such as the neck, jaw, or upper back; shortness of breath; diaphoresis; nausea	Emergency medical care • Reperfusion of the coronary artery • Rehabilitation • Improve risk factors to prevent reoccurrences
Cerebral artery disease Stroke	Facial droop; slurred speech; sudden numbness or weakness of the face, arm, or leg on one side of the body; dizziness; loss of coordination	• Emergency medical care • Reperfusion of the cerebral artery (if meets criteria) • Rehabilitation • Improve risk factors to prevent reoccurrences
Peripheral vascular disease	Weak peripheral pulses, intermittent claudication in the lower extremities, weakness and numbness of the feet or legs with gradual onset, sores and infections of the feet (may be on or between the toes), decreased growth of hair on extremities	Medications to improve blood flow and decrease risk of thromboses • Surgical reperfusion • Improve risk factors to prevent worsening of the condition
Diabetic foot ulcers	Sores on the feet especially on or between the toes, high risk for infection	Preventative measures: • Routine examination of the feet • Socks and shoes that prevent injury • Foot care and nail care Treatment measures: • Wound cultures and antibiotics if indicated • Rest (relief of pressure on the ulcer) • Evaluation for possible reperfusion procedure • Amputation

education, and coaching are aimed at prevention of disease. Diabetics who have abnormal ECG findings or who have symptoms should be screened for the presence of cardiovascular disease.

Hyperchloremic Metabolic Acidosis

Hyperchloremic metabolic acidosis is mainly a complication of DKA. It occurs as a result of several altered metabolic processes. Bicarbonate cannot be regenerated through the kidneys because of the lack of ketoanions. Bicarbonate that is regenerated moves rapidly to the intracellular space. Additionally, the chloride in IV solution contributes to hyperchloremic metabolic acidosis. This complication causes metabolic acidosis to continue longer than expected. However, with fluid therapy, insulin therapy, and correction of electrolytes, it is usually self-limiting, correcting itself within 24 to 48 hours.

NURSING ACTIONS

Nursing actions include maintaining fluid, electrolyte, and acid-base balance.

Fluid Volume Deficit

Goals: To improve vascular fluid volume to increase perfusion of essential organs and to replace interstitial and cellular fluid losses.

Interventions:

- Assess level of dehydration: physical appearance, neurological assessments, initial blood glucose, vital signs, urine output, and serum osmolality.

- Calculate corrected serum sodium.
- Administer fluids to correct fluid deficits.
- Review laboratory results for signs of organ failure and report abnormalities to the physician.
- Continue assessment of fluid balance: physical appearance, neurological assessments, lung sounds, strict intake and output, vital signs, hemodynamic monitoring, review current laboratory findings, and daily weights.

Altered Electrolyte Balance

Goals: To prevent life-threatening cardiac arrhythmias, improve cellular metabolism, improve smooth and skeletal muscle function, and maintain essential endocrine function.

Interventions:

- Determine initial electrolyte values and perform frequent reevaluation during therapies.
- Determine renal function by laboratory values.
- Perform continuous ECG monitoring.
- Perform frequent neurological assessments.
- Replace electrolytes as necessary.

Altered Acid-Base Balance

Goal: Transport of glucose into the cell, resulting in normal cellular metabolism evidenced by bicarbonate greater than 18 mEq/L, venous pH greater than 7.3, normal anion gap, and no ketones in the urine.

Interventions:

- Assess arterial blood gas results.
- Determine anion gap and recalculate when current laboratory results available.

- Provide insulin therapy.
- Measure urine ketones.

Identify Precipitating Factors

Goal: To identify and treat precipitating cause and prevent further complications.

Interventions:

- Assess for presence of infection.
- Culture any sites of infection to identify type of organisms.
- Assess for presence of deep vein thromboses, signs of acute myocardial infarction, and signs of stroke.
- Assess toxic screen (if completed).
- Assess for presence of pregnancy (women of child-bearing age).

Risk for Injury

Goal: To provide safety measures during critical illness.

Interventions:

- Assess skin integrity and risk for skin breakdown on admission.
- Consult the dietician in regard to providing adequate nutrition during critical illness.
- Provide basic hygiene: Keep the skin clean and dry, provide adequate oral hygiene, and, if immobile, reposition every 2 hours.
- Safety measures: Implement fall precautions; secure lines, cords, and equipment; and prevent the patient from dislodging lines and equipment.

Knowledge Deficit Related to Management of Diabetes and/or Acute Illness

Goals: To improve knowledge of precipitating factors of DKA and HHNS and improve knowledge of management of sick days and when to seek medical advice.

Interventions:

- Assess current knowledge level of the patient and significant others.
- Provide information regarding diabetes management.
- Consult the CDE if available.

Potential Noncompliance with Diabetes Management

Goals: To encourage compliance of diabetes management, ensure that hemoglobin A_{1C} is less than 6.5%, and encourage healthy lifestyle.

Interventions:

- Assess the results of the last hemoglobin A_{1C}.
- Provide positive reinforcement of application of knowledge to the patient and significant others.
- Coach the patient and significant others to help overcome barriers to healthy management of diabetes.

High Risk for Infection Related to the Effects of Hyperglycemia

Goal: To maintain blood glucose within normal ranges throughout the majority of the time during a critical illness.

Interventions:

- Perform bedside blood glucose monitoring.
- Assess concurrent treatments that increase or decrease the serum glucose (Box 14-6).
- Provide insulin therapy and adjust it based on blood glucose monitoring results.

Risk for Injury Related to Immobility

Goal: To provide safety measures during critical illness.

Interventions:

- Assess skin integrity and risk for skin breakdown on admission.
- Implement measures to prevent skin breakdown such as frequent repositioning, providing adequate nutrition during critical illness, and providing basic hygiene measures (keeping the skin clean and dry and providing adequate oral hygiene).

Potential Knowledge Deficit Related to Maintenance of Health

Goals: To improve knowledge of factors that lead to the development of type 2 diabetes and how to delay or prevent the onset of the disease.

Interventions:

- Assess for risk factors related to metabolic syndrome.
- Provide education related to diet and nutrition, exercise, and weight management.
- Consult the dietician.

Insulin Administration

Insulin is commonly given via the IV or SQ routes while in the critical care setting. However, a patient may be admitted to the critical care area using various insulin therapies such as insulin pens, injection aids, a continuous insulin pump, or inhaled insulin. The following is a short discussion of various

insulin therapies that are available to patients. More information on these therapies can be found at manufacturer websites such as those that are listed on the companion website.

Continuous Glucose Monitoring

A continuous glucose monitoring system allows the diabetic patient to record blood sugar levels throughout the day and night. There are several FDA-approved devices currently on the market. These monitors provide the patient with much more data than the patient has with traditional monitoring. Many continuous glucose monitors provide glucose results every 5 minutes, 24 hours a day. These monitors also provide the diabetic with trend information. For instance a diabetic will know if the blood sugar is rising or falling rapidly, and some monitors have an audible alarm to alert the diabetic to high and low blood sugars. The newer sensors are discreet. The diabetic inserts a sensor into the skin that measures blood sugars up to 1 week at a time. The monitor needs to be within about 5 feet of the sensor to receive data, but newer monitors are no longer attached to the sensor via a wire. Instead, the monitor receives information remotely. The diabetic can use continuous monitoring to assess the effects of medications, diet, and exercise. It can help the diabetic to identify trends that would otherwise go unnoticed. Many insurance plans are now covering continuous glucose monitoring.

Insulin Pump

A continuous SQ insulin infusion pump closely imitates the physiologic secretion of insulin by delivering a basal rate of insulin. The basal rate can be programmed to automatically change based on the time of day, and the diabetic can also deliver boluses of insulin for meals. Insulin doses are accurate, the pump is small and discrete, and programming can be changed as needed. It also eliminates the need for frequent insulin injections. The pump enables the diabetic to have a more flexible lifestyle. The insulin pump is worn externally with a catheter that is inserted into the SQ tissue. The catheter is changed every 2 to 3 days. The pump includes an insulin reservoir and a computerized control mechanism. Many newer pumps have programs that can be downloaded to a computer or other mobile devices, and some can even be programmed wirelessly. The insulin pump is powered by a battery and has alarms that alert the user if the catheter is occluded or the insulin reservoir is low. The reservoir is usually filled with 300 units of rapid-acting insulin. The insulin pump would not be the optimal tool for those who are not willing to monitor blood glucose frequently and make frequent dose adjustments. The pump requires a user who is knowledgeable, motivated, and able to do simple calculations. Because the pump uses rapid-acting insulin, if it does not deliver insulin for several hours, diabetic ketoacidosis can result. The pump is also

more expensive than other products. Though the insulin pump may be the best option for some diabetics, it may not be the optimal method of insulin delivery for others. The nurse should encourage the diabetic to keep insulin and syringes on hand in case the insulin pump malfunctions.

Insulin Pens

Many patients use insulin pens. They are easy to use, are inexpensive, allow for consistent measurement of insulin, are portable, and the user does not need as much manual dexterity as is required for the traditional vial and syringe. In addition, insulin pens are available in many of the premixed insulin preparations, preventing the patient from having to mix two types of insulin into one syringe. Insulin pens look like a writing pen. There are two types of pens: nondisposable and disposable. When using the nondisposable pens, a disposable cartridge of insulin is replaced when empty. In contrast, the disposable pens are discarded when the insulin cartridge in the pen is empty. The needle is disposed of and replaced after each injection. To use the pen the patient simply dials in an insulin dose and gives the injection SQ. There are three important considerations that the nurse should know about insulin pens in order to teach or reinforce teaching. First, the patient should detach the needle after each injection and use a new needle for each new injection. In addition to preventing infection, this step prevents leakage of the pen. Second, the patient should dial the dose into the pen slowly to ensure accuracy of the dose. This is especially true when higher doses are used. Third, the patient should leave the needle in place 5 to 6 seconds after injecting the insulin. This is done to ensure that the correct dose is administered from the pen.

Injection Aids

Injection aids help patients give insulin safely and accurately. Some of the aids available include syringe magnifiers, devices for patients who cannot see, needle guides and vial stabilizers, and devices to simplify injections (Austin, Boucher, & Peragallo-Dittko, 2006). Syringe magnifiers attach to the insulin syringe and magnify the syringe so users can accurately draw up a dose or see air in the syringe. A product for those who cannot see called Count-A-Dose makes an audible click as each unit of insulin is drawn into the syringe. Needle guides and vial stabilizers hold the insulin syringe and the vial together. These devices prevent the needle from coming out of the vial while the diabetic fills the syringe and are helpful for those with limited dexterity. Devices used to simplify the process of giving an injection include spring-loaded syringe holders. The filled syringe is loaded into the device. Some models hide the needle from view. The needle inserts into the skin when a button is pushed, or some devices even insert the needle when pressure is put

against the skin. Once the needle is in the skin, some devices inject the insulin automatically, whereas others require the diabetic to push the plunger. Many spring-loaded syringe holders accommodate prefilled insulin pens. These devices are optimal for those who have a fear of injecting insulin as well as those who do not have the dexterity to administer an injection.

New Technologies

Several new methods of insulin delivery are still being researched. Alternative sites of insulin delivery such as

oral, buccal, ocular, and transdermal routes are being studied. New technologies, including implantable insulin pumps, are under investigation. Islet cell transplantation is also being researched. For many years insulin was available only in a vial and was drawn up and administered for each injection using a traditional syringe. Diabetics had to deal with the inconvenience of storage and fear or social stigma related to using a needle and syringe. Currently there are many new technologies to help diabetics administer insulin safely, more discreetly, and with more convenience.

Problems in Glucose Metabolism Summary

Because diabetes is a very common condition with increasing prevalence in the United States, it is important for the nurse to know how to manage common diabetic emergencies such as DKA and HHNS. In addition to managing the critical events associated with DKA and HHNS, glucose control is an important aspect of nursing care. Short-term glucose control is important to prevent the creation of pro-inflammatory cytokines that cause insulin resistance and

stimulate the release of counterregulatory hormones. Long-term glucose control is important to prevent the long-term complications of diabetes such as systemic vascular disease, renal failure, neuropathies, and retinopathy. Once the critically ill patient is stable it is important to focus on helping the patient to maintain glucose control at home by providing knowledge and coaching the patient to make lifestyle changes.

Why/Why Not?

Mr. P, a known type 2 diabetic, is admitted to the intensive care unit with a blood glucose level of 653 mg/dl and is started on an insulin drip at 14 units/hour. He has cellulitis on his left leg and after wound cultures are obtained he is also started on IV antibiotics. Mr. P's serum potassium is 2.9 meq/L

and an infusion of potassium chloride 20 meq in 100 ml is planned. Determine if it would be appropriate or not to administer potassium via the IV route? Why or why not?

See answers to Why/Why Not? in the Answer Section.

Case Study 1

Type 1 Diabetes with Diabetic Ketoacidosis

Mrs. D is a 27-year-old accountant who comes to the emergency department (ED) with complaints of fever and extreme fatigue. She states that she has had the "flu" for 2 days and is also diabetic. The triage nurse notes that Mrs. D appears dehydrated and lethargic and that her breath has a fruity odor. The following rapid history and assessment is obtained in the ED.

Her vital signs are blood pressure 96/70, pulse 104, respiratory rate 28, oral temperature 100.8, and pulse oximeter reading 99%. Mrs. D states that she has some abdominal pain, which is not localized to a specific area,

but has no other pain. Her height is 68 inches and her weight is 155 pounds. She has no allergies.

- Her medications include:
 - Lantus 30 units every morning; patient did not take this medication today.
 - Humalog 5 units with meals, patient took 2 units of Humalog 2 hours ago and 4 hours ago.
 - Prinivil 5 mg every morning; patient did not take this medication today.
 - No vitamins, no herbals.
 - Pulse oximeter reading 99%.

- Past health history:
 - Type 1 diabetes since age 11 (16 years).
 - Denies any other medical problems.
 - Last hemoglobin A_{1C} was 6.2 (2 months ago).
1. Name five interventions that would be important to initiate.
 2. Based on the following laboratory findings, calculate the serum osmolarity.
 - Na⁺ 132, K⁺ 6.2, chloride 100, glucose 644, creatinine 1.4, BUN 31, bicarbonate 17.
 3. What is your assessment of the serum osmolarity? What physiologic changes, if any, will result?
 4. Calculate the anion gap based on the laboratory findings from question #2.
 5. What is your assessment of the anion gap? What do you anticipate the findings of arterial blood gases to show?
 6. Calculate the corrected serum sodium based on the laboratory findings from question #2.
 7. Based on the results of the corrected serum sodium, what intervention would you expect to implement?
 8. Calculate the 24-hour fluid replacement based on severe fluid losses of 100 mL/kg. How would the total fluid replacement be administered?
 9. Answer the following questions regarding insulin administration.
 - a. What type of insulin would you anticipate giving Mrs. D?
 - b. What route would you expect insulin to be administered?
 - c. Calculate an initial bolus dose and an initial hourly dose.
 - d. When would this dose be adjusted?
 - e. What should you assess prior to giving insulin?
 10. Once Mrs. D recovers, what topics would be important to focus on during diabetic teaching?

See answers to Case Studies in the Answer Section.

Case Study 2

Type 2 Diabetes with Hyperglycemic Hyperosmolar Nonketotic Syndrome

Mr. S is a 67-year-old retired professional who comes to the ED with complaints of shortness of breath, extreme fatigue, and headache. He states that he has had a cold for the past 2 weeks. He also has diabetes and heart failure. The triage nurse notes that Mr. S appears weak with fever. He also has a cough with production of tan, yellow sputum. Diagnostic findings include the following: sodium 128, K⁺ 5.7, chloride 95, glucose 1074, creatinine 2.4, BUN 62, and bicarbonate 21. During his stabilization in the ED Mr. S experiences respiratory failure, is intubated, and is placed on mechanical ventilation. Mr. S is diagnosed with HHNS, pneumonia, and heart failure and is admitted to the ICU. Fluid replacement and insulin therapy are started.

11. In addition to HHNS, pneumonia, and heart failure, what other problem is evident based on Mr. S's lab values.

How will this affect the care that you provide? What additional intervention might be anticipated?

12. Mr. S has been prescribed albuterol and dexamethasone to treat his pneumonia. How do these medications specifically help Mr. S's pneumonia? What would you anticipate as a result of these medications?
13. Upon review of the history and physical, you note that the last outpatient hemoglobin A_{1C} was 10.5. What was Mr. S's average blood sugar based on this result? What is the significance of this finding? What action would be appropriate based on this finding?
14. What three complications is Mr. S at high risk to develop? Why? What assessments or diagnostics would point to the development of these complications? would indicate their presence?

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 14.1 What medications can increase insulin resistance and the blood sugar in patients with type 2 diabetes?
- 14.2 Why is treatment of hyperglycemia during critical illnesses important?
- 14.3 What four assessment findings are associated with metabolic syndrome? Why is it important to assess serum glucose on patients with metabolic syndrome?
- 14.4 What are the most common precipitating factors for ketoacidosis (DKA) and hyperosmolar nonketotic syndrome (HHNS)?
- 14.5 What are two differences in the assessment findings between patients with DKA and HHNS?
- 14.6 What intervention(s) is (are) critical prior to the administration of insulin to the patient with DKA?
- 14.7 What are the most common complications that may occur during management of DKA or HHNS?

See answers to Chapter Review Questions in the Answer Section.

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Chapter 15

Care of the Patient with Acute Kidney Injury

Carrie Edgerly MacLeod, PhD, APRN-BC

Abbreviations

AKI	Acute Kidney Injury	ESRD	End Stage Renal Disease
ATN	Acute Tubular Necrosis	FENa	Fractional Excretion of Sodium
CKD	Chronic Kidney Disease	GFR	Glomerularfiltration Rate
CRRT	Continuous Renal Replacement Therapy	IHD	Intermittent Hemodialysis
CVVH	Continuous Venovenous Hemofiltration	PD	Peritoneal Dialysis
CVVHD	Continuous Venovenous Hemodialysis	RRT	Renal Replacement Therapy
CVVHDF	Continuous Venovenous Hemodiafiltration	SCUF	Slow Continuous Ultrafiltration



Learning Outcomes

Upon completion of this chapter, the learner will be able to:

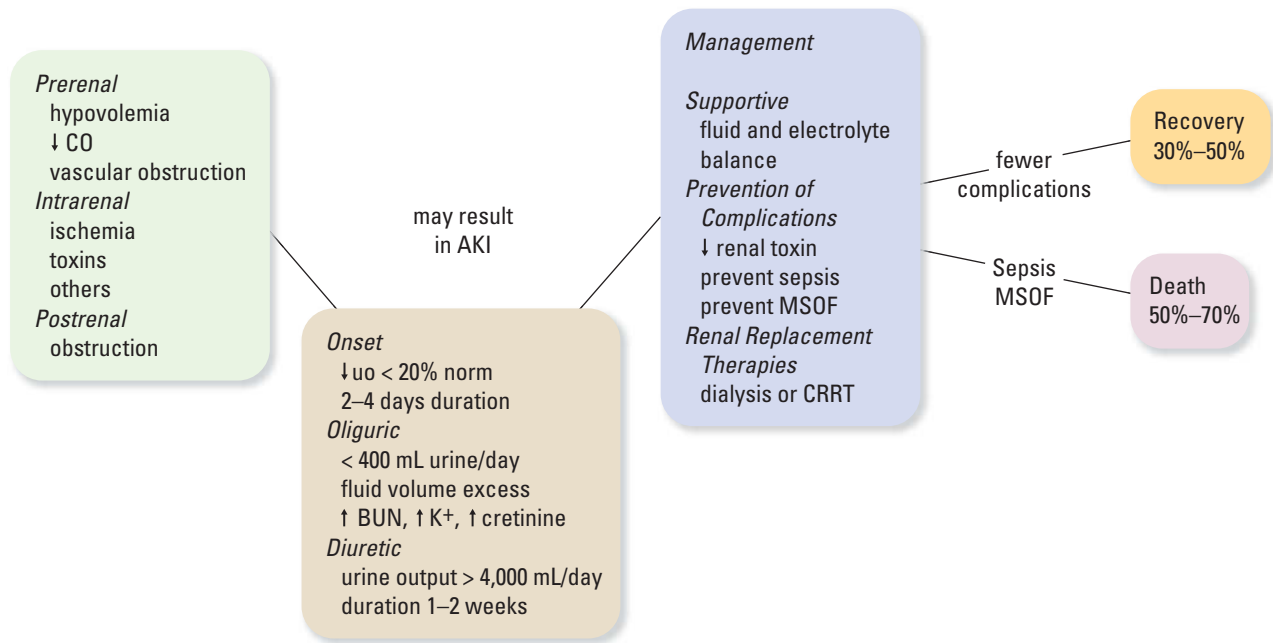
1. Interpret the difference between normal renal anatomy and physiology and the pathology of acute kidney injury.
2. Compare and contrast the etiologies and diagnoses of prerenal, intrinsic and post renal acute kidney injury.
3. Articulate the collaborative care and nursing actions for prerenal acute kidney injury.
4. Articulate the collaborative care and nursing actions for intrinsic acute kidney injury.
5. Critique the priority management of the patient with acute kidney injury.
6. Compare and contrast renal replacement therapies and the associated nursing responsibilities.

Introduction

Acute kidney injury (AKI) is “the abrupt increase (within 48 hours) in serum creatinine, resulting from an injury or insult that causes a functional or structural change in the kidney” (Molitoris et al., 2007). It is a broad clinical syndrome with various etiologies, including specific kidney diseases and nonspecific conditions including extrarenal

pathologies. It is staged based on serum creatinine level and urine output. It is essential that the cause of AKI be identified as quickly as possible for effective treatment.

Approximately 50% of patients admitted to intensive care units will develop AKI (Mandelbaum et al., 2011). In fact, the incidence of AKI is believed to be similar to that of an acute myocardial infarction. Unlike a myocardial infarction however, AKI may lack the signs and symptoms



Visual Map 15-1 Acute Kidney Failure Summary

needed to guide the early detection that is so critical for successful treatment (Kellum, Bellomo, & Ronco, 2012).

Causes of AKI are classified as prerenal (due to decreased renal blood flow), intrinsic or intrarenal (due to disturbances within the glomerulus or renal tubules), or postrenal (due to obstruction of urinary outflow). AKI results in retention of nitrogenous wastes, increases in serum creatinine, and disruptions in serum and urinary electrolytes. It may be oliguric, with the patient having a daily urine output of less than 400 mL per day, or non-oliguric. Management is based on supportive treatment, prevention of complications, and, if needed **renal replacement therapy (RRT)**. Of patients who develop AKI, 50% to 70% die, usually after developing sepsis or multisystem organ failure (MSOF). The majority of patients who survive regain renal function. (Visual Map 15-1)

Renal Anatomy, Physiology and Pathology

The kidneys are complex organs responsible for multiple metabolic functions, including the regulation of:

- Fluid volume status
- Acid-base balance
- Electrolyte concentrations in the body
- Clearance of nitrogenous and other wastes.

These functions are performed by the approximately 2 to 3 million nephrons, the functional unit of filtration and

reabsorption, in the human kidney (♦ Figure 15-1). Nephrons are composed of a glomerulus, Bowman's capsule, and renal tubules. The glomerulus is a system of tiny capillaries with thin walls. Filtration of particles and wastes occurs as blood

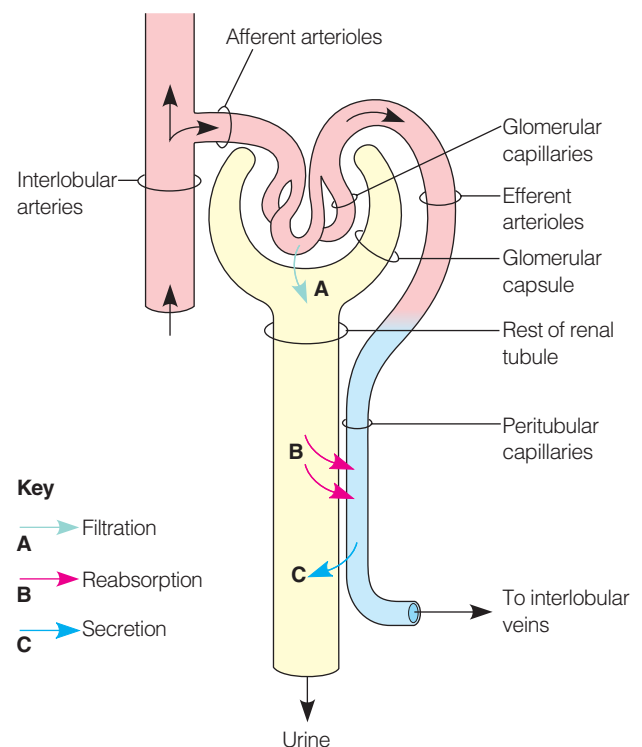


Figure 15-1 Schematic view of the three major mechanisms by which the kidneys adjust to the composition of plasma: A, Glomerular filtration. B, Tubular reabsorption. C, Tubular secretion.

passes through the glomerulus. The renal filtrate then enters Bowman’s capsule, the structure surrounding the glomerulus, and is channeled into the renal tubules. Most of the water and electrolytes contained in the filtrate are reabsorbed into the blood from the renal tubules. To regulate electrolyte concentration in the body, electrolytes may be actively secreted into the filtrate along the tubules. The filtrate, containing nitrogenous and other wastes as well as the secreted electrolytes, is then excreted as urine.

The **glomerulofiltration rate (GFR)** refers to the rate at which the filtrate is formed, approximately 125 mL/min in a healthy adult. The GFR is driven by the pressure gradient between the pressure in the renal capillaries and the pressure within the renal tubules. Factors that decrease pressure within the renal capillaries or increase pressure within the renal tubules may decrease the GFR. The most accurate measurement of GFR is the creatinine clearance rate (normally 85 to 135 mL/min).

However, the regulation of fluid electrolyte and acid-base balance is not the only essential function the kidneys perform. By producing erythropoietin, the kidneys stimulate production of red blood cells. By secreting renin, which is converted to angiotensin I and II, they also have a role in the regulation of blood pressure. In addition, they produce active vitamin D, assisting in the regulation of calcium balance.

Etiologies of Acute Kidney Injury

AKI is an abrupt reduction in kidney function defined as:

- an absolute increase in creatinine of 0.3 mg/dL within 48 hours
- increase in serum creatinine by 1.5 times of baseline in the past 7 days
- documented oliguria of 0.5 mL/kg/hr for more than six hours (Kellum et al., 2012)

The critical care nurse assesses the patient in AKI for the development of fluid and electrolyte imbalances, institutes supportive care, and attempts to prevent additional renal injury.

Risk Factors for Development of Acute Kidney Injury

The causes for AKI can be divided into three categories: prerenal, intrinsic renal and postrenal. Patients who have underlying **chronic kidney disease (CKD)** are particularly vulnerable. In fact, CKD is the most important risk factor for AKI, increasing the odds of acute injury by as much as 10 times those of individuals without CKD. There is also an increase risk of **end stage renal failure (ESRD)** by nearly

40 times, and an increase in the risk for mortality (Chawla, Eggers, Star, & Kimmel, 2014). Table 15-1 lists the wide spectrum of potential etiologies for AKI for all three categories.

Prerenal

Prerenal causes account for 60% of cases of AKI. Although an obstruction in renal blood flow resulting from renal vascular diseases or obstruction of a renal artery may cause prerenal failure, it more commonly results from a pronounced reduction in cardiac output due to severe hypotension, hypovolemia, or severe vasoconstriction. In prerenal dysfunction, the nephrons and glomeruli are structurally and functionally normal, and the GFR decreases due to the reduction in renal blood flow.

The patient with a prerenal cause of AKI often is hypotensive and may demonstrate severe vasoconstriction due to a pronounced decrease in cardiac output. The nephrons and glomeruli are still relatively normal, so the patient responds to the decreased kidney perfusion by retaining

Table 15-1 Cause of Acute Kidney Injury (AKI)

Prerenal	Intrarenal vasoconstriction • Medications: ◦ Nonsteroidal Anti-Inflammatories ◦ ACE inhibitors ◦ Angiotensin receptor blockers ◦ Cyclosporine/tacrolimus • Cardiorenal/Hepatorenal syndrome • Abdominal Compartment Syndrome • Hypercalcemia Systemic Vasodilation • Sepsis • neurogenic shock Volume Depletion • Renal Loss: Diuretics, osmotic diuresis • Extrarenal Loss: vomiting, diarrhea, burns, blood loss
Intrinsic Renal	Glomerular: post infection, glomerulonephritis Interstitial • Medications: Penicillin, cephalosporins, sulfonamides, ciprofloxacin, acyclovir, rifampin, Dilantin, proton pump inhibitors, nonsteroidal anti-inflammatories • Infection: direct renal or systemic • Viruses: Epstein-Barr, cytomegalovirus, HIV • Bacteria: Streptococcus, Legionella • Fungi: candidiasis, histoplasmosis • Systemic disease: sarcoidosis, lupus Tubular • Ischemia: prolonged hypotension • Nephrotoxic: toxins, radiographic contrast agents, Vascular • Renal vein thrombosis, malignant hypertension, renal infarction • Rhabdomyolysis
Postrenal	Extrarenal obstruction: prostate hypertrophy, neurogenic bladder, bladder, prostate or cervical cancer Infrarenal obstruction: stones, crystals, clots, tumor

Source: Kellum, J.A. & Lameire, N. (2013). Diagnosis, evaluation, and management of acute kidney injury: a KDIGO summary (Part 1). *Critical Care*, 17(204), 01–15.

Gerontological Considerations

Normal aging results in a significant reduction in renal reserves—as much as a 30% reduction between the ages of 30 and 75. Older, infirm patients, due to their reduced muscle mass, may show only modest increases in serum creatinine, even with severe AKI. The elderly are at high risk for AKI and demonstrate an increased vulnerability to poor health outcomes. Some factors that contribute to this high risk include the aging kidney, nephrotoxic

medications, radiocontrast material used for diagnostic procedures, polypharmacy and the high risk of sepsis in the debilitated elderly individual (Haim & Paixao, 2015).

Older patients are less likely to recover renal function following AKI and more likely to develop chronic kidney disease and to need long-term dialysis. They are also more likely to die following AKI than younger patients.

sodium and by producing less than 400 mL/day of concentrated urine. Urinalysis typically shows concentrated urine with a high osmolality (greater than 500 mOsm/L) and decreased urine sodium (less than 20 mmol/L). The **fractional excretion of sodium (FENa)**, a test for assessing how well the kidneys can concentrate urine and conserve sodium, is usually less than 1%. There are rarely more than a few casts and/or a little sediment present in the urine.

Intrinsic causes account for 30% to 40% of the cases of AKI. In critically ill patients, the most common etiology is prolonged hypoperfusion of the kidney. So, prerenal failure is a strong precursor of intrinsic AKI. If prerenal failure is not treated adequately and promptly, oxygen delivery to the kidneys does not meet the demand. The renal tubules respond to hypoxia with dysfunction, inflammation, and, possibly, necrosis. With the release of inflammatory mediators and death of cells there is further disruption in renal blood flow and damage to the basement membranes and renal tubules. In 30% to 70% of cases, renal tubular epithelial cells are shed in the urine. There is tubular dysfunction with impaired sodium and water reabsorption. Thus, intrinsic renal failure can be differentiated from prerenal dysfunction by urinalysis (Table 15-2). In intrinsic renal failure, urinalysis typically reveals an osmolality less than 350 mOsm/L, an increased urine sodium (greater than 40 mmol/L), and a FENa greater than 1% with granular casts and sediment. Although these tests are useful in differentiating between prerenal and intrarenal causes of AKI, they are not early indicators or markers of renal injury (Martin, 2010).

Postrenal

Postrenal causes, accounting for 5% to 10% of cases of AKI in hospitalized patients, are usually obstructions in the urine collection system. The most common reason is the development of benign prostatic hypertrophy in the older male. Other causes include tubular obstruction from crystals (caused by uric acid or acyclovir), bilateral ureteral obstruction, or other sources of urethral obstruction such as prostatic cancer. Renal failure results when the obstruction causes an increase in the tubular pressure, resulting in a decrease in the GFR. Most commonly it is characterized by the sudden onset of anuria (production of less than 50 mL of urine/day). It may be identified quickly and accurately by renal ultrasound. Postrenal failure usually resolves rapidly with removal of the urinary tract obstruction, and the return of renal filtration pressure.

Prerenal Acute Kidney Injury

Prerenal dysfunction most commonly results from a significant decrease in cardiac output or an obstruction in a renal artery resulting in a decrease in renal perfusion pressure. It is identified when the patient demonstrates signs of decreased cardiac output and oliguria with characteristic changes in urinalysis. Management involves improving the cardiac output, usually by volume administration, or relieving the renal artery obstruction. With prompt return of renal perfusion, most patients regain renal function. If inadequate renal perfusion is prolonged, patients may develop intrinsic renal failure.

Table 15-2 Contrasting Laboratory Findings in Prerenal and Intrinsic Renal Failure

Laboratory Finding	Prerenal	Intrinsic Renal
Urine osmolality	>500 mOsm/L	<350 mOsm/L
Urine sodium	<20 mmol/L	>40 mmol/L
FENa	<1%	>1%
Casts and/or sediment	Rare	Present

NURSING ACTIONS

The acutely ill patient may develop a decrease in cardiac output, leading to prerenal dysfunction in a variety of ways. The nurse reviews the patient's history for potential sources of decreased cardiac output. Refer to Table 15-1 for common causes of prerenal AKI.

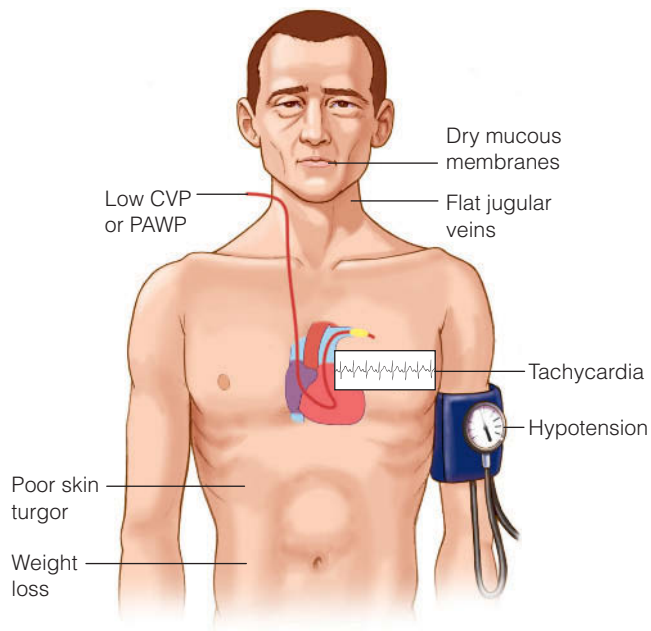


Figure 15-2 Manifestations of hypovolemia.

The nurse assesses the patient for manifestations of hypovolemia (♦ Figure 15-2), including:

- Hypotension or orthostatic hypotension
- Tachycardia
- Dry mucous membranes
- Poor skin turgor
- Flat jugular veins
- Weight loss
- Low central venous pressure (CVP) or pulmonary artery wedge pressure (PAWP)

The nurse looks for indications of increased extracellular fluid that may develop when the decreased renal perfusion is due to vasodilation or cardiovascular disease. These include:

- Edema
- Ascites
- Weight gain
- Increased CVP or PAWP

Additionally, the nurse reviews the following diagnostic studies.

1. **Urinalysis:** As described earlier, reviewing the urinalysis helps to differentiate between prerenal and intrinsic renal causes of AKI. To most accurately reflect the patient's situation, urine for evaluation should be obtained prior to the administration of a diuretic.
2. **Serum Blood Urea Nitrogen (BUN):** A normal level is 5 to 25 mg/dL. Urea is filtered freely, but its absorption from the tubules is a function of the urine flow rate. When the urine flow rate is reduced, more urea is absorbed. Thus, in prerenal failure, the rise in BUN may be out of proportion to the renal dysfunction.

3. **Serum Creatinine:** Serum creatinine provides a more accurate estimate of renal function than BUN. Normal levels in adults are 0.5 to 1.5 mg/dL. A doubling of serum creatinine normally indicates a 50% reduction in GFR. Be aware of the patient's baseline creatinine level and if a rise has occurred in the past 48 hours.
4. **Creatinine Clearance:** Creatinine clearance provides the most accurate estimate of GFR. Normal values range between 85 and 125 mL/min/1.73 m² for adult men, and 75 and 115 mL/min/1.73 m² for adult women. Values below normal indicate at least a 50% reduction in the number of functioning nephrons.
5. **BUN-to-Creatinine Ratio:** Because prerenal failure results in increased reabsorption of urea, the serum BUN-to-creatinine ratio may rise to greater than 20:1 in prerenal causes of AKI.
6. **Renal Ultrasound:** This test is performed to rule out obstruction, i.e., post renal causes, hydronephrosis or renal perfusion issues.
7. **Urine electrolytes:** Review FENa to assess if the etiology is prerenal or intrinsic. (Refer to Table 15-2.)
8. **Biomarkers:** Different urinary and serum proteins, molecules and microRNA enzymes such as Interleukin 18, Neutrophil Gelatinin-Associated Lipocalin, and Kidney-Injury Molecule-1 have potential for earlier detection of AKI. Currently, these biomarkers are used together with other parameters to optimize diagnostic accuracy and earlier injury-focused interventions to limit renal damage and assist with determining the response of AKI to treatment (Ronco & Ricci, 2013).

COLLABORATIVE CARE

The nurse works collaboratively with the physician to identify and then resolve prerenal dysfunction. The goal of therapy is restoration of normal renal perfusion by attaining normovolemia, increasing cardiac output, or relieving the obstruction in the renal artery.

Restoration of Normovolemia

For restoration of normovolemia, protocols suggest an isotonic intravenous fluid, normal saline, challenges until the patient's mean arterial pressure (MAP), central venous pressure (CVP) and mean perfusion pressure (MPP) reach optimal levels (Refer to Chapter 5, Cardiodynamics, for further details.). However, the fluid of choice may vary depending on the reason for the patient's fluid volume deficit.

- To deliver a fluid challenge, the nurse infuses 250 to 500 mL of normal saline rapidly. Although definitions of rapid administration vary depending on the circumstances, a fluid challenge of 500 mL may be administered over half an hour.

- The nurse anticipates that the patient's blood pressure (BP), heart rate, urine output, and CVP will begin to normalize quickly if the challenge has been successful. The urine output usually begins to increase within 10 to 20 minutes following the volume challenge and is maximal at 30 to 45 minutes if hypovolemia was the cause of the decreased urine output.
- The first fluid challenge may be followed by additional fluid challenges if it does not have the anticipated effect.
- When such a protocol is initiated, it is imperative that the nurse observe the patient closely and identify immediately any signs of fluid volume overload that could develop if an excessive amount of fluid was inadvertently administered. Fluid overload is particularly problematic for the patient with AKI since it may lead to increases in abdominal pressure resulting in abdominal compartment syndrome (Dirkes, 2011). As abdominal pressure increases, renal perfusion drops, and renal injury that is not responsive to attempts to decrease abdominal pressure can develop. The manifestations of fluid overload are described in the section titled "Assessment of Fluid Volume Status in the Patient with Acute Kidney Injury" and displayed in ♦ Figure 15-3.

Restoration of Adequate Renal Perfusion

One of the major determinants of renal perfusion is the mean arterial pressure (MAP). In a middle-aged or younger adult, maintaining the MAP above 70 to 80 mmHg usually allows for adequate renal perfusion. However, adults over the age of 70 may require a higher MAP (perhaps as high

as 100 mmHg) to maintain adequate renal perfusion (Wong et al., 2015). The most effective way to attain a MAP of 70 or greater is with adequate administration of volume. So the nurse should be attentive to the increase in the MAP when administering fluid challenges.

Essential for Evidence-Based Practice

The nurse should not rely on systolic and diastolic pressure to identify adequacy of tissue perfusion, rather the nurse should consistently assess the patient's mean arterial pressure and take measures to maintain it greater than 70.

In the past, dopamine was used to increase BP with the assumption that a dopamine infusion of 0.5 to 3 mcg/kg/min would increase renal perfusion. Current literature demonstrates the use of renal Dopamine to be associated with worse outcomes in patients with AKI and is no longer recommended. Thus, norepinephrine, rather than dopamine, is now indicated to increase the patient's MAP when adequate fluid administration does not result in a sufficient increase in MAP (Rahman et al., 2012).

Nephrology Consult

When patients do not respond to volume repletion and appropriate management of decreased cardiac output with improved urinary output and renal function, a consult to a nephrologist should be suggested. Studies have shown that early referral of patients with AKI to a nephrologist results in better patient outcomes. Patients with AKI who were seen by a nephrologist within 18 hours of AKI diagnosis had reduced risk of further renal function decline (Balasubramanian, Zayed, Abdul, Rauchman, & Zhang, 2012).

Relief of Renal Artery Obstruction

When the patient's decrease in renal function is accompanied by pronounced hypertension, the physician may suspect that the renal failure is caused by an obstruction in renal blood flow rather than hypovolemia or a decreased cardiac output. She may order renal Doppler flow studies or a renal angiogram to rule out such an obstruction. If an obstruction is identified, it may be treated invasively by angioplasty with stent placement or surgically. Usually prompt relief of the vascular obstruction results in return of renal function.

Avoidance of Additional Renal Injury

During the period of decreased renal perfusion and lowered GFR that occurs in prerenal AKI, the kidneys are vulnerable to insults from other sources such as medications, radiocontrast dyes, and toxins. These insults may precipitate the development of intrinsic AKI. Therefore,

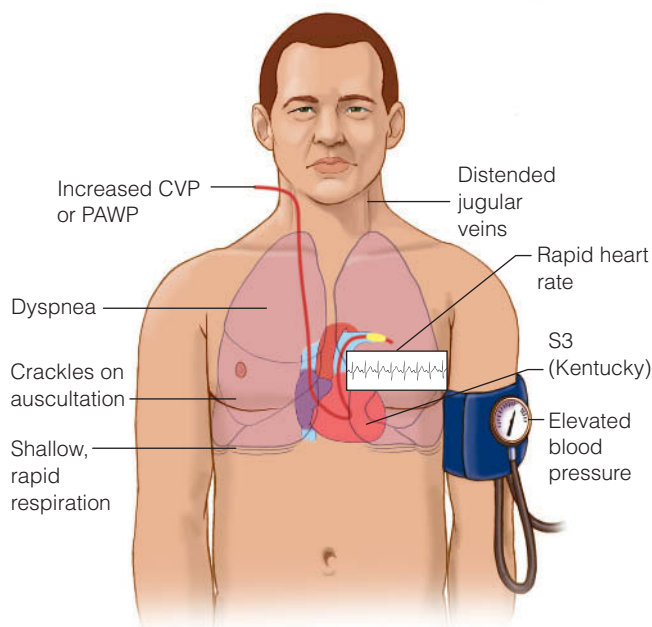


Figure 15-3 Manifestations of excess fluid volume.

Commonly Used Medications

Norepinephrine

Introduction

Norepinephrine is a potent α -adrenergic agonist with some β -adrenergic effects.

Dose and Desired Effect: Norepinephrine is administered by continuous intravenous (IV) infusion. Usual maintenance range of the infusion is 2 to 12 mcg/min. The usual maximum dose is 25 mcg/min. Due to its vasoconstrictive effects, norepinephrine usually results in a significant increase in MAP with little change in heart rate or cardiac output. Initial response should begin 1 to 2 minutes after IV administration is started.

Nursing Responsibilities:

- The nurse monitors the BP every 2 to 5 minutes after beginning a continuous infusion. Norepinephrine may be titrated up or down cautiously to obtain a MAP of 80. The lowest dose possible to attain the MAP should be utilized.
- In addition to monitoring the MAP, the nurse will keep an eye on the heart rate, monitor pattern, CVP, and urine output continuously during administration.

- Norepinephrine should be administered through a central venous catheter. If norepinephrine is administered through a peripheral IV line, severe vasoconstriction may result. Veins in the hands, ankles, and legs should be avoided but a large antecubital vein may be acceptable if necessary. Blanching along the vein is an indication of extravasation that can lead to severe tissue necrosis, sloughing, and gangrene. If extravasation does occur, phentolamine (Regitine) 5 to 10 mg diluted in 10 to 15 mL of normal saline is injected liberally throughout the tissue to prevent necrosis.

Potential Side and/or Toxic Effects: Side effects are rare when used as directed. However, previously there was concern that the vasoconstriction that occurs with norepinephrine would result in deterioration in renal functioning. This does not appear to be the case. Instead, by increasing the MAP, norepinephrine appears to increase glomerular filtration and renal function. However, the nurse should be certain to monitor the urine output and serum creatinine of the patient receiving norepinephrine.

- The nurse avoids administering nephrotoxic agents to the patient if possible.
- Drugs that are known to be excreted by the kidneys and potentially harmful to the kidneys are given in reduced doses based on the patient's creatinine clearance.
- The nurse monitors the peak and trough blood levels of medications known to be damaging to the kidney such as vancomycin and gentamycin. A trough level is usually drawn 1 hour prior to administration of the next dose of a medication, whereas a peak level is usually drawn immediately following the administration of a drug.
- If contrast dye must be administered, it is given sparingly or the physician may prescribe and the nurse may administer acetylcysteine for its renal protective effects.
- The nurse is scrupulous in his use of aseptic technique to prevent the development of an infection because removal of the by-products of infection and excretion of antibiotics may impose additional burdens on the damaged kidneys.

conditions, including nephrotoxic agents. Intrinsic AKI may be differentiated from prerenal failure by the previously described characteristic findings on urinalysis. Although there are normally four phases to the disease process—onset, oliguric, diuretic, and recovery—not all patients in AKI are oliguric.

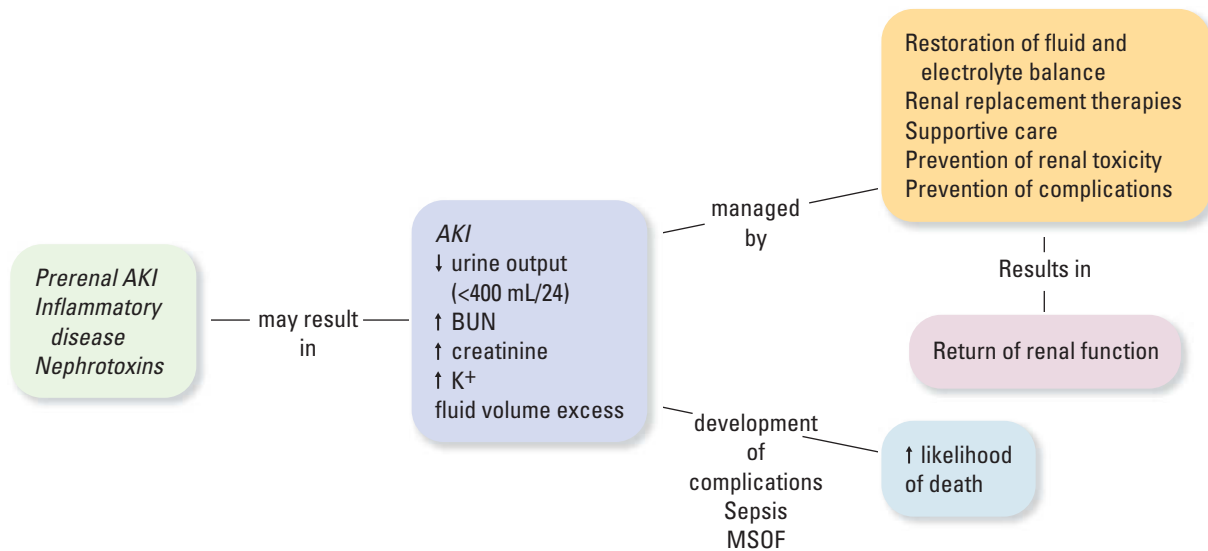
Predisposing Conditions

As noted earlier, the most common reason for development of intrinsic renal failure is prerenal failure. Other causes such as inflammatory disorders, infiltrative diseases, and nephrotoxins account for the remaining cases of intrinsic renal failure. Because inflammatory disorders and infiltrative diseases are less likely to be the cause of AKI in critically ill patients, they are not discussed further.

Individuals at higher than normal risk for AKI include those with pre-existing decreased renal function, such as diabetics, older adults, or patients who have decreased renal perfusion. Conditions that create endogenous toxins capable of causing renal failure are rhabdomyolysis and hypercalcemia rhabdomyolysis (accounting for as much as 25% of AKI). The conditions cause the breakdown of muscle fibers, resulting in the release of muscle fiber contents such as myoglobin into the circulation. Myoglobin, an oxygen-binding protein pigment found in the skeletal muscle, is normally filtered out of the bloodstream by the kidneys. When released in large amounts, myoglobin may block the structures of the

Intrinsic Acute Kidney Injury

In critically ill patients, intrinsic or intrarenal failure, sometimes called **acute tubular necrosis (ATN)**, is primarily caused by a prolonged reduction in renal perfusion (prerenal failure), but may also be precipitated by other



Visual Map 15-2 Intrinsic Renal Failure

kidney, causing AKI. The disorder may be caused by any condition that results in damage to skeletal muscle. Risk factors include trauma, severe exertion, ischemia or necrosis of muscles, crush injuries, burns, or seizures. The sequence of events during the development of rhabdomyolysis is hypovolemia, hyperkalemia, metabolic acidosis, and eventually AKI. In addition to the normal findings of AKI, the patient with rhabdomyolysis may manifest dark-red or cola-colored urine; muscle tenderness; and/or weakness of the affected muscle(s) or flu-like symptoms (Naqvi, Akhtar, Ahmed, Naqvi, & Rizvi, 2015). (Visual Map 15-2.)

Pathophysiology and Manifestations

Intrinsic renal failure usually has four distinct phases, the first of which is the **onset phase of acute kidney injury**. This phase immediately follows the period of renal injury and lasts approximately 2 to 4 days. During this phase, the patient's urinary output typically decreases to 20% of normal and the BUN and creatinine may begin to increase slightly.

Most patients then enter the second phase of AKI, the **oliguric phase of acute kidney injury**, during which the patient excretes less than 400 mL of urine/day. During this phase that typically lasts 10 to 14 days, the patient retains fluid, is unable to maintain electrolyte and/or acid-base balance, and cannot excrete sufficient amounts of nitrogenous wastes.

This renal dysfunction is probably the result of cyclical damage to the renal tubules. Decreased renal perfusion begins the process, resulting in decreased glomerular capillary pressure and a reduction in the GFR. There is concurrent ischemic injury to epithelial cells. The renal tubules respond to hypoxia with dysfunction, inflammation, and patchy tubular necrosis that does not correlate with the

level of renal dysfunction. With the release of inflammatory mediators and death of cells there is further disruption in renal blood flow and damage to the basement membrane. Persistent vasoconstriction and congestion from white cells and cell debris lead to ongoing hypoxia and necrosis (Chawla et al., 2014). In 30% to 70% of cases, renal tubular epithelial cells are shed in the urine; in the past this type of AKI was called acute tubular necrosis (ATN).

Some patients in AKI do not develop oliguria; this is termed **nonoliguric AKI**. They still have problems with electrolyte imbalances and uremia but their urine outputs are more than 400 mL/day, usually about 1000 mL/day. Studies have shown that patients with non-oliguric AKI have a higher mortality rate than patients with oliguric renal failure. The outcomes may be worse for these patients because they are producing what is measured as adequate amounts of urine so their referral to a nephrologist, management of electrolyte imbalances, and subsequent start of RRT are delayed.

As patients begin to regain renal function, they enter the **diuretic phase of AKI**. During this phase, patients usually begin to increase their urinary output, often producing up to 5 liters of urine each day. However, these patients still may not be able to completely excrete wastes and regulate electrolyte or acid-base balance. The BUN and creatinine usually decrease slowly toward normal over a 10-day period.

The final phase of AKI is the **recovery phase of acute kidney injury**. This phase typically lasts from 6 months to a year. During this phase, most patients' renal function returns to normal. Only 5% to 10% of patients who survive continue to require dialysis. However, patients are thought to be vulnerable to repeated insults to their renal function and are counseled to avoid renal toxins.

Assessment of Fluid Volume Status in the Patient with Acute Kidney Injury

The patient in the oliguric phase of ATN will usually have a fluid volume excess. To identify this fluid excess, the nurse assesses for the following:

- Rapid pulse of bounding quality
- Skin that is pale and cool to the touch
- Increased BP, possible decreased pulse pressure. One of the earliest indications of fluid excess, occurring prior to the development of edema, may be a gradual increase in BP.
- Jugular venous distention and/or hepatjugular reflex
- Nonpitting edema in dependent areas
- Increased CVP or PAWP
- Increased respiratory rate with shallow respirations
- Dyspnea on exertion or in supine position
- Crackles in bases on auscultation

Weight is a more accurate indicator of fluid volume status than many of the other assessment parameters. The nurse determines the patient's "dry" weight (the weight after the patient has been diuresed or had dialysis). Then, the patient should be weighed daily at the same time on the same properly calibrated scale with the same amount of clothing or bed linens. A patient will gain 0.5 kg (1 lb) for each 500 mL of extra fluid intake.

Although intake and output are carefully measured in most critically ill patients, they have been shown to be inaccurate. Still, the nurse attempts to maintain as accurate a record as possible. In the patient in AKI, the nurse usually measures sources of intake and output every 1 to 2 hours with a summation of total intake and output every 24 hours. Allowing for insensible loss, a nonventilated patient may be considered to be in balance if she has an intake of 500 to 700 mL of fluid more than she excretes in 24 hours. However, a ventilated patient does not have the same insensible losses, so intake and output should be roughly equivalent.

Essential for Evidence-Based Practice

The nurse examines laboratory values to identify hemodilution from fluid volume excess. It may be evident as a decrease in hemoglobin, hematocrit, and serum sodium values.

Prioritized Management of Acute Kidney Injury

Management is primarily supportive with an emphasis on achieving normal fluid and electrolyte balance by conservative methods or renal replacement therapies, avoiding

nephrotoxic agents, and preventing complications. Survival has not improved since the introduction of dialysis—50% to 80% of ICU patients with AKI die, usually following the development of sepsis and/or multisystem organ failure (MSOF).

Evidence-Based Interventions for Fluid Volume Excess

The most effective interventions for fluid volume excess in the AKI patient are fluid restriction and renal replacement therapies.

Diuretics

Once a mainstay of treatment, diuretics should be used carefully and not on a routine basis. There is no evidence that diuretic therapy improves morbidity, mortality or renal outcomes (Rahman et al., 2012).

Fluid restriction

Fluid restriction may be instituted in an attempt to prevent worsening fluid volume excess. The physician might prescribe a specific amount of fluid (such as 1000 mL) per 24 hours, or might calculate the intake for each day depending on the patient's previous day urine output plus the estimated insensible loss. For example:

Previous day's urine output (330) + insensible fluid loss estimate (400) = 770 mL. The fluid allotment may be divided over the course of the day. One common example is:

1/2 of calculated fluid may be allotted to days
1/3 for evening
Balance (1/6) at night

For the 770-mL fluid limit calculated, this would mean that the patient would receive 385 mL of fluid on days, 255 mL on evenings, and the balance (130 mL) at night.

It can be extremely difficult to maintain a conscious patient on a restricted fluid intake, especially if the patient is receiving IV fluids and/or medications.

Renal Replacement Therapy

Severe fluid volume excesses should be dealt with by some method of renal replacement therapy. RRT will be discussed later in the chapter under "Building Technology Skills."

Essential for Quality Assurance

The nurse reviews the patient's medication profile for evidence of use of loop diuretics. Despite the lack of evidence supporting the use of loop diuretics in AKI, they continue to be prescribed resulting in potential harm to patients (Murphy and Byrne, 2010).

Evidenced-Based Interventions For Electrolyte Imbalances

The critical care nurse must be prepared to assess her patient in AKI for development of the following electrolyte imbalances. The nurse recognizes that these serious imbalances may require immediate management on discovery.

Dilutional (Hypervolemic) Hyponatremia

The critical care nurse assesses the AKI patient for an abnormally low serum sodium due to fluid overload. Manifestations depend on the serum sodium level and how rapidly the level dropped. The manifestations of hyponatremia are most apparent when serum sodium levels fall from normal levels (135 to 148 mEq/L) to below 120 to 125 mEq/L in less than 12 to 24 hours. However, nearly all patients with serum sodium levels less than 120 mEq/L will demonstrate some symptoms, and patients with serum sodium levels less than 110 mEq/L will demonstrate severe symptoms. The nurse assesses the patient with the following serum sodium levels for these potential symptoms:

125 to 135 mEq/L	Generally, no manifestations
111 to 124 mEq/L	Headache, lethargy, disorientation
100 to 110 mEq/L	Confusion, hostility, nausea and vomiting, lethargy or violence
Less than 100 mEq/L	Delirium, convulsions, coma, Cheyne-Stokes respirations

Fluid restriction is the most effective method of treatment in asymptomatic patients. It allows for the gradual loss of free water with a subsequent rise in the serum sodium. If the patient is awake, the nurse will need to carefully explain the rationale for the fluid restriction, assist with the allocation of fluid intake as described previously, and carefully monitor the patient's intake.

If fluid restriction is not successful in increasing the serum sodium or if the patient is symptomatic, diuretics may be administered with a saline infusion. This must be done very carefully because the use of diuretics results not only in the loss of fluid but may result in the loss of sodium as well.

For a severely ill patient with a serum sodium less than 110 mEq/L, a loop diuretic may be cautiously administered in conjunction with hypertonic saline (3% or 5%). When administering these agents, the nurse checks urine output along with urine and blood electrolytes hourly.

Hyperkalemia (Serum Potassium Greater Than 5.5 mEq/L)

Hyperkalemia may represent a life-threatening complication. Although some patients complain of nausea or muscle

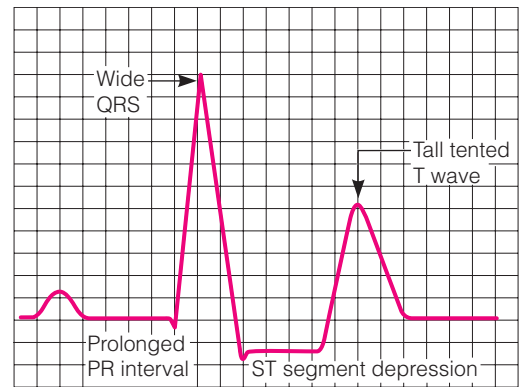


Figure 15-4 ECG in hyperkalemia.

weakness, usually hyperkalemia is identified through a laboratory test or an ECG. The changes that occur in the ECG are described next and shown in ♦ Figure 15-4.

$K^+ = 5.6$ to 6.0 mEq/L	tall, peaked Ts
6.0 to 6.5 mEq/L	prolonged PR and QT intervals
6.5 to 7.0 mEq/L	diminished Ps and depressed STs
7.0 to 7.5 mEq/L	bundle branch blocks may develop
7.5 to 8.0 mEq/L	Ps disappear, QRS widens

Death may occur when the potassium exceeds 7.0 mEq/L and is the result either of diastolic arrest caused by block of the distal Purkinje fibers or ventricular fibrillation caused by reentrant circuits.

If the serum potassium is less than 6.5 mEq/L and the patient is asymptomatic, the potassium level may be reduced by limiting potassium intake and increasing the potassium loss in the urine through the use of a diuretic. Once the level rises to above 6.5 mEq/L, the following efforts may be made to counteract or rapidly decrease the serum potassium:

- A. IV calcium may be administered to patients who are not receiving digitalis to counteract the potassium.

Dose and Desired Effect: The dose is dependent on whether calcium chloride or calcium gluconate is administered. Ten milliliters of 10% Ca^{++} gluconate (4.5 mEq) or 5 mL of Ca^{++} chloride (6.8 mEq) might be administered IV push over 2 minutes. The counteracting effect of IV calcium is short-lived; buying time for other types of interventions, the onset of action should be 1 to 3 minutes and the duration of effect 30 to 60 minutes. The nurse may recognize that calcium is having the desired effect by normalization of the ECG on the monitor.

Nursing Responsibilities: The nurse maintains the patient on a cardiac monitor and observes for bradycardia, stopping the administration of the calcium should bradycardia develop.

B. In an attempt to drive the excess potassium into the cell, three other medications might be utilized. None of the medications lowers the total body concentration of potassium, but all temporarily shift the potassium intracellularly, allowing time to institute other interventions that will decrease the total body concentration of potassium. These medications are:

1. Insulin plus glucose (5 to 10 units of regular insulin with 50 g of glucose); the onset of action is about 30 minutes and the duration 4 to 6 hours
2. Sodium bicarbonate (50 to 100 mEq total dose—or 1 mEq/kg); the onset of action is about 5 to 10 minutes with 1- to 2-hour duration of effect.
3. Inhaled beta agonists such as albuterol (2.5 mg/3 mL saline) may be given by nebulizer every 20 minutes. The dose is problematic because it is so much higher than what is normally used for treatment of reactive airway disease, and may result in central nervous system (tremors) and cardiovascular (tachycardia) symptoms. The onset of action is about 30 minutes and the duration 2 to 3 hours.

When administering medications that either counteract potassium or drive it into the cell, the nurse recognizes that the effect is temporary and definitive management is still required. Still, she should observe for the desired effects, an improvement in the ECG waveform, and the cardiac output. If insulin, glucose, and sodium bicarbonate, or inhaled beta-agonists have been utilized, there should also be a rapid reduction in serum potassium.

C. A cation exchange resin such as sodium polystyrene sulfonate (Kayexalate) might be utilized to decrease total body potassium through gastrointestinal transport.

Dose and Desired Effect: The usual dose is 15 to 50 g of the resin administered with sorbitol to hasten delivery. It may be administered either orally or rectally by retention enema. Onset is usually within 1 to 2 hours, but may be delayed as long as 12 hours with oral administration. Rectal administration results in more rapid reduction in serum potassium because most of the transporters are located in the colon.

Nursing Responsibilities: Because gastric ulceration may result from its administration, it may be administered either with ginger ale, if given orally, or in 500 to 700 mL of D₅W or saline, if given rectally.

The nurse observes for the reduction in serum potassium and monitors the patient until the potassium level has stabilized.

D. Finally, dialysis may be utilized to return the serum potassium to a normal level. This is the most effective method of adequately treating severe hyperkalemia. Care of the patient receiving dialysis is discussed in the “Building Technology Skills” section.

Hypocalcemia (Ionized Ca⁺⁺ Less Than 4.25 mg/dL, Which Is 1.2 mmol/L or Serum Ca⁺⁺ Less Than 8.5 mg/dL)

Calcium is found in the blood bound to albumin or free in its ionized form. Because only ionized calcium is metabolically active, it is the preferred value for identifying and treating hypocalcemia.

Manifestations of hypocalcemia may develop as soon as 48 hours after the onset of the oliguric phase of AKI because renal tubule disorders often result in excessive loss of calcium.

- With mild hypocalcemia, the patient may complain of some numbness and tingling in the digits or about the mouth.
- As the calcium level continues to drop, contraction of the corner of the patient’s eye and mouth may occur when the patient’s facial nerve is tapped gently. This represents a positive Chvostek’s sign and may indicate hypocalcemia (♦ Figure 15-5). However, the sign is not definitive for hypocalcemia because it occurs normally



Figure 15-5 Positive Chvostek’s sign.

Bariatric Considerations

Obesity is associated with increased AKI short and long-term mortality. For every 5.0 kg/m increase in BMI, there is an associated 10% increase for AKI (Danziger et al., 2015). The risk of acute kidney failure among overweight patients is nearly 90% greater than that for normal weight individuals. The most obese patients, with a BMI of 40 or above, have over seven times the risk of kidney failure (Druml, Metnitz, Schaden, Bauer, & Metnitz, 2009). Excess body weight may be associated with functional/

structural lesions of the kidney such as glomerulosclerosis, diabetic nephropathy, cancer of the kidney, nephrolithiasis, and abdominal compartment syndrome.

Research indicates the presence of an “obesity paradox” in patients with CKD: a higher body mass index (BMI) is associated with better survival. This survival advantage of large body size is relatively consistent for hemodialysis patients across racial and regional differences (Park et al., 2014).

in response to tapping of the facial nerve in 10% to 12% of the population, and it occurs in response to conditions other than hypocalcemia.

- Spasm of the hand and wrist, Trousseau’s sign, may become apparent when a BP cuff placed on an arm is inflated to 20 mmHg above the systolic pressure for at least 3 minutes.
- Serious physiological effects occur when total calcium levels are less than 7.0 and ionized calcium levels are less than 0.8 mmol/L; then the patient may develop tetany or convulsions.

Management of mild hypocalcemia is supportive and may involve oral supplementation, whereas serious hypocalcemia is managed as described next with IV calcium replacement.

Hyperphosphatemia (Serum Level Greater than 4.5 mg/dL)

In AKI, hyperphosphatemia, an increase in the serum phosphorus, does not usually occur until the GFR has decreased to 10% to 20% of normal. Because serum phosphorus and calcium are usually in an inverse relationship, hyperphosphatemia is usually accompanied by hypocalcemia. Patients experience few symptoms unless problems with hypocalcemia or soft tissue calcifications develop. Treatment includes ensuring adequate hydration, dietary restriction of phosphate (less than 200 mg/day), calcium supplementation, and administration of phosphate binders. Dietary restriction of less than 200 mg of phosphate is difficult for a patient to maintain, so elimination of phosphate is required.

Commonly Used Medications

Calcium for Supplementation

Introduction

Calcium, a therapeutic mineral and electrolyte replacement/supplement, is available as two separate forms of elemental calcium for IV use. It is imperative that the nurse recognize the differences in amounts of elemental calcium found in calcium gluconate and calcium chloride.

- One gram of Ca^{++} gluconate (10% injection) contains 90 mg of elemental calcium or 4.5 mEq.
- One gram of CaCl contains 272 mg of elemental calcium or 13.6 mEq.

Dose and Desired Effects: For tetany, a total dose of 4.5 to 16 mEq of calcium might be required for treatment. Often the initial dose is 1 to 2 amps of 10% solution of calcium gluconate (4.5 to 9 mEq) in 50 to 100 mL of D_5W administered IV over

5 to 10 minutes. The nurse should expect to see an improvement in serum calcium values drawn 4 to 6 hours later.

Nursing Responsibilities:

- It is preferable to administer the medication through a central line, although, if necessary, a large peripheral vein is acceptable. If a peripheral vein is utilized the nurse must assess the IV site closely for signs of extravasation and tissue damage.
- The nurse monitors the patient’s ECG during the infusion to detect the signs of hypercalcemia, a decreased QT interval, and an inverted T.

Potential Side and/or Toxic Effects: If the infusion is given too rapidly, pronounced hypotension, bradycardia, and cardiac arrest can occur.

Phosphate binders are the primary means for decreasing the serum phosphorus. Historically, aluminum-based antacids were used as phosphate binders. However, when patients began to develop symptoms that were attributable to aluminum toxicity, their use decreased dramatically. Calcium-containing phosphate binders such as calcium carbonate and calcium citrate might be used. However, studies have indicated that sevelamer (Renagel) or lanthanum carbonate (Fosrenol) may limit atherosclerosis in the chronic renal failure patient, and therefore should be the preferred phosphate binder.

Maintenance of Acid-Base Balance

AKI may lead to metabolic acidosis due to the inability of the renal tubules to secrete hydrogen ions into the renal tubules or to conserve bicarbonate. It may be identified by a pH less than 7.35, a HCO_3^- less than 23, with an initially normal pCO_2 . If the patient is able to compensate by increasing the rate and depth of respirations resulting in a decrease in the pCO_2 , the pH may return toward normal. This is the preferred method of compensation because there are fewer problems. As long as the patient is able to compensate, there is no need for further intervention. However, the nurse assesses the patient for the development of manifestations of metabolic acidosis, including:

- CNS effects such as confusion, hyperactive reflexes, tingling, and, possibly, tetany or seizures
- Effects associated with hyperkalemia such as dysrhythmias

The nurse continues to assess the patient's pH and bicarbonate levels as well. If any of the listed symptoms develop and/or the pH falls below 7.2 with the bicarbonate below 15, sodium bicarbonate might be given. Bicarbonate is given only if necessary because it may result in:

- Hyponatremia
- Hyperosmolality
- A decreased release of oxygen from hemoglobin to the tissues

The base deficit might be calculated by a formula such as the following to guide the total amount of bicarbonate administration:

$$\text{Deficit} = (\text{Desired bicarbonate-measured}) \times \text{weight (kg)} \times 0.5$$

Essential for Evidence-Based Practice

The most effective management strategy for intractable or severe acidosis in the patient with AKI is dialysis.

Removal of Nitrogenous Wastes

Serum creatinine provides an estimate of GFR. A doubling of serum creatinine normally indicates a 50% reduction in GFRs. Normal levels in adults are 0.5 to 1.5 mg/dL. When there is an abrupt cessation in GFR, the serum creatinine will usually rise between 1 and 2 mg/dL each day.

The rise in the BUN is more variable because it is not only dependent on the loss of renal function, but can also be affected by a change in the patient's protein intake, tissue catabolism, or gastrointestinal (GI) bleeding. A normal level is 5 to 25 mg/dL. Usually in intrinsic renal failure, the BUN and creatinine increase relatively proportionally and maintain approximately a 10:1 ratio.

Azotemia occurs when there is an accumulation of nitrogenous waste products in the blood, and is one of the clinical characteristics of uremia. Uremia can occur once

Commonly Used Medications

Calcium Carbonate and Calcium Citrate Phosphate Binders

Introduction

Calcium carbonate and calcium citrate serve a dual purpose: binding the excess phosphate and providing needed calcium simultaneously.

Dose and Desired Effect: Dosages of the binders are adjusted to bring the serum phosphate level to approximately 4.0 mg/dL.

Nursing Responsibilities: Phosphate binders are provided with meals, if the patient is eating, or down a nasogastric

tube otherwise to bind phosphate in the gastrointestinal tract where it will be excreted in the feces rather than absorbed into the blood.

Potential Side and/or Toxic Effects: When binders containing calcium are utilized, soft tissue calcification occasionally develops. Therefore, the nurse assesses the patient for the following indications of soft tissue calcification:

- A papular rash that may be itchy
- Painful deposits in the joints that may limit movement
- Red eyes from calcium deposits in the eyes

the creatinine clearance is below 10 to 20 mL/min. It is heralded by the clinical onset of the following symptoms:

- Mental status changes, visual disturbances
- Anorexia, nausea, and vomiting
- Pruritus
- Pericardial friction rub due to uremic pericarditis
- Muscle cramps
- Increased thirst
- Hypertension, fluid overload
- Metabolic acidosis, electrolyte imbalances

Dialysis should be initiated when signs or symptoms of uremia are present and are not treatable by other medical therapies.

Nursing Diagnoses

Patient with Acute Kidney Injury

- Ineffective tissue perfusion: renal related to hypovolemia and reduced arterial blood flow
 - Excess fluid volume related to renal failure
 - Imbalanced nutrition less than body requirements related to altered metabolic requirements
 - Risk for infection related to malnutrition, intubation, and indwelling catheters, drains, or IVs.
-

NURSING CARE

Nursing care of the patient with AKI includes ensuring that the patient receives adequate nutrition and promoting patient comfort.

Providing Nutrition

The goal of nutritional support in AKI is to ensure the delivery of adequate amounts of nutrients, to prevent protein-energy wasting and metabolic complications, to promote wound healing, to support immune system function, and to reduce mortality. Protein-energy wasting is common in AKI patients and can have a negative impact on recovery. Patients in AKI have increased energy expenditures and their energy requirements range between 25 and 35 kcal/kg/day. These patients should receive a minimum of 1.5 kcal/kg/day of protein and no more than 30 kcal nonprotein calories. It is also recommended that the patient receive trace elements and water soluble vitamin supplements. In addition to protein issues, patients are especially prone to complications of nutritional support, such as hyperglycemia, hypertriglyceridemia, fluid retention, and electrolyte and acid-base derangements, and need nutritional parameters monitored closely (Fiaccadori, Maggioro, Cabassi, Morabito, Castellano, & Regolisti, 2013).

According to the American Society of Parenteral and Enteral Nutrition, A.S.P.E.N., clinical guidelines, patients in AKI should have a formal nutrition assessment that includes an evaluation of inflammation prior to the development of a nutrition plan. Whenever possible, indirect calorimetry should be used to determine patients' caloric needs (Brown & Compher, 2010). Considerations that affect the provision of nutrition are the patient's protein requirements, the presence of inflammation, and whether and what type of RRT the patient is undergoing. Protein requirements can be calculated roughly based on the rise in the patient's BUN in 24 hours, and the amount of protein in the patient's diet can be based on this calculation. The A.S.P.E.N. guidelines call for protein intake to be adjusted according to catabolic rate, renal function, and dialysis losses. The remainder of the calories the patient requires is supplied as carbohydrates or lipids. As the degree of renal failure and the rate of tissue catabolism increases, the provision of appropriate nutrition becomes more complex. A renal dietician is needed to clarify patient needs and to distinguish the most appropriate therapy from among the products created for patients with special nutritional needs.

For patients in AKI, oral or enteral nutrition is preferred to parenteral (Brown & Compher, 2010). Enteral nutrition has the benefit of helping to maintain the GI mucosal barrier. There are usually fewer problems with volume overload and electrolyte imbalance for patients with AKI when enteral feedings are employed. Parenteral nutrition has been associated with increased morbidity and mortality in patients with AKI (Brown & Compher, 2010), and therefore is usually instituted only after a trial of enteral nutrition has failed. Potential problems of parenteral nutrition include infection, worsening of fluid volume and electrolyte problems, or an increase in tissue catabolism.

The nurse assesses the patient for indications that he is being adequately nourished. These include:

- Maintenance of body weight (with no evidence of excessive fluid intake or output)
 - Albumin level 3.5 to 4.0 g/dL
 - Total protein level 6 to 8 g/dL
 - Normalizing serum electrolytes
-

Reflect On

Why might the patient with AKI be more prone to complications or even death if they received parenteral rather than enteral nutrition?

Promoting Comfort

Patients who are in renal failure usually have multiple sources of discomfort, one of the most significant of which may be thirst. Other sources include discomfort from turning, itching, pain, and anxiety.

Thirst

Patients who are on restricted fluid intakes often complain of thirst. The free water they are allowed may barely be enough to administer their medications whether orally, by feeding tube, or parenterally. Thus, the nurse will need to allocate the fluid carefully, allowing for small amounts of oral fluids at regular intervals to increase patient comfort. Consistent, thorough mouth care also helps to maintain patient comfort. Although there have been no published studies detailing its use in patients with AKI, artificial saliva, especially if it contains mucin, may be effective in relieving a dry mouth that leads to complaints of thirst. However, the nurse may need to experiment with different varieties of artificial saliva to determine which one provides the most relief and is most palatable for the specific patient.

Pruritus

Pruritus may develop because some urea is excreted through the skin. As renal failure progresses, the skin may take on a yellowish tinge or actually show a “uremic frost.” Severe itchiness may be relieved with the onset of dialysis when the BUN decreases. Other methods that may be helpful include:

- Keeping the skin moist and cool by bathing the patient, then applying a thin layer of moisturizer over the damp skin and covering the patient with a superficial covering.
- Bathing the patient with tepid water only. Although hot water will initially be relaxing and may reduce any itching briefly, the itchiness will eventually worsen due to the vasodilation from the heat.
- Applying Stiefel Sarna anti-itching lotion with camphor and menthol. Some nurses report that patients get immediate relief from this over-the-counter reasonably priced lotion.
- Administering an H1 receptor antagonist such as clemastine fumarate (Tavist). The usual dose is 2.68 mg bid.

Pain

Pain is not likely to occur from the AKI itself but may result from the patient’s initial insult (e.g., surgery or trauma) or any invasive lines (e.g., dialysis access). The nurse identifies the source of the pain. Appropriate pain management will depend on the source of the pain.

Enhancing Communication

Anxiety is likely to be a concern for any critically ill patient and his or her family. Usually, the patient with

AKI has already had a series of problems, so the development of this additional one may be especially anxiety provoking for the patient and/or family. The nurse should explain how and why AKI probably developed and ensure that the patient understands the chosen management strategy. When the patient is anxious, this usually means repeated explanations of the same material. It is important that all members of the healthcare team repeat similar information. It can be very anxiety provoking to the patient and family when they hear contradictory information from healthcare providers. The amount of information provided should be determined by the patient’s readiness to learn. Explaining the equipment is helpful to some patients/families but is more information than others can tolerate.

Prevention of Complications

Prevention of complications is essential to ensuring good patient outcomes for the patient in AKI. Patients who are septic, whether they developed the AKI in response to the sepsis or the sepsis in response to the AKI, are more likely to die than patients without sepsis (Schroeder et al., 2004). In addition, sepsis is a major contributor to the development of MSOF. Patients with AKI who develop respiratory failure have an eightfold increase in the likelihood of dying, whereas patients with liver failure have a two to three times increase in mortality. Each additional failing organ results in an increase in mortality of 30%.

Nurses need to be vigilant in preventing, identifying, and treating infections in the patient with AKI. Sepsis is discussed in greater detail elsewhere in this book; however, a few of the evidence-based nursing interventions are listed next. The nurse:

- Ensures that she or he and other members of the healthcare team wash their hands prior to patient care.
- Uses strict aseptic technique when caring for invasive devices.
- Limits the use of invasive catheters as much as possible—for example, removes a Foley catheter and utilizes intermittent catheterization if the patient is anuric.
- Utilizes “bundling” to prevent ventilator-associated pneumonia.
- Monitors the patient’s temperature, complete blood count (CBC), and differential to detect indications of infection.
- If an infection is suspected, collects specimens from appropriate sites (usually urine, sputum, blood, and wound) to be sent to the lab for cultures and sensitivities prior to the administration of any antibiotic.

Building Technology Skills: Renal Replacement Therapies

Choosing the most appropriate method of RRT is a complex decision that is best made after careful consideration of a number of important factors. Approximately half of the patients with AKI cannot be managed with conservative treatment alone. In order to prevent or treat potentially life-threatening complications, these patients will require some form of RRT. Development of any of the following signs or symptoms, despite ongoing medical therapy, usually means RRT is indicated:

- Oliguria with fluid volume overload
- Hyperkalemia (serum K greater than 6.5)
- Hyponatremia (serum sodium less than 115)
- Severe acidemia (pH less than 7.1)
- Azotemia (BUN greater than 90 to 100)
- Symptoms of uremia such as mental changes, neuropathy, or pericarditis

Principles of Therapy

The principles underlying RRTs are diffusion, osmosis, and ultrafiltration. They are depicted in the following figures and defined as follows.

Diffusion

Solutes move across a semipermeable membrane from a solution where they are in higher concentration (the plasma) to a solution where they are in a lower concentration (the dialysate). Small molecules (such as electrolytes) are able to diffuse easily across the semipermeable membrane, whereas larger molecules (such as bacteria) are not. The amount of solute removal is proportional to the dialysate flow rate and the duration of dialysis (♦ Figure 15-6).

Osmosis

A solution (water) moves from an area of low solute concentration (the plasma) to an area of higher solute concentration (the dialysate). (♦ Figure 15-7.)

Ultrafiltration (Convection)

A pressure gradient is created between the sides of the semipermeable membrane. Solute is carried in solution across the semipermeable membrane in response to the pressure gradient, producing an ultrafiltrate. The rate of ultrafiltration depends on the porosity of the membrane and the hydrostatic pressure of the blood (which is dependent

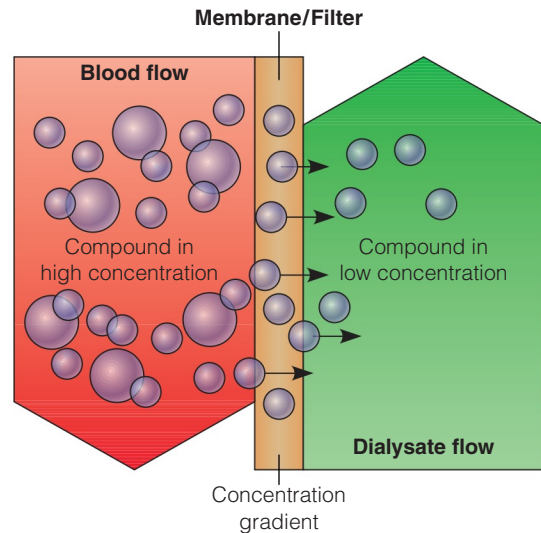


Figure 15-6 Diffusion.

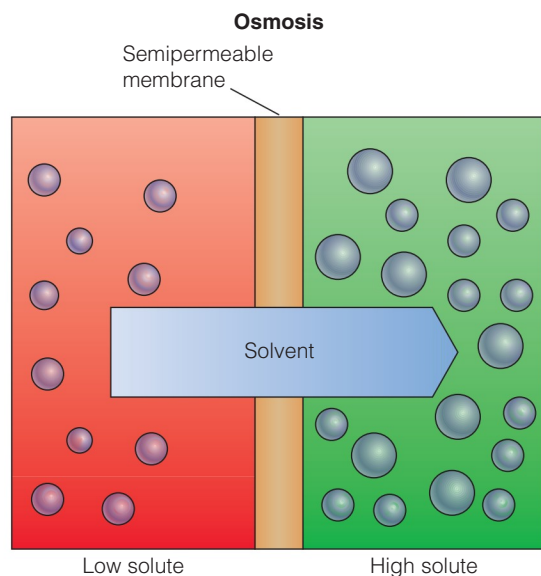


Figure 15-7 Osmosis.

on the blood flow). Astle (2005) recommends thinking of convection as a waterfall. A slow gentle waterfall will not pull much material along with it, whereas a rushing waterfall will drag more and larger particles (solutes) with it. Thus, ultrafiltration is able to remove fluid and the mid-sized solute molecules that are associated with uremia (♦ Figure 15-8). There are three forms of renal replacement therapy (RRT): intermittent hemodialysis, continuous renal replacement therapy (CRRT), and peritoneal dialysis.

Intermittent Hemodialysis

Intermittent hemodialysis (IHD) utilizes both ultrafiltration and diffusion to rapidly correct electrolyte imbalances, restore fluid balance, adjust acid-base balance, and remove wastes.

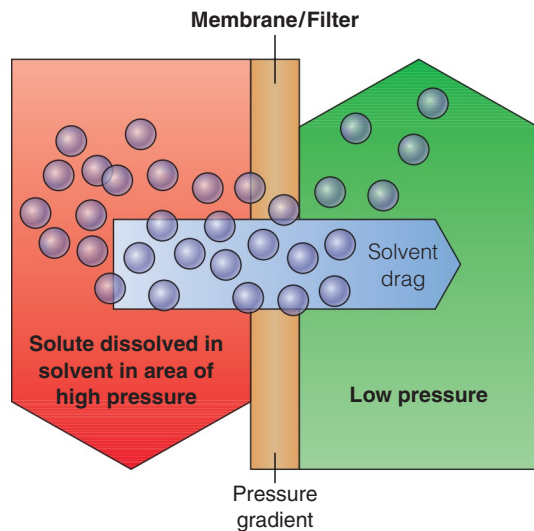


Figure 15-8 Ultrafiltration (convection).

Indications and Expected Outcomes

IHD is indicated in patients with:

- Severe fluid overload and electrolyte imbalances
- Acute or chronic renal failure
- Certain types of drug overdoses or poisonings
- Transfusion reactions

In the past, IHD for AKI patients was usually provided in 3- to 4-hour sessions that occurred two or three times a week at the ICU patient's bedside. The disadvantage to this approach is that fluid overload, waste accumulation, and electrolyte imbalances develop between each dialysis session. Multiple studies have suggested that daily hemodialysis results in a higher rate of return of renal function and decreased mortality for critically ill patients in AKI. Rauf and others (2008) have shown that intermittent hemodialysis is equally as effective, but less costly, than continuous forms of renal replacement therapy. Thus, in many circumstances it is the preferred treatment.

Technological Requirements: Dialysis Machinery

After the blood leaves the patient's body, it travels through a blood pump and an arterial pressure monitor to a dialyzer (filter). In the dialyzer, the blood is separated from the dialysate (deionized water) by a synthetic semipermeable membrane. The dialysate runs countercurrent to the blood flowing on the other side of the semipermeable membrane, allowing diffusion and ultrafiltration to occur. Then the blood travels through venous pressure and air leak detectors before being returned to the patient (♦ Figure 15-9). In hemodialysis, the blood flow rate is maintained between 200 and 400 mL/min, and the dialysate rate at 500 mL/hr. This high rate of flow is the reason why hemodialysis is able to correct

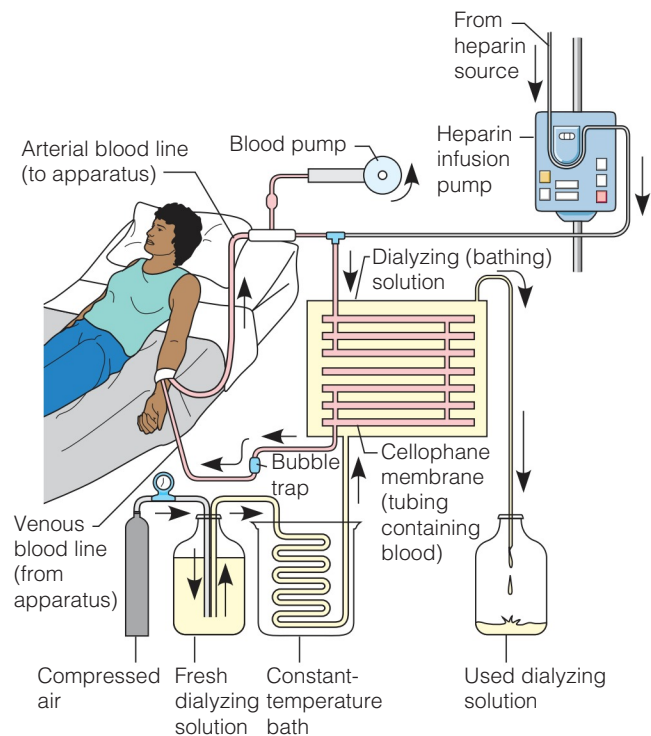


Figure 15-9 Components of a hemodialysis system.

imbalances so rapidly (♦ Figure 15-10). It is also the reason some critically ill patients have difficulty tolerating IHD.

Hemodialysis Requires Vascular Access. In AKI, hemodialysis access is usually through a double lumen venous catheter that may be located in the internal jugular (preferred), subclavian, or femoral veins. The catheter is normally inserted under full aseptic precautions at the bedside and is available for dialysis as soon as placement has been verified. The catheter is usually sutured to the skin so it will not be inadvertently dislodged and may remain in place and be utilized for dialysis for 3 to 4 weeks. There are more permanent versions of dual lumen venous catheters that may be utilized for months. Between dialysis treatments, the catheter is usually filled with a sodium citrate solution to prevent clotting and dressed aseptically with an occlusive, transparent dressing. Maintenance of the catheter and site are normally the responsibility of the dialysis nurse because these catheters are used only in extraordinary circumstances for any purpose other than hemodialysis (Smith, 2010).

In end-stage renal disease, there are more permanent forms of access for hemodialysis. The most commonly used types are arteriovenous (AV) fistulas and AV grafts. These are surgically created connections between an artery and a vein. The AV fistula is a direct connection, whereas the AV graft uses a graft to connect the artery and vein. These anastomoses create vessels that are capable of tolerating high flow rates. Although AV fistulas are the preferred access because they are usually more durable than grafts, a disadvantage is that it may take weeks to months

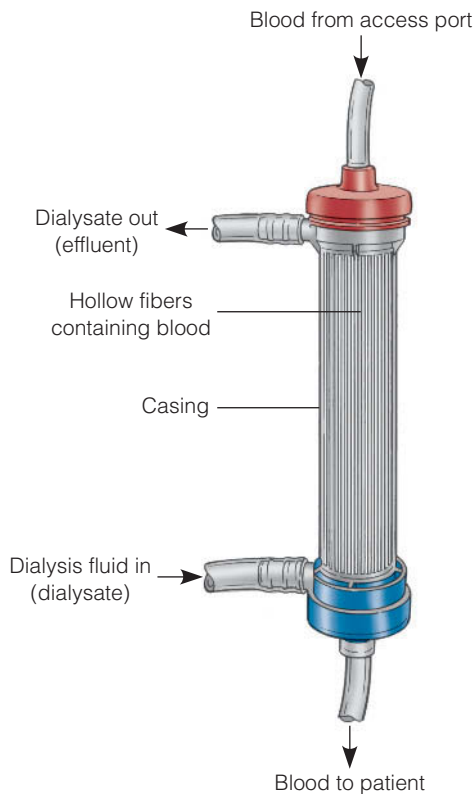


Figure 15-10 Hollow fiber dialyzer.

for the fistula to mature and be able to tolerate the flow rate needed for dialysis. See “Safety Initiatives—Fistula First: National Vascular Access Improvement Initiative.” Potential problems with these forms of access include clotting of the access, bleeding, and infection (♦ Figure 15-11).

When caring for a patient with an AV fistula or graft, the nurse assesses and maintains the patency of the access by:

- Palpating for the thrill or auscultating the bruit over the access

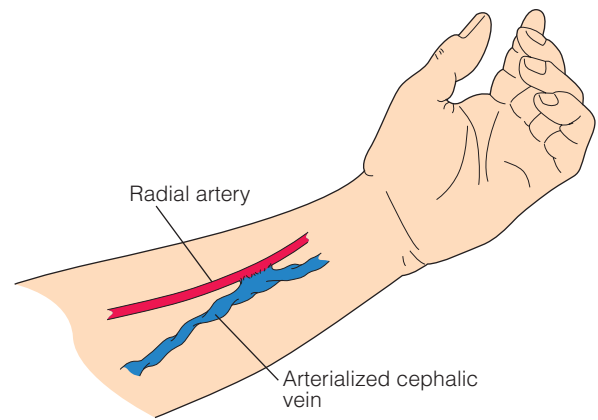


Figure 15-11 An arteriovenous fistula.

- Checking the CSM in the access extremity
- Avoiding any obstruction of blood flow in that extremity such as:
 - BP measurement
 - IV placement
 - Phlebotomy
 - Positioning the patient so there is pressure on the access
- Observing and preventing bleeding from the access by ensuring that gentle pressure is placed on the access site at the completion of a hemodialysis session, and reviewing laboratory bleeding and clotting studies
- Preventing infection by ensuring that aseptic technique is employed when the AV fistula or graft is in use. Usually, the nurse limits personnel in the area and wears a mask as well as sterile gloves when accessing the fistula or graft or completing the dialysis procedure.

Safety Initiatives

Fistula First: National Vascular Access Improvement Initiatives

PURPOSE: To increase the likelihood that every eligible patient will receive the optimal form of vascular access for that patient, usually an AV fistula, and that vascular access complications will be avoided through appropriate access, monitoring, and interventions.

RATIONALE: Central venous catheter accesses should be removed as soon as possible. Complications, including infection, catheter failure, thrombosis, inadequate dialysis, and central venous stenosis or occlusion, occur frequently with catheters—and the complication rate rises sharply with duration of use. Studies have also reported that catheter use is associated with an increased risk of mortality.

HIGHLIGHTS OF RECOMMENDATIONS:

- Routine CQI review of vascular access
- Timely referral to a nephrologist
- Early referral to a surgeon for “AVF-only” evaluation and timely placement
- Monitoring and maintenance to ensure adequate access functioning
- Education for caregivers and patients

Source: Fistula First: National Vascular Access Improvement Initiatives.

Nursing Responsibilities

Prior to dialysis, the nurse discusses the process with the patient and/or the patient's healthcare proxy. The nurse clarifies that the patient or proxy understands the benefits, risks, and likely outcomes of undertaking dialysis. By doing so, the nurse ensures that informed consent has been obtained and the patient's wishes are being respected.

Next, the nurse determines if there is a "dry weight" for the patient on record and determines the patient's current weight. The difference identifies the likely amount of fluid overload the patient is experiencing. A goal for the approximate amount of fluid to be removed by dialysis will be made by the nephrologist. Other measurements of fluid balance the nurse should assess before dialysis include BP, skin turgor, intake and output, breath sounds, and CVP or PAWP if available. The nurse also reviews the patient's laboratory results and identifies the goals the nephrologist has established for correction of electrolyte and acid-base abnormalities.

The nurse will hold certain medications prior to hemodialysis. The doses of medications that may cause hypotension such as beta blockers, angiotensin-converting enzyme (ACE) inhibitors, and calcium channel blockers that are due to be administered 2 to 4 hours prior to dialysis are usually held until dialysis has been completed. Medications that are removed from the body by dialysis (a current list is usually available from the dialysis center) are also held until the end of dialysis. Finally, prior to dialysis, the nurse may check the patient's temperature and should assess the patient's access.

Care of the critically ill patient during the dialysis session requires specialized knowledge and experience. Initiation and maintenance of the dialysis session is usually the responsibility of a nurse from a dialysis unit. All hemodialysis is a controlled hypotensive state but BP management is usually even a more significant concern in the patient with AKI. Thus, the patient's BP may be measured as frequently as every 2 to 5 minutes at the start of dialysis but may be taken every 15 to 30 minutes after the patient has stabilized. The entire dialysis session will usually take between 2 and 4 hours.

At the completion of the dialysis session, the nurse determines the patient's weight, ascertains that appropriate laboratory tests have been sent, monitors the patient's BP, assesses the patient's access site, and administers any medications that may have been held prior to or during dialysis. Later the nurse reviews the laboratory results, paying particular attention to clotting studies and electrolyte balance.

Potential Complications

Of patients in AKI who undergo IHD, 20% to 30% experience rapid shifts in plasma volume that can result in

hypotension. An additional 10% to 20% of patients experience sudden changes in electrolyte concentration that may cause dysrhythmias. The dialysis nurse usually manages these problems. The nurse might manage hypotension by administering normal saline boluses, providing volume expanders such as albumin or mannitol, and/or decreasing the rate of ultrafiltration on the dialyzer. Decreasing the rate of fluid removal may help to manage dysrhythmias. These problems also can be limited and even avoided by short periods of daily dialysis for the patient in AKI. Other frequent problems with the utilization of IHD for the patient in AKI include difficulties with vascular access, problems with anticoagulation, and dialysis membrane incompatibility.

Dialysis disequilibrium syndrome may also occur. It is especially common in patients undergoing their first or second dialysis treatment who experience sudden, large decreases in their BUN. The most likely explanation for this syndrome is that the levels of urea do not drop as rapidly in the brain as the plasma because of the blood-brain barrier. The higher levels of urea in the brain result in an osmotic concentration gradient between the brain cells and the plasma. Fluid enters the brain cells by osmosis until the concentration levels equal that of the extracellular fluid, resulting in cerebral edema and the dialysis disequilibrium syndrome. Manifestations of the syndrome include:

- Headache and mental impairment that may progress to confusion, agitation, and seizures
- Nausea that may lead to vomiting

The most effective management strategy is prevention with daily hemodialysis for 1 to 2 hours.

Continuous Renal Replacement Therapy

Continuous renal replacement therapy (CRRT) is an umbrella term for several methods of dialysis that allow for continuous fluid removal from the plasma (known as ultrafiltrate). The modalities differ in the principles, accesses, technology requirements, and nursing interventions.

Indications and Expected Outcomes

CRRT was originally proposed for use with patients who were hemodynamically unstable and could not tolerate the rapid fluid shifts that occur with traditional hemodialysis. With CRRT patients can be dialyzed over a 24-hour period, resulting in a controlled removal of fluid from the plasma. Because the methods differ in whether they utilize convection and/or diffusion, they differ in their abilities to remove different types of solutes and amounts of urea. Numerous articles describe the theoretical advantages of CRRT for

the patient in AKI. Yet randomized controlled studies have not demonstrated that CRRT is more likely to result in patients surviving or recovering renal function than IHD (Dirkes, 2011).

Technological Requirements

Although all types of CRRT remove fluid and some electrolytes and wastes through a filter, they vary in the process(es) they use to remove fluids and electrolytes, the types of access they require, the setup and technology utilized, and whether they require dialysate or replacement fluids. All forms are usually initiated and maintained by the ICU nurse rather than the dialysis nurse. Anticoagulation is required to prevent clotting of the circuits. This therapy must be carefully balanced to prevent clotting but avoid systemic bleeding. Heparin is the most commonly used anticoagulant. It is infused into the “arterial limb” of the circuit. Effective PTT levels of 1.5 to 2.0 times control should be maintained. However, the Kidney Disease Improving Global Outcomes (KDIGO) group recommends citrate rather than Heparin as the first-line anticoagulant for CRRT. Citrate chelates calcium and inhibits clotting thereby increasing hemofilter survival time. There is a significantly decreased bleeding risk in the critically ill with the use of citrate. If this is used as an anticoagulant, ionized calcium levels should be monitored; calcium should be removed from the dialysate fluid to prevent reversal of citrate effect; and bicarbonate and lactate should also be removed due to the alkali load of the citrate (Dickie, Tovey, Berry & Ostermann, 2015). A brief description of some types of CRRT follows.

Slow Continuous Ultrafiltration. **Slow continuous ultrafiltration (SCUF)** uses ultrafiltration alone to remove 100 to 300 mL of fluid hourly from the patient. It has little ability to remove urea and other midsized molecules, so its usefulness for patients in AKI is limited. SCUF utilizes both arterial and venous accesses and therefore does not require a pump but does require a MAP greater than 70. It does not require dialysate or replacement fluid.

Continuous Venovenous Hemofiltration. **Continuous venovenous hemofiltration (CVVH)** uses convection with replacement fluid flowing through the filter at a fast rate (1000 to 2000 mL/hr), creating a “solute drag” that removes fluids and small to midsized molecules. This results in removal of significant amounts of fluid (7 to 30 L/24 hr) and adequate amounts of urea and other midsized molecules. It utilizes venous accesses only and requires a pump as well as replacement fluid.

Continuous Venovenous Hemodialysis. **Continuous venovenous hemodialysis (CVVHD)** is a continuous process

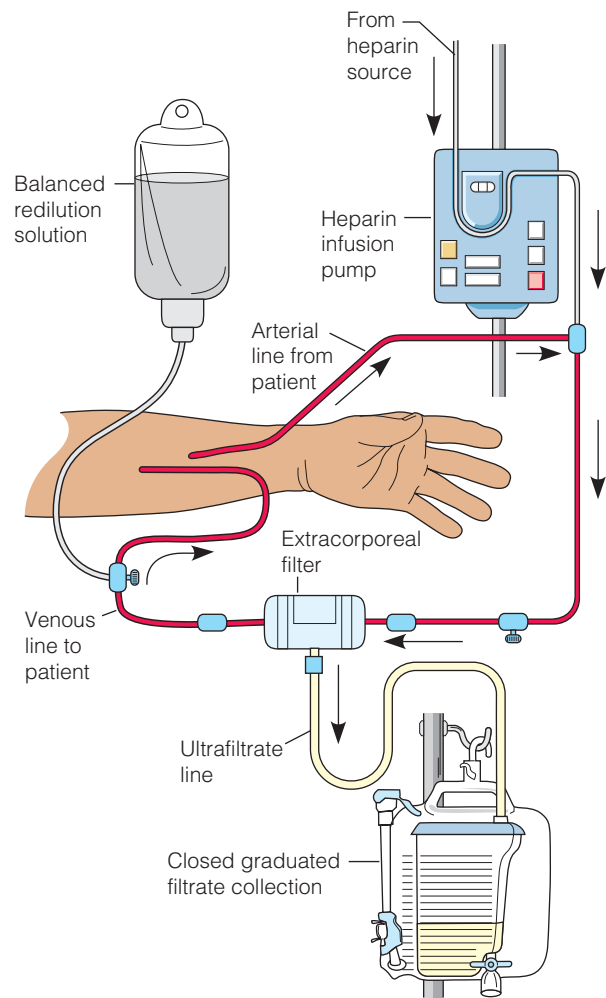


Figure 15-12 Continuous venovenous hemodiafiltration circuit (CVVHDF).

of slow dialysis that works by diffusion. However, because it removes mostly small molecules, its usefulness is somewhat limited. It requires dialysate fluid but does not require replacement fluid.

Continuous Venovenous Hemodiafiltration. **Continuous venovenous hemodiafiltration (CVVHDF)** is the most popular and efficient mode of CRRT because it is able to remove small and midsized particles and clear urea quickly. It uses both convection and diffusion simultaneously to remove fluids and solutes. It utilizes venous accesses only and requires a pump, dialysate fluid, and replacement fluid.

♦ Figure 15-12 displays a CVVHDF circuit.

Nursing Responsibilities

According to Richardson and Whatmore (2015), there are four essential principles for which the nurse is responsible: continuous assessment of the indications to influence the appropriate mode of CRRT; ensuring good vascular access; avoidance of unnecessary interruptions

and the preventions of complications. Prior to the start of any form of CRRT, the nurse assesses the patient's volume status and discusses initiation of the therapy with the patient and/or healthcare proxy as she or he would prior to hemodialysis. The nurse then clarifies with the nephrologist the fluid balance goal. Lab studies, including electrolytes, calcium, phosphorus, BUN, creatinine, glucose, PT/PTT, and CBC are drawn before the procedure is started. The ICU nurse is responsible for priming all lines in the circuit and expelling all air. Sterile technique should be maintained throughout the connection procedure and any time that the circuit is entered.

During the process, the nurse:

- Monitors the patient's vital signs and hemodynamic and fluid balance status every half hour.
- Assesses the ultrafiltration rate hourly and administers replacement fluid (if required by the CRRT method). The amount of replacement fluid administered each hour is determined by the hourly fluid balance goal set by the nephrologist. The hourly goal for fluid removal is added to all sources of fluid input in the previous hour. That sum is then subtracted from the sum of all sources of fluid output. The result is the amount of replacement fluid that needs to be provided in the subsequent hour. The type and composition of replacement fluid are prescribed by the physician and determined by the patient's electrolyte and acid-base status. Lab studies to monitor the patient's electrolyte status, BUN, and creatinine are checked at least twice a day but often every 4 to 6 hours.
- Maintains the patency of the system by ensuring adequate blood flow rates maintaining safe anticoagulation and assessing the hemofilter for clotting. If heparin is used, PTT and clotting studies may should be checked as often as every one to two hours.

Potential Complications

Frequent complications include hypotension, hypothermia, electrolyte imbalances, coagulation abnormalities or bleeding, and sepsis. Transient hypotension is not uncommon when CRRT is initiated. If this occurs, the nurse might assess the patient for blood loss, decrease the rate of ultrafiltration temporarily, then gradually increase it again, increase vasopressor support, and/or notify the physician. The patient may develop hypothermia and experience chills. This may be prevented or alleviated by a warmer on the blood return line to the patient or a warming blanket. Monitoring lab studies every 4 to 6 hours will help to prevent electrolyte imbalances and coagulation abnormalities or bleeding. Keeping all connections

securely tightened and all lines visible at all times will help to limit bleeding. Sepsis is the primary cause of death for patients in AKI; to assist in preventing it, all access dressings should be done aseptically, and all CRRT lines should be cared for meticulously.

Determining when the patient can be successfully weaned from RRT can be difficult. Studies suggest there is a relatively good chance of being successful in stopping RRT when the patient's spontaneous, meaning without the use of diuretics, urine output exceeds 400 mL in 24 hours (Ronco et al., 2015).

Peritoneal Dialysis

Peritoneal dialysis (PD) uses osmosis and diffusion to produce a steady, gradual restoration of fluid, electrolyte, and acid-base balance while removing nitrogenous and other wastes.

Indications and Expected Outcomes

The gradual restoration of fluid volume and electrolyte balance that results from PD can be an advantage because the patients are less likely to experience hypotension, dysrhythmias, or electrolyte imbalances than with hemodialysis (♦ Figure 15-13). However, PD is rarely used in AKI because there are a variety of disadvantages. First, PD is often not speedy or efficient enough to adequately remove the midsize wastes such as urea, which

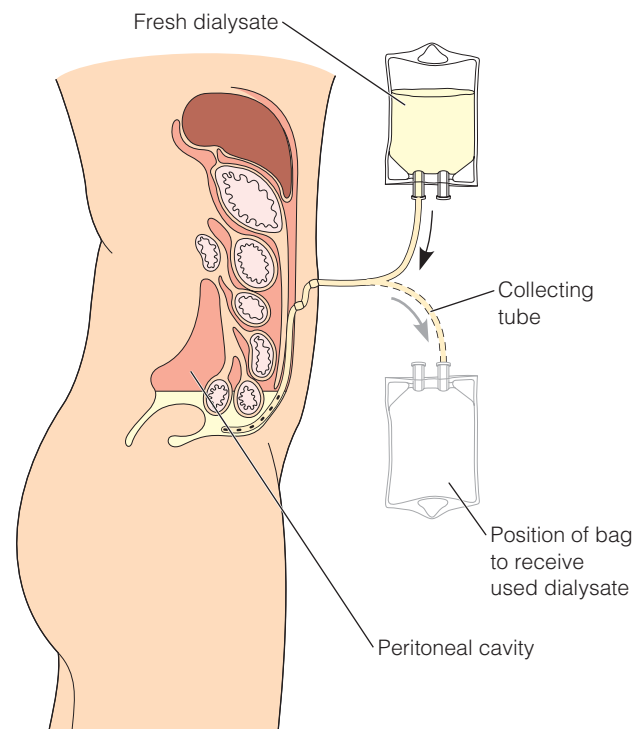


Figure 15-13 Peritoneal dialysis.

accumulate rapidly in catabolic AKI patients. Second, the volume of fluid that is placed in the peritoneum in PD tends to have a negative impact on respiratory function. Lastly, several studies have demonstrated poorer outcomes for patients who received PD rather than other modalities of treatment for AKI (Góes, Berbel, Balbi, & Ponce, 2014).

Technological Requirements

PD may be managed manually or by an automated PD system (cycler). In either case, there are three steps to the process and the critical care nurse is usually responsible for the functioning of the system.

During inflow, a specific amount of sterile, body temperature solution (dialysate) is allowed to infuse into the peritoneum over approximately 5 to 10 minutes. After completion of inflow, the dialysis tubing is clamped.

During the dwell stage, the second step in the process, the dialysate dwells in the peritoneum. Diffusion and osmosis occur between the blood and the dialysate across the peritoneal membrane. The duration of the dwell time is determined by the type of PD and prescribed by the nephrologist. In manual or continuously cycling PD, the dwell time is often 30 to 40 minutes.

At the completion of the dwell time, either the inflow tubing or tubing leading to a collection system is placed below the patient's body and unclamped so the dialysate drains from the patient's body by gravity. Drainage should occur rapidly and be completed within about 20 minutes. The entire cycle is repeated for the amount of time, or the total number of cycles prescribed by the nephrologist with new dialysate for each cycle. At the completion of the series of dialysis cycles, the patient may be left either with an empty peritoneum or with dialysate dwelling in the peritoneum.

ACCESS

Access to the peritoneum is by tunneled catheter (♦ Figure 15-14). The catheter has several sections, the first outside the body, the next located in the subcutaneous layer and having at least one dacron cuff or flanged collar to anchor the catheter, and a section in the peritoneal cavity with multiple lumens for rapid delivery of fluid. The nurse maintains aseptic technique when caring for the catheter and assesses the access site at least daily. Infections of both the access site and the tunneled catheter may develop. Indications of these infections include redness, swelling, and exudates at the catheter site or along the tunneled tract. If exudates or leakage occurs at the access site, by testing for the presence of glucose, the nurse can determine if the leaking fluid contains dialysate. If the fluid contains dialysate, it will test positive for glucose.

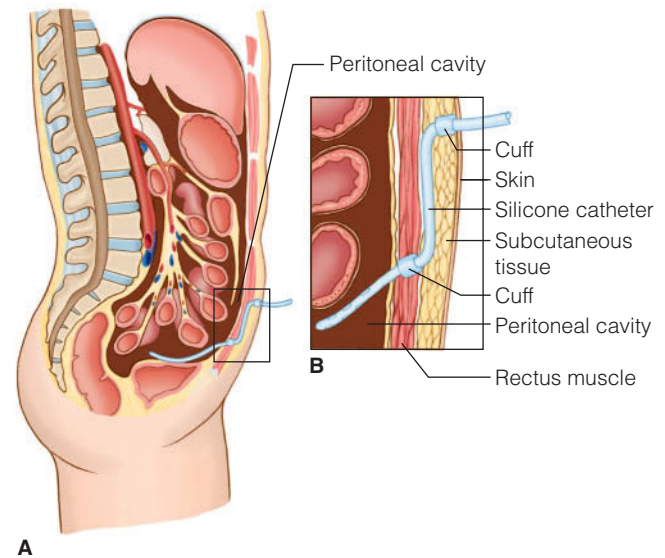


Figure 15-14 Peritoneal dialysis access.

Nursing Responsibilities

Prior to the onset of PD, the nurse assesses the patient's volume status as she would if the patient were receiving hemodialysis. The nurse also pays specific attention to the patient's temperature and laboratory studies to detect infection or severe electrolyte imbalances. The nurse identifies whether the patient should begin the dialysis process with the inflow step, or whether the patient has had dialysate dwelling in her peritoneum and should begin with the drain step.

The nurse is responsible for setting up the PD system, whether it is a manual system or a cycler. First, the nurse selects the appropriate concentration of dialysate solution. The nephrologist will have prescribed the concentration of glucose in the dialysate (1.5%, 2.5%, or 4.5%) based on the amount of fluid to be removed. The nephrologist may also prescribe additives such as antibiotics, potassium, insulin, or heparin for the dialysate, and the nurse ensures that they are present in the appropriate amounts. The dialysate must be warmed to body temperature. If the procedure is being performed manually, this is usually done before the dialysate bag is spiked. It should be warmed by an approved device, and not in a microwave or ever in a water bath. The connections are then made. At each step in the process, the nurse must ensure that all connections (between dialysate bags, tubings, and patient) are done aseptically according to agency policy.

After the connections are made, PD can begin. If a cycler is being used, it will warm the dialysate prior to inflow and ensure that the inflow, dwell, and drain times are maintained for the entire time of the dialysis. If manual dialysis is occurring, the nurse must time each of the

stages and monitor the process. At the completion of each dwell time, the dialysate return must be either measured (by the nurse) or weighed (by the machine) to determine the amount of return. If the dialysate return is less than the amount of dialysate infused, there may be an obstruction. This may be resolved by examining the tubing for a kink or helping the patient change position. If the catheter is starting to clot off, heparin may need to be added to the dialysate.

The nurse also inspects the dialysate return. It should be clear enough to read newspaper print through. Cloudy dialysate is suggestive of infection and should be reported immediately to the nephrologist or dialysis center.

After completion of the entire series of cycles, the tubing should be disconnected aseptically, the patient's access capped, the access site covered with a sterile dressing, and the patient weighed.

Potential Complications

The most significant risk to the patient with PD is the development of peritonitis. It is usually caused by contamination of the system, and that is why it is essential that aseptic technique be maintained consistently. The nurse continually observes the patient for signs that peritonitis may be developing. These include an elevated temperature or white blood count, malaise, cloudy dialysate return, and abdominal pain that is most pronounced on inflow. Treatment of peritonitis may involve adding an appropriate antibiotic to the dialysate fluid.

Recovery from Acute Kidney Injury

When patients respond to supportive treatment and do not develop complications such as sepsis, they usually regain renal function. As renal function returns, patients enter a diuretic phase when they may fail to concentrate urine and produce up to 5 liters of urine each day. In addition, they may not be able to adequately excrete wastes or regulate their electrolyte and acid-base balance. Their serum BUN and creatinine levels usually decrease slowly toward normal over a 10-day period. Patients may continue to need RRT during this phase until their GFR improves sufficiently to clear metabolic wastes. During this phase, the nurse needs to carefully monitor the patient's:

- BUN and creatinine
- fluid volume status for a fluid volume deficit
- serum electrolytes for hyponatremia and hypokalemia that may result from the diuresis.

The final phase of acute kidney injury is the recovery phase. This phase typically lasts from 6 months to a year. During this phase, most patients' renal function returns to normal. Only between 10% and 15% of patients who survive continue to require dialysis. However, 30% to 40% of patients have decreased renal function. These patients are thought to be vulnerable to repeated insults to their renal function and are counseled to avoid renal toxins to prevent them from developing chronic kidney disease. Table 15-3 contrasts chronic kidney disease with acute kidney injury.

Table 15-3 Contrast of Chronic Kidney Disease (CKD) with Acute Kidney Injury (AKI)

	CKD	AKI
Causes	Hypertension Diabetes Polycystic disease Analgesic abuse Glomerulonephritis Vascular disease Postrenal causes	Prerenal (due to decreased renal perfusion) Intrarenal or intrinsic (due to direct damage to the kidneys—e.g., drug or dye induced) Postrenal (due to obstruction of urine flow)
Phases	Phase 1 (decreased renal reserve) GFR > 90 Phase 2 (mild decrease in renal function) GFR < 90 Phase 3 (moderate decrease in renal function) GFR < 60 Phase 4 (severe decrease in renal function) GFR < 30 Phase 5 (ESRD) GFR < 15	Onset Oliguric Diuretic Recovery
Course	Slow progression to ESRD	Abrupt onset Rare progression to CKD Recovery or death dependent on development of sepsis and/or MODS
Management	Conservative Dialysis (hemo or peritoneal) Transplant	Hemodialysis Hemofiltration
Concerns	Better toleration of long-term electrolyte and fluid imbalances Disruptions in all body systems	Electrolyte, fluid, and acid-base imbalances increase rapidly and may be severe Sepsis MODS

Acute Kidney Injury Summary

Only recently has a consensus been reached on a definition, diagnostic criteria, and new name for acute renal failure now known as acute kidney injury. With the developing understanding of acute kidney injury there is the potential for utilization of biomarkers to allow early detection and intervention in AKI. In the meantime, the nurse attempts to

prevent AKI in critically ill patients by maintaining their fluid volume status and renal perfusion pressure while avoiding nephrotoxic agents and preventing sepsis. Unfortunately, once AKI develops, it is fatal approximately 60% of the time even when renal replacement therapies are utilized.

Why/Why Not?

The patient is recovering from aortic valve replacement surgery which was performed for bacterial endocarditis. He has developed acute tubular necrosis and a dialysis catheter has been placed in the left femoral vein for dialysis. The patient was seen by Infection Disease and has been on Vancomycin 1 gram IV BID and Ceftriaxone 4 gms IV q24 hours

for treatment of gram (+) blood cultures and presumed Staph Aureus endocarditis. Would you administer these medications as ordered? Why? Why not?

See answers to Why/Why Not? in the Answer Section.

Case Study

A 42-year-old woman who had been found by her neighbor lying in her closet was admitted to the hospital with the diagnosis of AKI and weakness. On admission, she was barely verbal but complaining of severe back and leg pain. Her vital signs were: BP of 78/50, pulse 56, respirations 22, and temperature of 102. She was unable to state how long she had been lying in the closet, but there were pressure ulcers forming on her right arm and leg. According to her medical record, she had a history of pneumonia complicated by renal failure, hepatitis C, and bipolar disorder. On admission, her labs revealed the following:

WBC 21.1, Hg 17.2, and HCT 52.7

In conjunction with her BP, what do these labs imply about her fluid status?

Her BUN was 88, creatinine 5, potassium 6, calcium 8.4, and CK 23,890.

Creatine kinase (CK) levels increase with skeletal and heart muscle breakdown; the normal level for women is 26 to 140 U/L.

1. What do these lab studies reveal about her renal status?
2. Why might her CK be so elevated?
3. What are the likely factors contributing to her current renal failure?
4. What should the immediate management strategies be for her AKI?
5. Is renal replacement therapy indicated at this time? Why or why not?

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 15.1 Why does urinalysis help to distinguish between the prerenal and intrinsic causes of acute kidney injury? How does it distinguish between them?
- 15.2 What are the major causes of intrinsic renal failure in the critically ill patient? What can the nurse do to prevent intrinsic renal failure in the critically ill patient?
- 15.3 What are the indications of fluid volume excess seen in the patient with AKI?
- 15.4 Which of the electrolyte imbalances is the most dangerous in AKI? Why? How is it managed emergently?
- 15.5 Why is peritoneal dialysis of limited use in patients with AKI?
- 15.6 What are the advantages of hemodialysis for the patient in AKI?
- 15.7 What is dialysis disequilibrium syndrome? Why does it occur?
- 15.8 What is the function of the replacement fluid utilized in some forms of CRRT?

See answers to Chapter Review Questions in the Answer Section.

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Chapter 16

Care of the Organ Donor and Transplant Recipient

Allanah M Bachman, MSN, ACNP-BC

Abbreviations

DGF	Delayed Graft Function	OPO	Organ Procurement Organization
MELD	Model for End-Stage Liver Disease	UNOS	United Network for Organ Sharing



Learning Outcomes

Upon completion of this chapter, the learner will be able to:

1. Interpret the concepts of basic immunology.
2. Distinguish the eligibility for and characterize the care of the transplant donor and family.
3. Critique the interrelationship between immunosuppression and rejection and how this is manifested in transplant recipients.
4. Compare and contrast the collaborative management of the transplant recipient patient.

Introduction

The demand for donated organs in the United States has far outstripped the supply of available organs. This is largely because the patient who receives a donated organ, such as a kidney, usually lives a longer life with better quality at less cost to the healthcare system. However, to achieve these results, the organ donor and transplant recipient require expert healthcare. Nurses provide skilled care to patients in all stages of organ donation. Nurses identify and care for potential donors, provide information to patients and families making a decision about donation or transplant, and provide nursing care for the organ recipient.

Review of Basic Immunology

When the body detects the presence of a foreign substance, the immune system responds in the two overlapping ways that are displayed in ♦ Figure 16-1.

Cell-mediated immune response results in the stimulation of T-lymphocyte activity. T lymphocytes recognize foreign antigens, bind to them, and produce sensitized clones of the T lymphocyte. These sensitized T lymphocytes migrate throughout the body to locate the antigen, bind with it, and release chemicals that result in lysis of the foreign cells.

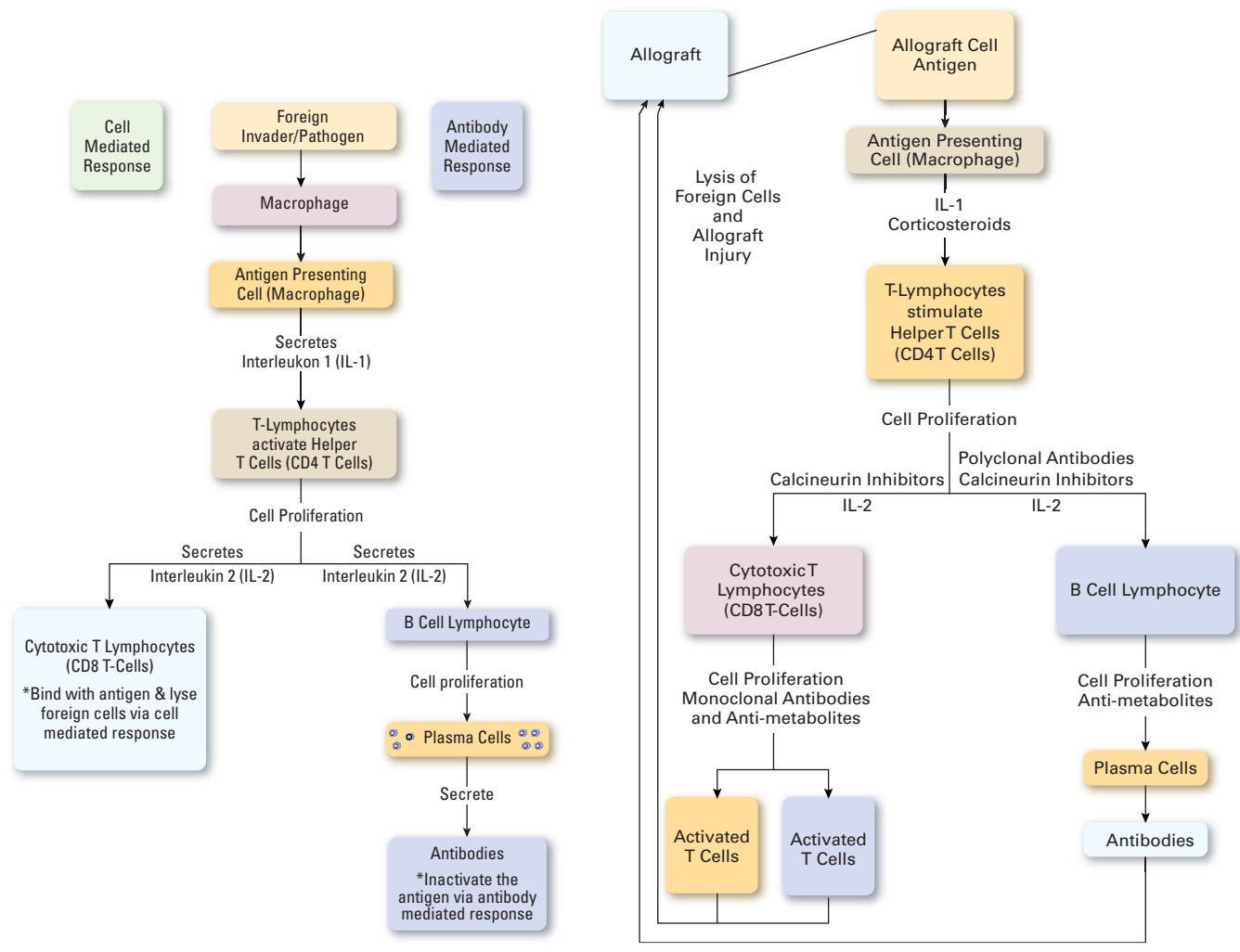


Figure 16-1 Overview of cell and antibody mediated immune response.

Antigens such as viral infected cells, cancer cells, and foreign tissue usually trigger cell-mediated responses.

Antibody-mediated (humoral) immune response results in the stimulation of B-lymphocyte activity. The B-lymphocyte differentiates into a plasma cell that produces antigen-specific antibodies that bind to the antigen and inactivate it. This initial response is followed by inflammation and phagocytosis. Bacteria, bacterial toxins, and some viruses are the usual triggers for antibody-mediated responses.

Antigens are substances that are recognized by the body as foreign or non-self. The human leukocyte antigen (HLA) system is found on Chromosome 6 and helps the immune system determine what tissue is foreign and what is self. **Tissue typing** is used to determine how much overlap exists between a potential donor's and the recipient's HLA antigens. Although there may be overlap in HLA antigens between siblings, and parents should each be half similar to their children, complete matches occur only with identical siblings.

A recipient may have antihuman antibodies in his or her blood, which may have developed from a previous blood transfusion, organ transplant, or during pregnancy, that would react with the tissue of a potential donor. The potential recipient cannot receive a transplant from that donor because the antibodies would immediately attack and damage the organ. Called *hyperacute rejection*, this type of rejection usually results in loss of the transplanted organ.

ABO typing identifies the blood group of a donor and recipient. Unmatched blood or organs that are transplanted across ABO compatibility will cause an immediate immune response. Type AB is considered to be a universal recipient because the person can receive blood or organs from any ABO group. Type O is a universal donor because any other group can accept the blood or organ. However, a person with type O can only receive blood or an organ from another person with type O. Types A and B may receive blood or organs from their own blood group or type O.

Eligibility and Care of the Transplant Donor and Family

There is a severe shortage of organ donors in the United States. Currently in 2015, there are approximately 122,000 people awaiting a lifesaving organs but there only about 14,000 donors (United Network for Organ Sharing [UNOS], 2015). A little over half of these donors were deceased individuals, and slightly under half were living donors. More specific data can be found on the UNOS website.

Living Donors

According to the UNOS (2015), over the past 5 years, living donors accounted for approximately 6000 transplants each year. Most of these living donors donated kidneys, although a small number donated a portion of their liver or lung.

Receiving an organ from a living donor provides a number of benefits for the recipient. First, living donation decreases the risk of organ **rejection** for the recipient. Because the operation can be scheduled in advance rather than as an emergency, the recipient may begin taking immunosuppressant drugs a day or two before the operation. As a result, the organ is less likely to be rejected. Also, when there is a better tissue match between the donor and the recipient, as is often the case in living donation, the recipient is less likely to experience an episode of rejection. In addition, the recipient does not have to be placed on the waiting list and wait for a match. The waiting period for an organ may be several years during which time the recipient's health often declines. In addition, the psychological benefit of organ donation can be one of the most important positive aspects of donation (UNOS, 2015). The recipient often experiences positive feelings knowing that the gift came from a loved one or a caring stranger, and the donor often has the satisfaction of having benefited another human being.

Qualifying as a Living Donor

UNOS (2015) states that the potential kidney donor must be 18 years or older, physically and mentally fit, in good general health and free from high blood pressure, diabetes, cancer, kidney disease, heart disease, and a variety of infectious diseases including the human immunodeficiency virus (HIV) and tuberculosis. Individuals considered for living donation must also be well-informed and willing to donate, and have good social and emotional support. Gender and race are not factors in determining a successful match.

Testing to Determine Eligibility for Donation

First, the potential donor's blood type is identified and compared to the recipient's to determine if they are compatible and a donation is possible. If the donor and recipient have compatible blood types, the donor undergoes a medical history review and a complete physical examination. According to UNOS (2015), the following tests may be performed:

- **Tissue typing:** The donor's blood is drawn for HLA typing.
- **Crossmatching:** A crossmatch is done just before every transplant to prevent acute rejection. In a crossmatch, the lymphocytes of the donor are mixed with the serum of the recipient. If the recipient has antibodies to the white cells of the donor, the cells are destroyed immediately. This is called a *positive crossmatch* and means that the transplant is contraindicated. If there is no reaction between the blood of the donor and the blood of the recipient (called a *negative crossmatch*), then the transplant may proceed.
- **Antibody screening:** A blood test that determines if the recipient has antibodies that could react to any of the donor's antigens.
- **Screening for transmissible diseases:** Blood tests that determine if the donor has any transmissible diseases such as HIV/AIDS, cancer, or hepatitis that could potentially be spread to the recipient or affect successful transplantation.
- **Renal function:** Serum creatinine, urea, electrolytes, urinary sediment, albuminuria, 24-hour proteinuria, creatinine clearance, and glycosuria are evaluated.
- **Renal Structure:** This set of tests includes a renal ultrasound and angiogram, allowing visualization of the blood vessels of the organ to be donated.
- **Diagnostic Screening:** Baseline tests including laboratory values, a chest X-ray and EKG to evaluate for possible heart or lung disease.
- **Psychiatric and/or psychological evaluation:** The donor and the recipient may undergo a psychiatric and/or psychological evaluation.

The Ethical Issues

Ethical questions arise when a healthy individual is asked to undergo a surgical procedure that removes a body organ solely for the benefit of another. Most agree that such a procedure is ethically acceptable when the patient provides fully informed consent. Informed consent means the living donor must be made aware of the physical and psychological risks before he consents to donate even though the risks are slim. The risk of death is low for kidney and liver donation; however, living donation involves major surgery and anesthesia. This can involve complications of postoperative

infections, pain, blood loss, blood clots, injury to the surrounding tissues or structures, reactions to anesthesia, or pneumonia (UNOS, 2015). If the donor has any concerns preoperatively, he should be encouraged to discuss them with a transplant professional and/or social worker.

Another important element of informed consent is that the donor has made the decision to donate voluntarily and has been offered opportunities to change his mind throughout the process. If the donor does change his mind during the process, he can postpone or cancel the donation at any time, and the donor's decision and reasons should be kept confidential (UNOS, 2015).

Post-Donation Care Requirements of Living Donors

Care of the donor following the most common living donor procedure, kidney donation, is discussed because procedures are different and more complex for donation of other organs. In the past, the surgery for living kidney donation was open nephrectomy. This procedure is performed less frequently now because the donor is more likely to experience pain and have a more extensive recovery period.

One of the major concerns for the donor following open nephrectomy is pain. Post-operative nephrectomy pain is usually managed with epidural analgesia. However, even with pain relief, because of the location of the surgery, the donor may splint and limit respiratory effort to diminish her pain. Therefore, the nurse must ensure

adequate pain control; routinely assess the donor's pain level; and instruct her to turn, cough, and breathe deeply. Movement is also essential, and several hours after the operation the donor is encouraged to get out of bed and walk around. The donor may remain hospitalized for 4 to 8 days before returning home. Post-operative restrictions include no heavy lifting or rough contact sports until at least 6 weeks and can usually return to normal activity within 4–12 weeks after surgery. In recent years, laparoscopy has been used more frequently to recover kidneys from living donors because it involves a smaller incision, less pain, better cosmetic results, and a shorter recovery time. In this procedure, 2 to 3 small incisions are made and the surgeon places a laparoscope, a device with a small video camera allowing an inside view of the abdomen, and removes the kidney through one of the other openings. Donors having laparoscopic surgery recover much more quickly and are usually discharged from the hospital in 1 or 2 days and can typically return to normal activity within 4 weeks (OPTN, 2015).

Deceased Donors

In the year 2014, there were approximately 8,596 deceased organ donors but a total of 30,160 organs recovered (OPTN, 2015). This is because when deceased donors are managed intensively, multiple organs may be transplanted from a single donor. Eligible donors should have been in good health previously and are usually less than 70 years of age. However, age is a relative contraindication, and people

Safety Initiatives

World Health Organization: Global Knowledge Base on Transplantation

PURPOSE: To ensure that transplant centers worldwide address their responsibilities to living organ donors

RATIONALE: Because it has demonstrated both cost-effectiveness and enhanced quality of life for recipients, kidney transplantation is by far the most frequently carried out transplantation globally. Although kidney donation by well-selected living donors with good health coverage carries negligible risks, this level of care is not always available worldwide. According to the World Health Organization (WHO), "The welfare of the live kidney donor is largely neglected in schemes where disadvantaged individuals are exploited and encouraged to sell their kidneys. In 2004, World Health Assembly Resolution 57.18 acknowledges the risk of exploitation of live kidney donors and urges Member States to protect the poorest and vulnerable groups from transplant tourism and the sale of tissue and organs" (WHO, 2016).

HIGHLIGHTS OF RECOMMENDATIONS: The World Health Organization's Global Knowledge Base on Transplantation

(GKT) is a database that brings together information on donation and transplantation around the world. The third component of the GKT reports on information regarding safety and ethics of transplantation practice by:

- Tracking and reporting adverse events and reactions incurred by recipients and living donors to enable appropriate decisions on safety measures to be taken.
- Providing vigilance, surveillance, and the rapid dissemination of information about the transmission of infectious agents by cell, tissue, and organ transplantation.
- Establishing links to vigilance and surveillance systems set up by developed countries so a single portal is available for information on transplant safety.

Source: World Health Organization, Global Knowledge Base on Transplantation (2016). More detailed information on the GKT and the full WHO Assembly Resolution 57.18 document may be found on the WHO website.

older than 80 have been organ donors. Absolute contraindications to donation include:

- Uncontrolled sepsis
- Active viral infections (e.g., hepatitis B or C and cytomegalovirus [CMV])
- HIV positive serology
- Any malignancy except a primary intracranial tumor

There are two types of donation from deceased donors: donation after pronouncement of brain death (death by neurological criteria) and donation after cardiac death (death by circulatory-respiratory criteria).

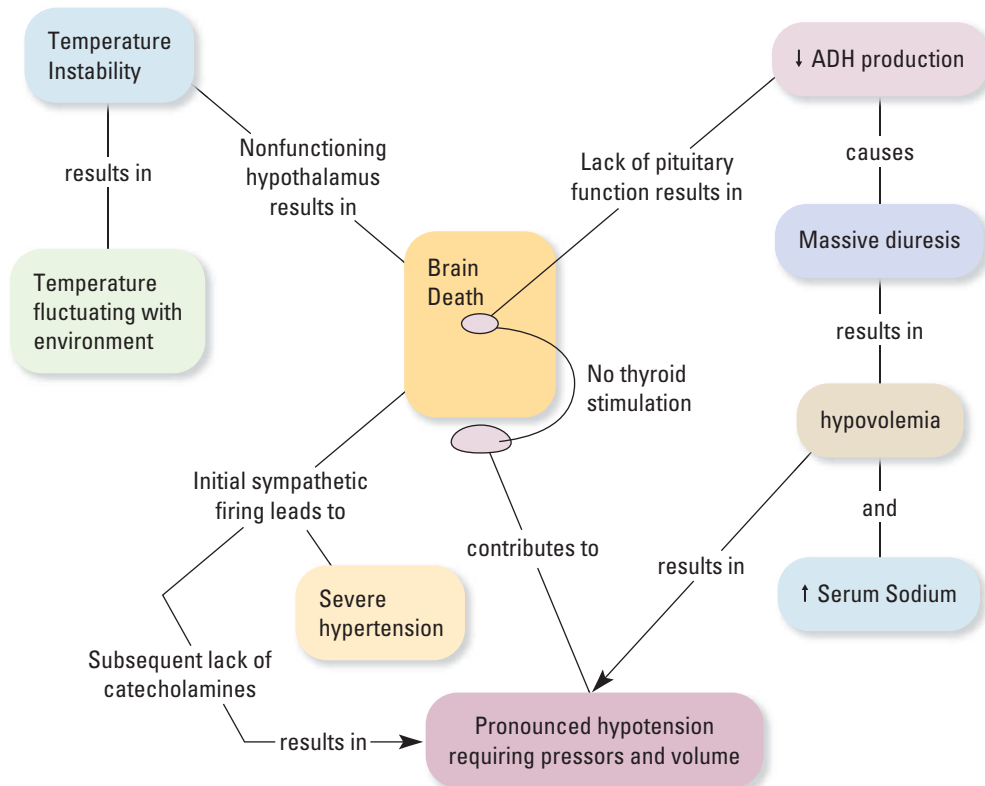
Imminent Death

Over the last several years, there has been a widening gap between the number of people waiting for organ transplantation and supply of transplantable organs. The majority of organ donation comes from deceased donors, and one organ donor can save up to eight lives (UNOS, 2015). Early notification to the Organ Procurement Organization (OPO) of any patient who meets the acute care hospital's definition of impending death or has been determined to meet brain death criteria is important. The OPO is responsible for identifying and evaluate potential donors, confirming brain death diagnosis, obtaining consent from the

family, assisting in the clinical management of the potential donor, and properly preserving and sending the organ for transplant (Finger, 2013). Organ donation should be considered as part of end-of-life decision-making and it is essential that the OPO should be notified in a timely manner. The OPO should be notified when a person has sustained a neurological injury that is not likely recoverable, when the brain death examination is being considered, and before the withdrawal of life-sustaining therapy has been discussed by the caregivers and family (Kotloff et al., 2015).

Criteria for Brain Death or Death by Neurological Criteria

Brain death is defined by the American Academy of Neurology as the irreversible loss of function of the brain, including the brainstem, or irreversible loss of circulatory or respiratory function. Common causes of brain death include severe head trauma, rupture of a cerebral aneurysm, hypoxic-ischemic brain insults, and fulminate hepatic failure (Visual Map 16-1). The most recent evidence-based recommendations for the clinical diagnosis of brain death were updated by the Quality Standards Subcommittee of the American Academy of Neurology in 2010. Subsequent testing and medical opinion have determined that they continue to be valid determinants of brain death in adult patients (Wijdicks, Varelas, Gronseth,



Visual Map 16-1 Brain Death

& Greer, 2010; Kotloff et al., 2015) when the following conditions are met:

- Evidence of an acute neurological catastrophe that is compatible with the clinical diagnosis of brain death. Neuroimaging such as a computed tomography (CT) scan is often essential for determining the cause of brain death.
- Exclusion of complicating medical conditions that may confound clinical assessment (no severe electrolyte, acid-base, or endocrine disturbance).
- Absence of drug intoxication or poisoning. Screens should be done as appropriate for alcohol, barbiturates, sedatives, and antiepileptic drugs that may affect findings on neurological examination.
- Core temperature 32°C (90°F). Hypothermia suppresses neurological activity. Pupillary response to light is lost at core temperatures of 28°C to 32°C, and brainstem reflexes disappear with core temperatures of less than 28°C. Therefore, the nurse may use thermal blankets and warm inspired air and/or intravenous solutions to treat hypothermia. A core temperature of greater than 36.5°C (97°F) is required before apnea testing is conducted.
- Normotension (systolic blood pressure [BP] greater than or equal to 90 mmHg) or euvolemia. Hypotension may suppress neurological activity and hence preclude the diagnosis of brain death. Hypotension should be corrected with appropriate therapy (usually fluid administration and vasopressors) before the apnea test is conducted.

Three Cardinal Findings in Brain Death

The three cardinal findings in brain death are coma or unresponsiveness, absence of brainstem reflexes, and apnea (Wijdicks et al., 2010; Kotloff et al., 2015).

Coma. The first cardinal finding, coma or unresponsiveness, is demonstrated by the absence of cerebral motor response in all extremities in response to noxious stimuli.

- There should be no spontaneous movement, no response to painful stimuli, and no decorticate or decerebrate posturing at any time in a brain-dead person.
- Spinal or deep tendon reflexes can occur in patients with brain death. The reflexes originate from the peripheral nerves or the spinal cord, and their occurrence does not require brain innervation. These reflexes may be generated spontaneously or by forceful flexion of the neck, body rotation, and respiratory acidosis. They include obvious reflexes such as the Babinski or knee jerk, but may also include flexion at

the waist and arm raising. It is often the role of the critical care nurse to explain the persistence of these movements to a distressed or perplexed family.

Absence of Brainstem Reflexes. The second cardinal finding, absence of brainstem reflexes, is demonstrated by the following:

No pupillary response to light:

- Pupillary reflexes should be assessed in a semidark room. A bright light is shone first into one then the other eye. Pupil size is assessed in both eyes each time. Any response to light by either pupil excludes the diagnosis of brain death. Most commonly, pupils in brain-dead patients are midposition and midsize (4–9 mm), but they may be dilated. Nonreactive round, oval, and irregularly shaped pupils are also compatible with brain death.

No ocular movement with either the oculoccephalic or oculovestibular response:

- Oculoccephalic reflex testing (doll's eyes testing) is performed only when there is no fracture or instability of the cervical spine. The patient's head is turned to one side and the eyes are examined for movement. Any movement of the eyes indicates that some pathways are intact.
- For oculovestibular reflex testing, or cold caloric testing, the ears must be free of significant wax or clotted blood, which may diminish the response to cold stimulation. The eardrums should be evaluated to determine that they are not perforated. When brain death is present, no deviation of the eyes occurs when either ear is irrigated with 50 mL cold water. A neurologist normally performs this test and any movement of the eyes indicates that some brain pathways are intact.

Absence of facial sensation and facial motor response as evidenced by the following:

- No corneal reflex to touch with a cotton swab
- No jaw reflex
- No grimacing to noxious stimuli

Absence of pharyngeal and tracheal reflexes:

- No response after stimulation of the posterior pharynx (gag reflex) with a tongue blade (Because this may not be adequate stimulus to cough for some patients, suctioning is recommended.)
- No cough response to bronchial suctioning

Apnea. The third cardinal finding, apnea, is demonstrated by apnea testing. Apnea testing must be performed according to specific parameters. As noted previously,

the patient should be normotensive and normothermic. In addition, the patient should have a normal $p\text{CO}_2$ and a normal $p\text{O}_2$. The patient should be preoxygenated with 100% FiO_2 for greater than 10 minutes to obtain an arterial $p\text{O}_2$ greater than or equal to 200 mmHg. The steps in the process according to the American Academy of Neurologists are the following:

- Connect a pulse oximeter and disconnect the ventilator.
- Deliver 100% O_2 , 6 L/min, into the trachea. Or place a cannula at the level of the carina.
- Look closely for respiratory movements (abdominal or chest excursions that produce adequate tidal volumes) for approximately 8–10 minutes.
- Measure arterial $p\text{O}_2$, $p\text{CO}_2$, and pH after approximately 8 minutes and reconnect the ventilator.
- If respiratory movements are absent and arterial $p\text{CO}_2$ is greater than or equal to 60 mmHg, the apnea test result is positive; it supports the diagnosis of brain death.
- If respiratory movements are observed, the apnea test result is negative; it does not support the diagnosis of brain death. If the test is negative and the patient remained hemodynamically stable through the first test, then a second test may be performed over a longer period of time, of approximately 10 to 15 minutes.

Confirmatory Testing. Brain death is a diagnosis for which a repeat clinical evaluation is recommended but not required. Usually the repeat evaluation is performed after 6 hours, but this interval is arbitrary. In addition, confirmatory tests are often performed. Although it is not mandatory to perform a confirmatory test, it is desirable because frequently not all components of the clinical test can be reliably performed or evaluated. The American Academy of Neurology in the most recent update to guidelines (Wijdicks et al., 2010) described the following confirmatory tests in the order of the most sensitive test first:

- A. Conventional angiography.** In brain death, there is no intracerebral filling at the level of the carotid bifurcation or circle of Willis. The external carotid circulation is patent, and filling of the superior longitudinal sinus may be delayed.
- B. Electroencephalography (EEG).** In brain death, there is no electrical activity during at least 30 minutes of recording
- C. Transcranial Doppler ultrasonography**
- D. Technetium-99m hexamethylpropyleneamine oxime brain scan.** In brain death, there is no uptake of isotope in brain parenchyma (“hollow skull phenomenon”)
- E. Somatosensory evoked potentials**

The EEG is the most commonly used confirmatory test. It is considered to be somewhat unreliable because it assesses only cerebral, not brainstem, function. Additionally, because it is usually performed at the bedside, monitors, infusion pumps, and ventilators may produce artifacts.

Reflect On

How would you explain to a distressed family that their family member is truly brain dead when the patient appears unharmed, breathing peacefully on a ventilator, and has stable vital signs?

Approaches to Obtaining Family Consent for Transplantation of Organs

In the majority of cases, the potential donor has not signed a donor card or indicated intent to donate prior to death. In these cases, the consent of a family member or person with healthcare proxy is obtained before donation.

Declaration of Death First

Before the potential organ donor’s family is approached about donation, they should have been informed about and should understand that their family member has died. Death is death whether it is pronounced by neurological criteria or the absence of a heartbeat and respiration. It is important that family members realize that once the patient has been pronounced dead, the person will either be removed from life support or support will be maintained only long enough for organ donation. It may help the family to understand that their family member is truly dead if they are told that, even with the ventilator breathing for him, a person who has been pronounced brain dead can only maintain a heart rhythm for a very limited amount of time. Many brain dead donors will experience cardiac arrest as a result of physiological instability due to significant cardiovascular, respiratory, endocrine and hematologic changes that occur following brain stem death. Intensive management of brain death donors is essential in increasing the number of transplantable organs (McKinlay, 2012).

Required Request

An individual may specifically opt in to the donor system by signing an organ donor card stating that she desires to have her organs donated, if it is possible, after she dies. Because only about 20% of the American population has indicated such a preference, Congress enacted required request legislation. **Required request** stipulates that when a person is imminently dying, the regional organ donor

bank must be notified so the organ bank can determine if the patient qualifies as an organ donor. If the patient does qualify but has not signed a donor card, the family must be asked if they are willing to donate the patient's organs.

Reflect On

How would you legally obtain the consent for organ donation when the family members disagree on what they believe the patient would want?

Nurses' Role in Organ Donations

Within the current system, nurses have a significant role in enhancing donations. Nurses are often the healthcare providers most intimately involved with the bereaved family. The amount of trust that is established between the family and the healthcare team during the period just preceding the patient's death is a factor in which families decide to donate organs. Irving et al. (2012) reviewed multiple studies that looked at factors that affect the decision to donate organs. The primary factors associated with a family's refusal of organ donation revolved around eight main themes. These being relational ties, religious beliefs, cultural beliefs, family influence, body integrity, healthcare system interaction, knowledge about donation, and participant(s) reservations or misgivings about organ donation.

When and how the family is approached about organ donation can also affect whether they will donate. Families are more likely to donate organs if:

- they are NOT asked to donate at the same time that they are informed their family member has died.
- they perceive that the timing of the request is optimal.
- they view the requestor as sensitive to their needs.
- they are approached in a quiet, private place where they can consider their options.
- an organ procurement specialist from an organ donor bank makes the request.

These simple interventions can increase the percentage of families who decide to donate.

Barriers to Organ Donation

Normally, between 30% and 50% of families asked to donate organs refuse. Some of the reasons are lack of time and private space as described previously, but in the absence of a donor card or a discussion with the patient about donation, many families believe they cannot be sure of the patient's wishes. Therefore, many families hesitate and refuse to donate. In addition, despite the American Academy of Neurology's guidelines of brain death, there is

a lack of standardization regarding the definition, legal interpretation, and declaration of brain death from state to state. To make matters more complicated, global guidelines amongst other countries are also widely variable. Racial and ethnic, as well as religious considerations play an important role in the decision to be an organ donor. Most major religions support organ and tissue donation but within each religion there can be different perspectives (DaSilva & Frontera, 2015). It is important for the nurse to have cultural and religious sensitivity and understand that the use of faith-based counseling may be helpful in end-of-life decisions-making and organ donation.

NURSING ACTIONS

The critical care nurse is actively involved in the identification, evaluation, and provision of care to the deceased organ donor. It is essential for the critical care nurse to know how to identify a potential donor, to recognize when a patient meets the agency's definition of impending death, and to promptly notify the appropriate Organ Procurement Organization (OPO). In addition, nurses should partner with their OPO, introducing themselves to the OPO coordinators and working with them to establish protocols. Nurses should serve as advocates for their patients and families by honoring the patients' last wishes, upholding the families' right to be offered the option of organ donation, and promoting compassionate communication between the healthcare team and the potential donors' families.

Communication between family and healthcare team is essential. Although it is the responsibility of the neurologist or critical care intensivist to initially offer the explanation of brain death and the certified organ requestor to approach the family about donation, it is the nurse who must reinforce the explanations and ensure the family's understanding. When families have someone with whom they can consistently communicate their concerns and ask their questions, they are more likely to donate their family members' organs.

Care of the Donor's Family

Although the bedside nurse will not be the healthcare professional who initially explains brain death or requests the donation from the family, there is a great deal the critical care nurse does to influence the family's decision about donation. First, the nurse will need to be sensitive to what the family is experiencing. Does the family perceive the nurse as being harried or uncaring? Or, does the nurse have time to welcome them, spend time with them, and answer their questions? Are they hearing differing pieces of information and opinions from different healthcare providers? Or, have the healthcare providers spoken with

each other and does the nurse reaffirm the information that has been given to the family? One of the major issues that family members may need to keep revisiting is: What happened? How did the patient go from being a healthy and productive person to suddenly being brain dead?

When speaking with the family, another of the specific things the nurse may need to explain or reinforce is that the time of death was when the patient was declared dead and not when the patient was taken to the operating room (OR). This reassures the family that donation was not the cause of the patient's death. The family might not understand testing for brain death, particularly apnea testing. The nurse may need to explain that a brain-dead person will not have the urge to breathe. Family members can also be confused by the occurrence of spinal reflexes, muscle fasciculations, and ventilator autotriggering and may think that these represent actual patient movement. The nurse may need to explain that they are only reflexes and do not depend on the function of the brain to occur. It is important that the nurses and clinicians taking care of the patient and family use clear and consistent terminology and definitions to help families understand brain death, so as to minimize any confusion or misunderstanding (Arbour, 2013).

Families need privacy and a chance to say goodbye. They benefit from a quiet space with minimal disruption. Most families want time alone with the donor at some time. Some stay in the hospital and say goodbye just before the patient is transported to the OR for organ retrieval. Many ask for some private time to say goodbye after the patient has been declared brain dead and they have signed the consent form for donation. After that, they may leave the hospital, perhaps hours before organ retrieval is scheduled in the OR.

COLLABORATIVE CARE

The nursing and medical care of the deceased individual awaiting organ recovery is extensive. To maximize the number and viability of transplanted organs, a representative of the OPO is usually responsible for completing the evaluation and managing the care of the prospective donor. The nurse may need to reassure the family that it is the organ procurement organization that assumes the financial cost of the evaluation and management, not the family.

Because nursing care of the deceased donor is complex and time consuming, the nurse should recognize that she is providing care not to just a single patient (the donor) but to multiple patients (the recipients). By providing optimal, intensive care to the donor, the nurse is investing in better outcomes for many other patients. During this time, the nurse's focus is on attaining the goals of hemodynamic stability and body homeostasis until the organs are retrieved. The longer it takes to declare brain death and obtain authorization for

organ donation, the more likely the risk of organ loss due to hemodynamic instability (Arbour, 2013).

Achieving these goals requires intensive care because brain death is a catastrophic event associated with disruption in autonomic and other basic organ functions. Shortly before herniation, a massive sympathetic outflow occurs in response to cerebral ischemia. The donor's BP rises dramatically in an attempt to maintain cerebral perfusion, resulting in an increase in afterload, possible cardiac ischemia, and decreased cardiac output. A neurogenic pulmonary edema may also develop. Concurrently, ischemia and dysfunction of the hypothalamus and pituitary gland result in temperature and endocrine instability. After herniation occurs there is a profound reduction in sympathetic activity, resulting in pronounced vasodilation and a sudden drop in BP.

Management of Hemodynamic Instability

Hypotension is a common development in organ donors and often requires vasoactive medications for management. It may be due to the reduction in sympathetic tone described previously, or it may result from hypovolemia, decreased cardiac output due to left heart failure, and/or endocrine instability. According to Kotloff et al. (2015), the goal of therapy is to maintain patient stability as evidenced by:

- Mean arterial pressure (MAP) greater than or equal to 60.
- Urine output greater than or equal to 1 mL/kg/hr.
- Ejection fraction greater than or equal to 45%.
- Vasoactive drug requirement at a minimum.

Because hypovolemia occurs frequently in the organ donor, invasive monitoring techniques are often used to guide fluid administration. Central venous and pulmonary artery (PA) catheters may be placed in order to attain central venous pressures, pulmonary capillary wedge pressure (PCWP), cardiac output (CO), cardiac index (CI), stroke volume and mixed venous oxygen saturation to help guide therapy in brain-dead patients. Depending on the patient's serum sodium level, sufficient volumes of either lactated Ringer's or normal saline may be administered to help achieve a euvolemic state in the donor (Kotloff et al., 2015).

If the donor continues to experience hemodynamic instability despite volume replacement and appropriate vasoactive medications, therapy with thyroid hormone (T_4), corticosteroids, and vasopressin may be instituted. Although there is inconsistent support in the literature for the use of thyroid hormone, many providers believe hormone replacement therapy with a combination of thyroid hormone and corticosteroids is helpful in stabilizing the hemodynamically unstable donor, and some are evaluating

using it for all donors. Brain death can cause hormonal imbalances and place donors at risk for adrenal insufficiency and cortisol deficiency. This hormonal imbalance can trigger a cascade of pro-inflammatory mediators, which can affect the viability of graft function after donation (Kotloff et al., 2015).

Management of Diabetes Insipidus

One of the reasons for hypovolemia in organ donors is **diabetes insipidus (DI)**. DI develops in 40% to 70% of deceased donors due to insufficient levels of antidiuretic hormone (ADH) and results in the production of large volumes of dilute urine. Typical findings for a patient with DI include:

- Urine output greater than or equal to 300 mL/hr
- Urine specific gravity less than or equal to 1.010
- Serum sodium greater than or equal to 150
- Absent urine glucose

If the donor is producing about 200 mL/hr of urine, the nurse usually measures the hourly urine output and replaces that volume in the subsequent hour with an equivalent amount isotonic crystalloid solution like 0.9% normal saline and lactated ringers solution. Both isotonic crystalloid and colloid solutions are acceptable for intravascular volume replacement. However, crystalloid is the preferred choice. Hetastarch colloid is associated with coagulopathy and kidney injury so it should not be routinely used (Kotloff et al., 2015). Once the donor's urine output exceeds 300 mL/hr, then either desmopressin or vasopressin is also usually administered.

Management of Ventilation

If the transplant team is considering recovering the donor's lungs, it is essential to avoid excessive fluid replacement and to prevent atelectasis. Providing sufficient fluid to maintain a CVP of 6 to 8 or PAWP of 8 to 12 while replacing fluid lost due to diuresis usually helps to prevent fluid overload and limit pulmonary edema. Aggressive pulmonary management is needed to prevent atelectasis. Many potential lung donors have a bronchoscopy to assess their lungs for infection or aspiration and aid in the prevention of atelectasis. Donors receive vigorous pulmonary hygiene. Ventilator settings should be reviewed carefully. The intent is to attain the highest level of oxygen concentration in the donor's blood (PaO_2) with the lowest amount of inspired oxygen (FiO_2). A $\text{PaO}_2/\text{FiO}_2$ ratio of more than 300 mmHg is typically the minimal acceptable level for lung donation. Ventilator strategies using lung protective methods, similar to strategies used in ARDS, use tidal volumes of 6–8 mL/kg and PEEP of 8–10 cm has shown to improve lung recovery rates and is recommended (Kotloff et al., 2015). In addition, the use of bronchodilators and the administration of antibiotics have been shown to improve the likelihood of successful lung transplantation.

Management of Electrolyte Imbalances

The goal is to maintain the serum sodium less than 150 and the serum potassium greater than 4.0 with any acidosis corrected by mildly hyperventilating the donor to maintain a pCO_2 of 30 to 35.

Commonly Used Medications

Vasopressin (Pitressin)

Introduction

Vasopressin (Pitressin) is an antidiuretic hormone used to counteract DI caused by a deficiency of endogenous vasopressin. Vasopressin has two primary functions; it acts directly on stimulating smooth muscle receptors resulting in vasoconstriction of blood vessels and it increases water reabsorption in the kidney. Its effectiveness should be identifiable within minutes as shown by a decrease in urine output and an increase in urine specific gravity. The alternative to treatment with vasopressin is administration of desmopressin (DDAVP). DDAVP has the primary effect of being anti-diuretic, with less of the vasopressor response. Therefore, DDAVP is a good drug choice for a patient with DI that does not also have hypotension (Kotloff et al., 2015). Desmopressin can be given nasally at 2 to 4 mcg every 4 to

6 hours, orally at 0.1 to 1 mg/day in two or three divided doses, or IV/SC at 1 to 4 mcg/day in two to four divided doses.

Nursing Responsibilities:

- Vasopressin may be administered SC, IM, or IV. Vasopressin SC or IM is dosed 5 to 10 units BID to QID. Vasopressin by IV infusion is usually administered at a rate beginning at 0.01 to 0.1 unit/min. It may be titrated depending on urine output, blood pressure, and systemic vascular resistance (SVR).
- Monitoring hourly urine output and urine specific gravity or osmolality. If the donor's urine output drops below 1 mL/kg/hr, he may be given a minimal (20 mg) dose of furosemide to increase the urine flow.

Management of Hypothermia

Following brain death, dysfunction of the hypothalamus often occurs. The donor becomes poikilothermic, which means that his body temperature fluctuates with the temperature of the environment. If the room is cool, the donor is predisposed to hypothermia, which can be worsened by administration of cool IV fluids or ventilator air. Because hypothermia can lead to problems with coagulation and cardiac irritability and can reduce oxygen delivery to the tissues, the donor's temperature is closely monitored. The nurse might adjust the room temperature to about 90°F and administer warm IV fluids or cover the patient with warming blankets to maintain a body temperature of approximately 36.5°C (98°F).

Donation After Cardiac Death

Another possibility for organ donation from deceased individuals is known as donation after cardiac death. Potential donors, known as **nonheartbeating donors**, are patients who do not meet the criteria for brain death but who are gravely ill and dying. Most have sustained a devastating nonrecoverable neurological injury that did not result in brain death. The patient, or more likely the patient's proxy, has determined in conjunction with the healthcare team that the patient would wish to have life-sustaining therapy withdrawn so she could die and would want to donate her organs after death.

Evaluation of the Donor

The first step in the process is the determination that the patient would want to have life support withdrawn. After that decision has been made, the patient is evaluated first to determine if he is medically suitable as an organ donor, but additionally if death will most likely occur within an hour following the withdrawal of life support. An hour is considered the maximum acceptable interval between withdrawal of support and recovery of organs for minimizing ischemic damage. The University of Wisconsin has developed a cardiac death evaluation tool to determine if a patient is likely to have cessation of cardiorespiratory function within an hour following withdrawal from a ventilator. The evaluation includes the following steps:

Step 1: Recording the patient's type of intubation and vasopressor status

Step 2: Recording the patient's vital signs and height and weight

Step 3: Disconnecting the patient from the ventilator for 10 minutes then reassessing the respiratory effort (respiratory rate, tidal volume, and inspiratory force) and vital signs. The assessment is stopped if the patient's O₂ saturation drops to less than 70% or his systolic BP to less than 80.

Utilizing the scoring criteria associated with the evaluation allows prediction of approximately 83% accuracy of those patients who will die within 60 minutes following withdrawal of life support (Kotloff et al., 2015). If cessation of cardiorespiratory effort seems likely and the patient otherwise is medically suitable as an organ donor, the family is counseled about the possibility of organ donation. Next, authorization for donation and medical examiner clearance are obtained, and the organ recovery team is notified. While awaiting the arrival of the organ recovery team, the patient is cared for by the hospital's normal critical care team, not the OPO. Goals of care include maintaining:

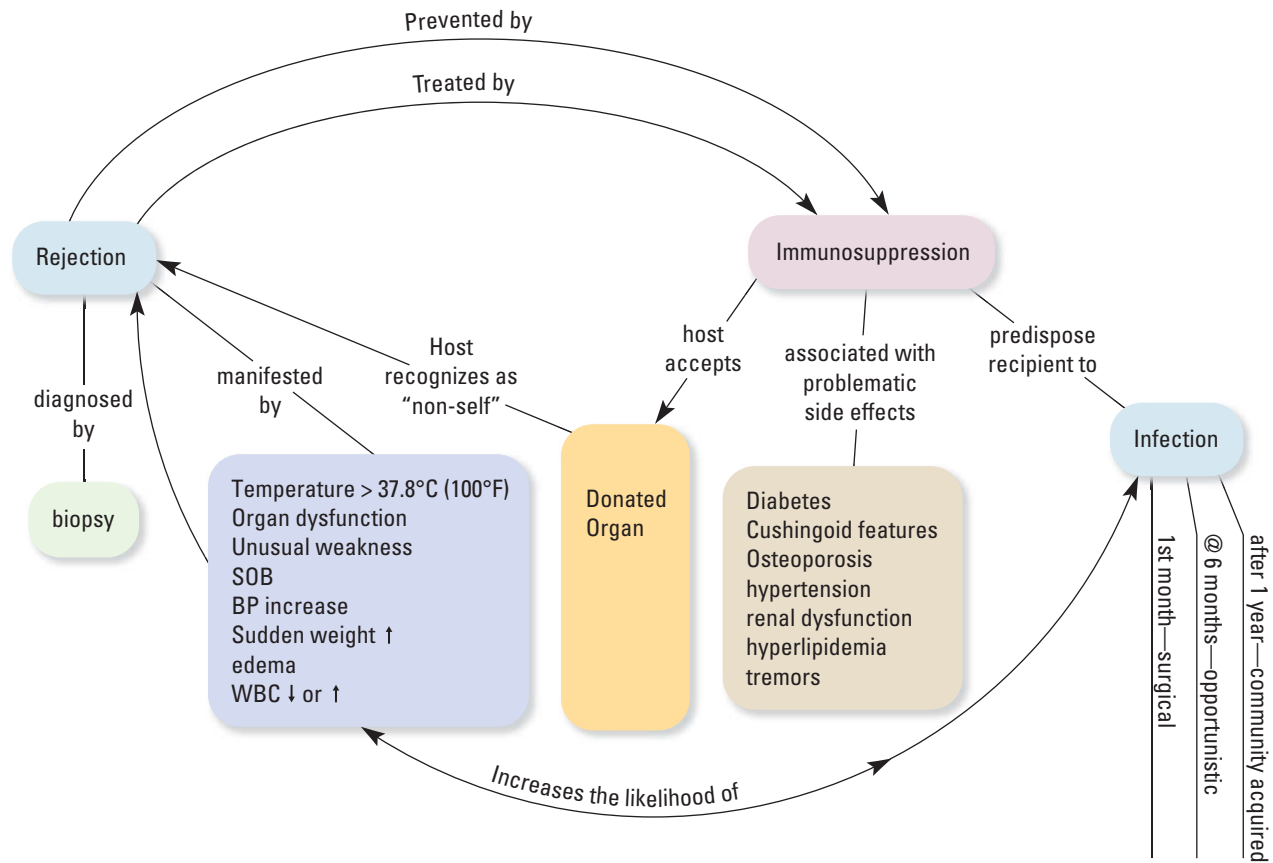
- Hemodynamic stability (which is usually not as difficult as for the brain-dead donor because herniation and the disruption in autonomic response have not occurred)
- Good tissue oxygenation
- Normal electrolyte balance
- Coagulation profiles within normal limits

Collaborative Procedure

When the recovery team is in place, the patient is extubated. The team waits until the patient can be declared dead. The definition is dependent on the specific institution but is often 5 minutes of asystole on the cardiac monitor, no arterial waveform indicative of a BP or pulse, and no respiratory effort. When death occurs, the patient is pronounced dead by a physician who is not a member of the transplant team. If the patient is pronounced dead within 60 minutes of extubation, then the team begins the organ recovery process. However, if the patient continues to breathe and has a heart rhythm after 60 minutes, the patient is transported back to the critical care unit for continuing care and family support.

NURSING ACTIONS

Families of patients who become nonheartbeating donors should receive support during the decision to withdraw life-sustaining technology from the patient and throughout the process of organ donation. Depending on the family, the support may be provided by the nurse, physician, social worker, or hospital chaplain. Most sources recommend that families be given the opportunity to say their farewells to the patient at a time and place of their choosing. This may take place in the ICU prior to the patient's extubation and pronouncement of death in the OR. However, some families request to stay with the patient while he is extubated and until he is pronounced dead. Therefore, arrangements may be made for the extubation and pronouncement to occur in a room close to the OR or for the family to be



Visual Map 16-2 Transplant Recipient

present in the OR until the patient is pronounced dead. In either case, it is recommended that a limited number of people be present while waiting for the pronouncement of death. Those present usually include the OPO representative responsible for caring for the donor, the physician responsible for pronouncing the patient dead, and the ICU nurse who has been providing care to the patient and who will be responsible for providing emotional support to the family. Additional emotional support will be required if the patient is one of those who do not have a circulatory arrest during the 60-minute time frame consistent with successful donation. These patients and their families will need to be provided with appropriate palliative care.

Organ Recipients

Currently, there are over 120,000 people in the United States waiting for organ transplants (UNOS, 2015). Because there are so many people waiting for only a few organs, potential organ recipients are evaluated carefully to ensure they will benefit from the transplant. Often, the risk of transmission of a malignancy or disease will need to be

weighed against the risk of the potential recipient not to receive the organ. The following is a list of contraindications to most organ transplants. Some are relative and might be waived depending on the patient's circumstances.

- Age greater than 70 years
- Untreated malignancies or cancer within the last 5 years
- Active infectious process, which has not responded to prescribed therapy
- Confirmed HIV positive status
- Active hepatitis
- Active substance abuse
- Active tuberculosis
- Severe COPD when the risk of damage to the patient from anesthesia is high
- Diffuse atherosclerotic or coronary artery disease that is not repairable, ejection fraction less than 20% for patients not receiving a heart transplant
- Psychosocial or behavioral abnormalities, which would prevent adequate post-transplant treatment
- Morbid obesity (body mass index [BMI] greater than 35)

Bariatric Considerations

In the past, obesity was considered a contraindication for transplant. However, with the rising rates of overweight and obese patients requiring transplant, there has been a reevaluation of the practice. The outcomes of transplant for overweight and obese patients appear to be dependent on the degree of obesity and the specific organ being transplanted.

The World Health Organization defines body mass index (BMI) from 25 to 29.9 as overweight, a BMI of 30 to 34.9 as class 1 obesity, BMI 35 to 39.9 as class 2, and BMI of >40 class 3 obesity. Obesity is associated with increased risk of peri-operative complications and decreased long-term survival after transplantation (Martin, DiMartini, Feng, Brown, & Fallon, 2014). A BMI greater than 30 may decrease short-term survival

following lung transplantation. However, BMI does not appear to have an adverse effect on short- or long-term outcomes following liver transplantation until it exceeds 35 or 40 kg/m². Class 3 obesity is considered a relative contraindication to liver transplantation. (Martin et al., 2014). However, patients with a BMI greater than 35 have been shown to have longer OR times, longer hospital stays, and more complications following renal transplantation.

The decision for transplantation should be based on the transplant center's complication rate, the available donor pool, and the type of organ to be transplanted. Whenever possible, the obese patient should be in a weight-loss program supervised by a registered dietitian prior to transplantation.

Eligibility Criteria and Evaluation for Specific Organ Transplants

In addition to the general requirements for any organ transplantation, there are specific requirements that recipients must meet depending on the organ(s) to be transplanted.

Kidney Transplantation

There are few additional contraindications to renal transplantation, so many adults with end-stage renal disease (ESRD) are potential transplant candidates. However, some people with ESRD prefer to continue dialysis and choose not to become candidates for renal transplantation. If the patient chooses to be evaluated for transplant, he should know that candidacy requirements as well as the evaluation process for kidney transplant vary

according to the transplant center. Patients interested in kidney transplant should be referred to a transplant center once the estimated glomerular filtration rate (GFR) is less than 30 ml/min and before renal function has irreversibly deteriorated, ideally before the need for maintenance dialysis (Rossi & Klein, 2015). Given that it can be hard to determine the rate of kidney function deterioration, early referral allows for enough time for a thorough evaluation.

Reflect On

The process of organ transplantation and management of treatment, medications, and complications is complex and ongoing throughout the life of a transplant recipient. Given rising healthcare costs, should under- or uninsured patients be candidates for transplantation?

Gerontological Considerations

Eligible donors should have been in good health previously and are usually less than 70 years of age. However, age is a relative contraindication and people older than 80 have been organ donors.

Individuals considered for living donation are usually between 18 and 60 years of age.

Age greater than 70 is a contraindication to some organ transplants. However, it is relative and might be waived depending on the patient's circumstances.

Medicare covers the cost of a renal transplant and expenses during the first 3 years. However, depending on the patient's age and circumstances, after 3 years the patient may no longer qualify for Medicare and must rely on another source of funding to cover the cost of medications and other transplant-related expenses.

The Candidate for Renal Transplantation

Once a person has been accepted as a candidate for renal transplantation, he is entered into the computer system maintained by UNOS. There are complex guidelines for allocation of the available kidneys to candidates on the waiting list with two overarching principles:

First, blood type O and B kidneys must be transplanted only into blood type O or B recipients except in the case of zero antigen mismatched candidates who have blood types other than O or B.

Second, it is mandatory that zero antigen-mismatched kidneys be shared. A zero antigen mismatch is defined as occurring when a candidate on the waiting list has an ABO blood type that is compatible with that of the donor and the candidate and donor both have all six of the same HLA-A, B, and DR antigens. When this occurs, the kidney must be offered to that individual.

For other candidates there is a point system for kidney allocation. When information about a donor is entered into the computerized UNOS Match System, all candidates who have an ABO blood type that is compatible with the donor and who are listed as active on the waiting list are assigned points and priority based on:

- **Time of waiting:** The time of waiting begins when the candidate either (a) has a creatinine clearance less than or equal to 20 mL/min or (b) begins chronic maintenance dialysis.
- **Quality of antigen mismatch:** Points are assigned to a candidate based on the number of mismatches between the candidate's antigens and the donor's antigens at the HLA-DR locus.
- **Donation status:** A candidate is given additional points if she has donated an organ or an organ segment to a person within the United States.
- **Proximity:** Kidneys are allocated first locally, then regionally, and, finally, nationally.
- No points are assigned for severity of illness for allocation of kidneys on the regional or national level although physicians on the local level may take that into consideration.

Liver Transplants

In general, a donor is matched to a potential liver recipient on the basis of several factors: ABO blood type, body size, degree of medical urgency, and Model for End-Stage Liver Disease (MELD) score. The priority of any patient on the UNOS waiting list for a donor liver depends on the following factors:

- Region (donor organs are first offered locally).
- ABO type (priority is for an identical ABO blood type but compatibility is essential).

- Body size (the acceptable body range is determined by the transplant surgeon).
- Degree of medical urgency as determined by the MELD score (highest priority given to sickest patients). The MELD is a numerical scale used for adult liver transplant candidates. The individual score determines how urgently a patient needs a liver transplant within the next 3 months. The number is calculated using the most recent laboratory tests for the following lab values:
 - Bilirubin
 - INR
 - Creatinine (If the patient has been dialyzed recently, the creatinine is entered as 4 mg/dL.)

After the score is calculated, potential liver recipients are grouped at four levels on the scale (less than 10, 11 to 18, 19 to 24, and greater than or equal to 25). Patients with the highest scores are the sickest and have the most urgent need for a liver transplant. Waiting times at each MELD level are tracked. If all other factors between potential recipients are equal, waiting times at levels of illness are the determining factor in which the person receives the liver.

Heart Transplant

Hearts are allocated to candidates locally based on medical status, then regionally. Every candidate older than 18 years of age awaiting heart transplantation is assigned a status code, which corresponds to how medically urgent it is that the candidate receive a heart transplant. The most severely ill candidates have at least one mechanical circulatory support device in place such as a left and/or right ventricular heart assist device, an intra-aortic balloon pump, or a total artificial heart. Candidates who are less severely ill may have implanted ventricular assist devices or continuous infusions of inotropes. In nonhospitalized candidates, a reproducible VO_2 max (the amount of oxygen required by the body as a patient exercises on a treadmill) of less than 14 mL/kg/min is an objective indication that the patient may require a transplant. ABO compatibility and body size are also factors that determine whether a candidate will receive a heart transplant from a specific donor.

In addition to the exclusion criteria mentioned previously, a heart transplant candidate might be excluded from the transplant list for:

- age more than 65 years.
- severe irreversible pulmonary hypertension.
- irreversible kidney or liver dysfunction not explained by underlying heart failure.
- symptomatic peripheral, renal, or cerebrovascular artery disease.

Table 16-1 Median Waiting Time in Days for a Transplanted Organ by Organ Transplanted and Blood Group of Recipient

Year	Organ	Blood Group O	Blood Group A	Blood Group B	Blood Group AB
2011–2014	Heart	535	192	178	70
2011–2014	Liver	1638	1481	385	127
2011–2014	Kidney	Not calculated as less than 50% of 106,393 on waiting list have not been transplanted.			

Source: Based on Organ Procurement and Transplantation Network (OPTN) data, <https://optn.transplant.hrsa.gov/>, as of January 27, 2017.

- insulin-dependent diabetes mellitus with evidence of damage to other organs.
- smoking.

Likelihood of Receiving an Organ from a Deceased Donor

The number of deceased donors has remained relatively flat since 1994, whereas the number of people awaiting transplants has continued to rise. The average waiting time for various organs arranged by blood group and year as listed by UNOS (2017) is displayed in Table 16-1. Often, a potential transplant patient may ask about their chances of obtaining a transplant once they are placed on the waiting list. The answer is variable because it is based on the organ being transplanted, medical necessity, blood and tissue type, size match, time on the waiting list, and geography. Geography plays a large part in determining where an organ is allocated. The United States is divided into 11 geographic areas that each have an OPO, there are then 58 total local donor service areas. Generally, the organ goes to a recipient in the same OPO region as the donor. If there is no suitable candidate in that region, it would then be offered to a patient in the next nearby region (Gift of Life Donor Program, 2015).

In addition, the wait for a donated kidney for minorities may be longer than for Caucasians. This probably occurs for two reasons. First, African Americans and other minorities are three times more likely to suffer from ESRD than are Caucasians. Second, transplant success rates increase when organs are matched between members of the same ethnic and racial group because they are more likely to share a common HLA phenotype. When an ethnic or racial group lacks information, has specific religious convictions, or mistrusts the medical community, they are less likely to agree to organ donation. Consequently, a lack of organs donated by minorities can contribute to longer waiting periods for transplantation.

Immunosuppression and Rejection

When delivering care to the transplant patient, the nurse should be aware of certain unique aspects of the patient's care. Those features of care include rejection and the use of

immunosuppressive drugs, the increased risk for infection, specific concerns related to the organ transplanted, and psychosocial issues related to accepting an organ from another human being.

Overview of Rejection

When a patient receives an organ from a donor who is genetically different than him, his immune system recognizes the organ as foreign and mounts an immune response to eliminate or neutralize the grafted organ. This process is known as rejection. If a solid organ transplant recipient does not receive immunosuppressant medications, then the graft will be rejected and the organ transplant will fail.

Rejection is more likely to occur when the recipient has been sensitized. Sensitization events such as previous transplants, blood transfusions, and pregnancies create antibodies that also make it harder for people to pass the crossmatch test.

Types of Rejection

There are four types of rejection that result from different immune responses and occur at varying times post-transplant. The first type of rejection, hyperacute rejection, is rare. It occurs only when the recipient has preformed antibodies to donor antigens. Thus, it develops almost immediately after the graft is transplanted into the recipient. Severe thrombosis and graft necrosis result in visible damage to the organ, the organ fails, and it must be removed.

Accelerated rejection, the second type of rejection, occurs within the first week after transplantation when the recipient has been sensitized to some of the donor antigens. Although it is usually treated aggressively with steroids and immunosuppressants, it is associated with a poor prognosis for graft survival.

Acute rejection develops in 15% to 60% of transplant recipients during the first year post-transplant. Acute rejection is a cell-mediated immune response that results in T lymphocytes infiltrating the donated organ and damaging it by secreting lysosomal enzymes and lymphokines. Antirejection doses of immunosuppressants are provided; these usually reverse the process of acute rejection, and the prognosis for graft survival is good. ♦ Figure 16-2 displays the process of acute rejection with the sites of action for immunosuppressive agents.

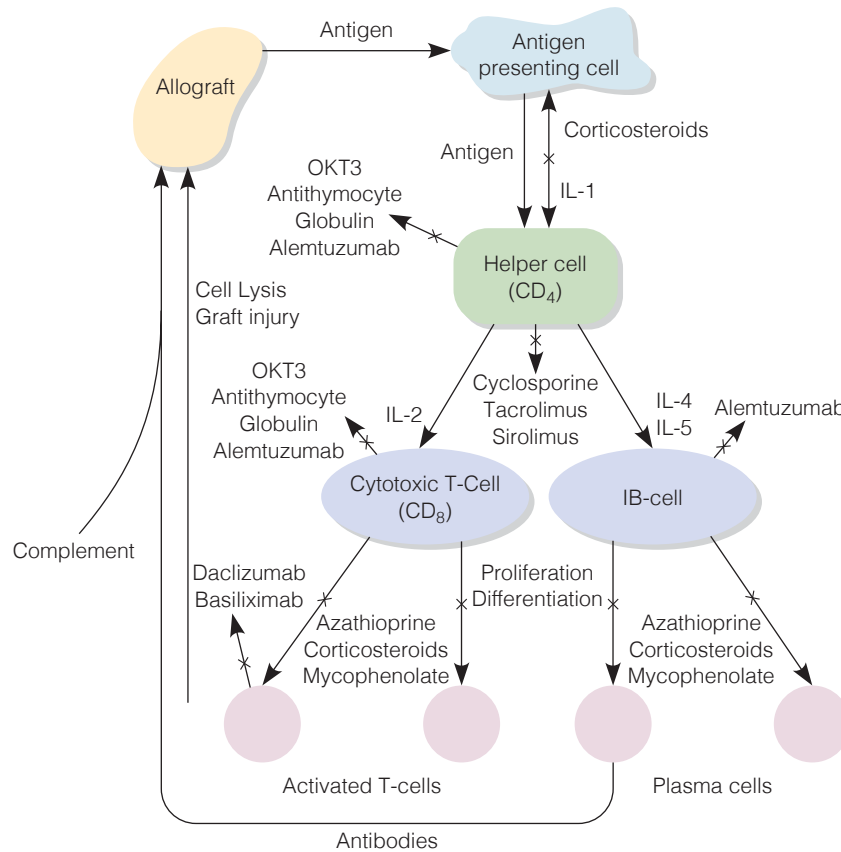


Figure 16-2 Progression of acute rejection with actions of immunosuppressants identified.

Chronic rejection occurs months to years after organ transplantation. The process is not completely understood but probably involves a combination of humoral and cell-mediated immune responses. There is usually progressive deterioration in organ function that does not respond well to immunosuppression.

Patients who receive solid organ transplants will need to be concerned about preventing and detecting rejection for as long as they have their transplanted organ.

Donor-Recipient Compatibility Testing

Hyperacute and accelerated rejection are now rare due to requirements for ABO compatibility testing and crossmatching before all transplants. UNOS calls for retesting the ABO compatibility of the donor and recipient blood types a second time prior to transplant to prevent the tragedy of a mismatched organ. Crossmatching is also performed prior to the transplant to prevent hyperacute rejection. In a crossmatch, the blood of the donor is mixed with that of the recipient. If the blood of the recipient attacks the cells in the blood of the donor, it means that the recipient has antibodies to the tissue type of the donor. A positive crossmatch means that the transplant is contraindicated. If there is no reaction between the blood of the

donor and the blood of the recipient, called a negative crossmatch, then the transplant may proceed.

Immunosuppression

There are three types of immunosuppressive therapy. The first two types, induction and maintenance immunosuppression, are used to prevent rejection of the transplanted organ. The third is anti-rejection treatment, which calls for higher and more potent doses of immunosuppressants to resolve an acute episode of rejection. The goal induction and maintenance immunosuppression is to prevent acute and chronic graft rejection, and to prevent and minimize risk of infection or risk of drug toxicity. The choice of the specific drug regimen for any type of immunosuppression depends on the transplant center but also on the organ transplanted. Medications are described by classification in the following sections. All organ transplant patients receiving immunosuppressants should be assessed for signs of rejection and monitored for the development of infection.

Induction

Induction immunosuppression refers to the temporary use of high-dose immunosuppressants in the perioperative period when the likelihood of rejection is highest. Its goal is to not only prevent the risk of acute rejection but to also

delay the use of maintenance drugs that can cause nephrotoxicity in the immediate post-operative phase when the patients kidneys are recovering perfusion injury as a result of surgery (Moten & Doligalski, 2013). It is often used for patients who have had kidney, intestine, and pancreas transplants, occasionally for patients with heart and/or lung transplants, and less often for patients with liver transplants. Induction therapy can be started pre-or intra-operatively and most commonly involves a brief course of antibody therapy such as rabbit ATG (thymoglobulin), calcineurin inhibitors (CNIs), antimetabolites or antiproliferative medications, and glucocorticoids.

Polyclonal and monoclonal antibodies are primarily used in induction therapy. **Polyclonal antibodies** are antibodies produced by multiple immune cells to target a specific antigen. Most commonly, a polyclonal agent called *rabbit antithymocyte globulin (ATG)* is used. Antithymocyte globulin antibodies are created by injecting animals (rabbit or horse) with human lymphoid cells, then they are harvested and refined to create the ATG agent (Pellegrino, 2015). **Monoclonal antibodies** are produced by a single immune cell and target a specific antigen. They include muromonab-CD3 (OKT3), daclizumab (Zenapex), and basiliximab (Simulect). These may be used for induction or antirejection immunosuppression but are less likely to be used than rabbit ATG. OKT3 can induce a cytokine response syndrome (fever, chills, wheeze, dyspnea, headache, rigor, chills, tremor, joint pain, rash) in most patients. However, 5% to 15% of patients develop a severe response with symptoms that may include pulmonary edema, hypotension or hypertension, decreased urinary output, respiratory failure, and cardiac arrest. Other common adverse effects of monoclonal antibodies are GI distress and hypertension.

According to the 2009 Kidney Disease: Improving Global Outcomes (KDIGO) clinical practice guideline on the management and treatment of kidney transplant recipients, antibody induction is utilized for the majority of kidney transplant patients. More practice and guideline information can be found on the KDIGO website. Rabbit ATG (Thymoglobulin) is typically the immunosuppressant administered. However, monoclonal antibodies such as daclizumab and basiliximab (Simulect) are utilized in some circumstances.

Maintenance

Maintenance therapy refers to the specific immunosuppressant medications that the organ recipient will continue to take either for as long as he lives or until the transplanted organ is removed. Most patients receive two to three different immunosuppressant medications that inhibit T-cells at various points of the activation pathway and which complement each other in an attempt to improve the effectiveness of the therapy. Current therapy

regimes often include corticosteroids, calcineurin inhibitors, and antimetabolite or antiproliferative agents, like azathioprine or mycophenolate mofetil (Moten & Doligalski, 2013; Chandraker, 2015).

Antirejection

Antirejection treatment is the use of higher doses of immunosuppressants and/or more potent IV immunosuppressants to resolve an acute episode of rejection. Treatment usually involves high-dose corticosteroid and antibody therapies.

Identification and Management of Rejection

With the advent of induction protocols and the use of several new immunosuppressant agents, fewer patients are developing acute rejection. Still, it is essential the nurse and patient know how to identify the symptoms of acute rejection and the patient know when to contact the transplant center.

- To avoid rejection, the patient should contact the transplant center if the patient cannot take the immunosuppressants or has missed doses.
- Because many medications interact with immunosuppressants, the patient should also contact the center when any medications are changed.
- Most transplant centers request that they be notified if one of their patients has a temperature above 37.8°C (100°F) and be called immediately if the patient's temperature exceeds 38.4°C (101.5°F).
- The recipient should notify the center promptly of the development of persistent, unusual weakness or fatigue; shortness of breath; sudden weight gain accompanied by swelling in the hands and feet; or a feeling of "not feeling right" accompanied by aches and pains, as well as any of the indications of rejection of a specific organ that are listed next.

Identification and Management of Rejection in Kidney Transplant Patients

In 2014, the United States Renal Data System (USRDS) reported a 7% to 9% incidence of acute rejection in the first year following kidney transplantation (USRDS, 2015). Indications that a person might be experiencing an acute rejection of a transplanted kidney include:

- Increase in blood urea nitrogen (BUN) and creatinine levels with a decrease in urine output
- Weight gain, edema, and an increase in BP
- Fever, chills, and elevated white blood cells (WBCs)
- Graft swelling and tenderness

Assessment of the patient should include:

- Accurate intake, output, and daily weights
- BP
- Serum levels of electrolytes, BUN, and creatinine
- Complete blood count (CBC)
- Evaluation for indications of a urinary tract infection
- Ultrasound of the bladder if postvoiding residuals are suspected
- Renal transplant ultrasound

Identification and Management of Rejection in Heart Transplant Patients

The percentage of patients requiring treatment for acute rejection after heart transplantation has remained consistent at approximately 40% over the past decade. Rejection is diagnosed via endomyocardial biopsies that are performed at regular intervals post-transplant. Immediately post-op, the biopsies are performed at least weekly with the frequency decreasing gradually to yearly. The degree of rejection is graded depending on the amount of intercellular infiltrate and the presence of myocyte damage or necrosis. Biopsies are essential because the patient may be asymptomatic until late in the process when the ejection fraction drops and the patient develops heart failure and dysrhythmias. Acute rejection in heart transplant patients is primarily treated with corticosteroids with less than 20% of patients receiving antibodies.

Chronic rejection is the major reason for death of those heart transplant recipients who survive beyond the first

year. It occurs in at least 60% of heart recipients 5 years after transplant and leads to progressive myocardial fibrosis and heart dysfunction.

Identification and Treatment of Rejection in Liver Transplant Patients

Acute rejection in liver transplant patients continues to steadily decline; rejection rates are approximately 15% to 25% (Cotler, 2015). Acute rejection is primarily treated with a short course of corticosteroids and is successful in 65% to 80% of transplant recipients; however, some patients, approximately less than 20% that are thought to be steroid resistant, require antibody therapy (Luu, 2014).

Indications that a patient may be rejecting a liver and the transplant center should be notified include

- Malaise, fatigue, and fever
- Abdominal discomfort with a swollen, tender graft
- Cessation of bile flow, change in color of bile from golden to colorless
- Dark- or tea-colored urine and clay-colored stools
- Jaundice
- Elevated bilirubin, liver enzymes, and international ratio (INR)

Assessment of the patient should include results of liver function tests, creatinine, urine output, prothrombin time (PT), INR, and bile drainage. A liver biopsy is the optimal test to distinguish between rejection of a transplanted liver, infection, or recurrent disease.

Commonly Used Medications

Corticosteroids, Calcineurin Inhibitors, Antimetabolites

Corticosteroids

Steroids are currently used in both maintenance and anti-rejection immunosuppressant regimens. Methylprednisolone/prednisone (Deltasone, Orasone) interferes with T-cell differentiation and macrophage function, impairing antigen recognition and cytotoxic response. It may be used for maintenance and antirejection therapy so the dose varies depending on the use. Oral prednisone doses may range from 2.5 to 100 mg/day in a single or divided dose, whereas IV methylprednisolone doses may vary from 250 to 1000 mg/day.

Common Side Effects:

- The development of diabetes
- Cushingoid appearance
- Mood swings
- Sodium and water retention

Longer-term side effects include:

- Weight gain
- Dyslipidemia
- Osteoporosis
- Aseptic necrosis of the hip
- Muscular atrophy
- Gastric ulceration
- Cataracts

Nursing Responsibilities:

- Administering the oral medication with food
- Monitoring baseline weight and BP
- Suggesting the patient have regular eye examinations
- Observing for signs of infection
- Patient education on the importance of weight-bearing exercises and taking calcium supplementation because

steroids have been associated with bone loss, which develops rapidly beginning in the first 3 months post-transplant

Calcineurin Inhibitors

Most post-transplant patients receive a calcineurin inhibitor for maintenance immunosuppression. This class commonly includes tacrolimus (FK-506) and cyclosporine (Neoral, Sandimmune). Per Pellegrino (2015) over 50% of new transplant patients are placed on tacrolimus (FK-506) and of those approximately 80% are liver and pancreas, 65% kidney and less than 50% are heart and lung transplants. The majority of renal transplant patients receive tacrolimus (FK-506) because it is associated with fewer episodes of acute rejection kidney transplant than cyclosporine (Kaufman, 2015). However, cyclosporine is often still used for patients following cardiac transplant.

Tacrolimus (Prograf) exerts its immunosuppressant effect through inhibition of cytokine production by T-cells (calcineurin inhibition). It is heavily metabolized in the liver and has a narrow therapeutic window. There is wide variability between its rate of absorption and its bioavailability. Oral doses usually range between 0.075 and 0.15 mg/kg every 12 hours.

Adverse Effects: Adverse effects that occur more commonly with tacrolimus than cyclosporine include:

- Insulin-dependent diabetes mellitus.
- Diarrhea and vomiting.
- Neurological toxicity may develop and is seen as tremor, headache, numbness, and tingling.
- Nephrotoxicity is also a possibility.

Nursing Responsibilities:

- Oral therapy is preferred because of the risk of anaphylactic reactions with intravenous (IV) tacrolimus.
- Tacrolimus should be taken on an empty stomach, at least 1 hour before a meal or 2 hours after a meal, ideally in the morning, because food intake can significantly limit its absorption.
- The patient will need regular monitoring of BP, blood sugar, and renal function.
- Given that many patients may develop diabetes, they may need education about dietary modification, oral hypoglycemic medications, or insulin.

Cyclosporine (Neoral, Sandimmune) inhibits T-cell proliferation and differentiation without affecting other immune cells. The drug is absorbed erratically after oral administration and

is metabolized in the liver. Therefore, the dose varies from patient to patient and may range from 3 to 10 mg/kg/day in divided doses.

Common Side Effects:

- The development of nephrotoxicity that can be acute and dose-related. Prolonged use can cause kidney fibrosis.
- The majority of patients develop hypertension and hyperlipidemia.
- Neurological toxicity is not as common as with tacrolimus.
- Hirsutism is a troublesome cosmetic side effect that has caused some patients to discontinue the drug.

Nursing Responsibilities:

- It is important that patients have their drug levels monitored as scheduled because metabolism varies among individuals and cyclosporine has a narrow therapeutic window.
- Patients also need to have their BP, weight, and kidney function monitored at regular intervals.
- Nurses need to be aware and alert patients that there are many drug interactions that can increase cyclosporine levels, particularly calcium channel blockers, antifungal agents like fluconazole or ketoconazole, antibiotics like erythromycin, and grapefruit juice. They should let their transplant center know of any changes to their medications.
- If possible, nephrotoxic agents, such as contrast medium, should be avoided.

Antimetabolites

Mycophenolate mofetil (CellCept) has replaced azathioprine and is a widely used adjunct immunosuppressant in solid organ transplant.

Mycophenolate mofetil (CellCept) is an antiproliferative agent. It more selectively inhibits the B- and T-cell proliferation than the older agent azathioprine. Doses range between 500 and 1500 mg twice a day.

Common Side Effects: The drug is normally well tolerated. The most common side effects—nausea, vomiting, and diarrhea—usually respond to a reduction in dose. Neutropenia and anemia are also common. Patients may be more prone to opportunistic infections than with azathioprine.

Nursing Responsibilities: The medication should be administered on an empty stomach and should be swallowed whole, never crushed or chewed.

Infection

The factor most responsible for the predisposition to infection in recipients of transplanted organs is the use of immunosuppressants. They help prevent transplanted organ rejection but alter the body's normal protective responses, which can lead to the development of various infections that can range from mild to severe. Other major contributing factors to the patient's immediate post-transplant likelihood of infection are the preoperative condition of the

patient and nosocomial factors. Nosocomial factors include surgical and/or invasive procedures, the hospital and critical care environments, the presence of multiresistant hospital organisms, and administration of antibiotics. In addition, there are factors regarding emerging infections in the donor (i.e., drug resistant TB, West Nile virus, lymphocytic choriomeningitis virus) that likely have an impact of graft survival as well (Asim, 2014). The American Society of Transplantation helps to maintain and refine guidelines

Commonly Used Medications

Rabbit Antithymocyte Globulin

Introduction

Rabbit antithymocyte globulin (Thymoglobulin) depletes lymphocytes and may work by modulating T-cell activation. The dose is 1.5 mg/kg/day IV for 3 to 14 days depending on whether it is used for induction or as an antirejection dose.

Common Side Effects:

- Fever, chills, dyspnea, joint pain, headache
- Gastrointestinal (GI) disturbances
- Hypertension
- Thrombocytopenia and neutropenia

Nursing Responsibilities: Rabbit antithymocyte globulin (ATG) is administered by central line with a filter over 6 hours for the first dose and 4 hours for subsequent doses.

for screening, monitoring, and reporting infectious complications in organ transplant patients.

Prevention of Infection

Prevention of post-transplant infection begins with pre-transplant screening. As noted previously, the patient is screened for the presence of a variety of infections. Some, such as active tuberculosis (TB), preclude the transplant. Other more common infections can be treated or prevented with prophylactic regimens. Screening for CMV is essential because it is latent in as many as 80% of donors and recipients and remains dormant in an infected host. CMV can cause infection at any point post-transplant, but it is estimated that more than 50% of transplant recipients will come down with infection within the first 3 months after transplantation (Asim, 2014). Although CMV only causes a flu-like illness in immune-competent individuals, it may develop into a serious illness that can result in invasive disease and organ dysfunction in transplant recipients. Therefore, many centers treat all their transplant recipients prophylactically with either acyclovir or ganciclovir. Nearly all centers treat a recipient who has never been exposed to CMV (is seronegative) and receives an organ from a donor who has been exposed and has latent CMV (is seropositive) with ganciclovir or valganciclovir.

Other preoperative strategies to prevent post-op infections may include the administration of vaccinations while the candidate is awaiting transplant. The potential recipient is usually seen by a dentist to assess oral hygiene, treat dental cavities, and extract any diseased teeth. Finally, the candidate is assessed for any other potential sources of infection, and they are treated or eliminated.

Identification and Management of Infection

How frequently a recipient develops infection post-operatively depends on the type of transplant he has received and the interval post-transplant. There are three time frames during which different infections occur in transplant recipients: first month, second through sixth month, and beyond the sixth month. The three time frames provide a guide to infection prevention and management.

Infections During the First Month Post-Operatively. Infections that occur in the first month after transplantation are usually nosocomial infections similar to infections occurring in any surgical patient. According to Asim (2014), the patient is at greatest risk of healthcare-associated infections within the first month following transplantation. These infections are often procedure or device related. The most common sites for infection in the first month are:

- Surgical incision
- Lungs, especially if the person remains intubated or is mechanically ventilated, the patient will be at risk for ventilator associated pneumonia or aspiration
- Urinary tract via a catheter
- Biliary tree due to catheters
- Blood, due to invasive IV or arterial catheters

The high level of immunosuppression during this first month can exacerbate these infections in transplant recipients.

Post-operatively, a clean, but not sterile, environment and frequent hand washing are the foundations of infection prevention for the organ transplant recipient. Nurses should also emphasize maintaining the integrity of skin and mucous membranes, limiting the number and duration of invasive devices, and providing optimal nutritional support. Routine post-operative measures to prevent infections such as the use of incentive spirometry, deep breathing, and early ambulation are also required. In order to limit the likelihood of infection, some centers require all people entering the recipient's room to wear masks, whereas others merely exclude visitors with respiratory infections. Many centers preclude patients from having plants or flowers in their rooms because the standing water may be a source of infection.

Family and patient teaching at the time of patient discharge from the hospital should focus on self-care practices that prevent infection. Some strategies that may help prevent infection are to:

- Wash their hands before putting anything in or near their mouth and after toileting.
- Wear a mask when in a crowd for the first few months post-transplant.
- Remove plants and flowers in vases from their homes.

- Wash, peel, and/or cook all fruits and vegetables.
- Avoid exposure to live vaccines.
- Avoid environments contaminated by mold, fungus, or water damage.
- Obtain prophylactic antibiotics prior to dental procedures.

Additionally, recipients and their families are taught to report any indications of infection to the transplant center immediately. Healthcare providers should be suspicious anytime a recipient reports unusual symptoms because the normal signs of infection have been suppressed by the patient's required medications. Although an increase in WBC count is usually associated with infection, leukopenia may develop in response to infection of the transplant recipient. Fever and chills may not develop so the nurse may need to look at subtle changes in lab values and patient symptoms to determine the source of the infection.

Infections Occurring from the Second to Sixth Month Post-Operatively. Opportunistic infections are the most common cause of infections after the first month until the sixth month post-transplant. Any episode of rejection that results in an increased dose of immunosuppressant medications can set the stage for the recipient to develop an opportunistic infection. The development of an opportunistic infection is due to the presence of an organism and suppression of the patient's innate immunity, but also may be compounded by the presence of an immunomodulating virus. Immunomodulating viruses make the patient more susceptible to opportunistic infections. Examples of immunomodulating viruses are hepatitis B and C, Epstein-Barr virus, and CMV.

CMV is the most common and most significant viral infection in solid organ transplant recipients. The seronegative recipient who receives an organ from a seropositive donor is most likely to develop a primary CMV infection after transplantation. However, disease may also occur with reactivation of infection in the patient who was CMV seropositive before transplantation. One of the earliest signs of active CMV infection is the nonspecific lab finding of leukopenia. However, CMV may present in a variety of ways:

- In most cases, CMV infection results in mild to moderately severe disease manifesting as episodic fevers accompanied by joint pain, fatigue, anorexia, abdominal pain, and diarrhea.
- CMV infection can also result in tissue-invasive disease causing pneumonia, gastritis, colitis, hepatitis, retinitis, and endocarditis.
- The development of CMV disease predisposes the patient to additional risk for opportunistic bacterial, viral, and fungal infections (Asim, 2014).

One common type of fungal infection in transplant recipients is candidiasis. Oral candidiasis, also known as thrush, can result in pronounced discomfort, preventing the patient from eating and leading to nutritional deficiencies. Therefore, frequent assessment of the patient's mouth and throat is essential. The nurse looks for white lesions scattered over the tongue and oropharyngeal mucous membranes and a red, sore throat and gums. Thrush can often be prevented by application of a topical antifungal agent.

Infections Occurring Later than 6 Months Post-Operatively. After the first 6 months, the majority of patients (approximately 80%) are at risk and most likely to develop community-acquired bacterial infections like staphylococcal pneumonia and pneumococcal pneumonia or viral infections such as influenza, respiratory syncytial virus or other acute infections like urinary tract infections. Patients with these infections generally have sufficient transplant functioning with minimal immunosuppression and are without chronic infection. Approximately 15% of the patients in the late post-transplant period develop persistent chronic infections that may require higher doses of immunosuppressive agents, and 10% of patients will develop more frequent episodes of acute rejection or have a combination of acute infection and the development of chronic rejection (Asim, 2014).

Collaborative Management of the Transplant Recipient

Organ transplants are complex surgical procedures. Patients need to have a multidisciplinary approach from all healthcare providers (nurses, advanced practice nurses, physicians, pharmacists, case coordinators, and social/financial support coordinators) in order to have successful transplant outcomes. Collaborative nursing care of the patient following a transplant varies depending on the transplanted organ and the specific surgical procedure.

Kidney Transplant

The majority of patients who receive a donated organ receive a kidney. The number of people awaiting kidney transplants has increased steadily because the quality of life is usually better for patients with ESRD who receive renal transplants than those who remain on dialysis.

Surgical Procedure

Since 1954, transplanted kidneys have been placed extraperitoneally, which is in the right iliac fossa as shown in

- ♦ Figure 16-3. Placing a transplanted kidney in the iliac

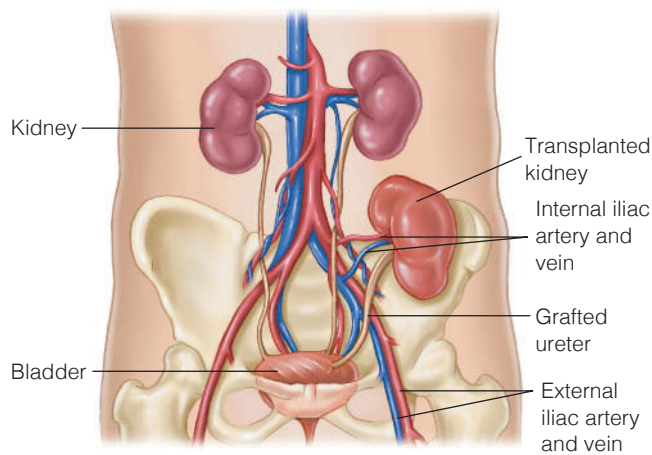


Figure 16-3 Placement of a transplanted kidney in the iliac fossa.

fossa is advantageous because of its close proximity to major blood vessels and the bladder. It also facilitates assessment of the transplanted kidney by palpation, auscultation, and biopsy. Finally, if removal of the graft is necessary, it can be performed with less risk to the patient. The patient's own kidneys usually remain in place following a transplant. The exceptions are for uncontrolled hypertension, tumors, recurrent infections, or stones of the kidney. In such cases, one or both of the native kidneys might be removed.

At the beginning of surgery, the donor's and recipient's renal arteries and veins are anastomosed to the iliac artery and vein. Once the kidney is perfused, the donor ureter is attached to the recipient's bladder. Different surgical techniques are available; one more common method is attaching the ureter to the bladder by-passing the donor ureter into a tunnel carved through the posterior bladder wall and sewn to the inner bladder mucosa. Often, to maintain the patency of the ureter, an indwelling ureteral stent is placed for the first few days to weeks after surgery. The stent will need to be removed within a few weeks following transplantation in an effort to reduce infection, hematuria, and stone formation (Bradley, 2015). The donor kidney usually begins to function immediately. The procedure typically takes about 3 hours after which the transplant recipient is transferred first to the post-anesthesia care unit (PACU) then to a transplant unit.

NURSING ACTIONS

The post-operative care of the renal transplant patient includes all of the essentials of good post-operative care for any patient. However, as previously noted, strategies to prevent post-op infections are even more essential than normal. In addition, the nurse must monitor the function of the transplanted kidney and detect early signs of any complications.

The amount of urine the transplanted kidney produces immediately after it is perfused varies greatly, depending on how well the kidney was perfused prior to transplant. The nurse monitors the patient's hourly urine output and evaluates his renal function tests. On occasion, delayed graft function (DGF) may occur. DGF is defined as oliguria that necessitates dialysis during the first 7 days after kidney transplantation, and the dialysis might be needed for days to weeks until full function of the kidney returns. Patients who receive kidneys from deceased donors are more likely to have delayed graft function.

According to Kaufman (2015), surgical complications after kidney transplant include:

- Urologic complications, which include urine leakage, reflux, and ureteral stenosis or obstruction
- Vascular complications, which include renal artery or vein thrombosis or bleeding due to anastomosis rupture
- Wound complications, which include infection or lymphocele (collection of lymph in the retroperitoneal space)

An obstruction from blood clots or kinking of the urinary catheter may develop early in the postoperative period. If the obstruction persists, the bladder could become overdistended, the implanted ureter might separate from the bladder, and a urine leak could develop. Therefore, the nurse maintains the urinary catheter free of clots and monitors the urine output carefully. Obstruction usually presents as a sudden decrease or cessation of urine output rather than a gradual decline. In addition, the patient may complain of cramping and clots, or fibrin may be visible in the catheter. After recognizing the obstruction and determining nothing is visibly kinked, the nurse might milk the urinary catheter in an attempt to dislodge any clots. If milking is not successful and the catheter requires flushing, it is done aseptically with small volumes (approximately 30 mL) and only per agency protocol.

A lymphocele is a wound complication that involves the collection of lymphatic fluid in either the donor or recipient lymph nodes surrounding the transplant. The fluid builds up and may obstruct the proximal ureter, resulting in decreased urine flow, or may obstruct venous drainage from the leg, resulting in edema or discomfort in that leg. The patient may complain of edema of the leg on the side of the graft, tenderness over the incision, and/or mild-to-moderate abdominal discomfort. In addition, fluid collection may also cause compression or obstruction of the transplant ureter, which may result in renal dysfunction (Kaufman, 2015). If the lymphocele has an opening to the skin surface, there is usually copious, clear drainage. An ultrasound is used to diagnose a lymphocele. Treatment, depending on the size of the lymphocele and the

patient's symptoms, might include watchful waiting, aspiration, or surgical creation of a window into the peritoneal cavity.

NURSING CARE

Pain management is essential for all postoperative patients, and there are no critical differences for pain management in the kidney transplant recipient. Pain relief must be adequate so the patient will engage in deep breathing and coughing and be ready to get out of bed and walk the day after surgery.

Imbalanced Nutrition

The nutritional goals of the transplant recipient depend on the specific patient's weight and current nutritional status. Patients with organ failure prior to transplantation are at risk for malnourishment. Protein-energy malnutrition is associated with higher morbidity and mortality rates. As much as 40% of patients with chronic renal failure who require dialysis suffer from malnutrition (Hwai-Ding, 2015). A variety of factors that can affect a patient's overall nutritional status include, blood loss/anemia, catabolism from chronic illness, protein and nutrient loss from dialysis, insufficient oral intake due to altered taste sensation or medication side effects or depression. Most patients begin taking sips of liquids within the first 24 hours after transplant surgery and begin eating as soon as bowel function resumes. For those patients with immediate return of renal function, there is no reason to restrict protein or potassium in the diet. The patient's caloric needs are increased due to the stress of surgery, high-dose steroids, and possible preexisting malnutrition; therefore, the patient is usually provided a diet with 30 to 45 kcal/kg of dry weight (Hwai-Ding, 2015). If the patient's oral intake is poor, then ongoing nutritional assessment and possible oral nutritional supplements may be required.

Recovery

Patients typically remain in the hospital for 3 to 5 days. Psychosocial issues are not uncommon in the first year post-transplant and will be discussed later in this chapter.

Most graft failures and patient deaths are the result of chronic rejection. The survival rates for kidney transplant grafts and patients from 2008 until 2015 are displayed in Table 16-2.

Heart Transplant

Heart transplant surgery is a time-consuming intricate procedure that is performed approximately 2000 to 2300 times each year in hospitals in the United States (OPTN, 2015).

Surgical Procedure

After completion of the pre-transplant procedure and in synchrony with arrival of the donor heart, the heart transplant candidate is connected to a heart-lung machine. In the orthotopic technique, the recipient's heart is removed from its connections to the great arteries, leaving in place the back parts of the right and left atria. The new heart is carefully fitted and sewn to the remaining portions of the recipient atria. Anastomoses of the aorta and pulmonary artery complete the procedure. Another approach, the bicaval technique, preserves the entire donor right atrium, attaching it to the recipient's superior and inferior venae cavae. This technique has led to less atrial distortion, fewer conduction abnormalities, and less tricuspid and mitral valve regurgitation. It has also resulted in fewer thromboembolic events, less need for permanent postoperative pacemaker placement, and an overall improvement in heart function in the early postoperative period. Thus, the bicaval technique is now commonly used. Both techniques are displayed in ♦ Figure 16-4.

After the new heart is completely sewn in place and begins to function, the patient is removed from the heart-lung machine. With the completion of the operative procedure, one or more chest or pleural tubes are placed. Epicardial pacing wires are brought out of the chest cavity through the skin's surface, in case there is a temporary need for pacing of the heart postoperatively. The sternum is brought together with stainless steel wires, and the fatty tissues and skin are closed with absorbable sutures. Patients are then transported to the ICU where they are stabilized, continuously monitored, and weaned from vasopressors and the mechanical ventilator.

Table 16-2 Survival Following Kidney Transplant 2008–2015 Identified as Percentage of Patients or Grafts Surviving and Noted by Type of Donor (Living or Deceased)

	Patient Survival			Graft Survival		
	1 year	3 years	5 years	1 year	3 years	5 years
Post-Transplant Duration						
Living Donor	98.7%	96%	91.9%	97.5%	92.5%	85.5%
Cadaveric Donor	96.2%	91.1%	82.9%	93.2%	85%	74.3%

Source: Based on Organ Procurement and Transplantation Network (OPTN) data, <https://optn.transplant.hrsa.gov/>, as of January 27, 2017.

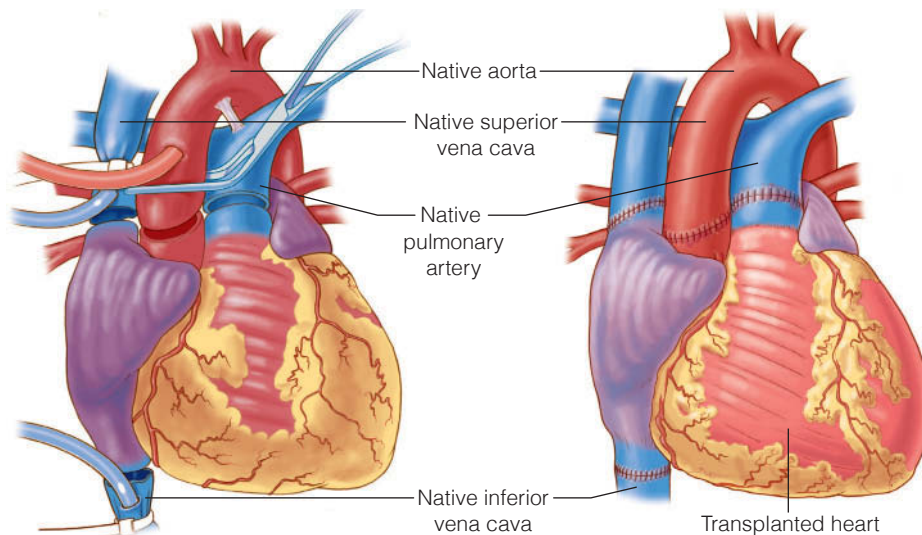


Figure 16-4 Orthotopic heart transplant bicaval technique.

Post-Operative Problems Related to the Surgical Procedure

Perioperative management of the heart transplant patient is in many ways similar to that of patients undergoing other major cardiovascular procedures. However, there are specific problems that occur only in patients undergoing heart transplantation.

The donor heart that is placed in the recipient's chest was denervated during removal from the donor. This means that the transplanted heart will not be able to respond to autonomic stimulation. Thus the patient's resting heart rate usually ranges between 90 and 110 beats/min. The heart can still respond to circulating catecholamines with an increase in heart rate and cardiac output. However, the heart's response to stress and exercise is delayed, as is the return to a resting heart rate after exercise. The lack of innervation also means that the heart will not respond to certain medications, such as atropine, that increase heart rate by blocking vagal stimulation of the heart. Instead, isoproterenol may be used during the postoperative period because it has a beta-adrenergic effect and can overcome sinus node dysfunction while inducing peripheral vasodilation.

Although a resting tachycardia is common post-cardiac transplant, sinus bradycardia may occur if ischemia or edema of the donor sinoatrial (SA) node developed during procurement, transfer, or transplant. The bradycardia is usually temporary, resolving within 1 to 2 weeks. However, the temporary pacing wires placed during surgery may be used to pace the patient in the short term and, if the bradycardia does not resolve, then placement of a permanent pacemaker may be necessary. Additionally, post-transplant patients may develop dysrhythmias, such as atrial fibrillation, commonly associated with all cardiac surgeries.

If the older orthotopic approach was utilized to attach the atria, the patient will have two existing SA nodes. Impulses

from each of the nodes (the recipient's as well as the donor's) will result in P waves on the electrocardiogram (ECG). The P waves from the recipient's native SA node are generally slower than the donor P waves and will respond to stimulation and increase or decrease in rate. However, the recipient's native P waves will be unrelated to the QRS complexes.

Post-Operative Complications

The most common causes of death following a heart transplant are infection or rejection of the transplanted heart that were described earlier. Immediate post-transplantation complications of concern include bleeding from suture lines, hyperacute rejection, graft failure, and infection. However, another major cause of death is cardiac allograft vasculopathy (CAV), which is an aggressive, diffuse form of arteriosclerosis, common in heart transplant recipients, occurring as early as 3 months following transplantation. CAV is one of the top three causes of transplant failure occurring after the first year of transplantation (Pham, 2015). Unlike the stenoses that develop in CAD that are localized to one section of an artery, CAV can affect the entire length of an artery in the transplanted heart. The exact pathogenesis is unknown but is characterized by diffuse narrowing of the arteries leading to eventual obliteration and cardiac allograft failure. The diffuse nature of the stenoses makes them less likely to be responsive to angioplasty and stenting.

In addition, since the transplanted heart lacks nerve innervation, the patient will not exhibit the typical symptoms of ischemic heart disease, like chest pain or pressure. Instead, CAV often presents as silent myocardial infarction, arrhythmia, or sudden death. Once CAV has developed, the only treatment is reimplantation (Botta, 2014). Therefore, early detection and prevention is the focus on reducing the risk of CAV. Heart transplant recipients are encouraged

to maintain a heart healthy lifestyle beginning as soon as possible postoperatively, and many transplant centers require routine coronary angiography to determine the existence or extent of CAV.

NURSING CARE

Heart transplant recipients may be debilitated and malnourished at the time of surgery. However, the focus of their nutritional support is adequate oral nutrition as soon as possible following extubation. They are started on a heart-healthy diet as soon as they are able to eat after surgery.

Recovery

Most heart transplant recipients are able to resume regular activities and their normal lifestyle despite cardiac denervation, but there are cautions for patients when resuming activities. Patients are instructed on their need to have a warm-up period prior to exercising to establish circulating catecholamines. They also need to cool down slowly after exercising to allow the catecholamine levels to taper off and their heart rate to return slowly to baseline. They should also be cautioned to change their position gradually to avoid orthostatic hypotension. Most patients are encouraged to enroll in a cardiac rehabilitation program within 2 to 3 months following their transplant. After the initial post-op period, patients have no limits on their prescribed amount of exercise, and heart transplant patients have been known to run marathons and climb mountains.

The prognosis following heart transplant depends on many factors, including age, general health, and status of the donor. Survival rates for the first 5 years following heart transplant are displayed in Table 16-3.

Liver Transplant

Liver transplant is a complex surgical procedure that requires a well-coordinated team for the best patient outcomes.

Surgical Procedure

The surgical procedures for liver transplantation differ depending on whether the recipient's liver is totally or

partially removed, and whether a whole or partial organ is transplanted. When the entire liver is removed, all ligaments that hold it in place as well as the common bile duct, hepatic artery, and portal vein are identified and clamped. Usually a portion of the inferior vena cava is removed along with the liver. When the hepatectomy is complete, the donor liver is implanted in the recipient's abdominal cavity. After blood flow to the new liver is restored by anastomoses of the inferior vena cava, portal vein, and hepatic artery, the bile duct is anastomosed, either to the recipient's own bile duct or to the small intestine. Usually a T tube or biliary stent is placed at that time. ♦ Figure 16-5 shows the surgical technique in liver transplantation.

The surgery usually takes between 5 and 6 hours but may be longer or shorter depending on the difficulty of the operation and the experience of the surgeon. Following surgery, the patient is usually transported to the ICU where they may remain for several days while continuing to recover from surgery.

Post-Operative Problems Related to the Surgical Procedure

One of the lesser understood problems in liver transplantation is primary nonfunction of the graft—a graft that simply fails to function. In most cases, it is clinically evident almost immediately in the OR because a functioning liver graft usually produces bile as soon as it is perfused, and pre-existing coagulopathies begin to improve almost immediately. The absence of bile production and the continuation of coagulation problems are early indications of graft failure.

Assessment of graft function in the post-operative period is one of the responsibilities of the nurse. The nurse carefully reviews laboratory tests and notes the appearance and amount of biliary drainage to assess graft function. The most sensitive laboratory indices of liver function are the coagulation indicators, PT (or INR), and partial thromboplastin time (PTT). A steady downward trend toward normalcy in coagulation studies (as well as in the following laboratory tests) indicates that the transplanted liver is beginning to function. Hyperglycemia in the immediate postoperative period is an indication that the liver is responding normally by storing glycogen and converting it to glucose in response to the metabolic effects of corticosteroids.

- Alanine aminotransferase (ALT)
- Aspartate aminotransferase (AST)
- Total bilirubin
- Lactic dehydrogenase
- Ammonia

The most common postoperative problems following liver transplant are acute rejection or early graft failure, vascular thrombosis, biliary stricture or leak, infection,

Table 16-3 Survival Following Heart Transplant 2008–2015 Identified as Percentage of Patients or Grafts Surviving.

Post-Transplant Duration	% of Patients Surviving	% Grafts Surviving
1 year	90.5%	90.0%
3 years	85.0%	84.0%
5 years	78.0%	76.0%

Source: Based on OPTN Data as of January 27, 2017.

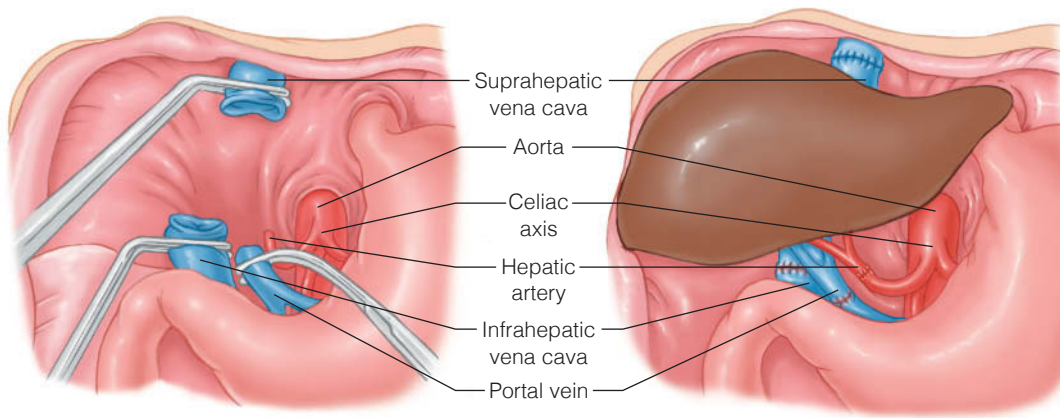


Figure 16-5 Surgical technique in liver transplant.

malignancy, or side effects to immunosuppressant medications (Luu, 2014). More specifically, issues involve:

- Disruption of flow through the hepatic artery, portal vein, or inferior vena cava. This can be due to vessel thrombosis or stenosis, pseudoaneurysm, or arteriovenous fistula.
- Biliary complications, including bile duct strictures, bile fluid accumulation (or bilomas), infected bilomas, or bile leaks
- Allograft nonfunction, as mentioned previously
- Acute rejection, which often occurs in 20% to 70% of patients after transplant (Luu, 2014). Elevated liver functions test, jaundice, fever, and abdominal tenderness are common findings in rejection.
- Infection, particularly bacterial infections, are the most common in the first month following transplant. Viruses and opportunistic infections occur more frequently in months 1 to 6 after transplant. Viral infection or reinfection with CMV occurs in 25% to 85% of liver transplants, and frequently between month 1 and 3 following transplant (Luu, 2014).
- Primary or recurrent hepatic malignancy. Some liver transplants are performed on patients who have or are at risk for hepatocellular carcinoma and these patients are at risk for developing recurrent malignancy in the allograft. In addition, transplant patients are at a much higher risk of developing cancer due to chronic immunosuppression. Patients are at risk for lymphomas, squamous cell carcinoma and post-transplant lymphoproliferative disorders (PTLDs). PTLDs are disorders characterized by an abnormal proliferation of lymphocytes that can occur in immunocompromised patients. Skin cancer is a common malignancy that develops post-transplant and is more aggressive in transplant recipients than in non-immunocompetent individuals. Recipients should be instructed to avoid the sun, to examine their own skin, and to have a yearly formal skin evaluation by

a healthcare provider with referral to a dermatologist for any suspicious lesion.

In addition, liver transplant recipients are also at high risk for the development of pulmonary complications. The reasons include:

- Atelectasis that develops from the patient spending the long surgical procedure in the supine position.
- Mechanical ventilation is necessary for a period postoperatively, predisposing the immunosuppressed patient to pulmonary infections.
- The patient may be immobile for extended periods.
- Pleural effusions are common on the right side following liver transplant.

To prevent these possible pulmonary complications, nurses need to mobilize the patient as much as possible and cough or suction the patient as needed. Aseptic technique when suctioning the patient's endotracheal (ET) tube is always appropriate as is the use of a ventilator bundle to prevent complications associated with mechanical ventilation. Frequent assessment of the lungs is necessary, especially for diminished sounds at the right base caused by a pleural effusion or atelectasis. If the effusion is large enough, it may require thoracentesis or placement of a chest tube for drainage.

NURSING CARE

Pain is common in postoperative patients, and nurses must be vigilant in treating it in their patients. Nurses should continually assess the patient for postoperative pain to ensure they not only have pain relief, but that it is adequate and to ensure the patient is able to cough and breathe deeply and increase activity as tolerated.

Imbalanced Nutrition

Malnutrition is a preoperative problem associated with ESLD and a postoperative complication for many liver

transplant patients. Patients with ESLD have a high incidence of protein and energy malnutrition with subsequent weight loss. Malabsorption of fat, and accelerated protein breakdown typically occur in patients with ESLD because of bile or pancreatic insufficiency and decreased fat absorption. This results in anorexia, early satiation, and diminished stomach capacity for a normal-size meal (Hwai-Ding, 2015). The goal of nutritional support post-operatively is to provide adequate protein and energy from carbohydrates or fats equivalent to or slightly less than energy expenditure. If at all possible, nutritional requirements are provided enterally because parenteral nutrition is associated with increased risk of infection in the liver transplant recipient. Management is consistent with any program of weight management: careful reduction in caloric intake accompanied by an increase in exercise.

Recovery

The first to fifth year survival rates for graft and patient post-liver transplant are displayed in Table 16-4.

Nursing Diagnoses

Transplant Patient

- Risk for infection related to inadequate secondary defenses (immunosuppression), malnutrition, indwelling catheters and drains or the presence of IVs and endotracheal tubes
- Risk for fluid volume excess related to renal insufficiency, steroid therapy, or decreased cardiac output
- Risk for decreased cardiac output related to cardiac muscle disease
- Interrupted family processes related to illness of family member and situational crisis of transplant
- Disturbed body image related to permanent changes in body due to immunosuppressant medications and presence of donated organ

Psychosocial and Psychological Issues in Transplant Recipients

During the first year following a transplant, many recipients experience some degree of psychosocial distress,

psychiatric symptoms, or psychiatric disorders. Psychiatric disorders can affect the patients overall coping and health following transplantation. Common nursing diagnoses during this period are risk for ineffective individual coping and ineffective management of the therapeutic regimen.

Risk for Ineffective Individual Coping

Psychosocial and psychological issues arise during the first year following organ transplantation. Common issues for patients and families relate to:

- Coping with the fear of rejection of the transplanted organ.
- Accepting the changes in physical appearance that result from steroid therapy. Especially bothersome to female recipients may be the “moon face” and excessive facial hair growth that may develop from medications.
- Mood disorders like depression and anxiety can be common following transplantation. Enduring the psychological highs and lows that sometimes occur as side effects of medications can be challenging and stressful for both patients and families.
- Managing the complex post-transplant regimen. Health-related side effects and the difficulty of continuing and maintaining the post-transplant treatment can affect the patient’s compliance with the treatment regime.
- Integrating the transplant experience. Transplant-related events, the transplant surgery, and the ICU stay can produce new-onset post-traumatic stress disorder (PTSD). The symptoms can range from having flashbacks about the transplant, to nightmares, hallucinations, anxiety, delirium, and the inability to recall elements of the transplant, to feelings of detachment from others and restricted affect.
- Managing with economic and financial issues such as the high cost of medications and healthcare.

High levels of emotional distress, as well as clinically significant depression and anxiety, are more common during the first year after a transplant than during later years for all patients having organ transplants. In addition, as the patient goes through the transplant process, families and caregivers may experience stress with the illness and hospitalization, as well distress within the dynamic of the patient-caregiver relationship.

Ineffective Management of the Therapeutic Regimen

Compliance after transplantation, the degree to which patients’ behaviors coincide with transplant team recommendations, is a major concern for transplant patients.

Table 16-4 Survival Following Liver Transplant 2008–2015 Identified as Percentage of Patients or Grafts Surviving.

Post-Transplant Duration	% of Patients Surviving	% Grafts Surviving
1 year	91.0%	88.5%
3 years	82.5%	80.0%
5 years	75.0%	72.0%

Source: Based on OPTN Data as of January 27, 2017.

Unfortunately, nonadherence with medications is common, “with studies repeatedly demonstrating that only about half of the people prescribed medications take at least 80% of the medications prescribed” (Stuber, 2010). In an effort to minimize noncompliance with the treatment regime, patients undergo psychiatric assessment and transplant education prior to possible transplantation but this does not ensure or predict a patient’s compliance with the complex post-transplant regimens.

Noncompliance appears to be relatively common during the first year after transplantation and tends to worsen as time from transplantation increases. Studies have demonstrated:

- As many as 20% of heart transplant recipients and 50% of kidney transplant recipients have been noncompliant with prescribed immunosuppressant medications during any given 12-month period.
- Approximately 10% to 40% of prior smokers resume smoking after transplantation despite counseling and pharmacotherapy. Smoking by both donors and recipients affects outcomes. In comparison to non-smokers, kidney function is worse in smokers one year following transplantation and have increases in cardiovascular events. Liver transplant recipients who smoke have higher rates of hepatic artery thrombosis, biliary complications and malignancy. Heart transplant recipients have higher risk of coronary artery disease (Corbett, Armstrong & Neuberger, 2012).
- 20% to 44% of liver transplant recipients resume alcohol consumption and excessive alcohol consumption after transplantation negatively affects long term survival (Faure et al., 2012).

Adolescents are at particular risk to be noncompliant following transplantation. This noncompliance is

partly due to developmental desire for independence and questioning of authority in combination with the complexity of the treatment regime and the negative cosmetic and physiological side effects of the medications (Stuber, 2010). Because compliance with the medication regimen is essential for graft survival, it is important for the nurse caring for the post-transplant recipient to develop an accurate understanding of how often the recipient is complying with therapy and what factors are contributing to the recipient’s difficulty in complying.

Support Groups

Many transplant patients and their families gain initial assistance and encouragement from support groups. Depending on the type of support group, patients and families may share their concerns and fears, receive educational information, share coping strategies, ask personal questions of people who have had similar problems, or receive assistance from social workers and transplant coordinators. Guest speakers may be invited to speak to the group about topics such as trends in transplantation or medications. Groups might share practical advice such as how to deal with insurance companies and where to purchase medications.

Talking with others with similar conditions can provide the new recipient with a feeling of security and comfort while assuring him that he and his family are “not alone.” It is encouraging for new recipients to see how others who have had transplants for years and their families are coping. As a patient’s transplant experience progresses, he may gain confidence and begin to provide assistance to others.

Transplant Summary

Organ donation has been demonstrated to be an effective therapy for end-stage cardiac, liver, and renal disease. Unfortunately, due to the shortage of organs, many potential recipients spend a long time on the transplant waiting list. During their time on the waiting list, their physiologic status often declines and some candidates die. The shortage of organs and resultant deaths of candidates waiting for organs has led healthcare providers and ethicists to consider ways to expand the pool of potential donors.

Caring for the potential donor and his family requires skill and compassion from the critical care nurse. When delivering care to the transplant recipient, the nurse recognizes there are some unique aspects to the patient’s care. These include rejection, which requires the use of immunosuppressive drugs; an increased risk for infection; specific concerns related to which organ was transplanted; and psychosocial issues related to receiving an organ from another human being.

Why/Why Not?

Mr. M. is a 62 year old male who has recently undergone a renal transplant. He is currently taking tacrolimus (FK-506). The nurse receives an order for 0.075 mg/kg IV q8h. Should the nurse administer this medication as ordered?

1. Vasopressin? Why? Why not?

2. Corticosteroids? Why? Why not?
3. Calcineurin Inhibitors? Why? Why not?
4. Antimetabolites? Why? Why not?
5. Antithymocyte Globulin? Why? Why not?

See answers to Why/Why Not? in the Answer Section.

Case Study

David Johnson, a 22-year-old senior in college, went to see his physician complaining of not feeling well. He stated that he had headaches that were increasing in frequency and that he was getting up three to four times each night to urinate. His BP was 138/96. David had been diagnosed with glomerulonephritis in high school but had avoided his healthcare provider since entering college.

His urinalysis revealed proteinuria, and his renal ultrasound with biopsy confirmed rapid deterioration in renal function. During the next few months, his decline in renal function continued. He became depressed and stated that he was not able to attend class because he was so tired.

Some of his lab studies were creatinine 6.1, BUN 63, potassium 5.7 (despite a low-potassium diet), Hct 26, and blood group AB positive.

David considered dialysis. However, both his sister and brother indicated that they would be willing to donate a kidney and the nephrologist concurred that if early donation was possible, David might do better. Initial testing was performed; David's sister was B positive and a 0 antigen mismatch. His brother was AB positive with no matching antigens.

1. Why is it a benefit for David to receive an organ from one of his siblings?
2. Which of David's siblings is the better choice for a donation? Why?
3. What other factors will need to be assessed about the potential donor?

David was scheduled to receive a transplant. Immediately prior to surgery, his BP was 167/92. His lab studies revealed that his creatinine was 8.1 and BUN 83. His potassium was 5.5 and Hct 26. His donated kidney began working immediately and by later that day his creatinine had decreased to 6. On day 3, he was feeling well, was up and walking, and his creatinine was 1.3, BP 132/84. He was discharged home, taking prednisone, CellCept, and tacrolimus.

4. For what symptoms should David be advised to contact his healthcare provider?
5. What precautions should David take to prevent infection?

About a month after discharge, David began to complain of pronounced hand tremors.

6. What is most likely to be causing the tremors?

Seven months after his transplant, David is feeling "lousy," he has gained 3 pounds in a few days, his BP is 150/90, and his creatinine is 3.4.

7. What is the most likely cause of his change in condition? What will be the probable management?

Five years following his kidney transplant, David has had three hip replacements.

8. What is the likely cause of his hip dysfunction?

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 16.1 Differentiate between the four types of rejection. Which of the four types is associated with the highest morbidity and mortality for transplant recipients?
- 16.2 What is opting in for consent for organ donation?
- 16.3 How is brain death diagnosed?
- 16.4 How can a nurse increase the likelihood that a family will donate a deceased person's organs?
- 16.5 Why is the calcineurin inhibitor, tacrolimus (FK506), preferred over cyclosporine?
- 16.6 Why are transplant recipients predisposed to develop infection?
- 16.7 Why is cytomegalovirus (CMV) a problematic infection in the transplant patient?
- 16.8 Why is parenteral nutrition a poor choice for provision of nutrition in the transplant recipient?
- 16.9 Why is the development of coronary artery disease of particular concern in the heart transplant recipient?
- 16.10 What factors affect patient compliance post transplantation? How can nurses improve patient compliance?

See answers to Chapter Review Questions in the Answer Section.

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Chapter 17

Care of the Acutely Ill Burn Patient

Ernest J. Grant, PhD, RN, FAAN

Linda S. Edelman, RN, PhD

Abbreviations

ABLS Advance Burn Life Support
BMR Basal Metabolic Rate
IAP Intra-Abdominal Pressure

TBSA Total Body Surface Area
VAC Vacuum-Assisted Closure

Learning Outcomes

Upon completion of this chapter, the learner will be able to:

1. Explain the common etiologies of burn injuries.
2. Evaluate the severity of a burn injury.
3. Explain the changes within the body system that occur following a burn and prepare for immediate care of the burn patient.
4. Describe the initial assessment of a patient with a minor burn injury.
5. Prioritize the care of the patients with major burns during the resuscitation, acute and rehabilitative phases.

Introduction

Burn injuries occur as a result of exposure to heat, chemicals, radiation, or electric current. The resulting transfer of energy from the heat source to the body initiates local changes to the integumentary system, including tissue destruction and initiation of the inflammatory cascade, which, in severe burns, can lead to a systemic response involving all body systems. Treatments range from over-the-counter topical agents for minor burns to specialized care for major burns provided in a burn intensive care unit. Specialized burn care involves skin grafting and

other surgical procedures, hemodynamic monitoring, and ventilator support.

Over one million fires occur in the United States each year, and burn injuries result in approximately 486,000 emergency department (ED) visits and 450,000 hospital admissions (American Burn Association, 2015). Approximately 3,200 of these injuries are fatal, making burns the seventh leading cause of death from unintentional injury (American Burn Association, 2015). The majority of burn injuries and deaths occur as a result of residential fires (Runyan, Bangdiwala, Linzer, Sacks, & Butts, 1992) and scalds (American Burn Association, 2015). Approximately

40% to 50% of these deaths could be prevented by the installation of working smoke alarms in the home (Ahrens, 2001). Most burn injuries are unintentional; however, intentional burns such as self-harm and child and elder abuse may also occur (Pruitt, Wolf, & Mason, 2012).

Men are at increased risk for burn injury. The 2014 National Burn Repository report states that nearly 70% of burn patients admitted to U.S. burn centers are male (National Burn Repository, 2014). Gender differences in rates of burn injury occur early, with males accounting for 60% of children under the age of one year admitted to U.S. burn centers, and remain throughout the lifespan (National Burn Repository, 2014). The patterns of injury are different between genders. Men are burned more often by fire/flame, whereas women experience more scald injuries (Kerby, McGwin, George, Cross, Chaudry, & Rue, 2006). Men tend to have larger cutaneous burns and are more likely to sustain inhalation injury.

Children and the elderly are at increased risk of burn injury and are more likely to die as a result of their injury. Children under 4 years of age are at particular risk for burn injury because of impulsivity and the inability to move away from a burn source. The majority of pediatric burn injuries and deaths are a result of scald injuries and residential fires. The elderly are at risk because of sensational and mobility impairments associated with aging and because of environmental risks such as living in older homes, using older appliances, and not having operational smoke alarms (Istre, McCoy, Osborn, Barnard, & Bolton, 2001; Pruitt et al., 2012; and Pham, 2012). Other factors associated with increased risk of burn injury include living in rural areas, poverty, and race.

Burn rates have declined over the last several decades as have the number of hospitalizations for burn treatment. Most burns are minor and can be treated in outpatient clinics and EDs. More severe burns are treated in hospitals or burn centers. Burn care for these patients has also advanced dramatically during the past few decades, resulting in declining death rates. Because the majority of burns are minor, nurses in outpatient and ED settings must be familiar with how to assess the severity of a burn, provide basic wound care, and support the unique psychosocial needs of burn patients and their caregivers. Nurses working in specialized burn centers are part of a multidisciplinary team that comprehensively treats patients through the resuscitation, acute care, and rehabilitation phases of a burn injury.

Etiology of Injury

The causes of burn injury may be thermal (heat) or nonthermal (electricity, chemical, or radiation). These etiologies all result in tissue damage; however, the causative agents and the initial treatment measures differ.

Thermal Burns

The most common burns are thermal in nature and are caused by exposure to heat. Thermal burns include fire/flame injuries, scald injuries from exposure to hot liquids or steam, and contact/friction injuries. Scald injuries are the most common form of burns, but fire/flame injuries account for the majority of deaths. The majority of thermal burns occur in the home, and the young and elderly are at particular risk.

Scald injuries may be caused by hot liquids or steam. Exposure to 120°F (49°C) water for five minutes can cause a scald in a healthy adult (American Burn Association, 2008). The exposure time required to sustain a scald injury is decreased to one second when the water temperature is 155°F (see “Safety Initiatives: Burn Prevention Education” and Table 17.1, Risk of Scald Injury). Severe burns can occur in children and the elderly at lower temperatures and exposure times. The majority of scalds occur in children less than 4 years of age. In this population, scalds are caused by pulling hot liquids onto themselves or by being placed in bath water that is too hot. The elderly and disabled individuals are at risk for scalding by hot bath water because of impaired sensation, slower reaction times, and decreased mobility. Grease and hot cooking liquid injuries are common scald injuries in adults. Kitchen-related scalds and hot tar burns are common work-related injuries.

Residential fires cause the majority of fire/flame injuries. Many of these injuries are caused by the misuse of fuels and flammable liquids. Other fire/flame injuries are vehicle-related, including flash burns sustained while repairing carburetors and starting boat motors, and burns resulting from ignition of fuel following an automobile crash. Fire/flame injuries tend to be larger and more severe than other burn etiologies. These injuries account for the majority of admissions to specialized burn centers and are more likely to result in death (Pruitt et al., 2012).

Ignition of clothing is a common source of thermal injury in all age groups and is the second most common cause of fatal burns (Pruitt et al., 2012). In particular, ignition of synthetic fabrics that melt and adhere to the skin results in more severe burns.

Other thermal burns include contact and friction injuries. Contact injuries occur when the body comes in contact with a hot object. Common sources of contact burns are motorcycle mufflers and campfire coals. The young, elderly, and disabled are at risk for burns caused by heating pads and blankets as well as other hot appliances. Common sources of friction burns are “road rashes” from motor vehicle accidents and, particularly in children, contact with treadmills or other fast-moving machinery.

Safety Initiatives

Burn Prevention Education

PURPOSE: There are several organizations whose mission is to provide burn injury prevention education and advocate for those at greatest risk. Detailed educational offerings are geared for the consumer, burn survivor, healthcare professionals and fire and life safety educators. Examples of such organizations include The American Burn Association's Burn Prevention Committee (Ameriburn.org), The Burn Prevention Network, (burnprevention.org); the National Fire Protection Association (NFPA.org); the CDC (CDC.gov); United States Fire Administration (USFA.fema.gov), and The Phoenix Society (phoenix-society.org). The American Burn Association resource website is designed to provide comprehensive up-to-date information for burn care professionals around the world.

RATIONALE: In the United States, it is usually the poor, very old, and very young who experience a burn injury. In a report from 2014, a fire death occurred every 2 hours and 41 minutes, and a civilian fire injury occurred every 33 minutes (Hayes, 2015). These fires resulted in nearly \$11.6 billion in property damage, and fire and other burn injuries resulted in \$7.5 billion in injury-related costs. Burn prevention education can prevent costly injuries and immeasurable suffering among the most vulnerable Americans (Pruitt et al., 2012)

HIGHLIGHTS OF RECOMMENDATIONS: Cooking is the leading cause of home fires in North America. Unattended cooking is a leading cause of kitchen fires and child scald injuries. Kitchen fires can be prevented by:

- Keeping flammable objects (such as pot holders and dish towels) away from heating sources
- Turning off cooking appliances as soon as the cooking is done
- Avoiding loose sleeves while cooking because they may come in contact with a heating source and catch fire

House fires may also develop from smoldering cigarettes or ignition of flammable substances stored in or near the home. These fires can be prevented by:

- Never leaving lit cigarettes unattended and always disposing of cigarettes properly after smoking
- Keeping cigarette lighters and matches in a safe place where children cannot reach them
- Installing smoke alarms and testing them on a regular basis to make sure they are still working
- Labeling and locking up flammable liquids, such as gasoline or kerosene, that may ignite

Scald injuries may occur from hot food and drink but also from excessively hot tap water. Adults can develop a full-thickness burn wound from exposure to liquids of the temperatures listed in Table 17-1 in the indicated amount of time. The exposure times needed for tissue damage are shorter in children and older adults.

The following may prevent scald wounds:

- Adjusting the hot water heater so the water temperature delivered from all faucets does not exceed 49°C (120°F)
- Replacing bath faucets and showerheads with anti-scald equipment
- Turning pot handles to the back of the stove (cook on back burners)
- Being aware that hot soup, coffee, tea, and hot chocolate can cause serious burns
- Keeping small children out of the cooking area and not holding babies while cooking or drinking hot liquids
- Supervising children in the bathroom

Sources: American Burn Association. (2012). Burn Prevention Fact Sheet/ Educator's guide. Retrieved from <http://ameriburn.org/preventionEdRes.php>.

Table 17-1 Risk of Scald Injury

Temperature (°F)	Burn Risk Exposure Time	Possible Exposure
110°	7 hours	Comfortable bathing 90°–110°
120°	5–9 minutes	Hot water heater setting
130°	30 seconds	Hot drinks for adults
145°	2.5 seconds	Too hot for most to drink
150°	1.8 seconds	Dishwasher heat cycle
170°	0.03 second	Temperature of hot drinks from some brewing units

Source: Courtesy of the American Burn Association. (2008). *Scald injury prevention: Educator's guide*. Chicago, IL: Author.

Electrical Burns

Each year, approximately 1,000 deaths occur as a result of electrical injuries (Pruitt et al., 2012). Low-voltage (alternating current) household current is responsible for one third of electrical injuries and occur most often to young

children who insert foreign objects into outlets or bite on electrical cords. High-voltage injuries are often work related; however, nearly 100 people die each year in the United States from lightning injuries (Pruitt et al., 2012). It is hard to assess the severity and extent of electrical burns



Figure 17-1 Electrical injury results in exit wounds, which often occur in the extremities such as in this patient with exit wounds to the fingers.

St. Stephen's Hospital, London/Science Source

because often only small entry and exit wounds are visible (♦ Figure 17-1). However, electricity follows the path of least resistance through the body and there may be widespread internal tissue damage, including tissue and bone destruction, which is not easily assessed. In addition, electrical injury may result in loss of consciousness and/or cardiac dysrhythmias that can lead to cardiopulmonary arrest. Electrical injuries have been associated with post-injury psychiatric sequelae including depression, anxiety and post-traumatic stress disorder (Ramati et al., 2009).

Chemical Burns

Chemical burns are common injuries seen in EDs (Pruitt et al., 2012). Chemical injuries are caused by direct skin contact with acidic or alkaline agents often found in household cleaning agents or industrial chemicals. Common household agents that can cause burns include drain cleaners, lye, ammonia (industrial and household strength), oven cleaners, toilet bowl cleaners, dishwasher detergents, and bleach. Alkalis, such as oven cleaners, result in more serious burns than acids because they are more difficult to neutralize, resulting in deeper tissue damage. The extent of damage from a chemical is related to the type and concentration of the agent and the amount and duration of contact with the skin. National, state, or local poison control centers should be contacted for information on how to neutralize the specific chemical agent. In addition, healthcare workers should be aware of risk to themselves or other patients and take appropriate cautions by remembering to wear personal protective equipment.

In the early part of the 21st century, EDs and burn centers began reporting increased numbers of injuries resulting from the production of methamphetamines. Although most of these injuries are flame burns, approximately 25% are chemical burns (Danks et al., 2004). Methamphetamine-related burn injuries have been reported to be more severe and result in increased risks of pneumonia and respiratory failure resulting in longer hospital stays (Burke et al., 2008; Spann et al., 2006). Individuals with methamphetamine-associated burns often do not disclose the cause of their

injury and, therefore, may not be properly decontaminated, putting healthcare workers at risk of exposure to the toxic chemicals involved in methamphetamine production. In 2006, the Federal Combat Methamphetamine Epidemic Act of 2005 was enacted, which resulted in a transient decrease in methamphetamine-related burn injuries. Recently, methamphetamine-related injuries have increased, particularly in areas of the Midwest, in part because of new methods of preparation (Davidson et al., 2013; MMWR, 2015).

Radiation Burns

Radiation burns from sun exposure or radiation treatment for cancer tend to be localized. Exposure to extensive doses of radiation from industrial and medical equipment or from nuclear power accidents is rare. Radiation burns are similar to other burns except that the time between exposure and clinical manifestations may be days to weeks.

Burn Classification and Severity

Burns are classified by total body surface area (TBSA) involved and depth of injury. Other contributing factors include the type and body location of the burn, patient age, gender, and past medical history, as well as the presence of inhalation and/or other injuries. Together these factors help form the clinical management of burn patients.

Size of Injury

Burn size is determined by the percentage of TBSA involved. Several methods are used to estimate TBSA. The **rule of nines** is an easy-to-remember method often used in the prehospital, emergency, or clinic settings (♦ Figure 17-2). In this method, the body is divided into five areas, each with increments of 9% TBSA. The areas are the head and neck (9%), arms (9% each), anterior and posterior trunk (36%), anterior and posterior legs (18% each), and the perineum (1%).

The rule of nines is not accurate for extremes of height and weight or for children. Therefore, a more comprehensive calculator of TBSA burned is the Lund and Browder chart (♦ Figure 17-3). This chart determines the percentage TBSA burned for each body part according to the age of the patient. It also allows clinicians to document the percentage TBSA according to burn depth.

The calculation of burn size is very subjective. Therefore, computer programs, such as the web-based Sage diagram, have been developed to increase the reproducibility of burn size assessments (Neuwalder, Sampson, Breuing, & Orgill, 2002). Digital photo planimetry software

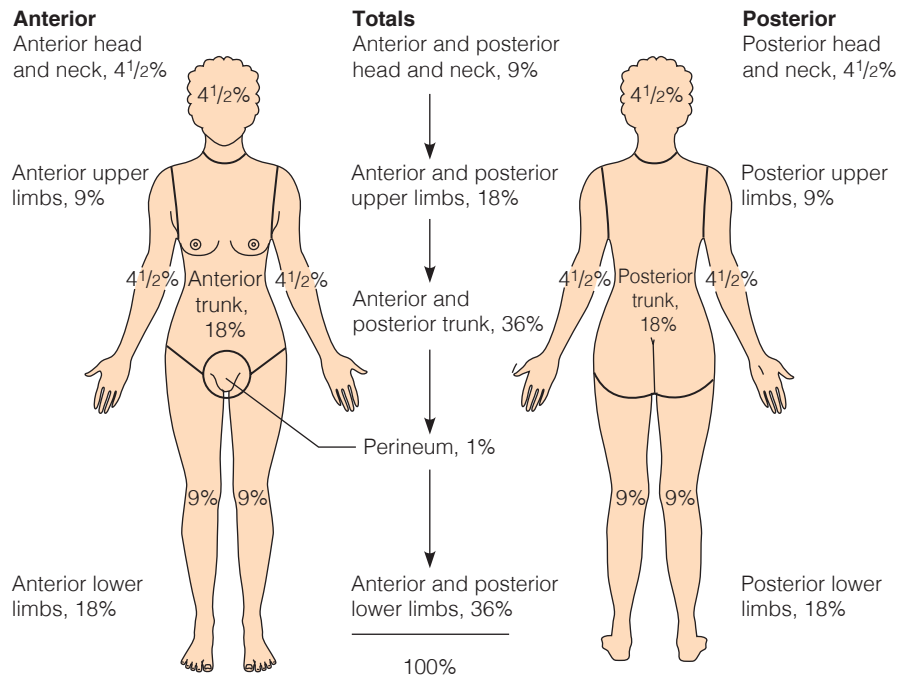


Figure 17-2 Rule of nines. The “rule of nines” is one method for quickly estimating the percentage of TBSA affected by a burn injury. Although useful in emergency care situations, the rule of nines is not accurate for estimating TBSA for adults who are short, obese, or very thin.

is increasingly used to calculate burn size and recently 3D photography has been shown to be an accurate and noninvasive alternative (Gee Kee, Kimble, & Stockton, 2015). Laser Doppler imaging can be used to accurately assess burn depth, although it is not commonly used in clinical settings (Jaskille, Ramella-Roman, Shupp, Jordan, & Jeng, 2010).

Depth of Injury

The depth of burn injury is determined by the depth of tissue destruction (♦ Figure 17-4). A number of factors contribute to burn depth, including the etiology, temperature, and duration of contact with the burning agent, and skin thickness.

Superficial, or first-degree burns, involve only the epidermal layer of the skin, leaving the skin intact (♦ Figure 17-5). The involved skin is pink to red in color and slightly edematous. Blisters will not form until after 24 hours, if at all. The TBSA of first-degree burns is not usually included in burn size estimates because these burns will heal without scarring in 3 to 6 days. However, extensive first-degree burns may cause chills, headaches, nausea and vomiting, and considerable pain. Common etiologies of first-degree burns are sunburns and minor flash burns.

A partial-thickness, or second-degree, burn involves the epidermal and dermal layers of the skin, but the hair follicles, sebaceous glands, and epidermal sweat glands remain intact (♦ Figure 17-6). A partial-thickness burn is further classified into either a superficial or deep second-degree

burn by the amount of dermis involved. A superficial partial-thickness burn does not involve the entire depth of the dermis. The burn wound is often bright red in color and edematous. Capillary refill is near normal. The surface is moist and thin-walled, fluid-filled blisters appear within minutes. A superficial partial-thickness burn is very painful and sensation to even very superficial pressure, such as air currents, is increased. Healing occurs within 21 days with little to no scarring. A deep partial-thickness burn involves the entire dermis. The wound appearance is white and waxy and capillary refill may be decreased. The surface may be wet or dry and blisters can range from large and fluid filled to flat. When nerve fibers are destroyed, there is less pain and decreased sensation even to deep touch. A deep partial-thickness burn heals in approximately 3 weeks and scarring is likely. Initially, a deep partial-thickness burn may appear to be a full-thickness burn, but after 7 to 10 days, skin buds and hair growth will be noticeable, indicating that the skin appendages are intact. However, some burns that initially appeared to be deep second-degree burns may progress to full-thickness injuries due to decreased blood supply reaching the injured area or infection.

A full-thickness, or third-degree, burn involves all layers of the skin, including the hair follicles, sebaceous glands, and the epidermal sweat glands (♦ Figure 17-7). The wound color ranges from very pale to bright red; but, unlike a second-degree burn, there is little to no capillary refill and thrombosed blood vessels may be evident. The surface of the wound is dry, firm, and may have a leathery feel and may also be referred to as *eschar*. A third-degree

Area	Age (years)					% 1°	% 2°	% 3°	% Total
	0-1	1-4	5-9	10-15	Adult				
Head	19	17	13	10	7				
Neck	2	2	2	2	2				
Ant. trunk	13	13	13	13	13				
Post. trunk	13	13	13	13	13				
R. buttock	2½	2½	2½	2½	2½				
L. buttock	2½	2½	2½	2½	2½				
Genitalia	1	1	1	1	1				
R.U. arm	4	4	4	4	4				
L.U. arm	4	4	4	4	4				
R.L. arm	3	3	3	3	3				
L.L. arm	3	3	3	3	3				
R. hand	2½	2½	2½	2½	2½				
L. hand	2½	2½	2½	2½	2½				
R. thigh	5½	6½	8½	8½	9½				
L. thigh	5½	6½	8½	8½	9½				
R. leg	5	5	5½	6	7				
L. leg	5	5	5½	6	7				
R. foot	3½	3½	3½	3½	3½				
L. foot	3½	3½	3½	3½	3½				
Total									

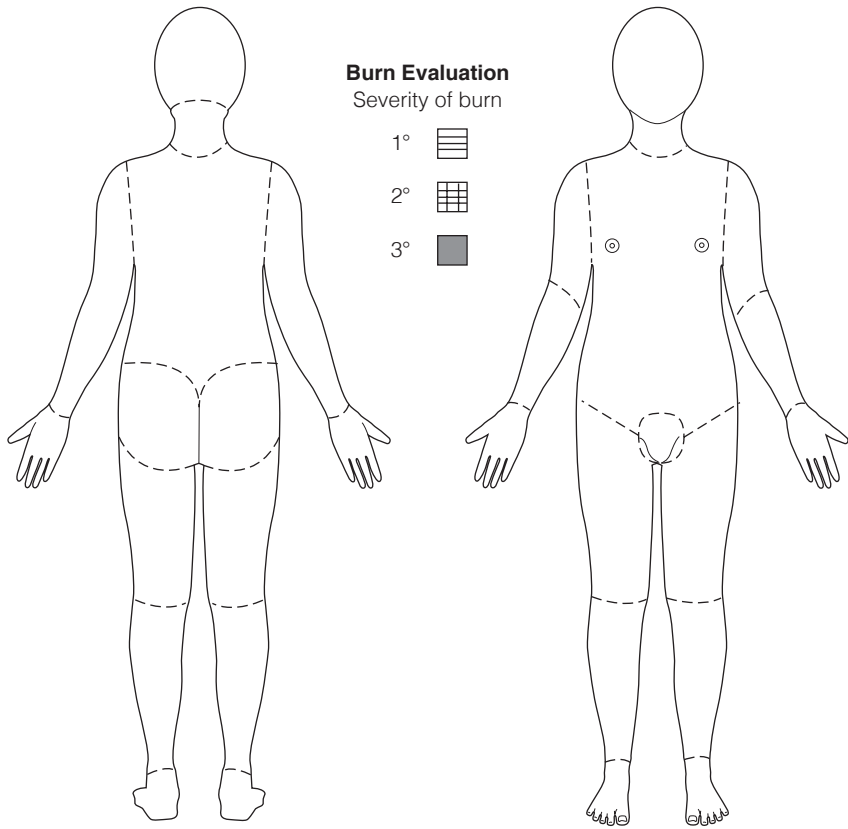


Figure 17-3 Lund and Browder burn assessment chart. This method of estimating TBSA affected by a burn injury is more accurate than the “rule of nines” because it accounts for changes in body surface area across the life span.

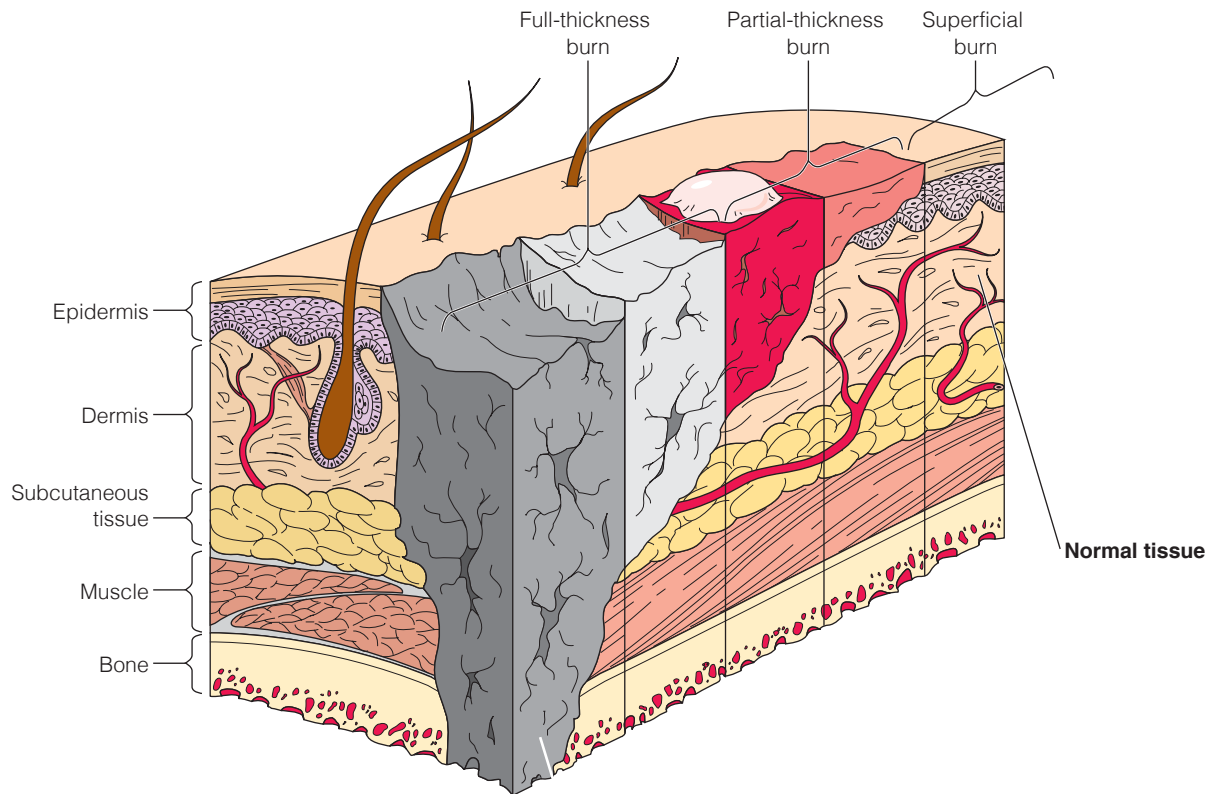


Figure 17-4 Depth of burn injury. Burn injury classification according to the depth of the burn.



Figure 17-5 First- and second-degree burns. The patient on the left sustained first degrees burn to the back (sunburn) where the skin is red but intact. The picture on the right shows a second degree burn to the palmar surface.

A, Michelle Del Guercio/Science Source B, Scott Camazine/Science Source



Figure 17-6 Partial-thickness burn injury.

Dr. P. Marazzi/Science Source

burn has no sensation to deep pressure. There is no pain because the nerve endings have been destroyed. Skin grafting is required for a third-degree burn to heal and for the patient to regain functionality to extremities.

If the burn extends into the subcutaneous tissue, muscle, or bone, it is classified as a fourth-degree burn. Such burns may be caused by prolonged exposure to a fire or flame, high voltage electrical injuries or exposure to highly concentrated chemicals. The extremities may appear charred



Figure 17-7 Full-thickness burn injury.

Dr. M.A. Ansari/Science Source

and mummified. Amputation is usually required. Fourth-degree burns are associated with high morbidity and mortality.

Other Factors Contributing to Burn Severity

Although the percentage of TBSA involved and the depth of injury are the primary classifications of burn severity, several other factors also contribute to the severity of the burn.

Location of Injury

The area of the body that is burned can contribute to severity of injury. Edema from burns to the face can cause acute airway compromise. Scarring is always a concern for individuals with burns to the face. Other body areas of concern are the genitalia, hands, and feet because of scarring and loss of function. A burn that extends over major joints also can be more serious because of the risk of developing joint contractures, resulting in loss of mobility. Injury to all of these areas can result in long-term morbidity and often require the specialized treatment provided by burn centers.

Age and Gender

Historically, mortality rates following burn injury have been the highest in the very young and the elderly. Pediatric patients can now survive very large and deep burns due to advances in pediatric burn care. However, mortality rates in the elderly remain high due to a number of reasons. The elderly individual with decreased mobility may have trouble extinguishing or escaping the heat source, and thus may sustain a more extensive and deep burn. Elderly skin is thinner and less elastic, resulting in deeper depth of burn. Chronic health problems in the elderly decrease the ability of the body to handle the systemic stress placed on it by a burn injury. The impact of burn injuries on psychosocial and physical functioning increases with age, resulting in loss of independence and quality of life (Klein et al., 2011).

Women are at decreased risk for burn injury but they have been reported to have increased mortality rates; female gender has been associated with higher mortality following a burn injury. This higher mortality is independent of age, burn size, or the presence of inhalation injury. The findings are most conclusive in women of reproductive age, suggesting that sex hormones may play a role (George et al., 2005; Kerby et al., 2006). However, a recent study of patients with larger burns found the association of female gender with mortality strongest in pediatric patients where the influence of sex hormones would be negligible (Summers et al., 2014). This study also found the sex differences increased with burn size (Summers et al., 2014).

Etiology of injury, pre-burn health status, and social factors may also influence the increased risk of mortality seen in women.

Concurrent Health Problems

The patient with chronic health problems has increased morbidity following a burn injury. It is important that a complete medical history is obtained from the burn patient. Particular attention should be paid to the patient with a history of cardiac, pulmonary, or renal disease because of the stress a burn injury places on these systems. Individuals with a history of (poor or malnutrition), diabetes, peripheral vascular disease, or impaired skin integrity may have decreased wound healing of even minor burns. Immunosuppression from disease or steroid use also increases morbidity.

Inhalation Injury

Approximately one-third of patients admitted to burn centers have concomitant smoke inhalation injury (Traber, Herndon, Enkhbaatar, Maybauer, & Maybauer, 2012). The patient with an inhalation injury has a mortality rate of 5% to 8%; this risk increases to over 20% if the patient also has cutaneous burns (Sen & Gamelli, 2005). Inhalation injury results in pulmonary edema, which predisposes the burn patient to pulmonary failure, infection, and long-term pulmonary complications; these will be discussed in detail later in this chapter.

Pathophysiology of Burn Injury

A burn injury rapidly invokes a multisystem physiologic response (Visual Map 17-1). The extent of this response depends on the size and depth of injury. Minor burns produce a localized response involving the integumentary system. Moderate and more severe burns result in changes that are systemic and long lasting. Major burns—those involving more than 20% TBSA—produce a pathophysiologic response involving all body systems. As a rule of thumb, therapeutic intervention is necessary in order for an individual with a major burn to survive.

Integumentary System

The skin is the largest organ in the body, accounting for 20% of the body's weight. It serves as a barrier to infection, ultraviolet radiation, and fluid loss. An important function of the skin is body temperature regulation. Touch and pressure receptors in the skin help protect the body from the environment and mechanical injury.

Anatomy of the Skin

The skin has three layers: the epidermis, the dermis, and the subcutaneous layer (refer to the layers of the skin depicted in Figure 17-4.) The outermost layer, the epidermis, is a protective layer that is continually regenerating. The epidermis varies in thickness depending on location. Therefore, the sole of the foot will receive a less severe burn than the perineum when exposed to the same heat source. The epidermis is thinner in the very young and the elderly, putting them at increased risk for more serious burn injury. Keratinocytes, the primary cells of the epidermis, are produced in the basal layer and then move upward, first becoming enlarged then flattened and stacked. At the skin surface the keratinocytes are dead and cornified, and they function as a protective barrier and in preventing evaporative water loss. The epidermis contains other cells that provide further protection. Melanocytes produce melatonin that provides skin color and shields the body from ultraviolet radiation. Langerhans cells, which migrate from the bone marrow, initiate an immune response against environmental antigens.

The second layer of the skin is the dermis, which is composed primarily of connective tissue. Also located in the dermis are fibroblasts, mast cells, and macrophages, which are important in immune regulation and the inflammatory response. The dermis provides the elasticity necessary for movement. The dermal appendages, including sweat and sebaceous glands and hair follicles, are located in the dermis. The papillary capillaries, located in the dermis, provide blood to the skin and dermal appendages. Sweat glands and arteriovenous anastomoses in the dermis are important in thermoregulation. The dermis also contains nerves and lymphatic vessels.

The third layer of the skin is the subcutaneous layer, which is composed of lobules of fat cells separated by walls of collagen and large blood vessels. The subcutaneous layer serves as a heat insulator, shock absorber, and nutritional reserve.

Burn Injury

The skin damage resulting from a burn injury depends on the amount and duration of heat as well as the body location. Because the thickness and vascularization of the skin vary, skin dissipates heat from a burn differently depending on location. If the microcirculation of the skin is not damaged by the heat source, such as in a first-degree burn, the dermis is protected. When the microcirculation is damaged, the skin cannot cool itself after the heat source is removed and the burning process continues, resulting in damage to the dermis.

Zones of Injury. A burn injury causes a bull's-eye pattern of injury on the skin surface. The deepest part of the burn is

Gerontological Considerations

According to the Centers for Disease Control and Prevention (CDC, 2015), over 25,000 Americans over the age of 65 experience a non-fatal burn injury each year, and more than 1,000 die from a burn injury, accounting for almost 40% of all fire-related deaths. For each decade over the age of 65, the death rate from a fire increases one-fold. Because older adults are more likely to die from a major burn injury, they are best treated in a specialized burn center.

Older adults with impaired vision and hearing often do not have smoke alarms that compensate for their impairment, thus, they may not hear an alarm, and they may not be able to evacuate a burning building promptly.

Fires caused by smoking are the leading cause of death in older adults. Fires due to smoking are especially dangerous in older adults because older adults are more likely to have concurrent respiratory problems (chronic obstructive lung disease, asthma, or lung cancer) and may be on home oxygen, which supports combustion. In addition, when the older person who is on home oxygen therapy has been smoking, his face, clothing or bedding is likely to be ignited early in the fire because of the increase levels of oxygen that may be trapped in their facial hair or clothing.

Approximately 3,000 older adults are injured in residential fires each year, with cooking fires being the leading cause of injuries. Because they have thinner skin, when older adults receive burn injuries they often get more severe burns at lower temperatures and in less

time than younger adults. In addition, the older adult may delay seeking treatment for the burn due to a diminished sense of pain. The effect of such a delay can be devastating; when treatment is delayed from 2 to 5 hours, the mortality increases fivefold.

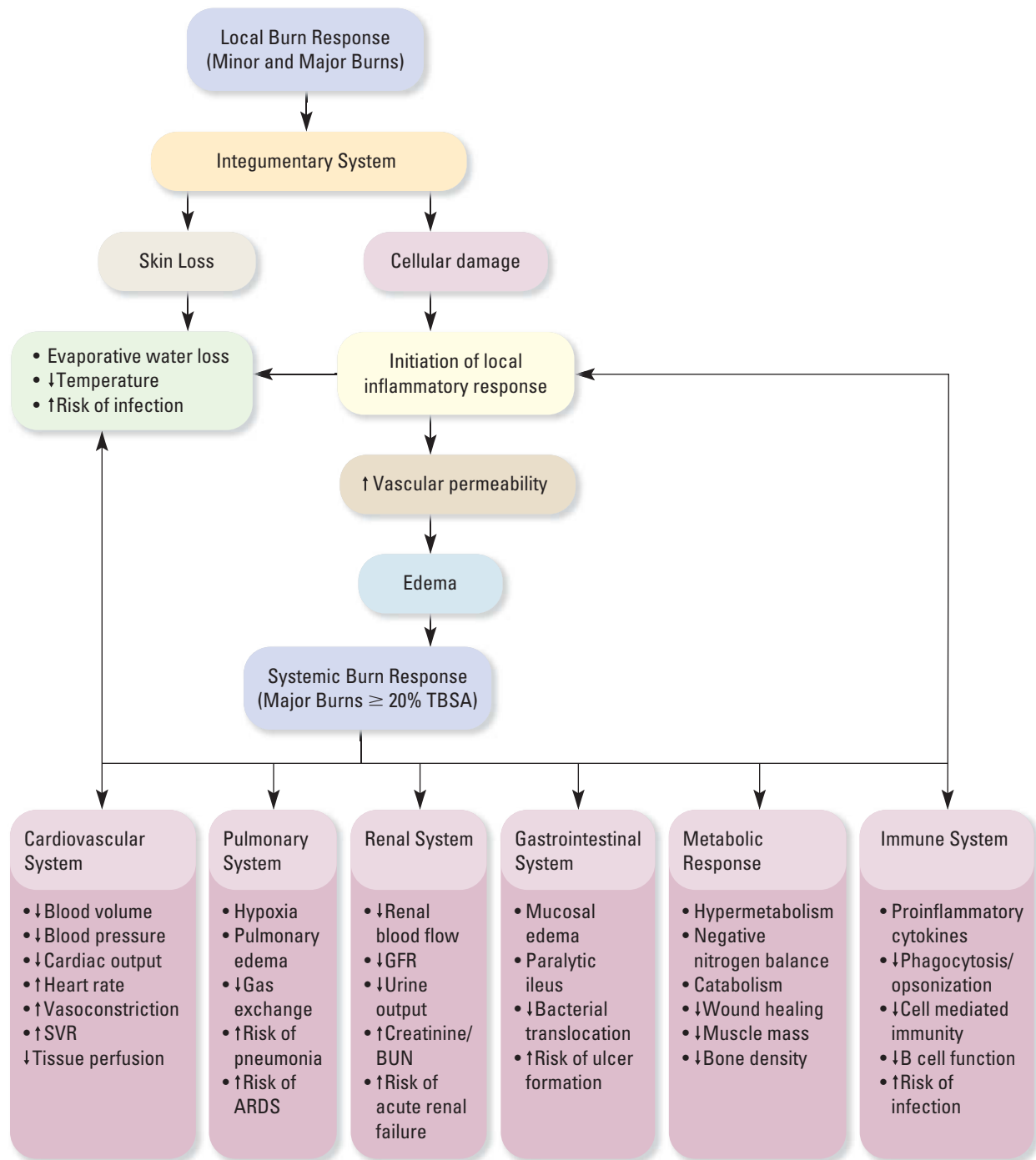
Tap water injuries may cause fatalities in older adults if the person falls, faints, or remains in a bathtub with water that is too hot. The recommended maximum setting for home water heaters is 120°F (49°C). Heating pads are another common cause of minor burns that may lead to major burns in older adults with sensory or cognitive deficits.

Preexisting health conditions influence how the older adult responds to the acute injury. The patient with heart problems must be closely monitored during fluid resuscitation, and a balance must be maintained between providing adequate resuscitation to the tissues and further stressing the heart. Older adults with dementia are at increased risk of experiencing burn injuries; the burn injuries they experience are more severe and result in longer hospital stays than older adults without dementia. The older adult with an inhalation injury may require more ventilatory support and is at increased risk for developing pneumonia or sepsis. Wound healing is delayed in the older adult, and finding good donor sites for skin grafting can be a problem. The older adult becomes debilitated more rapidly after a burn injury and return to pre-burn function is more difficult.

in the center with less deep areas surrounding it to form the periphery of the injury. One to three zones of injury develop depending on the depth of injury (♦ Figure 17-8). Third-degree burns develop in all three zones, beginning with the inner **zone of coagulation**, which is the deepest part of the burn. The cells are nonviable and the microcirculation is destroyed, leaving the skin dark colored and leathery. The hard crust that forms over the nonviable necrotic tissue is called *eschar*. The medial **zone of stasis** is composed of viable and nonviable cells. Tissue destruction in the zone of stasis can occur even after the source of heat is removed. Burns that result in deep partial or full thickness injury cause apoptosis of dermal cells, which contributes to tissue destruction in the zone of stasis that can continue for up to 3 weeks post-injury (Shupp et al., 2010). There is damage to the microcirculation, resulting in vasoconstriction and ischemia due to the production of inflammatory mediators such as oxygen-derived free radicals, cytokines, and complement. The skin initially appears moist or blistered and is red in color. Capillary refill is delayed. Spontaneous

healing can occur, but persistent inflammation or infection can result in further loss of perfusion and subsequent conversion with the zone of coagulation. The outer **zone of hyperemia** is tissue with intact microvasculature that heals spontaneously within days. The area initially appears pink, and capillary refill may be increased due to vasodilation induced by local inflammatory mediators.

Alterations in Skin Function. The skin is sterile immediately after a burn injury. Eschar develops over the burn wound and provides the perfect medium for bacterial growth. The burn wound soon becomes colonized with low concentrations of bacteria and yeast, which are normal flora of the accessory glands of the skin, respiratory, and gastrointestinal tracts (Sharma, 2007). Burn patients are at risk for developing nosocomial infections because they are immunosuppressed. Hospital infection protocols should be followed, aseptic technique should be used when handling the burn wounds, and dressings should be changed regularly. The wound should be assessed for



Visual Map 17-1 Burns Pathophysiology

signs and symptoms of infection at each dressing change and appropriate treatment initiated. Examples of infection may include redness extending well beyond the border of the burn, delayed healing, or changes in the wound appearance.

Patients with large burns are more susceptible to infection and have difficulty regulating body temperature even after the burn wound is healed. Healed burned areas are more susceptible to mechanical injury as a consequence of changes in the texture of the skin and loss of sensory

perception. Sun exposure should be avoided because burned areas are more susceptible to ultraviolet radiation. In addition, patients with deep partial-thickness and full-thickness burns have a decreased ability to synthesize vitamin D when exposed to sunlight.

Cardiovascular System Changes

A burn injury can cause a variety of cardiovascular system changes, particularly in individuals with preexisting

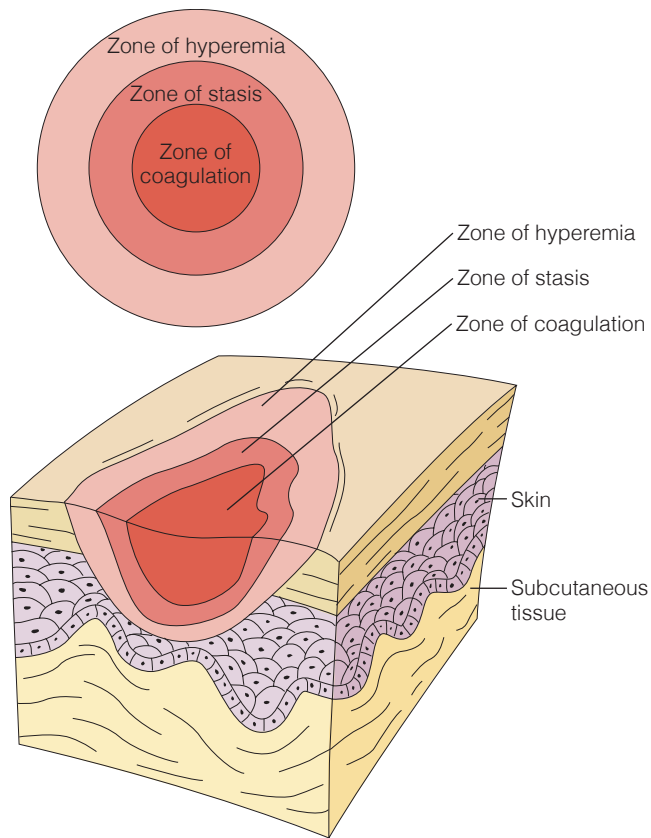


Figure 17-8 The zones of injury.

cardiovascular disease. The most common cardiovascular change is hypovolemic burn shock. Other changes include alterations in cardiac rhythm and peripheral extremity vascular compromise.

Within minutes of a burn injury, increased capillary permeability in burned areas where the microcirculation is not destroyed results in edema formation. The extent of edema depends on burn severity and body parts affected. Burn edema is greatest over areas where the skin is less elastic, such as the face and genitals, and less over areas with increased elasticity, such as the extremities. Increased vascular permeability is present for approximately the first 24 hours after injury. Without intervention, burn edema decreases over the first 72 hours following injury as circulating blood volumes increase and diuresis occurs. Factors that contribute to sustained burn edema are extremes of age, burn severity, and cardiovascular compromise. In addition, fluid resuscitation can contribute to increased and sustained burn edema.

Burn Shock

Burn edema is generalized in the patient with major burns. Resulting burn shock has two components. The cellular component of burn shock is the result of damage to cells in the burn wound. Damaged cells have altered membrane

potential that results in metabolic and immune changes, which are discussed in subsequent sections. The hypovolemic cardiovascular component is a result of the massive decrease in circulating blood volume as systemic edema develops. The loss of plasma blood volume results in hyperviscosity of the blood and slowed capillary circulation. Burn shock occurs when blood pressure and cardiac output decrease to compensate for blood volume losses. As a result, tachycardia and vasoconstriction occur, increasing systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR). The release of inflammatory mediators suppresses myocardial contractility. Burn injuries are catabolic. Increased oxygen consumption further contributes to the cardiovascular response. Electrolyte imbalances can arise and contribute to the development of cardiac dysrhythmias.

White blood cells (WBCs) and platelets accumulate in the tissue surrounding the burn. The combination of vasoconstriction with the accumulation of WBCs and platelets contributes to decreased tissue perfusion. Decreased perfusion to the burn causes ischemia, which can progress to necrosis. Prolonged ischemia and lack of oxygen to damaged tissue can cause burn depth to increase. Large burns require fluid resuscitation to reverse burn shock and prevent further complications such as acute renal failure and cardiovascular collapse. However, despite adequate fluid resuscitation, cardiac output remains decreased in patients with large burns during the acute burn phase. Fluid status assessment and prompt alterations in resuscitation parameters are important nursing functions.

Other Cardiovascular Changes

Burns can cause myocardial changes. Electrical injuries can result in dysrhythmias or cardiac arrest by interfering with the electrical activity of the heart or by direct heat to the myocardium. Inflammatory mediators produced in response to a large thermal injury can cause myocardial ischemia, which leads to decreased contractility and cardiac output.

Peripheral vascular compromise occurs in individuals with extremity burns. Peripheral vasoconstriction from burn shock and edema contribute to decreased perfusion. Circumferential extremity burns are at risk for developing compartment syndrome in which the pressure within the muscle compartments is greater than within the microvasculature.

Respiratory System Changes

Pulmonary damage occurs either by direct inhalation injury or as part of the systemic response to burn injury. Patients exposed to smoke or heat within an enclosed place, or those near an explosion, are at increased risk for

inhalation injury that can occur in the absence of cutaneous burns. Individuals with singed scalp or facial hair should be evaluated for inhalation injury.

Inhalation injury is caused by exposure of the airways to heat or toxic substances, resulting in damage to the upper and lower airways. Inhalation of heat immediately causes direct injury to the mucous membranes of the upper airways, resulting in inflammation and edema and subsequent airway compromise. Immediate signs of inhalation injury are changes to the mucosal lining of the oropharynx and larynx and include the presence of soot, hoarseness, edema, or blisters. Symptoms of direct injury occur over the first 24 hours and include stridor, hypoxia, and respiratory distress.

Injury to the lower airways is a result of the inhalation of noxious gases and particulate matter, and rarely of direct thermal injury. The resulting chemical irritation of the alveolar tissue causes loss of ciliary action, increased mucus production, edema, and bronchospasm. In addition, carbon monoxide (CO), which is a compound in smoke, rapidly diffuses into the bloodstream and directly impairs oxygen utilization by displacing oxygen on hemoglobin. The patient with a lower airway injury may not initially show signs of respiratory difficulty and have normal arterial blood gases and chest x-rays for the first 24 hours, after which chest x-rays may show the development of patchy atelectasis. Inhalation injury increases the risk of developing infection, respiratory failure, and acute respiratory distress syndrome (ARDS). Morbidity and mortality are greatly increased.

Burn shock contributes to pulmonary changes following burn injury. Patients with major burns may develop pulmonary edema as a result of increased PVR. Immune system changes can increase the risk of developing pneumonia and ARDS, particularly in patients requiring mechanical ventilation for extended periods. Prophylactic use of steroids and antibiotics should not be used when an inhalation injury is suspected.

Gastrointestinal System Changes

The systemic vasoconstriction caused by burn shock often results in the development of paralytic ileus and mucosal edema. The patient with paralytic ileus is at risk for abdominal distention, nausea, vomiting, and hematemesis. A nasogastric tube is placed in the patient with a major burn to prevent abdominal distension and aspiration if emesis occurs. Bowel ischemia can lead to increased intestinal mucosal permeability, which allows bacterial translocation from the lumen of the gut to the mesenteric lymph nodes and systemic tissues. Bacterial translocation increases the risk of systemic infection and multiple-organ dysfunction syndrome (MODS) (Magnotti & Deitch, 2005). Stress ulcers in the stomach or duodenum are common in burn patients.

Symptoms of a stress ulcer, which can occur after the acute phase of the burn injury, include abdominal pain, hematemesis, and melanic stools. Symptoms can arise quickly, and close attention should be paid to hematocrit levels because patients often require blood transfusions. The patient should be started on proton pump inhibitors to minimize the potential formation of stress ulcers.

Renal System Changes

Renal blood flow and glomerular filtration rates are greatly decreased during the hypovolemic burn shock phase of burn injury. As a result, urine output is decreased and serum creatinine and blood urea nitrogen increase. Hemoglobinuria (free hemoglobin from destroyed red blood cells [RBCs]) and myoglobinuria (myoglobin released from damaged muscle cells) can cause urine to be dark brown. Acute renal failure can develop if fluids are not administered and renal blood flow normalized.

Urine output is the most reliable monitor of adequate burn shock resuscitation. Every patient with a burn large enough to warrant fluid resuscitation, generally 20% TBSA or larger, should have a urinary catheter placed immediately. Urine output should be monitored hourly throughout the resuscitation phase. Fluids should be administered to maintain urine output greater than 30 mL/hr in adults or 1 mL/kg/hr in children.

Immune System Changes

The cellular component of burn shock causes immediate and severe changes to the immune system. Activation of the inflammatory cascade occurs immediately after a burn injury. This systemic inflammatory response syndrome (SIRS) results in the release of pro-inflammatory cytokines and other inflammatory mediators (Murphey, Sherwood, & Toliver-Kinsky, 2012). Products released from activated inflammatory cells increase vascular permeability and local tissue ischemia. The function of WBCs and other phagocytic cells is altered and the ability to opsonize and phagocytose bacteria is decreased. The number of circulating T cells is decreased and T helper (Th) cell production shifts to Th2 lymphocytes. A decrease in Th1 cells results in decreased cell-mediated cytotoxicity. Levels of circulating antibodies are diminished. Together, suppression of the humoral and cellular immune responses results in immune dysfunction that can last for over a month, putting the patient at risk for infectious complications.

Metabolic Changes

Cellular metabolism is decreased the first few days following burn injury. Oxygen consumption is decreased during hypovolemic burn shock. Cellular metabolism increases

once hypovolemia has been reversed by fluid resuscitation. The basal metabolic rate (BMR) can be twice the normal rate. Even so, the body expends up to 100% more energy than it produces. Hypermetabolism results in protein catabolism, lipolysis, and gluconeogenesis. Urea and creatinine levels in the urine increase. Body weight drops quickly. Delayed wound healing, muscle wasting, and bone loss can occur. Hypermetabolism persists until wound closure. It is imperative that burn patients receive early and adequate oral or gastrointestinal nutrition to combat hypermetabolism.

COLLABORATIVE CARE

Burn care is multidisciplinary. Physicians, nurses, physical and occupational therapists, pharmacists, dietitians, social workers, respiratory therapist, chaplains, recreation therapist, psychiatrists, and case managers work together to plan and administer care. Nurses are a vital member of the burn team and often manage the continuum of care required for full recovery. Burn care is often broken down into three stages: the resuscitation, acute, and rehabilitation stages. Burn care must be as dynamic as the burn injury itself and the plan of care must be individualized and continually reevaluated.

The following section discusses the primary assessment of the burn that will determine if the burn is minor or major and where care will be delivered. Minor burns are often treated in the ED or outpatient clinic, and the focus of care is pain management and wound care. Large burns require hospitalization, often in specialized burn centers, for the resuscitation and acute care phases. Burn care does not end when the patient is discharged from the hospital but continues through rehabilitation, scar management, and aftercare.

The first step in burn care, whether at the scene or in a medical facility, is to stop the burning process. The patient should be removed from the burn source as quickly as possible while protecting the patient from further injury or endangering others. The burned area should be cooled with cool (not cold) water for at least five minutes. Chemical burns should be lavaged with large amounts of water immediately. Any powdered chemicals should be brushed from the clothing and skin prior to lavage. Clothing and jewelry should be removed to prevent further burning; however, clothing that adheres to the skin should be left in place.

Once the burning process is stopped, the burns should be covered with dry bandages and the patient wrapped in blankets to prevent unnecessary heat loss. One exception is chemical burns that should be lavaged repeatedly with water until a determination is made how the chemical agent should be treated.

Primary Assessment

A burned patient's appearance can be startling to individuals unused to working with burn injuries. Normal skin can be covered in soot. Injured skin may range in color from white to dark brown or may be covered in large weeping blisters. The burn wound is not itself an immediately life-threatening injury, but systemic effects may be, particularly if the patient experienced inhalation or electrical burns. Therefore, the first assessment of a burn patient, whether at the scene or in the ED, should be a primary trauma survey (while maintaining c-spine stabilization) beginning with the ABCs (airway, breathing, circulation).

First, the patient's airway and breathing should be assessed. If the patient is not breathing, immediate steps should be taken to establish a patent airway. The patient is evaluated for signs and symptoms of inhalation injury, including the presence of facial burns or singed facial and scalp hair, progressive stridor, and hoarseness. Humidified oxygen is administered, particularly if the patient has signs of inhalation injury. The patient may be prophylactically intubated if there are signs of progressing respiratory stress or airway edema. Procuring a secure endotracheal tube is very important because it is very difficult to reintubate a burn patient due to severe airway edema and neck swelling. The Advanced Burn Life Support Course (ABLS) recommends the use of umbilical tape ties to secure and stabilize endotracheal tubes, rather than commercially available devices. Adhesive tapes and Velcro closures commonly used on commercial devices do not adhere well to a burned face, thus increasing the potential for endotracheal tube dislodgement (American Burn Association, 2011).

Assessment of the burn patient's circulation must be performed immediately. Heart rate and blood pressure should be reassessed frequently. At least one large-bore intravenous line (preferably through non-burn peripheral tissue) should be placed and well-secured. Cardiac monitoring should be initiated for all major burns. Electrical burn patients should have cardiac monitoring for 24 hours following injury to assess for dysrhythmias. Tachycardia and hypotension are expected findings immediately after a burn injury. Body temperature can decrease rapidly secondary to the loss of intact skin, and the patient should be kept covered with warm blankets. The administration of warm IV fluids and increasing the room temperature may also help diminish hypothermia. Extremities should be elevated.

Once the initial ABCs have been assessed, neurological status should be examined. A burn patient should be awake and able to follow commands. Decreased neurological status or unconsciousness may indicate anoxic injury or an additional neurological injury. If the patient was involved

in a fall or high-voltage electrical injury, a cervical spine collar and long board should be applied. The patient's vital information should be obtained as soon as possible, particularly if the patient has major burns and may be unable to provide information if sedation or intubation is required later.

Initial Burn Assessment

A secondary burn assessment may be started at the scene and repeated in more detail once the patient reaches the ED. Often the burn assessment is done in parallel with the primary assessment. A complete burn history should be obtained either from the patient or individuals present at the burn scene. Injury details such as etiology, location, and circumstances should be described in as much detail as is available. Initial vital signs and neurological status should be recorded. Part of the burn assessment is to examine the circumstances of the injury. Burns are a common form of child and elder abuse, and the patient should be evaluated for patterns of injury that are not congruent with the burn history. Adult burn patients with suspicious patterns of burns that are not in agreement with the burn history may have self-inflicted or assault-related burns, or have been involved in illegal activities such as methamphetamine production. Classic patterns of burn injury that are suspicious of abuse in children include scalds with uniform depth, bilateral symmetry, and the absence of splash marks, indicating that the child was held still; multiple superficial well-defined circular burns, indicating cigarette burns; and small punctate burns indicative of electrocution with small live wires (Greenbaum, Horton, Williams, Shah, & Dunn, 2006; Toon et al., 2011) (♦ Figure 17-9). Children with suspicious burn wounds often have signs of previous abuse (Greenbaum, Donne, Wilson, & Dunn, 2004). Patterns are similar for elder

abuse. Hospital and state guidelines for reporting inflicted burns should be followed.

The physical assessment of the burn should be head-to-toe and should include examination for any signs of co-trauma. The eyes should be examined for corneal injury prior to the onset of severe edema. Signs of inhalation injury should be reassessed. Carboxyhemoglobin levels should be obtained if the patient was burned within an enclosed space or with signs indicating inhalation injury. If inhalation injury is suspected, a bronchoscopy may be performed.

The location and appearance of all burn wounds should be assessed as to size and depth of injury. The patient with deep or circumferential chest burns is at increased risk for developing respiratory problems and may require escharotomies to ensure adequate respiration. Such procedures should not be attempted in the pre-hospital or ED setting without consulting with the receiving burn center. All extremities should be assessed for circumferential burns to identify areas at risk for loss of perfusion as burn edema progresses. Peripheral nerve stimulation is difficult to perform on edematous areas but should be attempted if circumferential extremity burns are present.

A diagram of the burn injury should be drawn during the burn assessment using either the rule of nines or the Lund and Browder chart (Figures 17-2 and 17-3). The percentage TBSA of second- and third-degree burns and the total percentage of TBSA burned should be calculated. The percentage of TBSA burned guides fluid resuscitation and helps in the determination of whether the patient should be transferred to a specialized burn center. The burn assessment should end with a decision as to whether the patient has a minor injury that can be treated in the outpatient setting or if the burn is severe enough to warrant hospitalization.

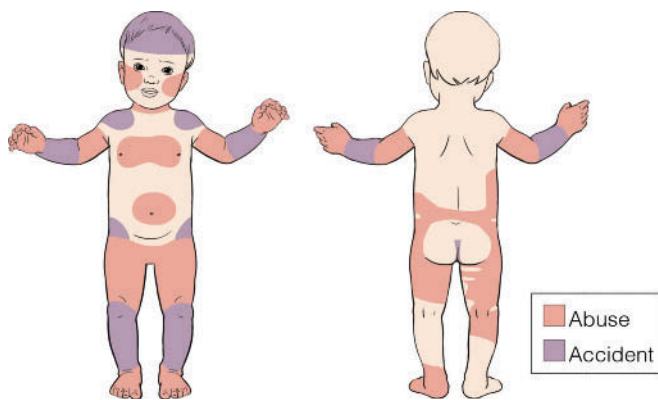


Figure 17-9 The child has a burn injury pattern that is suspicious of intentional scalding. The burns on the buttocks and feet suggest that the child was placed into hot water.

Transfer of the Patient to a Specialized Burn Center

The patient with a major burn injury is best treated in a specialized burn center. Approximately 60% of patients admitted to a U.S. hospital for treatment of a burn injury are admitted to burn centers (American Burn Association, 2015). The American Burn Association uses the guidelines established and updated by the American College of Surgeons Committee on Trauma (2014) to describe the burn patient who should be transported to a burn center for treatment (Box 17-1). Criteria evaluated to determine if referral is warranted include burn size, burn etiology, pre-existing medical conditions, concomitant traumas, children first seen in hospitals without pediatric care capabilities, and individuals with specific psychosocial needs.

Box 17-1 American College of Surgeons Burn Center Referral Criteria

A burn center may treat adults, children, or both.

Burn injuries that should be referred to a burn center include the following:

1. Partial-thickness burns of greater than 10% of the total body surface area.
2. Burns that involve the face, hands, feet, genitalia, perineum, or major joints.
3. Third-degree burns in any age group.
4. Electrical burns, including lightning injury.
5. Chemical burns.
6. Inhalation injury.
7. Burn injury in patients with pre-existing medical disorders that could complicate management, prolong recovery, or affect mortality.
8. Burns and concomitant trauma (such as fractures) when the burn injury poses the greatest risk of morbidity or mortality. If the trauma poses the greater immediate risk, the patient's condition may be stabilized initially in a trauma center before transfer to a burn center. Physician judgment will be necessary in such situations and should be in concert with the regional medical control plan and triage protocols.
9. Burns in children; children with burns should be transferred to a burn center verified to treat children. In the absence of a regional pediatric burn center, an adult burn center may serve as a second option for the management of pediatric burns.
10. Burn injury in patients who will require special social, emotional, or rehabilitative intervention.

Source: Excerpted from American College of Surgeons. (2014). Guidelines for trauma centers caring for burn patients. In Committee on Trauma & American College of Surgeons (Eds.), *Resources for optimal care of the injured patient* (pp. 100–106). Chicago, IL: Author.

The closest burn center should be contacted when burn patients meet any of the referral criteria. The nurse should be prepared to provide the burn center with the following information:

- Patient demographics and past medical history, including allergies, medications, and immunization status
- Burn circumstances
- Burn size estimate and the presence or absence of inhalation injury and/or co-traumas
- Appearance of burn wound and dressings in place
- Vital signs
- Airway management
- Pain management
- Fluids and medications administered since the burn injury
- Presence of venous or arterial lines, urinary catheter, nasogastric tube
- Social history and special needs

The burn center will advise the referring ED or hospital on how to prepare the patient for transport, including fluid resuscitation requirements and airway management.

Burn centers are regional centers of care; therefore, patients are often transported over great distances to the nearest burn center. Air transport is the primary mode of transport for patients with major burn injuries or with co-traumas. Less severe burns may be transported by ground

ambulance or private vehicle for burn center admission or evaluation in the burn outpatient clinic. Ideally, transport personnel should have critical care transport certification for optimal care during the transport process.

The Patient with Minor Burns

The majority of burn injuries are minor burns involving less than 20% TBSA and no full-thickness injuries. In this textbook, a minor burn is defined as any adult burn less than 20% TBSA and any pediatric burn less than 10% TBSA. However, other factors such as age, co-traumas, or preexisting medical conditions can contribute to the seriousness of a burn injury. Minor burns do not usually require hospitalization; however, some minor burns should be evaluated in a specialized burn center because of associated factors that require more intensive treatment. Therefore, a patient with a minor burn involving 10% to 20% TBSA should be considered for burn center referral. A patient with a minor burn injury who has burns involving the face, hands, feet, genitalia, perineum, or major joints should be referred to a burn center. Likewise, a patient with a minor burn caused by electricity or chemicals, or with inhalation injury, is best evaluated in a burn center. The minor burn patient with pre-existing medical conditions, co-traumas, or requiring special psychosocial interventions will benefit from specialized burn care.

COLLABORATIVE CARE

The patient with a minor burn may present to the ED or outpatient clinic immediately after burn injury, or days later when he or she has concerns about pain, burn severity, or infection. The patient with a minor burn that does not meet the criteria for referral to a burn center does not require formal resuscitation but should be encouraged to increase oral fluid intake for the first 24 hours post-burn to replace fluids lost by localized edema. Oral electrolyte replacement solutions will help to restore fluid balance. Minor burns can be very painful and anxiety invoking for the patient and family. Nursing care should encompass burn wound care and pain management. Time should be spent discussing the resources of the patient and family and concerns they have about caring for the burn. The ability of the patient to care for the wound and to be safe at home should be assessed. The patient should be given detailed written instructions on wound care, pain management, and signs and symptoms of infection prior to discharge to home.

Management of Pain

The patient with a minor burn may be experiencing intense pain by the time he or she presents at a healthcare facility. Pain should be assessed and treated prior to beginning wound care using developmentally appropriate pain scales. The patient undergoing the initial wound evaluation and debridement may require an intravenous narcotic such as morphine to achieve adequate pain control. Anxiolytics such as midazolam and lorazepam may be required if the patient is experiencing high anxiety. Nurses, working with social workers and child-life specialists, often use nonpharmaceutical methods to decrease the pain and anxiety associated with wound care. Techniques involving guided imagery, hypnosis, and relaxation can decrease the amount of pain medication needed. Oral pain medications should be prescribed for the patient to take as needed at home.

Reflect On

Have you ever had a patient who was experiencing extreme anxiety regarding procedures such as a dressing change? What were the triggers for the anxiety? Did you consider any nonpharmaceutical interventions to decrease his or her anxiety? Have you ever considered how a patient's culture may influence how they deal with pain?

Wound Care

Wound care of minor burns involves cleaning and dressing the wound. The first step is to debride the burned area of loose tissue, wound debris, and eschar. First, the wound is gently washed with a mild antimicrobial soap or

wound-cleansing solution. Washing can be done with gauze pads or wet washcloths. Whether to debride intact blisters is controversial. Usually intact or broken blisters are removed to decrease the risk of infection (Moss, 2010). Eschar or loose skin that cannot be trimmed with a scissors or removed with gentle washing should not be removed. Body hair in the wound and immediately around it should be shaved. The wound is rinsed with warm water or saline and patted dry with gauze or warm cloths.

Minor burn wounds that have developed thick eschar or necrotic tissue may require enzymatic debridement. A number of commercially available topical enzymes are available. The nurse should follow the directions on the product label. Generally, the enzyme is applied to the burn wound and covered with gauze followed by a wet dressing.

Dressings for Minor Burns

A wound dressing should achieve two goals: It should maintain a moist environment to facilitate wound healing and it should prevent infection. These goals are accomplished by applying a topical ointment or cream directly to the clean wound, and covering the wound with a dressing. A number of topical agents are used to treat minor burn wounds. Two common topical burn wound creams are Bacitracin ointment and silver sulfadiazine (Silvadene), which have antimicrobial properties. Bacitracin is used to treat first- and second-degree burns; silver sulfadiazine is used for deep-second-degree burns. Ointments containing vitamins A, D, and E are commonly used to dress minor burns of the face. Once the minor wound has healed, vitamin-enhanced ointments are used to keep the skin moist.

The topical agents applied directly to the minor burn wound can be left open to air or covered with a semi-open or closed dressing (♦ Figure 17-10). Minor burns, particularly on the face and head, may be covered with a thin layer of antimicrobial cream and left open to air. Silver



Figure 17-10 Closed method of dressing a burn.

Antonia Reeve/Science Source

Sulfadiazene should not be applied to facial burns. The topical agent must be reapplied frequently so the wound does not become dry. Wounds left open to air may be more painful because nerve endings are exposed to air currents. Therefore, most wound agents have nonadherent dressings placed over the topical agent. The nonadherent dressings can be covered with netting so the wound remains semi-open to the environment. The application of gauze pads or rolls over the nonadherent dressing provides a closed dressing to the environment. Patients with closed dressings have greater mobility and are less prone to infection.

The nurse providing wound care for a minor burn should invite the patient to participate in each step. Participation can decrease pain and anxiety by allowing the patient to control the timing of each step. Participation, along with demonstration, also shows the patient how wound care should be performed. The patient and caregivers should be educated about the wound appearance and how to conduct wound care at home. It is important the patient is informed about the signs and symptoms of infection and any other concerns specific to the wound.

The Patient with a Major Burn

Major burns differ from minor burns in that they require more intensive acute burn care, usually in the hospital setting. Burns involving greater than 20% TBSA in adults, or greater than 10% TBSA in children and adults older than 50 years, are considered major. However, third-degree burns, inhalation injury, electrical burns, or burns in individuals with preexisting health conditions may also require burn resuscitation. The patient with a major burn injury should be referred for evaluation and treatment in a specialized burn center.

Collaborative Care

Nurses are vital members of the burn team that manages and cares for major burns. An important component of nursing care for the burn patient is to discuss the care plan goals with the patient and family and to adjust goals accordingly. In many burn centers, nurses are responsible for coordinating the care provided by burn team members.

The care of a patient with major burns can be divided into three phases: the fluid resuscitation phase, the acute care phase, and the rehabilitation phase. The resuscitation phase begins with emergency medical services (EMS) and continues until fluid resuscitation is complete,

approximately 72 hours after injury, and the acute phase continues. The nurse is vital in administering fluids, monitoring the patient's status, and adjusting care accordingly. The acute phase lasts until the burn wound is closed, which can be weeks to months after the burn injury occurred. Nursing care focuses on promoting wound healing, preventing complications, and providing psychosocial support to the patient and family. The final rehabilitation phase focuses on restoring the burn patient's ability to function in society and can last for several years following a burn injury. Most of the rehabilitation phase occurs outside the hospital and nursing interventions are limited. Many burn centers have established burn aftercare programs that work in conjunction with organizations, such as the Phoenix Society for Burn Survivors, to help provide psychosocial support for the burn survivor and their family.

Essential for Evidence-Based Care

A 4-year-old otherwise healthy child presents with a 10% TBSA burn from touching a hot stove surface on the left palmar surface of his hand, left forearm, and anterior trunk. The nurse should know what information should be obtained for a burn unit consultation and if this child meets the criteria for transfer to a burn center.

Resuscitation Phase

The fluid resuscitation phase of burn care begins at the time of burn injury and ends with successful fluid resuscitation. Patients with major burns entering this phase should have large-bore intravenous access for fluid administration. A urinary catheter should be placed prior to administering large boluses of fluids. In men, the foreskin should be reduced to avoid paraphimosis from excess edema. A nasogastric tube should be placed and suction applied to prevent aspiration. Heart rate and blood pressure monitoring should be available.

NURSING DIAGNOSES

Patient with a Burn Injury

- Ineffective airway clearance related to inhalation injury
- Impaired gas exchange related to inhalation injury
- Deficient fluid volume related to third-spacing of fluids
- Pain related to burn injury
- Impaired skin integrity related to burn injury
- Anxiety related to sudden change in health status
- Body image disturbance related to change in physical appearance
- Risk for infection related to inadequate defense mechanisms
- Risk for imbalanced nutrition less than body requirements due to increased metabolic needs following burn injury

Nursing care during the resuscitation phase is intensive. Refer to the “Nursing Diagnoses: Patient with a Burn Injury” boxed feature for a list of common diagnoses.

Airway Management

Laryngeal swelling and upper airway obstruction from edema can occur anytime within the first 24 hours following a major burn injury. Early intubation is encouraged because obstruction can occur rapidly, making intubation of the patient in respiratory distress difficult and risky. Intubated patients are heavily sedated and sometimes chemically paralyzed to provide both patient comfort and decrease the risk of extubation. Management of the ventilated patient is described in Chapter 3 .

Airway management during the fluid resuscitation stage should begin with a thorough respiratory status assessment followed by continual monitoring. An initial chest x-ray should be taken to assess for signs of atelectasis, pneumonia, and edema. If the patient is intubated, the endotracheal tube placement should be noted. Arterial blood gases and vital signs should be obtained throughout the resuscitation phase of treatment and ventilatory adjustments made as needed. Other nursing interventions include elevating the head of the bed and turning the patient, if possible; frequent suctioning; and administering humidified oxygen as ordered. Patients with burns to the neck, chest, and abdomen should be evaluated frequently for the development of compartment syndrome that can lead to ineffective breathing patterns. Compartment syndrome will be discussed further in this chapter.

Inhalation Injury. The patient with head and neck burns or who was burned in an enclosed space should be further evaluated for signs and symptoms of inhalation injury. Bronchoscopy may be performed. During the resuscitation phase, manifestations of inhalation injury include high carboxyhemoglobin levels, upper airway obstruction, and pneumonitis. Nursing interventions, in addition to those described earlier, include monitoring of carboxyhemoglobin levels and observing for signs of respiratory distress associated with airway obstruction (increased respiratory rate, tachycardia, increased work of breathing, wheezing/hoarseness). If the patient was not initially intubated, the nurse should be prepared for emergent intubation. The placement and security of endotracheal tubes should be continually assessed as burn edema waxes and wanes.

Carbon Monoxide. Although all components of fire smoke are capable of damaging airway mucosa, CO can cause additional damage because it interferes with oxygen delivery. CO has a stronger affinity for hemoglobin than does oxygen, and it displaces oxygen as it binds to the hemoglobin, thus impairing oxygen transport and tissue oxygenation. The primary targets of CO poisoning are the heart

and brain. The level of CO bound to hemoglobin in the blood determines the symptoms. Elevated carboxyhemoglobin levels are associated with the following symptoms:

0% to 5%: Normal value, no symptoms

10% to 20%: Headache, nausea, dizziness, lightheadedness

20% to 40%: Confusion, headache, tachycardia, angina, hypertension, dysrhythmias

40% to 60%: Syncope, hypotension, seizures, and coma

Greater than 60%: Ventricular dysrhythmias and death

The rate at which CO is excreted by the lungs is related to the fraction of inspired oxygen in the air the person is breathing. If a patient is breathing room air, CO will have a half-life of 3 to 4 hours. However, if the patient is breathing 100% oxygen, the half-life of CO is reduced to 30 to 40 minutes. Therefore, patients with suspected CO poisoning are provided with high-flow oxygen (90% to 100%). Many burn centers recommend the use of hyperbaric oxygen therapy (2.5 atm) if the patient has pronounced changes in his level of consciousness or if his levels of carboxyhemoglobin are high. When hyperbaric oxygen is utilized, the nurse must monitor the patient for the development of tension pneumothorax, tympanic membrane rupture, and sinus or ear pain.

Fluid Resuscitation

Burns involving greater than 20% TBSA require fluid resuscitation to counteract the intravascular fluid losses resulting from increased capillary permeability. Patients should begin receiving fluids as soon as possible to avoid the development of hypovolemic shock. Research has shown that patients who do not receive fluids within 2 hours of injury have complicated resuscitation and increased mortality (Latenser, 2009). The goal of fluid resuscitation is to perfuse the organs; however, optimal fluid resuscitation is a balance between under resuscitation, which can result in decreased perfusion, kidney failure, and organ dysfunction and over resuscitation, which can increase burn edema, elevate compartment pressures, and lead to ARDS and multiple organ dysfunction (Pham, Cancio, & Gibran, 2008).

Due to increased capillary permeability and subsequent intravascular volume deficits, fluid replacement therapy should be started in adults and children with burns greater than 20% TBSA. Intravenous (IV) access should be established through a non-burned peripheral site using a crystalloid solution. Although a number of resuscitation formulas exists, the ABA's Burn Consensus formula (2 to 4 ml/kg body weight/%TBSA) is the most common formula used during the first 24 hours post burn (Pham, Cancio & Gibran, 2008). Because of limited glucose stores, maintenance fluids should be administered to

children in addition to the calculated amount indicated by the Burn Consensus Formula. Patients with full thickness burns, inhalation injury, or whose resuscitation was delayed may require additional resuscitation fluids.

The amount of fluid administered is dependent on body and burn size. Several formulas are used to fluid resuscitate burn patients (Warden, 2012). The most commonly used formula is the Parkland Formula that recommends 4 mL per kg body weight per % TBSA administered during the first 24 hours following a burn injury. Half of the total resuscitation volume is given in the first 8 hours. An example of how to calculate the Parkland Formula for an adult burn patient is shown in Table 17-2. Pediatric patients require more fluids per body weight. Pediatric formulas often follow the Parkland Formula but add basal fluid requirements, which are an additional 1500 cc for each m² of body surface area (BSA). Some burn centers also add basal fluid requirements to the Parkland Formula when resuscitating adult burn patients. Recent studies have found that many burn patients receive fluid volumes exceeding recommendations, a phenomena called “fluid creep,” that can contribute to burn edema (Warden, 2012). Because fluid resuscitation volumes are calculated for the first 24 hours post-burn, patients transferred from great distances to a burn center must have resuscitation fluid rates adjusted to account for the total amount of fluids administered prior to admission.

The goal of resuscitation is for the patient to maintain adequate urine output on a maintenance rate of fluids. The fluid maintenance volume is determined by the patient’s recommended fluid intake (basal fluid requirement) plus evaporative water loss. Maintenance fluid calculations take into account the patient’s BSA and the % TBSA burned. The following formula is used to calculate the daily maintenance fluid rates following successful resuscitation for adult burn patients: $(1,500 \text{ mL/m}^2) + [(25 + \% \text{ TBSA burn}) \times \text{m}^2 \times 24] = \text{total maintenance fluid (mL) to be given over 24 hours}$ (Warden, 2012). An example of how to calculate maintenance fluids for an adult burn patient is shown in Table 17-2.

Crystalloid fluids are most commonly used for burn resuscitation. Crystalloid fluids are electrolyte-based fluids that diffuse easily from the bloodstream. Lactated Ringer’s solution is the fluid of choice for burn resuscitation because it is slightly hypotonic, treating both intravascular volume losses and extracellular sodium losses. Hypertonic saline is occasionally used for burn resuscitation because it draws water back into the intravascular space by osmosis. Several studies have suggested that hypertonic saline results in hypernatremia and subsequent acute renal failure.

Colloids, such as albumin, plasma, or dextran, have been used to replenish the plasma proteins lost by the administration of high volumes of crystalloids during burn resuscitation (Pham et al., 2008). Colloids given after the

Table 17-2 Calculating Fluid Needs for an Adult Patient

Formulas: Parkland Formula (for resuscitation)	
Maintenance Formula: (Basal fluid requirement + Evaporative water loss)	
Example of How to Calculate Fluid Rates for an Adult Burn Patient	
Patient: 71-year-old male burned in a house explosion	Weight: 86 kg Height: 188 cm TBSA: 26% BSA ^a : 2.1 m ²
Resuscitation Fluids (First 24 hours)	
Parkland Formula + Basal requirements^b	Calculation
Parkland Formula (24-hour total)	4 mL × 86 kg × 26% TBSA = 8,944 mL
Basal requirements (24-hour total)	1,550 mL × 2.1 m ² = 3,150 mL
24-hour fluid total	8,944 + 3,150 = 12,094 mL
Fluid administration rate	
1st 8 hours (½ of 24-hour total)	6,047 mL/8 hrs = 756 mL/hr
2nd and 3rd 8 hours (½ of 24-hour total)	6047 mL/16 hrs = 378 mL/hr
Maintenance Fluids	
Maintenance Formula (24-hour total)	(1,500 × 2.1 m ²) + [25 + 26% TBSA] × 2.1 m ² × 24 hr] = 5,720 mL
Fluid administration rate	5720 mL/24 hr = 240 mL/hr

Source: From Mosteller, R. D. (1987, October 22). Simplified calculation of body surface area (Letter). *New England Journal of Medicine*, 317(17), 1098.

^aBSA can be calculated with a BSA calculator (many are available on the Internet) or by using a formula. One common formula is the Mosteller formula: $\text{BSA (m}^2\text{)} = (\text{Height(cm)} \times \text{Weight(kg)})/3,600)^{1/2}$.

^bNot all burn centers add basal requirements to the Parkland Formula when resuscitating burn patients. The nurse should be familiar with the practice of the burn center treating the patient.

first 12 to 24 hours post-burn may decrease overall fluid requirements and edema formation (Pham et al., 2008). Nonetheless, the use of colloids remains controversial; a recent Cochrane review of the literature states that there is no evidence that resuscitation with colloids reduces the risk of death in burn, trauma, or surgical patients (Perel & Roberts, 2007). Colloids may be beneficial in reducing complications associated with excess fluid resuscitation (Saffle, 2007). Antioxidant therapy, such as administration of ascorbic acid (vitamin C), may decrease overall fluid requirements following burn injury (Pham et al., 2008).

Monitoring of Resuscitation. Nursing roles during resuscitation include administering fluids and continually monitoring and assessing the patient’s status. Hourly urine

output measurement is the gold standard by which burn resuscitation is monitored. A urinary output of 0.5–1.0 mL/kg/hr in adults and 1.0–1.5 mL/kg/hr in children is recommended by the ABA practice guidelines to maintain adequate renal perfusion during resuscitation. Fluid type and rates are adjusted hourly based on urine output. Depending on the protocol used, fluid rates are gradually decreased as urine output is maintained. Burn resuscitation is considered successful when the patient has adequate urine output after 2 hours of fluids administered at maintenance rate. Because fluid resuscitation is often managed by burn care nurses, fluid resuscitation protocols are increasingly used to guide monitoring and management (Fahlstrom, Boyle, & Makic, 2013; Latenser, 2009; Saffle, 2007). Protocols may prevent over resuscitation, or fluid creep, by identifying patients with increased resuscitation needs early so that physicians can order appropriate adjustments in the rate and type of fluids (Latenser, 2009).

Vital signs are also monitored. Acceptable parameters include a heart rate less than 120 beats per minute, a blood pressure that is normal to slightly hypertensive, and clear lung sounds. Arterial blood gases are useful indicators of the patient's status during resuscitation. Elevated base deficit and increased lactate levels occur when systemic perfusion is compromised (Ahrns, 2004). Base deficit and lactate levels can remain elevated in burn patients considered adequately resuscitated based on urine output measurements, and mortality is increased in patients whose levels do not correct over time (Ahrns, 2004; Pham et al., 2008). Whether base excess and lactate levels should be used to determine resuscitation endpoints remains controversial.

Invasive cardiac output monitoring is sometimes used in patients with massive tissue edema, elderly patients, or patients who are not responding to standard treatment. A pulmonary artery wedge pressure less than 18 mmHg is an indicator that resuscitation is adequate, but this measurement should always be used in adjunct to urine output and other parameters. Noninvasive hemodynamic monitoring methods, such as transesophageal echocardiography (TEE) or arterial pressure waveform analysis, are increasingly used to monitor cardiac output in burn patients who do not respond to fluid resuscitation or who are hemodynamically unstable.

Electrolyte status should be monitored closely during burn resuscitation. Burn shock can result in hyper- or hypokalemia. Hyperkalemia can occur during the initial fluid shift when potassium leaks out of damaged cells into the vasculature. Correcting metabolic acidosis will also temporarily resolve hyperkalemia. Hypokalemia usually results later from fluid loss at the burn wound or because of hemodilution from fluid resuscitation. Potassium should be replaced with careful monitoring. Hyponatremia can occur in conjunction with either hyper- or hypokalemia. Fluid

administration rates should be adjusted if fluid excess is present. Careful administration of crystalloid fluids and restriction of free water will usually correct sodium deficits.

Fluid resuscitation is considered complete when the patient's fluid rates are at maintenance levels for 2 hours with adequate urine output. At that time dextrose-containing crystalloid solutions may be used.

Abdominal Compartment Syndrome. Fluid resuscitation of patients with large burns and or inhalation injury often results in fluid creep where the total resuscitation fluid requirements greatly exceed Parkland formula calculations (Luckianow, Ellis, Governale, & Kaplan, 2012; Saffle, 2007). A complication of excess fluid resuscitation is an accumulation of abdominal fluid, resulting in high intra-abdominal pressure. Subcutaneous edema and eschar in patients with abdominal burns contribute to further increase intra-abdominal pressures (IAPs). Intra-abdominal hypertension places the burn patient at risk for developing secondary abdominal compartment syndrome. If abdominal compartment syndrome remains untreated, the risk of organ failure and death is high. The patient undergoing fluid resuscitation for major burns should be monitored for development of intra-abdominal hypertension. Bladder pressure, measured via the patient's urinary catheter, is the most common measurement of IAP. Intra-abdominal hypertension occurs when bladder pressures exceed 30 mmHg, but patients with bladder pressures as low as 15 mmHg may have intra-abdominal hypertension and be at risk for abdominal compartment syndrome (Hershberger, Hunt, Arnoldo, & Purdue, 2007; Kramer, 2012). The treatment for intra-abdominal hypertension includes decreasing crystalloid fluid rates and administering colloid fluids such as albumin (Hershberger et al., 2007). The patients with circumferential abdominal burns may require abdominal escharotomies to decrease IAP and prevent abdominal compartment syndrome.

Abdominal compartment syndrome is diagnosed by sustained intra-abdominal hypertension along with abdominal tightness, difficulty in providing adequate ventilation due to increased inspiratory pressures, and increasing oliguria in spite of fluid therapy. Abdominal compartment syndrome often requires abdominal decompression either by the insertion of a percutaneous catheter to drain the excess fluid or by laparotomy to relieve abdominal pressure. Decompression laparotomy results in an open abdomen that is covered by a dressing until the wound can be surgically closed.

COLLABORATIVE CARE

Initial management of large burn wounds involves debridement, assessment, and wound coverage. The goals of wound management at this stage are to decrease fluid

and electrolyte losses, prevent infection, and decrease the risk of developing compartment syndrome.

The initial wound management usually occurs in the patient's room. The nurse should have multiple towels, drapes, wound care solutions, and wound dressings available. Wound care can result in the patient becoming chilled, so precautions should be taken, such as warm blankets and increasing the room temperature, to maintain the patient's body temperature. The patient should be pre-medicated intravenously for pain. Wound care involves moving the patient; therefore, all lines and the endotracheal tube should be secured. Members of the burn team performing wound care wear protective gowns, masks, and gloves to decrease the risk of nosocomial infections. Dressings applied at the scene or ED should be gently removed.

Debridement of the major burn wound involves cleaning the burn wound with warm water or sterile saline and antimicrobial soaps or wound cleansers. Loose skin and debris is removed using wet washcloths or gauze. Fluid-filled blisters and skin tags are trimmed with a scissors. Hair in or around the wound should be shaved. The debrided wound is washed with warm water or saline.

Assessment of the major burn wound includes description of the body location, size, and depth of injury. Circumferential burns of the neck, chest, abdomen, and extremities should be assessed further for the development of compartment syndrome. The patient with facial burns should be assessed for corneal injuries, and ocular lubrication should be applied to prevent corneal ulceration.

The major burn wound is dressed similarly to a minor burn. Silver sulfadiazine is the most common topical agent used. Bacitracin is commonly used on facial burns. Large gauze pads or gauze wrap are used to cover the wounds and secured in place with netting. Extremity burns are wrapped circumferentially starting at the distal end of the extremity and moving proximally. Individual fingers and toes are wrapped separately. Gauze dressings on the extremities can be secured with elastic bandages. Bandages must be loose enough to allow for edema formation. Windows into the bandages are often made to give nurses access to extremity pulse points. Depending on the products used, wound care is performed once or twice daily. After the patient's wounds are dressed, the patient should be placed under warm blankets and the temperature closely monitored.

Compartment Syndrome

Circumferential burn wounds to the neck, chest, abdomen, and extremities are at risk for developing compartment syndrome. The burn eschar constricts the burned area at the same time that edema is causing subcutaneous fluid expansion. The net result is impaired circulation to the involved area. Compartment syndrome can arise rapidly

at any time during the resuscitation phase. The nurse continuously assesses areas with circumferential burns for tautness, lack of color or capillary refill, coolness, and numbness and tingling. Pulses peripheral to burned extremities are assessed. Doppler ultrasound is used to assess for blood flow in the extremities. The nurse should assess the patient with circumferential burns to the neck and torso for signs indicative of compartment syndrome including decreased lung sounds, respiratory distress, and increased ventilatory needs.

Compartment syndrome is prevented by performing an escharotomy. The physician uses a scalpel or electrocautery to cut through the eschar, which releases tension and permits blood flow to the area. Escharotomies are usually performed at the bedside. The nurse should be prepared to assist in draping and monitoring the patient during the procedure. The escharotomy incisions are packed with gauze for at least 24 hours after which the incisions can be treated similarly to the burn wound. Nursing interventions following an escharotomy include monitoring the patient for blood loss and assessing for further signs of compartment syndrome. The original escharotomy incisions may need to be extended if edema progresses and pressures continue to rise. Occasionally, pressure below the fascia develops and fasciotomies are performed.

Pain Management

The patient with a large burn injury experiences excruciating pain, particularly in areas where the burn is not full thickness. During the resuscitation phase, the burn patient is treated with intravenously administered narcotics. Continuous infusions of morphine or fentanyl are common. Narcotics are titrated according to the patient's pain responses, and boluses are given for anticipated procedures such as wound care. Anxiolytics are often prescribed prior to procedures or may be used in conjunction with IV analgesic therapy. Ventilated patients who are chemically paralyzed also require medication for pain. It is important that the nurse continually assesses the patient for pain using developmentally appropriate pain and sedation scales. Hospital- or burn unit-specific protocols for assessing and treating pain in these patients should be followed.

Acute Phase

The resuscitation phase ends within 24 to 72 hours post-burn injury. Intravenous fluid requirements decrease as the extravasation of intravascular fluids ends. Clinically, the acute phase of burn care begins when the burn patient can maintain adequate urine output while receiving only maintenance rates of intravenous fluids. Following burn resuscitation, the patient enters into a hypermetabolic state

that lasts until after the wound is healed. Characteristics of hypermetabolism are increased body temperature, tachycardia, increased caloric needs, and weight loss.

Burn care during the acute phase focuses on supporting the patient as the burn wounds heal. Support includes maintenance of respiratory and circulatory status, wound care, pain management, infection control, nutritional support, physical therapy, and psychosocial support. Every member of the burn team is actively enrolled in this phase of burn care. Nursing coordinates patient care throughout this phase, maintaining constant communication with other members of the burn team, the patient, and the patient's family.

The burn patient requires ongoing assessment of circulatory and respiratory status during the acute burn phase. Fluid and electrolyte requirements can be variable during this stage depending on surgical procedures, infections, and nutritional status. Transfusions may be required to correct for blood loss. Tachycardia and fever are common throughout this stage because of the hypermetabolic response. Ventilatory requirements gradually decrease as burn edema subsides. Frequent suctioning is required to clear the lungs. The patient with inhalation injury may require prolonged ventilation and is at increased risk for pneumonia and other pulmonary complications. The patient is extubated after gradual periods of weaning. Tracheostomies are often performed if the patient fails ventilator weaning or requires prolonged ventilatory support for other reasons.

Wound Management

Once burn resuscitation is complete, the burn team can begin aggressive wound care management. Areas of first- and second-degree burns can be managed with wound care. Deep second-degree burns can heal without surgical intervention unless the wound becomes necrotic due to impaired circulation or infection. Third-degree, or full-thickness, burns require surgical intervention to heal.

Surgical Management in the Acute Phase

Perhaps the most important progress in modern burn care is early excision and coverage of full-thickness burn injuries (Lee et al., 2012). The purpose of excision and coverage is to remove the eschar and other debris of a full-thickness burn, leaving viable tissue exposed. Morbidity is decreased if the patient with major burns undergoes excision and closure within the first week of injury. Early surgical intervention slows the inflammatory response, decreases wound colonization, and attenuates the hypermetabolic response (Lee et al., 2012; Sheridan, 2002).

The patient with major burns may require multiple excisional procedures staged over several days. Excision is performed using electrocautery or a dermatome. Burned tissue is removed in thin slices until bleeding occurs and viable dermis or subcutaneous tissue is visible. Excision can result in considerable blood loss, and blood transfusions may be necessary.

Once the burn wound is excised, it is covered with either a skin graft (autograft) or other biological or biosynthetic coverings (Table 17-3). Coverage decreases insensible water loss and allows healing to begin. The type of coverage is dependent on the amount of unburned skin the patient has to donate for skin grafting, patient status, and the preferences of the burn surgeon.

Autografting. **Autografting** is a procedure that involves removing thin slices of unburned skin from an unburned "donor" site and placing it on top of the excised burn wound. Autografts are intended to be permanent. Donor sites may be "harvested" more than once if the patient requires multiple skin grafting procedures. Donor skin can be placed directly onto the burn (sheet graft) or meshed to increase the burn surface area covered. Meshed autografts result in more scarring and are placed, when possible, on the back, buttocks, and thighs. The patient with large areas to be grafted and limited donor sites may have meshed autografts placed on more visible body areas such as the trunk, arms, and lower extremities. Sheet grafts are applied to the hands and face to decrease the risk of scarring, loss of function, and its cosmetic effect.

Biological and Biosynthetic Dressings. Biological skin coverings can be used to cover excised burn wounds when the patient does not have enough donor sites to provide autograft coverage. Cadaver skin (allograft) is often used to cover excised skin or widely meshed autograft. Tissue typing is not performed; therefore, allograft results in a



Figure 17-11 Widely meshed autograft covering excised skin.

Source: Robert Ingelhart/E+/Getty Images

Table 17-3 Wound Dressings

BIOLOGICAL WOUND DRESSINGS			
Name	Description	Type	Manufacturer
Autograft	Graft of patient's own skin. Can be full or partial thickness. Can be "reharvested" multiple times from the same donor site.	Permanent	
Allograft	Graft from human cadaver. Can be fresh or cryopreserved. Often placed over autograft. Covers large wound areas and aids in revascularization of the wound. Eventually rejected.	Temporary	
Cultured epithelial autograft (CEA)	Composed of patient's own keratinocytes cultured with mouse fibroblasts to form an epidermal autograft, and then attached to a petrolatum gauze backing. Used as a permanent skin replacement in patients with large burns. Requires a small biopsy of healthy skin. May take several weeks to grow before use on graft sites.	Permanent	Genzyme Corporation
Biosynthetic Wound Dressings			
Integra	Bilayer skin replacement system comprising dermal and epidermal layers. Collagen-based dermal layer serves as matrix for revascularization. The silicone-based epidermal layer controls moisture loss. When revascularization is adequate, the silicone layer is removed and covered with a thin layer of autograft.	Permanent	Integra Life Sciences Corporation
Alloderm	Acellular dermal matrix made from donated human skin that is placed directly on the wound bed and covered with a thin layer of allograft. Provides matrix for revascularization of the allograft.	Permanent	Life Cell Corporation
Biobrane	Biosynthetic dressing applied to second-degree burns or other partial-thickness wounds. Collagen-bound dressing that adheres firmly to the wound until epithelialization occurs.	Temporary	Smith & Nephew
Synthetic Wound Dressings			
Name	Description	Type	Manufacturer
Aquacel	Silver-impregnated dressing used on partial-thickness burns, skin grafts, and donor sites. Can be left in place for up to 14 days, reducing the need for painful dressing changes.	Temporary	Convatec
Acticoat	Antimicrobial barrier dressing consisting of three layers: absorbent core sandwiched between silver-coated adherent polyethylene net layers. Antimicrobial properties effective for 3 days. Low wound adherence decreases pain of dressing changes. Used on partial-thickness burns, skin grafts, and dermal substitutes. Moisten daily with sterile water to activate antimicrobial effect.	Temporary	Smith & Nephew
DuoDerm	Hydrocolloid dressing that adheres to moist wounds.	Temporary	Convatec
Mepilex® AG	Silver impregnated foam. Antimicrobial properties effective for up to 7 days. Wound margins are sealed but dressing does not adhere to moist wounds. Used on superficial burns and donor sites.	Temporary	Molnlycke Healthcare

temporary coverage until it sloughs from the wound bed. An allograft provides a barrier against mechanical trauma, bacterial infection, and water loss (Sheridan, 2002).

Cultured autologous epithelial cells manufactured by Genzyme are the only commercially available permanent skin covering available in the United States. The cost of autologous epithelial autograft is considerable and use is reserved for patients with very large burns and few donor sites. Specific wound care protocols are implemented when cultured epithelial autografts are used, and only experienced wound care nurses should care for these wounds.

A number of commercial biosynthetic and synthetic skin substitutes are available (Table 17-3). Permanent biosynthetic products, such as Integra and Alloderm, are composed of biological and synthetic materials and serve as a dermal replacement over which autograft is placed. Other products, such as Biobrane, Aquacel, and Acticoat,

serve as temporary protective coverings over wide-meshed autografts, donor sites, and partial-thickness burns.

Major Burn Dressings

The patient with large burns requires extensive wound care. Wound care is often conducted in a hydrotherapy room where the patient is placed on a shower table or in a hydrotherapy tank for cleansing using antimicrobial soap or wound cleansing agents. Nursing interventions include pre-medicating the patient for pain and anxiety and monitoring the patient's pain, vital signs, and temperature during the dressing change. The burn wounds can be in varying stages of healing and include wounds that have not been excised, grafted wounds, and donor sites, all of which require specific care. Each dressing change should include a visual assessment of the wounds for signs of infection, wound healing, or graft loss.

Burn wounds that have not been excised or that will heal without surgical intervention are usually treated with silver sulfadiazine or other silver-coated dressings. Facial burns are treated with antimicrobial ointments such as Bacitracin or vitamin A, D, and E cream. Burns to the ear can result in chondritis and loss of the ear cartilage. The burned ear should be treated with antimicrobial agents and protected from mechanical pressure caused by wound dressings, pillows, or endotracheal tube ties.

Vacuum-assisted closure (VAC) is frequently being used on partial-thickness wounds. A sponge connected to a tube is placed over the wound and covered with an adhesive occlusive dressing. When the tube is attached to the VAC pump, a negative pressure is created over the wound. VAC continually removes exudates and promotes granulation tissue formation.

Skin grafts require meticulous wound care and each burn center has developed protocols for graft care. In the operating room, the fresh skin graft is covered with a moist dressing such as impregnated mesh gauze or silver-impregnated dressings. The dressing is covered with bulky gauze and wrapped securely. The initial dressing may remain in place for up to 3 days if no complications such as bleeding or signs of infection are noted. Initially the grafted skin is very fragile, so mechanical forces should be avoided when cleaning and dressing the wound.

Donor sites can be very painful. Wound care and dressings are similar to minor burns. Dressings that can remain in place for several days are commonly used to decrease the pain associated with dressing changes. Donor sites heal within 10 to 14 days, at which time they can be reharvested.

Essential for Patient-Centered Care

It is essential the nurse know the nursing concerns regarding comfort and wound care following excision and grafting of a burn wound.

NURSING CARE

During the acute phase of burn care, the nurse is a vital member of the burn team and continuously interacts with team members and the patient to ensure appropriate patient care. The nurse is involved with wound management as discussed earlier. Other nursing concerns during this phase include pain management, prevention of infection, nutritional support, involvement of physical therapy, and psychosocial support. Find these and other common nursing diagnoses in the "Nursing Diagnoses" boxed feature earlier in this chapter.

Promoting Comfort

Pain is a constant for the patient with large burns. In fact, pain control becomes a more significant issue as the acute phase of burn care progresses and the wounds include

donor sites. Background pain is a result of exposed nerve endings within the burn injury and donor sites, as well as pain associated with straightening and flexing burned areas during routine movements. Background pain can be interrupted with periods of increased breakthrough pain that is difficult to relieve with pain medications. The patient experiences additional procedural pain related to surgeries, dressing changes, and physical/occupational (or just therapy) therapy sessions. As the patient becomes more alert, anticipatory pain and anxiety can become overwhelming to the point where patient progress is limited. Therefore, it is important to assess pain regularly using appropriate pain scales, especially before scheduled procedures such as dressing changes. Narcotics are the most common medications used to treat burn pain. Continuous infusion of intravenous morphine is standard of care for ventilated patients. Increasingly, sedatives, such as propofol (Diprivan) or dexmedetomidine hydrochloride (Precedex), are used in place of benzodiazepines, such as lorazepam (Ativan) or midazolam (Versed), to treat anxiety in ventilated burn patients. Once a patient is extubated and able to swallow, intravenous narcotics are transitioned to intranasal or oral forms. Nonsteroidal anti-inflammatory drugs are often used in conjunction with narcotics. Pain is often decreased if patients are allowed to control some aspects of their pain management. Anxiety resulting from fear and loss of control can increase pain levels if not treated. Anxiolytics are commonly administered in conjunction with pain medications. Donor site itching contributes to patient discomfort and should be treated with oral medications and topical emollients.

Pain should be continually monitored during the acute burn phase. Treatment should be individualized and take into account patient history, burn size, anticipated procedures, and patient coping strategies. Nursing interventions include providing adequate background and procedural pain and anxiety control and assisting the patient with nonpharmacological pain-reducing therapies such as distraction or guided imagery. Nurses can help alleviate anxiety by providing the patient with information about procedures and encouraging participation in wound care and physical therapy.

Preventing and Managing Infection

Infections are responsible for approximately 75% of deaths following burn injury (Sharma, 2007). Burn patients are at risk for developing infections secondary to altered skin integrity, respiratory endothelium damage from inhalation injury, and alterations in the immune response. The presence of blood and urinary catheters and prolonged intubation contributes further to the risk of infection.

Staphylococcus aureus and *Pseudomonas aeruginosa* are the most frequently isolated organisms from patients

in burn units (Sharma, 2007). Fungal and antibiotic-resistant organisms can result in life-threatening infections. Infections arise from endogenous sources, including normal flora of the skin, upper respiratory tract, and gastrointestinal tract. Exogenous sources from the hospital environment are responsible for the majority of infections. The most common mode of transmission of exogenous organisms is via hospital personnel or contaminated equipment.

The ABA has published guidelines to define infections and sepsis in burn patients (Greenhalgh et al., 2007). Sepsis is defined as “a change in the burn patient that triggers the concern for infection” and is the point at which antibiotics are started and the search for the cause of infection initiated. Causes of infection include organisms from the respiratory tract (pneumonia), bloodstream or catheter, urinary tract, or burn wound. Clinical signs of a burn wound infection include changes in wound appearance such as erythema, early separation of burn eschar, or progression of the wound depth. Wound infections are diagnosed by objective findings from wound biopsies or swabs and tissue histology. Antibiotic sensitivities are performed to guide the pharmacological treatment. Infected wounds are sometimes treated with surgical excision to remove the infected tissue.

Infections are a serious complication for burn patients and contribute to increased mortality and morbidity. Therefore, infection control practices are very important, especially when treating patients with large burns or who are at increased risk due to age, weakened immune systems, or chronic disease. As in any critically ill hospitalized patient population, the prevention and control of nosocomial infections is a primary nursing concern. Multidrug resistant (MDR) bacteria, fungal, and *Clostridium difficile* infections in patients who have had prolonged exposure to antibiotics for treatment of burn infections are associated with higher morbidity and mortality (Ballard et al., 2008; Crabtree et al., 2011; Keen et al., 2010). The complications of these serious nosocomial infections and the difficulty in effectively treating them warrant the implementation of stringent infection control practices in hospital settings where patients with large burns are treated (Crabtree et al., 2011; Ravat et al., 2011).

Essential for Safety

A burn unit has a patient with MRSA. It is essential the nurses in the unit know what infection precautions (policies/procedures) should be taken to protect this patient and other patients in the burn unit.

Providing Nutrition

During the acute phase of burn care, the patient is in a hypermetabolic state and total energy needs can be as much as 100 times normal. Early and aggressive nutritional

support within 24 to 48 hours of burn injury is required to promote wound healing and decrease the risk of sepsis. The goal of nutritional support is to meet the patient's caloric needs and maintain a positive nitrogen balance.

Nutritional support can be provided orally if the burn patient is able to eat. However, the patient with a large burn usually requires enteral nutritional support in order to meet caloric requirements. Enteral feeds can be delivered to the stomach or the small intestine. Postpyloric feeding decreases the risk of aspiration. The burn patient receiving enteral feeds should have a nasogastric tube placed and feeding should begin slowly to assess the patient's tolerance. Enteral nutritional support protects bowel mucosal integrity and prevents stress ulcer formation. Recent research supports initiating enteral nutritional support even during the first 24 hours of injury when fluid resuscitation is ongoing (Mosier et al., 2011).

Nutrition requirements are based on the patient's age and burn size. A number of formulas are used to estimate daily caloric needs, including the Curreri and Harris-Benedict formulas (Saffle, Graves & Cochran, 2012). Formulas vary widely and no single formula has been shown to correlate well with actual energy expenditure. In addition, burn patients' caloric needs fluctuate widely during the acute burn phase. Formulas are used as a guide to estimate caloric needs, but must be adjusted according to measurements of the patient's nutritional and clinical status.

Enteral nutrition must contain complete nutrition for the burn patient. A variety of commercially available nutritional products are available. The main source of calories is from carbohydrates. The increased caloric needs put the burn patient at risk for hyperglycemia. Tight glucose monitoring and control is important, even in patients with no history of glucose intolerance. Protein catabolism is a result of burn hypermetabolism. Enteral nutrition must be high protein to prevent catabolism. The current protein recommendation is 1.5 to 2.0 grams protein per kilogram body weight per day (Saffle et al., 2012). Enteral nutrition contains a small quantity of fat to prevent essential fatty acid deficiencies. Micronutrients are often included in enteral nutrition. Vitamins A, C, D, iron, zinc, and selenium are considered beneficial to the burn patient. The amino acids glutamine and arginine are commonly added supplements because of their positive roles in immune function and wound healing (Saffle et al., 2012).

Patients with large burns may develop hyperglycemia and require intensive insulin therapy. Recent research in intensive care and burn patients stress the importance of tight glycemic control (Ballian, Rabiee, Andersen, Elahi & Gibson, 2010; Mann, Mora, Pidcoke, Wolf, & Wade, 2009; Gibson et al., 2009). Nurses often are responsible for maintaining target glucose ranges from 80 to 110 mg/dL. Many burn units have implemented nurse-driven intensive insulin therapy protocols.

Essential for Evidence-Based Practice

A 55-year-old, mildly obese male patient with a 45% TBSA burn has a morning blood sugar level of 145 mg/dL. It is essential that the nurse know the current evidence based practice guidelines concerning the treatment of hyperglycemia in obese burn patients.

A patient with a large burn injury often experiences dysphagia as a result of prolonged intubation. Speech

therapists assess the patient's ability to swallow without aspiration before the patient is allowed to take liquids or food by mouth. As the patient is able to tolerate oral feeding, enteral feeds are gradually decreased until oral intake matches caloric needs. Daily calorie counts help determine when the patient no longer requires enteral nutritional support. Because hypermetabolism lasts well past healing of the burn injury, the burn patient must be encouraged to eat high-caloric, high-protein diets supplemented with vitamins and minerals in order to meet their nutritional needs.

Commonly Used Medications

Topical Burn Medications

Silver Sulfadiazene (Silvadene, SSD, Thermazene)

Desired Effect: Used for both partial- and full-thickness burns, it has broad-spectrum bactericidal effects against many gram-negative and gram-positive bacteria. Application is painless, but the drug does not penetrate eschar.

Nursing Responsibilities:

- Monitoring the patient's white blood count
- Identifying any signs of infection
- Monitoring for the development of a rash and discontinuing the medication if one develops. Do not apply to facial burns.

Side and/or Toxic Effects: Some patients develop transient leucopenia, which usually improves over the course of therapy. A few patients develop hypersensitivity.

Mafenide Acetate (Sulfamylon)

Desired Effect: Used for partial- and full-thickness burns, it is an antimicrobial cream or solution containing mafenide (which is related to the sulfonamides). It has broad-spectrum bacteriostatic action against many gram-positive and gram-negative bacteria. It does penetrate eschar, it is absorbed systemically, and application can be painful.

Nursing Responsibilities:

- Monitoring for signs of fungal infection because no fungal coverage is provided
- Assessing and treating pain associated with dressing changes
- Monitoring for acid-base imbalances—especially metabolic acidosis
- Assessing the patient's respiratory function
- Discontinuing use if a rash develops

Side and/or Toxic Effects: Patients may develop metabolic acidosis if they are unable to hyperventilate to compensate for the metabolic acidosis that the drug induces. Patients may develop a hypersensitivity reaction manifested by a rash on their unburned skin.

Bacitracin

Desired Effect: Applied to superficial and facial burns that are left open to the environment, it is effective against gram-negative organisms and application is painless.

Nursing Responsibilities:

- Monitoring for signs and symptoms of hypersensitivity and discontinuing use if a rash develops
- Monitoring for signs of infection
- Reapplying frequently to prevent the wound from becoming dry; washing face prior to applying new application to prevent accumulation of old application festering in pores.
- Controlling the risk of infection in patients with large burns is an important part of nursing care and should include the following goals:
 - Practicing standard precautions when working with all burn patients
 - Prevention of transmission of organisms to patients or personnel. Strict hand-washing protocols should be implicated for personnel and visitors entering patient rooms. Some burn centers require that anyone coming in contact with patients with large burns must wear gloves, masks and protective gowns. All equipment should be cleaned prior to patient contact.
 - Aggressive management of the burn wound. Aseptic technique should be followed. Wound care should progress from sites at most risk of infection to sites at least risk, and care should be taken to not contaminate the burn wound with normal flora from the bowel. The wound should be monitored for signs and symptoms of infections and treated appropriately. The nurse should be familiar with the actions of topical and systemic antibiotics.
- Close monitoring of signs and symptoms of infection. Bacterial cultures should be ordered per hospital protocol and systemic antibiotics prescribed appropriately. Infected lines should be removed or replaced.

Nutritional status is assessed in several ways (Saffle et al., 2012). Changes in body weight over time can indicate whether a patient is receiving enough calories to maintain body weight. Body weight is not an accurate day-to-day predictor of nutritional status because of edema. Indirect calorimetry, which measures resting energy expenditures, is the gold standard for measuring nutritional status (Prelack, Dylewski, & Sheridan, 2007). When done weekly, indirect calorimetry shows changes in clinical status over time and allows for caloric intake to be adjusted accordingly. Indirect calorimetry is not available in many hospitals. Therefore, nitrogen balance is widely used to assess nutritional status. Twenty-four-hour urine samples are collected at least weekly to determine urea nitrogen levels and calculate nitrogen balance. A positive nitrogen balance indicates that the patient is taking in more protein than he is using.

Enteral feeding is not tolerated in patients with a number of conditions, including ileus, duodenal ulcers, or pancreatitis. Total parenteral nutrition (TPN) is administered to patients who cannot tolerate enteral feeds. TPN should be used judiciously in burn patients because it has been associated with increased infection rates in trauma patients and increased mortality in burn patients (Saffle et al., 2012).

Fostering Patient and Family Centered Care

A burn injury is a frightening and traumatic event for the patient regardless of severity. The patient's preinjury cognitive status, psychological health, coping skills, and social support networks all contribute to the way the patient responds psychologically to the burn injury (Attioe & Pounds-Cornish, 2015; Blakeney, Rosenberg, Rosenberg, & Faber, 2008). The nurse assesses preinjury psychological status upon admission, obtaining information from the patient and the family. Information about how the patient has handled stressful events in the past is helpful in planning care. The patient with a large burn initially may not be aware of the extent of injury. As the patient becomes more alert, the patient becomes cognizant of the fact that a life-changing, if not life-threatening, event has occurred. This realization can bring feelings of fear that contribute to the pain and anxiety caused by the burn injury. The nurse works to alleviate these feelings by providing the patient information about the plan of care and allowing the patient and family some control in care decisions. The patient's room should be made as comfortable as possible. Placing favorite objects in the room and playing the patient's favorite music can help orient the patient and decrease anxiety.

Psychosocial Support

The traumatic nature of burns, and the physical changes that result, often requires psychosocial adjustments for

patients and families. The patient with a large burn injury may use strategies such as dissociation, disintegration, and depersonalization to cope with the burn injury (Blakeney et al., 2008). The nurse must be aware of the coping strategies the patient and family are using and work with them to adopt strategies that are conducive to healing. It is not uncommon for the burn patient to experience feelings of hopelessness, despair, and depression as body image changes and functional limitations become apparent. Antidepressants are often prescribed to treat depression. The nurse helps the patient and family adapt and set realistic goals toward which to work.

The psychosocial aspects of burn care are often physically and emotionally difficult for the nurse. As the patient recovers, dependence on nursing staff can increase. Hopelessness and despair can cause the patient to act out in anger or to withdraw in tears. The nurse must be compassionate yet firm in setting boundaries with the patient. Most importantly, the nurse should encourage the patient to verbalize feelings followed by reassurance that the patient's feelings are normal after a burn injury. The nurse should avoid personal judgment of the patient's actions. The burn team supports the patient, family, and each other as the patient adapts psychologically.

Post-traumatic stress (PTSD) is an anxiety disorder that arises after a traumatic event. Symptoms of PTSD include flashbacks (reliving the event over and over), extreme emotional lability, and withdrawal from society. PTSD can occur following even a minor burn injury, particularly if the injury event was perceived as life threatening. Extreme or unmanaged pain has also been associated with the development of PTSD in burn patients. PTSD symptoms can occur soon after injury or years later. PTSD is predictive of poorer burn outcomes including decreased social functioning and quality of life (Corry, Klick, & Fauerbach, 2010). Therefore, it is important that the burn team assesses for signs of PTSD during the acute and rehabilitation phases of burn treatment and provide appropriate referrals for treatment.

Most patients do recover from the emotional trauma of a burn injury and return to productive fulfilling lives. The nurse provides the patient with resources to help complete this recovery, including further psychological evaluation and therapy and burn survivor networks. Burn survivor groups include support groups led by psychosocial burn team members at most burn centers and burn survivor networks. Often the first burn survivor a burn patient meets is him- or herself. Networking with other burn survivors can alleviate feelings of isolation. Numerous online and national burn survivor networks exist. The Phoenix Society is one such network that provides many psychosocial resources for burn survivors and their family.

Reflect On

Burn patients often worry about reintegration into society and the stares and questions people will have about their injuries. What are some ways you can think of to prepare them? What are some community, national, and online resources that can be helpful?

Supporting the Family

Following a major burn, the nurse provides immediate comfort and support to the patient's family as they see their loved one for the first time. The first visit is frightening for family members, as the patient may be heavily sedated, on a ventilator, and so edematous as to be unrecognizable. The nurse should work with other members of the burn team to educate the family to the burn unit layout, the sounds and smells, and the need for infection-control practices. A child-life specialist, if available, can prepare small children to see their family member, using dolls and role-playing. Once the family is at the patient's bedside, the nurse educates the family to the lines and machines attached to the patient. The family is encouraged to talk to and touch the patient. The nurse must be prepared to answer the family's questions about the patient's status and prognosis in a way that instills hope without being misleading. The nurse is often responsible for introducing family members to other members of the burn team and ensuring the family comprehends the plan of care for the patient.

Once the burn patient enters the acute phase of burn care, the nurse works with the family to provide emotional support to the patient. It is natural for family members to go through periods of hopelessness even as the patient improves. The nurse helps the family develop coping strategies and connects the family with support services as needed. The nurse should be aware of issues specific to the family such as financial concerns, family dynamics, and the ability to cope. Discord among family members is not uncommon. Hospital security policies are put in place if the nurse perceives that the safety of hospital staff or the patient is threatened by family members or visitors.

Many burn centers have support groups for family members. Families should be encouraged to attend sessions even if the patient is not stable enough to attend. Communication with other family members helps validate feelings. Burn unit social workers or trained volunteers facilitate support group dialogue and answer questions about the burn healing process.

COLLABORATIVE CARE

Psychosocial Support

A major burn injury can result in a psychological crisis for the patient and family. Fear, loss of control, and

disruption of the family structure contribute to the psychological devastation. Often the burn patient or family members have mental health histories or addiction problems that are exacerbated by the trauma of burn injury. The nurse works with members of the burn team, including social workers, psychologists, and child-life specialists, to provide psychological support to the patient and family.

Physical/Occupational Therapy

Physical therapy is an important part of the acute phase of burn care. The nurse works closely with physical therapists to prevent pressure wounds, promote wound healing, and prevent the formation of scars and joint contractures. The patient with a major burn injury is often immobile for periods secondary to chemical paralysis or sedation. The immobilized patient is at risk for developing pressure wounds. The nurse continually assesses and repositions the patient. In the immobilized patient, physical therapy is limited to range of motion.

The burn patient with burns over joints can develop contractures as the burn wound heals that impede mobility. The nurse works with the physical or occupational therapist to properly position the patient and apply splints that prevent the formation of joint contractures. As the burn wounds heal, the physical or occupational therapist assesses the need for pressure garments, custom-made elastic garments that apply uniform pressure over the wound. Pressure garments, if worn correctly, reduce scarring and prevent the formation of hypertrophic scars. The patient may wear pressure garments for up to 2 years after sustaining a major burn injury.

The physical therapist ambulates the burn patient as soon as the patient is stable. As the patient becomes stronger, physical therapy exercises to increase strength and improve function are implemented. Therapy sessions can be very painful for the patient, and the burn nurse works with the therapist to ensure the patient is properly medicated for pain and anxiety prior to each session.

Rehabilitation Phase

The rehabilitation phase of burn care is a collaborative process and begins when the patient is no longer acutely ill and most of the burn wounds are covered. The rehabilitation phase begins in the hospital and continues post discharge as the patient regains function and recovers emotionally. For the patient with large burns, the rehabilitation phase may continue for several years during which the patient wears pressure garments, continues physical and occupational therapy, and has reconstructive surgeries.

NURSING ACTIONS

Burn patients with very large burns are now surviving. Restoring quality of life and appearance have become important measures of successful burn care. Nursing care during the resuscitation phase can help the burn patient achieve optimal recovery. The primary nursing intervention during this phase is to support the patient's physical and emotional healing. This includes preparing the patient and family for discharge and ensuring that the patient is connected to resources in his or her home community to facilitate continued healing.

During the rehabilitation phase, physical healing focuses primarily on wound healing. There are additional factors that the nurse should address with the patient and family during this phase. Because altered immune function is sustained throughout the healing phase, the burn patients may be susceptible to infections and colds. Appropriate precautions should be taken, including avoiding unnecessary exposure to people with colds or infections and maintaining up-to-date immunizations. The recovering burn patient should be educated about signs and symptoms of infection and should seek prompt medical treatment. Hypermetabolism is sustained for 9 to 12 months following burn injury (Williams, Herndon & Jeschke, 2009). The patient's weight should be monitored closely throughout the rehabilitation phase. The patient may still require supplemental enteral nutrition or high-caloric nutritional supplements to maintain a positive energy balance. Large burn injuries have been associated with bone density losses, and the patient may be at increased risk for osteoporosis and pathologic fractures (Evans, 2012). Disturbances in thermoregulation continue throughout the rehabilitation phase, and the patient may experience heat intolerance.

Wound and Scar Management

The rehabilitation phase of burn care begins when the patient's wounds are primarily covered. Wound management consists of final closure of the burn wound, assessing for signs of infection, and scar management. The patient and family are taught wound care prior to discharge from the hospital. Post discharge the patient is followed as an outpatient regularly for the duration of the rehabilitation phase.

During the rehabilitation phase, the burn wound becomes a scar. The burn scar continues to develop for up to 2 years post injury. Proper wound and scar management helps the burn scar become lighter in color and more pliable. The patient with large burns is at risk for developing a number of scar-related complications during this time. The nurse works with physical and occupational therapists to assess scar development and develop appropriate treatment modalities to prevent complications. The patient's

tolerance for exercise is measured over time. Active exercise is encouraged to maintain or rebuild muscle mass, decrease edema, and reduce the risk of deep vein thrombosis.

Hypertrophic Scarring

Second-degree burn wounds extend into the dermis, synthesizing collagen to form a scar. In areas of the wound with granulation tissue, collagen deposition can be disorganized, resulting in the development of a hypertrophic scar that is erythematous and raised. Hypertrophic scarring occurs most often in nongrafted burns that took longer than 2 weeks to heal or along the edges of skin grafts. Children and individuals with darker skin pigments are more susceptible. Hypertrophic scarring can be prevented or greatly reduced by the application of pressure garments. Pressure over time helps the collagen bundles align in a more parallel fashion and reduces hypervascularity and cellularity. Keeping the skin soft and pliable with ointments and massage also helps reduce the hypertrophic scar.

The burn patient may be required to wear pressure garments continuously for up to 2 years following injury. Physical or occupational therapists measure the patient at different times to ensure the garments fit correctly and provide continuous pressure. The patient is educated about the importance of wearing the garments continuously during this time and how to protect fragile skin under the garments with lubrication. Pressure garments can be uncomfortable to wear, causing itching and heat intolerance. Therefore, patient compliance can be an issue. The nurse works with the burn team to encourage compliance. Over-the-counter or prescription medications to decrease itching are often prescribed.

Wound Contractures

Wound contractures occur as a result of scar formation over joints, which limit joint movement. Contractures may be prevented by exercise, body positioning, joint splinting, and pressure dressings. Prevention begins during the acute phase of burn care and continues through the rehabilitation phase. During the rehabilitation phase, the nurse works with physical and occupational therapy to ensure the patient is exercising and appropriate body positioning is maintained using splints and other devices.

Contractures are treated by applying splints to the affected area and by specific range-of-motion exercises. The splints are worn for prescribed times. An important nursing intervention is to assess the patient's compliance. Contractures that are not responding to exercise and positioning may require surgical incision of the scar tissue to "release" the contracture of a joint. The excised scar is closed by primary closure, z-plasty, or skin grafting (Huang & Yang, 2012).

Other Wound Complications

The maturing burn scar is fragile and susceptible to wound breakdown from shearing and pressure. The burn nurse assesses the patient for signs of wound breakdown and ensures wound dressings and pressure garments are fitted properly. Wound breakdown can occur after the patient is discharged from the hospital. The patient and family are provided education about protecting the wound from breakdown and how to care for open wounds that develop.

Neuropathies can develop as a result of scar formation, edema, or improper positioning of splints or dressings. The nurse assesses peripheral pulses and sensation throughout the rehabilitation phase. Symptoms of neuropathies include severe burning pain, increased or decreased sensation, abnormal sensation, and cold intolerance (Warden & Warner, 2012). The nurse works with physical and occupational therapists to position splints and dressings appropriately. Edema can be prevented by elevating edematous limbs.

Heterotopic bone ossification, the abnormal formation of bone, is a rare complication of burn injury. Heterotopic formation is more likely to occur in a joint that had an overlying deep burn and that was immobilized for a long period. Early joint mobility in the acute burn phase is important in preventing this complication. During the rehabilitation phase, the patient is assessed for range of motion. A symptom of heterotopic bone formation is the patient's reluctance to move a joint that previously had good range of motion. The patient is taught the importance of continuing range-of-motion exercises for joints near the burn injury.

Pain During the Rehabilitation Phase

Pain during the rehabilitation phase of burn care is characterized as an aching pain (Meyer, Weichman, Woodson, Jaco, & Thomas, 2012). As in the acute phase, the burn patient experiences procedural pain and background pain. Oral opioids, acetaminophen, or nonsteroidal anti-inflammatory medications provide adequate pain relief for most patients. Once the patient's pain is treated, itching and anxiety should be addressed as separate issues. Pain and anxiety medications should be tapered during the rehabilitation phase. The nurse, working with psychosocial burn team members, can use nonpharmacological therapies to decrease pain and anxiety.

Psychosocial Support

During the rehabilitation phase, the burn patient becomes aware of the extent of the burn injury and the effect on appearance, function, and activities of daily living. The patient experiences feelings of grief and loss for the "pre-burn self." Feelings of hopelessness and loss of autonomy

often result in the patient becoming angry with the burn team and family members (Blakeney et al., 2008). The burn team works with the patient to combat these feelings by setting limits, defining achievable goals, and providing as much autonomy as possible. The patient and family are provided with information about the psychological recovery from a burn injury and future disfigurement and functional abilities. Often PTSD arises after a burn patient has been discharged from the hospital. Patients and family members should be informed of the symptoms of PTSD, such as flashbacks, emotional lability, and social withdrawal, and encouraged to seek medical treatment if symptoms occur. The patient and family may benefit from attending a burn support group where they meet other burn survivors who validate their own experiences and instill hope for the future.

Reintegration into Society

The burn team begins to prepare the patient for reintegration into society prior to discharge. For the patient with a large disfiguring burn, reintegration is a daunting, anxiety-provoking step. The patient fears rejection and ridicule from acquaintances and strangers seeing the burn scars for the first time. Both the patient and family worry that they will not be able to manage activities of daily living and burn-specific care at home. The burn team works to alleviate these concerns by encouraging the patient and family to be independent prior to discharge. The patient and family are encouraged to practice interacting with others by taking short trips outside the hospital prior to discharge. The burn team may also work with the patient's community to ease social reintegration. School age children may benefit from school re-entry programs. Groups important to the patient can be provided with education about burns and be given the opportunity to ask questions about how to best assist and encourage the patient.

After discharge the burn patient faces many physical and psychosocial challenges. The nurse assesses the patient's coping abilities during outpatient clinic visits. During this time the patient may begin experiencing heightened levels of depression, anxiety, or signs of PTSD. The patient who has altered behavior may benefit from consultation with a psychiatric or mental healthcare provider.

The patient's family members must also learn to cope with the long-term consequences of the burn injury. A major burn injury often results in loss of income and changed family roles. The family may feel ill equipped to care for the patient's physical and psychosocial needs. Prior to discharge, the burn team assesses the family's ability to care for the patient at home and provides the family with references for local services that can ease the transition.

As the survivability of burn injuries increases, quality of life post-burn recovery has become an important focus of burn care. Despite the psychosocial challenges of burn injury, patients and families are quite resilient, particularly with focused and consistent psychosocial support from time of injury through reintegration to society. Patients and families, especially parents of burned children, can experience significant stress and depression for several years after the burn injury. However, most long-term burn survivors report that their quality of life is high. Burn patients with a history of psychiatric disorders or who experienced more psychological stress, including PTSD, post-burn are at risk for poorer quality of life, stressing the importance of early assessment and treatment (Rosenberg, Lawrence, Rosenberg, Fauerbach, & Glakeny, 2012). Older adults may not be able to live independently immediately following discharge from treatment for a burn injury. However, recent research suggests that the impact of a burn on the functional and psychosocial aspects of quality of life for older adults continues to improve 2 years following burn injury (Klein et al., 2011).

The majority of burn patients return to work within a year of injury, although patients with large burns may need prolonged rehabilitation and/or job retraining that slows their return to work (Mason, Esselman, Fraser, Schomer, Truitt, & Johnson, 2012; Schneider, Bassi, & Ryan, 2011). Although major burns significantly impact the lives of patients with a major burn injury, with time they can go on to lead full and rewarding lives. The nurse can help patients and families recognize that quality of life can be good and can provide them with support services to help them achieve this goal.

NURSING CARE

Nursing care of the burn patient is challenging yet rewarding. Even minor burns can require complex care in the ED or clinic setting. Nursing interventions for the patient with a minor burn injury include wound care, assessing for signs and symptoms of infection, pain management, and psychosocial evaluation.

Bariatric Considerations

Obese patients, specifically those with BMI greater than 35 kg/m², have increased hospital lengths of stay and mortality following burns compared to people of normal weight (Dries, 2009).

Post-burn, obese patients have an increase in metabolic rate similar to non-obese patients, and actual weight may be used to calculate energy expenditure (Iretton-Jones, 1997).

Overweight and obese adults older than 55 with a BMI greater than 27 are less likely to return home post-burn and are more likely to have difficulty with locomotion and transfer (Ghanem, Sen, Philp, Dziewulski, & Shelley, 2011).

Obese burn patients often require more staff to perform dressing changes and toileting. Specialized equipment such as larger beds and operating tables, hoists, and adaptations to ventilator tubing may be needed (Ghanem et al., 2011).

Care of the patient with serious burns is often delivered in specialized burn centers by a dedicated multidisciplinary burn team. The burn team comprises the following members: physicians, nurses, physical and occupational therapists, dietitians, pharmacists, psychologists, and social and child-life workers. Other disciplines, such as recreational therapists, speech therapists, and other medical specialists, are consulted as needed. Nursing members of the burn team include advance practice nurses, burn nurses, and case-managers to name a few. Most nurses working with burn patients are Advance Burn Life Support (ABLS) certified and many are certified in intensive care or pediatric specialties. The nurse caring for the burn patient is a vital member of the burn team. The nurse provides bedside care and communicates the patient's status to other members of the burn team and the family. The nurse provides continuity of care throughout the three phases of burn care. See the "Nursing Diagnoses" boxed feature for a specific list.

Burn Summary

Burns are painful injuries that involve every major body system. Even a minor burn can be a very stressful event for a patient. A major burn is often a life-threatening injury. The patient endures countless medical procedures, intense physical/occupational therapy, and much pain. In addition, the physical scarring and loss of function associated

with a large burn contribute to psychological stresses. The nurse caring for the burn patient must be aware of both the physical and psychological issues associated with burn care. Most burn injuries are minor and treated in EDs or clinics, where the nurse assesses and cares for the wound while monitoring and treating pain and anxiety.

The nurse working in a specialized burn center is part of a multidisciplinary team providing intensive care to patients with burn injuries. In this setting, the nurse cares for the burn patient through the continuum of burn care: from resuscitation to rehabilitation. Because of the broad depth

of knowledge, a nurse must have to care for a major burn, burn nursing is challenging. However, the rewards of following a patient with severe burns from intensive care to the time when the patient is functioning fully in society are many and great.

Why/Why Not?

A man fell asleep while cooking, and a pot with fat caught fire on his stove. He awoke when the smoke detectors went off and smoke had filled the room where he was sleeping. After calling 911, he stayed in the house attempting to put out the fire. The EMTs transported him to the ED for evaluation. He had soot about his face and mouth but there were no indications of respiratory difficulty when he arrived in the ED. His initial blood gases and chest x-ray were normal. While the nurse was preparing

him for discharge, the nurse noted that his respiratory rate had increased to 30, he appeared to be using accessory muscles to breathe, and he seemed to be more irritable than earlier. When the nurse asked if he is experiencing difficulty breathing, he said "No" and demanded to leave immediately. Should the nurse ask him to stay so she could reevaluate him? Why or why not?

See answers to Why/Why Not? in the Answer Section.

Case Study

Jon Lewis, a 21-year-old student at a university, was trapped in his room during a house fire. In an attempt to escape the fire, he opened the door from his room, burning his forearms, face, and chest, then turned and jumped arms first through his second-floor window. On the ground he was met by firemen who rushed him to the burn trauma center at the university hospital 5 minutes away.

Jon arrived in the ED of the burn trauma center 10 minutes post-burn.

1. Should the burning process have stopped? If not, what care is indicated to stop the process?
2. What findings would lead the burn team to suspect an inhalation injury?
3. What should the ED team do immediately if they suspect an inhalation injury?
4. How should the extent and depth of the burns on his chest, forearms, and face be assessed?
5. Why is Lactated Ringer's the preferred fluid for fluid resuscitation? How is the amount of fluid required calculated? How will the nurse know if fluid resuscitation is adequate?

On further evaluation, Jon was found to have compression injuries of his T5–T7 vertebrae and a severed muscle and tendons in his right arm.

6. How did he probably sustain these injuries?

Following initial management in the ED, Jon was transported to the burn trauma unit of the hospital. On admission to the burn trauma ICU, he was ventilated, had dressings applied to his chest, arms, and face, and was receiving 800 mL/hr of Lactated Ringer's. His vital signs were BP 184/92, heart rate 110 bpm, respirations 12, oxygen saturation 94%.

7. What additional information should the nurse obtain from the ED team?
8. What complications might the nurse anticipate?
9. What preventive strategies should the nurse initiate immediately?

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 17.1 What are the most common etiologies of burn injury for each of the following age groups?
 - A. Children
 - B. Adults
 - C. Older adults
- 17.2 What are the differences in manifestations between a partial- and a full-thickness burn?
- 17.3 What findings would indicate a possible inhalation injury?
- 17.4 What changes occur within the vasculature following a burn injury? What implications do these changes have for patient management?
- 17.5 What are the priorities of care immediately after a patient with a severe burn injury arrives at a hospital?

- 17.6 How are full-thickness burn wounds managed?
- 17.7 What are some of the psychological issues that confront burn victims?

- 17.8 Why are very young and very old people likely to have more severe burn injuries?

See answers to Chapter Review Questions in the Answer Section.

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Chapter 18

Care of the Patient with Sepsis

Kathleen Perrin, PhD, RN, CCRN

Abbreviations

APC	Activated Protein C	IL	Interleukin
DIC	Disseminated Intravascular Coagulation	MRSA	Methicillin-Resistant <i>Staphylococcus Aureus</i>
FDPs	Fibrin Degradation Products	TNF	Tumor Necrosis Factor



Learning Outcomes

Upon completion of this chapter, the learner will be able to:

1. Discuss the inflammatory/immune process resulting in sepsis.
2. Describe evidence-based prevention strategies for hospital acquired infections.
3. Analyze the significance of assessment findings in patients with SIRS and severe sepsis.
4. Evaluate the elements of the severe sepsis bundles and describe other supportive therapies.
5. Explain collaborative management and nursing care for complications of sepsis.

Introduction

Severe sepsis, the presence of infection and organ dysfunction, is a complex, costly, and frequently lethal syndrome with a hospital mortality rate that approached 29% worldwide in 2001. It is such a disturbing health problem that an international task force was mobilized to study and recommend evidence-based solutions to the problem. Infection prevention strategies and evidence-based interventions advocated by the Surviving Sepsis Campaign in 2004, and revised repeatedly since, are improving outcomes for septic patients.

Inflammatory Immune Response in Septic Shock

Severe sepsis is a complex process that is not completely understood. However, it seems the body responds to sepsis in three general ways: inflammation, thrombosis, and fibrinolysis. This process is displayed in ♦ Figure 18-1.

Pathophysiology Review

The inflammatory phase of sepsis begins in response to an infectious organism as white blood cells release

the age of the population increases, and there is increasing use of transplantation, immunosuppression, chemotherapy, and invasive devices (Mayr et al., 2014).

Outcome of Sepsis

According to Hall and colleagues (2011), only 2% of hospitalizations in 2008 were for sepsis, yet sepsis accounted for 17% of all hospital deaths. Mortality rates for sepsis are estimated to range from 23% to 46% depending on the patient's age, underlying condition, and site of infection. Sepsis is more likely to be lethal in the older adult. In Angus and colleagues' 2001 study, the overall hospital mortality rate was 28.6%, but it approached 38% in those 85 years or older. The impact of age on outcome was most apparent for older adults who had been healthy prior to the onset of sepsis.

The majority of septic patients receive care in an intensive care unit (ICU). In fact, sepsis is the leading cause of death among critically ill patients in noncoronary intensive care units in the United States (Mayr et al., 2014). The care of a septic patient in a critical care unit is expensive. Per patient costs for the condition are highest in infants, non-survivors, ICU or surgical patients, and patients with dysfunctions of four or more organ systems. However, because sepsis affects the elderly and those with chronic health problems disproportionately, more than half of all annual costs in the United States—\$8.7 billion—were spent on care of septic patients 65 years or older.

International Sepsis Campaign

The Surviving Sepsis Campaign established in 2004 is a joint effort of the European Society of Intensive Care Medicine, the International Sepsis Forum, and the Society of Critical Care Medicine. The focus of the campaign is improving the diagnosis, survival, and management of patients with sepsis by addressing the challenges associated with it. The campaign is responsible for developing and revising international guidelines for management of severe sepsis and septic shock (Surviving Sepsis Campaign, 2015).

Predisposing Factors and Causes

There are a variety of factors that increase an individual's risk for developing sepsis. These factors include:

- Advanced age (older than 85 years)
- Compromised immune status such as:
 - Alcohol misuse
 - Cancer
 - Immunosuppressant therapy
 - Positive human immunodeficiency virus (HIV) status
- Chronic illness such as:
 - Diabetes mellitus
 - Chronic renal failure
 - Hepatitis
- Invasive interventions such as central line catheters and endotracheal intubation
- Malnutrition
- Surgical/invasive procedures, especially intra-abdominal surgery
- Sequential infections treated with broad-spectrum antibiotics

Mayr, Yende, and Angus (2014) determined that the most common sites of infection for severe sepsis varied by gender. The sites and frequency were:

Respiratory	42% men	36% women
Bacteremia (site unspecified)	21% men	34% women
Abdominal	9% men	10% women
Genitourinary	10% men,	9% women
Wound/soft tissues	9% men	8% women
Other sources	9% men	10% women

Among older patients, the urinary tract was the most common source of infection.

Prevention of Hospital-Acquired Infections

Hospital-acquired infections are associated with increased lengths of hospital stay as well as increased costs. At the very least the development of an infection can be uncomfortable for the patient and worrisome for the patient and family. Catheter-related bloodborne infections and surgical site infections increase the likelihood that the patient will develop severe sepsis or multisystem organ dysfunction and spend time in intensive care. Other hospital-acquired infections such as ventilator-associated pneumonia are linked to an increased risk of patient death. Therefore, nurses and other healthcare providers need to be vigilant about utilizing evidence-based practices to prevent nosocomial infections.

Ventilator-Associated Pneumonia Prevention Protocols

Ventilator-associated pneumonia (VAP) or infection-related ventilator complication is an airway infection that develops

Gerontological Considerations

Rates for severe sepsis rise sharply with age. There are 0.2 case per 1,000 population among children, but 26.2 cases per 1,000 adults over the age of 85.

Mortality is also greater in older individuals. The mortality rate is 10% in children and 38% for adults over the age of 85.

Among older adults, the most common source of infection is the urinary tract but pneumonia is also common and fever in patients with pneumonia may be blunted or absent (Umberger, Callen, & Brown, 2015).

Multiple organ dysfunction syndrome (MODS) is more likely to occur following severe sepsis in older patients when they have pre-existing organ dysfunctions.

Cognitive impairment may be a presenting manifestation of sepsis in the older adult. Such impairment accompanied by functional disability is more likely to continue in elders who survive sepsis, with declines persisting for years. "The magnitude of these new deficits was large, likely resulting in a pivotal downturn in patients' ability to live independently" (Iwashyna, Ely, Smith, & Langa, 2010, p. 1787).

More than half of the \$8.7 billion in annual cost for severe sepsis is spent on care of septic patients 65 years or older.

at least 48 hours after a patient is intubated. It is identified when a patient:

- Has a period of stability on a ventilator, then has increasing requirements for FiO_2 and/or PEEP
- On or after day 3 develops a fever higher than 38°C or WBC greater than $12,000 \text{ cells/mm}^3$
- Has purulent secretions or positive culture of lavage or lung tissue (Magill et al., 2013)

Preventing pneumonia associated with ventilator use is of special importance because VAP was the leading cause of death from hospital-acquired infections. Deaths from VAP exceeded the rate of deaths from central line infections, severe sepsis, or respiratory tract infections in the nonintubated patient. In addition to making it more likely that the patient will die, development of a VAP prolongs the patient's illness and hospitalization. It results in more time spent on the ventilator, more days in the ICU, and more time in the hospital after discharge from the ICU.

In 2007, VAP developed in between 9% and 27% of intubated patients (Seckel, 2007). Bacteria usually enter the respiratory tract after colonizing the oral cavity, sinuses, trachea, or stomach. The normal flora of the oral cavity changes within 48 hours of hospital admission to more virulent flora, whereas stomach contents become colonized with bacteria when the pH of the stomach is altered for stress ulcer prophylaxis. VAP may develop when there is aspiration of these contents or when bacterial colonies develop on the endotracheal tube (Seckel, 2007). Augustyn (2007) divided the onset of VAP into two types: early and late onset. Early-onset VAP occurs between 48 and 96 hours after intubation and is associated with antibiotic-susceptible organisms. Late-onset VAP occurs after 96 hours and is usually associated with antibiotic-resistant organisms.

Two crucial steps in reducing the morbidity and mortality due to ventilator-associated pneumonia are application of evidence-based practices to reduce the occurrence of pneumonia and early recognition and treatment of VAP should it develop. The Institute for Healthcare Improvement (IHI) recommends that every healthcare institution implement the "Ventilator Bundle, a series of interventions related to ventilator care that, when implemented together, will achieve significantly better outcomes than when implemented individually." Since the institution of the Ventilator Bundle, VAP rates have decreased dramatically, and in fact many ICUs have reported consecutive months without a VAP (IHI, 2012b). The key components of the IHI (2012b) ventilator bundle are:

- Elevation of the head of the bed
- Daily sedation interruption and daily assessment of readiness to extubate
- Peptic ulcer disease prophylaxis
- Deep venous thrombosis prophylaxis
- Daily oral care with chlorhexidine

Additional interventions recommended to prevent VAP include:

- Washing hands before and after contact with each patient (Tolentino-DelosReyes, Ruppert, & Shyang-Yun, 2007)
- Use of a continuous-suctioning endotracheal tube (Damas et al., 2015)
- Change of ventilator circuit no more often than every 48 hours (Tolentino-DelosReyes et al., 2007) or no longer routinely changing the patient's ventilator circuit (Seckel, 2007)

Elevation of the Head of the Bed

Elevation of the head of the bed (HOB) to between 30 and 45 degrees is recommended partially because it most likely reduces the risk of aspiration of oropharyngeal contents but also because it may increase patient ventilation. IHI (2012b) guidelines advocate that it be standard procedure for all patients (especially for those receiving gastric feedings) to have their HOB elevated to at least 30 degrees unless there is a compelling reason for them to have their HOB lower, such as hemodynamic instability or the presence of an intra-aortic balloon pump. Because the constant elevation of the HOB may result in increased lumbosacral pressure, nurses need to be vigilant about assessing skin breakdown and providing pressure relief for patients.

Daily Sedation Interruption/Assessment of Readiness to Extubate

Daily sedation interruption and assessment of readiness to extubate are recommended because reducing the amount of time on a ventilator decreases the patient's potential for exposure to VAP. There is concern among some nurses about the safety of interrupting sedation; therefore, most institutions have specific policies outlining for whom and when sedation interruption should be attempted. The evidence demonstrates that appropriate use of daily interruption of sedation to determine readiness to wean decreases patients' time on a ventilator, length of stay in ICU, and length of hospital stay without increasing unplanned extubations (Aragón & Sole, 2006).

Peptic Ulcer Prophylaxis

Peptic ulcer prophylaxis is recommended for ventilated patients for two reasons. First, there is a higher incidence of stress ulceration in critically ill patients. Next, critically ill intubated patients lack the ability to protect their airway, and the effects of aspirating acidic contents may be worse than aspirating material with a higher pH. Increasing the pH of gastric contents with H₂-receptor inhibitors may protect against a greater pulmonary inflammatory response if gastrointestinal contents are aspirated. The Surviving Sepsis Campaign Guidelines concluded that histamine receptor inhibitors are the preferred agents because they are more effective than sucralfate. However, recently there have been calls for further study about the benefits of both proton pump inhibitors and H₂ antagonists in stress ulcer prophylaxis because they have been implicated in the development of nosocomial pneumonia (Miano et al., 2009) and *Clostridium difficile*-associated diarrhea.

Deep Vein Thrombosis Prophylaxis

The IHI (2012b) recommends deep venous thrombosis (DVT) prophylaxis because when it is applied as part of

"a package of interventions for ventilator care, the rate of pneumonia decreases precipitously." The IHI believes that interventions to decrease DVTs remain excellent practice in the general care of ventilated patients. When determining which type of prophylaxis is appropriate, the IHI recommends balancing the increase in the risk of bleeding that may occur if anticoagulants are used with the fact that sequential compression devices are often not consistently applied to patients. For example, the devices may be removed for extensive periods when patients are away from the ICU for procedures. For additional information about DVT prophylaxis, see the safety initiative "Reducing Surgical Complications: Venous Thromboembolism (VTE) Prophylaxis" in Chapter 10.

Daily Oral Care with Chlorhexidine

The provision of routine oral care with products containing chlorhexidine decreases the incidence of VAP (Ames, 2011). The American Association of Critical-Care Nurses (AACN, 2006) noted in a practice alert that although several studies that included oral care as part of a VAP prevention bundle showed a decrease in the incidence of VAP, the specific contribution of oral care to the reduction in VAP was unknown. Most ICUs have specific policies for the delivery of oral care, usually requiring tooth brushing at least twice a day with a chlorhexidine product and differentiating comprehensive oral care (including tooth brushing) from oral cavity moisturizing.

The American Dental Society recommends toothbrushing at least twice a day to limit dental plaque, a source of bacteria, whereas chlorhexidine mouth care appears to prevent the overgrowth of more virulent bacteria in the mouth. Munro and others (2009) found that mouth care with chlorhexidine twice daily reduced the incidence of VAP while tooth brushing alone did not. A clinical trial is underway to determine the optimal mouth care (Munro, 2014). While awaiting definitive studies, AACN (2010) recommends that nurses ensure their ICU has a written oral care policy and that they adhere to that policy. See ♦ Figure 18-2 for an example of an oral care cleansing system.

Additional Approaches to Preventing VAP

IHI (2012b) specifically states that the VAP bundle is not comprehensive, and additional therapies may be beneficial and should be provided. Among them, appropriate hand hygiene has been demonstrated to reduce infection rates (Aitken, Blot, Marshall, Moloney-Harmon, & Johnson, 2011). Hand hygiene includes antisepsis prior to patient care, gloves for patient care, and antisepsis after care. The Centers for Disease Control and Prevention (CDC) recommends alcohol-based products for hand hygiene in the interest of improving compliance. Nevertheless, nurses'



Figure 18-2 An oral cleansing and suctioning system.

self-reported compliance for hand hygiene only reaches 84%, while in observational studies compliance ranges between 50% and 60%. When asked, critical care nurses state that time factors are the major reason they are unable to clean their hands between each patient (De Wandel, Maes, Labeau, Vereecken, & Blot, 2010).

Reflect On

Why do you believe the incidence of hand washing among nurses is most likely only 50% to 60%? What could be done to improve this situation?

The use of endotracheal tubes with subglottic suctioning results in significant reductions in VAP and antibiotic use (Damas et al., 2015). (♦ Figure 18-3.)

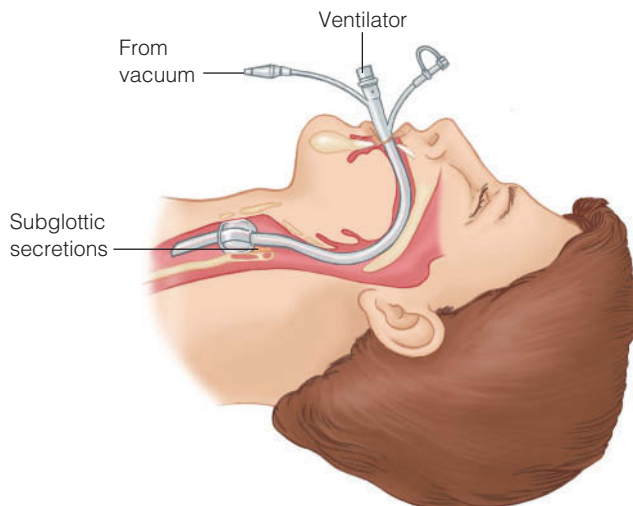


Figure 18-3 Endotracheal tube with evacuation lumen.

The use of an endotracheal tube (ET) with a dorsal lumen above the cuff (CASS tube) permits continuous suction and prevents accumulation of tracheal secretions in the subglottic area. If a CASS tube is not inserted when the patient is initially intubated, the tube should not be changed to a CASS tube due to the increased likelihood of infection from reintubation.

In the past, ventilator circuits were changed at regular intervals, ranging from every 8 hours to every 2 days. Current recommendations are to change ventilator circuits no more than every 48 hours (Tolentino-DelosReyes et al., 2007) or not to routinely change the patient's ventilator circuit (Seckel, 2007). Maselli and Restrepo (2011) recommend that ventilator circuits be changed only when they are visibly soiled or are not functioning properly.

Essential for Evidence-Based Practice

Providing mouth care with chlorhexidine to critically ill ventilated patients is a documented way to decrease the incidence of VAP.

Preventing Central Line Associated Bloodstream Infections

Central venous catheters (CVCs) are being used more frequently in patient care. The IHI estimates that 48% of ICU patients have CVCs, accounting for 15 million central venous catheter-days every year. Compared to peripherally inserted catheters, CVCs carry a significantly greater risk of infection, accounting for nearly 90% of catheter-related bloodstream infections. Todd noted that in 2006, approximately 80,000 such infections occurred annually in ICUs. Mortality attributable to these infections was between 4% and 20%, so between 500 and 4,000 patients in the United States died in 2006 due to CVC-related bloodstream infections. In addition, nosocomial bloodstream infections prolonged hospitalization by a mean of 7 days and increased the cost of a patient's care during a hospitalization by \$3,700 to \$29,000.

The IHI (2012a) has been advocating the use of the central line bundle, a group of evidence-based interventions for patients with intravascular catheters that, when implemented together, result in better outcomes than when implemented individually. The key components of the IHI central line bundle are:

- Hand hygiene: Hands should be cleansed prior to contact with patients with waterless cleaning solution or, if visibly soiled, with soap and water.
- Maximal barrier precautions upon insertion: Full barrier precautions means that cap, mask, sterile gown, sterile gloves, and full-body drapes are required during insertion of CVCs.

- Chlorhexidine skin antisepsis: Chlorhexidine 2% in alcohol 70% has been demonstrated to be the most effective agent for cleansing the skin. Cleansing should include a friction scrub for 30 seconds followed by 2 minutes of drying the site (Aragon & Sole, 2006).
- Optimal catheter site selection, with subclavian vein as the preferred site for non-tunneled catheters site: Selection is determined based on individual patient issues. However, when possible, use of the subclavian vein has been associated with fewer bloodstream infections.
- Daily review of line necessity with prompt removal of unnecessary lines.

The IHI (2012a) acknowledges that their bundle is not a comprehensive list of strategies to limit central line associated infections. The American Hospital Association, the Joint Commission, and other health organizations have an extensive listing of strategies for prevention of central line associated infections available on-line (Marshall et al., 2014). They recommend strategies that are not part of the IHI bundle. Among the strategies they recommend: indications for CVC line insertion should be posted on each unit, and CVCs should be inserted only when indicated by healthcare personnel who have been appropriately educated about insertion.

Once the CVC has been established, hand hygiene and sterile gloves are required for dressing changes. There is insufficient evidence to support the recommendation for a specific type of dressing. However, frequency of dressing change is dependent on dressing type. It is recommended that gauze dressings be changed every 2 days or as needed, whereas transparent dressings may be left in place for up

to 7 days. There is evidence to suggest that IV tubings should be changed no more frequently than every 72 hours but should be replaced by at least 96 hours. However, tubings should be changed more frequently when blood products, propofol, or lipids are administered (AACN, 2005).

Essential for Evidence-Based Practice

The nurse should review the necessity of every IV on a daily basis.

Surgical Site Care

Surgical site infections are common in hospitalized patients, occurring in 2% to 5% of clean extra-abdominal surgeries and up to 20% of intra-abdominal surgeries. Patients who develop surgical site infections are 60% more likely to spend time in ICU, average 7 days more in the hospital, and have twice the mortality of patients who do not develop infections. Two methods that have been advocated for prevention of surgical site infections are the administration of an appropriate antibiotic and the avoidance of shaving of the surgical site. Clear evidence exists that providing an appropriate antibiotic no more than 1 hour prior to the surgical incision and discontinuing the antibiotic 24 hours after surgery results in fewer surgical site infections (Bratzler et al., 2013). There is no convincing evidence to recommend in favor of hair removal from the surgical site prior to surgery. However, strong evidence exists that if hair removal is considered necessary, shaving should not be performed. Instead, hair removal with a depilatory or an electric clipper is recommended immediately before surgery (Bolton, 2006).

Bariatric Considerations

- In sepsis, the inflammatory response of the host is as important as the infectiousness of the agent. There is growing evidence that obesity may be associated with chronic inflammatory disease resulting in exaggerated inflammatory and pro-thrombotic responses in obese patients.
- Factors associated with an increased risk of infection in the obese patient include increased duration of mechanical ventilation, difficulty in obtaining vascular access, more frequent and longer use of indwelling urinary drainage catheters, and an increased incidence of skin breakdown.
- The distribution, metabolism, and elimination of antimicrobial agents are altered in the obese person. Unfortunately, "little is known about the dosing of most antibiotics in patients with morbid obesity" (King & Velmahos, 2010, p. S480). Moreover, Mayr, Yende, and Angus (2014) believe that obese patients

receive lower doses of antimicrobial agents (adjusted for weight) compared to normal weight patients.

- Obesity has been linked to an increase in surgical site infections possibly because pre-operative antibiotic doses have not been appropriately adjusted for the patients' weight (Bratzler et al., 2013). Obese patients with sepsis have higher morbidity, longer hospital stays, and higher healthcare costs than leaner individuals.

Intensive care units in the United States were quick to respond to the challenge of preventing CVC infections by instituting the central line bundle. The effectiveness of the bundle can be seen directly in the 58% reduction in the number of CVC infections in ICUs between 2001 and 2009 (CDC, 2011). According to the CDC, this represented an estimated savings of 6000 lives and \$414 million.

Preventing Urinary Tract Infections

Indwelling urinary catheters are inserted in approximately four million Americans each year and are used in the majority of critical care patients. Not surprisingly, because they are used so frequently, catheter-associated urinary tract infection (CAUTI) is one of the most common nosocomial infections in ICUs worldwide. Nosocomial bacteriuria develops in 25% to 40% of patients requiring a urinary catheter for more than 7 days. Although CAUTI may result in a moderate increase in cost and length of stay, it is not usually associated with an increase in mortality.

Risks for Development of Catheter-Associated Urinary Tract Infections

Laupland et al. (2005) identified the following factors and noted the associated relative risks for development of a CAUTI. The factors marked with an asterisk (*) were identified by Gray (2010) as especially significant for patients in critical care.

Prolonged catheterization (longer than 6 days)	5.1–6.8
Female gender	2.5–3.7
Catheter insertion outside operating room	2.0–5.3

Other active sites of infection*	2.3–2.4
Diabetes	2.2–2.3
Malnutrition*	2.4
Azotemia (creatinine greater than 2.0 mg/dL)*	2.1–2.6
Monitoring of urine output	2.0
Drainage tube below level of bladder and above collection bag	1.9
Antimicrobial drug therapy*	0.1–0.4

Guidelines for Prevention of Catheter-Associated Urinary Tract Infections

Guidelines to prevent UTIs were developed by the CDC in 1981 and updated in 2009. Although there have been few randomized controlled clinical trials demonstrating the effectiveness of these practices (sometimes called a CAUTI bundle), their use has been associated with reductions in CAUTI rates of 19% in cardiothoracic ICUs and 67% in medical-surgical ICUs (Lo et al., 2014). The recommended practices include:

- **Avoiding unnecessary catheterizations:** The use of indwelling urinary catheters should be limited to circumstances when they are absolutely necessary (such as for monitoring urine output accurately in a critically

Safety Initiatives

Reduction of Methicillin-Resistant *Staphylococcus Aureus* Infections

PURPOSE: To significantly reduce methicillin-resistant *Staphylococcus aureus* (MRSA) infection by reliably implementing five components of care recommended in the IHI guidelines

RATIONALE: Healthcare-associated infections remain a major cause of disability, death, and increased cost of healthcare. Treatment of infections has become more complex because of the alarming rise in antibiotic resistance. Infections caused by MRSA are particularly problematic because their incidence has increased in the past decade and they are often lethal. Moreover, MRSA accounted for 63% of *S. aureus* infections acquired in ICUs in the United States in 2004.

HIGHLIGHTS OF RECOMMENDATIONS: In order to reduce MRSA infections, the IHI recommends that organizations start with these five components of care:

1. Hand hygiene: Adequate hand hygiene involves wearing gloves during patient care and properly washing hands after the gloves are removed.
2. Decontamination of the environment and equipment: Because MRSA can survive on table tops, bed rails, and computers, these surfaces must be adequately decontaminated.

3. Active surveillance cultures: These cultures identify colonized patients and allow for implementation of isolation and adequate decontamination of surfaces to prevent transmission. Although the nares are the most common site for colonization, 15% to 25% of patients harbor MRSA in their axillae, 30% to 40% in their perineum, and 40% on their hands and arms.
4. Contact precautions for infected and colonized patients: Contact precautions interrupt the important modes of MRSA transmission. If nurses wear gloves during contact with patients with MRSA or while in the immediate environment of patients with MRSA, the likelihood that the nurses will contaminate their hands is decreased. Wearing a gown can protect nurses from contaminating their clothing when caring for colonized or infected patients.
5. Device bundles: These include ventilator and central line bundles.

Source: Institute for Healthcare Improvement. (2008). *Getting started kit: Reduce methicillin-resistant Staphylococcus aureus (MRSA) infection how-to guide*. Cambridge, MA: Author. Available at www.ihl.org

ill patient, perioperative or end of life urinary drainage, and promotion of wound healing [Gray, 2010]). Catheters should be removed as soon as they are no longer needed. Studies have indicated that as many as 40% of catheters are inserted unnecessarily or left in place too long.

- **Insertion using aseptic technique:** Catheters should be inserted by trained healthcare professionals using aseptic technique, including sterile gloves, a fenestrated sterile drape, and an antiseptic, such as 10% povidone-iodine or 2% aqueous chlorhexidine.
- **Maintaining a closed drainage system:** After a catheter is inserted, strict maintenance of a closed drainage system can limit the overall risk of CAUTI to less than 25% for up to 2 weeks.
- **Ensuring dependent drainage and an unobstructed urine flow:** The collection tubing and bag should always remain below the level of the patient's bladder with the drainage tubing always above the level of the collection bag to ensure constant drainage.
- **Minimizing manipulation of urine collection system:** Caregivers should wash their hands before touching the drainage system. The catheter and the drainage system should be manipulated as little as possible, and urine output should be monitored hourly only when clearly indicated by the patient's condition.

Essential for Evidence-Based Practice

The occurrence of CAUTIs can be reduced simply by instituting a daily evaluation of the continuing necessity of each urinary catheter, and removing it when there is no longer an approved indication for leaving it in place (Elpern et al., 2009).

Potential future solutions include the use of antimicrobial urinary catheters. Johnson, Kuslowski, and Wilt (2006) state that there is fair quality evidence that antimicrobial urinary catheters can prevent bacteriuria in hospitalized patients during short-term catheterization. However, the effectiveness of the catheters depends on the antimicrobial coating, and the cost implications remain undefined.

Assessment of the Septic Patient

Early recognition of the septic patient is key to the initiation of early therapy and better patient outcomes. In hospitals reporting a systematic approach to identifying sepsis, screening for sepsis was usually prompted by a deterioration in laboratory values, admission to ICU, or changes in vital signs.

Nurses (rather than physicians or medical residents) usually identified the concerns and were responsible for screening patients and identifying the presence of sepsis (Durthaler, Ernst, & Johnston, 2009). Therefore, it is essential that critical care nurses recognize the manifestations of SIRS, sepsis, and septic shock and assess their patients for them (Aitken et al., 2011). In addition, Aitken and colleagues recommend the institution of daily screening for sepsis in ICU patients. This screening can be accomplished with a simple screening tool, including the patient's white blood count, respiratory rate, temperature, and heart rate (Moore et al., 2014).

Recognition of the Patient with Systemic Inflammatory Response Syndrome

The first phase of the septic process, the **systemic inflammatory response syndrome (SIRS)**, has been described as the presence of two or more of the following:

- Temperature greater than 38°C (100.4°F) or less than 36°C (96.8°F)
- Pulse greater than 90 beats per minute
- Respiratory rate greater than 20 breaths/minute or PaCO₂ less than 32 torr
- White blood cells (WBCs) greater than 12,000/mm³ or less than 4000/mm³

An international consensus panel reaffirmed the definition of SIRS in 2001 and used the presence of 2 or more of SIRS criteria as the indication of sepsis. Recently, there has been dissatisfaction with using SIRS criteria to identify sepsis because the criteria are nonspecific. Although mortality increases as patients meet more of the SIRS criteria, requiring 2 or more to diagnose sepsis failed to identify 12% of patients who eventually developed severe sepsis and died (Kaulonen, Bailey, Pilcher, Cooper, & Bellomo, 2015). Therefore, new definitions for severe sepsis and septic shock not based on SIRS criteria were established in 2016.

Essential for Evidence-Based Practice

The route by which a patient's temperature is obtained is very important. Axillary measurements consistently underestimate core body temperature while tympanic measurements are very inaccurate. Oral and temporal artery measurements more closely reflect core body temperatures of critically ill patients but should not be substituted for core temperatures when precision is required (Lawson et al., 2007).

Recognition of the Patient with Sepsis

Sepsis is now defined as "life threatening organ dysfunction caused by a dysregulated host response to infection"

(Singer et al., 2016, p. 801). In addition to infection, patients who have sepsis have organ dysfunction that can be identified by using the Sequential Organ Failure Assessment (SOFA). Included in calculating the SOFA are:

- Respiratory function: evaluated by $\text{PaO}_2/\text{FiO}_2$ mmHg
- Cardiovascular function: evaluated by MAP and whether or not the patient is receiving vasopressors
- Coagulation: evaluated by platelet count
- Renal function: evaluated by urine output and creatinine level
- Central nervous function: evaluated by Glasgow Coma Scale
- Liver function: evaluated by Bilirubin

If the above information is not available, Singer et al. (2016) recommend the Quick SOFA (qSOFA) be used to identify patients with infection who are likely to have poor outcomes. The presence of any two of the following three clinical variables: altered mental status, systolic blood pressure of 100 mmHg or less, and respiratory rate 22/min or greater, are nearly as predictive of severe sepsis as the full SOFA score.

The patient with sepsis should also be assessed for localizing signs of infection. Some of the common origins of infection for hospitalized patients are displayed in ♦ Figure 18-4.

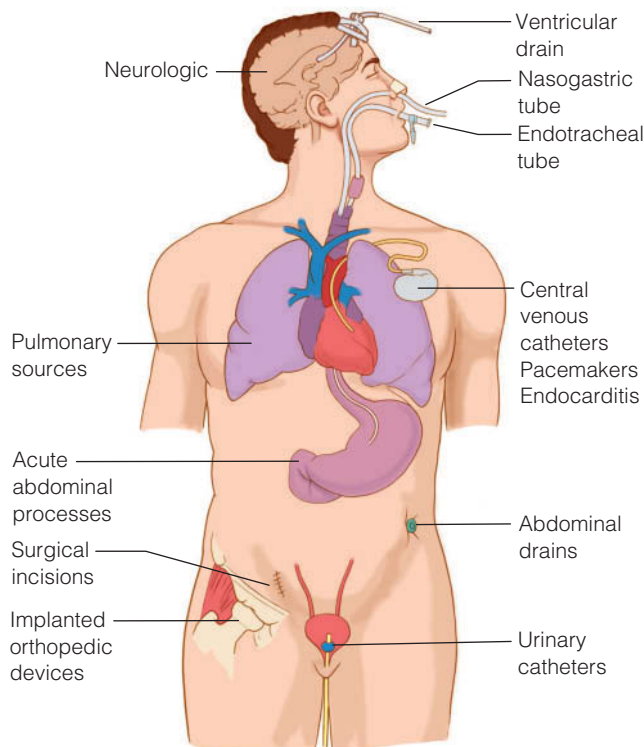


Figure 18-4 Possible sources of infection in the hospitalized patient with sepsis.

In order to assist in identifying the source of infection the nurse should consider the following:

- Does the patient have a cough or a change in the character of his sputum that might indicate a pulmonary source of infection?
- Is the patient experiencing nausea, vomiting, or diarrhea? Does he have abdominal pain or rebound tenderness that might indicate an abdominal source of infection? Is the patient tube fed?
- Has the patient been experiencing dysuria, frequency of urination, or pain at the costovertebral angle that could be indicative of a renal source of infection?
- Does the patient have a new murmur or a change in a murmur, possibly indicating endocarditis?
- Does the patient have a headache or a change in mental status or neurological findings that might indicate a neurological focus of infection?
- Does the patient have redness, tenderness, or drainage at the sites of any implants, catheters, or medical devices? (Henker & Carlson, 2007)

Recognition of the Patient with Severe Sepsis and Septic Shock

Severe sepsis occurs when dysfunction of two or more organ systems develops in response to infection and hypoperfusion. It can develop either slowly or suddenly but once it has developed, patient outcomes are not as likely to be positive. Even modest organ dysfunction, noted when the patient is first believed to have an infection, is associated with a 10% increase in mortality (Singer et al., 2016). Therefore, the ICU nurse caring for a patient with an infection needs to be vigilant about monitoring the patient for the development of any of the following signs of organ dysfunction:

- Acute lung injury indicated by tachypnea and/or hypoxemia
- Coagulation abnormalities or thrombocytopenia
- Neurological dysfunction indicated by a sudden change in mental status with possible confusion or psychosis
- Renal dysfunction evidenced by urine output less than 0.5 mL/Kg/hr for at least 2 hours (or less than 500 mL/day) and a serum creatinine greater than 2.0 mg/dL
- Liver dysfunction indicated by jaundice, coagulopathy, and increased bilirubin levels,
- Gastrointestinal injury indicated by stress ulceration, ileus (absent bowel sounds), and malabsorption
- Cardiac dysfunction indicated by tachycardia, dysrhythmias, or hypotension (MAP less than 70) especially if accompanied by lactic acidosis

Septic shock, the most severe form of sepsis, is defined as the subset of sepsis in which profound circulatory, cellular and metabolic abnormalities are associated with a greater risk of death than with sepsis alone. Patients with septic shock require vasopressors to maintain a MAP greater than 65 mmHg and have serum lactates greater than 2 mmol/L in the absence of hypovolemia (Singer et al., 2016).

Diagnostic Criteria

A number of laboratory abnormalities are also correlated with the presence of sepsis. These include:

- Leukocytosis (white count greater than 12,000/mm³) and a shift to the left of neutrophils. However, a peaking fever appears to be more sensitive than leukocytosis to predict bacteremia.
- Thrombocytopenia (platelet count less than 100,000)
- Hyperglycemia (plasma glucose greater than 140 mg/dL)
- Plasma C reactive protein level greater than 2 SD above normal
- Coagulation abnormalities (international ratio [INR] greater than 1.5 or an activated partial thromboplastin time (aPTT) greater than 60s)
- Hyperbilirubinemia (plasma bilirubin greater than 4 mg/dL)
- Increased lactate (greater than 2 mmol/L)
- Procalcitonin (PCT) level of greater than 2 ng/mL indicates that sepsis is probable while a PCT greater than 10 ng/mL indicates a high likelihood of sepsis or septic shock. When the PCT drops by 50% or more during treatment, the patient is more likely to survive. (Karlsson et al., 2010)

In addition, computerized tomography (CT) scans may be used to identify possible abscesses, and chest or abdominal x-rays may reveal infectious processes.

Collaborative Care of the Patient With Severe Sepsis or Septic Shock

Collaborative management of sepsis is based on the understanding that interventions to optimize the patients' hemodynamic status instituted early in the septic process reduce mortality. It appears that accurate diagnosis and definitive management of severe sepsis and septic shock must occur within the first 6 hours of the onset of symptoms for the best outcomes (Otero et al., 2006). This makes sense because the purpose of the interventions is to prevent further organ

dysfunction. If interventions are delayed until after organ and cellular dysfunction develop, then the strategies designed to provide the cells with more oxygen are unlikely to be helpful. Therefore, many healthcare institutions have developed guidelines based on the Surviving Sepsis Campaign Guidelines to ensure patients with severe sepsis are identified early and are managed appropriately.

Severe Sepsis Bundle

When a patient is identified with severe sepsis, septic shock, and/or lactate greater than 4 mmol/L (36 mg/dL), there are four evidence-based practices that the Surviving Sepsis Campaign states should be completed within the first 3 hours. For the best outcomes, the appropriate care should be instituted in the emergency department (ED) even before the patient is transferred to an ICU (Dellinger et al., 2013). Aitken et al. (2011) recommend that nurses be empowered via standing orders to initiate the sepsis bundle immediately for hypotension unresponsive to volume repletion and elevated lactate so that there is no delay in the management of sepsis.

Bundle Element 1: Measure Serum Lactate

A serum lactate greater than 2 mmol/L is often present in patients with severe sepsis or septic shock and may develop in response to anaerobic metabolism due to hypoperfusion. Although elevated lactate levels may result from other causes such as decreased clearance by the liver, obtaining a serum lactate is essential to identifying inadequate tissue perfusion in patients who are not yet hypotensive but who are at risk for septic shock. Identification of septic patients prior to the deterioration of their vital signs allows for initiation of the severe sepsis bundle and decreases both morbidity and mortality.

Bundle Element 2: Obtain Blood Cultures Prior to Antibiotic Administration

Of patients presenting with a clinical syndrome of severe sepsis or shock, 30% to 50% have positive blood cultures. Therefore, blood should be obtained for culture in any critically ill septic patient. The Surviving Sepsis Campaign (2012) recommends that blood cultures be obtained as soon as possible after the onset of fever and chills and prior to the administration of any antibiotic. Durthaler et al. (2009) found that 92.8% of hospitals complied with this element of the sepsis bundle, the highest for any element of either sepsis bundle. Russell (2006) recommended also culturing the patient's sputum, urine, and other fluids such as a draining wound or possibly cerebrospinal fluid. Obtaining cultures prior to antibiotic administration offers the best hope of identifying the organism that caused the patient's sepsis.

Bundle Element 3: Administer a Broad-Spectrum Antibiotic

Administration of an appropriate antimicrobial agent within the first few hours after the development of sepsis is one of the few interventions that clearly reduce mortality in sepsis (Liberman & Whelan, 2006). Each hour that the administration of an appropriate antibiotic is delayed may result in an 8% increase in mortality (Powers & Jacobi, 2006). The choice of agent depends on the suspected source of the infection and the susceptibility of the likely pathogen but also on the patient's history. After 48 to 72 hours, the multidisciplinary team reassesses the appropriateness of the antimicrobial regimen by reviewing the results of cultures and sensitivities and the clinical status of the patient. The eventual goal is to use a single narrow-spectrum antibiotic to prevent the development of resistance, to reduce toxicity, and to limit costs. Patients should begin therapy with a full loading dose of each antimicrobial. However, because patients with sepsis often have abnormal renal or hepatic function, the pharmacist should be consulted to ensure the prescribed dose results in serum concentrations that are both clinically effective and minimally toxic.

Essential for Evidence-Based Practice

Nurses can improve patient outcomes for septic patients by ensuring that prescribed antibiotics are administered as soon as possible after blood cultures are obtained.

Bundle Element 4: In the Event of Hypotension and/or Serum Lactate Greater than 4 mmol/L

The severe sepsis bundle calls for an initial fluid challenge of a minimum of 30 mL/kg of crystalloid in cases of suspected hypovolemia or actual cases of serum lactate greater than 4 mmol/L (36 g/dL). The fluid administration should be started as soon as possible in the course of septic shock.

- To deliver a fluid challenge, the nurse infuses the prescribed fluid volume (usually 500 to 1,000 mL) over approximately 30 minutes.
- The nurse anticipates that the patient's BP and heart rate will begin to normalize quickly if the challenge has been successful. An adequate response to a fluid challenge for the patient with sepsis is defined as a MAP greater than 65 with a heart rate less than 110 beats/min.
- Fluid volume requirements vary greatly among septic patients and may be difficult to predict. Therefore, the fluid challenge may be repeated if the patient does not

respond adequately. The 30 mL/kg of crystalloid merely represents the minimally acceptable initial fluid volume. If repeated fluid boluses are required, the patient's fluid status will need to be reassessed as described in Bundle Element 6.

- Nearly all septic patients will require appreciably more intake than output so the nurse recognizes that I&O alone cannot guide fluid replacement.
- The nurse is responsible for monitoring the patient closely during the fluid challenge for evidence of fluid volume overload because fluid overload during this phase is associated with poorer long-term patient outcomes. If fluid overload develops, the fluid challenge is ended. The nurse is vigilant in assessing for weight gain, generalized or pulmonary edema, and an elevated CVP while providing the fluid challenge. Boyd and colleagues (2011) found that optimal fluid replacement during the fluid challenge phase resulted in a weight gain of 3 pounds during the first 4 days and a CVP of 8 in the first 12 hours.

Severe Sepsis Bundle: To Be Completed Within Six Hours of the Time of Presentation

The resuscitation bundle includes as many as seven key interventions. The four bundles described above are to be completed within three hours. For patients with severe sepsis, the following three bundles must be accomplished within 6 hours of the time of presentation.

Bundle Element 5

In the event of persistent hypotension despite fluid resuscitation (septic shock), bundle element 5 indicates delivery of vasopressors to maintain a mean arterial pressure greater than or equal to 65 mmHg. It is important to follow the MAP rather than the systolic BP because it is more likely to reflect actual tissue perfusion. Because the vasopressor will be titrated to the MAP, a reliable BP should be continuously available. Therefore, direct monitoring of the BP through the radial artery is usually indicated. The insertion of the arterial line may be delayed until the patient has been transported to the ICU, whereas the delivery of vasopressors should be through a CVC as soon as one is available.

Although vasopressor agents are recommended, there remain concerns about their potential for inappropriate or detrimental use (Dellinger et al., 2013). When vasopressors are used in patients who have not received adequate fluid resuscitation, they produce additional vasoconstriction that may worsen organ perfusion. Even if the patient has received adequate fluid volume, there is concern that vasopressors may increase BP and perfusion to some organs at the expense

of perfusion to the intestine and kidneys. Finally, too high a BP has the potential to increase left ventricular workload to an unsustainable level and worsen heart failure in the patient with preexisting heart disease.

Essential for Evidence-Based Practice

The nurse needs to continually and carefully assess renal and cardiac functioning when vasopressors are added to the treatment regimen.

Choice of Vasopressors. The choice of vasopressor varies with the institutional protocol or the specific physician. A meta-analysis by De Backer, Aldecoa, Njimi, and Vincent (2012) suggested that norepinephrine is more appropriate than dopamine because dopamine is associated with a higher mortality and more dysrhythmias than norepinephrine when administered to septic patients. Consequently, the Surviving Sepsis Campaign recommends norepinephrine as the vasopressor of choice (Dellinger et al., 2013). Vasopressin use might be considered in patients with refractory shock despite adequate volume replacement and high-dose conventional vasopressors.

Commonly Used Medications

Norepinephrine and Vasopressin

Norepinephrine: A Potent Alpha-Adrenergic Agonist with Some Beta-Adrenergic Effects

Desired Effects: Due to its vasoconstrictive effects, norepinephrine usually results in a significant increase in MAP with little change in heart rate or cardiac output. It seems to be more effective than dopamine at reversing hypotension in septic shock patients resistant to fluid resuscitation. Initial response should begin in 1 to 2 minutes of IV administration.

Nursing Responsibilities: Norepinephrine is administered by continuous IV infusion. Usual maintenance range of the infusion is 2 to 12 mcg/min. The usual maximum dose is 25 mcg/min.

The nurse should monitor BP at least every 2 to 5 minutes after beginning a continuous infusion. However, it is preferable the BP be monitored continuously through direct measurement by an arterial line. Norepinephrine may be titrated up or down cautiously to obtain a MAP of 65. The lowest dose possible to attain the MAP should be utilized.

In addition to monitoring the MAP, the nurse will assess the heart rate, monitor pattern, and urine output continuously during administration.

If at all possible, norepinephrine should be administered through a CVC. If norepinephrine is administered through a peripheral IV line, severe vasoconstriction may result. Veins in the hands, ankles, and legs should be avoided but a large antecubital vein can be acceptable if necessary. Blanching along the vein is an indication of extravasation that can lead to severe tissue necrosis, sloughing, and gangrene.

If extravasation does occur, phentolamine (Regitine) 5 to 10 mg diluted in 10 to 15 mL of normal saline is injected liberally throughout the tissue to prevent necrosis.

Potential Side and/or Toxic Effects: Side effects are rare when used as directed. Previously there was concern that the vasoconstriction occurring with norepinephrine could

result in deterioration of renal functioning. However, this does not appear to be the case. Instead, by increasing the MAP norepinephrine appears to increase glomerular filtration and renal function. However, the nurse should be certain to monitor the urine output and serum creatinine of the patient receiving norepinephrine.

Septic shock may be accompanied by a reduced vascular response to catecholamines. Thus a patient may have had adequate volume replacement and be receiving high doses of vasopressors but still remain hypotensive. In such circumstances, a continuous infusion of low-dose arginine vasopressin might be utilized.

Vasopressin: A Synthetic Hormone, Antidiuretic, and Potent Vasoconstrictor

Desired Effects: An increase in MAP without an increase in cardiac index or heart rate should be detectable within 1 hour of the institution of a continuous infusion of vasopressin 0.04 unit/min. At this dose, there should not be a decrease in urine output.

Nursing Responsibilities: Vasopressin has been associated with a decrease in cardiac output in some studies of patients with septic shock. Therefore, the nurse should continue to observe the patient for adequate tissue perfusion and cardiac output.

End-organ perfusion, specifically gastrointestinal (GI) and liver perfusion, has been reduced in some patients in septic shock receiving vasopressin. Therefore, the nurse should routinely assess liver and GI function.

Potential Side and/or Toxic Effects: In higher doses, vasopressin has been associated with arrhythmias, bradycardia, hypertension, myocardial infarction (MI), and abdominal cramping.

Overdose can result in water intoxication.

Bundle Element 6

In the event of persistent hypotension despite fluid resuscitation (septic shock) and/or lactate greater than 4 mmol/L, bundle element 6 indicates reassessing volume status and tissue perfusion and document findings. The nurse should repeat a focused assessment of the patient's volume status including vital signs, pulmonary status, capillary refill, pulse, and skin findings to identify ongoing indications of hypoperfusion. Alternately, the nurse might review two of the following: the CVP, ScvO₂, bedside cardiovascular ultrasound, or dynamic assessment of responsiveness with passive leg raises or fluid challenge. See Chapter 5 for a detailed explanation of hemodynamic assessment.

Bundle Element 7: Re-measure Lactate if Initial Lactate Was Elevated

The mortality rate is high for patients with hypotension and serum lactate greater than 4 mmol/L since inadequate tissue perfusion is continuing. Monitoring blood pressure, indications of septic shock, and serum lactate assist the nurse to determine how well the patient is progressing. An increasing MAP and decreasing serum lactate would likely indicate patient improvement. Conversely, a decreasing MAP and increasing serum lactate would necessitate a search for another cause of decreased perfusion (such as pneumothorax or increased intra-abdominal pressure), additional interventions (perhaps other vasopressors or the addition of an inotrope such as dobutamine), and/or discussions about goals for the patient's care. For an overview of the possible progression and management of sepsis, see Visual Map 18-1.

Severe Sepsis Bundles: Other Supportive Therapies

Other supportive therapies for the patient with severe sepsis may include maintain blood glucose monitoring and maintenance, and mechanical ventilation.

Maintain Glucose Control

Following initial stabilization of patients with severe sepsis, blood glucose should be maintained at less than 180 mg/dL with a goal of approximately 150 mg/dL. Initially, blood glucose should be monitored according to protocol. If two consecutive blood glucoses are greater than 180 mg/dL, an insulin infusion may be started (Dellinger et al., 2013). The rationale and a protocol for instituting a continuous infusion of insulin are available in Chapter 14. Concerns have arisen about the cost-effectiveness of tight glycemic control as well as the potential for adverse effects, especially hypoglycemia, when it is utilized for septic patients (Martin, 2007). Sandrock and Albertson (2010) clarify that the controversy about glucose control in the septic patient is not about the need for glucose control; rather, it is about the target blood glucose for insulin therapy.

Mechanical Ventilation of Sepsis-Induced Acute Respiratory Distress Syndrome

Patients with sepsis are at an increased risk for developing acute respiratory failure, and nearly 50% of patients with severe sepsis develop acute respiratory distress syndrome (ARDS). High tidal volumes should be avoided in patients prone to the development of ARDS. Current recommendations are for "low" tidal volume (6 mL/kg/lean body weight) as a goal in conjunction with the goal of maintaining end-inspiratory plateau pressures of less than 30 cm H₂O to prevent further lung injury. The nurse should utilize a sedation assessment scale and medicate the patient with the minimal amount of sedative or pain medication necessary to attain the desired pain and sedation scores (Dellinger et al., 2013).

Reflect On

Several of the elements of the sepsis management bundle are currently undergoing reconsideration. After weighing the evidence do you think these elements should be maintained in the bundle or removed? Why?

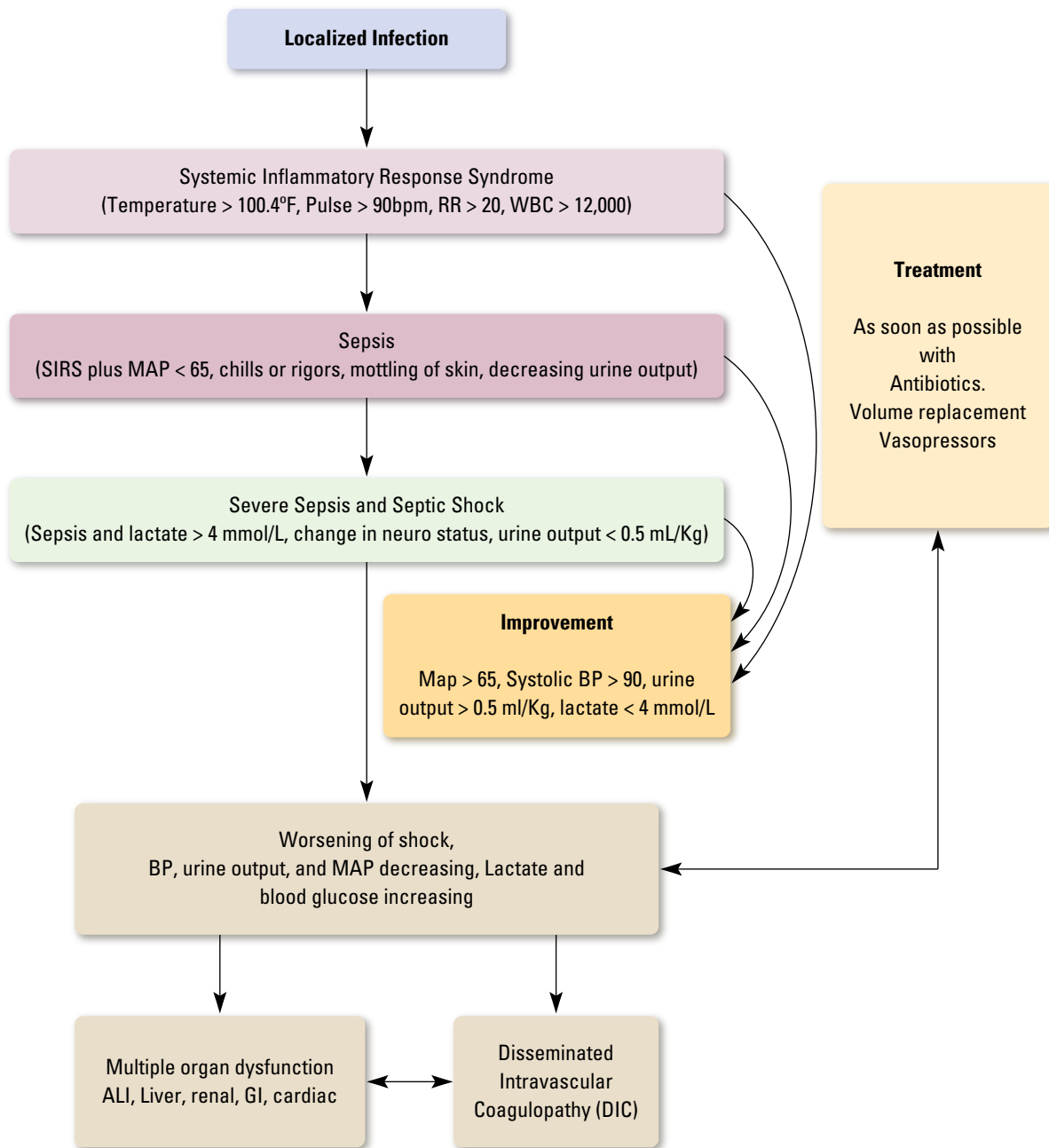
COLLABORATIVE CARE

Source control

In addition to the bundle elements, an essential collaborative intervention for management of the septic patient is source control and identification and eradication of the source of infection. As previously described, patients with sepsis should be evaluated to determine the source of the infection. In addition to identifying localizing signs of infections, reviewing the exposure history with the patient or family and performing diagnostic tests such as CT or magnetic resonance imaging (MRI) may be useful. When the source of infection is identified, it should be eliminated if possible. Source control might involve the drainage of an abscess, the debridement of infected necrotic tissue, the removal of a potentially infected device, or the definitive control of a source of ongoing microbial contamination. The nursing management of the patient is dependent on the specific site of the infection and the type of surgery performed for source control.

NURSING CARE

The patient with sepsis may experience a variety of sources of discomfort, including discomfort from medical devices and pain at the site of the infection. Management of a septic patient's pain will vary depending on the specific source of the pain. In addition, most septic patients are febrile and may experience discomfort from their fever or the efforts of healthcare providers to reduce their fever.



Visual Map 18-1 Progression of Sepsis

Enhancing Comfort

Uncomplicated fever is an important immunologic defense mechanism. In fact, an elevated body temperature in patients with acute respiratory distress syndrome has been shown to decrease mortality (Schell-Chaple, Puntillo, Matthay, & Lui, 2015). Although the benefit of fever disappears when the fever exceeds 39.4°C (about 103°F), a higher temperature does not appear to confer an increased risk of death (Taccone, Saxena, & Schortgen, 2014). Therefore, attempts are made to actively reduce a fever only in specific situations, which include:

- the fever is severely elevated,
- the patient is very uncomfortable,
- the patient has a neurological disorder,
- the patient is post cardiac arrest, or
- the patient is at risk for or is compromised from the fever.

Unfortunately, there are a number of unresolved questions about the management of fever (Taccone et al., 2014). The first is what constitutes a fever that is too high and requires treatment. Patients are usually defined as febrile when their fevers reach 38.3°C (101°F), and interventions are often instituted for fevers higher than 39°C (approximately 102°F).

Because there is disagreement about when to institute treatment for fever, critical care nurses need to observe their patients with fever to determine if the fever is resulting in cardiovascular compromise or excessive metabolic demands. Fevers are more likely to result in compromise in very old patients, neonates, neurological patients with increased intracranial pressure or seizures, and patients with cardiovascular disease.

A second question is, which method is better for controlling fever, medication or physical cooling? Commonly used interventions to reduce fever such as the use of antipyretic drugs and external cooling should be evaluated in terms of potential risks. Although antipyretics are usually instituted for fevers higher than 39°C (102°F), reducing such fevers does not necessarily improve patient outcomes (Henker & Carlson, 2007). Acetaminophen, one of the most commonly used antipyretics, is erratically absorbed when administered via nasogastric tube, often resulting in elevated drug levels in critically ill patients. Repeated therapy at recommended doses can result in drug accumulation, and hepatotoxicity has developed in some patients. Thus, some healthcare providers recommend alternating doses of acetaminophen with ibuprofen. However, such a scheme can result in the potential for excessive doses. When acetaminophen is administered every 4 hours with ibuprofen every 6 hours, not only is it possible for the recommended daily dose to be exceeded, it is unclear which antipyretic should be administered at the twelfth hour (acetaminophen or ibuprofen, or both). If acetaminophen and ibuprofen are alternated every 2 hours, the recommended number of total daily doses is exceeded. When administering antipyretics, the critical care nurse needs to be attentive to the total daily dose of acetaminophen that the patient is receiving as well as to the patient's liver function.

Henker and Carlson (2007) recommend that exogenous cooling be considered when a critically ill patient's fever exceeds 39.5°C (103°F). ICU nurses rarely use tepid sponges to cool patients because tepid sponges are unlikely to be successful and most patients find them very uncomfortable.

- More commonly an external cooling (hypothermia) blanket is utilized to cool the patient.
- Of particular concern when instituting exogenous cooling is the potential for the patient to shiver. Shivering causes an increase in the metabolic rate of 100% to 200% and increases body temperature. Thus, it should be prevented.
- In order to prevent shivering, the nurse might:
 - Use higher temperatures on the cooling blanket.
 - Keep the patient's hands and feet off the blanket.
 - Sedate and paralyze the patient. Sedating or paralyzing a patient with drugs that suppress shivering during external cooling results in a more rapid reduction of fever and reduced energy expenditure than if the patient were treated with antipyretic drugs alone (Axelrod, 2000).

- The utilization of an external cooling blanket requires continuous, accurate assessment of the patient's core temperature.
- Usually the blanket is turned off when the patient's temperature is about 1°C above the desired temperature.

Providing Nutrition

The Surviving Sepsis Campaign (Dellinger et al., 2013) states that it is important to provide nutritional support for the septic patient early in order to maintain gut integrity and prevent bacterial translocation. The campaign recommends beginning low dose (approximately 500 Kcal/day) oral or enteral feeding within the first 48 hours and advancing the feeding only as the patient tolerates. They do not recommend routine administration of immunomodulating supplementation to feedings.

Unfortunately, it is not always possible to meet the nutritional needs of septic patients with enteral feedings, in which case parenteral feedings are indicated. Standard total parenteral nutrition (TPN) solutions may not be metabolized well in septic patients so specialized solutions may be used. Because the nutritional requirements of septic patients are complex and undergoing revision, a dietitian or a nutrition protocol should be consulted to determine each patient's specific requirements.

Facilitating Communication

The Surviving Sepsis Campaign (Dellinger et al., 2013) recommends that the prognosis and goals of care for the septic patient be discussed with the family and patient within the first 72 hours following presentation of sepsis. Although it may be difficult to determine the prognosis immediately, many patients with severe sepsis and septic shock will not survive. Therefore, the SSC recommends that palliative care principles be included in the patient's care or palliative care specialists be consulted.

Prevention, Detection, and Management of Complications

The most common complications of sepsis are **multiple organ dysfunction syndrome (MODS)** and **disseminated intravascular coagulation (DIC)**. MODS is defined as altered organ function in an acutely ill patient such that homeostasis cannot be maintained without intervention.

One definition of DIC is an acquired syndrome characterized by intravascular activation of coagulation arising from a variety of different causes (Becker & Wira, 2009). It can cause damage to microvasculature, result in simultaneous thrombotic and hemorrhagic complications, and

produce organ dysfunction. The most effective way to prevent both of these complications is to identify the septic patient early and initiate definitive management of severe sepsis within the first 6 hours of the onset of symptoms.

Multiple Organ Dysfunction Syndrome (MODS)

MODS develops in severe sepsis in response to a series of factors. First, older patients may have pre-existing organ dysfunctions that make organ failure more likely to develop. Initially in all patients, tissue damage develops at the site of an infection, resulting in damage to the endothelial layer of blood vessels with release of vasoactive and pro-coagulant mediators. This results in localized increased vascular permeability and shunting. When SIRS develops, the response to the circulating mediators becomes systemic. A major contributor to the development of tissue ischemia may be that in response to the circulating pro-coagulants, platelets, red cells, and thrombin form microaggregates in blood vessels and impede blood flow to vital organs. Additionally, pro-inflammatory cytokines and other mediators may cause generalized endothelial dysfunction, resulting in extensive capillary leakage in organs such as the lungs, liver, heart, and kidney. As more organs fail, the mortality rate from MODS increases. When one organ fails the mortality rate is about 7%, whereas when four organs fail, the mortality rate approaches 70%. Therefore, the nurse caring for a septic patient needs to be vigilant about monitoring the patient for the development of any of the following signs of organ dysfunction:

- Acute lung injury (ALI) indicated by tachypnea, hyperventilation (arterial pH less than 7.35 and $p\text{CO}_2$ less than 32), and/or hypoxemia
 - Assessment and management of the patient with ALI/ARDS is discussed in Chapter 3. As noted in the sepsis management bundle, high tidal volumes should be avoided in septic patients. Current recommendations are for “low” tidal volume (6 mL/kg/lean body weight) as a goal in conjunction with the goal of maintaining end-inspiratory plateau pressures of less than 30 cm H_2O to prevent further lung injury.
- Coagulation abnormalities or thrombocytopenia
 - Assessment and management of the patient with DIC will be discussed later in this chapter.
- Neurological dysfunction indicated by a sudden change in mental status with possible confusion or psychosis progressing to coma
- Renal dysfunction evidenced by a decreased urine output (less than 0.5 mL/kg/hr) and elevated serum creatinine
 - Assessment and management of the patient with acute renal failure is discussed in Chapter 14.

Evidence suggests that consultation with a nephrologist as soon as renal dysfunction is suspected results in better patient outcomes.

- Liver dysfunction indicated by jaundice, coagulopathy, decreased protein C levels, and increased D-dimer levels
 - Assessment and management of the patient with liver failure is discussed in Chapter 14.
- Gastrointestinal injury indicated by stress ulceration, ileus, and malabsorption. Damage to the GI tract may worsen the septic response by allowing translocation of bacteria and endotoxins from the gut.
- Cardiac dysfunction indicated by tachycardia, dysrhythmias, hypotension with decreased CVP or PA pressures, and either high or low cardiac outputs

If the nurse recognizes the patient is developing organ failure, she should notify the physician and prepare to institute the appropriate interventions. There is no specific care for the patient with MODS other than the supportive care indicated for the dysfunctional organ(s) and management of the underlying condition. However, the earlier interventions are instituted, the more likely the outcome is to be positive.

Disseminated Intravascular Coagulation (DIC)

DIC occurs in approximately 1% of all hospitalized patients but may occur in 30% to 50% of patients with sepsis (Becker & Wira, 2009). Although the incidence of DIC has decreased over the past decade, the mortality rate has remained the same (Singh et al., 2013). Sepsis is the most common cause of DIC but it may also develop following a variety of other conditions. Common other precursors of DIC (often called triggers) include:

- Traumatic injuries, especially multiple trauma, burns, and crush injuries
- Obstetric complications such as abruptio placentae, placenta previa, retained dead fetus, and eclampsia
- Embolisms, whether fat, amniotic fluid, or pulmonary
- Immunologic disorders such as transfusion reactions, transplant rejection, rheumatoid arthritis, and lupus
- Malignancies
- Shock accompanied by acidosis

◆ Figure 18-5 displays how DIC progresses once it has been triggered. In DIC, the trigger causes excessive activation of the normal coagulation cascade. Either the extrinsic coagulation pathway is activated by massive tissue destruction (e.g., crush injury) or the intrinsic pathway is activated through endothelial cell injury (e.g., shock with acidosis). Next, thrombin production, which should be balanced by chemical mediators such as proteins S and C, is not and continues unrestrained. The excessive thrombin

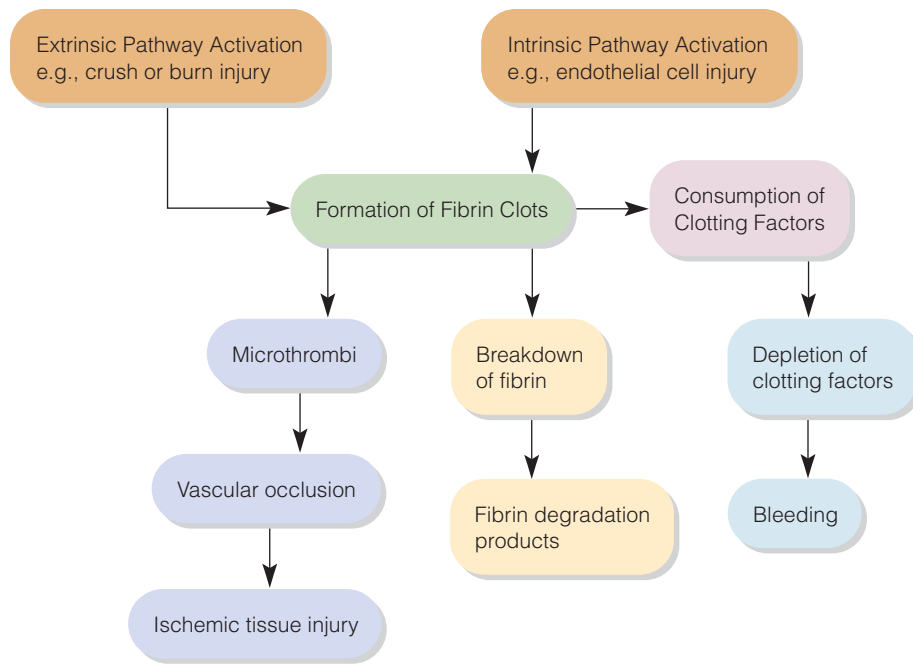


Figure 18-5 Overview of disseminated intravascular coagulopathy.

production activates fibrinogen, resulting in the formation of fibrin. Fibrin is deposited throughout the microcirculation, leading to obstruction of blood vessels and ischemic tissue damage. As the fibrin deposits are formed, platelets and coagulation factors are consumed, leading to a deficiency of clotting factors, and the patient begins to bleed. The bleeding may be intensified as the clots are lysed, resulting in fibrin degradation products (FDPs) because the FDPs are anticoagulants. Thus, the patient develops simultaneous thrombotic and hemorrhagic complications.

Assessment

Patient assessment is geared at identifying the manifestations that result from the excessive thrombosis formation and bleeding. These signs may appear acutely as a profound clotting/bleeding disorder or may develop slowly as a chronic, partially compensated process. How rapidly DIC develops is partially dependent on the intensity of the trigger but is also related to the condition of the patient's liver, bone marrow, and endothelium. Some patients present primarily with bleeding abnormalities; others present with manifestations from microemboli. The critical care nurse must be aware of the various possible manifestations of DIC and must be prepared to notify the physician if DIC is suspected.

Initially, the patient may show manifestations of thrombosis and microemboli:

- Occlusion of blood vessels may be seen as cyanosis and/or gangrene, especially of the digits. There may actually be a demarcation line between the viable and

necrotic tissue visible on the fingers or toes, and the patient may complain of pain in the digits.

- Pulses may be diminished.
- Inadequate perfusion of the brain may result in a CVA or present as an altered level of consciousness, especially confusion or disorientation.
- Inadequate renal perfusion may be identified by a decrease in urine output, an increase in serum creatinine, or pain at the costovertebral angle.
- The patient may complain of abdominal tenderness and have diminished or absent bowel sounds that may indicate an ileus or a bowel infarction.

As clotting factors are depleted, the patient begins to ooze or bleed from multiple sites:

- In chronic DIC, there may be only a slight oozing of blood.
- In acute DIC, the patient may bleed heavily from IV sites, arterial lines, urinary catheters, surgical sites, the GI tract, and mucous membranes.
- The patient may also have ecchymoses on the palate, gums, and skin.
- Bleeding in the pulmonary tree may be evidenced as hemoptysis.
 - The patient may develop hematuria or metrorrhagia.
- If the patient begins to hemorrhage from any of these sites, the signs and symptoms of hemorrhagic shock may become evident.

Diagnosis of Disseminated Intravascular Coagulation

The diagnosis of DIC is made based on the presence of an underlying disorder that is likely to lead to DIC, the clinical presentation of the patient, and laboratory values.

There is no gold standard for diagnosing DIC (Wada et al., 2013), so the nurse examines a series of lab tests to determine the likelihood of DIC. The tests include:

- Platelet count (normally 150,000 to 400,000). A value of less than 50,000 is suggestive of DIC. However, a drop of 50% from baseline may also be indicative.
- FDPs (normally less than 10 mcg/mL). A value greater than 40 mcg/mL or rising values are suggestive of DIC.
- D-dimer (normally less than 250 ng/mL). Higher values may be indicative of DIC but the test is less sensitive than FDPs because a positive D-dimer may result whenever a large clot is being dissolved.
- Fibrinogen (normally 200 to 400 mg/dL). Levels may be normal very early in the disorder or in mild DIC. A value of less than 100 or falling values are suggestive.
- Prothrombin time ([PT], normally 11 to 12.5 seconds). Any elevation is suggestive of DIC.
- Activated partial thromboplastin time (normally 30 to 40 seconds). Prolongation is suggestive of DIC; critical values are greater than 70 seconds.

COLLABORATIVE CARE

The most important intervention for DIC is identification and treatment of the underlying disorder.

Identifying and Treating the Trigger

When the trigger is an easily identifiable and treatable cause, such as an abruptio placentae, the outcome is likely to be better than if the trigger cannot be identified or is difficult to treat. Aggressive treatment of the underlying disorder is always indicated because with successful treatment of the trigger, DIC may spontaneously resolve (Wada et al., 2013). If treatment fails, the mortality rate for DIC is high.

Restoring the Balance of Clot Formation, Dissolution, and Inhibition

Geiger (2003) notes that maintaining hemostasis is not an easy task and requires delicate balancing of clot formation, dissolution, and inhibition. To this end, clotting factors that have been consumed in the process of DIC may be replaced.

Replacement of these factors is more likely to aid in rebalancing the coagulation disorder if the trigger has been identified and removed.

Platelet replacement may be provided if the patient is actively bleeding and the platelet count is dangerously low. There is no uniform agreement about an appropriate level for platelet transfusions in DIC, with recommendations ranging between 10,000 and 50,000 depending on whether or not the patient is actively bleeding (Wada et al., 2013). As with all blood transfusions, consent is required prior to platelet transfusions. Platelets are administered using filter tubing at an infusion rate of 4 to 8 mL/kg/hr.

Depending on laboratory findings, and if the patient continues to bleed, other clotting factors may also be replaced. The PT/INR may be used to guide administration of fresh frozen plasma. If the fibrinogen level is less than 100 mg/dL, cryoprecipitate may also be given because it contains 5 to 10 times more fibrinogen than plasma. Theoretically, administering heparin should decrease the consumption of clotting factors and slow the process of DIC. Although heparin has been used for many years, the beneficial effects of heparin have not been convincingly established (Becker & Wira, 2009). However, there probably is a therapeutic indication for heparin when the patient demonstrates primarily coagulation abnormalities in DIC and severe organ dysfunction from microemboli. If heparin is used, the patient should have an obvious thrombosis and it should be administered in low doses such as 5 to 10 units/kg/hr or 80 to 100 units/kg sc. During administration, the nurse observes the patient closely to detect any increase in the amount or rate of bleeding. If heparin therapy is successful, the nurse will recognize that end organ function stabilizes and eventually begins to improve.

Supporting Organ Perfusion and Function

Supporting and maintaining adequate organ and tissue perfusion are crucial to patient survival. Specific interventions depend on the patient's underlying condition and the degree of hemorrhage:

- IV fluids may be sufficient to maintain adequate perfusion in some patients. However, if blood loss is pronounced, then packed red blood cells may be administered.
- The nurse monitors the patient's BP, heart rate, hydration status, and oxygen saturation at least every 2 hours and more often if necessary.
- The physician should be notified if the heart rate changes more than 10% from baseline or if the MAP drops below a specified level (often 65 to 70 mmHg).

The nurse also closely monitors the function of the patient's various organ systems, especially neurological and renal functioning:

- Neurological screening, such as the Glasgow Coma Scale, should be performed at least every 2 hours.
- Urine output should be measured every 1 to 2 hours, and creatinine should be reviewed on a daily basis.
- The nurse checks the adequacy of peripheral perfusion by assessing and documenting the pulses, sensations, movement, capillary refill, and warmth of each of the extremities at least every 2 hours.
- The nurse closely observes the patient for any increased bleeding, noting the presence of ecchymoses, documenting the presence of obvious bleeding, and testing any suspicious secretions for the presence of blood.

The patient should also be protected from further tissue injury:

- The nurse avoids inserting any invasive devices (including rectal thermometers) unless absolutely necessary.
- After an IV is removed, the nurse holds pressure for 3 to 5 minutes to ensure hemostasis.
- Mouth care is provided gently with soft massage of the gums to remove debris.
- Soft clots should not be removed but should be allowed to fall away on their own.

NURSING CARE

Patients with MODS and DIC are critically ill requiring considerable nursing attention just to maintain their physiologic integrity. Yet, they have other needs as well.

Enhancing Comfort

It is essential to assess the patient at regular intervals for pain because pain is one of the hallmarks of tissue ischemia. The nurse questions the patient hourly, when possible, concerning the presence, location, intensity, and duration of any pain. The development of pain in a new location may be an indication of new microemboli. Pain may also be the

result of bleeding into confined spaces, especially joints. After carefully evaluating the pain and notifying the physician, if necessary, the nurse provides the appropriate medication.

Facilitating Communication

Patients who develop DIC and their families are often highly anxious. Some patients were already critically ill, and DIC represents a serious, potentially lethal complication. Other patients, especially pregnant women with abruptio placentae, were anticipating a joyous event and have encountered a crisis. Pronounced bleeding from multiple sites can be very frightening for both the patient and the family. The nurse needs to be willing to explain repeatedly to the patient and family what is occurring, what the likely cause is, and how the healthcare team is attempting to intervene. Additional guidelines about how to communicate with critically ill patients and their families are provided in Chapter 2.

Nursing Diagnoses

Patient with Sepsis and Disseminated Intravascular Coagulation

- Infection related to inadequate primary defenses
- Decreased cardiac output related to decreased ventricular filling (decreased preload)
- Deficient fluid volume related to failure of regulatory mechanisms and increased metabolic rate
- Interrupted family processes related to serious injury of family member
- Risk for ineffective tissue perfusion: peripheral, renal, GI, cardiovascular, cardiopulmonary, and cerebral related to hypotension and hypovolemia

Essential for Patient Centered Care

Patients who develop DIC and their families are usually very frightened, and they often need to tell their illness narrative repeatedly in an attempt to understand what is happening. Obtaining a member of the healthcare team or appropriate volunteer to listen to them can assist families to integrate the event.

Sepsis Summary

Severe sepsis is a complex illness that develops after a localized infection triggers the inflammatory response syndrome, resulting in the failure of one or more organ systems. In the past, it was associated with high morbidity and mortality. Recently, early goal-directed therapy has led

to improved outcomes for septic patients. However, treatment of severe sepsis remains complicated, especially if patients are not diagnosed promptly, do not receive adequate volume resuscitation, or are not treated with appropriate antibiotics within the first 3 hours.

Why/Why Not?

The nurse has just obtained the midnight assessment of an 81-year-old patient with pneumonia. His heart rate has risen to 98, his respiratory rate to 26, and his temperature to 102°F. His BP is currently 95/50 (65). The resident has just gone to sleep. The nurse is trying to decide whether to

awaken the resident for notification about the patient's assessment. Should the nurse notify the resident now? Why or why not?

See answers to Why/Why Not? in the Answer Section.

Case Study

Allen Hale, 27 years old, was admitted to the eight-bed ICU of a community hospital after running a red light in his car and colliding with another car. He is being transferred to the operating room (OR) to evacuate a left parietal subdural hematoma. His concomitant injuries include a flail chest with pulmonary contusions, for which he is being mechanically ventilated, and a fractured left femur. His blood alcohol level on admission was 200 mg/dL (0.2% weight/volume), and there was indication of marijuana on his toxicology screen. Currently, he is receiving Diprivan (propofol) 45 mcg/kg/min. He responds to noxious stimuli by withdrawal. He withdraws all his extremities from noxious stimuli except his left leg, and his pupils are equal and reactive to light.

On arrival in the ICU following surgery for removal of a subdural hematoma, Allen remains on Diprivan (propofol) 45 mcg/kg/min. His neurological signs are unchanged from pre-op, and his left leg fracture has not been repaired but an immobilizer is in place (dorsalis pedis [DP] and posterior tibial [PT] pulses are palpable in the left foot).

Some of his other assessment findings are BP 109/71, HR 111/min, RR 18, Temp 100.6°F, urine output 20–30 mL/hr, pH 7.28, pCO₂ 46, pO₂ 99, O₂ saturation 97%, Hgb 10, and Hct 30.

His ventilator settings are Assist Control with a rate of 18, tidal volume (TV) 800, FiO₂ 50%, PEEP 5. Lung sounds are diminished on the left with scattered rhonchi on the right. He has a left chest tube in place and has bilateral equal chest expansion.

In addition to the Diprivan and ventilator settings, some of his post-op orders include IV antibiotics, morphine sulfate 2 mg q 2h for pain, Foley catheter to gravity drainage, nasogastric tube to low continuous suction, maintain left leg in immobilizer.

1. What are the three top priorities for nursing care? List at least three nursing or collaborative interventions for each priority.

2. What additional concerns would there be for this patient?
3. What are the priority concerns when managing a patient with a chest tube?
4. What measures should be instituted at this time to prevent VAP?

On hospital day 6, Allen became increasingly unstable. His chest x-ray revealed pulmonary contusions and pneumonia on the left as well as the fractured ribs. He began to meet the criteria for ARDS with diffuse bilateral infiltrates and refractory hypoxemia. His ventilator settings at this time were FiO₂ 100%, TV 800, Assist Control 24, and PEEP 7. His Diprivan had been increased to 55 mcg/kg/min but he was becoming increasingly restless.

Other findings included BP 80/50, HR 120 bpm, RR 28, urine output 18–20 mL/hr, temperature 101.4°F, pH 7.22, pO₂ 109, pCO₂ 54, WBC 32,000, Hgb 8, Hct 28.

5. What are the possible sources of infection?
6. What is SIRS?
7. Is it likely that Allen is developing SIRS? Why?
8. Has he developed severe sepsis or septic shock? Why or why not?
9. Explain the rationales, indications, and contraindications for the following interventions and state whether the interventions would be appropriate for Allen:
 - Continuous IV infusion of norepinephrine bitartrate (Levophed)
 - Increase in PEEP
 - Increase in the amount of sedation
10. Are there indications that Allen is in danger of developing failure of other organs? If yes, what are the indications?

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 18.1 Why is severe sepsis a costly illness? Why is it likely to become more costly?
- 18.2 How does elevating the head of the bed for a ventilated patient decrease the incidence of ventilator-associated pneumonia?

- 18.3 What interventions are included in evidence-based management of a central venous catheter?
- 18.4 Why is shaving a surgical site preoperatively no longer indicated?
- 18.5 What assessment findings would indicate to the nurse that the patient had developed systemic inflammatory response syndrome (SIRS)?
- 18.6 What questions should the nurse consider when attempting to localize the source of a patient's infection?

- 18.7 What are the elements of the 3 hour sepsis management bundle?
- 18.8 What are the elements of the 6 hour sepsis management bundle and why are they essential to patient survival?
- 18.9 When should a patient's fever be treated?
- 18.10 Why does disseminated intravascular coagulation result in both thrombosis and bleeding?

See answers to Chapter Review Questions in the Answer Section.

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Chapter 19

Caring for the ICU Patient at the End of Life

Kathleen Perrin, PhD, RN, CCRN

Mary Kazanowski PhD, APRN, ACHPN

Abbreviations

DNAR Do Not Attempt Resuscitation
ENA Emergency Nurses' Association

RDOS Respiratory Distress Observation Scale



Learning Outcomes

Upon completion of this chapter, the learner will be able to:

1. Describe healthcare provider responses to the various categories of death in the ICU.
2. Examine the needs of families of dying patients.
3. Prioritize collaborative and nursing strategies used to manage patients' symptoms at the end of life.
4. Explain sources of conflict at the end of life.

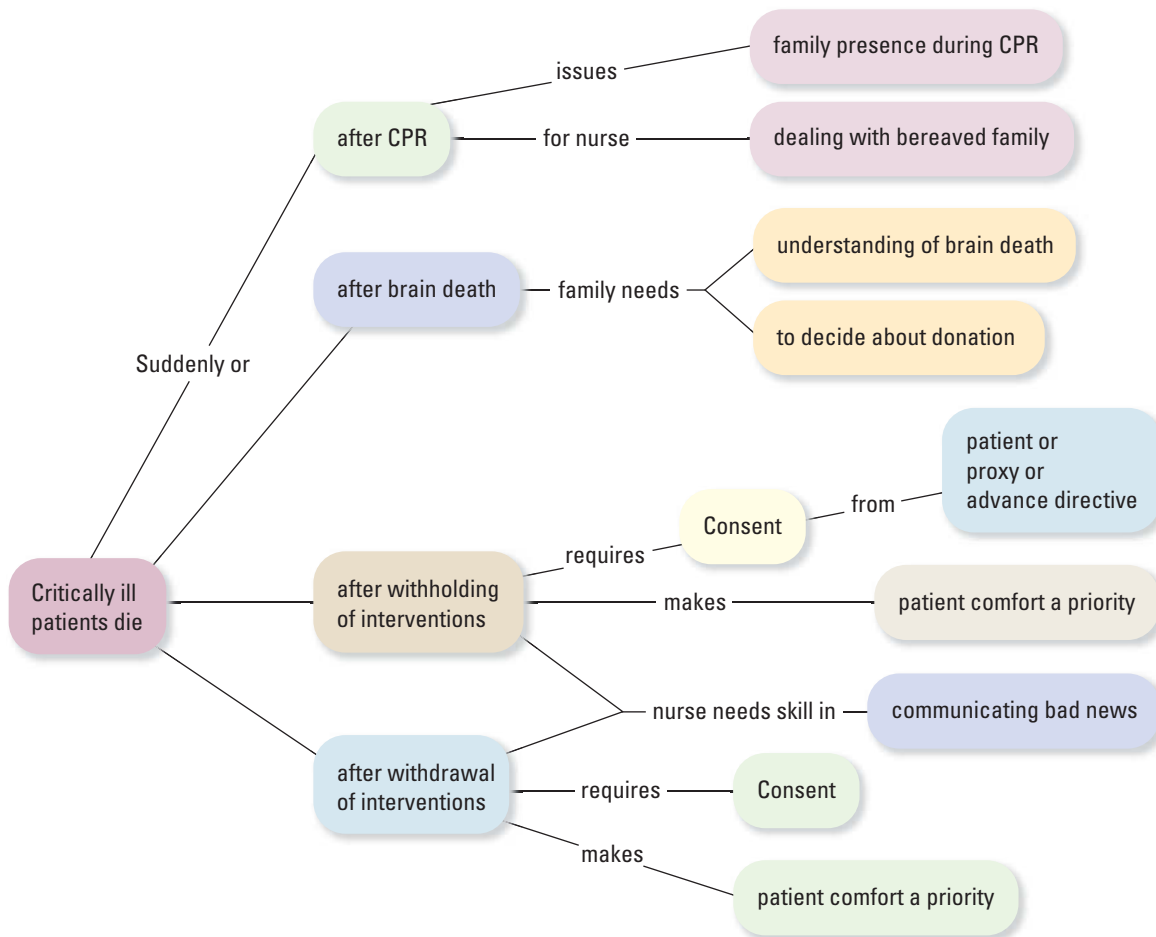
End of Life in the ICU

When critically ill patients die, it may be unexpected. However, it is more likely to be following a discussion with the patient, family, or healthcare proxy resulting in the withdrawal or withholding of life-sustaining technology. Care of the dying patient in the intensive care unit (ICU) focuses on meeting the needs of the dying patient and his family (Visual Map 19-1).

Review of Some Ethical and Legal Concepts

For the past 20 years in the United States, medical ethics has placed an emphasis on the principle of respect for persons. A derivation of this principle is that the patient should be the primary decision-maker about his healthcare as long

as he is competent. **Advance directives** were developed to allow competent patients to indicate in advance the kind of healthcare treatment they would desire should they become incapacitated at the end of their lives. Patients may use an advance directive to indicate a preference for specific treatments to be provided or withheld (medical directive) and/or to designate an individual (healthcare proxy) to make treatment decisions for them. Once signed the directives are legally enforceable. However, patients or their proxies may change their minds about the treatments described in the directive at any time, even at the end of the patient's life. Recently, there has been a movement away from a reliance on patient autonomy toward a shared decision paradigm stimulated in part by studies showing that patients want their families involved in their healthcare decision-making and that many favor joint decision-making with their healthcare providers (Carlet et al., 2004).



Visual Map 19-1 End of Life Summary

When considering what care should be provided to a patient, many healthcare providers and patients weigh the benefits and burdens of the treatment. Some ethicists refer to this balancing of effects as proportionalism, derived from the medieval principle of double effect. The principle of double effect acknowledges that most treatments not only have benefits (good effects) but also impose substantial burdens (bad effects). In proportionalism, not only must the benefits outweigh the burdens, but the person choosing the treatment also must intend the good effect rather than the bad one. When using this methodology, a person weighs the circumstances, his intentions, and the consequences to determine the least harmful result (McGhee, 2007). For example, when a nurse is providing morphine for dyspnea to a dying patient, the nurse intends the medication to decrease the patient's anxiety and dyspnea, thus improving the patient's comfort, and the nurse accepts the possibility that it may decrease the patient's respiratory rate.

Palliative Care

Many ethicists and healthcare providers believe that placing comfort as a priority or delivering palliative care is

ethically and legally appropriate, even necessary for all people with life-threatening illnesses. Palliative care is multidisciplinary care intended to focus on control of patients' symptoms, relief of patients' and families' suffering, and enhancement of patients' quality of life. Historically, palliative care was associated with end-of-life care and there was a forced dichotomy between curative care and palliative care. Now, there is a growing realization that for all seriously ill patients, palliative care should be delivered on a continuum. At one end of the continuum, the patient's care would be aggressive and curative with less emphasis on patient comfort while at the other end of the continuum, the patient's care would involve little or no aggressive/curative treatment but have an emphasis on enhancing patient comfort. Freysteinson (2010) summarizes this idea well when she says, "Possibly the dichotomous notion that there is care of the living and hospice care for the dying will be replaced by an overarching palliative paradigm of respect for life, dignity, and personal choice across the life span" (p. 71).

In its definition of palliative care, the World Health Organization (WHO) states that although palliative care embraces life, it realizes that death is a normal process.

Therefore, palliative care makes no effort to either speed up or slow down death. Instead, it offers a system to help patients live as fully as possible until death by providing relief from symptoms and integrating psychosocial and spiritual aspects of care. Finally, the WHO definition states that palliative care offers support to the patient's family to help them cope with the patient's death. The patient's family is defined (and this definition will be used in this chapter) as whomever the patient considers to be his family whether related or not. In the United States, palliative care is often initiated in the form of a palliative consult, where a physician or APRN specializing in palliative care sees the patient and family to facilitate discussions of goals of care, assist in decision-making, and facilitate symptom management.

Essential for Patient-Centered Care

In the context of palliative care, the patient's family is considered to be whomever the patient considers to be family.

Categories of Death in the ICU

Although families and patients usually associate critical care with the promise of modern medicine and the expectation of a cure for the patient, the limits of medicine may be reached and the patient may die. Angus and others (2004) found that 22% of deaths of hospitalized patients followed ICU admission; that means that about 540,000 American patients die after ICU admission each year. Thus, ICU nurses routinely encounter dying patients and need to be prepared to provide expert care to them.

Copnell (2005) noted that although ICU deaths may be classified as planned or unplanned, a better approach is to view the deaths as falling into four categories:

- Death that occurs despite all efforts on the part of clinicians. (This may be called "failed CPR" and accounts for approximately 10% of ICU deaths. It includes patients who die suddenly following traumatic injuries and patients who have been receiving maximal support for days or even weeks.)
- Death that occurs following demonstration of lack of brainstem functioning. (This is usually known as **brain death** and represents between 6% and 8% of deaths in critical care units.)
- Death that occurs following a decision not to initiate one or more interventions such as ventilation, intubation, dialysis, enteral feeding, or vasopressors. (This may be known as *limitation* or *withholding* because a decision is made not to institute a treatment that is unlikely to benefit the patient.)
- Death that occurs after a decision to withdraw one or more therapies that have already been initiated but are not benefiting the patient. (This is often termed *withdrawal*.)

The vast majority of ICU deaths in the United States, approximately 70% to 90%, follow either limitation or withdrawal of medical interventions. Limitation and withdrawal usually have two phases: the first is a shared decision-making process that leads from a focus on care that is in pursuit of a cure to care that focuses on palliation and comfort. The second phase focuses on providing the appropriate care to the patient and family and requires humanistic and technical skills from the members of the healthcare team.

Death That Occurs Despite All Efforts on the Part of Clinicians

Approximately 10% of the time, patients receive aggressive interventions, including **cardiopulmonary resuscitation (CPR)**, until the end of life. Weil and Fries (2005), in a review of current management and outcomes of CPR concluded, "there is no secure evidence that the ultimate outcomes after cardiopulmonary resuscitation in settings of in-hospital cardiac arrest have improved in more than 40 years (p. 2825)." They indicated that the current survival rate may be as high as 17%, a modest increase from 14% documented in the past. In 2008, Peberdy and associates agreed, adding that some groups of patients such as patients with renal failure, cancer, and multisystem organ failure, rarely survive following a cardiac arrest. When Tian and others (2010) reviewed the outcome of CPR in critically ill patients, they determined that 15.9% of patients were discharged from the hospital alive with approximately half of those able to go home. In their study, patients who received vasopressors were half as likely to survive and half as likely to go home if they survived as other critically ill patients.

Because the survival rate is so low, increasing numbers of ethicists and healthcare providers have begun to argue that CPR is a futile intervention and ought not to be offered to every patient. Daly (2008) stated that patients and their families rarely understand how poor the survival rates are for CPR. She argues that even though the American Heart Association guidelines for CPR renamed "Do Not Resuscitate" to "Do Not Attempt Resuscitation" to emphasize the likelihood that resuscitation would not be successful, the likelihood that resuscitation will not be accomplished has not been conveyed to patients and families. Marco and Larkin (2008) verified the public's misperception, learning that 54% of people estimated that they would survive CPR with full return to baseline functioning. Moreover, resuscitation is most likely a painful or at least an uncomfortable process. The patient's ribs may be broken, endotracheal intubation is performed, and electric shocks may be delivered. Yet CPR remains the default option for intervention for hospitalized patients, even those patients with multisystem organ failure. It is provided unless the patient or the patient's proxy agrees to a written **Do Not Attempt Resuscitation (DNAR)** order. Daly (2008) argues that instead of CPR being the default in

all hospitals in the United States, CPR should be limited to those patients who are likely to benefit from CPR, have been offered the option by their physician, and have consented to CPR in advance.

Bishop, Brothers, Perry, and Ahmad (2010) also recommend a change in presumption for CPR to a presumption not to resuscitate. They recommend that physicians discuss CPR status routinely with patients during hospital admission explaining the physician's recommendation for or against CPR. If the physician recommended and the patient consented to resuscitation, a CPR order would then be included as part of a patient's admission orders. As is the current situation, the decision could be readdressed if the patient's status changed. However, if a CPR order was not present, the patient would not receive resuscitation.

Family Presence During Resuscitation

Currently resuscitation is attempted on all patients in American hospitals unless they have a DNAR order in place. More than 80% of those patients who have resuscitation attempted die in the hospital. Patients who die in ICUs following CPR usually fall into two groups: patients who were admitted within the previous 2 days with devastating but potentially survivable injuries such as severe traumatic injuries, and patients who refuse or for whom their families refuse to have a DNAR order written. In both of these circumstances the family may not have had the opportunity to come to terms with the devastating nature of the injury and the realization that modern healthcare is unable to cure the patient. This is one of the reasons why many healthcare practitioners are advocating that family members be present during CPR. There are a number of benefits to family presence during resuscitation such as:

- Family members may be active participants able to provide information about the patient and to ask questions and gain a sense of reality about the critical illness (Baumhover & Hughes, 2009).
- Family members may be able to provide comfort and support to the patient.
- Family members usually realize that the healthcare team has tried heroically to keep the patient alive and are better able to accept the team's decision to end resuscitation (McClement, Fallis, & Pereira, 2009).
- After seeing the patient's last moments, family members are less likely to be haunted by what they imagine might have occurred during that time.
- If the patient dies, family members usually cope better with their loss (Halm, 2005).
- Staff are reminded of the patient's personhood (Halm, 2005; McClement et al., 2009).

Few patients survive and communicate following resuscitation, so it is difficult to determine the patient's perspective on having the family present during CPR. In one study (Eichorn, Meyers, & Guzzetta, 2001) that examined a few patients who survived resuscitation attempts, patients reported feeling "comforted, supported, and less afraid" when family members were in the room during resuscitation.

Opponents of family presence usually are concerned that the healthcare team will not be able to function optimally if family members are present (McClement et al., 2009). They worry that family members may not be aware of all of the patient's medical conditions and there may be a breach of confidentiality. Opponents of family presence are also worried about the effects of the family on the hospital staff. They fear that more staff will be needed during CPR because someone will need to meet the needs of the family. They believe that the staff will experience more stress, resulting in performance anxiety (Halm, 2005). Opponents also believe that family members may misunderstand what they see and hear during the code, possibly resulting in lawsuits. Current research does not support these concerns (Jabre et al., 2013). Thus, there has been steadily growing interest in allowing family members to be present during resuscitation of their loved one under certain circumstances.

Essential for Evidence-Based Practice

Multiple studies have demonstrated that it is beneficial to family members and not a significant problem for healthcare providers to have family members present during a patient's resuscitation attempt.

Nursing Actions

The nurse works with families to support, inform, guide, and provide comfort through their decision-making process.

Helping the Family to Decide

When the nurse is helping family members decide whether or not they should be present during their loved one's resuscitation, seven steps should be followed (using the mnemonic device *in-or-out*) (Riwwitis & Twibell, 2006, p. 14).

1. **I:** Introduce yourself to the family.
2. **N:** Now: Explain the patient's current status.
3. **O:** Outcome: Explain the possible results.
4. **R:** Relationships: Learn who makes the decisions.
5. **O:** Option: Provide the choices.
6. **U:** Understanding: Assess comprehension.
7. **T:** Time: Take action.

Guidelines for Family Presence

If a family member decides that she wants to be present, Mason (2004) recommends the following guidelines be followed:

- The nurse should initially find out the family member's relationship to the patient and ask for identification.
- The nurse should determine if the family member is able to follow instructions about being in the room, such as stepping away from the bed during defibrillation. If family members want to go in but are very emotional, it may be possible to get them to cooperate by looking them in the eye and saying, "You'll need to pull yourself together if you want to go into the room and be of help to your loved one." Emergency Nurses' Association (ENA) guidelines recommend that people who appear to be under the influence of drugs or alcohol or who are abusive should not be permitted in the room.
- If the patient is conscious, the nurse should ask the patient's permission. If not, and if the hospital does not have a written policy on family presence, the nurse should get the permission of the person responsible for the care, such as the physician in charge of the code.
- A trained facilitator such as a nurse, social worker, or chaplain should prepare the family for how their loved one will appear, what they will see in the room, and what they can do. The facilitator should stay with the family while they are in the room to provide them with explanations and instructions (such as to stay away from the bed during defibrillation).
- Even if the family stays in the room for only 5 or 10 minutes, they need to be debriefed afterward. The facilitator should ask them how they are feeling and encourage them to describe their experience or to ask any questions they may have about what they witnessed.

Working with the Bereaved Family

Families of patients who have died in ICU stress the importance of bereavement care (Nelson et al., 2010) stating that they felt lost and did not know what to do or where to go after the patient had been pronounced dead. According to Truog and others (2008), best practices when speaking with bereaved families and loved ones include:

- If the family has not been at the hospital and the death is unexpected, contacting them and explaining they should come to the hospital rather than saying outright over the phone that the patient has died
- Providing privacy for the family and loved ones during the discussion
- Beginning conversations with words of condolence such as "I'm so sorry for your loss" unless such words

are not appropriate in the family's religious tradition (e.g., Judaism)

- Maintaining eye contact and touching the family member if appropriate
- Avoiding the use of jargon such as *CPR* or *vent*—instead the nurse should use words like "heart stopped" and "we tried to restart it" or "the breathing machine"
- Listening to the family rather than rushing to speak, and particularly avoiding the use of clichés such as "She lived a long (or good) life" because they may not be well-received by the family
- Allowing the family time to recover. Offering either private time or time with a chaplain or social worker for the family to be with the patient after death and before the body is removed from the unit

Essential for Evidence-Based Practice

Families have identified feeling lost after the patient is pronounced dead especially if the nurse rushes away after completing post-mortem care (Nelson et al., 2010). Nurses should attempt to find an appropriate support person to remain with the bereaved family after the patient's death.

Brain Death

Brain death is defined as the irreversible loss of brain function including the function of the brainstem. The frequency with which a nurse encounters a patient declared dead by neurological criteria will depend on the type of institution where the nurse is employed, with nurses employed in tertiary medical centers encountering more patients. However, the total number of brain-dead potential donors in the United States is estimated to be between 10,500 and 13,800 annually (Ehrle, 2006).

Meeting the Criteria. In 2010, the Quality Standards Subcommittee of the American Academy of Neurology reviewed and updated the 1995 guidelines (American Academy of Neurology, Quality Standards Subcommittee, 1995) for the determination of brain death. The guidelines are only appropriate for use with adults over the age of 18 who do not have severe electrolyte, metabolic, or acid-base imbalances; have not ingested drug overdoses or poisons; and have a core body temperature greater than 32°C (90°F). There are three cardinal findings that are necessary for the determination of brain death. These are coma, absence of brainstem reflexes, and apnea:

- Coma is determined by the lack of motor responses or grimacing to noxious stimuli.
- Absence of brainstem reflexes is determined by pupils 4 mm or larger that do not respond to light, and the

absence of all of the following reflexes: oculoccephalic, oculovestibular, corneal, gag, and cough.

- Apnea is identified by removing the patient from the ventilator for approximately 8 minutes and placing him on 100% oxygen through a catheter at the level of the patient's carina or by T piece. The patient is observed to determine that there are no respiratory movements.

The determination of brain death may be confirmed by using additional criteria. Once these tests have been completed and confirmed, the patient may be declared dead using brain death criteria. The 2010 review of the guidelines reassured healthcare providers that "in adults recovery of neurologic function has not been reported after the clinical diagnosis of brain death has been established using the criteria given in the 1995 AAN practice parameter" (Wijdicks, Varelas, Gronseth, & Greer, 2010, p. 1912).

Communicating with Families about Brain Death. When the patient has been declared dead by neurological (brain death) criteria, healthcare providers must be very careful about their choice of words (Arbour, 2013; Copnell, 2014). One common mistake for a healthcare provider is to say something such as, "Your (family member) has been diagnosed with brain death. He will die shortly after we take him off the ventilator." This may confuse the family, who may not understand that the patient has been pronounced dead and removing the ventilator is essentially removing a useless mechanical device from a person who has already died. Instead a nurse might confirm, "Your family member has been pronounced dead because he has no functional brain activity. We will be removing the ventilator shortly but would like to answer any of your questions before we proceed." It is especially important that the nurse convey to the family that brain dead is dead. Copnell (2014) notes that only 28% of family members could give a correct definition of brain death and 60% believed the patient was still alive. This may be because brain death usually follows a sudden catastrophic event but also because the family finds themselves facing someone who does not look dead. Consistency, an empathetic approach, and allowing family members to ask repeated questions is helpful (Arbour, 2013).

Donation Request. The United States has a system of required request, which means that all institutions receiving Medicare or Medicaid funds must notify the organ donor bank of impending deaths and ask the families of potential donors for permission to procure organs and tissues. The request for organs and tissues should be done separately from the notification to the family that the patient has died, and is most effective when done by a specially trained individual from the Organ Procurement Organization

(OPO). Thus both members of the OPO and hospital staff will be communicating with the patient's family.

To ensure the entire healthcare team communicates the same message to the family, the OPO recommends *huddles*. Huddles are short coordinated, frequent discussions between healthcare providers and OPO personnel whose goal is to meet the unique needs of the potential donor's family. Huddles have been identified as one of the key evidence-based practices to improve donation rates (Ehrle, 2006).

Whether or not the family agrees to donation, it is important that the nurse spend time with the family, preparing them for the patient's imminent death and explaining to them what will occur (Matzo, 2015). Families need to believe that the potential donor is valued as a person. Therefore, patients who are considered to be brain dead should be treated in such a way as to maximize integrity and minimize discomfort, even though by definition they do not feel pain (Evans, 2004). Evaluation and care of the prospective deceased donor is described in Chapter 16 .

Death That Occurs Following a Decision to Limit Therapy

Most patients who die in critical care units in the United States have had some potential therapies withheld prior to their death. Therapies that may be withheld prior to a patient dying include, among others, CPR, dialysis, ventilation, intubation, and vasopressors. Withholding therapies is not usually considered until a patient's prognosis is very grave. Many Americans prefer to have aggressive medical care delivered and resources expended until the patient's prognosis becomes apparent, and physicians, nurses, patients, and families are certain that it is appropriate to limit additional interventions.

One of the most difficult issues in critical care is to determine when a patient is actually dying. The Support Principal Investigators (1995) reported in a landmark study that it is not apparent that a patient is dying until about 2 days before the patient's death. More recently, Curtis and colleagues (2011) concluded that HCPs still have difficulty determining when patients may be dying and are often unaware of or have difficulty communicating about patients' preferences for end-of-life (EOL) care. Unfortunately, since many still believe that palliative care is only appropriate for dying patients, this means it may not be initiated until the very end of a patient's life, resulting in unnecessary suffering for patients and families. It is important to refer patients early for palliative care consultation to facilitate good symptom management and limit patient suffering. Palliative care has also been shown to improve patient outcomes (Festic et al., 2010). Therefore, Weissman and Meier (2011) recommend the following evidence-based triggers for initiation of palliative care so it is not unnecessarily delayed.

Patients should be referred for palliative care when they have a potentially life limiting condition and:

- the healthcare provider would not be surprised if the patient died in the next 12 months.
- the admission was prompted by or involved difficult to control physical or psychological symptoms.
- the patient had an out of hospital cardiac arrest.
- the patient has been in the ICU for more than 7 days.
- there are disagreements among the patient, staff, or family concerning medical decisions, resuscitation preferences, or non-oral feeding and hydration.

Essential for Evidence-Based Practice

Presence of a set of triggers for a palliative care consult may result in timely initiation of palliative care and improved patient and family comfort and satisfaction with care.

Once comfort has been established as the primary goal for care, then the aggressive/curative care may still continue but ideally it will be reviewed for appropriateness. If patients and families decide to forgo some medical care, they might consider a DNAR order before limitation of other medical interventions. Allowing a natural death with a DNAR order is often the most acceptable plan for patients and families because it allows treatment to continue and only comes into play when the patient's heart beat or breathing actually stop.

Do Not Attempt Resuscitation. In 1995, the Support Principal Investigators found that, "nearly half of the patients who had a desire for CPR to be withheld did not have a DNAR order written during their hospital stay. Nearly one-third of those patients died before discharge" (Support Principal Investigators, 1995). The 10 years following the study showed no significant improvement, with CPR still being infrequently addressed in the hospital orders or medical records of patients who underwent CPR during their hospital stay (Mirza, Kad, & Ellison, 2005). Because CPR has a small chance (17%) of being successful, it is not clearly futile. Thus, the decision about resuscitation is value laden, is not purely medically determined, and is one in which patients and families should participate. Resuscitation is clearly an intrusive, expensive, and, possibly, painful medical intervention, so healthcare providers, ethicists, and lawyers are in agreement that patients and families may decline the intervention if they believe it will offer minimal benefit or be too burdensome. Therefore, if a patient is in the hospital, either the patient or the patient's family ought to be asked if CPR should be performed or withheld should the patient have a cardiac or respiratory arrest.

Unfortunately, many healthcare providers are uncomfortable initiating a discussion about DNAR orders with patients and families. Often, it is the nurses who have been listening to the patients' and families' concerns about prognoses and treatment choices who alert the physicians to the patients' and families' willingness to discuss resuscitation. Nurses can begin such discussions by interjecting questions about prognosis and DNAR beliefs into the casual conversation of daily care and developing trust before attempting to identify the patient's wishes (Perrin, 2015b). Nurses may start by providing some patient education, saying, "This is what is happening now and we're going to try this treatment." Then they might ask the patient, "Have you thought about what you would want if this didn't work or if you didn't get better? What would you want to happen?"

Many ICU patients are incapable of making a decision for themselves, causing nurses and other healthcare providers to turn to advance directives or families and loved ones to learn the patient's preferences. Applicable advance directives are uncommon; so the nurse may need to carefully query the family to help them articulate the patient's desires. A nurse might approach family members as she had the patient, educating the families about the treatments and asking similar questions. If the nurse believes that death could be imminent, she might directly and immediately ask about the patient's wishes for resuscitation. Asking specific questions about CPR is essential. When patients or family members are asked if they want "everything done," they will often say "Yes" only to declare later, "I didn't mean that."

Saying they do not want everything done for a dying family member or loved one often makes people feel as if they are abandoning the dying patient. There are other ways to tactfully ask if a patient should be resuscitated. A nurse could say something like: "We are doing all that we can to try to help your family member get better, however, she is not improving. We would like to keep her comfortable rather than try to restart her heart if it stops, do you agree?" The response may be very different than if the nurse asked, "Do you want everything done?" Nurses may need to spend a significant amount of time explaining the concept of DNAR in understandable terms to patients and families so that they can give informed consent and listening to what the patient and family understand (Perrin, 2015a).

Essential for communication

When talking with families who are attempting to make difficult decisions about care for their family member, the healthcare provider should listen more than she speaks (Truog et al., 2008).

Nurses should emphasize to patients and families that consent to a DNAR order does not imply consent to limit other medical interventions or a decision to switch to a

goal of comfort rather than cure. These are separate issues that may be discussed simultaneously or at a later time. Consenting to a DNAR does not imply that the patient will receive less care. Patients with DNAR orders in ICUs frequently receive more nursing time and nursing care.

Although the decision to withhold CPR is often the first limitation of treatment, it is common for patients and surrogates to decline other therapies that will negatively impact the patient's quality of life and provide minimal benefit. These therapies are usually withheld because although it is possible to provide them and they might offer some marginal benefit to the patient, instituting them is more likely to result in prolonging the patient's dying than survival with meaningful quality of life. In other words, the patient or surrogate weighs the benefits and burdens of treatment, and declines the treatment because the burdens outweigh the benefits.

Limitation of therapy usually follows a discussion among members of the healthcare team, the patient, and family. In the best situation, patients can state their wishes for limitation of therapy or, if they are incapacitated, they have advance directives that are clearly pertinent to the situation so that family and healthcare providers are able to follow the patient's wishes. However, it is uncommon for patients to have made their wishes for therapy known and legally enforceable prior to hospitalization.

Advance Directives. After 20 years of use, problems continue to exist with advance directives and they have failed to meet their intended goals (Duke, Yarbrough, & Pang, 2009). For the most part, they have had little demonstrated impact on the provision of end-of-life care (Tulsky, 2005). Reasons for this limited impact include:

- First, despite considerable publicity since the advent of the Patient Self Determination Act in 1991, the majority of Americans have failed to complete advance directives; in fact, the majority of patients an ICU nurse encounters will never have made one (Boyle, Miller, & Forbes-Thompson, 2005; Johnson, Zhao, Newby, Granger, & Granger, 2012).
- Second, if the patient has completed a directive the responsible person may be unable to locate it, it may be out-of-date, or it may not provide adequate guidance in the clinical situation.
- Third, if the directive appoints a healthcare proxy who is empowered to make decisions for the patient, the healthcare proxy may or may not have a clear understanding of the patient's preferences (Engelberg, Patrick, & Curtis, 2005). The patient may never even have discussed his wishes with his proxy (Johnson et al., 2012). In such circumstances, the most commonly recommended means for the healthcare proxy to make a decision are by the substituted judgment or best interests standards:

- In the substituted judgment standard, the healthcare proxy imagines himself as the patient in the particular situation and determines what the patient would want. This can be difficult depending on the ability of the healthcare proxy to see the situation from the point of view of the patient.
- In the best interests standard, the healthcare proxy makes a decision based on what he believes is in the best interests of the patient. Bailey (2006) argues that when using the best interests standard, the decision-maker is essentially making the determination that the quality of life the patient would experience is, or would be, so poor that it is worse than no life at all. It can be extremely difficult for a healthcare proxy or family member to objectively determine when the anticipated degree of the patient's body integrity and function will have deteriorated to such a point.
- Finally, and perhaps most importantly, a completed directive is no substitute for a person who has had a conversation with the patient who understands the patient's goals and values. When patients have had discussions with their prospective healthcare proxy, the potential healthcare proxy displays a better understanding of what is important to the patient at end of life and may be more likely to accurately follow patient preferences.

There are a few questions that are useful for the nurse to ask that might help to clarify the wishes of the patient. The nurse might wish to know:

- Has the patient spoken to anyone about the terms of the advance directive?
- With whom did the patient speak?
- What was discussed?
- What are the patient's wishes in her or his own words?

Patients generally want their healthcare proxies to be family members and although they want the family member to voice their perspective about what type of care should be provided, most want the decision-making to be shared between their loved ones and their physician (Nolan et al., 2005).

Thus the nurse may be responsible for coordinating a discussion between members of the healthcare team, the family, and the patient about goals of patient care. Conflict may develop when families, patients, or healthcare team members have differing views about what is in the patient's best interests or what ought to be the goals for patient care. For example, is the patient dying and should interventions be limited? The nurse has an important role in promoting effective communication and preventing conflict between healthcare providers, patients, and families. Tulsky (2005)

states that developing trust is an essential first step in developing a relationship with a potentially dying patient and her family, and an important factor in avoiding conflict and coming to a realistic decision about end-of-life care. Tulskey (2005) recommends these steps that the nurse can follow for establishing trust:

- Encourage patients and families to talk and identify and acknowledge the family's feelings as well as the difficulty of the situation by asking something such as: "I'm sure this illness has been a lot for you to absorb quickly. How are you coping with it?"
- Maintain a higher ratio of family member-to-healthcare provider speaking time; therefore, listening, asking clarifying questions, and tolerating silence are important skills for nurses.
- Do not contradict or put down other healthcare providers yet recognize patient concerns by saying something such as: "I hear you saying that you don't feel you are being heard by _____. I'd like to make certain you have a chance to voice all your concerns."
- Acknowledge errors.
- Be humble by setting a nonthreatening tone.
- Demonstrate respect for family members by saying something such as: "I'm so impressed by how involved you have been with your father throughout his illness." Also show respect by assuming that family members are operating in what they believe to be the patient's best interests unless there is proof to the contrary.

Conveying Bad News. Once trust has been established, the nurse and other members of the healthcare team must assist the family to understand what is happening to their family member so they can plan for the future. When dealing with potentially dying, critically ill patients, this often involves conveying or helping the family to accept bad news. Peel (2003) notes that patients and families expect to hear bad news initially from the physician either in an individual meeting or during a family or multidisciplinary team meeting. However, people in crisis, as these families and patients are, usually need repeated explanations of

the same information (Perrin, 2015a). Thus, the nurse will need to continue to convey and reaffirm the bad news. The challenge is to deliver the news consistently in a sensitive manner at an appropriate time.

Best practices for discussing bad news were developed by Buckman (1992) and have been reiterated in numerous sources including the Critical Care Communication (C3) Course (Arnold et al., 2010). These include:

- Finding out what the patient and family understand about the issue
- Finding out how much information the patient and/or family want to know (Asking permission before giving information shows respect and helps to develop a relationship.)
- Sharing information, starting from the patient's and family's viewpoints and step by step bringing them closer to medical facts
- Responding to their reactions and emotions, using an empathic approach
- Explaining the treatment plan and prognosis, summarizing, and making a contact

Truog et al. (2008) add the following recommendation:

- Not forcing decisions, that is, allowing the patient and family time to absorb information and discuss their concerns. Or, as Arnold and associates (2010) say in slightly different terms, allowing the family to begin to grieve their loss before requiring them to make a decision.

In addition, Downey, Engelberg, Shannon, and Curtis (2006) recommend that the nurse talk with the patient and family about their cultural needs and attempt to meet them. Truthfulness and how to deliver bad news are often culturally derived values so the Society for Critical Care Medicine (SCCM) recommends that when there is a choice of providers, the provider's culture should be matched to the patient's (Davidson et al., 2007). In some cultures, it may be traditional for the patient's family to be the sole recipients of information. If the patient prefers not to hear information and defers to his family, the informed refusal should be respected. Information about the patient's illness and its

Gerontological Considerations

There are significant demographic and clinical variations in the rates of ICU use at the end of life. Older patients and those with more chronic illnesses have the highest rate of end-of-life ICU use, consuming a significant proportion of its resources.

However, the proportion of patients who die in the ICU and the proportion of patients who receive

mechanical ventilation decline after patients reach age 85 (Seferian & Afessa, 2006).

A specific type of advance directive, the living will, does appear to have an impact on the type of care delivered to older patients (Silveira, Kim, & Langa, 2010). Older patients with living wills are more likely to receive comfort care and less likely to receive care in a critical care unit.

prognosis should then be delivered in a culturally relevant and appropriate manner to his family and the outcome of such discussions documented in his medical record.

Evidence is accumulating that frequent multidisciplinary team and family meetings to set goals for patient care during an ICU stay are most likely to result in an appropriate and coordinated plan of care and to reduce conflict. Boyle and associates (2005) and Arnold and associates (2010) recommend an initial meeting of the multidisciplinary team, patient (if possible), and family within 48 hours of the patient's ICU admission if the patient is at risk for death. Arnold emphasizes how important it is that members of the healthcare team prepare for the meeting by reviewing any issues, arranging a private location, and coordinating the timing so the important members of the team can be present. Coombs and Long (2008) suggest the review of issues include nurses' perspectives on the difficult or distressing daily patient care decisions that they are already making. During the meeting, what family members understand about the illness should be assessed, medical facts and treatment options should be reviewed, the patient's and family's values and goals should be discussed, a plan of care devised, and criteria to judge whether the plan is succeeding or not developed. Arnold notes that some clinicians use the following mnemonic SPIKES to facilitate family meetings:

- S = Set up the situation (Privacy, tissues, all appropriate participants)
- P = Find out patient or family Perception of the medical situation
- I = Invitation—find out how much information the family wants to hear
- K = Knowledge (Foreshadow, speak in simple terms, stop and check for understanding)
- E = Use Empathic statements when responding
- S = Strategize (Plan what is going to be done next)

Additional meetings may be held to judge the success or failure of the plan and to determine the goals of patient care. To avoid potential conflict, Boyle et al. (2005) also recommend using a screening tool to identify patients and families who are at high risk for conflict. These families are further evaluated by a social worker and recommended for additional interventions, such as regular family meetings or an ethics consult, if appropriate.

Limitation of Care

Once the family, patient, and healthcare team acknowledge that the patient may be dying, there are a number of treatments that may be withheld so the dying process is not prolonged. However, this does not mean the patient does not receive nursing care. The goal of the nursing care will gradually shift from an emphasis on cure to one on comfort but

most studies demonstrate that ICU nurses spend more time with their dying patients, not less. There may be no more that medicine can do to cure but there is a great deal that nursing can do to comfort and care.

Essential for Patient-Centered Care

When medical/technological care is going to be limited, it is essential to family members that members of the healthcare team, especially nurses, continue to show compassion and maintain the privacy and dignity of the patient (Nelson et al., 2010).

Common interventions that may be withheld include ventilation, dialysis, and enteral feeding. There is little controversy surrounding limiting the first two of these because both interventions are aggressive and intrusive. However, there has been a resurgence of controversy surrounding the limitation of food and fluid to the terminally ill. Because food is associated with nurturance and care in many cultures and religions, withholding food and fluid may seem unacceptable and unethical to some families and healthcare providers. Many people believe that food and fluids should always be provided to patients because they represent simple, ordinary means of care. This is certainly true for food and fluids provided to a patient who desires them by mouth. It is ordinary, expected care to provide oral nutrition to a patient who wishes to eat. However, at some point in the dying process, additional food and fluid appear to offer little benefit and may be very burdensome to a patient. Studies conducted over the past 30 years of aggressive nutritional support, including tube feeding, of patients with cancer have consistently demonstrated no benefits in length of survival (Emanuel, Ferris, & von Gunten, 2010). In fact, qualitative studies and practitioner experiences suggest that such aggressive feeding may harm the patient.

When Van der Riet, Good, Higgins, and Sneesby (2008) reviewed a series of studies on hydration at the end of life, they found that providing additional hydration had mixed results at best. Although some patients experienced less delirium when they were hydrated, most experienced discomfort from fluid retention manifested as edema, ascites, incontinence, urinary retention, pleural effusion, or pulmonary secretions. The natural process of the patient not wanting to eat or drink at the end of life may be helpful in relieving or preventing this constellation of symptoms. There is also evidence that dehydration, which may occur at end of life, does not cause suffering, provided that good oral hygiene is maintained (Van der Riet et al., 2008). More importantly, Emanuel, Ferris, and von Gunten (2010) assert that providing enteral or parenteral hydration to a dying patient does not reverse the patient's perception of thirst.

Palliative care specialists stress it is more important to manage symptoms such as thirst and hunger of the patient

than to adhere to any general formula. Byock (1997) includes in his promises to dying patients, “We will always offer you food and water.” This accentuates the importance of offering oral feedings but permits the patient to determine whether he can tolerate them. When the patient is unable to swallow or does not desire food and fluids but is complaining of thirst or a dry mouth, the nurse may teach caregivers to moisten the mouth and lips or provide ice chips. Good lip and mouth care as well as stopping medications with anticholinergic side effects have both been shown to decrease a dying patient’s perception of thirst (Emanuel et al., 2010).

Needs of the Families of Dying Patients

While the patient is probably dying, the nurse should attend to the needs of both the family and the patient. Truog et al. (2001) and Nelson et al. (2010) reviewed research studies and summarized the needs of families of patients dying in ICUs.

Families Need to Be with the Dying Patient

Evidence suggests providing a private room with unlimited visiting hours if possible. Restrictions on the number of people visiting and children and pets visiting should be lifted if at all possible. Dying patients emphasized that they were aware of and derived comfort and strength from the presence of their family (Nelson et al., 2010). Families may need to be given permission and shown how to communicate with the dying person. It is probable that the person hears more than he is able to respond to so no one should communicate over the patient. Everyone should talk to the person as if he was present. Nurses can encourage family members to say what they believe they need to say. For example, it is appropriate for a family member to tell a patient at this time that he has been forgiven (if that is the case) and that he is loved and will be remembered (Emanuel, Ferris, von Gunten, Hauser, & von Roenn, 2015). If, however, the family is unable to be with the patient, they should be reassured that it is acceptable for them to remain at home and that a nurse will stay with the patient until the end (if this is likely to be true).

There is ongoing discussion about whether palliative care should be continued in the environment of the ICU. Some argue that the high-tech environment is not appropriate for dying patients and that they would be better served on a palliative care or medical surgical unit. Nurses often argue that if the patient and family have formed a bond with the nursing staff, death is likely to occur soon, and the bed is not urgently needed, that allowing the patient to die in the ICU promotes continuity of care and comfort for

patient and family. Some ICUs have established palliative care packages to convert the high-tech environment into a more comfortable space for the dying patient and family.

Families Want to Be Helpful to the Dying Patient

The nurse can show the family how to provide care to the patient. It will depend on the family what care they will want to provide. A few families may want to bathe the patient. However, for most patients, the nurse can show the family how to moisten the patient’s lips, reposition the patient, or soothe the patient. The nurse might encourage the family to bring in music or read to the patient so they feel they have been able to provide some comfort.

Families Need to Be Assured of the Patient’s Comfort

Some healthcare providers and ethicists argue that if the patient is showing agonal respirations or having a “death rattle” even if the patient does not appear to be uncomfortable, it may be appropriate to medicate her for the benefit of the family (Truog et al., 2008). Otherwise, all appropriate medications for pain, nausea, and dyspnea should be administered aggressively. Even if there is no obvious source of pain, there is evidence that dying patients, especially older adults, may experience significant pain from arthritis so assessment with a validated instrument at regular intervals and management of pain is essential (Smith et al., 2010). Evidence-based strategies for management of symptoms in a dying patient will be discussed in detail later in the chapter.

Families Need to Be Informed about the Patient’s Condition

Family members indicate that nothing is as important as being able to get information every day about what is happening. They state that being in the dark “is like being in oil” (Nelson et al., 2010). There are many different ways to keep the family informed:

- Family members can be provided with an electronic pager when they leave the unit so they can be notified of any significant event or need for them in the unit.
- A family member should be provided with the telephone number of the unit and told that one member of the family is welcome to call if the family is anxious and needs an update.
- Families should be able to identify the physician and nurse who have primary responsibility for caring for the patient. A family member should feel free to ask the physician and nurse to explain the patient’s treatment at any time.

The information that is provided to the family about the patient's treatment must be in terms that they can understand. They are confused by medical jargon and the "alphabet soup" that sometimes accompanies medical diagnoses. They ask for information specifically about benefits and burdens in laymen's terms so they can make informed decisions about treatment options (Nelson et al., 2010).

It is not just the ability to receive information that is important to the family; the family must believe the information is accurate and trustworthy. To ensure this, members of the healthcare team should offer a consistent message to the family. Nurses and all healthcare providers should be careful of the way they use language; families will often interpret the phrase "He had a good day" as the patient is getting better. The team should discuss the likely prognosis and agree about what they will say to the family. However, they will have to acknowledge that medical prognoses are not always certain and help the family to understand that it is not possible to know exactly how things will progress or when. Tolerating such ambiguity can be difficult for families, and the support of clergy and social workers is often essential.

Families Need to Be Comforted and Allowed to Express Their Emotions

According to Morse (2001), people who are suffering have two distinct patterns of behavior requiring different types of nursing interventions. The first behavior pattern is enduring. In this phase, the family member has not acknowledged the full impact of the situation and is attempting to control her emotions and the situation. She may appear rigid, does not display any emotion, and interacts minimally with people around her. The second phase is emotional suffering. During that phase, the person acknowledges what is occurring and starts to respond emotionally. In order to move forward, the person must enter the phase of emotional suffering. However, any person may move between the phases depending on the situation. For example, a family member may display emotional suffering in the waiting room but enduring when in the room with the patient. The nurse should comfort people differently depending on which behaviors they are displaying.

Morse's research (2001) has indicated that while people are enduring, the nurse should comfort them by:

- Respecting the person's need for physical space and not touching or hugging him or her. Morse calls touching or being empathetic to the person at this stage "side swiping" because it will undermine the person's enduring.
- Reinforcing enduring strategies by saying: "You are doing such a good job"—talking to the patient, sitting by the bedside—or "You're holding up well."

- Explaining what is happening, offering anticipatory guidance, and providing potential time frames for what is occurring.
- Being with the person in silence.

When the person is displaying emotional suffering, Morse's evidence suggests that the nurse may comfort her by becoming engaged with the sufferer's emotion (2001). The nurse might utilize the following:

- Positioning (The nurse places herself directly in line with the person's vision so eye contact must be made between the two.)
- Touching (The nurse might hug, stroke, or pat the person to create a physical connection with her.)
- Appropriate use of verbal responses (The nurse might use therapeutic empathy, informing reassurance, humor/distraction, or confrontation.)
- Sharing in the experience (The nurse might convey sympathy, commiseration, compassion, consolation, or reflexive reassurance to the patient.)

Essential for Evidence-Based Practice

The nurse should choose how to approach the patient and family depending on the type of suffering they are manifesting (Morse, 2001).

Care of the Patient During Limitation and Withdrawal of Therapy

The withdrawal of medical interventions already in progress has been practiced in critical care units in the United States for the past 20 years. To understand why such a position is ethically justifiable, one must acknowledge that modern medicine is not capable of curing all conditions and that it is capable of imposing inordinate suffering on patients in attempts to prolong their lives. If an ongoing medical treatment is not capable of benefiting the patient, most ethicists do not view withdrawing the treatment as the direct cause of the patient's death. The cause of death in such a circumstance would be the relentless progression of the patient's disease. Thus, when an intervention is neither enhancing the care of the patient nor promoting the patient's recovery, the intervention may be withdrawn. Therapies that support a variety of physiologic functions may be withdrawn. The following is an abbreviated list:

Cardiovascular:	Vasopressors
	Pacemakers and implantable cardioverter/defibrillators
	Intra-aortic balloon pumps
	Ventricular assist devices
Respiratory:	Mechanical ventilation
	Supplemental oxygen
	Artificial airways
Renal:	Dialysis
	Hemofiltration
Neurological:	Cerebral spinal fluid drainage
Immunologic:	Treatment of infection with anti-infectives

The guideline suggested by Truog and others (2008) to determine if it is appropriate to continue an intervention at the end of life is whether the intervention will provide symptom relief; enhance functional status; or lessen emotional, psychological, or spiritual distress. Thus, simple laboratory tests or chest x-rays that might cause discomfort and would only provide unneeded information might be discontinued. However, a more sophisticated intervention such as cerebral spinal fluid drainage might be continued even when it is not curing the patient but it is providing symptom relief. The decision to continue each intervention is based on the specific patient's comfort, so an intervention that might be used for one patient might be withdrawn from another. For example, a patient might have an antibiotic continued if it decreased the pain and fever from otitis media, whereas a different patient might have an antibiotic withheld if there is no available access and the patient was not experiencing discomfort or fever from the infection. For interventions other than ventilator therapy, there is no evidence to believe that withdrawing them gradually results in any more comfort for the patient, so they are usually discontinued abruptly (Truog et al., 2008).

There has been an ongoing debate about the best method for withdrawing mechanical ventilation (Paruk et al., 2014). One approach, terminal weaning, advocated by Campbell, Bizek, and Thill (1999), involves the gradual reduction in settings on the ventilator: a change to synchronized intermittent mechanical ventilation with pressure support, a decrease in the respiratory rate, a reduction in the FIO₂, and finally discontinuation of the ventilator and provision of humidified room air by T piece. The other common approach, terminal extubation, involves removal of the endotracheal tube (ET) and discontinuation of the ventilator. Campbell and others (1999) recommended rapid terminal weaning, believing that it is a humane process in the unconscious patient and the patient is usually comfortable. Other authorities suggest that the family may find a terminal wean less morally troublesome than terminal

extubation and that it may be a better choice if the patient has large quantities of oral or respiratory secretions. Advocates of terminal extubation state that it does not prolong the dying process, that it cannot be mistaken for a therapeutic wean when the patient may be improving, and that when the patient has had the ET tube removed, he or she appears more normal to the family. There is not sufficient data to determine which method is preferable (Truog et al., 2008). Approximately 44% of physicians practice immediate withdrawal of ventilation, 33% prefer terminal weaning, and 12% prefer immediate terminal extubation (Paruk et al., 2014). Seventy percent of physicians state they eventually extubate the patient but the method they choose to reach extubation is dependent on the individual patient (Paruk et al., 2014).

Dialysis is another therapy that may be withdrawn either following a patient's request or when death appears imminent in a critically ill patient. Unlike terminal extubation, where the median length of patient survival is hours following withdrawal of therapy, the mean length of survival following discontinuation of dialysis is 8 to 9 days (Brody, Campbell, Faber-Langedoen, & Ogle, 1997) although some patients may survive for weeks. Most patients, nearly 65%, appear to die comfortable deaths, providing excessive IV fluid administration is avoided and the patient is permitted oral fluids as desired for comfort. If the patient does receive excessive fluid administration, he or she may develop dyspnea.

Families of patients who are about to have life-sustaining technologies withdrawn have been demonstrated to benefit from concrete and objective information about what is likely to occur. According to Kirchhoff, Palzkill, Kowalkowski, Mork, and Gretarsdottir (2008) that information can be delivered in three parts:

- Initial generic part that includes information about the uncertainty about how soon death will occur, assurance that care will continue to be provided, provision of privacy, information about possible physiologic changes, and an emphasis on priority management of any signs of distress
- Tailored part that is specific to the patient's physiologic status, likely signs and symptoms, and probable time until death.
- Final generic part that includes permission to the family to stay or leave as well as to show emotion and to be engaged with the dying patient

NURSING CARE

Once the decision is made that therapy will be limited or withdrawn and there will be no attempt to stop the dying process, the goal of patient care is comfort. The nurse needs to recognize that each time he cares for an actively dying

patient, it will be a unique event. The symptoms will vary and the nurse needs to adapt institutional guidelines and the recommendations described next to the needs of the specific patient.

Nursing Diagnoses

Intensive Care Unit Patient at the End of Life

- Anticipatory grieving related to perceived impending death
- Ineffective breathing related to the dying process
- Interrupted family processes related to illness of family member and possible conflict among family members
- Impaired verbal communication related to endotracheal intubation, dyspnea, and fatigue
- Risk for ineffective coping related to diagnosis of serious illness and personal vulnerability
- Risk for spiritual distress related to challenged beliefs and value systems

Essential for Patient Centered Care

Although a variety of protocols have been established for end-of-life care, it is most important that the nurse respond to the specific needs of each patient and family.

Promoting Comfort

The primary sources of discomfort for dying patients in the ICU are pain, thirst, and dyspnea (Puntillo et al., 2014). Anxiety and delirium may also be significant sources of discomfort. Unfortunately, a study by Singer and colleagues (2015) showed the prevalence of these symptoms in dying patients has increased over the last decade. Critical care nurses have the knowledge and ability to manage these symptoms and it is imperative they do so. When nurses focus on relieving the distress associated with these symptoms, they can significantly improve their dying patients' quality of life (Hermann & Looney, 2011). Opioids remain the mainstay for treating pain and pain is managed aggressively. Morphine is administered freely as noted in the "Commonly Used Medications" section in this chapter whenever indicated by a pain scale, such as the Critical Care Pain Observation Scale (CPOT). Thirst is managed with non-pharmacological interventions as discussed earlier in this chapter.

Managing Dyspnea

"Dyspnea is a form of suffering and is probably the most important symptom that must be relieved for patients dying in the ICU" (Truog et al., 2001, p. 2339). The responsive, cognitively intact patient should be questioned about breathlessness since self-report is the most reliable indicator of discomfort. However, more than half of patients are

unable to respond to questions with a simple yes or no (Campbell, Templin, & Walsh, 2010). To assess for dyspnea when the patient cannot self-report, Puntillo et al. (2014) recommend the use of the Respiratory Distress Observation Scale (RDOS), a behavioral scale (Campbell, Templin, & Walsh, 2010). The scale is composed of eight variables: heart rate, respiratory rate, restlessness, paradoxical breathing patterns, accessory muscle use, grunting, nasal flaring, and look of fear, with numerical ranking of each variable and total possible scores ranging from 0 to 16. Scores of 0-2 suggest little or no dyspnea while scores greater than 3 imply moderate to severe distress and the need for additional interventions. This scale is reliable and valid can be used to quantify the degree of dyspnea as well as to guide and evaluate interventions for dyspnea (Campbell & Templin, 2015). See Table 19-1.

When a ventilator is withdrawn, regardless of whether terminal wean or terminal extubation is utilized, most patients receive anticipatory dosing of sedatives or pain medication to prevent them from experiencing a sudden increase in dyspnea. "As a general rule, the doses of medications that the patient has been receiving hourly should be increased by two or threefold and administered acutely before withdrawing mechanical ventilation" (Truog et al., 2001). With appropriate dosing, patients should appear comfortable and have a score on the RDOS of less than 3. The optimal dose of medication is "determined by increasing the dose until the patient responds" (Brody et al., 1997, p. 653). Research indicates that administration of high doses of medication to relieve symptoms does not hasten patients' deaths (Campbell et al., 1999; Vitella, Kenner, & Sali, 2005; Emanuel, Ferris, von Gunten, Hauser, & von Roenn, 2015).

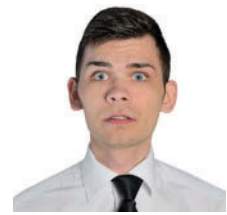
One possible way to use morphine and lorazepam to relieve dyspnea during terminal weaning or extubation is described under "Commonly Used Medications." However, at this time, there is no evidence to support one set of interventions over another (Truog et al., 2008). Additionally, it is essential that medication and dose be individualized to relieve the symptoms of the specific patient. The nurse may utilize nonpharmacological means in conjunction with medication to relieve dyspnea. Positioning the patient to encourage chest expansion may offer the patient some sense of relief. The most comfortable position for the patient will depend on her underlying condition and might be sitting up, leaning over a bedside table, or lying on a side. The administration of oxygen may enhance the comfort of some patients. However, there is evidence that patients who have been terminally extubated may be less responsive and appear more comfortable if they become hypoxic before becoming hypercarbic. Thus, some clinicians prefer not to provide oxygen to dying patients who are being weaned or extubated. If an electric fan is allowed in the unit, the sensation of coolness and air blowing on the

Table 19-1 Respiratory Distress Observation Scale

Variable	0 Points	1 Point	2 Points	Total
Heart rate per minute	<90 beats	90-109 beats	≤110 beats	
Respiratory rate per minute	≤18 breaths	19-30 breaths	>30 breaths	
Restlessness: nonpurposeful movements	None	Occasional, slight movements	Frequent movements	
Paradoxical breathing pattern: abdomen moves in on inspiration	None		Present	
Accessory muscle use: rise in clavicle during inspiration	None	Slight rise	Pronounced rise	
Grunting at end expiration: guttural sound	None		Present	
Nasal flaring: involuntary movement of nares	None		Present	
Look of fear	None		Eyes wide open, facial muscles tense, brow furrowed, mouth open, teeth together	
Total				

Instruction for use:

1. RDOS is not a substitute for patient self-report if able.
2. RDOS is an adult assessment tool.
3. RDOS cannot be used when the patient is paralyzed with a neuromuscular blocking agent.
4. Count respiratory and heart rates for one-minute; auscultate if necessary.
5. Grunting may be audible with intubated patients on auscultation.
6. Fearful facial expressions:



A Respiratory Distress Observation Scale for Patients Unable To Self-Report Dyspnea Margaret L. Campbell, Ph.D., R.N., F.A.A.N.,¹⁻³ Thomas Templin, Ph.D.,² and Julia Walch, R.N., M.S.N., F.N.P.³ (page 290)

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patient's face may produce some relief of breathlessness. The goal of collaborative management is to have a patient who appears to both healthcare providers and family to be dying peacefully without untoward respiratory difficulty.

Alleviating Anxiety

There are multiple causes of anxiety at the end of life, including poorly controlled pain, hypoxia, dyspnea, metabolic imbalances, medication side effects, and psychological distress. When the patient appears anxious or morphine alone does not adequately control a patient's dyspnea, a benzodiazepine may be used for its effect on anxiety, fear, and the autonomic responses that accompany dyspnea.

The nurse may utilize nonpharmacological means in conjunction with medication to relieve anxiety. It may help to dim the lights and silence the alarms in the patient's room, thus limiting stimulation around the patient. The family may be allowed to visit without restrictions and clergy or social services may be contacted to provide support and alleviate psychological distress. In a dying patient who is less responsive, anxiety may be caused by urinary

retention or constipation. Urinary retention might be treated with a Foley catheter but constipation in the immediate period near death is only treated if the patient is experiencing pronounced discomfort. It is not appropriate to search for metabolic causes for anxiety by performing laboratory tests at this time because the procedures may inflict pain and they are not usually successful in establishing the cause (Kazanowski, 2012). The goal of collaborative management is to utilize pharmacological and nonpharmacological interventions so the patient appears to both healthcare providers and family to be dying peacefully without undue restlessness, anxiety, or fear.

Reflect On

What nursing interventions did you find provided the most comfort to the dying patients for whom you have provided care? What nursing interventions provided the most comfort to their families? Were the beneficial interventions similar for different patients or did the interventions that provided the most comfort differ from patient to patient?

Commonly Used Medications

Morphine Sulfate—an Opioid

Introduction

Morphine is the preferred analgesic for use with a dying patient in ICU because it is potent, has a wide therapeutic range, acts rapidly when given intravenously, is relatively inexpensive, and may induce a feeling of well-being. An alternative, if needed, would be fentanyl, but much lower doses would be utilized because of its high potency.

Desired Effects: Desired effects that should become apparent 5 minutes after IV administration and last for 1½ to 2 hours (longer in patients with renal or hepatic impairment) include analgesia, sedation, respiratory depression, vasodilation, and relief of air hunger.

Nursing Responsibilities:

- Anticipatory dosing prior to weaning or extubation. The usual dose is 5 to 10 mg IV or if the patient has been receiving morphine, two to three times the patient's usual bolus dose.
- Initiation of an ongoing morphine infusion, usually at a rate of 50% of the bolus dose/hour, immediately following the anticipatory bolus dose.
- Assessment of the patient's level of calmness/agitation, physical movement, muscle tone, heart rate, respiratory effort, and facial tension. If the patient is displaying

symptoms indicative of discomfort, the morphine dose should be titrated up slowly every 5 to 15 minutes with the goal of eliminating tachypnea, coughing or choking, agitation, excessive movement of the head and torso, diaphoresis, and grimacing.

- Consistent documentation of the patient's symptoms as an indication for additional morphine administration as well as a description of the patient's response to the initial dose and any necessary increases in dose.
- Morphine might be avoided in patients with concurrent renal and hepatic failure because the accumulation of the metabolites can have a neurotoxic effect increasing the potential for terminal delirium (Brecher & West, 2013). Emanuel and associates (2015) suggest stopping continuous infusions or routine dosing of morphine if the patient becomes anuric and using fentanyl as an alternative.

Side and/or Toxic Effects:

- Hypotension and decreased level of consciousness are anticipated side effects and should not result in a reduction in the dose of morphine.
- Morphine is associated with histamine release that may result in urticaria, pruritus, or flushing and may be relieved with antihistamine therapy.

Fostering Patient- and Family-Centered Care

It is often difficult for the family to wait with the patient after life-sustaining technology has been withdrawn. Family members often anticipate that the patient will die immediately after the removal of the technology. However, this is frequently not the case. In one study the median patient

survival was 2.3 hours, and in another 3.5 hours following terminal weaning from a ventilator. The family usually needs assistance coping while the patient is actively dying. Most intensive care units try to have patients remain in the same bed in the same unit after life-sustaining technology is withdrawn so they can receive care from nurses who have known and cared for them previously (Kirchhoff & Kowalkowski, 2010). ICU nurses are more likely than other

Commonly used Medications

Lorazepam—a Benzodiazepine

Introduction

Lorazepam is more rapidly acting, less expensive, and may have fewer respiratory and cardiovascular depressive effects than other benzodiazepines such as diazepam or midazolam but has no analgesic effect.

Desired Effects: Desired effects that should peak about 30 minutes after IV administration include sedation and decreases in anxiety and restlessness. Benzodiazepines have a synergistic sedative effect with morphine and 75% of patients undergoing withdrawal of life support receive a

combination of the two drugs. According to Brody et al. (1997), benzodiazepines should be first-line pharmacological therapy for anxiety in the dying patient.

Nursing Responsibilities:

- Administration of an anticipatory bolus dose (often two times the patient's normal bolus dose given IV) prior to terminal weaning or extubation
- Documentation of the patient's level of anxiety and response to medication
- Titration of the dose dependent on the benzodiazepine utilized and the patient's symptoms

healthcare providers to be present during the withdrawal of technology and are likely to continue providing care to the patient and family following withdrawal until death (Kirchhoff & Kowalkowski, 2010). During this time, nurses may offer assistance to the family and patient by:

- Creating a quiet environment with soft lighting and turning off or dimming all monitors and alarms in the patient's room
- Assuring the family that the patient is being provided with all possible pharmacological and physical means to promote comfort
- Assessing the patient and family frequently and responding to needs as soon as possible, thus reassuring the patient and family that the patient will not be abandoned
- Explaining the signs of the dying process that the patient is exhibiting and providing some anticipatory guidance about when death might occur
- Acknowledging how difficult it may be for the family to tolerate the uncertainty and to wait while the dying process progresses
- Allowing the family to choose the option not to remain with the patient
- Offering to help arrange for religious or cultural observances that might comfort the patient and family

The family also needs to be aware that a small subset of patients, between 6% and 11%, do not die for a significant period following extubation and a few patients may even survive for weeks following hospital discharge.

Essential for Evidence-Based Practice

Studies have indicated that family members would prefer to have monitors turned off while the patient is dying but are afraid to ask (Kirchhoff et al., 2008). Thus, nurses should ask the family what they would prefer or act proactively to turn monitors off in a dying patient's room.

Meeting the Spiritual Needs of the Dying Patient and Her Family

The SCCM recommends that the spiritual needs of patients be assessed by the healthcare team, and any findings that affect health and healing be incorporated into the plan of care (Davidson et al., 2007). However, when Emecoff, Curtin, Buddadhumaruk, and White (2015) assessed 13 ICUs, they found that spiritual or religious considerations were addressed only 20% of the time during goals of care discussions. The SCCM defines *spiritual support* as encouraging and respecting prayer and adherence to cultural traditions so patients and families may cope with

illness, death, and dying (Davidson et al., 2007). Hospitals may have formal spiritual counseling by a chaplaincy service or more informal efforts by staff to accommodate the spiritual traditions and cultural needs of patients and families.

In either case, at the end of life, it is important to offer the patient and family an opportunity to consider the spiritual dimension of life. Some of the spiritual issues that a patient or family may want to explore are:

- The role of suffering in life
- Questions about meaning, purpose, and hope
- Ethical considerations
- Grief and loss issues

Patients and families may also need someone to assist them with either religious or nonreligious rituals of healing and closure. A hospital chaplain can assist with the appropriate rituals or may find a suitable religious adviser. However, if the nurse is comfortable and the patient and/or family requests it, the nurse might pray with the patient or assist with the religious ritual, especially if she is from the patient's cultural and religious tradition.

Conflict at the End of Life

Approximately 30% of the time, the healthcare team, patient, and family are not able to agree about how best to care for the dying patient (Gupta, Goyal, Chauhan, Mishra, & Bhatnager, 2008). The patient, a member of the patient's family, or a member of the healthcare team may want to continue lifesaving treatment even though the others involved in treatment decision-making believe the patient is dying. Infrequently, conflict may arise when either the patient or a family member asks that the patient be assisted to die.

Continuation of Life-Sustaining Treatment

Most of the time, when death is imminent, consensus can be reached among the healthcare providers, the patient, and the family so life-prolonging interventions are not provided and the patient is able to die peacefully. However, sometimes consensus cannot be reached and conflict develops about what life-sustaining technology to provide. One underlying reason for the development of conflict is that trust was not established early in the hospitalization and the family does not believe what the healthcare providers are saying. If a nurse suspects that lack of trust is the reason for conflict, he might validate his suspicion by saying, "From the experiences you describe, I imagine it may be difficult for you to trust us." If lack of trust is the primary

reason the patient and family want to continue life-sustaining care, the nurse should act to reestablish trust; the nurse might emphasize what is being done for the patient, and the nurse can offer to facilitate getting another opinion from a healthcare provider who is trusted.

Disagreement about treatment may also result when decision-makers do not share a common understanding of the patient's prognosis and therefore a common goal for the patient's care (Gupta et al., 2008). Arnold and others (2010) counsel looking for and resolving possible sources of misunderstanding. Possible sources of misunderstanding include:

- The family has not been told anything about the patient's situation.
- The information the family was provided was not in a format that they could understand.
- The family has been receiving conflicting information or mixed messages from multiple members of the healthcare team.

Because nurses are more consistently available to patients and their families, they are in a unique position to be able to uncover and correct these misunderstandings. Nurses can begin by asking the family "What do you understand is happening to the patient?" and exploring how the family came to that conclusion. Nurses can build on what the family understands is happening and "present a realistic picture" clarifying what the "silent patient" is experiencing (Robichaux & Clark, 2006). To help clarify the goal of therapy, a nurse might ask, "What do you think will likely happen if we continue this therapy?" Nurses need to find the time to learn what the patient and family wish would happen and help them to separate their wishes from the actuality.

Sometimes the issues underlying the conflict about continuation of life-sustaining therapy are difficult to uncover or resolve. The conflict may result from a family member or a healthcare provider experiencing guilt about the care of the patient. For example, the spouse or physician may have ignored a symptom, which if treated earlier could have resulted in a better outcome for the patient. The family is likely to be under stress and in denial; it may be too soon for them to accept reality and reach a decision. Simply allowing a bit more time before asking the family to make a decision may help to resolve the conflict if this is the problem. An evidence-based mnemonic that can be used to uncover and respond to the family's emotions is NURSE:

- **Name the emotion:** You seem (insert appropriate emotion—e.g., worried)
- **Understand the emotion:** I can see that this is difficult for you.
- **Respect:** I can see that you are trying to _____.

- **Support the participants:** I (or we) will help you to _____.
- **Explore possibilities:** Tell me what options you can envision.

Arnold et al. (2010) insist that conflict arises not from family members' misunderstandings about prognosis, but instead from the meanings and values that they place on the various types of support. For example, the patient and family may come from a religious tradition that does not permit the withdrawal of life-sustaining therapy. Many observant Orthodox Jews believe that everything possible must be done to prolong a patient's life, and termination of a life-sustaining-treatment such as mechanical ventilation is prohibited (Carlet et al., 2004). Or the family may be hoping for and believing that a miracle will occur. In such situations, it may be very difficult to reach a consensus about limiting care.

The situation becomes even more complex when a family member demands an intervention that the healthcare team considers to be futile. Medical futility may be defined as any treatment that is without benefit (that does not provide palliation, restoration, or cure) to the patient. The problem with such a definition is that most life-sustaining interventions can be shown to have some very small possibility of benefit and the futility of their use is not always obvious to family members (Pfeifer & Kennedy, 2006). In addition, Zier et al. (2009) have shown that surrogate decision-makers doubt physicians' abilities to predict medical futility. For these reasons alone, arguments based on medical futility are unlikely to convince families and surrogates that care should be withheld once conflict has developed.

However, Day (2009) believes there is another, more important reason why futility arguments do not affect surrogates' decisions. She believes that the patient or family is making a distinction between what is effective (which can be determined empirically and is meant by medical futility) and what the family believes is beneficial (which is a question of values and goals). Day states that families or patients seeking futile care have defined a goal that the healthcare team does not support. She uses the example of a patient who wants his life sustained despite any amount of suffering using every possible medical technology for every possible moment. The patient's goal is clear and to reach that goal his chosen interventions are useful. However, the healthcare team can rarely support such a goal and the chosen interventions.

On occasion, it is a member of the healthcare team or the culture of a specific critical care unit that does not support the withdrawal of medical interventions. Using technology to rescue a patient each time a new complication develops is more common in surgical ICUs where the focus may be on treating everything and rarely, if ever, acknowledging that a

patient may die (Nelson et al., 2012). Some surgeons believe they have contracted for aggressive postoperative care informally with patients pre-operatively by explaining to their patients that they would decline to operate on them if the patients wanted to limit postoperative treatments (Schwarze, Redmann, Alexander, & Brasel, 2013). In such an environment, instituting withdrawal of interventions is difficult and even obtaining a palliative care consult can be problematic. Aslakson and colleagues (2012) found that when surgeons did try to open discussions about palliative care with their patients, the discussions were often quick, inadequate, and ineffective.

The healthcare providers, especially the nurses, who are continuing the treatment, realize how extremely slim the possibility of long-term benefit is and how much the patient appears suffering while enduring it. The nurses providing the care are also likely to be considering their fiduciary responsibilities to their current and future patients (Day, 2009). Therefore, they may object to continuing to provide the extensive care requested. In fact, the continuation of life-sustaining therapies in patients who are dying is one of the major sources of moral distress for critical care nurses (Browning, 2013).

When the healthcare team, patient, and family cannot reach a consensus about the continuation of life-sustaining treatment, the issue should be referred to the hospital's ethics committee. The Joint Commission requires hospitals to have an ethics committee to aid in the resolution of such difficult issues. Some states, such as Texas, have laws that delineate procedures that must be followed prior to, during, and after ethics committee deliberations about futile treatment. After reviewing the case, the ethics committee might recommend either supporting the continuation of life-sustaining therapy or limiting treatment. In Texas, if the ethics committee recommends discontinuation of life-sustaining treatment, the patient and proxy must receive written notification of a 10-day treatment limit with an option to request transfer to another facility. If they cannot find another facility to accept the patient, then the life-sustaining treatment is discontinued after the 10th day (Pfeifer & Kennedy, 2006).

Assisted Death

When death is assisted, the healthcare provider purposely takes an action that directly results in the patient's death. If it is **voluntary euthanasia**, the patient requests the action be taken to end his life; in **involuntary euthanasia**, the action to end the patient's life is taken without the patient's consent. A study by Emanuel, Fairclough, Daniels, and Clarridge (1996) suggests that approximately 12% of oncologists have assisted a patient to die but the study does not distinguish between voluntary and involuntary euthanasia. In one of the first published studies of nurses' practices of

assisted death, Matzo and Emanuel (1997) compared oncology nurses' and oncology physicians' practices. More physicians than nurses assisted their patients in dying (11% versus 1%). However, nurses were more likely than physicians to have performed patient-requested euthanasia (4% versus 1%). In a controversial study by Asch (1996) regarding assisted death in the ICU, nurses were asked about their practices regarding the use of large doses of narcotics. By the way the question was worded it may have been difficult for respondents to determine if they were being asked about pain management or about assisting a patient to die. However, Asch reported that 16% of critical care nurses had assisted their patients to die.

Most healthcare providers who assist a patient's death state that they are acting out of compassion. They argue that they assisted a patient's death because the patient's life was no longer worth living and that the burdens of the life far outweighed the benefits. They cite such reasons as the patient was suffering unbearably or the family and healthcare team were unable to reach a decision to withdraw futile, life-sustaining treatment so the patient was forced to suffer needlessly. They may also declare that the patient would never have wanted to live in her or his current condition and would have requested interventions be halted if she or he had a voice.

Although the public supports the general concept of assisted death by physicians, oncology patients in pain usually do not (Emanuel et al., 1996). Perhaps the reason is that people who are dying, whose quality of life may appear limited to others, do not want to have healthcare providers empowered to determine if their lives are worth living or not. In fact, only 3% of Americans would want their healthcare providers to make a decision and commit involuntary euthanasia on their behalf if they were not conscious and could not express their wishes (Blendon, Szalay, & Knox, 1992). Thus, there is little support for those healthcare providers who contend that in assisting their patients to die they are acting in accordance with their patients' wishes.

There are numerous other arguments against the use of assisted death. First, most medical and nursing professional organizations, including the American Nurses Association and the American Association of Critical-Care Nurses, state that their members have a primary duty not to harm, which prohibits them from intentionally causing a patient's death. Most religious groups profess that human life should be respected and healthcare providers should never intentionally cause their patients' death. Advocates for the poor and homeless wonder how long it would take before sick, vulnerable, poor people would begin to be euthanized if euthanasia were allowed in this society, where 42 million Americans lack health insurance and cost containment is becoming one of the most important medical values. Hospice nurses question whether palliative care would

continue to receive adequate funding and referrals, or whether patients would experience pressures to die quickly. Caring for a deteriorating or dying person takes time, a scarce commodity in our society. Finally, assisting a patient to die is illegal in most of the United States.

It is not clear how often ICU nurses are asked by patients or their families to end a patient's life. Studies of Japanese and Australian nurses (Tanida et al., 2002) indicate

that 50% of nurses have been asked at least once to end a patient's suffering by ending his life. Thus, it is appropriate for a nurse to think about how she would respond to the request by either the patient or a family member to end the life of a suffering patient. One possible response is, "I cannot help you to end your suffering by ending your life, but I will work to relieve your suffering and to help you live the rest of your life with dignity."

End of Life Summary

Although some critically ill patients die unexpectedly or following CPR, the majority of ICU patients die following withdrawal or withholding of life-sustaining technology. Caring for a grieving family and a dying patient takes

excellent communication skills. The nurse must be able to help the patient and his family understand that the patient is probably dying and make decisions about the type of care the patient desires.

Why/Why Not?

A nurse is caring for a 72-year-old patient with COPD, heart failure, and pneumonia who has recently been extubated and comfort measures have been instituted. The patient has a morphine drip infusing at 4 mg/hr and a lorazepam infusion at 2 mg/hour. The patient is able to moan but is unable to speak. Her vital signs at the time of extubation were: BP 100/60, P 88, RR 16, and they are currently BP 84/55, P 100,

RR 26. The patient is slightly restless and displaying some use of accessory muscles but no nasal flaring or look of fear. Her RDOs is scored as a 4. The nurse can titrate the dose of morphine and lorazepam for patient comfort. Should the nurse increase the dosage? Why or why not?

See answers to Why/Why Not? in the Answer Section.

Case Study

Armand Gregoire is a 78-year-old widower whose only significant medical history was a permanent pacemaker placed 4 years previously for a sinus bradycardia of 38–42 beats per minute. The pacemaker had been placed so he could continue to scuba dive. His only over-the-counter (OTC) medication was an occasional Tylenol for joint pain and he was taking no prescribed medications.

At 6 a.m. on a Friday morning Armand experienced severe left arm pain. He drove himself to the Emergency Department (ED) of the nearest hospital, but by the time he arrived the pain was crushing and he was showing signs of heart failure. He was prepared immediately for cardiac catheterization then transferred to the operating room (OR) for a quadruple bypass. After transfer to the cardiac surgical ICU, he did not regain consciousness. Over the next 2 days, he developed worsening heart failure as well as acute renal failure and could not be weaned from the ventilator.

Armand had never completed an advance directive. So the cardiac surgeon approached Armand's next of kin, his stepson, for permission to begin dialysis. The stepson's response was immediate, "My Dad would not want to be dialyzed. He wouldn't even want that breathing machine. He's been depressed, saying he wants to die and waiting for my Mom to come and get him for the past 4 years. No, I won't consent to it."

The cardiac surgeon believes that Armand has cardiogenic shock from which he can recover. The surgeon wants to start dialysis without the stepson's approval because Armand never completed an advance directive. The surgeon says that if he doesn't provide dialysis, he would not be giving Armand every chance to live and would feel as if he were killing Armand. However, the stepson has now decided that not only does he not want his father to be dialyzed but he wants the ventilator to be withdrawn as well.

1. What should the role of the nurse who is caring for Armand be?
2. What further information does the nurse caring for Armand need before making a decision about what to do?
3. How should the nurse decide what ought to be done?
4. What are the nurse's possible courses of action?
5. What do you believe the nurse ought to do in this circumstance?

See answers to Case Studies in the Answer Section.

Chapter Review Questions

- 19.1 Do you believe that families should be allowed to witness resuscitation attempts on their family member? Why or why not?
- 19.2 What are the advantages of advance directives?
- 19.3 Why are advance directives currently of limited use for patients in critical care?
- 19.4 What are the needs of families of patients who are dying in ICU and how can ICU nurses meet those needs?
- 19.5 Why do most healthcare providers believe that it is ethical to limit and/or withdraw medical interventions from critically ill patients?
- 19.6 What is the difference between terminal weaning and terminal extubation?
- 19.7 How do most healthcare providers differentiate between withdrawal of medical interventions and assisted death?
- 19.8 How might a nurse act to resolve conflict about goals of care at the end of life?

See answers to Chapter Review Questions in the Answer Section.

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Appendix A

Normal Laboratory Values

According to the National Council of State Boards of Nursing, nurses should know the normal values and

recognize deviations from normal for the following laboratory tests.

Laboratory Test	Normal Value
Arterial Blood Gases	
pH	7.35 to 7.45
PaO ₂	75 to 100 mmHg
SaO ₂	93% to 96%
PaCO ₂	35 to 45 mmHg
HCO ₃	24 to 28 mEq/L
Mixed Venous Gases	
SvO ₂	60% to 75%
Serum Levels	
Glucose (fasting)	60 to 100 mg/dL
Blood urea nitrogen (BUN)	5 to 25 mg/dL
Creatinine	0.5 to 1.5 mg/dL
Sodium	135 to 145 mEq/L
Potassium	3.5 to 5.3 mEq/L
Magnesium	1.5 to 2.5 mEq/L
or	1.8 to 3.0 mg/dL
Total calcium	9 to 11 mg/dL
Phosphorus/phosphate	1.7 to 2.6 mEq/L
or	2.5 to 4.5 mg/dL
Lactate	
Arterial blood	0.5 to 2.0 mmol/L
Venous blood	0.5 to 1.5 mmol/L
Cholesterol	
Total	desired less than 200 mg/dL
LDL	60 to 160 mg/dL
HDL	29 to 77 mg/dL
Albumin	3.5 to 5.0 g/dL
Total protein	6.0 to 8.0 g/dL
ALT (SGPT)	10 to 35 units/L
AST (SGOT)	8-35 units/L
Ammonia	5 to 45 mcg/dL
Bilirubin	
Indirect	0.1 to 1.0 mg/dL
Direct	0.1 to 0.3 mg/dL
PT	11 to 15 seconds
INR	for anticoagulant therapy utilized after the person has been stabilized on anticoagulant therapy—depending on the reason for anticoagulation 2.0 to 3.5 × the control

Laboratory Test	Normal Value
PTT	60 to 70 seconds
APTT	20 to 35 seconds
Bleeding time (Ivy method)	3 to 7 minutes
Hemoglobin A ₁ C (nondiabetic)	2% to 5%
Hematocrit	
Male	40% to 54%
Female	36% to 46%
Hemoglobin	
Male	13.5 to 18 g/dL
Female	12 to 15 g/dL
Erythrocyte sedimentation rate (ESR)	
Wintrobe method	
Male	0 to 9 mm/hr
Female	0 to 15 mm/hr
Platelet count	150,000 to 400,000 μ L
White blood cell count	4,500 to 10,000 μ L (mm^3)
Differential	
Neutrophils (total)	50% to 70% or 2,500 to 7,000/ μ L (mm^3)
Segmented	50% to 65% or 2,500 to 6,500/ μ L (mm^3)
Bands	0% to 5% or 0 to 500/ μ L (mm^3)
Eosinophils	1% to 3% or 100 to 300/ μ L (mm^3)
Basophils	0.4% to 1% or 40 to 100/ μ L (mm^3)
Monocytes	4% to 6% or 200 to 600/ μ L (mm^3)
Lymphocytes	25% to 35% or 1,700 to 3,500/ μ L (mm^3)
Urine	
pH	4.5 to 8 (average 6)
Specific gravity	1.005 to 1.030
	1.015 to 1.024 with normal fluid intake
White blood cell count	3 to 4
Protein	2 to 8 mg/dL

Appendix B

Medication Infusion Calculations

It is important to recalculate all IV infusions to be certain they are infusing at the appropriate rate even though most IV pumps now perform the calculations. The pump may be in error or the person who programmed the pump may have made an error.

The two methods that are commonly used to calculate the rate and dosage of medication infusions are proportion and dimensional analysis. **Using a consistent method for calculating and not switching between methods is highly recommended because switching methods is likely to cause a calculation error.** In the sections that follow, the same medication infusions are calculated using each method. **Please choose the method you use and review the calculations only according to your preferred method.**

Proportion Method

To calculate medication infusion rates, the following formula can be utilized:

$$\text{Dose} = \text{Rate} \times \text{Concentration}$$

With two of the factors, the third can always be calculated. It is rarely necessary to calculate concentration because that is nearly always given. Dose is often prescribed so the common way this formula is utilized is to determine a rate. To calculate the rate, the formula becomes:

$$\frac{\text{Dose}}{\text{Concentration}} = \text{Rate}$$

In addition, sometimes it is necessary to add conversion factors for minutes/hour, micrograms/milligrams, and/or kilograms.

Dose

The drug to be administered determines the dose. It may be prescribed in mg/hr, mg/min, or mcg/kg/min. It is necessary to review the prescriber's order and normal dosing for the medication to determine the dose.

Rate

Because IV pumps are utilized to administer these medications, the rate will always be in mL/hr. When the dose and concentration are known, then it is the rate that is being calculated (which is most often the case). If the rate is

known and it is the dose that is uncertain, the rate may be identified by checking the IV pump. Depending on the medication and the specific pump, pump rates may be set at 0.1-mL intervals.

Concentration

The pharmacy will always provide the drug in a known concentration, and the concentration should be clearly visible on the label of the IV bag or bottle. The concentration may be in mg/mL, units/mL, or mcg/mL. It is particularly important to verify the concentration of the drug at the beginning of a shift or whenever a new bag or bottle is hung.

Calculation Units/Hour

Example: Heparin

The prescribed dose is 1,200 U/hr of heparin. The label states that there are 20,000 U/500 mL of fluid. To calculate the rate to set the IV pump, complete the following steps:

The first step is optional: Many people prefer to simplify the concentration before they begin calculations. Often the simplified concentration will be available on the IV label.

$$\frac{20,000 \text{ U}}{500 \text{ mL}} = \frac{40 \text{ U}}{1 \text{ mL}}$$

When the formula $\text{Dose} = \text{Rate} \times \text{Concentration}$ ($D = R \times C$) is used, to calculate rate, the formula must be converted to:

$$\frac{\text{Dose}}{\text{Concentration}} = \text{Rate/hour} \quad \text{or} \quad \frac{1,200 \text{ U}}{40 \text{ U/mL}} = \text{Rate/hour}$$

By canceling out one of the zeros in the units in the top and bottom of the fraction, the fraction can be simplified to

$$\frac{120}{4 \text{ mL}} = \text{Rate/hour}$$

After dividing, the rate is determined to be 30 mL/hr

Calculation Milligrams/Hour (mg/hr)

Example: Midazolam

Calculating milligrams per hour is similar to calculating units per hour. An example would be a midazolam infusion. If a midazolam infusion was running at 5 mL/hr

(5 mg/hr) and the dose was to be decreased by 20% to determine the minimum effective infusion rate, how should the dose and rate be adjusted?

To determine 20% of 5, multiply 5 by 0.2 ($5 \times 0.2 = 1$)

To reduce the rate by 20%, subtract 1 from 5 ($5 - 1 = 4$)

The new dose for the infusion is 4 mg/hr

The formula needed for the adjusted rate is:

$$\frac{\text{Dose}}{\text{Concentration}} = \text{Rate/hour}$$

$$\text{or } \frac{4 \text{ mg}}{1 \text{ mg/mL}} = \text{Rate hour}$$

Therefore, the rate is 4 mL/hr

Calculation Milligrams/Minute (mg/min)

Example: Lidocaine

To calculate the rate for medications delivered in milligrams per minute, it is necessary to add an additional step and a conversion factor for minutes to hours because the medication is prescribed in mg/min and IV pumps are set to infuse in mL/hr.

A lidocaine infusion is prescribed to run at 3 mg/min. The available concentration is 1 g in 250 mL. This is equivalent to 1,000 mg/250 mL and can be simplified to 4 mg/mL.

Rate (mL/hr)

$$= \frac{\text{Dose} \times \text{conversion factor (60 minutes/hour)}}{\text{Concentration}}$$

$$\text{Rate (mL/hr)} = \frac{3 \text{ mg/min}}{4 \text{ mg/1 mL}} \times 60 \text{ minutes/hour}$$

After division and canceling of minutes and milligrams,

$$\text{Rate} = 45 \text{ mL/hr}$$

Calculation Micrograms/Minute (mcg/min)

Example: Norepinephrine (Levophed)

This calculation is similar to milligrams per minute except it is necessary to perform an additional step and convert from milligrams to micrograms. This time, instead of calculating for rate, the example will demonstrate how and why one might calculate for dose.

A nurse has been titrating a Levophed drip upward to maintain a MAP greater than 60 for a patient in septic shock. The concentration of the Levophed is 4 mg in 500 mL and the medication is currently infusing at

30 mL/hr. How many mcg/min of Levophed is the patient receiving?

The concentration can be simplified by converting milligrams to micrograms and reducing the fraction. It is 4,000 mcg/500 mL or 8 mcg/mL.

The appropriate formula because it is the dose that is desired is:

$$\text{Dose} = \frac{\text{rate} \times \text{concentration}}{\text{conversion factor (60 minutes/hour)}}$$

$$\text{Dose (mcg/min)} = \frac{30 \text{ mL/hr} \times 8 \text{ mcg/mL}}{60 \text{ minutes/hour}}$$

After division and canceling of units,

$$\text{Dose} = 4 \text{ mcg/min}$$

Calculation Micrograms/Kilogram/Minute (mcg/kg/min)

Example: Dopamine

A physician has prescribed a dopamine infusion to increase a patient's blood pressure. The infusion is prescribed to begin at 5 mcg/kg/min. The patient weighs 70 kg and the dopamine is available 400 mg/250 mL. At what rate should the nurse begin the infusion?

There are numerous additional steps needed for this calculation, including converting milligrams to micrograms, adjusting for the patient's weight, and converting minutes to hours (to set the IV pump).

To simplify the concentration 400,000 mcg/250 mL = 1,600 mcg/mL

$$\text{Rate (mL/hr)} = \frac{\text{dose (5 mcg/kg/min)} \times \text{pt's weight (70 kg)} \times 60 \text{ min/hr (conversion)}}{\text{concentration (1,600 mcg/mL)}}$$

$$\text{Rate} = 13.125 \text{ mL/hr}$$

Dimensional Analysis

The same elements that are important for calculating dosages or rates of medicated infusions by the proportion method are also needed for calculating by dimensional analysis. These are the prescribed (or currently infusing) dose, the rate of infusion, and the concentration of the drug.

Dose

The drug to be administered determines the dose. It may be prescribed in mg/hr, mg/min, or mcg/kg/min. It is necessary to review the prescriber's order and normal dosing for the medication to determine the dose.

Rate

Because IV pumps are utilized to administer these medications, the rate will be in mL/hr. When the dose and concentration are known, then it is the rate that is being calculated (which is most often the case). If the rate is known and it is the dose that is uncertain, the rate may be identified by checking the IV pump. Depending on the medication and the specific pump, pump rates may be set at 0.1-mL intervals.

Concentration

The pharmacy will always provide the drug in a known concentration, and the concentration should be clearly visible on the label of the IV bag or bottle. The concentration may be in mg/mL, units/mL, or mcg/mL. It is particularly important to verify the concentration of the drug at the beginning of a shift or whenever a new bag or bottle is hung.

When calculating in dimensional analysis, always start with what is prescribed (or the rate at which the infusion is running if you are trying to determine the dose). Next, add the concentration. You will then need to add additional conversion factors depending on the specific calculation. Conversion factors are added as fractions in such a fashion that the unnecessary units cancel out. You stop adding conversion factors when all that is left is the information needed to calculate the dose.

Commonly used conversion factors are:

- Conversion for the weight of one patient: 1 patient/weight in Kg
- Conversion for mgs to mcg: 1 mg/1000 mcg
- Conversion for minutes to hour 60 min/1 hr

Calculation Units/Hour

Example: Heparin

The prescribed dose is 1,200 U/hr of heparin. The label states there are 20,000 U/500 mL of fluid. Calculate the rate to set the IV pump (mL/hr):

To solve, start with what is ordered (1,200 Units/hr) then add the concentration to the equation (500 mL contains 20,000 Units). The concentration should be entered in such a manner that the Units will cancel out when the equation is solved.

$$\frac{1,200 \text{ Units}}{1 \text{ hr}} \times \frac{500 \text{ mL}}{20,000 \text{ Units}}$$

To solve

1. multiply 1,200 by 500 which equals 600,000.
2. divide 600,000 by 20,000 which equals 30
3. Cancel out the units. (Units cancel out leaving mL/hr)

The rate is determined to be 30 mL/hr.

Calculation Milligrams/Hour (mg/hr)

Example: Midazolam

Calculating milligrams per hour is similar to calculating units per hour. An example would be a midazolam infusion. If a midazolam infusion was running at 5 mL/hr (5 mg/hr) and the dose was to be decreased by 20% to determine the minimum effective infusion rate, how should the dose and rate be adjusted?

To reduce the current dose of 5 mg/hour by 20%:

- 1) $5 \text{ mg} \times 20\% (0.2) = 1 \text{ mg}$
- 2) $5 \text{ mg} - 1 \text{ mg} = 4 \text{ mg/h}$

The new dose is determined to be 4 mg/h.

To solve, start with the new dose, 4 mg/hr and add the concentration of the infusion (100 mg/100 ml or 1 mg/1 ml). The concentration should be entered in such a manner that the Units will cancel out when the equation is solved:

$$\frac{4 \text{ mg}}{\text{hr}} \times \frac{1 \text{ mg}}{1 \text{ ml}}$$

The new rate is determined to be 4 ml/h.

Calculation Milligrams/Minute (mg/min)

Example: Lidocaine

A lidocaine infusion is prescribed to run at 3 mg/min. The available concentration is 1 g in 250 mL.

To solve, start with the prescribed dose (3 mg/min), add the concentration as a fraction (250 mL contains 1 Gm), next add the conversion factor for the number of milligrams in a gram (1 Gm is 1,000 mg), and finally the conversion factor for the number of minutes in an hour (60 mins/1 hr). Make certain to enter the conversion factors in such a way that the unnecessary units cancel out.

$$\frac{3 \text{ mg}}{1 \text{ min}} \times \frac{250 \text{ mL}}{1 \text{ Gm}} \times \frac{1 \text{ Gm}}{1000 \text{ mg}} \times \frac{60 \text{ min}}{1 \text{ hr}}$$

1. Multiply 3 by 250 by 1 by 60 which equals 45,000
2. Multiply 1 by 1000 by 1 which equals 1,000
3. Divide 45,000 by 1000 which equals 45
4. Cancel out the units. (Grams, minutes, and milligrams cancel out leaving mL/hr)

Rate = 45 mL/hr

Calculation Micrograms/Minute (mcg/min)

Example: Norepinephrine (Levophed)

A nurse has been titrating a Levophed drip upward to maintain a MAP greater than 60 for a patient in septic shock.

The concentration of the Levophed is 4 mg in 500 mL and the medication is currently infusing at 30 mL/hr. How many mcg/min of Levophed is the patient receiving?

This time it is necessary to begin with what is known (30 mL/hr), next add the concentration to the equation (4 mg/500 mL) then the conversion factor for time (1hr/60 mins) and the final conversion factor (1,000 mcg/1 mg)

$$\frac{30\text{mL}}{1\text{hr}} \times \frac{4\text{mg}}{500\text{mL}} \times \frac{1\text{hr}}{60\text{min}} \times \frac{1,000\text{mcg}}{1\text{mg}}$$

1. Multiply 30 by 4 by 1 by 1,000 which equals 120,000
2. Multiply 1 by 500 by 60 by 1 which equals 30,000
3. Divide 120,000 by 30,000 which equals 4
4. Cancel out the units (Milliliters, hours, and milligrams cancel out leaving mcg/min)

$$\text{Dose} = 4 \text{ mcg/min}$$

Calculation Micrograms/Kilogram/Minute (mcg/kg/min)

Example: Dopamine

A physician has prescribed a dopamine infusion to increase a patient's blood pressure. The infusion is prescribed to

begin at 5 mcg/kg/min. The patient weighs 70 kg and the dopamine is available 400 mg/250 mL. At what rate should the nurse begin the infusion?

To solve: start with what is prescribed (5 mcg/kg/min), next add the weight of the patient as a fraction (70 kg/1 patient), the concentration of the drug (250 mL has 400 mg) the conversion for mgs to mcg (1 mg/1000 mcg) and conversion for minutes to hour (60 min/1 hr)

$$\frac{5 \text{ mcg}}{\text{Kg min}} \times \frac{70 \text{ Kg}}{1 \text{ patient}} \times \frac{250 \text{ mL}}{400 \text{ mg}} \times \frac{1 \text{ mg}}{1000 \text{ mcg}} \times \frac{60 \text{ min}}{1 \text{ hr}}$$

1. Multiply 5 by 70 by 250 by 1 by 60 which equals 5,250,000
2. Multiply 1 by 400 by 1000 by 1 which equals 400,000
3. Divide 5,250,000 by 400,000 which equals 13.125
4. Cancel out the units (micrograms, Kilograms, minutes, and milligrams cancel leaving mL/hr)

$$\text{Rate} = 13.125 \text{ mL/hr}$$

Appendix C

Answer Key

Chapter 1

Why/Why Not?

Why?

The surgery is urgent, the nurse is only witnessing the signature of the patient and not that the patient was actually informed. The patient did make the X on the consent form so the nurse could sign to validate the X was the patient's signature.

Why Not?

It would have been preferable to have the patient learn about the revascularization procedure and the potential for amputation prior to the administration of pain medication.

Currently there is reason to wonder if the patient has the capacity to consent. The nurse might want to speak with the surgeon, determine exactly when the surgery is scheduled, and suggest the surgeon return when the patient is more awake so it is clear the patient understood the risks and benefits of the procedure. Or, if the surgery is imminent, that a surrogate be asked to consent to the procedure since the patient may not currently have capacity.

Case Study

1. In situations where emergent care is required, if the team is unable to obtain informed consent, then consent is presumed due to the urgency of the situation. The healthcare team should provide treatment.
2. The nurse might explain to the sister that there are two common ethically accepted modes for making surrogate decisions. The first is the best interests standard in which the sister would decide what she believes is in the best interests of Allen. For example, she might decide that because Allen needed an adequate airway and nutrition to stay alive, it would be in his best interest to have the procedures. The second method is substituted judgment in which the surrogate (in this case the sister) decides what she thinks Allen would have decided had he been able to make the decision. For example, she might say, "Allen never wanted to live with a tracheostomy, ventilator, or tube feeding. He would never have consented to these interventions, so I will not either."
3. Although nurses are often told that they should be able to provide care without discrimination to all

patients, sometimes this is very difficult to do. In Angela's case, she is most likely a conscientious nurse who recognizes biases toward Allen that she might not be able to hide completely while providing care. Asking for another assignment in this situation would seem to be a legitimate request. However, the nurse leader would want to suggest that Angela take an opportunity to discuss her unresolved feelings about caring for Allen.

Review Questions

- 1.1 In a "closed ICU," a dedicated ICU team that includes a critical care physician provides patient care. Because the critical care team knows one another and has common expectations and an established communication pattern, patient outcomes appear to be better when patients are cared for in a closed system.
- 1.2 In part, because of the complexity and high degree of coupling of care, healthcare errors are most common in critical care areas. Complex care is usually also highly technological care, and technology also contributes to the error rate.
- 1.3 An underlying assumption of the synergy model is that optimal patient outcomes occur when the needs of the patient and family are aligned with the competencies of the nurse.
- 1.4 Many critically ill patients are intubated and unable to communicate even their most basic needs clearly. Thus, even though they may have a clear preference, they may be unable to communicate it. Other patients may be incapacitated temporarily due to pain, depression, or unconsciousness.
- 1.5
 - a. Could treatment of another problem obviate the need for a restraint?
 - b. Is the restraint the least restrictive and invasive alternative possible?
 - c. Will the use of the restraint outweigh the risks (physical, emotional, and ethical) associated with its use?
 - d. Are the restraints absolutely necessary for patient safety?
- 1.6 The AACN believes there is evidence that the moral distress experienced by critical care nurses has a substantial impact on healthcare. According to its evidence, as many as one half of all critical care nurses may have left a unit due to moral distress. In addition,

nurses who experience moral distress may lose the capacity to care for their patients and experience psychological and physiologic problems.

- 1.7 There are a variety of self-care practices that nurses can employ for their own health and well-being but also because they may aid in the prevention of compassion fatigue. These include:
- Make a commitment to self-care.
 - Develop strategies for letting go of work.
 - Develop strategies for acquiring adequate rest and relaxation.
 - Plan strategies for practicing effective daily stress reduction.

Chapter 2

Why/Why Not

Why?

Aspirating 200 mL of tube feeding leaves the nurse in a gray zone. Some experts recommend continuing tube feedings unless the patient has a residual of 500 mL or is nauseated, vomiting or aspirating. For a one time residual of 200 mL, the nurse might consider advancing the tube feeding to 60 mL/hour. However, this is the second time the nurse has aspirated 200 mL.

Why Not?

This is the second time the residual has been 200 mL. The tube feeding is still being advanced and the residual is questionable. A better approach than increasing the rate now might be to continue the tube feeding at 50 mL/hour and determine if a motility agent such as metoclopramide can be prescribed.

Case Study

1. 4.5 mg
2. 9.1 mg/hour
3. These doses are at the high end of therapeutic doses. They usually are reduced by 30% to 50% if the patient is also receiving an opioid (and Harold is). Thus, these doses might be too high.
4. The nurse should:
 - a. Notify the physician.
 - b. Adjust the rate of midazolam infusion based on the appropriate concentration and physician's recommendation.
 - c. Continue to monitor the patient and titrate the rate to the desired response level of the patient.
 - d. Report the occurrence in the appropriate manner and complete the appropriate forms.

5. The standard concentration used for calculation of the drip rate on the pump was 0.5 mg/mL, whereas the dose of midazolam prepared by the pharmacy was 1 mg/mL. Thus, the patient received twice the intended dose of midazolam per hour.
6. Reporting errors can allow for system-wide changes that improve patient care. Things that might have helped in this situation include:
 - Automatic notification by the pharmacy or pump when a drug is to be given at a high dose
 - Use of only one concentration of each medication by the hospital with the pumps programmed for that concentration
 - Checking the concentration of medication in the bag and on the pump each time a medication is hung
 - Having two nurses check sedative and narcotic doses
 - Thorough education concerning new products, particularly IV pumps
 - Appropriate use of pain and sedation assessments
7. The initial high level of pain coupled with the provision and subsequent infusion of a dose of midazolam at the high end of the therapeutic spectrum and lack of ongoing sedation assessment initiated the problem. The nurse's unfamiliarity with a new pump compounded the problem.

Review Questions

- 2.1 The AACN states that the characteristics of critically ill patients are resiliency, vulnerability, stability, complexity, predictability, resource availability, participation in care, and participation in decision making.
- 2.2 Patients describe their needs as:
 - Being thirsty
 - Having tubes in their mouth and nose
 - Not being able to communicate
 - Being restricted by tubes/lines
 - Being unable to sleep
 - Not being able to control themselves
- 2.3 Responsive patients indicate that they need their nurses to be kind, patient, and attentive to their needs; to offer frequent verbal assurances; and to know what information is important, and provide it. Nurses should recognize how frustrating communication can be for ventilated patients. When communicating with ventilated patients, nurses should do the following:
 - Routinely ask patients about their feelings and their state of mind.
 - Ask permission before beginning nursing care and procedures.
 - Evaluate patients' understanding of the information conveyed to them by asking simple yes/no questions.

- 2.4 By using a valid, reliable pain assessment tool such as the CPOT displayed in Table 2–1 .
- 2.5 By using a valid, reliable tool such as the AACN Sedation Assessment Scale displayed in Table 2.5 and by titrating the sedative to a predetermined level on the chosen sedation scale.
- 2.6 Nutritional support should be administered enterally if at all possible because it is associated with a much lower risk of infection and better outcomes than parenteral nutrition. In part, this lower infection rate may occur because enteral feeding prevents translocation of bacteria from the GI tract, which can occur when the GI tract is inactive.
- 2.7 The advantages of open visiting hours include family satisfaction; improved communication between family, patient, and staff; more emotional support for the patient as well as physiologic benefits for the patient; and opportunities for enhanced family and patient education. Disadvantages include discomfort of staff and the staff's unfounded concerns that the patient will be stressed or unable to receive needed care.
- 2.8 Information should be provided in a timely manner to families in an appropriate setting and delivered by a caring individual, preferably a physician, at least initially. The healthcare team should deliver a consistent message. In addition, regular family conferences, written instructional guides, a consistent family contact member, and a consistent nurse providing care may also help to meet the communication needs of families.

Chapter 3

Why/Why Not?

Why?

- NIV has been used effectively in COPD and will likely improve gas exchange.
- A sign of severe hypercapnea is decreased alertness. Ms. Jones is obtunded and improved gas exchange is likely to improve her level of consciousness.
- A trial of NIV may improve Ms. Jones respiratory status enough to avoid intubation and invasive mechanical ventilation.

Why Not?

- Is it safe to use a tight fitting mask for a patient who is obtunded and may not have a high enough level of consciousness to protect her airway? Ms. Jones is at an increased risk of aspiration. NIV should be used with caution in patients who are not able to protect their own airway.

- Positive pressure ventilation can reduce venous return and cause hypotension, particularly in hypovolemic patients. Ms. Jones' BP is low and positive airway pressure may further reduce her BP. An assessment of her fluid volume status is indicated.
- NIV may not be sufficient for patients with severe respiratory distress and patients with acute respiratory distress syndrome. With Ms. Jones' admission diagnosis of pneumonia, is it possible that she is in acute respiratory distress syndrome? Her RR is very high and a delay in adequate gas exchange may further worsen her condition. Should ABGs be drawn to assess the severity of her condition?
- You should discuss your concerns with the provider and suggest obtaining ABGs early in the treatment course to assess the appropriateness of this mode of ventilation support. NIV may be an appropriate intervention, but if Ms. Jones' condition does not improve quickly, the nurse should be prepared to assist with intubation. Positioning the patient at 30–45 degrees may reduce the risk of aspiration and work of breathing.

Case Study

1. The medication that he received for the seizures or for his intubation might have caused his blood pressure to drop. It is also possible that it interacted with the Toprol he was taking on a routine basis.
2.
 - Elevate the HOB to at least 30 degrees.
 - Monitor for gastric distention.
 - Maintain a proper cuff pressure in the ET tube (greater than 20 cm H₂O).
 - Change the ventilator circuit only when it is soiled.
 - Limit secretions in the oropharynx; use chlorhexidine for oral care.
 - Use narcotics and sedatives carefully; provide daily sedation vacation.
 - Wash hands.
 - Provide stress ulcer prophylaxis.
 - Turn the patient at least every 2 hours.
 - Institute DVT prevention strategies.
3. Most institutions would recommend supporting his blood pressure with volume and/or vasopressors so that his HOB could be maintained at 30 degrees of elevation or higher.
4. He would not be a candidate for attempting a sedation vacation or withdrawing sedation at this time.
5. His ET tube will need to be repositioned (it should be 2 to 3 cm above the carina), so the nurse will need to notify the appropriate people in the agency and anticipate that the tube will be repositioned.

6. The nurse has indications that the patient has infiltrates and a consolidation, so aggressive pulmonary hygiene with frequent repositioning is warranted. The nurse should check the patient's WBC (it was 21,000), determine if a sputum culture had been sent, and notify the appropriate people.
7. Mr. Donnelly's misuse of alcohol and cigarette smoking could predispose him to the development of ARDs.
8. He is just mildly hypercapneic (norm PaCO_2 35 to 45) but he has been able to compensate with a slightly elevated bicarbonate (norm 22 to 26), thus maintaining his pH in the normal range. His oxygen saturation and PaO_2 are acceptable, maintained by a FiO_2 that is within the recommended range.
9. In this mode, the patient sets the rate independently; however, when breathing in excess of the set rate patients receive the tidal volume they can pull using their own strength. They are controlling the rate as well as volume with the ventilator serving as a "backup." The client is not allowed to breathe at less than the set rate, ensuring an adequate minute volume, but can breathe additional breaths if he has a drive to breathe. The mode is called synchronized because if the patient initiates a breath when it is almost time for a set volume breath, the ventilator will synchronize with the patient's respiratory effort. This prevents the machine from trying to deliver a breath at the same time a patient is trying to exhale.

Review Questions

- 3.1 a. Oxygen concentration (FiO_2): Amount of oxygen in gaseous admixture delivered to the patient
 b. Tidal volume: Volume of gas delivered in one cycle
 c. Rate: Minimal number of breaths per minute
 d. Inspiratory:expiratory ratio: Ratio of time of inspiration to time of expiration; may be reversed in conditions in which lungs are noncompliant
 e. High-pressure limit: Ventilator will not exceed this pressure in delivering volume; pop-off mechanism prevents excessive pressure
 f. Pressure support: Positive pressure used to decrease the patient's work of breathing
 g. Positive End Expiratory Pressure (PEEP): Positive pressure left in the lungs at the end of expiration; prevents atelectasis and may enhance oxygenation at higher levels
- 3.2 ALI may be caused by such direct insults to the lungs as chest trauma with pulmonary contusion, pneumonia, aspiration, near drowning, pulmonary edema, inhalation injury, pulmonary embolus, radiation, or eclampsia. It may also be caused indirectly by insults occurring from sepsis, burns, multiple transfusions, drug overdose, acute pancreatitis, and intracranial hypertension.
- Although all of these have a different initial mechanism, they result in a common pathway of inflammation with damage to the capillary and alveolar membranes and ARDS.
- 3.3 The progression of ALI is frequently divided into three stages: exudative, proliferative, and fibrotic. Disruption of alveolar capillary membrane permeability is at the crux of ALI pathophysiology. During the first phase, known as the exudative phase, the capillary membrane begins to leak, and protein-rich fluid fills the alveoli, profoundly disrupting gas exchange.
- The proliferative phase begins 7 to 10 days after onset. Type II alveolar cells sustain damage, limiting the production of surfactant, resulting in further loss of alveolar function. This decrease in available alveolar surface area results in VQ mismatch and hypoxemia.
- The final phase is called the fibrotic phase because the altered healing process results in development of fibrotic tissue in the alveolar capillary membrane. The resulting alveolar disfigurement contributes to decreased lung compliance and worsening pulmonary hypertension.
- 3.4 The early manifestations of ARDS are tachycardia and tachypnea that seem incongruent with the rest of the patient findings. The hallmark is hypoxemia that is unresponsive to oxygen administration.
- 3.5 Historically mortality rates for older adults with ARDS (69% to 80%) were higher than for younger adults. More recently, older adults have not been shown to have significantly higher mortality rates from ARDS unless they have underlying renal, cardiac, and/or neurological disease.
- 3.6 Tidal volumes of 4 to 6 mL/kg of body weight reduced mortality as well as decreasing days on the ventilator. The lower tidal volumes result in alveolar hypoventilation, resulting in a rise in serum CO_2 levels. PEEP provides additional surface area in the alveoli to enhance gas exchange; setting PEEP at levels between 14 and 16 cm H_2O can return collapsed alveoli to a functional status. In addition, FiO_2 is set at less than 60%, if possible, to minimize the potential for oxygen toxicity, and plateau pressures are maintained below 30 cm H_2O , if possible.
- 3.7 Three nursing priorities when caring for the patient with ARDS are:
 - Provision of effective ventilation
 - Maintenance of tissue oxygenation
 - Prevention of sepsis and potential MSOF

Chapter 4

Why/Why Not?

Why

It is important that this patient stays in normal sinus rhythm. AF in the setting of a low ejection fraction can cause hemodynamic compromise, which clearly it has for Mr M. The combination of beta-blockade for heart rate control and the anti-arrhythmic effect of Amiodarone is proving effective. The increase in the QTc is worrisome though. QTc prolongation can produce VT and Torsades de points. The nurse should contact the ordering healthcare provider to clarify if these medications should be given at the current dose or held.

Why Not

The QTc exceeds the upper limit of normal and should concern the nurse. Particularly since the nurse can trace an increase since the patient has started amiodarone. Contact the prescribing healthcare provider and inquire about the following: A change in dosing on the amiodarone, sometimes the oral load needs to be lessened with the elderly population; orders to hold either or both medications; any testing to determine if the amiodarone may be deleterious to this elderly patient, which may include liver and thyroid function tests.

Case Study

1. Epinephrine 1 mg IV, repeated every 3 to 5 minutes.
2. Epinephrine is a peripheral vasoconstrictor; therefore, the implication is that this drug may increase coronary and cerebral perfusion pressure during CPR.
3. Assess the patient for a pulse, as it appears his dysrhythmia may have converted to a sinus rhythm.
4. An antidysrhythmic will be important to prevent a return of the ventricular dysrhythmia; amiodarone 150 mg IV bolus followed by a 1 mg/min infusion for 6 hours then a 0.5 mg/min maintenance infusion over 18 hours.
5. Possible causes include hypovolemia, hypoxia, acidosis, hyper- or hypokalemia, hypomagnesemia, hypoglycemia, cardiac tamponade, tension pneumothorax, or thrombosis.

Labs should include arterial blood gases to check for hypoxia and acidosis, potassium, magnesium, and glucose. The patient should also have a chest x-ray to rule out a pneumothorax and should be assessed for cardiac tamponade and thrombosis.

Review

- 4.1 An electrocardiogram (ECG) is used to determine narrow and wide complex tachycardia which is based on the QRS complex.

Narrow complex is when the QRS is less than 0.12 seconds (120 ms). Examples include sinus tachycardia, atrial fibrillation, atrial flutter, supraventricular tachycardia.

Wide complex is when the QRS is greater than or equal to 0.12 seconds (120 msec). Examples include ventricular tachycardia, a narrow complex tachycardia with an aberrant conduction system, pacemaker-tracked or mediated tachycardia.

- 4.2 Most patients have an underlying cardiac problem such as a valvular disease or electrolyte imbalance. Also, they may be unable to tolerate the rapid rhythm that occurs at the onset of AF.
- 4.3 Patients with AF are at high risk for embolic strokes.
- 4.4 The common treatment modalities for atrial fibrillation:
 - a. Rate control with chronic anticoagulation is recommended for most AF patients.
 - b. Coumadin (warfarin) is used unless the patients are in a low-risk group or have contraindications to its use.
 - c. Beta blockers (atenolol, metoprolol) and calcium channel blockers (diltiazem, verapamil) are recommended for rate control as an outpatient; digoxin should be considered as second-line therapy for patients who do not respond to these medications.
 - d. For patients requesting cardioversion, either synchronized cardioversion or pharmacological conversion is appropriate.
 - e. To minimize the risk of embolic stroke in patients electing cardioversion, either transesophageal echocardiogram (TEE) or 3 weeks of anticoagulation prior to cardioversion is acceptable. Both strategies are followed by 3 to 4 weeks of anticoagulation.
- 4.5 Third-degree heart block, also known as complete heart block, occurs when the impulses from the atria are not conducted through the AV junction. Therefore, there is no relationship between the atria (P waves) and the ventricles (QRS waves). The PR interval varies and the QRS is usually wide and the heart rate slow (less than 40 to 60).

Amiodarone is indicated to treat both supraventricular tachycardias and ventricular tachycardias.
- 4.6 Amiodarone is an antiarrhythmic agent. It has actions similar to calcium channel blockers on slowing conduction and prolonging refractoriness at the AV node. Amiodarone also prolongs the duration of cardiac action potential via the sodium and potassium channels, raising the threshold for SVT and ventricular fibrillation, and may prevent its recurrence.

Amiodarone is indicated in (a) stable irregular narrow complex tachycardia, such as atrial fibrillation, (b) stable regular narrow-complex tachycardia,

- (c) to control rapid ventricular rate due to accessory pathway conduction in pre-excited atrial arrhythmias,
- (d) hemodynamically stable monomorphic VT, and
- (e) polymorphic VT with normal QT interval.

4.7 Pacemakers can fail by failing to pace or capture as well as by oversensing and undersensing.

Chapter 5

Why/Why Not?

Why?

PA catheters are used to establish the presence of heart failure in hemodynamically unstable patients. The PA catheter may be utilized to evaluate the patient's volume status during diuresis, and perhaps to guide medication therapy if needed.

Why Not?

This patient presents with a known diagnosis of heart failure, and a known etiology of his heart failure exacerbation – fluid overload after several missed Lasix doses. The placement of a PA catheter is invasive and not without risks, including ventricular dysrhythmias, pulmonary capillary rupture, pulmonary infarction, pneumothorax/hemothorax, and infection. Additionally, the patient's respiratory status is compromised by his volume overload, and it will be difficult to safely oxygenate him while maintaining a supine and flat position for the placement of the catheter. The nurse and providers must carefully consider the risks and benefits of any procedure, and especially how performing the procedure will change management of the patient. At this time, the risks of placing a PA catheter outweigh the benefits, and will provide little new information to guide therapy. The patient should be given Lasix, oxygen support (via noninvasive ventilation if needed) and his volume status should be closely followed. If diuresis does not improve his heart failure, a PA catheter may need to be placed for further evaluation.

Case Study

1. Indications for placement of a pulmonary artery catheter for this patient are:
 - Assessment of oxygen supply and demand
 - Monitoring during surgery in a patient with a cardiovascular history compounded by renal failure
 - Guidance during fluid or medication therapy
 - Heart failure
2. This is a PA tracing. Once the catheter leaves the RV it is advanced across the pulmonary valve to the PA.

The PA waveform has a dicrotic notch that identifies the closure of the pulmonary valve. The systolic pressure is fairly consistent with the RV systolic pressure; however, the diastolic pressure increases once the catheter is in the PA. Normal PA pressure value is 15 to 25 mm Hg (systolic), 5 to 15 mm Hg (diastolic), 9 to 16 mm Hg (mean)

3. This is a pulmonary artery wedge pressure, also known as a pulmonary artery occlusive pressure.
4. The catheter has most likely drifted farther along the pulmonary artery and become stuck in the wedge position. The complication of a catheter stuck in wedge is that blood flow distal to the catheter has stopped, thus risking pulmonary tissue ischemia.
5. Nursing actions include:
 - Ensure that all the air is out of the syringe.
 - Try repositioning the patient.
 - NEVER flush the PA catheter when the catheter is stuck in wedge as this can lead to PA rupture.
 - Notify the physician or advanced practice nurse to reposition the catheter.
 - If the nurse's institution allows, withdraw the PA catheter until the PA waveform appears.
6. The PAWP and RA are both elevated indicating that the patient's preload is elevated.
7. The SVRI and PVRI are both elevated indicating that the patient's afterload is also elevated.

Overall, how would you evaluate his stroke volume index and cardiac index?

Both the CI and SVI are decreased indicating that the patient's cardiac output is decreased. The combination of elevated preload, increased afterload, and decreased cardiac output indicate that the patient is likely in heart failure.

8. The SvO₂ is normally 60% to 75%. Decreased values of venous oxygen saturation indicate increased oxygen extraction by the tissues. This may be due to either a decrease in oxygen delivery or an increase in tissue oxygen demands. In this case, it is likely due to the decrease in oxygen uptake and delivery associated with the patient's heart failure.

Review Questions

- 5.1 The four determinants of cardiac output are heart rate, preload, afterload, and contractility.
- 5.2 Myocardial fiber stretch from an adequate end diastolic volume or preload is required to optimize contractility. If the volume becomes too large, the myocardial fibers are overstretched, leading to failure.
- 5.3 a. Nursing actions are to ensure that the pressure bag is inflated to 300 mmHg. Ensure there is adequate saline solution in the flush bag. Use the fast flush

device on the pressure monitoring system to clear the tubing of blood.

b. This will cause the arterial line to be overdamped.

5.4 Factors elevating the RA pressures are:

- Fluid volume excess
- Right ventricular failure
- Pulmonary hypertension
- Tricuspid stenosis or insufficiency
- Pulmonic stenosis or insufficiency
- Pulmonary embolism

Chapter 6

Why/Why Not?

Why?

Currently Ms M shows no signs of acute ischemia or infarct. However, she has at least one major risk factor for CAD and subsequent ACS: hypertension that appears poorly controlled. A pathologic Q wave with no evidence of ST-segment elevation or T-wave inversion) indicates that she had experienced an MI sometime in the past. Her coronary anatomy should be visualized at some point to assess for disease.

Why Not

Risk stratification indicates that Ms M has risk factors for CAD and ACS but is not currently experiencing an acute episode. An early conservative approach, including a stress test, is indicated. Medication therapy should be maximized including a statin, beta blocker, anti-hypertensives, SL nitroglycerin and aspirin. If her stress test is positive she should progress towards cardiac catheterization.

Case Study

1. Common causes of chest discomfort are acute myocardial infarction, peptic ulcer disease, acute coronary syndrome, pericarditis, and aortic dissection. A nurse in the emergency department needs to assist in a quick assessment to assist with the differential diagnosis.
2. These leads point to damage in the anterior wall of the left ventricle. This is usually due to an occlusion of the left main or the left anterior descending coronary artery. Infarctions in this area of the myocardium carry the highest mortality rate.
3. STEMI is an infarction of the full thickness of a region of the myocardium. It is caused by thrombosis over an atherosclerotic plaque occluding a coronary artery and starving the supplied area of the myocardium of oxygen.

Acute coronary syndromes are a spectrum of conditions of myocardial ischemia in which there are possibly no ST elevations on the ECG and can include unstable angina and an NSTEMI.

4. This patient has multiple risk factors for the development and progression of acute coronary syndrome. They include hypertension, obesity, stressful work environment, stressful family life, increased caloric intake, no exercise, high blood cholesterol, and borderline diabetic.
5. The patient will need to have a CK-MB to assess for specific myocardial muscle damage. A hemoglobin A1C is needed to assess for glycemic control. Troponin T is needed for a measurement of the muscle damage, myoglobin, especially if it is elevated with the first 2 hours of chest pain. C-reactive protein will be used to assess inflammation of the coronary system. Chest x-ray is needed to rule out pulmonary causes of chest pain.
6. Upon arrival to the emergency department the patient is placed in a semi-Fowler's position to allow for comfort and proper cardiovascular functioning. An intravenous line should be started to keep the vein open for immediate access for the administration of emergency drugs. Medications and treatments are then started in a standard progression.
 - Aspirin—Aspirin is given first and consists of four "baby strength" aspirin or the equivalent of 325 milligrams. It is used to prevent platelet aggregation and the formation of blood clots in the coronary vasculature.
 - Nitroglycerin—This is given if the patient's systolic blood pressure is above 90 mmHg. It can be given in either the sublingual form, which will reduce the pain that is currently present, or the intravenous form to prevent future episodes.
 - Supplemental oxygen—Oxygen is administered at 2 to 4 liters per nasal cannula to increase myocardial oxygenation. The oxygen saturation level of 92% is used as a guideline for the proper amount of oxygen to be administered.
 - Morphine—Morphine sulfate is given in a dose of 2 to 5 mg intravenous every 5 to 30 minutes as needed. Morphine is used for both pain management and as an antianxiety medication. If this medication is given first it can mask the patient's perception of pain and mask the underlying cause of ischemia.
 - Vital signs should be taken at frequent intervals.
 - Electrocardiogram—This should be obtained within the first 10 minutes of arrival.

The patient will also be evaluated with a series of blood tests and cardiac markers.

Review

- 6.1 An STEMI is caused by occlusive thrombus composed of platelets and thrombin. Aspirin is an antiplatelet agent that stops platelet aggregation.
- 6.2 Troponins are molecules that regulate the interaction of actin and myosin in muscle. Troponins are released into the blood from damaged muscle. Cardiac troponins I and T are specific to cardiac muscle, are highly sensitive to ischemia and necrosis, and correlate with the amount of myocardial damage. The main drawback to the use of troponins is that the level may not rise until after 12 hours of myocardial damage.
- 6.3 The main electrolyte disturbances are hypercalcemia, hypocalcemia, hypokalemia, and hyponatremia. Calcium, sodium, and potassium are all needed for proper function of the heart. The most important level for the nurse to monitor during hospitalization and treatment of ACS is the potassium level. Many of the cardiac drugs can interfere with the body's absorption of potassium. If the level of potassium falls below 3.5, the patient is at risk for developing lethal cardiac rhythm disturbances, such as ventricular tachycardia.
- 6.4 The pain of ACS is treated with aspirin for its antiplatelet effects; nitroglycerin as a vasodilator; and morphine sulfate, an opioid, to control pain and anxiety.
- 6.5 A coronary artery bypass graft is indicated for the patient who has failed medical management, has more than two diseased coronary vessels with significant blockage, may not be a candidate for PCI, or has failed a PCI attempt with ongoing chest discomfort.
- 6.6 Fibrinolytic agents such as eptifibatide (Integrilin), tirofiban (Aggrastat), and abciximab (ReoPro) are agents that break down fibrin. When used in conjunction with early aspirin therapy, these agents will dissolve the thrombus blocking the coronary artery and restore blood flow to the vessel, and thus oxygen to the heart muscle.
- 6.7 The most common complications of a myocardial infarction are:
 - Heart failure due to left ventricular dysfunction from decreased myocardial blood supply and decreased ventricular wall motion.
 - Dysrhythmias, especially atrial and ventricular, are very common post myocardial infarction. These are caused by the release of catecholamines, hypokalemia, and the parasympathetic response.
 - Pericarditis, which is the inflammation of the pericardial sac, can also occur. This happens frequently after invasive cardiac surgery and is self-limiting.

Chapter 7

Why/Why Not?

Why?

In the setting of stable hemodynamics the milrinone could be discontinued but this medication must be weaned slowly and the patient monitored very closely. One would NOT just shut off this medication.

Why Not?

Milrinone should be weaned slowly due to its long half-life (2.3h) and the potential for the patient to decompensate. Milrinone promotes vasodilation, resulting in decreased preload and afterload and reduced cardiac workload. Because the drug causes vasodilation, the patient's blood pressure is monitored closely. In addition, the nurse would want to monitor for increasing pulmonary pressures, increasing systemic vascular resistance and decreasing cardiac output during the drip wean. There is also potential for ventricular dysrhythmias as this drug delays AV conduction.

Case Study

1. The patient needs BiPap because of the oxygen saturation of 90% and pO₂ 63 on 6 LNC. Her CXR and physical assessment data are suggestive of heart failure. The echocardiogram demonstrates an EF of 25%, which is indicative of decreased ventricular function. A PA catheter will reveal crucial hemodynamic data that will guide the clinical management.

Mrs. E's hemodynamics are CVP 16, PA systolic 50, PA diastolic 28, and PAWP 26. Cardiac output is 4.0 with a cardiac index of 1.8. What does Mrs. E's hemodynamic profile indicate? What other physical exam findings and lab values support your analysis?

The hemodynamic profile is one of fluid overload. Physical exam findings that support this include (+) JVD, crackles 2/3 up bilaterally; 3+ pitting edema ankle to midcalf bilaterally; (+) hepatojugular reflux. Diagnostic data include BNP 1100; CXR: pulmonary edema. An echocardiogram is done which reveals LVEF 25%, mild LV hypertrophy, valvular function intact.

2. Atrial fibrillation in the setting of a HFrEF can precipitously drop a patient's cardiac output. Atrial kick, may supply as nearly as 20% of the volume entering the ventricle, is compromised and contributes to a lessening ventricular volume and subsequent decreased CO. When it is unclear how long a patient has been in AFIB, it is crucial to obtain an echocardiogram before cardioversion to rule out atrial clot.
3. The physician prescribes Lasix 20 mg IV push. What should you check before and during the administration

of this drug? How do you evaluate if this intervention is successful? What would be your expectation if the patient did not respond appropriately to the medication?

You should monitor BP and know your patient's serum potassium level. If the patient does not have a Foley catheter in place, you may want to obtain an order for this. If there is a foley catheter in place, be sure to empty it before administering the Lasix for accurate measurement of effective diuresis. IV diuresis would be considered effective if you have a generous increase in urine output and resolution of fluid overload symptoms. If your patient does not respond effectively to the intervention, your expectation should be to contact the healthcare provider for further orders which may include administration of the diuretic at a higher dose.

4. Dobutamine is a beta-adrenergic agonist that produces the following physiologic effects that would be appropriate for Mrs. E: increased contractility and reduced afterload. You should see increase in CO, decrease in PAWP, and decrease in SVR. Dobutamine also decreases AV conduction and has less potential for precipitating arrhythmias so the nurse should monitor the heart rate and rhythm.
5. Lisinopril is an ACE inhibitor. ACE inhibitors cause suppression of the RAS resulting in vasodilation. With decreased systemic vascular resistance (SVR), it is easier for the LV to unload volume to the systemic circulation. The nurse should expect that patients with heart failure due to systolic dysfunction (HFrEF) will be on ACE inhibitors unless there is a contraindication. Coreg is a beta blocker. By inhibiting the effects of the SNS stimulation, betablockers reduce heart rate, blood pressure, and cardiac contractility. Beta blockade reduces cardiac hypertrophy, arrhythmias, and tachycardia leading to heart failure. Betablockers are indicated for all patients with stable heart failure and reduced ejection fraction. The earlier this therapy is prescribed, the more positive effects there may be on the decreasing disease progression and the improvement of systolic function. Lasix is a loop diuretic. Sodium and water retention lead to fluid volume excess. The patient in HF may present with signs and symptoms of peripheral and pulmonary edema. Typically a loop diuretic like furosemide (Lasix) is used to control the pulmonary and peripheral edema with usual starting doses of 20 to 40 mg PO daily.

Review

- 7.1 The primary neurohormonal mechanisms involved in response to heart failure are the sympathetic nervous system and the renin-angiotensin-aldosterone system.

In response to a decreased cardiac output, activation of the sympathetic nervous system releases norepinephrine, causing increases in vasoconstriction, heart rate, and contractility. Activation of the renin-angiotensin-aldosterone system further increases vasoconstriction through the activation of angiotensin II, a potent vasoconstrictor. Activation of the renin-angiotensin-aldosterone system also stimulates aldosterone, expanding fluid volume through sodium reabsorption. These actions may be effectively blocked with medications such as beta-blockers, ACE inhibitors, angiotensin receptor blockers, and aldosterone inhibitors.

- 7.2 HFrEF, or systolic dysfunction, is impaired left ventricular contractility with a reduced ejection fraction. The ejection fraction is the percentage of blood ejected from the left ventricle with each contraction—normal is 55% to 75%. This impaired contractility leads to a reduction in cardiac output. HFpEF or, diastolic dysfunction appears when the ventricle is normal size but hypertrophied, leading to loss of left ventricular diastolic relaxation and distensibility. This preserves the normal ejection fraction but impairs filling, leading to elevated filling pressures and pulmonary artery wedge pressures.
- 7.3 This patient would be classified as NYHA class 3A.
- 7.4 The signs of deterioration include dyspnea at rest, tachycardia, reduced oxygen saturation, crackles on lung auscultation, hypotension, worsening cough, new dysrhythmias, elevation of PAWP, and reduction in CO/CI.
- 7.5 Aggressive oxygen administration, administration of venodilators and loop diuretics, and possible administration of positive inotropic therapy
- 7.6 Maintenance of adequate ventilation and tissue oxygenation.

Chapter 8

Why/Why Not?

Why?

- Extended periods of hypotension lead to end organ dysfunction.
- Titrating the Dopamine to keep systolic blood pressure over 90 mmHg and mean arterial pressure over 65 mmHg will ensure tissue perfusion of the major organs.

Why Not?

- Tachycardia is a common side effect of Dopamine administration. Tachycardia increases the myocardial oxygen demand.

- Discuss concerns about the tachycardia and new dysrhythmias with ordering provider. Other available vasopressor infusions including norepinephrine, epinephrine, phenylephrine may be considered. These preparations do not increase the heart rate as much as dopamine.

Case Studies

Case 1

1. Signs and symptoms indicating hypovolemia in this patient include:
 - Hypotension
 - Tachycardia
 - Pale skin color
 - Delayed capillary refill
 - Absent distal pulses
 - Change in mental status
2. Lab tests indicating hypovolemia include:
 - Elevated BUN
 - Elevated sodium
 - Decreased hemoglobin
 - Decreased Hct
3. As the patient's circulating volume and blood pressure decrease, the baroreceptors in the carotid arteries and aortic arch send signals to the medulla in the brainstem. The cardiac accelerator center and the vasoconstrictor center are stimulated. The cardiac accelerator center causes an increase in heart rate and stroke volume. The vasoconstrictor center causes arterioles and veins to constrict, delaying capillary refill and reducing the quality of the peripheral pulses.
4. At face value the saturation of 96% appears within normal limits. However, in this patient the amount of hemoglobin available for saturation is reduced. Therefore, the saturation level is 96% of the available hemoglobin of 5.6. The supply side of the tissue oxygenation is reduced in this patient.
5. Immediate collaborative management in this patient includes rapid fluid resuscitation, first with isotonic crystalloids such as 0.9% normal saline, then blood products. The nurse must ensure that the large-bore intravenous catheters placed by EMS are patent and functional. The nurse should anticipate that the physician or advanced practice nurse will place a central line for additional vascular access. Use of a rapid fluid delivery device should be anticipated. Delivery of high-flow oxygen should continue and the nurse should anticipate the possibility of intubation and mechanical ventilation. Placement of a large-bore nasogastric tube is indicated to decompress the stomach, preventing aspiration as well as quantifying the amount of GI bleeding.

6. The nurse would recognize that a hypovolemic patient had received adequate volume replacement by CVP 8–12 mmHg or back to baseline, MAP of at least 60–70 mmHg, and urine output greater than 0.5 to 1 mL/kg/hr

Case 2

7. The signs and symptoms suggestive of cardiogenic shock in this patient include dyspnea diaphoresis, and pallor; vital signs of pulse 120 and irregular; BP 78/52; RR 28; pulse oximetry of 88% on room air; crackles bilaterally on chest auscultation; and ST elevation in the anterior leads
8. The pathophysiology of this patient's dyspnea and hypoxia is that the increased filling pressures in the left ventricle are reflected back into the left atrium and the pulmonary vasculature, leading to vascular congestion and pulmonary edema with impaired gas exchange.
9. The likely etiology of his cardiogenic shock is a large anterior MI.
10. Priorities in collaborative management for this patient include correction of the underlying cause (in the case of this patient, reperfusion of the myocardium during his myocardial infarction), improvement of myocardial contractility and cardiac output, and management of complications such as pulmonary edema and dysrhythmias.

Case 3

11. The signs and symptoms indicating that this patient has developed anaphylactic shock include that she is obviously dyspneic with audible wheezing; her lips and eyes are swollen and her face is flushed; her complaints of abdominal cramping and vital signs of BP 82/40; pulse 132; RR 42; with a pulse oximetry of 86% on room air.
12. Collaborative priorities of care for this patient in the emergency department include establishment of a patent airway and restoration of circulating volume and tissue perfusion. Epinephrine is widely advocated as the main treatment in those individuals experiencing anaphylaxis since it produces bronchodilation improving the respiratory status and causes peripheral vasoconstriction improving the blood pressure and tissue perfusion.

Case 4

13. Signs and symptoms suggesting that the patient has developed a tension pneumothorax with obstructive shock rather than a simple pneumothorax include diaphoresis, BP 70/40, pulse 142, and absent breath sounds on the left with only marginal sounds on the right.

14. Tension pneumothorax may cause hypotension when the increased pressure in the chest results in impaired ventricular filling. This occurs because the pneumothorax has become so large that the mediastinum has shifted and compressed the cardiac chambers resulting in impaired filling, decreased cardiac output, and hypotension.
15. In addition to preparing for chest tube insertion, the nurse might need to assist with the insertion of a large-bore needle for immediate decompression. Once the lung is re-expanded, the nurse should reassess the patient, expecting almost immediate improvement in the patient's status.

Review

- 8.1. Airway and breathing should be the priority for treatment. This patient may need to be intubated. It is unclear if he has had a cervical spinal cord injury that may be affecting his thoracic excursion and his ability to breathe.

His heart rate is not tachycardic because neurogenic shock occurs when a spinal cord injury to the cervical and upper thoracic spinal cord causes a temporary interruption in the sympathetic innervation leaving the parasympathetic innervation unopposed. Unopposed parasympathetic innervation regulation of the heart rate results in profound bradycardia.

The other parameters you need to assess to determine if the patient has neurogenic shock are:

- Hypotension without compensatory tachycardia
- Bradycardia
- Warm, dry skin
- Flaccid paralysis below level of injury
- Loss of cutaneous and deep tendon reflexes below level of injury
- Bowel and bladder dysfunction
- Priapism

The loss of the autonomic reflexes below the level of spinal cord injury results in vasodilation and redistribution of the blood volume. Interventions to correct the vasodilation include crystalloid administration initially. If volume resuscitation does not correct the hypotension, then vasopressors administered intravenously should be instituted. Vasopressor infusions such as dopamine, norepinephrine, and phenylephrine are preferred as they stimulate the alpha receptors resulting in vasoconstriction.

- 8.2. • Patients on beta blockers may not respond to epinephrine. Glucagon 1–2 mg IV over 5 minutes followed by infusion of 5–15 micrograms per minute to counteract the beta blocking effect may be needed.
- The nurse should note the concentration of epinephrine. The ordered routes (IM, IV) require different concentrations.

- Concurrent administration of intravenous isotonic crystalloid boluses of 1–2 liters are recommended in the hypotensive patient.

Chapter 9

Why/Why Not?

Why?

A patient with a spinal cord injury would normally demonstrate neurogenic shock which is hypotension accompanied by bradycardia. This patient is tachycardic, so he is very likely bleeding from the penetrating wound of his abdomen. Permissive hypotension, maintaining the trauma patient's systolic blood pressure at 90, is one of the components of damage control resuscitation. It means that the patient will not be provided with large volumes of fluid until control of the patient's bleeding has been achieved either in the ED, angiographically, or surgically. The surgeon has indicated that he intends to take the patient immediately for scanning then to the OR to control bleeding. As long as the patient's BP remains at 90 systolic and it is possible to take the patient immediately to scanning and the OR, infusing the IV at 150 mL/hr would be an evidence based, logical, approach.

Why Not?

As noted above, the patient is clearly bleeding, probably in his abdomen. Volume replacement in the hypovolemic patient using rapid infusers, large-bore IVs, or intraosseous access to rapidly replace the volume of the exsanguinating trauma patient in a timely manner has been standard treatment for a long time. Although rapid infusion has been the standard, it is only one acceptable option and not the one the surgeon has chosen in this situation. The patient meets the criteria for permissive hypotension and the surgeon is preparing to take him immediately to scanning and the OR. The nurse might ask the surgeon to explain the rationale if s/he was unfamiliar with permissive hypotension and damage control resuscitation but should begin the fluid administration.

Case Study

1. His ABCs have been quickly reviewed. It is now time for a full set of vital signs and further assessment (and reevaluation) of his breathing, circulation, and neuro status to account for the increased confusion and belligerence.
2. The indications of a flail chest are dyspnea, pain in the chest wall, and paradoxical movement of the chest wall. The size and severity of the pulmonary contusion often determine the treatment that a person with a flail chest will require.

3. A flail chest may be splinted initially. The patient may need intubation and mechanical ventilation to stabilize the ribs and to prevent pneumonia. James's level of consciousness is deteriorating and he may need emergent intubation.
4. He most likely has extensive blood loss. James may have lost large amounts of blood into both his left leg fracture and the pulmonary contusion under his left chest wall injury. He may also have damaged his spleen. He will need volume and possibly blood replacement.
5. James's confusion will need additional assessment. It could be due to hypoxia, hypotension and poor cerebral perfusion, head injury, intoxication, or other possibilities.
6. Initially, pulses will need to be checked below the level of the fracture. It will be x-rayed and splinted. When and how it will be treated will depend on the severity of his other injuries and the nature of the fracture.
7. James's increasing confusion and belligerence will complicate his abdominal assessment. His abdomen will be observed for any indication of injury and his bowel sounds will be auscultated before his abdomen is palpated.

Review

- 9.1 The nurse should verify that the cervical spine is adequately stabilized while assessing the airway. If the nurse determines the airway is not open, it must be opened using cervical spine precautions.
- 9.2 The patient needs to be assessed for blood at the urinary meatus and any indication of pelvic trauma before the catheter is inserted. The physician should perform a digital rectal exam before the catheter is inserted.
- 9.3 The primary assessment includes Airway, Breathing, Circulation, and Disability. The secondary assessment includes Exposing the patient to check for other injuries, getting a Full set of vital signs, Giving comfort, doing a Head-to-toe assessment, obtaining an accurate History, and Inspecting the patient's posterior.
- 9.4 A variety of factors might contribute to the trauma score dropping. The drop could be due to the patient receiving pain medication with a subsequent drop in BP and respiratory rates coupled with a change in neuro status. With initial catecholamine release following trauma, some patients sustain their vital signs and do not deteriorate until they are medicated and their pain is relieved. This is not uncommon in patients with chest wall injuries. It could also be due to inadequate volume repletion and the beginnings of shock, which can become more apparent after pain medication.

- 9.5 Chest tubes may be inserted to allow air or blood to drain from the pleural space.

What would your teaching plan include for a patient who has undergone a fasciotomy?

The patient and family would need to understand the reason for the fasciotomy, that sensation and circulation should have returned to the affected limb so that any recurrence of symptoms should be reported, that there will be daily moist sterile dressings, and that additional surgery and possibly skin grafting will be needed to close the wound.

Chapter 10

Why/Why Not?

Why?

The 3% saline infusion is currently going at 1 mL/Kg through a central line. This is an appropriate dose by the appropriate route. It is important that the patient be euvolemic when 3% saline is administered and his BP and urine output indicate he is. The patient's serum Sodium and osmolality are within the desired range. The nurse will anticipate continuing the hypertonic infusion for the time being.

Why Not?

Although his ICP is marginally below 20, the level at which interventions for increased ICP are often started, 3% saline may be continued for as long as 7 to 10 days postinjury or last ICP spike. When the decision is made to discontinue hypertonic saline, the infusion should be weaned off. The dose should be halved every 6 hours. During the weaning, serum sodium levels should be checked every 12 hours.

Case Study

1. These findings most likely indicate a lesion on the left side of his brain and an increase in intracranial pressure. An increase in the systolic blood pressure occurs with increased intracranial pressure and increases cerebral perfusion. This compensatory mechanism can occur as long as the brain's ability to autoregulate is intact.
2. Mannitol decreases intracranial pressure while reducing cerebrovascular resistance and increasing cerebral blood flow.

Nursing responsibilities are the following: If the 15%, 20%, or 25% solutions are utilized, the nurse administers mannitol through an in-line filter. Mannitol may cause hypotension after rapid administration, especially in volume-depleted patients. Therefore, it is important that the patient be euvolemic and the nurse administer a bolus dose no more rapidly than over 15 to

30 minutes. Mannitol increases the osmolality of the blood, with optimal osmolality between 300 and 320 mOsm. If repeated doses of mannitol are given, the nurse monitors the serum osmolality every 4 to 6 hours and ensures that it remains less than 320 mOsm. The nurse also monitors the urine output to ensure that it is at least 30 to 50 mL/hr and reviews the lab results, looking specifically for hyponatremia and hypokalemia.

3. Because the origin of the bleeding is arterial, the hematoma accumulates rapidly and must be evacuated immediately. Patient outcomes are often good if the condition is identified promptly and the patient is taken to the OR for immediate surgical evacuation of the hematoma.
4. Sedation is often used to control agitation associated with ventilation in head-injured patients although it has not been shown to affect patient outcomes. Propofol is a sedative-hypnotic anesthetic, which decreases ICP but does not improve mortality. One advantage of propofol is that, in most patients, the dose may be lowered or discontinued and within about 10 minutes an accurate neurological assessment can be obtained. Long-term and high-dose uses are associated with significant morbidity, specifically the propofol infusion syndrome.
5. The nurse has specific responsibilities when caring for a patient whose CSF is being drained through an external ventricular drain, including:
 - First the transducer and stopcock must be leveled at the tragus of the ear or higher as determined by the neurosurgeon.
 - If continuous drainage is being utilized, it is essential that the level is maintained as the patient moves or is repositioned.
 - The amount of drainage as well as the color, clarity, and presence of sediment are noted at least hourly.
 - Also hourly, the nurse accurately determines the ICP by temporarily turning off continuous drainage.

Adam's ICP suddenly increased to 40. His pupils became fixed and he exhibited decerebrate posturing. How should the nurse have responded?

Depending on the institution, the nurse might have standing orders to provide additional stat mannitol or to acutely hyperventilate Adam while notifying the physician.

- Compressing veins and shunting of blood out of the venous sinuses

These compensatory mechanisms allow for small increases in the volumes of components in the skull to occur without a significant increase in intracranial pressure.

- 10.2 Secondary injury produces ongoing increases in intracranial pressure and additional damage to the patient who has sustained a brain injury. These secondary insults are remediable—thus, preventing or limiting them is one of the major ways that the long-term outcomes from cerebral injury can be improved.
- 10.3 Admission motor score on the Glasgow Coma Scale.
- 10.4 An intracranial pressure of 34 is significantly elevated and a cerebral perfusion pressure of 50 is low. The nurse will need to check standing orders and to notify the physician. Actions that might be indicated include draining CSF, providing mannitol, changing the patient's position, changing the amount or type of IV fluid, or providing sedation carefully.
- 10.5 In the United States, an average of 1.4 million traumatic brain injuries (TBIs) occur each year, resulting in 235,000 hospitalizations and 50,000 deaths. Head injuries represent 2% to 3% of all causes of deaths and 26% to 68% of trauma deaths. These large numbers of injuries are especially problematic because TBI is one of the most disabling of injuries. Approximately 5.3 million Americans are living with long-term or lifelong disability following hospitalization for a TBI. In addition, in the first 3 years postinjury, patients recovering from a TBI are more likely to report binge drinking, develop epilepsy, and die.
- 10.6 Epidural hematomas usually occur in conjunction with a skull fracture and result from a laceration of the middle meningeal artery causing bleeding between the dura mater and the skull. Approximately half of the patients who suffer this injury demonstrate the classic presentation of an initial loss of consciousness followed by a lucid interval then a sudden reloss of consciousness with rapid deterioration in neurological status. Because the origin of the bleeding is arterial, the hematoma accumulates rapidly and must be evacuated immediately. Patient outcomes are often good if the condition is identified promptly and the patient is taken to the OR for immediate surgical evacuation of the hematoma. Subdural hematomas usually are the result of countercoup injuries, occurring on the opposite side of the skull from the injury just below the dura. In contrast to an epidural hematoma, this hematoma is usually the result of venous bleeding, often originating with the stretching of bridging veins. Subdural hematomas are classified as acute, subacute, and chronic. The presentation and management depend on the specific type.

Review Questions

- 10.1 Normally, the intracranial pressure is kept in a range between 5 and 15 mmHg by a mechanism known as compliance. When the volume of one of the components increases, the body may respond by:
 - Displacing CSF into the lumbar cistern
 - Reabsorbing more CSF

- 10.7** The initial priorities in the management of any trauma patient are the same—the ABCs with assessment and treatment of any injury upon discovery. When these are completed then D (Disability), the neuro assessment, begins.
- 10.8** In order to prevent shivering, the nurse might:
- Use higher temperatures on the cooling blanket (as high as 23.9°C [75°F]) so the patient's temperature does not drop as rapidly.
 - Wrap the patient's arms and legs in cloth towels and position them so they are off the blanket.
 - Place a bath blanket between the patient and the hypothermia blanket to prevent burning the patient's skin.
 - Sedate or even paralyze the patient.
- 10.9** The classic presentation of meningitis includes fever; headache; neck stiffness; photophobia; nausea; vomiting; and changes in mental status such as irritability, lethargy, confusion, and coma. When a patient complains of the symptoms and displays the classic signs of meningitis on focused neurological assessment, the nurse should bypass the usual history taking and proceed immediately to definitive diagnosis and emergent management.
- 10.10** Between 10% and 25% of patients experiencing a severe TBI develop seizures during the first week after a traumatic injury. Seizure activity in the first week may cause secondary brain damage as a result of increased metabolic demands, raised intracranial pressure, and excess neurotransmitter release. Therefore, the nurse needs to be vigilant in observing for the development of such seizures, notify the physician immediately should a patient experience a seizure, and anticipate that anticonvulsant therapy may be indicated.

Chapter 11

Why/Why Not?

Why?

The daughter has accurately identified one of the elements of the Cincinnati Prehospital Stroke Scale, a facial droop. She is also correct that there is a time limit on how long after the development of symptoms rt-PA may be administered.

Why Not?

There are a number of criteria that a stroke survivor must meet in order to be eligible for rt-PA. However, at this point there is not enough evidence to determine if this woman meets the first criterion which is that the thrombolytic be given 3 hours from the onset of symptoms. The onset of

symptoms is not the time of discovery of the symptoms. The daughter returned from work to discover the symptom but it is unclear when the symptom actually developed. That is why it is important to determine when the patient was last seen well (last seen without symptoms). If a neighbor had seen the patient well 15 minutes before the daughter returned from work, that would have established a last seen well time which could be used to calculate the time frame during which the patient would be eligible for a thrombolytic.

Case Study

1. **A.** Monitor respiratory status. (The patient is at risk for impaired breathing due to possible damage of cerebral tissues.)
B. Monitor cardiac status. (The patient's BP is already elevated and further increase may cause complications. Cardiac monitor will reveal if arrhythmia is present.)
C. Monitor neurological status. (Frequent monitoring of neurological status is needed to detect changes and impending complications.)
D. Monitor temperature. (The patient's temperature is already elevated. Hyperthermia develops when the hypothalamus is affected.)
E. Initiate NPO status. (The patient may be at risk of a swallowing deficit and further assessment will be needed when stable.)
2. Accurate time of symptom onset
Any recent medical, surgical, or trauma events
Risk factors (Note any of the following as risk factors: male gender, over 55 years of age, prior stroke, TIAs or MI, carotid or other artery disease, hypertension, atrial fibrillation, hyperlipidemia, diabetes mellitus, tobacco use, excessive alcohol use, obesity, inactivity, and sickle cell anemia.)
Medications (particularly antihypertensive, antiplatelet, and anticoagulant agents and over-the-counter medications such as aspirin)
3. The admission assessment revealed a history of hypertension, carotid stenosis, and TIAs.
4. Neuroimaging (ideally, a CT or MRI should be ordered within 4 to 5 minutes and read within 20 minutes of patient arrival to the ED). Lab services: Blood tests that should be obtained immediately are CBC, chemistry, and coagulation profiles. A chest x-ray and ECG should be performed within 45 minutes of arrival to ED.
5. His treatment options depend on the time since his arrival in the ED and the capabilities of the hospital to which he has been transported. If his arrival is timely and the hospital has immediate radiological and neurological consultation, an IV thrombolytic might be provided. In a tertiary center, the thrombolytic might

be provided to the appropriate cerebral artery, or another invasive method of ensuring arterial patency might be attempted. If he arrived outside the safe time frame for thrombolysis, treatment would involve watchful waiting with management of increased intracranial pressure and prevention of complications.

6. Some post-stroke complications and teaching needs include:

- Risk for falls
- Dysphagia
- Dysphasia
- Immobility
- Incontinence
- Constipation
- Skin breakdown

Review

11.1 The most common generalized symptoms of a brain tumor are headache, nausea, vomiting, drowsiness, visual problems, and changes in personality. Focal symptoms are more specific symptoms that result from tumor irritation of brain tissue. Focal symptoms include hearing problems such as ringing or buzzing sounds or hearing loss, decreased muscle control, lack of coordination, decreased sensation, weakness or paralysis, difficulty with walking or speech, balance problems, or double vision.

11.2 The decrease in cerebral edema may occur because glucocorticoids directly affect vascular endothelial cell function and restore normal capillary permeability. In addition, dexamethasone may cause cerebral vasoconstriction.

Nursing responsibilities include the following: Side effects from glucocorticoid therapy are common, so it is important to determine that the patient is among the 70% to 80% of patients who benefit from the therapy if it is to be continued. To avoid adverse effects, the dose is adjusted to the minimum that will control the patient's symptoms. Even at the lowest appropriate dose, typical doses given to patients with brain tumors have the potential to suppress the hypothalamic-pituitary-adrenocortical (HPA) axis. If the dose is to be discontinued, it should be tapered to allow the HPA axis time to recover. In addition, if the steroids are abruptly discontinued, rebound edema may occur and the patient may have an abrupt return of neurological symptoms. Tapering schedules for dexamethasone vary depending on the length of time the patient has been on the steroid and the patient's symptoms.

Potential Side/Toxic Effects: Adverse effects are dose and time dependent, with as many as 50% of patients experiencing at least one toxic symptom.

Common effects include euphoria, with excessive feeling of well-being and insomnia; increased appetite, especially for sweets; weight gain; hyperglycemia, particularly in diabetics; hypertension; muscle weakness in the legs (the patient may complain of inability to climb stairs or arise from chairs); stomach ulcer; and increased risk of infection.

The nurse may need to educate the patient and family concerning muscle weakness because they may fear it is an indication of worsening neurological function. Dietary counseling is important to prevent excessive weight gain and high blood sugars. Nurses should encourage the patient to take steroids with food and avoid aspirin and nonsteroidal anti-inflammatory agents to prevent gastric ulceration and bleeding. When patients with brain tumors receive steroids for a prolonged period, their CD4 count may drop sufficiently to predispose them to opportunistic infections. Nurses should inspect the mouths of these patients to detect the presence of oral and esophageal candidiasis. To prevent the development of pneumocystis pneumonia, Bactrim might be administered.

11.3 The nursing responsibilities include:

- Maintaining head of the bed elevated 30 to 45 degrees
- Providing a moustache dressing
- Assessing for CSF leakage on the dressing
- Discouraging the patient from sneezing and blowing the nose
- Assessing the patient for a visual field defect
- Assessing pituitary function and identifying the presence of diabetes insipidus

11.4 Pneumatic compression boots and graduated compression stockings have been shown to decrease the occurrence of VTEs without increasing ICP. An alternative is the use of compression boots prior to, during, and for 24 hours after the surgery followed by low-dose heparin 5,000 units twice a day or enoxaparin 40 mg/day.

11.5 The two major categories of CVA, hemorrhage and ischemia, are totally opposite conditions. Whereas hemorrhage is defined as rupture of a blood vessel and too much blood within the closed cranial cavity, ischemia is defined as too little blood supply with inadequate oxygen and nutrients to part of the brain.

11.6 The four major neuroanatomic stroke syndromes are middle cerebral artery occlusions, anterior cerebral artery occlusions, posterior cerebral artery occlusions, and vertebrobasilar artery occlusions.

11.7 A deterioration in mental status, such as restlessness or lethargy and the development of focal neurological deficits, most likely hemiparesis or dysphasia, suggest that the patient might be developing vasospasm.

Symptoms may wax and wane, changing from minute to minute. They tend to become more apparent when the patient's blood pressure drops and less obvious when the blood pressure increases.

11.8 Screening for dysphagia is essential in a stroke survivor because it is a very common complication and is associated with the development of aspiration pneumonia. To determine if a patient has a swallowing deficit, the nurse might use the following swallow screening criteria. Prior to swallow screening, the nurse should:

- Evaluate lung sounds and obtain the most recent vital signs, including temperature.
- Evaluate the ability of the patient to follow directions.
- If the patient demonstrates any of the following problems at any time during the assessment, the nurse should cease the evaluation, keep the patient NPO, and ask the MD for a speech therapy order for a swallowing evaluation: coughing before, during, or after a swallow.
- Gurgly/wet vocal quality or any voice changes
- Need to swallow two or more times to clear
- Excessive length of time to move food to the back of the throat to swallow
- Pocketing of food
- Excessive secretions

The nurse should consider each of the following when doing a swallow screening:

- a. Does the patient have facial weakness or a droop?
- b. Does the patient have difficulty with arousal?
- c. Does the patient have an absent gag reflex?
- d. Given one bite of applesauce, does the patient cough or clear her throat?
- e. Given one sip of water, does the patient cough or clear her throat?
- f. Given consecutive sips of water, does the patient cough or clear her throat?
- g. Given a graham cracker or saltine, does the patient have difficulty chewing, any oral residue, and can the patient cough/clear the throat?
- h. Does the patient need to swallow more than one time per bite/sip?

Chapter 12

Why/Why Not?

Why?

The nurse might consider that the patient is currently bleeding and the blood is available. It is not possible to determine the patient's baseline blood pressure and the hemoglobin is less than 10 g/dL. So, the nurse might wonder if blood would be appropriate.

Why Not?

The patient's MAP is greater than 65 and his heart rate is less than 100 which indicate she is still hemodynamically stable. Half of the patients with a variceal bleed spontaneously stop bleeding, resulting from hypovolemia triggering splenic vasoconstriction and a decrease in portal pressure. When a patient is hemodynamically stable, caution is taken to avoid fluid overload, which can trigger a rebound increase in portal pressure, resulting in the development of early rebleeding regardless of the initial treatment. According to practice guidelines, the goal is to keep the hemoglobin 7-8 g/dL. The nurse should continue to observe this patient for any indication of continued bleeding or the development of hemodynamic instability. If those developed transfusions might be indicated.

Case Study

1. The first priority in Barbara's care should be the maintenance of her airway while she is vomiting. If her airway has been ensured, then the nurse must attend to her hemorrhagic shock.
2. The nurse will need additional assessment information, especially a rapid full physical assessment, including the following: What is her respiratory status, how well is she perfusing (manifestations of shock) her MAP, and is she still oriented? The nurse will also want to review some lab data such as how many units of blood have been typed and crossed for her and how many are available, what are the rest of her lab studies—specifically her clotting studies? What is the current plan? Will Barbara be undergoing endoscopy? If so, when? If not, what measures will be attempted to decrease the bleeding?
3. Barbara has longstanding liver disease and it is likely that her clotting studies will reveal an abnormality. She is currently retching and vomiting and that will make bleeding from varices more severe.
4. She has severe liver disease, including a previous bleed, and she is continuing to drink alcohol. All of these factors contribute to the likelihood that she would rebleed.
5. Gastric lavage might be performed prior to endoscopy or erythromycin might be administered in an attempt to clear the stomach of blood and clots, thus making it easier to locate the source of the bleeding. Additionally, the critical care nurse should:
 - Maintain NPO status ideally.
 - Ensure informed consent.
 - Remove dentures.
 - Monitor baseline vital signs, including temperature and oxygen saturation.
 - Provide patient/family education.

- If the patient is being mechanically ventilated, consult the endoscopist and respiratory therapy related to temporarily adjusting ventilator setting to maximize oxygenation during sedation.
- 6. Esophageal variceal ligation (EVL) involves suctioning the varix into the scope cylinder and deploying a band around the varix. The band strangulates the varix, causing thrombosis and obliteration. It is an effective therapeutic modalities in stopping the bleeding in the majority of the patients. However, in situations in which there is severe active bleeding and poor visibility, banding may be more difficult. The use of endoscopic therapy in combination with a vasoactive agent (octreotide) is more successful than either therapy alone.
- 7. Barbara has only been home for about a week. It is unclear how much she was able to drink during that week and she may not undergo alcohol withdrawal during this hospitalization.
- 8. The nurse should complete an assessment of Barbara, review the lab findings, and notify the physician.
- 9. Because Barbara has had repeated esophageal bleeding and multiple episodes of ascites, she might be considered for TIPS.

Barbara has most likely developed hepatic encephalopathy. The collaborative management involves identifying and treating precipitating factors (in this case the upper GI bleed and hypokalemia), determining Barbara's serum ammonia level, and using lactulose or oral antibiotics to lower the level.

- 10. Barbara is at high risk for either an invasive or surgical procedure to lower portal pressure. However, once she is no longer actively bleeding and has stabilized, only reduction in portal pressure will be likely to prevent her from recurrent bleeding.

Review

- 12.1 When exposed to repeated doses of alcohol, the central nervous system (CNS) becomes accustomed to the depressant effects of the alcohol and produces adaptive changes in an attempt to function normally. In the absence of or with a significant decrease in the amount of alcohol, chaos erupts within the CNS. When alcohol is no longer acting as a depressant, the compensatory actions cause excessive CNS excitability. It is analogous to having an accelerator without a brake. The time course of withdrawal is determined by the time it takes to restore balance.
- 12.2 The nurse should obtain a complete history in a non-threatening manner from the patient and/or family. It is important to consider a patient's nonverbal responses, anxiety, and presence or absence of eye contact for clues. The nurse questions the patient/family related to:

- Current and past alcohol use and family history of alcohol problems
- Abuse and dependence are more prevalent in families in which first-degree relatives have been afflicted.
- Quantity and frequency/pattern of alcohol use
- Severity can depend on duration and quantity of consumption.
- Patients/families may underreport consumption and abuse or deny it.
- History of liver disease or other alcohol-related illnesses and previous withdrawals

Additionally, a standardized questionnaire should be utilized to detect dependency. There are several reliable and valid questionnaires available. One such questionnaire is the CAGE. It is a simple, fast, short, reliable, and valid questionnaire. The acronym helps the clinician to recall the following questions:

- Have you ever felt the need to CUT down on drinking?
- Have you ever felt ANNOYED by criticism of your drinking?
- Have you ever had GUILTY feelings about your drinking?
- Have you ever had an EYE opener first thing in the morning to steady your nerves or get rid of a hang-over?

- 12.3 The CIWA-Ar is the scale most commonly used to assess the intensity of withdrawal and to provide appropriate medical therapy.
- 12.4 DTs are the most severe complication of alcohol withdrawal and patients who develop DTs may die.
- 12.5 Alcoholic hallucinosis can manifest 12 to 24 hours after the last alcohol ingestion. With alcohol hallucinosis the patient experiences perceptual disturbances—usually visual, auditory, or tactile phenomena—without sensorial alterations. Patients are fully conscious and aware of their environments, acknowledging that the hallucinations are related to the substance dependence and withdrawal. The distinction from DTs is that in DTs the patient experiences disorientation and global confusion.
- 12.6 The following are all safety concerns for a patient with AWS who may be confused, disoriented, and hallucinating:
 - Accurate assessment and appropriate medication administration per protocols (vital signs and LOC)
 - Implementation of one-to-one continuous observation and monitoring
 - Institution of fall protocols relative to disorientation and sedation
 - Seizure precautions
 - Aspiration precautions
 - Physical restraints to prevent injury to patient and staff

12.7 Benzodiazepines are the foundation of pharmacological therapy for AWS. These agents serve as a substitute for alcohol by acting on inhibitory GABA mediators replacing the depressant effects on the CNS. As a substitute for alcohol, they counteract hyperactivity, thus known to have “cross tolerance.” In addition to their sedative-hypnotic effect, they also have anticonvulsant properties and less adverse effects than other drugs in this classification. Benzodiazepines have been proven to be safe and effective in preventing and reducing withdrawal severity, including seizures and delirium.

The nurse anticipates that the route of administration and dosing will be guided by the clinical picture and the kinetic properties of the benzodiazepine chosen. The therapeutic goal is to achieve light somnolence. Evidence of light somnolence is that the patient sleeps when not stimulated, yet is easily arousable when sleeping. Intravenous administration has the quickest onset. For severe withdrawal, the nurse can expect to be administering larger doses of intravenous benzodiazepines.

12.8 When AWS develops in critically ill patients, it may compound and confuse an already difficult situation.

12.9 In acute liver failure, there is sudden dramatic loss of liver function. It is a medical emergency with a high mortality rate. Treatment is dependent on the specific cause. Chronic liver failure is usually the result of excessive alcohol misuse, viral hepatitis, or nonalcoholic fatty liver disease. It may eventually result in cirrhosis.

12.10 Acetaminophen overdose is dangerous because, if untreated, it has the potential to result in acute liver failure and/or death.

12.11 N-acetylcysteine (NAC) (Mucomyst) is an antidote that counteracts the effects of acetaminophen toxicity. When administered within 12 hours of ingestion of a single dose it can eliminate significant hepatic injury. Oral dosing is initiated on nonpregnant patients with a functioning GI system and no indications of hepatotoxicity. The loading dose is 140 mg/kg followed by 17 doses of 70 mg/kg every 4 hours. It is typically available in a 20% solution (200 mg/mL) and is diluted to a 5% solution with fruit juice, a carbonated beverage, or water.

12.12 Portal hypertension develops over time, is asymptomatic, and is responsible for an array of complications that can markedly reduce the life expectancy of patients. The following complications are directly attributable to portal hypertension: ascites, variceal hemorrhage, hepatic encephalopathy (HE) (portosystemic encephalopathy [PSE]), and hepatorenal syndrome.

12.13 The nurse will need to assess the patient’s hemodynamic status and neurological functioning and complete an abdominal assessment, specifically noting any indications of liver disease. The nurse will also want to review liver enzymes, serum albumin, clotting studies, serum electrolytes, and CBC.

12.14 The nursing responsibilities for a patient with ascites include:

- Assisting with paracentesis as appropriate
- Maintaining sodium and fluid restriction
- Providing diuretic therapy
- Evaluating the effectiveness of the therapy
- Educating the patient and family about therapy

The nursing responsibilities for a patient with hepatic encephalopathy include:

- Assessing the patient’s respiratory, safety, and neurological status
- Reviewing the patient’s blood ammonia
- Identifying and correcting precipitating factors
- Providing medical therapy (lactulose or oral antibiotics as indicated)

Chapter 13

Why/Why Not?

Why?

The patient is clearly in pain, as shown by the increase in the CPOT scores. The dose for the fentanyl bolus and increase in fentanyl infusion are reasonable for a ventilated patient and would be not be surprising doses for a patient with severe pancreatitis.

Why Not?

The nurse will most likely need to provide the fentanyl bolus and increase in the infusion. However, that should not be the only response by the nurse to the increase in the patient’s pain. The patient had previously had good pain control, the increase in pain is accompanied by a drop in BP and an increase in pulse rate. The nurse should assess the patient fully for worsening pancreatitis accompanied by hypovolemia.

Case Study

1. He is dehydrated and requires immediate fluid resuscitation.
2. It is confirmed by his vital signs; his blood pressure is low and his heart rate is high despite volume replacement. His CVP is equivocal.
3. His recent heavy alcohol intake is likely the precipitating factor.

4. His pancreatitis is likely severe based on his age, his pain, his hypoxia, volume status, and WBC. His Hct at 45 would seem to indicate hemoconcentration and the potential for pancreatic necrosis. His serum amylase indicates that he most likely has pancreatitis but the amylase level is not a reliable predictor of the severity of pancreatitis.
5. Sudden, severe epigastrium pain often peaking within 30 to 60 minutes and lasting hours to days is the hallmark of pancreatitis. It results from irritation and edema of the inflamed pancreas and, in severe cases, the release of pancreatic enzymes into the surrounding tissues.
6. The two essential collaborative interventions are fluid resuscitation and pain relief.
7. Deficient fluid volume related to severe fluid shift.

Review

- 13.1 The most frequent cause of GI bleeding in older adults is the development of GI ulcers in people who are taking NSAIDs for arthritis. NSAIDs can cause local and systemic effects that promote mucosal damage.
- 13.2 Older adults are more likely to present with nonspecific signs such as dehydration and abdominal cramping. The most common manifestations of GI bleeding in other age groups are hematemesis, hematochezia, and melena.
- 13.3 Typically, the patient with a UGI bleed presents with hematemesis and/or melena, whereas an LGI bleed is suspected in patients with hematochezia. Though helpful in evaluating the source, stool color is not absolute. Both UGI and LGI bleeders can have melena and a patient with a massive UGI bleed can have hematochezia.
- 13.4 Total fluid deficit cannot be accurately predicted and fluid resuscitation should remain rapid as long as blood pressure remains low. Other clinical parameters guiding resuscitation are vital signs, urine output, mental status, and peripheral perfusion. The nurse anticipates that the patient's vital signs shall begin to normalize and the urine output will increase within 15 to 20 minutes of the challenge.
- 13.5 The important components of discharge teaching include: education of the patient to eliminate precipitating factors such as NSAIDs, aspirin, or Plavix; explanation of how long an acid suppression agent such as a PPI should be taken, preparation for testing for *H. pylori* and the possibility of an eradication program.
- 13.6 Postprocedural nursing responsibilities of the critical care nurse include:
 - If not intubated, maintain recovery position (left lateral decubitus) to protect the airway until the patient is fully awake:
 - EGD patients may have a topical anesthetic sprayed to the throat area, which may impair swallowing.
 - Monitor vital signs, including temperature and oxygen saturation, level of pain, and consciousness until the patient returns to baseline (typically every 10 to 15 minutes for 30 minutes to an hour, then per ICU protocol, more frequently depending on acuity).
 - Monitor for potential complications:
 - Gastrointestinal blood loss (rebleeding)
 - Perforation
 - Aspiration
 - Adverse reaction to procedural sedation medications
- 13.7 The exact mechanism as to how the different predisposing factors induce pancreatitis is uncertain. General consensus regarding pathogenesis is autodigestion. The acinar cells are damaged, causing inappropriate activation of trypsinogen to trypsin. Trypsin activates a cascade of other enzymes that begin digestive functions in the pancreas, resulting in inflammation and tissue damage. The normal defense and inhibitory mechanisms are overwhelmed by the large amounts of activated enzymes.

The release of inflammatory mediators causes increased vascular permeability with varying degrees of edema, hemorrhage, and necrosis.
- 13.8 The acute physiology and chronic health evaluation (APACHE II) and Ranson's early prognostic signs are two commonly used systems to determine the severity of pancreatitis. In addition, there is a CT scan severity grading system. The most critical markers of severity are organ failure (especially MSOF) and pancreatic necrosis. Most recently the hematocrit is being investigated as a possible prognostic marker for the presence of necrotizing pancreatitis.
- 13.9 The two priorities of care are providing fluid resuscitation and pain relief.

Chapter 14

Why/Why Not?

Why?

Mr. P's serum potassium is well below normal levels which can lead to serious heart arrhythmias.

In addition to following up on Mr. P's serum potassium after the infusion, it will also be important to monitor other electrolytes such as magnesium and calcium.

Infuse this medication using an infusion pump, and give it slowly. The usual IV rate of potassium is 10 meq/hour, with a maximum rate (reserved for critical situations) of 20 meq/hour.

Why Not?

Before giving this IV infusion, the nurse will want to assess kidney function as renal insufficiency and failure are a common complication of prolonged hyperglycemia. Review glomerular filtration rate, and creatinine levels before giving this medication.

Potassium chloride 20 meq in 100 ml is very concentrated. Do you want to give this through a peripheral line or a central line? Would it be possible to dilute it further in 200 ml of fluid?

Is the IV in place? Extravasation of potassium can cause severe tissue damage. A central line should be verified by X-ray before use. Positive blood return through a central line should also be checked prior to the administration of IV potassium.

Case study 1

- Initial, concurrent interventions may include the following:
 - Perform a more in-depth assessment, including all body systems.
 - Initiate ECG monitoring.
 - Obtain blood glucose (at bedside).
 - Obtain laboratory studies:
 - Blood for CBC, chemistry panel (electrolytes, glucose, creatinine, BUN), glomerular filtration rate, liver function studies, clotting studies, arterial blood gases
 - Urine for glucose, ketones, and culture
 - Possible cultures: wound, sputum, blood
 - Start intravenous fluids for hydration. Normal saline would be anticipated based on the low blood pressure and high pulse rate.
 - Insert urinary catheter for strict assessment of output.
 - Reassure the patient.
 - Call next of kin or significant other.
- The following laboratory results are needed to calculate serum osmolarity:
 - Na^+ 132, Glucose 644, BUN 31
 - Serum osmolarity calculation: $(2 \times \text{sodium}) + (\text{serum glucose}/18) + (\text{BUN}/2.8)$
 - $(2 \times 132) + (644/18) + (31/2.8) =$
 - $(264) + (35.7) + (11.07) = 310.77$
 - Serum osmolarity = 311 mmol/liter
- The serum osmolarity of 311 is elevated. Normal serum osmolarity is 280 to 300 mmol/liter. This elevated serum osmolarity is a result of elevated glucose.

Fluid will shift from interstitial and cellular tissues to the vascular space in an attempt to dilute the serum. Osmotic diuresis will result in dumping of glucose and electrolytes, causing electrolyte depletion.
- The following laboratory results are needed to calculate the anion gap:
 - Na^+ 132, K^+ 6.2, Chloride 100, Bicarbonate 17
 - Anion gap calculation: $(\text{Sodium} + \text{potassium}) - (\text{chloride} + \text{bicarbonate})$
 - $(132 + 6.2) - (100 + 17) =$
 - $138.2 - 117 = 21.2$
 - Anion gap = 21.2
- The results of the anion gap show metabolic acidosis. The normal anion gap (using this calculation method) is 10 to 17 mEq/L. Arterial blood gas results will also find metabolic acidosis.
- The following laboratory results are needed to calculate the corrected serum sodium:
 - Na^+ 132, Glucose 644
 - CSS calculation: $\text{Serum Na}^+ + \{[(\text{Serum glucose (mg/dL)} - 100)/100] \times 1.6\}$
 - $132 + \{[(644 - 100)/100] \times 1.6\}$
 - $132 + \{544/100\} \times 1.6\}$
 - $132 + \{5.44 \times 1.6\}$
 - $132 + 8.7 = 140.7$
 - Corrected serum sodium = 140.7
- Based on the corrected serum sodium results, the nurse would expect to change IV fluids to 0.45% normal saline solution because the corrected serum sodium is within normal limits.
- This patient weighs 155 pounds. To calculate kilograms, 155 is divided by 2.2. Mrs. D weighs 70.45 or 70 kg. The 24-hour fluid replacement equals 100 mL/kg of weight, or 100 mL $\times$ 70 kg, or 7,000 mL in 24 hours. The nurse would anticipate giving one half of the total fluid volume in the first 8 hours, equal to 3,500 mL. Often, a fluid bolus of 1,000 cc is delivered in the first hour, leaving 2,500 mL to be administered in the remaining 7 hours. The hourly IV rate would be 357 mL/hr $(2,500 \div 7 \text{ hours})$. The other 3,500 mL would be administered in the following 16 hours. The hourly IV rate would be 219 mL/hr $(3,500 \div 16 \text{ hours})$.
- The nurse would anticipate giving regular insulin.
 - The IV route is used for critically ill clients in DKA or HHNS.
 - A common bolus dose is equal to 0.15 unit/kg. Because Mrs. D weighs 70 kg, the nurse would anticipate a bolus dose of 11 units of Regular insulin via the IV route $(70 \text{ kg} \times 0.15 \text{ unit} = 10.5 \text{ rounded to } 11 \text{ units})$. Insulin infusions are often started at 0.1 unit/kg/hr. The nurse would anticipate giving 7 units of Regular insulin via the IV route per hour $(0.1 \text{ unit} \times 70 \text{ kg per hour} = 7 \text{ units per hour})$.

- d. The nurse would expect to adjust the insulin infusion after 1 hour based on hourly blood glucose results. The goal is to decrease blood glucose slowly to prevent complications by infusing 50 to 70 mg/dL per hour.
 - e. The nurse would assess potassium levels and level of dehydration prior to giving insulin. IV fluids should be initiated before starting an insulin infusion. If potassium levels are low, the nurse would anticipate replacing potassium before giving IV insulin.
10. It would be important to assess Mrs. D's basic knowledge of diabetes. The admission history includes an important clue that Mrs. D was in very good control of her diabetes prior to this acute illness. She reported that 2 months ago, her hemoglobin A1C was 6.2. As a result, the focus of teaching during this hospitalization would include awareness of the specific precipitating factor that caused the present DKA episode and a plan to prevent future occurrences. Teaching would focus on the management of diabetes during acute illness and the importance of early communication with the physician during acute illness to prevent ketoacidosis or to treat it early.

Case Study 2

- 11. Mr. S's creatinine and BUN are both elevated, pointing to renal failure. With the presence of both renal failure and heart failure, the nurse will expect fluid intolerance and third spacing of fluid into the lungs and interstitial spaces. Fluid volume overload will be a problem even in the presence of severe dehydration. Because of this complication the nurse would anticipate placement of a Swan-Ganz catheter to accurately measure the outcomes of fluid volume replacement.
- 12. Albuterol causes bronchodilation by relaxing the smooth muscles of the respiratory tree. It is used to improve oxygenation. Dexamethasone is a glucocorticoid steroid. It has anti-inflammatory effects and is also used to improve oxygenation during pneumonia. Both albuterol and dexamethasone increase the blood sugar. The nurse would anticipate the use of increased doses of insulin to meet the goal of decreasing the blood glucose by 50 to 70 mg/dL per hour.
- 13. Based on a hemoglobin A1C of 10.5, Mr. S's average blood sugar for the preceding 2 to 3 months ranged between 275 and 310 mg/dL. Mr. S is at significant risk for long-term complications such as retinopathy, peripheral and autonomic neuropathy, systemic vascular disease, and renal failure. Mr. S already has vascular complications, including a history of myocardial infarction, heart failure, and renal failure. The nurse would plan education and encouragement regarding the poor control of his blood glucose readings. A multidisciplinary approach would be helpful. The physician will review the prescribed medications. A certified diabetic educator (CDE), if available, would be very helpful to help with teaching and guiding decisions. It would also be very important to involve close family members or significant others in the teaching and coaching sessions.
- 14. The following complications are listed with rationales for their development and assessments that would indicate their presence:
 - a. Fluid volume overload would be a common complication for Mr. S due to his preexisting heart failure and renal failure. Important assessments would be decreasing oxygen saturation, decreasing arterial oxygenation, high-pressure alarms on the ventilator, worsening lung sounds, decreased urine output, and changes on chest x-ray.
 - b. Adult respiratory distress syndrome (ARDS) is a possibility due to pneumonia in addition to intracellular fluid shifts during rehydration. Either one of these conditions by themselves could predispose Mr. S to ARDS, though Mr. S has both problems. Initial assessments indicating the presence of ARDS include diffuse rales and diffuse infiltrates on chest x-ray. In contrast, pneumonia is detected by coarse rhonchi in a specific area of the lungs.
 - c. Cerebral edema might develop as a result of intracellular fluid shifts during rehydration. The nurse would assess for headache, lethargy, confusion, and irritability to identify early signs of cerebral edema. A CT scan can also help to identify cerebral edema.
 - d. Thrombosis is a complication that Mr. S might develop as a result of severely high serum osmolality in addition to preexisting vascular disease (evidenced by prior heart attack, heart failure, and renal failure). Thromboses could cause a variety of problems including:
 - Myocardial infarction, identified by chest pain or pressure, and changes on the 12-lead ECG
 - Stroke, identified by inability to speak, one-sided facial droop, and/or one-sided muscle weakness
 - Deep vein thrombosis, evidenced by unilateral swelling of an extremity and abnormal peripheral vascular studies
 - Pulmonary embolism, evidenced by chest pain, hypoxia, and abnormal pulmonary vascular studies

Review

- 14.1 Medications that can increase the blood sugar include steroids, thiazide diuretics, calcium channel blockers, propranolol, phenytoin, and sympathomimetics.

- 14.2** Hyperglycemia has been shown to cause an inflammatory response, resulting in damage to the endothelium of blood vessels. Even short periods of hypoglycemia during critical illness have been linked to poorer patient outcomes. Therefore, attempts are made to maintain the blood glucose between 80 and 110 mg/dL.
- 14.3** The risk factors include central obesity, abnormal lipid panel, fasting blood glucose greater than 110 mg/dL, and hyperinsulinemia. Glucose monitoring should be implemented in order to detect hyperglycemia in this population. Hyperglycemia causes endovascular inflammation and results in increased complications, increased length of stay in the critical care unit, and increased mortality.
- 14.4** Infection, conditions that induce stress (MI, CVA, trauma, or surgery), pregnancy, medications (especially steroids), and misuse of alcohol are the most common precipitating factors.
- 14.5** The patient with DKA often presents with metabolic acidosis, whereas the patient with HHNS usually does not. The patient with HHNS will often have visual disturbances such as blurred vision due to the increased blood osmolality.
- 14.6** The nurse must assess the patient's potassium level and prepare for potassium replacement. The nurse must also assess the patient's volume status and must have initiated volume replacement. In addition, the nurse checks the patient's pH, bicarbonate level, serum sodium, calcium, phosphate, and magnesium.
- 14.7** Common complications following treatment of DKA and HHNS include hypoglycemia, hypokalemia, fluid volume overload with heart failure, cerebral edema, and ARDS.

Chapter 15

Why/Why Not?

Why?

It is essential that the patient receive the appropriate IV antibiotics for the treatment of his endocarditis and he will likely need at least six weeks of antibiotic therapy.

Why Not

The nurse should question the doses and timing of these antibiotics. Antibiotic dosing needs to be closely adjusted as standard dosing may lead to toxicity in the patient with AKI and in those patients receiving hemodialysis. This patient is at high risk for toxicity. The nurse should not administer the Vancomycin until there is a serum level available. Vancomycin levels should be drawn predialysis;

levels taken during dialysis are not accurate. If the serum level is within therapeutic range, the nurse should administer the vancomycin after dialysis or during the last 2 hours of a dialysis run. The ceftriaxone dose is above the maximum dosing for renal failure (1-2 GM IV q12-24 hours) and should be adjusted. Ceftriaxone can be administered anytime during hemodialysis. It is crucial that the nurse be aware of the potential for adverse drug reactions with any medications when a patient has an acute kidney injury.

Case Study

1. She is dehydrated and requires immediate volume replacement. Her BUN, creatinine, and potassium indicate that she is in renal failure.
2. Her CK might be so elevated from muscle breakdown due to her prolonged period on the floor.
3. The most likely cause of her renal failure is rhabdomyolysis, which can result in myoglobin blocking structures in the kidney and cause renal failure. However, her volume depletion and subsequent hypotension probably also contributed to the AKI.
4. Volume replacement (her H & H indicate that she is in need of volume replacement) would be the first essential collaborative management step and should also increase her BP.

If her renal perfusion and urine output improved following volume replacement, then she would most likely be observed to determine that her BUN, creatinine, and CK returned to more normal levels. Hydration is key to management of rhabdomyolysis.

5. Renal replacement therapy would most likely be delayed until her response to hydration was determined and her BP increased.

Review

- 15.1** In prerenal causes of AKI, urinalysis typically shows a concentrated urine with a high osmolality (greater than 500 mOsm/L) and a decreased urine sodium (less than 20 mmol/L). The fractional excretion of sodium (FENa), a test for assessing how well the kidneys can concentrate urine and conserve sodium, is usually less than 1%. There are rarely more than a few casts and/or a little sediment present in the urine. In intrinsic renal failure, urinalysis typically reveals an osmolality less than 350 mOsm/L, an increased urine sodium (greater than 40 mmol/L), and FENa greater than 1% with granular casts and sediment.
- 15.2** Intrinsic or prerenal failure is primarily caused by a prolonged reduction in renal perfusion but may also be precipitated by other conditions, including nephrotoxic agents. By maintaining an adequate MAP and ensuring renal perfusion as well as avoiding

administration of nephrotoxic agents, the nurse may help to prevent AKI.

15.3 Indications of volume excess include: rapid pulse of bounding quality, skin that is pale and cool to the touch, an elevated BP, possibly with decreased pulse pressure.

- One of the earliest indications of fluid excess, occurring prior to the development of edema, may be a gradual increase in BP.
- Other signs of volume excess include: jugular venous distention and/or hepatojugular reflex, nonpitting edema in dependent areas, increased CVP or PAWP and an increase in weight.
- Respiratory indications of volume excess include: increased respiratory rate with shallow respirations, dyspnea on exertion or in supine position and crackles in bases on auscultation.

15.4 Hyperkalemia. It may result in death from diastolic arrest or ventricular fibrillation.

Potassium may be counteracted with IV calcium or driven intracellularly with insulin and glucose or sodium bicarbonate. Longer term reductions in potassium might be achieved with a cation exchange resin or renal replacement therapy.

15.5 Peritoneal dialysis is often not speedy or efficient enough to adequately remove the midsized wastes such as urea that accumulate rapidly in catabolic AKI patients. Second, the volume of fluid that is placed in the peritoneum in PD tends to have a negative impact on respiratory function. Lastly, several studies have demonstrated poorer outcomes for patients who received PD rather than other modalities of treatment for AKI.

15.6 Daily hemodialysis results in a higher rate of return of renal function and decreased mortality for critically ill patients in AKI. Excess fluid can be removed as quickly as the patient can tolerate and electrolyte imbalances may be returned to normal more quickly.

15.7 Dialysis disequilibrium is especially common in patients undergoing their first or second dialysis treatment who experience sudden, large decreases in their BUN. The most likely explanation for this syndrome is that the levels of urea do not drop as rapidly in the brain as the plasma because of the blood-brain barrier. The higher levels of urea in the brain result in an osmotic concentration gradient between the brain cells and the plasma. Fluid enters the brain cells by osmosis until the concentration levels equal that of the extracellular fluid, resulting in cerebral edema and the dialysis disequilibrium syndrome. Manifestations of the syndrome include:

- Headache and mental impairment that may progress to confusion, agitation, and seizures
- Nausea that may lead to vomiting

15.8 Convection occurs when replacement fluid flows through the filter at a fast rate (1,000 to 2,000 mL/hr), creating a “solute drag” that removes fluids and small to mid-sized molecules.

Chapter 16

Why/Why Not?

1. Why?

Vasopressin will help to counteract DI but due to its vasoconstrictive effects, it may also stabilize the donor’s BP and is typically the first or second line agent used when the patient is hypotensive. Having a good blood pressure post-operatively will help ensure good perfusion to the allograft.

Why Not?

If the patient is hypertensive and requires treatment for DI, DDAVP might be a better choice than vasopressin. Having a high blood pressure might put stress on fresh suture lines of a newly transplanted graft, increasing the risk for intra or post-operative bleeding.

2. Why?

Transplanted graft survival is dependent on immunosuppression following transplantation. High dose corticosteroids are often used for induction therapy, the dose is then often lowered for maintenance therapy. Steroids are still generally prescribed for the overwhelming majority of transplant recipients; however, there is increasing interest in developing protocols that either avoid or minimize the use of steroids. Steroid-free protocols, however, may increase the risk of chronic rejection.

Why Not?

Though corticosteroids diminish the risk of transplant rejection, they are associated with many long-term adverse effects. The general goal is to minimize drug toxicity and long-term adverse effects without placing the patient at risk for short- or long-term transplant rejection. The 2009 KDIGO clinical practice guideline reports that patients with low immunologic risk who have received induction therapy should consider having steroids discontinued shortly after transplantation in an effort to minimize long-term adverse effects (KDIGO, 2009). Despite this, however, many transplant centers will often continue low dose corticosteroid therapy to reduce risk of allograft rejection and will only avoid steroids on a patient-specific basis.

3. Why?

Appropriate dosing of immunosuppressants is associated with decreased acute rejection rates following transplantation.

Why Not?

Tacrolimus and cyclosporine, like other immunosuppressants in the same class, have a narrow therapeutic range and multiple side effects and toxicities. The therapeutic trough levels of tacrolimus are usually 8-12 ng/ml in the first 3 months and 3 to 7 ng/ml thereafter. For cyclosporine, therapeutic levels are usually 800 to 1000 ng/ml in the first 1 to 3 months and 400 to 600 ng/ml thereafter (Chandraker, 2014). To minimize toxicities and side effects, know the goal level of each medication for the patient as well as the most recent level. Is your patient exhibiting any side effects or toxicities? Has the patient been started on any new medications that may increase plasma concentrations of either cyclosporine or tacrolimus? Perhaps consider discussing checking a level with the ordering healthcare provider before the dose is given, especially if there has not been a level check in a while or the patient has new complaints or symptoms suspicious of toxicity.

4. Why?

Mycophenolate is used for both maintenance and chronic rejection. Studies have shown it works well to prevent graft rejection and coronary stenosis in heart transplantation and improve long-term kidney graft survival when compared to azathioprine (Pellegrino, 2013).

Why Not?

Is the patient exhibiting gastrointestinal side effects? If so, consider discussing dose decrease or change to enteric coated Cellcept with the ordering healthcare provider. Be aware that many non-generic medications might be more expensive for the patient. Is the cost of the medication worth the gastrointestinal side effects? Will the patient continue to take the medication if it is expensive?

5. Why?

Acute rejection risk is highest within the first 3 months following transplantation and so immunosuppression should also be at the highest (Hardinger & Brennan, 2015). ATG is given for induction therapy as well as for acute rejection.

Why Not?

The more immunosuppressed a patient, the more risk of serious side effects. Infection and malignancy are of the most concern with high immunosuppression. Nosocomial infection risk, being greatest in the immediate

post-operative period. The nurse needs to be aware of all strategies to prevent infection in this population.

Case Study

1. Donation from a sibling would most likely provide David with a better match from a healthy donor. He would also be ensured of a kidney and most likely be able to receive the kidney at a much earlier date than if he were to receive one from a deceased individual. Donation from a live individual also allows for planning of the donation and immunosuppression to begin at the optimal time prior to donation.
2. David's sister is a better match because she is an O antigen mismatch. A person with B positive blood can donate to someone with AB positive.
3. The potential kidney donor must be physically fit; in good general health; and free from high blood pressure, diabetes, cancer, kidney disease, heart disease, and a variety of infectious diseases including the human immunodeficiency virus (HIV) and tuberculosis.
4. David should be told to contact his healthcare provider or transplant center for signs and symptoms of rejection or infection. Symptoms of acute rejection of a transplanted kidney include:
 - Weight gain, edema, and an increase in blood pressure
 - Fever, chills, and elevated WBC
 - Graft swelling and tenderness
5. Many transplant centers continue to instruct their patients to:
 - Wash their hands before putting anything in or near their mouth and after toileting
 - Wear a mask when in a crowd for the first few months post transplant
 - Remove plants and flowers in vases from their homes
 - Wash, peel, and/or cook all fruits and vegetables
 - Avoid exposure to live vaccines
 - Avoid environments contaminated by mold, fungus, or water damage
 - Obtain prophylactic antibiotics prior to dental procedures
6. The tremors are a common side effect of the tacrolimus that David is taking for immunosuppression.
7. David is most likely having an acute episode of rejection. He will need to consult his transplant center, will probably be hospitalized, will be provided with increased doses of immunosuppressant medications, and will probably require a change in his normal medication regimen.
8. Long-term use of steroids can result in aseptic necrosis of the hip.

Review

16.1 The four types of rejection associated with the highest morbidity and mortality for transplant recipients:

- Hyperacute rejection usually results in loss of the transplanted organ.
- Accelerated rejection occurs when the recipient has been sensitized to some of the donor antigens resulting in immediate damage to the transplanted organ.
- Acute rejection is a cell-mediated immune response that results in T lymphocytes infiltrating the donated organ and damaging it by secreting lysosomal enzymes and lymphokines.
- Chronic rejection is a combination of humoral and cell-mediated immune responses that usually results in progressive deterioration in organ function. Chronic rejection is more likely to result in loss of an organ or death.

16.2 An individual may “opt in” to the donor system by signing an organ donor card, stating that she desires to have her organs donated, if it is possible, after she dies.

16.3 Brain death is the irreversible loss of function of the brain, including the brainstem. The criteria for the clinical diagnosis of brain death include:

- Evidence of an acute neurological catastrophe that is compatible with the clinical diagnosis of brain death
- Exclusion of complicating medical conditions that may confound clinical assessment (no severe electrolyte, acid-base, or endocrine disturbance)
- Absence of drug intoxication or poisoning
- Core temperature of 32°C (90°F)

The three cardinal findings in brain death are coma or unresponsiveness, absence of brainstem reflexes, and apnea.

16.4 When and how the family is approached about organ donation affects whether they will donate. Families are more likely to donate organs if:

- They are NOT asked to donate at the same time that they are informed their family member has died
- They perceive that the timing of the request is optimal
- They view the requestor as sensitive to their needs
- They are approached in a quiet, private place where they can consider their options
- An organ procurement specialist from an organ donor bank makes the request

16.5 Tacrolimus (FK-506) is associated with fewer episodes of acute rejection 1 year after kidney transplant than cyclosporine.

16.6 The factor most responsible for the predisposition to infection in recipients of transplanted organs is the use of immunosuppressants, which alter the body’s normal protective responses. Two other major contributing factors to the patient’s immediate posttransplant likelihood of infection are the preoperative condition of the patient and nosocomial factors.

16.7 The development of CMV has been demonstrated to contribute to acute and/or chronic allograft injury and dysfunction as well as to an increased risk for opportunistic bacterial, viral, and fungal infections.

16.8 If at all possible, nutritional requirements are provided enterally because parenteral nutrition is associated with increased risk of infection in the transplant recipient.

16.9 Cardiac allograft vasculopathy (CAV), an aggressive diffuse form of arteriosclerosis, is common in heart transplant recipients, occurring in 40% to 50% of them 5 years after surgery. Unlike the stenoses that develop in CAD that are localized to one section of an artery, CAV can affect the entire length of an artery in the transplanted heart.

16.10 Healthcare providers have not been successful at identifying ways to predict a patient’s compliance with the complex posttransplant regimens. Therefore, it is unclear which patients are most likely to be non-compliant post transplant. Noncompliance appears to be relatively common during the first year after transplantation and tends to worsen as time from transplantation increases. Nurses can help by working with the individual patient to determine what issues (e.g., changes in physical appearance, cost of medications, the complexity of the regimen, feeling of split personality, or depression) may be making it difficult for the person to comply with therapy.

Chapter 17

Why/Why Not?

Why?

Yes, the nurse should reevaluate him. The patient with a lower airway injury may have normal blood gases and chest x-ray for as long as 24 hours after the burn injury. Inhalation injuries are associated with increased risks of infection, respiratory failure, and ARDs. The nurse should reevaluate the patient to determine if he has developed respiratory problems and to prevent potential complications.

Why Not?

It’s possible that the patient is highly anxious, because a house fire is a very frightening event. He may be concerned

about returning home and seeing the extent of the damage to his home. Thus, he may not have any underlying respiratory problem; he may simply be displaying the anxiety which is to be anticipated with such an event. Still, it would seem prudent for the nurse to reevaluate and suggest the physician reevaluate the patient's respiratory status prior to discharge especially considering the amount of time he spent in the sooty environment and the potential for respiratory difficulty to develop for up to 24 hours after a lower airway injury.

Case Study

1. Jon has no clothing on his upper body; the burn process has stopped. Attempting to cool him now may induce hypothermia.
 2. Immediate signs of inhalation injury are changes to the mucosal lining of the oropharynx and larynx, including the presence of soot, edema, or blisters. Symptoms of direct injury occur over the first 24 hours and include stridor, hypoxia, and respiratory distress.
 3. The ED team will secure an airway and most likely intubate the patient if they suspect inhalation injury. Carboxyhemoglobin levels should be obtained for individuals burned within an enclosed space or with signs indicating inhalation injury. If inhalation injury is suspected, a bronchoscopy may be performed.
 4. The location and appearance of all burn wounds should be assessed as to size and depth of injury. Patients with burns to the chest that are deep or circumferential are at increased risk for developing respiratory problems and may require escharotomies to ensure adequate respiration. All extremities should be assessed for circumferential burns to identify areas at risk for loss of perfusion as burn edema progresses. Peripheral nerve stimulation is difficult to do on edematous areas but should be attempted if circumferential extremity burns are present. A diagram of the burn injury should be drawn during the burn assessment using either the rule of nines or the Lund and Browder Chart (see Figures 17-2 and 17-3). The percentage TBSA of second- and third-degree burns, and the total % TBSA burned should be calculated.
 5. Lactated Ringer's is the preferred fluid for resuscitation because it is most like normal extracellular fluid. Normal saline contains a large amount of chloride and if a burn patient gets large amounts of chloride, there is a potential for metabolic acidosis. Fluid that contains dextrose is not used because it does not contain any electrolytes and the patient may be glucose intolerant. The amount of fluid that is required is calculated by whichever specific formula the burn unit recommends. Generally, adults and children with burns greater than 20% TBSA should undergo formal fluid resuscitation using estimates based on body size and surface area burned. Common formulas used to initiate resuscitation estimate a crystalloid need for 2 to 4 mL/kg body weight/%TBSA during the first 24 hours.
- The nurse will know that fluid replacement is adequate by the amount of urine that Jon is producing. He should be maintaining a urine output of 100 mL/hr.
6. Jon most likely sustained these injuries when he jumped through his second floor window. Although they will require treatment, he is currently able to move all his extremities and they are not life threatening.
 7. The nurse should obtain the following information at a minimum for the ED team:
 - Ventilator settings
 - Complete assessment, including a neurological assessment
 - Estimation of extent of burn
 - Calculation of fluid resuscitation needs
 - Medications administered (pain, tetanus, etc.)
 - History of the event
 - Availability of family
 8. The nurse should anticipate the following:
 - Potential for renal failure
 - Development of edema and potential need for an escharotomy
 - Potential for pneumonia and ARDS
 - Potential for development of peptic ulcer
 - Risk of infection
 9. The nurse should institute the following strategies:
 - Maintaining adequate fluid resuscitation
 - Initiating prophylaxis for peptic ulcer
 - Instituting VAP protocol
 - Instituting CVC protocol
 - Maintaining aseptic technique

Review

- 17.1 The most common burns in both adults and children are thermal in nature and are caused by exposure to heat. Thermal burns include fire/flame injuries, scald injuries from exposure to hot liquids or steam, and contact/friction. The majority of pediatric burn injuries and deaths result from scald injuries and residential fires. The most significant burn injuries in older adults are from flame injuries from smoking or scald injuries.
- 17.2 A partial-thickness, or second-degree, burn involves the epidermal and dermal layers of the skin, but the hair follicles, sebaceous glands, and epidermal sweat glands remain intact. A second-degree burn is further

classified by the amount of dermis involved into either a superficial or deep second-degree burn. A superficial second-degree burn does not involve the entire depth of the dermis. The burn wound is often bright red in color and edematous. Capillary refill is near normal. The surface is moist and thin-walled; fluid-filled blisters appear within minutes. A superficial second-degree burn is very painful and sensation to even very superficial pressure, such as air currents, is increased. Healing occurs within 21 days with little to no scarring.

A deep second-degree burn involves the entire dermis. The wound appearance is white and waxy and capillary refill is decreased. The surface may be wet or dry and blisters can range from large and fluid filled to flat. There is less pain and decreased sensation even to deep touch. A deep second-degree burn heals in approximately 3 weeks and scarring is likely. Initially, a deep second-degree burn may appear to be a full-thickness burn but after 7 to 10 days skin buds and hair growth will be noticeable, indicating that the skin appendages are intact. However, some burns that initially appeared to be deep second-degree burns may progress to full-thickness injuries due to decreased blood supply or infection.

A full-thickness, or third-degree, burn involves all layers of the skin, including the hair follicles, sebaceous glands, and the epidermal sweat glands. The wound color ranges from very pale to bright red; but unlike a second-degree burn, there is little to no capillary refill and thrombosed blood vessels may be evident. The surface of the wound is dry and firm and may have a leathery feel. A third-degree burn has no sensation to deep pressure and there is no pain because the nerve endings have been destroyed.

- 17.3** Immediate signs of inhalation injury are changes to the mucosal lining of the oropharynx and larynx, including the presence of soot, edema, or blisters. Symptoms of direct injury occur over the first 24 hours and include stridor, hypoxia, and respiratory distress.
- 17.4** A burn injury can cause a variety of cardiovascular system changes, particularly in individuals with pre-existing cardiovascular disease. The most common cardiovascular change is hypovolemic or burn shock. Other changes include alterations in cardiac rhythm and peripheral extremity vascular compromise.

Within minutes of a burn injury, increased capillary permeability in burned areas where the micro-circulation is not destroyed results in edema formation. The extent of edema depends on burn severity and body parts affected. Burn edema is

greatest over areas where the skin is less elastic, such as the face and genitals, and less over areas with increased elasticity, such as the extremities. Increased vascular permeability is present for approximately the first 24 hours after injury.

This increased capillary permeability explains the need for immediate fluid resuscitation and the potential for severe edema and altered tissue perfusion.

- 17.5** The immediate priorities when a burn victim arrives at a hospital are the same as for any trauma victim, the ABCs.
- 17.6** Full-thickness burn wounds require grafting.
- 17.7** Patients vary in how they respond to a burn injury depending on their previous coping skills and their social support. At first the burn survivor may not be aware of the seriousness of the injury. However, with realization may come fear, which can contribute to the patient's perception of pain. Patients may use dissociation, disintegration, and depersonalization to cope with the injury. As their body image changes and functional limitations become apparent, they may experience depression. PTSD may develop either soon or years after a burn injury; it is predictive of poorer outcome and quality of life. Therefore, it is important that members of the burn team assess for development of PTSD. While caring for burn patients, it is important for nurses to remember that most patients recover from the emotional turmoil and return to leading productive lives.
- 17.8** Children are at increased likelihood for burn injury due to their impulsivity. Both children and older adults may be unable to move away from a burn source, making them more likely to be injured. They both also have thinner skin, making it more likely that they will suffer more severe burn injuries at lower temperatures. Finally, older adults may be more likely to suffer burn injuries due to cognitive and sensory impairments.

Chapter 18

Why Why Not?

Why?

An 81-year-old patient is more likely to become septic and pneumonia is a frequent cause of severe sepsis in older adults. The vital signs all indicate that the patient is deteriorating and could be developing severe sepsis. Although the MAP is not yet less than 65, the trend is apparent and if the nurse waited the patient would likely deteriorate further. The earlier the onset of treatment, the better the outcome is likely to be.

Why Not?

The patient clearly meets the criteria for SIRs but does not display the criteria necessary for sepsis yet. The resident might say that the midnight assessment was likely in an 82-year-old patient with pneumonia who had not yet started to respond to the antibiotics administered. Yet, if the nurse wants to assure the patient is adequately assessed and treated as soon as possible, shouldn't the nurse notify the resident after the midnight assessment?

Case Study

1. The top three priorities are:
 - a. Maintenance of adequate air exchange
Check the patient's airway.
Determine his ventilation (check his O₂ saturation, note the effect of PEEP on his ICP and BP, check his ABGs, position him appropriately).
Check the functioning of his chest tube (note drainage and crepitus about the insertion site).
 - b. Maintenance of adequate circulation and perfusion
Monitor for blood loss (determine possible sites, check Hgb and Hct).
Monitor BP, pulse, and urine output.
Determine the appropriate position and level of HOB.
Provide volume replacement as indicated.
Provide pressors as needed.
 - c. Assessment of neurological status
Assess the level of neurological functioning (on and off sedation), compare to pre-op.
Maintain HOB level.
Check functioning of the Jackson-Pratt drain.
2. Additional concerns include:
 - Pain relief
 - Provision of adequate nutrition
 - Prevention of infection
 - Identification of potential alcohol withdrawal and prevention
 - Stabilization of leg and prevention of further injuries
3. Priority concerns for managing a chest tube are:
 - Ensure accurate setup.
 - Monitor drainage, tidaling, bubbling in suction control chamber.
 - Prevent dependent loops.
 - Assess the insertion site for crepitus and drainage on the dressing.
 - Position the patient upright if possible; turn every 2 hours for air removal.
4. The Ventilator Bundle should be instituted.

5. Possible sources of infection include:
 - Sinusitis
 - Neurosurgical site
 - Lungs from VAP
 - Central lines
 - Urinary catheter
 - Left leg
6. The criteria for SIRs includes
 - Temperature greater than 38°C (100.4°F) or less than 36°C (96.8°F)
 - Pulse greater than 90 beats per minute
 - Respiratory rate greater than 20 breaths/minute or PaCO₂ less than 32 torr
 - WBC greater than 12,000/mm³ or less than 4,000/mm³
7. Yes, he meets all of the criteria.
8. He meets the criteria. Severe sepsis occurs when dysfunction of two or more organ systems develops in response to hypoperfusion.
 - Acute lung injury indicated by tachypnea and/or hypoxemia
 - Coagulation abnormalities or thrombocytopenia
 - Neurological dysfunction indicated by a sudden change in mental status with possible confusion or psychosis
 - Renal dysfunction evidenced by a decreased urine output for at least 2 hours and an increase in serum creatinine greater than 0.5 mg/dL
 - Liver dysfunction indicated by jaundice, coagulopathy, decreased protein C levels, and increased D-dimer levels
 - Gastrointestinal injury indicated by stress ulceration, ileus (absent bowel sounds), and malabsorption
 - Cardiac dysfunction indicated by tachycardia, dysrhythmias, hypotension with decreased CVP or PA pressures, and either high or low cardiac outputs
 - Hypotension with lactic acidosis

He also meets the criteria for septic shock. The most severe form of sepsis is defined as the presence of sepsis and refractory hypotension. Refractory hypotension is a systolic BP less than 90 mmHg, a mean arterial pressure less than 65 mmHg, or a decrease of 40 mmHg in systolic BP that is unresponsive to a crystalloid fluid challenge of 20 to 40 mL/kg.
9. A continuous infusion of norepinephrine will be useful to increase his blood pressure and probably improve his kidney function.
 - PEEP might be increased with caution for ARDS to improve his oxygenation, but will likely affect his BP adversely so the nurse will need to monitor his blood pressure closely.

- Rather than increasing his current sedation, which does not appear to be working, it might be worth trying a different sedative.
10. Yes, he has already developed ARDS (see Respiratory assessment) and most likely also has acute renal failure.

Review

- 18.1 The majority of septic patients, approximately 70% of them, receive care in an intensive care, coronary care, or intermediate care unit. Sepsis affects the elderly disproportionately; more than half of all annual costs—\$8.7 billion—were spent on care of septic patients 65 years or older. Because sepsis rates rise sharply with age, as the nation ages the incidence of sepsis will increase. There will be almost a million cases of sepsis in the United States by the year 2010.
- 18.2 Elevation of the head of the bed (HOB) to at least 30 degrees is recommended because it reduces the risk of aspiration of oropharyngeal contents.
- 18.3 The elements of the central line bundle are hand hygiene, maximal barrier precautions on insertion, chlorhexidine skin antisepsis, optimal catheter site selection, and daily review of line necessity with prompt removal of unnecessary lines.
- 18.4 Evidence suggests that shaving a surgical site results in an increase in surgical site infections. Therefore, if the site must be cleared, either clipping the hair or using a depilatory is recommended.
- 18.5 Assessment findings that would indicate SIRs include:
- Temperature greater than 38°C (100.4°F) or less than 36°C (96.8°F)
 - Pulse greater than 90 beats per minute
 - Respiratory rate greater than 20 breaths/minute or PaCO₂ less than 32 torr
 - WBC greater than 12,000/mm³ or less than 4,000/mm³
- 18.6 Does the patient have a cough or a change in the character of his sputum that might indicate a pulmonary source of infection?
- Is the patient experiencing nausea, vomiting, or diarrhea? Does he have abdominal pain or rebound tenderness that might indicate an abdominal source of infection?
- Has the patient been experiencing dysuria, frequency of urination, or pain at the costovertebral angle that could be indicative of a renal source of infection?
- Does the patient have a new murmur or a change in a murmur, possibly indicating endocarditis?
- Does the patient have a headache or a change in mental status or neurological findings that might indicate a neurological focus of infection?
- Does the patient have redness, tenderness, or drainage at the sites of any implants, catheters, or medical devices?
- 18.7 The elements of the Severe Sepsis Bundle to be completed within 3 hours include:
- Measure serum lactate.
 - Obtain blood cultures prior to antibiotic administration.
 - Administer broad-spectrum antibiotic within 3 hours of ED admission.
 - In the event of hypotension or serum lactate greater than 4, administer 30 mL/kg crystalloid solution
- 18.8 The elements of the Severe Sepsis Bundle to be completed within 6 hours include:
- In the event of persistent hypotension despite fluid resuscitation deliver a vasopressor to maintain a MAP greater than 65.
 - Remeasure serum lactate if original lactate was elevated
- 18.9 Unless a fever is severely elevated, the patient is very uncomfortable, or the patient is at risk for compromise from the fever, there may be no attempt to actively reduce the fever. Critical care nurses need to observe their patients with fever to determine if the fever is resulting in cardiovascular compromise or excessive metabolic demands. Fevers are more likely to result in compromise in very old patients, neonates, neurological patients with increased intracranial pressure or seizures, and patients with cardiovascular disease.
- 18.10 A trigger activates the coagulation cascade. Thrombin production, which should be balanced by chemical mediators, is not and continues unrestrained. The excessive thrombin production activates fibrinogen, resulting in the formation of fibrin. Fibrin is deposited throughout the microcirculation, leading to obstruction of blood vessels and ischemic tissue damage. As the fibrin deposits are formed, platelets and coagulation factors are consumed, leading to a deficiency of clotting factors and the patient begins to bleed. The bleeding may be intensified as the clots are lysed, resulting in fibrin degradation products (FDPs) because the FDPs are anticoagulants. Thus, the patient develops simultaneous thrombotic and hemorrhagic complications.

Chapter 19

Why/Why Not?

Why?

The medication should be increased according to the parameters the nurse has available. The patient's RDOS score of 4 indicates that the patient is experiencing

sufficient breathlessness so that additional medication should be administered to promote patient comfort. Although some nurses fear that if they provide additional medication, the patient will die sooner, this has been demonstrated not to be true.

Why Not?

A nurse might wonder about administering additional medication because the patient's blood pressure has dropped and is low. Although the patient's BP has decreased, BP should not be used as a guide to determine when to give pain and dyspnea medication at end-of-life. If a low blood pressure is used as a parameter for administering medication, dying patients will experience unnecessary pain

Case Study

1. First, the nurse will need to establish trust with the son and the physician. Then, the nurse should attempt to gather the multidisciplinary team for a meeting. She should also ask social services to determine who is the legitimate spokesperson for Armand. In the meantime, she should be attentive to Armand, providing compassionate care and observing for any signs of returning consciousness.
 2. The nurse needs to have an understanding of what Armand might have wanted, who the appropriate spokesperson is for him now, and what his physiologic status is. How likely is he to benefit from the dialysis? What would be the burden of continuing care?
 3. The nurse might agree that Armand's stepson was using substituted judgment to decide. In this case, the nurse would be persuaded that Armand's stepson understood Armand and was speaking honestly and authentically on his behalf. The nurse might weigh the benefits and burdens of the treatment and concur with Armand's stepson that the benefits did not outweigh the burdens or with the surgeon that the potential benefit (Armand's continued life) was worth the burden of dialysis.
 4. The nurse can advocate at a multidisciplinary team meeting for what she believes Armand would have wanted or what was in his best interests. If she felt that the situation was not resolved after the team meeting, she could call for a meeting of the hospital ethics committee.
 5. This answer should reflect the learner's own evaluation.
- healthcare team. Most studies reveal few, if any, untoward effects.
- 19.2 When an advance directive is in place that clearly delineates what the patient would want done in the specific situation and the patient has not changed her mind, the directive presents a clear indication of what care should be provided at the end of life.
 - 19.3 The directive may be out of date, may not provide adequate guidance in the clinical situation, or the appointed healthcare proxy may not have a clear understanding of what the role entails or what the patient would have wanted to have done.
 - 19.4 Families of patients dying in critical care units have identified needs to be helpful to the dying patient and to be assured of the patient's comfort. They want to be informed about the patient's condition as well as what is being done for the patient. They want to be comforted and allowed to ventilate their emotions. They need reassurance that their decisions are correct. Nurses can assist families by being present for the patient and the family and acting to meet the identified needs of the family.
 - 19.5 If an ongoing medical treatment is not capable of benefiting the patient, most ethicists do not view withdrawing the treatment as the direct cause of the patient's death. The cause of death in such a circumstance would be the relentless progression of the patient's disease. Thus, when an intervention is neither enhancing the care of the patient nor promoting the patient's recovery, the intervention may be withdrawn.
 - 19.6 Terminal weaning involves the gradual reduction in settings on the ventilator: a change to synchronized intermittent mechanical ventilation with pressure support, a decrease in the respiratory rate, a reduction in the FiO_2 , and, finally, the discontinuation of the ventilator and provision of humidified room air by T piece. Terminal extubation involves removal of the endotracheal tube and discontinuation of the ventilator.
 - 19.7 Withdrawal of a medical intervention has been classified as a "passive" action, allowing death to occur from the disease process. However, assisted death is an active process in which the healthcare provider takes an action that directly results in the patient's death. It is illegal in all states and most countries.
 - 19.8 First, the nurse needs to establish trust among the patient, family, and other healthcare providers. Then she needs to develop a common understanding of the patient's prognosis and the benefits and burdens of treatment. A multidisciplinary team meeting can help this to occur. If conflict continues, the nurse can suggest an ethics committee consultation.

Review

- 19.1 Yes, families should be able to be present IF they desire to be present and there are resources available for their support during the resuscitation. The benefits to the family in most situations clearly outweigh the discomfort experienced by some members of the

Glossary

Absolute refractory period The brief period during depolarization of the cardiac cell membrane when the cardiac cells will not respond to further stimulation.

Accelerated rejection The recipient has been sensitized to some of the donor antigens.

Acute coronary syndrome Umbrella term used to describe conditions that cause chest pain due to inadequate blood flow to the myocardium.

Acute kidney injury (AKI) A broad clinical syndrome with various etiologies resulting in functional decline of the kidneys and an abrupt increase in serum creatinine.

Acute lung injury (ALI) Considered the pulmonary symptom of multiple organ dysfunction syndrome (MODS). The most severe form of ALI is **acute respiratory distress syndrome (ARDS)**.

Acute rejection A cell-mediated immune response that results in T lymphocytes infiltrating the donated organ and damaging it by secreting lysosomal enzymes and lymphokines.

Acute respiratory distress syndrome (ARDS) The most severe form of acute lung injury.

Acute respiratory failure (ARF) One of the most common disorders treated in critical care units, acute respiratory failure (ARF) occurs when the pulmonary system is unable to adequately exchange oxygen and remove carbon dioxide.

Acute tubular necrosis (ATN) Intrarenal failure, sometimes called ATN, is primarily caused by a prolonged reduction in renal perfusion (prerenal failure) but may also be precipitated by other conditions, including nephrotoxic agents.

Advance directives Allow competent patients to indicate in advance the kind of healthcare treatment they would desire should they become incapacitated at the end of their lives.

Adverse event Injury caused by medical management rather than the underlying condition of the patient.

Afterload The pressure (resistance) against which the right or left ventricle has to pump to eject the blood.

Alveoli Small balloon-type structures located at the end of the respiratory tree in which the exchanges of gases occur.

Anabolic hormone A hormone that synthesizes simple hormones into a more complicated hormone. Insulin is an anabolic hormone.

Anaphylactic shock An overwhelming allergic reaction to an antigen with a rapid onset, which may lead to death without appropriate interventions.

Angina pectoris An oppressive pain or pressure in the chest caused by inadequate oxygenation and blood flow to the heart muscle.

Anxiety A subjective feeling of distress and anguish.

Arterial blood gas (ABG) Procedure to measure the partial pressure of oxygen (O₂) and carbon dioxide (CO₂) gases and the pH (hydrogen ion concentration) in arterial blood.

Ascites The accumulation of a large amount of protein-rich fluid in the peritoneal cavity.

Assist control In this ventilator mode, a tidal volume and a minimum number of breaths/minute are set to be delivered by the ventilator. However, the patient is able to take additional breaths over the set rate as desired. With each breath, the ventilator delivers the set tidal volume.

Atrioventricular (AV) node A pacemaker of the heart that is part of the electrical conduction pathway between the atria and ventricles. If not stimulated from the SA node, the AV node can spontaneously generate electrical impulses at a rate of 40 to 60 beats per minute.

Autografting A procedure that involves removing thin slices of unburned skin from an unburned “donor” site on a patient and placing it on top of the excised burn wound. Autografts are the patient’s own skin and are intended to be permanent.

Automaticity The ability of certain cardiac cells to spontaneously initiate an electrical impulse.

Barotrauma Damage that results from excessive pressure, injuring alveoli and potentially resulting in a pneumothorax.

Beck’s triad Signs seen with a pericardial tamponade that include hypotension, distended neck veins, and muffled heart sounds.

Bilevel positive airway pressure (BiPAP) BiPAP has two levels of positive airway pressure, the higher one for inspiration (IPAP) and the lower for expiration (EPAP). Adding inspiratory pressure reduces the work of breathing and is more effective than expiratory pressure alone.

Brain attack Also known as a cerebrovascular accident (CVA) or stroke, it is a decrease in blood flow and oxygen to brain cells with the subsequent loss of neurological functioning.

Brain death The irreversible loss of function of the brain, including the brainstem.

Brain tumors Classified as primary or metastatic, most metastatic brain tumors arise from the lungs, breast, and skin. Primary brain tumors may be either benign or malignant.

B-type natriuretic peptide (BNP) A peptide released in response to increased ventricular filling pressures. Normal levels are 0 to 100 pg/mL.

Bundle of His The initial part of the ventricular conduction system that penetrates the AV valves, and then bifurcates into the right and left bundle branches to bring electrical stimulation to the right and left ventricles.

Cardiac cycle The sequence of events related to the flow of blood from the beginning of one heartbeat to the beginning of the next.

Cardiac index Cardiac output divided by the body surface area.

Cardiac output The volume of blood ejected by the left ventricle every minute, which is determined by heart rate and stroke volume.

Cardiac troponins There are two cardiac-specific troponin markers: Troponin T and Troponin I. Troponin I is the most accurate marker of myocardial injury. Troponin T measured 72 hours after an acute MI might be predictive of MI size, independent of reperfusion. Values greater than 0.01 ng/mL are elevated and values above 0.1 ng/mL are markedly elevated.

Cardiopulmonary resuscitation Provision of ventilation and compressions in an effort to sustain life.

Cardioversion Electrical shocks through the chest wall and heart that are synchronized with the QRS complex of the patient's cardiac rhythm.

Central venous pressure (CVP) Measures the right-sided preload via a catheter placed in a central vein such as the subclavian or internal jugular. Normal CVP is 2 to 6 mmHg (mean).

Cerebral vascular accident (CVA) Also known as a brain attack or stroke, a decrease in blood flow and oxygen to brain cells with the subsequent loss of neurological functioning.

Cerebrospinal fluid leakages Occur when a tear in the dura allows an opening to develop between the subarachnoid space and the outside.

Chemoreceptors Central chemoreceptors scrutinize the level of the hydrogen ion in the blood, while peripheral chemoreceptors monitor oxygen levels as well as carbon dioxide and hydrogen ion levels. The chemoreceptors respond to these changes by stimulating acceleration or slowing of the ventilation rate.

Chronic kidney disease (CKD) The progressive loss of renal function over months to years.

Chronic rejection A combination of humoral and cell-mediated immune responses that usually results in progressive deterioration in organ function.

Closed ICU An ICU in which patient care is provided by a dedicated ICU team that includes a critical care physician.

Colloids Solutions that have protein in them, either natural or synthetic. Blood is a colloid.

Compassion fatigue A state of tension and preoccupation with the suffering of those being helped that is traumatizing for the helper.

Compliance The expansibility of the thorax and lungs as measured by the increase in lung volumes in comparison to increases in intra-alveolar pressure.

Computed tomography angiography (CTA) Considered the "gold standard" for detecting cerebral aneurysms, arteriovenous malformations (AVMs), and arteriovenous fistulae (AVFs), it can also measure the exact degree of stenosis in extracranial and intracranial arteries and guide decisions regarding the use of recannulization therapy

Conductivity The ability of cardiac cells to transmit an electrical impulse to adjacent cardiac cells.

Continuous positive airway pressure (CPAP) CPAP delivers one continuous positive pressure throughout inspiration and expiration. Typical pressure settings are between 5 and 15 cm H₂O.

Continuous renal replacement therapy (CRRT) An umbrella term for several methods of dialysis that allow for

continuous fluid removal from the plasma (known as ultrafiltrate). The modalities differ in the principles, accesses, technology requirements, and nursing interventions.

Continuous venovenous hemodialysis (CVVHD) A mode of CRRT that is a continuous process of slow dialysis that works by diffusion. However, because it removes mostly small molecules, its usefulness is somewhat limited. It requires dialysate fluid, but does not require replacement fluid.

Continuous venovenous hemodiafiltration (CVVHDF) The most popular and efficient mode of CRRT. It is able to remove small and mid-sized particles and clear urea quickly. It uses both convection and diffusion simultaneously to remove fluids and solutes and utilizes venous accesses only.

Continuous venovenous hemofiltration (CVVH) A mode of CRRT uses convection with replacement fluid flowing through the filter at a fast rate (1,000 to 2,000 mL/hr), creating a "solute drag" that removes fluids and small to mid-sized molecules. This results in removal of significant amounts of fluid (7 to 30 L/24 hr) and adequate amounts of urea and other mid-sized molecules. It utilizes venous accesses only and requires a pump as well as replacement fluid.

Contractility The ability of cardiac cells to shorten in response to electrical stimulation.

Coronary artery disease (CAD) The development of fatty plaques in the coronary arteries. The growth of the atherosclerotic plaques narrows the vasculature, which then limits the blood flow and delivery of oxygen to the coronary muscle.

Coronary artery bypass grafting (CABG) A procedure in which the patient's diseased coronary arteries are bypassed with the patient's own venous (saphenous vein) or arterial (internal mammary artery or radial) vessels.

Cortical mapping Allows the surgeon to identify and avoid "eloquent" areas of the brain.

Creatine kinase (CK) An enzyme found in the brain, skeletal muscles, and heart that is released when the organ(s) are damaged. Might not be helpful in immediate diagnosis of an MI because of its lack of specificity and the 4 to 6 hours needed to see a significant rise. CK levels are more important in gauging the size and timing of an acute MI than the actual diagnosis.

Creatine phosphokinase-myocardial band (CK-MB) The version of CK used to specifically identify myocardial damage. Levels increase within 3 to 6 hours, peak at 24 hours, and return to normal within 36-72 hours after infarction. The CK-MB is considered positive when it is greater than 3% of the total CK.

C-reactive protein (CRP) Acute-phase reactant produced by the liver in response to inflammation.

Creatine kinase (CK) An enzyme of the transferase class in muscle, brain, and other tissues.

Critical care Direct delivery of medical care to a critically ill or injured patient.

Critical care nursing Specialty that deals specifically with human responses to life-threatening problems.

Critically ill patient A patient with an illness or injury that acutely impairs one or more vital organ systems such that the patient's survival is jeopardized.

Damage control resuscitation (DCR) Developed following military casualties, DCR has eight principles that have resulted in enhanced survival following traumatic injury.

Dead space unit The difference between anatomical dead space and physiologic dead space, representing the space in alveoli occupied by air that does not participate in oxygen-carbon dioxide exchange.

Defibrillation Unsynchronized electrical shocks administered through the chest wall and heart to depolarize myocardial cells in an attempt to restore sinus rhythm.

Delirium tremens (DTs) Also called alcohol withdrawal delirium, DTs are the most severe complication of withdrawal.

Depolarization The electrical state that exists in the cardiac cell membrane when the electrical charges are opposite the resting (polarized) state. The inside of the cell is more positively charged than the outside of the cell.

Diabetes insipidus Develops in 40% to 70% of people who receive kidneys from deceased donors and is caused by insufficient levels of antidiuretic hormone (ADH) resulting in the production of large volumes of dilute urine.

Diaphragm The primary muscle of ventilation.

Dicrotic notch A point on the arterial pressure waveform and the pulmonary artery pressure waveform that signals the end of systole.

Disseminated intravascular coagulation or coagulopathy (DIC) Systemic intravascular activation of the coagulation cascade with fibrin formation and deposition in the microvasculature, resulting in simultaneous thrombotic and hemorrhagic complications.

Diuretic phase of AKI In this phase of AKI, patients increase their urinary output, often producing up to five liters of urine each day. However, despite the increased urine output the patients' kidneys often do not excrete wastes or regulate electrolyte and acid-base balance.

Diverticular disease Results from weak points on the intestinal wall that herniate to form saclike projections called diverticula.

Do Not Attempt to Resuscitate (DNAR) The decision not to provide cardiopulmonary resuscitation.

Dysphagia Difficulty swallowing.

Echocardiogram A diagnostic, noninvasive visual representation that is produced through ultrasound waves of the great arteries and the heart's structure and movement.

Electrocardiogram (12 lead ECG) A graphic picture showing the electrical activity of the heart. Provides information related to heart rate, conduction disturbances, electrical impulse disturbances, the presence of myocardial ischemia, and electrical effects of medications and electrolyte imbalances.

Electrophysiology The study and intervention of cardiac dysrhythmias with the goal of restoring normal cardiac rhythm.

Embolic stroke Particles arise from another part of the body and flow through the bloodstream, resulting in blockage of arterial blood flow to a particular area of the brain.

End stage renal failure (ESRD) More recently known as chronic kidney disease, end stage renal failure (ESRD) is the progressive loss of kidney function over months to years.

Endoscopic retrograde cholangiopancreatography (ERCP) Direct visualization of the common bile duct utilizing an endoscope and a series of x-rays.

Endoscopy A procedure that uses a flexible fiber-optic endoscope to directly visualize the inside of a hollow organ or cavity.

Endotracheal tubes Tubes that are placed into the trachea via the nose or mouth in order to establish a patent and stable airway for patients.

Enteral nutrition Delivery of part or all of a person's caloric requirements through the gastrointestinal tract. Methods may include a normal oral diet, the use of liquid supplements, or delivery of part or all of the daily requirements by use of a tube (tube feeding) to the stomach or small intestine.

Epidural hematomas Usually occur in conjunction with a skull fracture and result from a laceration of the middle meningeal artery, causing bleeding between the dura mater and the skull.

Epiglottitis Provides airway protection during ingestion of food and fluids by closing off the tracheal opening during swallowing.

Esophagogastroduodenoscopy Upper GI endoscopy (EGD) that involves oral intubation with a flexible endoscope to visualize the esophagus, stomach, and proximal duodenum.

Ethical dilemma A situation that gives rise to conflicting moral claims, resulting in disagreements about choices for action.

Excitability A characteristic shared by all cardiac cells that refers to the ability to respond to an electrical impulse generated by pacemaker cells or other external stimuli.

Expiratory pressure (EPAP) EPAP is the lower pressure provided during expiration when a patient is receiving BiPAP.

Expiratory reserve volume (ERV) The maximal volume of air, usually about 1,000 milliliters, that can be expelled from the lungs after normal expiration.

Extubation The removal of a patient's endotracheal tube.

Fractional excretion of sodium (FENa) A test for assessing how well the kidneys can concentrate urine and conserve sodium, which is usually less than 1%.

Fraction of inspired oxygen (FiO₂) The percentage of oxygen in the mix of air delivered to a patient.

Functional residual capacity (FRC) The volume remaining in the lungs after a normal exhalation.

Glasgow Coma Scale Scale used to determine a patient's level of consciousness; assesses both level of consciousness and motor response to a stimulus. Scores range from 3 to 15 based on the patient's ability to open the eyes, respond verbally, and move normally. Repeated assessments aid in determining if the patient's brain function is improving or deteriorating.

Glomerulofiltration rate (GFR) The rate at which filtrate is formed in the glomerulus of the kidney—approximately 125 mL/min in a healthy adult.

Glycogenesis The conversion of excess glucose to glycogen.

Glycogenolysis The formation of glucose from amino acids and fats when there is limited carbohydrate intake.

Glycosuria Presence of glucose in the urine.

Golden Hour A term used in trauma care referring to the first hour following the initial traumatic injury. Patient survival rates increase with the proper care during the golden hour.

Heart failure (HF) The inability of the heart to pump sufficient blood to meet the demands of the body. The most common type of HF is LV systolic dysfunction.

Heart failure with preserved ejection fraction (HF-pEF) Heart failure with a LVEF greater than 50%.

Heart failure with reduced ejection fraction (HFrEF) Heart failure with a LVEF less than 40%.

Helicobacter pylori A highly mobile bacterium that avoids acid by burrowing underneath the mucosa. It causes the mucosa to be more susceptible to peptic acid.

Hematemesis Vomiting of blood that is either bright red or has a coffee grounds appearance, indicating UGI bleeding.

Hematochezia The passage of bright red blood from the rectum. It might or might not be mixed with stool. Hematochezia suggests a LGI bleed.

Hemodynamics The study of forces that aid in circulating the blood throughout the body. These forces can be monitored by blood pressure, pulse rate, mental status, urinary output, and cardiac output.

Hemorrhage Blood loss with organ compromise; threatens tissue perfusion or life.

Hepatic encephalopathy (HE) Also known as portosystemic encephalopathy (PSE), HE is a condition characterized by a wide range of potentially reversible neuropsychiatric manifestations that occurs in patients with advanced liver disease and portal hypertension.

High-flow oxygen therapy High-flow oxygen therapy is designed to deliver oxygen at a higher flow rate than the patient's inspiratory flow rate. High-flow delivery devices may also have some positive pressure effect and can create an oxygen reservoir in the physiologic dead space of the upper airways.

High-pressure limit A ventilator will not exceed this pre-set pressure when delivering volume.

Hollow organs Organs that have a cavity or space inside (e.g., bladder or stomach).

Hydrocephalus Blood in the subarachnoid space obliterates the arachnoidal villi, preventing absorption of cerebrospinal fluid (CSF), or blood within the ventricles blocks the foramen of Monroe, preventing drainage of CSF. It may lead to an increase in intracranial pressure and deterioration of the patient's neurological status.

Hydrolysis Splitting of bonded fats and the addition of water to produce fatty acids and glycerol.

Hypercapnea An elevated PaCO_2 .

Hypovolemic shock A shock state resulting from a loss of circulating volume.

Hypoxemia A decreased PaO_2 .

Implantable cardioverter-defibrillator (ICD) A device similar in size and the way it is implanted in a patient to a pacemaker. It monitors the patient's heart rate and rhythm and is multiprogrammable to deliver pacing, cardioversion, and/or defibrillation according to the patient's needs.

Infection A pathologic process induced by a microorganism.

Informed consent Has three components. The decision must be made voluntarily by a competent adult who understands his or her condition and the possible treatments.

Inotropic Positive inotropes increase the contractility of the heart. Negative inotropes decrease the contractility.

Inspiratory:expiratory ratio The inspiratory:expiratory ratio compares the time spent in inspiration to that in expiration. Normally, a person spends twice as much time in exhalation as in inspiration, so the ratio would be 1:2.

Inspiratory pressure (IPAP) The higher inspiratory pressure that is set for BiPAP.

Inspiratory reserve volume (IRV) The volume of air one is able to inhale in addition to the V_T .

Intermittent hemodialysis (IHD) Utilizes both ultrafiltration and diffusion to rapidly correct electrolyte imbalances, restore fluid balance, adjust acid-base balance, and remove wastes.

Intracerebral hemorrhage (ICH) Derived from bleeding of small arteries or arterioles directly into the brain, forming a localized hematoma that spreads along white matter tracts.

Intraosseous infusion Process of administering medications or other fluids into a catheter placed directly into the bone marrow.

Intrarenal acute renal failure Commonly results from prolonged hypoperfusion of the kidney. The renal tubules respond to hypoxia with dysfunction, inflammation, and, possibly, necrosis.

Intubation The insertion of an endotracheal tube.

Inverse ratio ventilation In disorders in which the lungs are "stiff" or noncompliant, the I:E ratio may be reversed in order to allow a slower delivery of the tidal volume.

Involuntary euthanasia An action to end the patient's life is taken without the patient's consent.

Ischemia A temporary deficiency of blood flow to an organ or tissue.

Ketoacidosis Occurs as a result of buildup of fatty acids. Metabolic acidosis happens when the blood becomes more acidic than body tissues.

Ketonuria Presence of ketones in the urine.

Korsakoff's syndrome Selective memory disturbances and amnesia.

Kussmaul's respirations Deep and rapid respirations that are the body's attempt to decrease carbon dioxide in order to compensate for metabolic acidosis.

Lactate clearance The trending of lactate values measured during resuscitation. Rapid improvement is associated with better patient outcomes, while failure to improve is associated with adverse clinical outcomes.

Left sided heart failure (HF) The pumping action or ability of the left ventricle (LV) to fill is impaired allowing blood to back up from the LV to the left atrium and into the pulmonary veins.

Left ventricular (LV) failure The pumping action or ability of the LV to fill is impaired, allowing blood to back up from the LV to the left atrium and into the pulmonary veins. Fluid eventually builds up in the lungs and pleural spaces causing pulmonary symptoms.

Lethal triad Refers to the spiraling effects of acute coagulopathy, acidosis, and hypothermia observed in exsanguinating patients.

Left ventricular ejection fraction (LVEF) Refers to the percentage of blood ejected from the left ventricle with each contraction—55% to 75% is normal.

Maximum inspiratory pressure is used to evaluate inspiratory respiratory muscle strength and readiness for weaning from mechanical ventilation.

Mean arterial pressure The average pressure over one cardiac cycle.

Mechanical ventilation Assists the breathing process in clients who cannot effectively ventilate independently.

Melena The passage of black tarry colored stool. It has a very characteristic foul odor.

Meningitis Inflammation of the meninges and the underlying subarachnoid space that contains the cerebrospinal fluid. Depending on the development and duration of symptoms, meningitis may be classified as acute, subacute, or chronic.

Mild traumatic brain injury Head injury with patient having no or very limited loss of consciousness and GCS 13 or higher.

Minute Volume Determined by totaling the tidal volumes over 1 minute, minute volume is indicative of readiness to wean.

Moderate head injury (GCS 9–12) may be associated with a loss of consciousness for up to a day.

Monoclonal antibodies Antibodies produced by a single immune cell to target a specific antigen.

Monro-Kellie hypothesis Intracranial Volume (VIC) = Volume brain + Volume blood + Volume CSF + Volume lesion.

Moral distress Occurs when a nurse knows the right thing to do, yet institutional constraints prevent her from doing it.

Multiple organ dysfunction syndrome (MODS) The presence of altered organ function in an acutely ill patient such that homeostasis cannot be maintained without intervention.

Muscle oxygen saturation (SmO₂) The amount of oxygen in venous blood after tissue oxygen extraction has occurred. It is measured through a near-infrared spectroscopy sensor placed on the patient's shoulder, thigh, or calf.

Myoglobin The iron-containing protein found in muscle cells that store oxygen for cells.

Nasopharyngeal airway A flexible, curved piece of rubber or plastic, with one wide, trumpetlike end and one narrow end that can be inserted through the nose into the pharynx.

Nasotracheal tube A tube inserted into the trachea through the nose and pharynx, which is used to deliver oxygen.

Necrosis The death of cells, tissues, or organs.

Negligence Composed of several specific elements, including a duty was owed, the duty was breached, the breach of the duty was the proximate cause of an injury to the patient, and damages resulted.

Neurogenic Shock Occurs when a spinal cord injury causes a temporary interruption in the sympathetic innervation leaving the parasympathetic innervation unopposed. This results in vasodilation with pronounced hypotension and bradycardia.

Noncardiac chest pain (NCCP) The term that is used to describe pain in the chest that is not caused by heart disease or a heart attack.

Non-heartbeating donor A donor who has sustained a devastating nonrecoverable neurological injury that did not result in brain death. The patient's proxy has determined in conjunction with the healthcare team that the patient would wish to have life-sustaining therapy withdrawn so that she could die and would want to donate her organs after death.

Noninvasive mechanical ventilation (NIV) NIV supports the ventilatory efforts of patients without the insertion of invasive airway devices.

Non-rebreather masks Non-rebreather masks have valves over the ports that allow exhaled air to escape but prevent room air from being inhaled. This mask is capable of delivering up to 100% O₂.

Nonoliguric phase of acute kidney injury (AKI) Patient produces more than 400 mL of urine/day, usually about 1,000 mL/day, but does not excrete metabolic wastes and regulate electrolytes or acid-base balance.

NSTEMI Non-ST-segment elevation myocardial infarction, which occurs as a result of a subtotal occluding thrombus. This type of infarction is associated with ST-segment depression or T wave inversion on the ECG.

Obstructive shock Circulatory failure caused by physical obstruction, for example, cardiac tamponade or pulmonary embolism.

Oliguric phase of acute kidney injury The patient excretes less than 400 mL of urine/day.

Onset phase of acute kidney injury Immediately follows the period of renal injury, the patient's urinary output typically decreases to 20% of normal and the BUN and creatinine may begin to increase.

Oropharyngeal airway Pharyngeal airways are designed to prevent the tongue from occluding the upper airway. To insert an oropharyngeal airway, the nurse displaces the patient's tongue downward using a tongue depressor and slides the airway over the patient's tongue into the oropharynx. Oropharyngeal airways are contraindicated in patients who are alert and have intact gag reflexes.

Oxyhemoglobin dissociation curve Oxygen is carried in the blood in two ways. About 97% is bound to hemoglobin, and about 3% is dissolved in serum. The oxyhemoglobin dissociation curve describes the balance of these two transport methods. A shift means that there is a change in the way oxygen is taken up by the hemoglobin molecule as well as a change in the way it is delivered to the tissues.

Oxygen content The total amount of oxygen in the blood, including oxygen dissolved, hemoglobin level, and oxygen saturation.

Oxyhemoglobin dissociation curve The relationship between the two transport methods, the oxygen bound to hemoglobin and the oxygen dissolved in the serum.

P/F ratio The relationship between the PaO_2 and the FiO_2 ($\text{PaO}_2/\text{FiO}_2$) with normal values in healthy adults ranging from 400 to 500 mmHg.

Pain “Whatever the person says it is, existing whenever he says it does.”

Pancreatitis A sudden inflammatory process of the pancreas.

Paracentesis The removal of fluid from the peritoneal space using a large-bore needle or catheter system.

Parenteral nutrition The infusion of nutrients using a venous catheter located in a large, usually central, vein.

Peak inspiratory pressure (PIP) The highest pressure during inspiration, whereas plateau pressure stays relatively constant through inspiration.

PEEP, positive end expiratory pressure PEEP is positive pressure that remains in the lungs at the end of expiration.

Peptic ulcer disease Ulcer located in the stomach or the first part of the duodenum.

Peritoneal dialysis (PD) Uses osmosis and diffusion to produce a steady, gradual restoration of fluid, electrolyte, and acid-base balance while removing nitrogenous and other wastes.

Pharyngeal airways Airways designed to prevent the tongue from occluding the upper airway.

Phlebostatic axis The reference point for the left atrium when leveling a pressure monitoring system. It is located at the fourth intercostal space halfway between the anterior-posterior diameter of the chest.

Plateau pressure Pressure measured during an inspiratory pause on the ventilator. The goal is less than 30 cm. H_2O to prevent volutrauma.

Polarization The electrical state that exists at the cardiac cell membrane when the cell is at rest. The inside of the cell is more negatively charged than the outside of the cell.

Polyclonal antibodies Antibodies produced by multiple immune cells to target a specific antigen. Most commonly, a polyclonal agent called rabbit antithymocyte globulin (ATG) is used.

Postrenal acute kidney injury Characterized by the sudden onset of anuria (production of less than 50 mL of urine/day) due to obstruction of the urinary tract.

Preload The stretch on the ventricular myocardium at end diastole.

Prerenal acute kidney injury The nephrons and glomeruli are structurally and functionally normal and the glomerular filtration rate decreases due to reduction in renal blood flow.

Pressure control ventilation This ventilator mode provides a set pressure instead of a set tidal volume. On inspiration, the ventilator delivers gas until a pressure limit is reached. An adequate tidal volume is not ensured and must be monitored frequently.

Pressure Support Positive pressure provided during inhalation intended to decrease the patient's work of breathing.

Primary survey Assessment of airway, breathing, circulation, and disability or neurological status.

Pulmonary artery wedge pressure (PCWP) The PCWP waveform is obtained when the balloon is inflated at the end of the PA catheter. This pressure reflects the left atrial pressure and indirectly the left ventricular end diastolic pressure. Normal PCWP is 8 to 12 mmHg (mean).

Pulse pressure The difference between the systolic and diastolic blood pressure.

Purkinje fibers The terminal filaments of the electrical conduction system of the ventricles, which can generate electrical impulses at a rate of 20 to 40 beats per minute.

Radiofrequency catheter ablation (RFCA) A catheter delivers high-frequency electrical current to obliterate the site of origin of a dysrhythmia in the heart. RFCA has successfully treated SVT, atrial flutter, and AF.

Rapid Sequence Intubation (RSI) An airway management technique that uses an induction agent to create immediate unresponsiveness and a neuromuscular blocking agent to produce muscular relaxation. It is the fastest and most effective means of controlling the emergency airway.

Rapid shallow breathing index (RSBI) A value utilized to determine readiness for weaning from ventilation. It is determined by dividing the respiratory rate by tidal volume in liters.

Recovery phase of acute kidney injury Renal function returns to normal.

Rejection The constellation of host immune responses evoked when an allograft tissue (organ, graft, tissue) is transplanted into a recipient.

Relative refractory period A brief period following the “absolute refractory period” of depolarization when some of the cardiac cells have repolarized. If they receive a stronger than normal stimulus, they may depolarize. It is also known as the “vulnerable period.”

Remodeling Structural changes to the myocardium leading to chamber dilation and hypertrophy.

Renal replacement therapy (RRT) There are three forms of renal replacement therapy: intermittent hemodialysis, continuous renal replacement therapy (CRRT), and peritoneal dialysis.

Repolarization The restoration of the polarized (resting) state at the cardiac cell membrane. The inside of the cell is more negatively charged than the outside of the cell.

Required request The regional organ donor bank must be notified when a patient is imminently dying so the organ bank can determine if the patient qualifies as an organ donor. If the patient does qualify but has not signed a donor card, the family must be asked if they are willing to donate the patient's organs.

Residual volume (RV) The volume of air that remains in the lungs following forced expiration beyond normal expiration, or the expiratory reserve volume (ERV).

Respiration The transport of oxygen and carbon dioxide between the alveoli and the pulmonary capillaries.

Revised trauma score Physiologic scoring system that predicts the likelihood of death following a traumatic injury.

Right atrial pressure (RAP) The RA pressure measures the right-sided preload through the proximal port of the PA catheter. Normal RA is 2 to 6 mmHg (mean).

Right ventricular failure In RV failure, the blood volume backs up from the right ventricle to the right atrium and out to the systemic circulation causing peripheral and organ edema. Right ventricular HF is often a consequence of left ventricular HF, but can also be a primary cause of HF.

Rule of nines The body is divided into five areas, each with increments of 9% TBSA. These areas are the head and neck (9%), arms (9% each), anterior and posterior trunk (36%), anterior and posterior legs (18% each), and the perineum (1%).

SBAR A technique to guide nurses' communication to physicians in critical situations.

Secondary survey Head-to-toe assessment done to identify injuries.

Seizures Stereotyped behavior associated with electrographic abnormalities in the EEG.

Septic shock The presence of sepsis and refractory hypotension (systolic BP less than 90 mmHg, mean arterial pressure less than 65 mmHg, or a decrease of 40 mmHg in systolic BP) that is unresponsive to a crystalloid fluid challenge of 20 to 40 mL/kg.

Severe head injury (GCS less than or equal to 8); usually associated with loss of consciousness for more than 24 hours.

Shock Syndrome marked by inadequate perfusion and oxygenation of cells, tissue, and organs, usually as a result of markedly lowered blood pressure.

Shunt unit When an alveolus is inadequately ventilated in the presence of perfusion.

Silent unit When both ventilation and perfusion are impaired.

Sinoatrial (SA) node The dominant pacemaker of the heart, located in the upper posterior wall of the right atrium, that spontaneously generates electrical impulses at a rate of 60 to 100 beats per minute.

Slow continuous ultrafiltration (SCUF) A method of CRRT that uses ultrafiltration alone to remove 100 to 300 mL of fluid hourly from the patient. It has little ability to remove urea and other mid-sized molecules, so its usefulness for patients in AKI is limited.

Spinal cord Part of central nervous system and extends from the medulla to the second lumbar vertebra within the spinal canal. It is protected by bone and enclosed in the meninges.

Spinal cord injury (SCI) When the vertebral column is deformed, there may be injury or secondary edema of the spinal cord, resulting in the loss of neurological function.

Spontaneous bacterial peritonitis (SBP) An infection of the ascitic fluid without indication of another intra-abdominal source such as a perforated viscus.

Square wave test A test performed on the pressure monitoring system to verify the ability of the pressure monitoring system to accurately reflect pressure values.

Stable angina Angina that occurs with exercise, is predictable, and is usually promptly relieved by rest or nitroglycerin.

STEMI ST-segment elevation myocardial infarction. It occurs as a result of a total occlusion of a coronary artery. This type of infarction is associated with ST elevations.

Stroke Also known as a brain attack or cerebrovascular accident, it is a decrease in blood flow and oxygen to brain cells with the subsequent loss of neurological functioning.

Stroke volume (SV) The volume of blood ejected from each ventricle with each heartbeat.

Subarachnoid hemorrhage Bleeding between the arachnoid and pia mater may result from rupture of a preexisting or a traumatic cerebral aneurysm.

Subdural hematomas Usually are the result of countercoup injuries, occurring on the opposite side of the skull from the injury just below the dura. They are usually the result of venous bleeding, often originating with the stretching of bridging veins. Subdural hematomas are classified as acute, subacute, and chronic.

Supernormal period The period during depolarization of the cardiac cell membrane that follows the relative refractory period. During this period a weaker than normal stimulus can cause depolarization of cardiac cells.

Supraventricular tachycardia (SVT) A general term to describe tachydysrhythmias that originate above the ventricles, of atrial, junctional, or of uncertain origin but with a rapid ventricular rate and narrow QRS complexes.

Surfactant Phospholipids secreted by the great alveolar cells into alveoli and respiratory air passages that contribute to the elastic property of pulmonary tissue by lowering surface tension and preventing collapse at end-expiration.

Sympathetic nervous system (SNS) One of the two main divisions of the autonomic nervous system. Activation of the sympathetic nervous system releases norepinephrine, causing increases in vasoconstriction, heart rate, and contractility. Activation is associated with changes in all body systems and is described in lay terms as the "flight or fight" response.

Synchronized cardioversion A shock that is synchronized with the QRS complex. The defibrillator must be placed in the synchronized mode so that it will recognize the QRS complexes and shock on an R wave, thus avoiding discharging during the vulnerable period of the T wave, which could cause VF.

Synchronized intermittent mandatory ventilation (SIMV)

In this ventilatory mode, the patient receives a minimum number of breaths with a set tidal volume, but may take additional breaths as determined by the patient. In these additional breaths, the patient determines how much volume to inspire and is able to use their respiratory muscles. The mandatory breaths are spaced evenly throughout each minute, timed or synchronized with the patient's inspiratory effort. This feature explains the intermittent in the name of the mode.

Systemic inflammatory response syndrome (SIRS) The presence of two or more of the following: temperature greater than 38°C (100.4°F) or less than 36°C (96.8°F); pulse greater than 90 beats per minute; respiratory rate greater than 20 breaths/minute or PaCO₂ less than 32 torr; WBC greater than 12,000/mm³ or less than 4,000/mm³.

Systemic vascular resistance (SVR) The resistance to the flow of blood in the blood vessels throughout the body. The SVR increases when vessels constrict (as in hypovolemia) and decreases when the vessels dilate (as in sepsis). A change in the diameter, elasticity, or number of available vessels influence the resistance to blood flow.

Thrombotic stroke A thrombotic stroke occurs when thrombus formation in an artery causes a stroke due to decreased blood flow.

Tidal volume (V_T) The volume of one inhalation/exhalation cycle.

Tissue typing Used to determine how much overlap exists between a potential donor's and the recipient's HLA antigens.

Total lung capacity (TLC) The maximum volume the lungs can hold.

Trachea The major tube connecting the upper and lower airways.

Tracheostomy Involves an incision in the neck accessing the trachea and creation of a stoma through which a tracheostomy tube is inserted.

Type I respiratory failure (hypoxemic) Stems from a disruption of O_2 transport from the alveolus to arterial flow. Respiration is impeded.

Type II respiratory failure (hypoxemic hypercapneic) Originates in musculoskeletal or anatomical lung dysfunction or suppression. Ventilation is not sufficient.

Unstable angina Angina that has changed to a more severe form. It can occur at rest and may be an indication of impending myocardial infarction.

Variant angina Chest pain that results from the spasm of coronary arteries rather than from exertion or other increased demands on the heart.

Vasospasm An angiographic narrowing of cerebral blood vessel(s) that can lead to delayed ischemia.

Venous thromboembolism (VTE) Deep vein thrombosis with the development of pulmonary embolism.

Ventilator-induced lung injury (VILI) Injury to the lung resulting from excessive pressure (barotrauma) or excessive volume (volutrauma).

Ventilation The mechanical act of moving air in and out of the respiratory tree.

Ventilator-associated pneumonia (VAP) A new lung infection developing within 48 hours of intubation.

Ventricular remodeling The reshaping of the walls of the myocardium after a myocardial infarction.

Venturi mask This mask provides precise FiO_2 levels when used properly. It has an adjustable dial imprinted with the desired FiO_2 and LPM flow. A port or blender is attached around the connection of the O_2 tubing and the adapter. The port must remain in place and uncovered in order to ensure accurate O_2 delivery.

Vital capacity (VC) The sum of the ERV, V_T , and IRV.

Voluntary euthanasia A person requests an action be taken to end his life.

Volutrauma Over-distention of the alveoli that results in inflammation and can reduce the patient's ability to recover respiratory function.

Weaning The process of determining the patient's readiness to resume spontaneous breathing and concluding mechanical support.

Wernicke's encephalopathy Confusion, abnormal gait, and paralysis of certain eye muscles.

Work of breathing (WOB) The effort expended in the act of inspiration.

Zone of coagulation Third-degree burns have all three zones, beginning with the inner zone of coagulation, which is the deepest part of the burn. The cells are nonviable and the microcirculation is destroyed, leaving the skin dark colored and leathery.

Zone of hyperemia The outer zone of hyperemia is tissue with intact microvasculature that heals spontaneously within days. The area initially appears pink and capillary refill may be increased due to vasodilation induced by local inflammatory mediators.

Zone of stasis The medial zone of stasis is composed of viable and nonviable cells. There is damage to the microcirculation, resulting in vasoconstriction and ischemia. The skin initially appears moist or blistered and is red in color.

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