PICU Protocol of EHA

First Edition 2024

Egyptian Clinical Practice Protocols

in

Pediatric Intensive Care Units

for

Egypt Healthcare Authority

First Edition

2024

Prepared by

Working Group for Development of

Egyptian Clinical Practice Protocols

in

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Egypt Healthcare Authority

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Disclaimer

Protocols and guidelines outline the recommended or suggested clinical practice; however, they cannot replace sound clinical judgment by appropriately trained and licensed physicians.

The physician is ultimately responsible for management of individual patients under their care.

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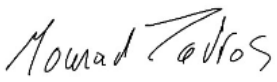

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PREFACE

Recently, there is an increasing need to provide programs with accurate competency-based assessments to ensure the delivery of high-quality healthcare. The aim of developing these Egyptian Clinical Practice Protocols in Pediatric Intensive Care Units is to unify and standardize the delivery of healthcare to any child at all health facilities.

The current status of healthcare in which avoidable failures are abound. “We train longer, specialize more, use ever-advancing technologies, and still we fail.” Part of the problem, is that the ever-increasing complexity of medicine makes uniform care delivery impractical or impossible. That is, unless there are protocols, checklists, or care paths that are readily available to providers.

Regarding Pediatric Intensive Care Units, busy clinicians have all felt the need for a concise, easy-to-use resource at the bedside for evidence-based protocols, or consensus-driven care paths.

In this protocol, we offer comprehensive reviews of selected topics and comprehensive advice about management approaches based on the highest level of evidence available in each case. Our goal is to provide an authoritative practical medical resource for pediatricians.

We hope that such an approach will encourage clinicians to apply available evidence to their practice and also track compliance with desired practices. We hope that practicing pediatricians, fellows and practitioners will find this protocol useful in delivering high-quality clinical care to their patients. We remain open to feedback and suggestions about how to improve this resource and how to make it maximally useful to those delivering care at the bedside in for patients in Pediatric Intensive Care Units.

Members of the Working Group

For Development of the Egyptian Clinical Practice Guideline

In Pediatric Intensive Care Units

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Standards for PICU Preparation

Human resources needed for every five beds:

- ✓ **Doctors**; one consultant, one specialist and four residents
- ✓ **Clinical pharmacy**; one
- ✓ **Nurses**; one head nurse, three nurses and three assistant nurses
- ✓ **Two porters**
- ✓ **One infection control specialist**

PICU structural composition:

1-Take in consideration the proximity to OR
2-also to be near to ER
3-8 meters are the area needed for each ICU bed
4-PICU should contain: procedure room, operating room and preparatory room
5-AC system with hepa filter
6-Entrance different from exit
7-Central sterilization unit

Laboratory requirements for any PICU

- Emergency lab 24 hours 7days or lab to lab deal
- CBC, blood gases, electrolytes coagulation profile, LFT, KFT

Pharmacy:

- Emergency stock of medication for each unit
- Internal pharmacy 24 hours /7 days
- Each governorate should contain TPN center preferred to be in largest hospital
- Blood bank for each governorate

Security cameras for PICU beds

Doctors& nurse rooms with toilet and for each five beds one toilet

Store room

For each five beds one sterilization basin

Equipment:

1-Central station

2- Portable x ray and U/S with duplex probe

3-For each PICU bed;

Monitor +4 syringe +2 infusion pump+ 1 warmer+ 2 oxygen outlets +1 air outlet +1 suction outlet +12 electric outlets on UPS with stabilizer

4-For every five beds we need 5 MV +2 high frequency MV +2 noninvasive ventilation +1 crash car with D/C unit +ECG + augmented EEG

5- Air mattress

Transportation:

- Separate portable ventilator or one of the unit ventilators if it also could be used as portable ventilator
- Four oxygen cylinders
- Two portable monitors
- Two incubators

NB:

- **Two CRRT machines for each governorate in same place or if there is a dialysis unit it should be in it**

Pediatric Resuscitation

Scope:

- Applicable to individuals up to 12 years of age
- Resuscitation of neonates in the delivery room is NOT within the scope of this protocol
- This protocol applies to healthcare settings. Basic life support by lay by-standers is not the intended scope

Recognition:

- CPR is indicated when there is no reliable central pulse with a rate of at least 60/min. in a patient who is unconscious and unresponsive
- Central pulse may be assessed in the Carotid, Brachial or Femoral arteries
- Duration of assessment may NOT exceed 10 seconds
- Hospitalized critical patients will be already on continuous monitoring. Note that those with pulseless electrical activity will show cardiac electrical activity (ECG pattern) on the monitor in absence of a central pulse. CPR is still necessary
- Hemodynamically stable patients with HR less than 60/min may require alternative approaches
- Rescue breathing is indicated
  (a) Together with chest compressions during CPR
  (b) In patients with absent or grossly ineffective (eg gasping) breathing even if there is a reliable pulse Airway opening is necessary for rescue breathing
- Immediate defibrillation is needed for those with recognized pulseless shockable rhythm (VF, VT) once it is available.
- CPR should be performed until a shockable rhythm is recognized and a defibrillator is ready
  - CPR is not indicated when there are obvious signs of life such as a reliable central pulse ≥60/min., regular breathing, eye opening, speech, cough, etc.
  - However, this does not rule out a critical or unstable condition. System support may be required. Recognition and management of system failures before cardiac arrest occurs and after return of spontaneous circulation are keys to survival
- CPR is a team process; Pre-assignment of teams and appropriate training improve outcomes. While CPR can and should be initiated by a single individual, calling for help is then necessary to continue effective management. Required equipment, medications and supplies must be readily available and checked regularly and after each use

Sequence of Actions:

1. Recognize the case, ensure safety, call for help and position the patient

- It is important to ensure the patient is not in ongoing danger and it is important that the rescuer approach safely so he would not be endangered. Approach with care
- Inside the hospital, scene safety should be easy to maintain
- A sole rescuer may initiate a cycle of CPR first if calling for help requires him to leave the patient
- A defibrillator/ monitor should be available or brought with coming help
- Patient should be positioned supine on a flat hard surface. A bed mattress is too soft; a board may be used under the patient, but make sure it can take the force of chest compressions and its edge is away from the patient's back
- If the patient needs to be moved or turned, do that with care to avoid injury

2. If there is no central pulse ≥60/min., start chest compressions

3. Open airway, check breathing and start rescue breaths

- If there is no pulse, starting chest compressions immediately, followed by airway-breathing support is recommended (C-AB). If there is a pulse, the usual ABC approach; starting with airway & breathing, is recommended. Conscious patients with a FB airway obstruction will require specific management

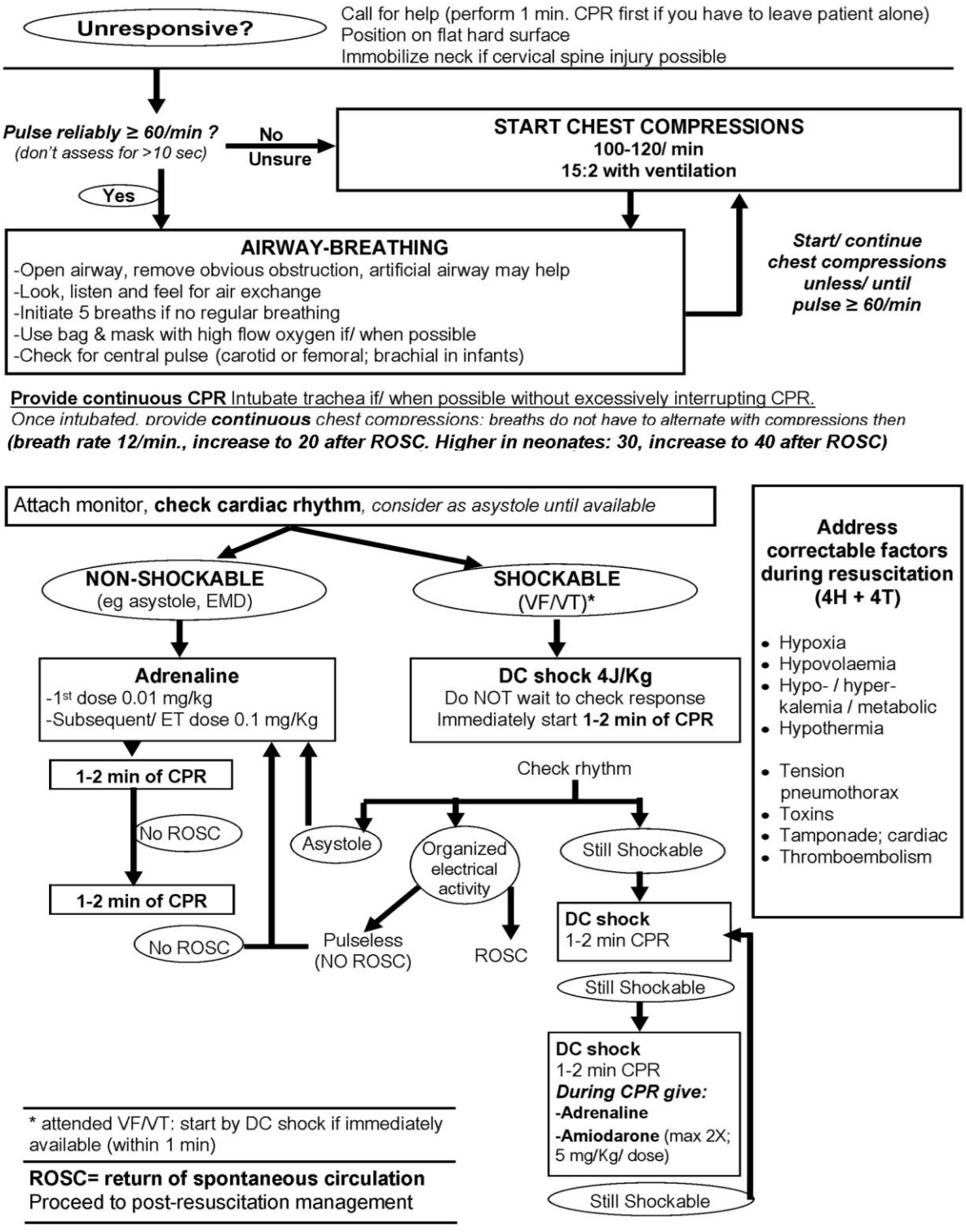
4. Identify a shockable rhythm (VF/VT) & give DC shock

5. Immediate vascular access for drug ± fluid administration

- Shockable rhythms are not the commonest causes of pediatric cardiac arrest; however, early recognition & treatment could markedly improve outcome. Do NOT unnecessarily interrupt CPR but identify rhythm as soon as available. When cardiac arrest is associated with an attended VF/VT (in a monitored patient), an immediate DC shock is given if available

6. Check and correct correctable factors

- This should occur throughout resuscitation



- **EMD (electromechanical dissociation) = pulseless electrical activity**
- **The first DC shock may be 2-4J/Kg**

Chest Compressions

Site: midline on the lower sternum

- NOT the xiphisternum, NOT on the left precordium

Rate: 100-120/min

Depth/ force: need to depress 1/3 the AP chest diameter

- Two hands for older children
- One hand for younger children
- Only use the heel of the hand(s), fingers should NOT be applied to the chest
- Two fingers for infants
- Both thumbs with hands encircling the chest
- Index and middle fingers (preferred for single rescuer)

Quality: compression NOT rocking motion, allow FULL RECOIL between compressions, MINIMAL INTERRUPTION (preplan before you interrupt)

Airway-Breathing Support

Airway

- Head tilt chin lift to open airway. Cannot use this technique if there is a need to immobilize the cervical spine. Only jaw thrust can be used in this case
- Check for breathing (look, listen and feel). Remove obvious FB. Suction secretions, blood, etc.
- Maintain airway
- Oropharyngeal, nasopharyngeal or laryngeal mask airways may be helpful
- Endotracheal intubation once possible
- ETT position must be confirmed
- Surgical airway is rarely necessary

Breathing

- With airway open, use bag & mask to ventilate
- Use the highest possible oxygen (100%)
- Check for a visible chest rise during ventilation
- Use bag and ETT once placed
- If you started with breathing & are not doing CPR, give 5 initial breaths then reassess circulation
- Oxygen recommendations are different in neonatal resuscitation
- Lack of adequate chest rise: verify airway, proper mask seal, absence of a leak or FB obstruction

Synchronizing breaths & chest compressions

- Generally, 15:2 (2 breaths after every 15 chest compressions). Five such sequences represent one cycle (1-2min)
- 30:2 has been suggested for child CPR with a single rescuer, although 15:2 can still be used
- With advanced airway (ETT): no need. Provide breaths continuously at 12/min. Increase to 20/min. if there is (return of) spontaneous circulation
- Hyperventilation during cardiac arrest is not beneficial and may be harmful

In case of choking/ FB obstruction

- An obvious FB can be removed, blind finger sweeps should be avoided
- A conscious patient with effective cough should be allowed to cough & monitored
- If there is no/ loss of effective cough in a conscious patient, apply alternately 5 back blows, 5 abdominal thrusts (children NOT infants), 5 chest thrusts (can use in infants)
- If patient unconscious/ lost consciousness start CPR
- Direct laryngoscopy & removal under vision may be possible

VASCULAR ACCESS

- The best time to establish vascular access is before cardiac arrest
- Peripheral lines can be used during resuscitation. A small bolus of normal saline can be given after each injection to improve drug delivery to the central circulation
- In absence of an alternative, intraosseous or femoral vein access will not significantly interfere with resuscitation. All resuscitation medications can be given intraosseous
- Volume (10-20mL/Kg isotonic crystalloid) is given in hypovolemic patients. This should not delay CPR, adrenaline administration or defibrillation
- Endotracheal and intracardiac routes for medications are no longer recommended

DRUGS

Adrenaline

- During CPR for cardiac arrest, give 10µg/ Kg adrenaline IV and repeat every 2 cycles (3-5min)
- For pulseless VT/VF, give adrenaline after the third shock (if patient still pulseless in shockable rhythm) and repeat every 2 cycles
- 10µg/ Kg equals 0.01 mg/Kg or 0.1mL/Kg using diluted solution (1/10,000 or 1 ampoule of 1 mg in 10mL). The max. adult dose is 1 mg
- There is NO evidence to support higher single doses
- Following return of spontaneous circulation, consider starting an adrenaline infusion and titrate rate to response

Amiodarone

- Used for pulseless VT/VF that has not responded after the third DC shock
- Dose: 5mg/Kg, may be repeated ONCE after the 5th shock
- Lidocaine (1mg/Kg followed by 20-50µg/ Kg/min) may be an alternative

"Glucose, Ca, Mg, bicarbonate and atropine are not routinely recommended during CPR"

Glucose

- Should be avoided except if there is hypoglycemia

Calcium

- Is indicated for hypocalcemia, massive transfusion, hyperkalemia, hypermagnesemia and calcium channel blocker toxicity

Magnesium

- Is indicated for hypomagnesemia and torsade de pointes

Sodium bicarbonate

- May be considered after establishment of adequate ventilation, chest compressions and giving adrenaline if there is severe metabolic acidosis, hyperkalemia, tricyclic antidepressant toxicity or prolonged CPR

"Naloxone reverses opioid-induced CNS depression but it cannot substitute breathing support and can precipitate withdrawal in opioid-dependent patients"

Shockable Rhythms

- Patients with cardiac arrest and a shockable rhythm (VT or VF) must receive immediate DC shock
- CPR must start/ continue until the rhythm is identified and defibrillator is ready
- Check rhythm after every CPR cycle if available
- DC may be delivered manually or using AED

  Manual: use 2-4J/Kg for the first shock and 4J/Kg for all subsequent shocks. Make sure the Synchronize button is not accidentally on with VF as it will lead to failure of giving the shock

  AED: when attached, it will analyze rhythm and display if a shock is needed. You still need to press the shock button to have it delivered. For children 1-8years of age, use an attenuator. For infants, use a manual defibrillator. If unavailable, use whatever is available.

  Either case, paddles must be properly positioned and must not contact each other. Smaller sized paddles are needed for those <10Kg. Apply conducting gel/ cream if available

- Confirm all is clear, with no one touching the patient or bed, before delivering the shock
- Once delivered, immediately remove paddles and resume CPR. do not stop to check response. Provide 1 cycle of CPR first.
- Make interruptions of CPR for checking rhythm as brief as possible. If another shock is needed, continue CPR until defibrillator charged and ready.
- **If the patient is still in cardiac arrest:**

  And rhythm still shockable➔ repeat a shock after every CPR cycle, give adrenaline & amiodarone after the 3rd shock, repeat adrenaline every 2 cycles and amiodarone only once after the 5th shock
  And rhythm changed to non-shockable➔ give adrenaline and continue CPR

- Antiarrhythmic drugs cannot replace a DC shock in the context of cardiac arrest

Correctable Factors

- ✓ Hypoxia
- ✓ Hypovolemia
- ✓ Hypothermia
- ✓ Hypo/hyperkalemia (& other metabolic)
- ✓ Tension pneumothorax
- ✓ Tamponade
- ✓ Thrombosis (PE, coronary)
- ✓ Toxic

Post-Resuscitation Management

- Assess and support for any system dysfunction, correct any correctable factors, address the underlying disease. Continue to monitor patient and determine the appropriate location for care (eg ICU). Key points include:

  Respiration: maintain adequate ventilation and oxygenation. Most patients will require an ETT and positive pressure ventilation. Obtain blood gases and use pulse oximetry to guide oxygen therapy.

  Circulation: assess cardiac rate and rhythm, pulses, BP and perfusion (capillary refill, peripheral temperature & color, distal pulses, etc). Apply shock protocol if needed. Patients may have myocardial dysfunction

  Neurological: patients may develop convulsions or cerebral oedema

  Metabolic: normothermia, glucose, fluid, electrolyte & acid-base status

  Surgery or interventions may be needed to manage trauma, achieve hemostasis or address other life-threatening emergencies. The correct timing and patient support are necessary to maximize benefit and minimize risk

Basic Fluid Therapy

Scope:

- This protocol addresses intravenous fluid therapy in infants and children.
- Neonates are not specifically covered.
- Although the overall principles are similar, some conditions may have different fluid requirements; such as diabetic ketoacidosis, perioperative fluid management, burns, etc.
- For details of fluid management in shock, refer to shock protocol.

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Objectives:

- ✓ Provision of maintenance requirements
- ✓ Replacement of ongoing (excessive/ abnormal) losses
- ✓ Correction of deficit/ dehydration
- ✓ Volume expansion (mainly shock)

Maintenance Requirements:

Normal maintenance per 24hrs

Weight	Calculation	Max.
-10 Kg (excludes neonates)	100 mL/Kg	1000 mL/ 10Kg
>10- 20 Kg	+ 50 mL/kg	1500 mL/ 20 Kg
>20 Kg	+ 20 mL/Kg	2400 mL
All patients	1500 mL/m2	

- Insensible losses are 400mL/m2/day
- Normal maintenance replaces normal urine and insensible losses; since sweat, stool and abnormal losses are normally negligible
- This calculation tends to overestimate requirements of most acutely ill patients after the resuscitation phase. It is suggested to use 70% of this as a starting point in these cases.

Normal maintenance applies provided that:

- Patient is normally hydrated (not dehydrated or oedematous; in which case a +ve or –ve 'respectively' balance relative to maintenance is required)
- No fluid restriction is necessary eg HF, SIADH, etc
  - Generally, 60-70% of normal maintenance is given in such cases
- There are no abnormal losses (radiant warmer, fever, diarrhea, drains, 3rd space, burns, etc). Measured or estimated non-urinary abnormal losses need to be added to normal maintenance.
- Urine output is normal
  - In anuric, oliguric or polyuric patients; maintenance shall equal insensible losses + patient's urine output + abnormal losses if present.

Amount and type of fluids:

	Condition	Amount	Type	Example
Normal maintenance	Normally hydrated, no restrictions, normal urine &no abnormal losses	1500 mL/m2/day Or check table above	0.5NS[(a)], 5-10% glucose[(b)], 10-20mEq/L K	glucose-saline 1:1 with KCl (0.5-1mL/100mL)
Restricted maintenance	Oedema, HF, SIADH, etc	Generally 60-70%, higher restrictions may apply	0.5NS-NS[(c)], 5-10% glucose[(b)], 10-20mEq/L K	Glucose-saline 1:1 or NS, with KCl (0.5-1mL/100mL)
Dehydration		Maintenance + deficit	0.5NS-NS, 2.5-5%[(b)] glucose, 20-40mEq/L K	Glucose-saline 1:1 or NS, with KCl (1-2mL/100mL)
Insensible loss only	Anuria	400mL/m2, may omit in case of overload	≥10% glucose	
Insensible loss + urine output	Oliguria	400mL/m2/day Plus actual patient's urine output per 1,4,12 or 24h[(d)]	As normal maintenance, with/without K	
	Polyuria		0.5NS, ≤5%glucose, 20mEq/L K[(e)]	Glucose-saline 1:1 with KCl (1mL/100mL)
Abnormal losses	Other than in urine	Add amount equal to losses in 1,4,12 or 24hrs[(d)]	Depends on type of fluid lost[(f)]	
PD/ CRRT		Consider patient & therapy balance together		

NOTES:

- Oral/ enteral fluids, medications, drug infusions, blood products, etc are part of maintenance/ replacement fluids. Only 85% of milk is fluid.
- Na, K should be monitored and adjusted accordingly. **Do Not** add maintenance potassium in anuric patients. All stated Na, K, glucose requirements are starting points. Modify based on actual level monitoring.

(a) In critically-ill children (not small infants), 0.5NS is be preferable in the first 24hrs, rather than lower Na (eg Neomaint, 4:1 glucose-saline used in neonates & small infants). Normal saline may be preferred with hyponatremia or CNS injury. Na content must be adjusted based on at least daily serum electrolyte checks.

(b) Many acutely ill children (>6Mo) will not require glucose initially. If needed to maintain blood glucose, prevent catabolism & ketosis, 10% glucose in maintenance fluids may be necessary if tolerated. Higher concentrations require central access unless for a very short term. Blood glucose should not consistently exceed 180-200 mg/dL. Higher infusion rates (eg in dehydrated patients) require reducing glucose concentration. Patients with liver cell failure, adrenal insufficiency or metabolic crisis should receive at least 10% glucose, even if insulin is required. Very rapid replacement (eg severly polyuric patients) may not tolerate even 2.5% so use glucose-free fluids (± low rate glucose 10% as a separate infusion).

(c) Unless sodium restriction is also necessary

(d) Interval depends on patient's condition and how changing it is. As a start, use 1hr for immediate postcardiac surgery, posttransplantation and those on CRRT; use 12-24h for values close to normal maintenance (i.e. slight increase or decrease) and 4h for most other cases. Increase interval when stable

(e) Extremely polyuric patients are expected to need higher Na and lower K content

(f) For example, upper GI losses by Ringer plus 20 mEq/l potassium, diarrhea by usual rehydration solutions, drains with Ringer lactate, etc

Dehydration & Deficit Therapy

Dehydration	Mild	Moderate	Severe
Description	History of ECF loss, acute wt loss, just detectable dehydration, score 1-2	Significant dehydration: score 3-7, or TWO of (sunken eyes, poor skin turgor, thirst, irritability)	Hypovolemic shock, lethargy* or severe metabolic acidosis* Score 8+ two of (sunken eyes, very poor skin turgor, lethargy* or inability to drink*) *of no other cause
Deficit (mL/Kg) 'higher values for smaller patients'	**30-50**	**60-90**	**100-150**

- Score allocates 2 points (0=normal, 1=mild, 2=severe) for each of fontanel, tongue, eyes & skin turgor. Two points are added for each of shock, lethargy and metabolic acidosis if present.

Deficit Therapy:

- Shock should be treated as needed. Patients with severe dehydration who are not shocked may still be given a rapid bolus of 10-20mL/Kg isotonic crystalloid (normal saline) over 30-60 min; which is then subtracted from deficit

There are rapid, slow & very slow methods for correction of dehydration:

Rate	Rapid	Slow	Very slow
Duration of correction	≤ 6hrs	24hrs	≥48 hrs
Indications	- Rapid fluid losses - Acute severe dehydration (eg GE) In children with no concerns from rapid hydration (eg HF, electrolyte disturbance, etc)	Most hospitalized cases	Concerns of osmotic shifts (eg DKA, hypernatremia)
Technique	Give deficit over 6h (? 3-4h in children >2yrs)	Give 24h maintenance + deficit over 24hrs Other approaches: - Give ½ deficit (+1/3 daily maint.) in 8h, then ½ deficit (+2/3 daily maint) in 16h - Give deficit + (½ daily maint) over 12h	Give maintenance + 30-50 mL/kg/day until corrected (typically will take 2-3 days)

- **Type of fluid:** 0.5NS –NS with glucose 2.5% (rapid)-5% (slow) and 20 mEq/l potassium (more if needed). Premixed rehydration fluids with 77—142mEq/L Na may be used (care of K content)
- **Potassium** should not be added in anuric or hyperkalemic patients although hypokalemia may still be corrected (by formula, without maintenance K) in anuric patients if needed.
- **Dehydration** should be reassessed frequently.
    - **Severity** may be different from initial assessment and patients may have abnormal losses or have/ develop acute kidney injury.

Hyponatremia

Hyponatremia denotes a free water excess relative to Na content; however, ECF volume may be increased, normal or decreased

ECF volume	Pathophysiology	Examples	Management strategy
Reduced (hyponatremic dehydration)	Na loss > water loss	GE, diuretics, adrenal insufficiency, salt-losing nephropathies, etc	Rehydrate with higher Na Crude: use NS or 3/4NS & modify according to rate of Na rise Accurate: add 10 ml/kg hypertonic saline to total daily fluids
Normal	Free water gain	SIADH	Water restriction (60-70%) Use 0.5NS - NS (unless Na restriction is also needed)
Increased	Water> salt retention	Congestive HF, hepatic failure, nephrotic syndrome	

- NS, 3/4NS & ½ NS refer to Na content. Appropriate glucose & K should be added.
- Correction rate at no more than 0.5 mEq/Kg hourly (12/day), preferably even slower (8/day). Check & modify rate of rise after 6h.
- Severe symptomatic cases may warrant initial partial correction with 5-10 ml/Kg hypertonic saline to raise Na 4-8 mEq/L which is adequate to reverse acute symptoms), followed by more gradual correction. Approaches include 5mL/kg over 1-2 h (30 min for severe brain oedema) which may be repeated once. Alternatively, 2mL/Kg over 15-30 min, which may be repeated once if symptoms persist and for a 3rd time after checking serum Na if symptoms still persist.

Hypernatremia

Hypernatremia denotes a free water deficit relative to Na content; however, ECF volume may be increased, normal or decreased

ECF volume	Pathophysiology	Examples	Management strategy
Reduced (hypernatremic dehydration)	Water loss> Na loss	GE	Give maintenance + 40mL/Kg/day with 1/3NS-½ NS Modify according to rate of Na drop: - If too slow: ↑rate or ↓Na content, & vice versa
Normal	Free water loss	DI	Give 40mL/Kg free water (eg G5%) per 24h and the remaining fluids as usual
Increased	Hypertonic Na intake	Concentrated formula, sea water, Na bicarbonate, etc	

- ½ NS & 1/3NS refer to Na content. Appropriate glucose & K should be added.
- Na content can be given as bicarbonate in acidotic patients (eg 30mL bicarbonate in 500 mL glucose 5% + 5mL KCl will be 60meq/L Na – between 1/3NS & ½ NS)
- Correction rate at no more than 0.5 mEq/Kg hourly (12/day). Check & modify rate of drop after 6h.
- An accurate calculation is possible, based on urine vol, urine Na, insensible loss & current ECF volume status to determine 24h maintenance water & Na requirements separately. Then, allocate 40 mL/kg of the total volume as free water and give the remainder with the proportional amount of calculated Na.
- PD is needed for refractory hyperNa, intractable acidosis or associated AKI with need for dialysis

Hypokalemia

- Interpret K together with pH (a low pH ➔ extracellular shift of K so level will increase; correction will lower K correspondingly)

- Mild hypokalemia which is non-progressive may be corrected by increasing K intake.
- Otherwise, correction can be achieved using the formula:

"Body weight X deficit (target-actual K) X 0.6 mEq (0.3 mL)"

- Amount to be diluted in normal saline & given over few hrs (correct 0.5mEq/l per hr).

- Never give concentrated KCL.

- Target K is 3.0 for chronic hypokalemia & where complete correction is risky or not necessary, 4.0 for those with or at risk for arrhythmia or during correction of acidosis & 3.5 for most cases

- An empiric correction using 0.5mEq/Kg (0.25mL/Kg), DILUTED and given over 1-2 hr may be used

Hyperkalemia

- Interpret K together with pH (correction of acidosis could correct hyperkalemia)
- Emergency measures for hyperkalemia: (K> 7 mEq/L, rising or with ECG changes)

(all may be temporary & K can rebound so if hyperkalemia is severe and K cannot be eliminated from the body, dialysis will be needed)

- Calcium gluconate IV to reverse membrane effects of K
- Intracellular shift with bicarbonate, iv or nebulized salbutamol &/or glucose-insulin
- **Potassium removal** can be achieved using loop diuretics, cation exchange resin (Ca or Na polystyrene sulfonate) or dialysis. The administration of corticosteroids & fluids in cases of hyperkalemia secondary to adrenal insufficiency is also effective.

Fluids & mixtures by sodium content

	Na (mEq/L)	Examples	Glucose & K content
Isotonic	±150	Normal saline Ringer & lactated Ringer (Na slightly less)	None K (4-5 mEq/L) in ringer & LR
3/4 NS	±110	Glucose: saline 1:3	1/4th source glucose (2.5% if using 10%), No K
	80-90	IV rehydration premixed solutions	Glucose <5% K varies by brand (8-30mEq/L)
½ NS	77	Glucose: saline 1:1 Half normal saline	½ source glucose (5% if using 10%, 2.5% if 5%), No K
1/3 NS	±50	Glucose: saline 2:1 Glucose: ½ NS 1:2 1:19 bicarb: glucose (25mL/500) 1:16 bicarb: glucose (30mL/500)	No K. Using 5%, final glucose: 3.3% (6.7% with 10%) 1.7% 5% 5% & higher Na(60mEq/L)
0.2NS	±30	Glucose 10%: saline 4:1 Glucose 10%: 25%: saline 3:1:1 Neomaint Pedimaint	8%, No K 11%, No K 12%, K 10mEq/L 10%, K 20mEq/L, higher Na (37)
	0	Glucose (all conc.)	

Glucose 25% can be used to increase final mixture glucose concentration. Kadalex is glucose 5% with 27mEq/L potassium. To increase K by 20 mEq/L add 5mL KCl per 500 mL, or 1 mL/100Ml

Formulae & Calculations

Body surface area (m^2) = $\frac{\text{weight (kg) x 4 + 7}}{\text{weight (Kg)+90}}$

10 Kg ≈ 0.5m^2, 20 Kg ≈ 0.8m^2, 30 Kg ≈1m^2, adult ≈1.7m^2

mL/day to drops/ min: divide by 100

eg 10 Kg with maintenance 100 mL/Kg/d = 1000 mL/day

Rate (drops per min. by ordinary IV set) = 1000 *(mL/day)*/ 100 = 10 *(drops/ min.)*

Limit of oliguria:

-400 mL/m^2/day
-25% normal maintenance
-1mL/Kg/hr up to 10 Kg, 0.5mL/Kg/hr >10Kg, 20mL/hr in those >40kg

eg 10 Kg: 1 mL/kg/h = 10 mL/h = 240 mL/day
30 Kg= 1 m^2 body surface area = 400 mL/day

Limit of polyuria:

-2000 mL/1.7m^2/d
- 0.8-1.0 x normal maintenance
- 3mL/kg/hr up to 10 Kg

eg 10 Kg: 30 mL/hr= 720 mL/day
30 Kg= 1m^2 body surface area = 1200 mL/day *(2000mL/1.7m^2)*

Universal fluid balance formula:

Obligatory intake (medications, infusions, transfusions) + enteral*&parenteral nutrition + IV fluids= Insensible loss + Urine output + Abnormal losses + Ultrafiltration
*only calculate oral/enteral fluids and water content of foods (eg 85% of milk formula)

Metabolic acidosis:

Weight x deficit x 0.33 = mEq $NaHCO_3$ (or mL 8.4% solution)

Deficit= base deficit -5, also ½ base deficit or 15- patient bicarbonate
eg 10 Kg, base excess -20 mEq/L
Deficit (base deficit - 5) = 20 - 5 = 15
Amount of 8.4% bicarbonate: 10 *(weight)* X 15 *(deficit)* X 0.33 = 50 mL

Hypokalemia:

Weight x deficit x 0.6 = mEq KCl (or 0.3 = mL KCl)

KCl MUST BE DILUTED in normal saline
Deficit= target K – patient K , Target K can be 3-4 meq/L depending on the condition
Correct a deficit of 0.5/hr

Hyponatremia:

Rapid correction 5-10 mL/ Kg hypertonic saline in 2hrs

Glucose: insulin for hyperkalemia:

MIX 0.5g/kg glucose (eg 5ml/kg glucose 10%, 2mL/Kg 25%) with 0.1 u/kg soluble insulin & give over ½ hr. May double both, i.e. 1g/kg glucose & 0.2 u/Kg insulin

eg 20 Kg
GLUCOSE: 40 mL of 25% or 100 mL of 10%
INSULIN: 2 u soluble insulin

24-hr infusion calculation:

Basic formula 1.44 mg/Kg/day (or 1.5 mg/25hrs) = 1 mcg/Kg/min
Body weight (Kg) x target dose (mcg/Kg/min.) x 1.44 mg ➔ dilute to 24mL & give at 1 mL/hr

eg 20 Kg, Noradrenaline 0.1 µg/Kg/min
20 *(body weight)* X 0.1 *(target dose)* X 1.44 ≈ 3 mg
3 mg diluted to 24 mL and give at 1 mL/hr
1 mL/hr = 0.1 µg/Kg/min. Adjust rate according *(eg 1.5mL/hr= 0.15 µg/Kg/min.)*

Shock

Definition:

Shock is an acute process characterized by the body's inability to deliver adequate oxygen to meet the metabolic demands of vital organs and tissues.

Table (1): Types of Shock (Nelson 20th edition)

Hypovolemic	Cardiogenic	Distributive	Septic	Obstructive
Decreased preload secondary to internal or external losses	Cardiac pump failure secondary to poor myocardial function	Abnormalities of vasomotor tone from loss of venous and arterial capacitance	Encompasses multiple forms of shock Hypovolemic; third spacing of fluids into the extracellular, interstitial space Distributive: early shock with decreased afterload Cardiogenic; depression of myocardial function by endotoxins	Decreased cardiac output secondary to direct impediment to right- or left- heart outflow or restriction of all cardiac chambers
Potential etiologies Blood loss: hemorrhage; Plasma loss: burns, nephrotic syndrome; Water/electrolyte loss: vomiting diarrhea	Congenital heart disease Cardiomyopathies: infectious or acquired, dilated or restrictive Ischemia Arrhythmias	Anaphylaxis Neurologic: loss of sympathetic vascular tone secondary to spinal cord or brainstem injury Drugs	Bacterial Viral Fungal (Immunocompromised patients are at increased risk)	Tension pneumothorax Pericardial tamponade Pulmonary embolism Anterior mediastinal masses Critical coarctation of the aorta

Table (2): Hemodynamic variables in different shock states

(nelson 20th edition)

Type of shock	Cardiac output	Systemic vascular resistance	Mean arterial pressure	Capillary wedge pressure	Central venous pressure
Hypovolemic	↓	↑	↔ or ↓	↓↓↓	↓↓↓
Cardiogenic*					
Systolic	↓↓	↑↑↑	↔ or ↓	↑↑	↑↑
Diastolic	↔	↑↑	↔	↑↑	↑
Obstructive	↓	↑	↔ or ↓	↑↑♦	↑↑♦
Distributive	↑↑	↓↓↓	↔ or ↓	↔ or ↓	↔ or ↓
Septic					
Early	↑↑↑	↓↓↓	↔ or ↓□	↓	↓
Late	↓↓	↓↓	↓↓	↑	↑ or ↔

Systolic or diastolic dysfunction
Wedge pressure, central venous pressure, and pulmonary artery diastolic pressures are equal
Wide pulse pressure

Sepsis:

- Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection (new definition JAMA 2016).
- Organ dysfunction can be identified as an acute change in total SOFA score 2 points consequent to the infection.
- The baseline SOFA score can be assumed to be zero in patients not known to have preexisting organ dysfunction.

SOFA Score:

Table (3): Sequential [Sepsis-related] organ failure assessment score *(JAMA 2017)*

Variables	Score[a] 0	1	2	3	4
Respiratory					
$Pao_2:Fio_2$[b] or $Spo_2:Fio_2$[c]	≥400	300-399	200-299	100-199 With respiratory support	<100 With respiratory support
	≥292	264-291	221-264	148-220 With respiratory support	<148 With respiratory support
Coagulation					
Platelet count, $\times 10^3/\mu L$	≥150	100-149	50-99	20-49	<20
Hepatic					
Bilirubin, mg/dL	<1.2	1.2-1.9	2.0-5.9	6.0-11.9	>12.0
Cardiovascular					
MAP by age group or vasoactive infusion, mm Hg or μg/kg/min[d]					
<1 mo	≥46	<46	Dopamine hydrochloride ≤5 or dobutamine hydrochloride (any)	Dopamine hydrochloride >5 or epinephrine ≤0.1 or norepinephrine bitartrate ≤0.1	Dopamine hydrochloride >15 or epinephrine >0.1 or norepinephrine bitartrate >0.1
1-11 mo	≥55	<55			
12-23 mo	≥60	<60			
24-59 mo	≥62	<62			
60-143 mo	≥65	<65			
144-216 mo	≥67	<67			
>216 mo[e]	≥70	<70			
Glasgow Coma Score[f]	15	13-14	10-12	6-9	<6
Renal					
Creatinine by age group, mg/dL					
<1 mo	<0.8	0.8-0.9	1.0-1.1	1.2-1.5	≥1.6
1-11 mo	<0.3	0.3-0.4	0.5-0.7	0.8-1.1	≥1.2
12-23 mo	<0.4	0.4-0.5	0.6-1.0	1.1-1.4	≥1.5
24-59 mo	<0.6	0.6-0.8	0.9-1.5	1.6-2.2	≥2.3
60-143 mo	<0.7	0.7-1.0	1.1-1.7	1.8-2.5	≥2.6
144-216 mo	<1.0	1.0-1.6	1.7-2.8	2.9-4.1	≥4.2
>216 mo[e]	<1.2	1.2-1.9	2.0-3.4	3.5-4.9	≥5

qSOFA Score:

- Patients with suspected infection who are likely to have a prolonged ICU stay or to die in the hospital can be promptly identified at the bedside with qSOFA, ie, alteration in mental status, systolic blood pressure 100 mm Hg, or respiratory rate 22/min.

Box 4. qSOFA (Quick SOFA) Criteria (JAMA 2016)

- Respiratory rate ≥ 22/min.
- Altered mentation
- Systolic blood pressure ≤ 100 mmHg

Septic shock

- Septic shock is a subset of sepsis in which underlying circulatory and cellular/ metabolic abnormalities are profound enough to substantially increase mortality.

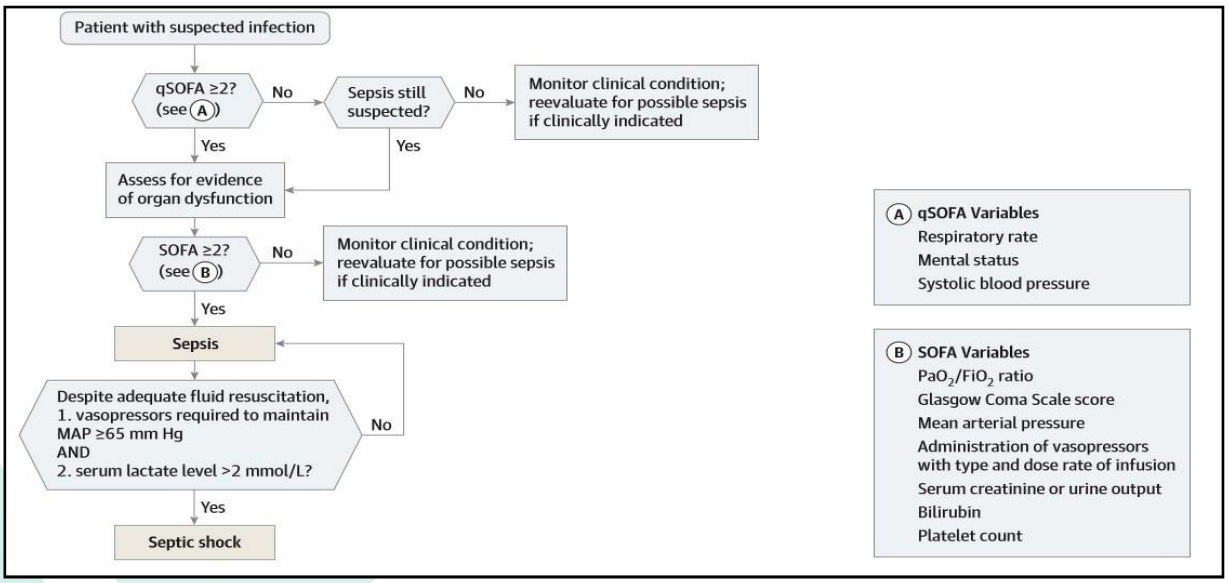
Patients with septic shock can be identified

- With a clinical construct of sepsis with persisting hypotension requiring vasopressors to maintain MAP 65 mm Hg and having a serum lactate level >2 mmol/L (18 mg/dL) despite adequate volume resuscitation. With these criteria, hospital mortality is in excess of 40%.

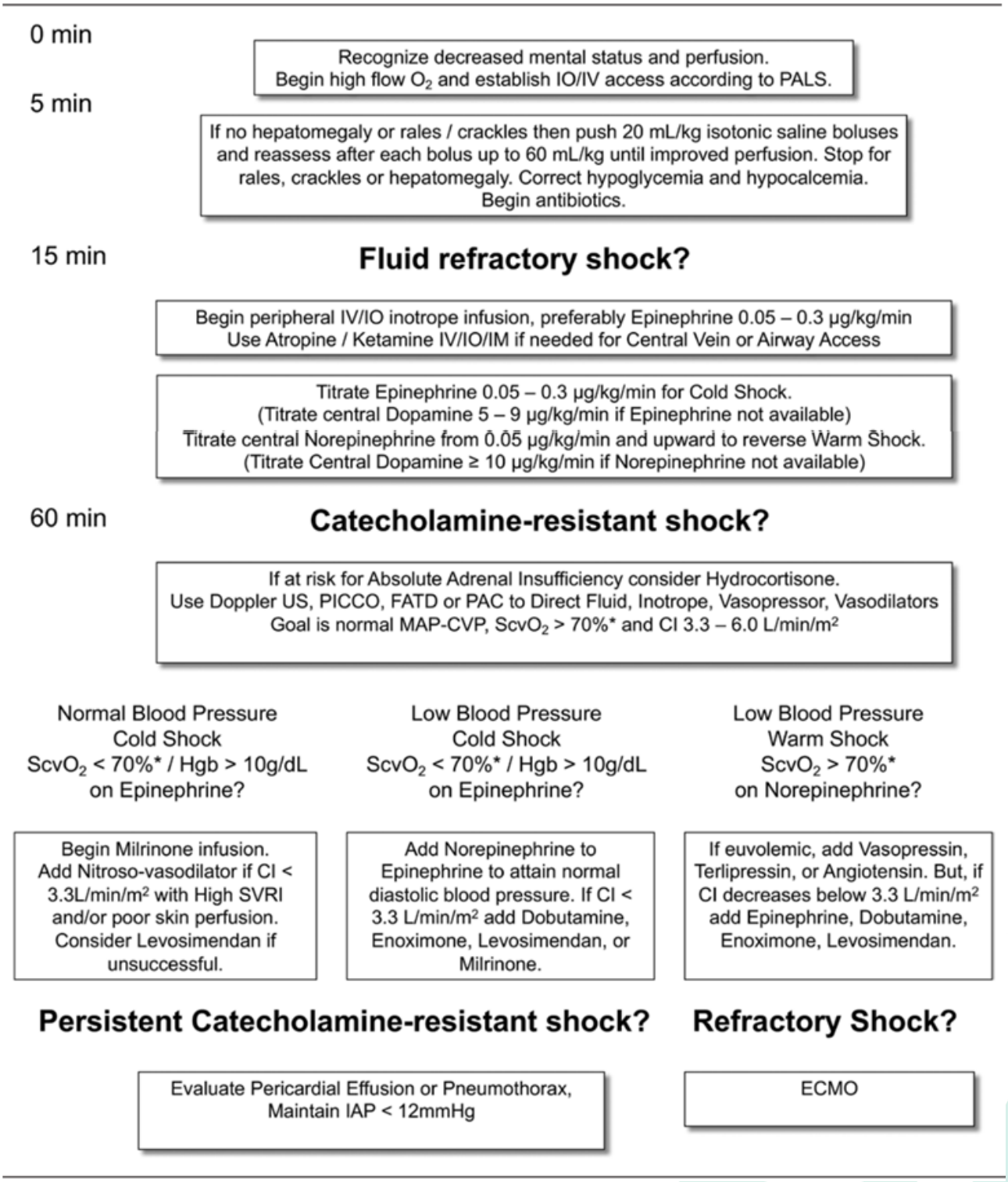
How to differentiate between cold and warm shock?

	Cold shock	Warm shock
Capillary refill	> 2 seconds	Flash capillary refill
Peripheral pulses	Diminished	Bounding
Mottling of skin	Present	Absent
Pulse pressure	Narrow	Wide

How to proceed? (JAMA 2016)



Management of Septic Shock (nelson 21th edition)



Early Goal Directed Therapy: (The 2012 Surviving Sepsis Campaign Guidelines)

During the first 6 hours of resuscitation, the goals of initial resuscitation of sepsis-induced hypoperfusion should include all of the following as one part of a treatment protocol:

- Central venous pressure (CVP) 8-12 mm Hg
- Mean arterial pressure (MAP) ≥65 mm Hg
- Urine output ≥0.5 mL/kg/hr
- Cardiac index > 3.3 < 6.0L/min/m2 in PICU
- Central venous (superior vena cava) or mixed venous oxygen saturation
- ≥70 percent or ≥65 percent, respectively

Therapeutic Endpoints

Clinical

- Heart Rate normalized for age
- Capillary refill < 2sec
- Normal pulse quality
- Warm extremities
- Blood pressure normal for age
- Urine output >1 mL/kg/h
- Normal mental status
- CVP >8 mmHg
- No difference in central and peripheral pulses

Threshold rates	Heart rate (bpm)	Mean arterial pressure
Term newborn	120-180	55
Up to 1 yr	120-180	60
Up to 2 yrs	120-160	65
Up to 7 yrs	100-140	65
Up to 15 yrs	90-140	65

Laboratory:

- Decreasing lactate
- SvO2 >70%

Table (4): Vasodilators/Afterload Reducers (nelson 20th edition)

Drug	Effect(s)	Dosing range	Comment(s)
Nitroprusside	Vasodilator (mainly arterial)	0.5-4.0 µg/kg/min	Rapid effect Risk of cyanide toxicity with prolonged use (> 96hr)
Nitroglycerin	Vasodilator (mainly venous)	1-20 µg /kg/min	Rapid effect Risk of increased intracranial pressure
Prostaglandin E1	Vasodilator Maintains an open ductus arteriosus in the newborn with ductal-dependent congenital heart disease	0.01-0.2 µg /kg/min	Can lead to hypotension Risk of apnea
Milrinone	Increased cardiac contractility Improves cardiac diastolic function Peripheral vasodilation	Load 50 µg /kg over 15 min 0.5-1.0 µg/kg/min	Phosphoodiesterase inhibitor – slows cyclic adenosine monophosphate breakdown

Table (5): Cardiovascular Drug Treatment of Shock (nelson 20th edition)

Drug	Effect(s)	Dosing range	Comment(s)
Dopamine	↑ Cardiac contractility	3-20 µg/kg/min	↑ Risk of arrhythmias at high doses
	Significant peripheral vasoconstriction at > 10 g/kg/min		
Epinephrine	↑ Heart rate and ↑ cardiac contractility Potent vasoconstrictor	0.05-3.0 µg/kg/min	May ↓ renal perfusion at high doses ↑ Myocardial O2 consumption Risk of arrhythmia at high doses
Dobutamine	↑ Cardiac contractility Peripheral vasodilator		
Norepinephrine	Potent vasoconstriction No significant effect on cardiac contractility	0.05-1.5 µg/kg/min	↑ Blood pressure secondary to ↑ systemic vascular resistance ↑ Left ventricular afterload
Phenylephrine	Potent vasoconstriction	0.05-2.0 µg/kg/min	Can cause sudden hypertension ↑ O2 consumption

Terlipressin dose in septic shock:

- Loading dose 20 mic/kg/ dose followed by continuous infusion 4- 20 mic/ kg/ hour

REFERENCES:

- David A. Turner and Ira M. Cheifetz, shock Chapter 70 Part IX 516:528 nelson textbook of pediatrics 20th edition 2015
- Mervyn Singer, Clifford S. Deutschman, Christopher Warren Seymour, Manu Shankar-Hari, Djillali Annane, Michael Bauer et al., The Third International Consensus Definitions for Sepsis and Septic Shock(Sepsis-3) JAMA.2016;315(8):801-810.
- Rodriguez-Nunez A, Lopez-Herce J, GilAntonJ, et al: Rescue treatment with terlipressin in children with refractory septic shock: A clinical study. Crit Care 2006; 10: R20 (doi:10.1186/cc3984)

Anaphylactic Shock:

Emergency Treatment:

- Patients with anaphylaxis should be placed on their back with lower extremities elevated. If short-of-breath and/or vomiting, patient should be placed semi-upright in a position of comfort with the lower extremities elevated.

  - Intramuscular epinephrine 1: 1000 (1 mg/ml) at a dose of 0.01 mg/kg body weight up to a maximum dose of 0.3 mg injected into the lateral thigh (vastus lateralis).
  - The dose can be repeated at 5-15 min intervals.
  - The intramuscular route is preferred because epinephrine has a vasodilator effect in skeletal muscle. After IM injection into the vastus lateralis, absorption is rapid, and epinephrine reaches the central circulation rapidly.
  - The maximum dose of epinephrine in anaphylaxis is lower than the dose used in cardiopulmonary resuscitation.
  - Failure to inject it promptly before the patient gets acute cardio-respiratory failure and shock potentially increases the risk of death and the risk of biphasic anaphylaxis (late phase reaction).

- Support the airway and ventilation

- Give supplementary oxygen 6-8 L/min

- Resuscitate with intravenous saline 0.9% (20 ml/kg body weight, repeated up to a total of 50 ml/kg over the first half an hour.

- Other lines of treatment:

  - Nebulized beta-2 stimulants: Decrease wheeze but are not life-saving H1-antihistamines: Decrease itch and hives but not life saving
  - Dose of diphenhydramine (Pirafene 50 mg/ml):
    - ✓ 2-6 years: 6.25 mg 6-12 years: 12.2-25 mg > 12 years: 25-50 mg
  - Corticosteroids: effects take several hours: not lifesaving. Used to prevent biphasic; however, there is no evidence that this occurs.
  - Dose of Hydrocortisone : 2.5- 5mg / kg

Refractory Cases:

- ***IV epinephrine:*** central line – 1:10,000 solution – infusion pump ¯ Intubation
- ***Cricothyrotomy***
- ***Vasopressors***
- ***Glucagon:*** exerts positive inotropic and chronotropic effects on the heart, independent of catecholamines. Therefore, glucagon, 1 mg intravenous bolus, followed by an infusion of 1 to 5 mg per hour, may improve hypotension in one to five minutes, with a maximal benefit at five to 15 minutes. (The U.S. Food and Drug Administration have not approved glucagon for this use.) Nausea and vomiting may limit therapy with glucagon.

Duration of Monitoring:

- Protracted or biphasic anaphylaxis (up to 72 hours; usually within 10 hours) occurs in up to 20% of adults and 6% of children. ¯ Patients should ideally be monitored for at least 4, and preferably 8-10 hr.
- Some cases require monitoring for ≥ 24 hours.

Place the patient on their back with lower extremities elevated.

If short-of-breath and/or vomiting, patient should be placed semi-upright in a position of comfort with the lower extremities elevated.

↓

Adrenaline I.M

1: 1000 (1 mg/ml) at a dose of 0.01 mg/kg body weight up to a maximum dose of 0.3 mg injected into the lateral thigh (vastus lateralis)

↓

Support the airway and ventilation

Give supplementary oxygen 6-8 L/min

↓

Resuscitate

Intravenous saline 0.9% (20 ml/kg body weight, repeated up to a total of 50 ml/kg over the first half an hour.

↓

Other lines of treatment (not lifesaving)

↙ ↓ ↘

Nebulized beta-2 stimulants | H1-antihistamines | Corticosteroids

↓

Refractory cases

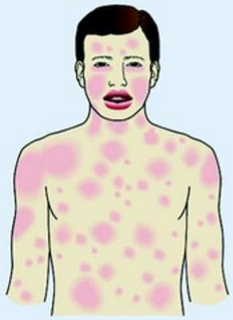
- ***IV epinephrine: central line – 1:10,000 solution – infusion pump***
- ***Intubation***
- ***Cricothyrotomy***
- ***Vasopressors: noradrenaline or dopamine***
- ***Glucagon***

Acknowledgment:

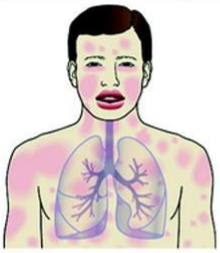
- **Thanks to Prof. Dr. Elham Hosni, for participating in this chapter.**

Anaphylaxis is highly likely when any one of the following three criteria is fulfilled:

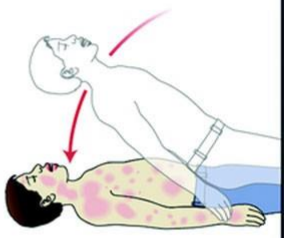
1 Sudden onset of an illness (minutes to several hours), with involvement of the skin, mucosal tissue, or both (e.g. generalized hives, itching or flushing, swollen lips-tongue-uvula)



AND AT LEAST ONE OF THE FOLLOWING:

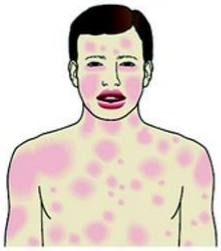


Sudden respiratory symptoms and signs (e.g. shortness of breath, wheeze, cough, stridor, hypoxemia)

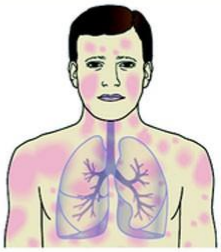


Sudden reduced BP or symptoms of end-organ dysfunction (e.g. hypotonia [collapse], incontinence)

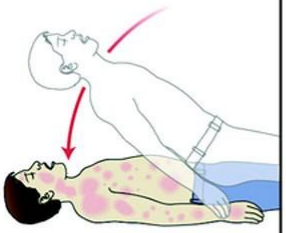
OR 2 Two or more of the following that occur suddenly after exposure to a *likely allergen or other trigger* for that patient* (minutes to several hours):



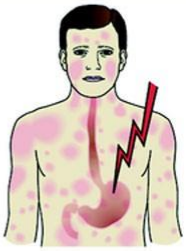
Sudden skin or mucosal symptoms and signs (e.g. generalized hives, itch-flush, swollen lips-tongue-uvula)



Sudden respiratory symptoms and signs (e.g. shortness of breath, wheeze, cough, stridor, hypoxemia)



Sudden reduced BP or symptoms of end-organ dysfunction (e.g. hypotonia [collapse], incontinence)



Sudden gastrointestinal symptoms (e.g. crampy abdominal pain, vomiting)

OR 3 Reduced blood pressure (BP) after exposure to a *known allergen** for that patient* (minutes to several hours):

Infants and children: low systolic BP (age-specific) or greater than 30% decrease in systolic BP***

Adults: systolic BP of less than 90 mm Hg or greater than 30% decrease from that person's baseline

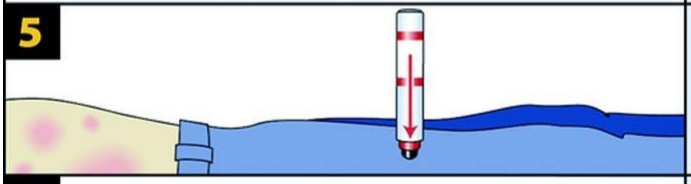
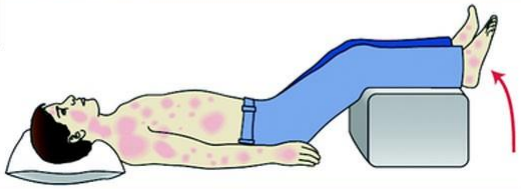
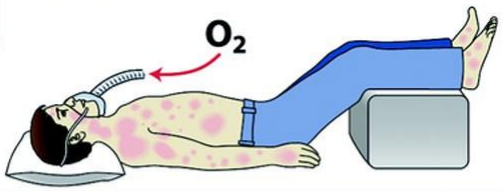
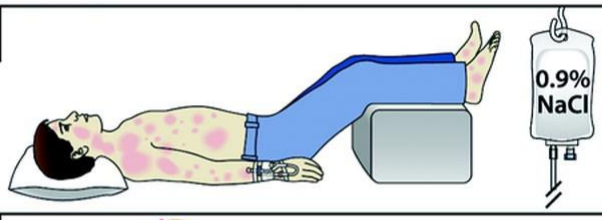
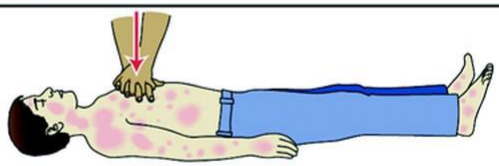
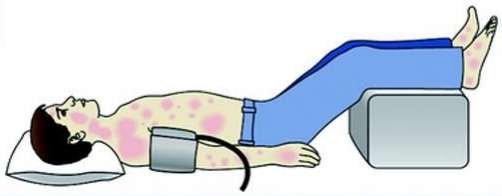
* For example, immunologic but IgE-independent, or non-immunologic (direct mast cell activation)

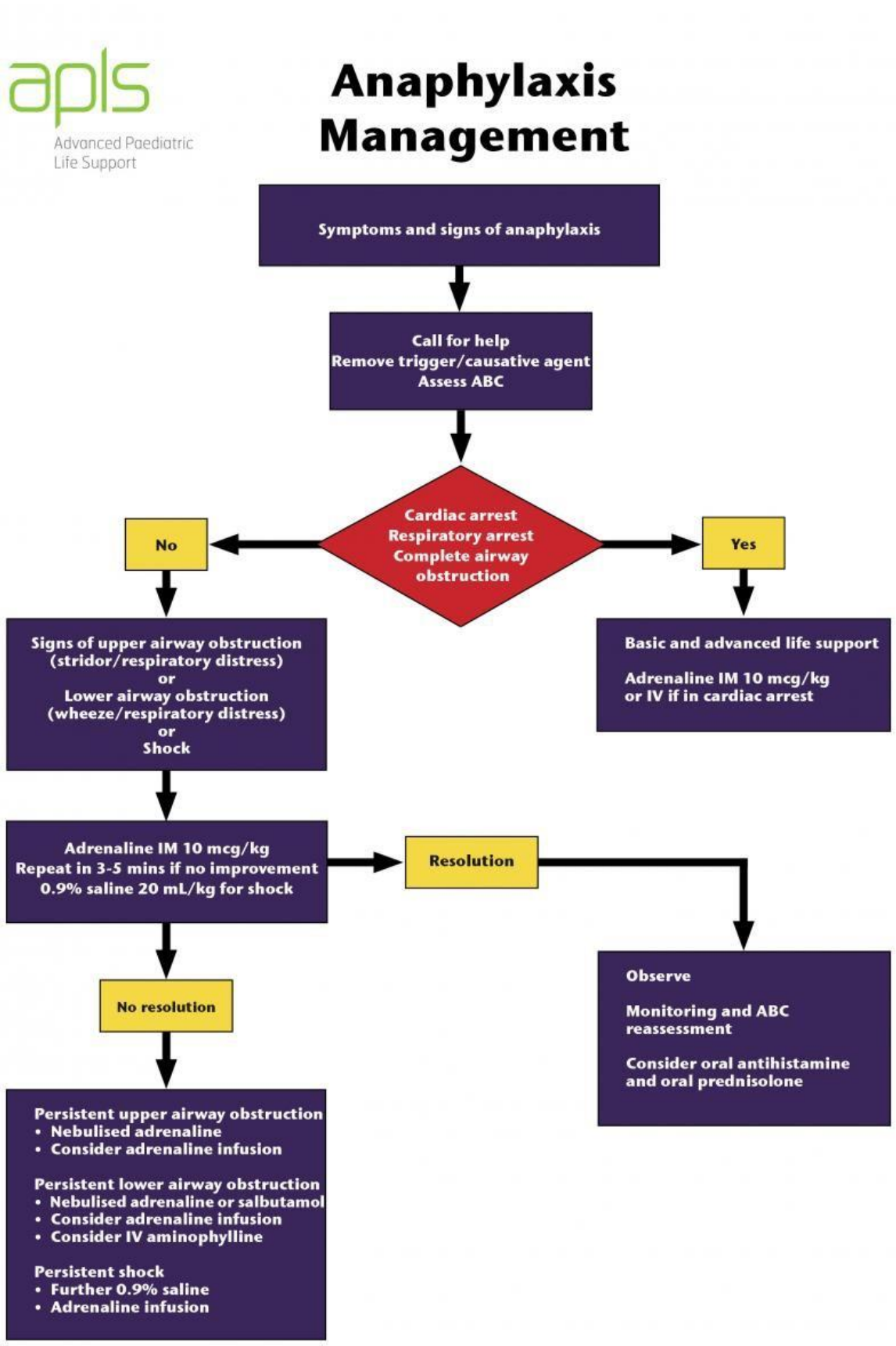
** For example, after an insect sting, reduced blood pressure might be the only manifestation of anaphylaxis; or, after allergen immunotherapy, generalized hives might be the only initial manifestation of anaphylaxis.

*** Low systolic blood pressure for children is defined as less than 70 mm Hg from 1 month to 1 year, less than (70 mm Hg + [2 x age]) from 1 to 10 years, and less than 90 mm Hg from 11 to 17 years. Normal heart rate ranges from 80-140 beats/minute at age 1-2 years; from 80-120 beats/minute at age 3 years; and from 70-115 beats/minute after age 3 years. In infants and children, respiratory compromise is more likely than hypotension or shock, and shock is more likely to be manifest initially by tachycardia than by hypotension.

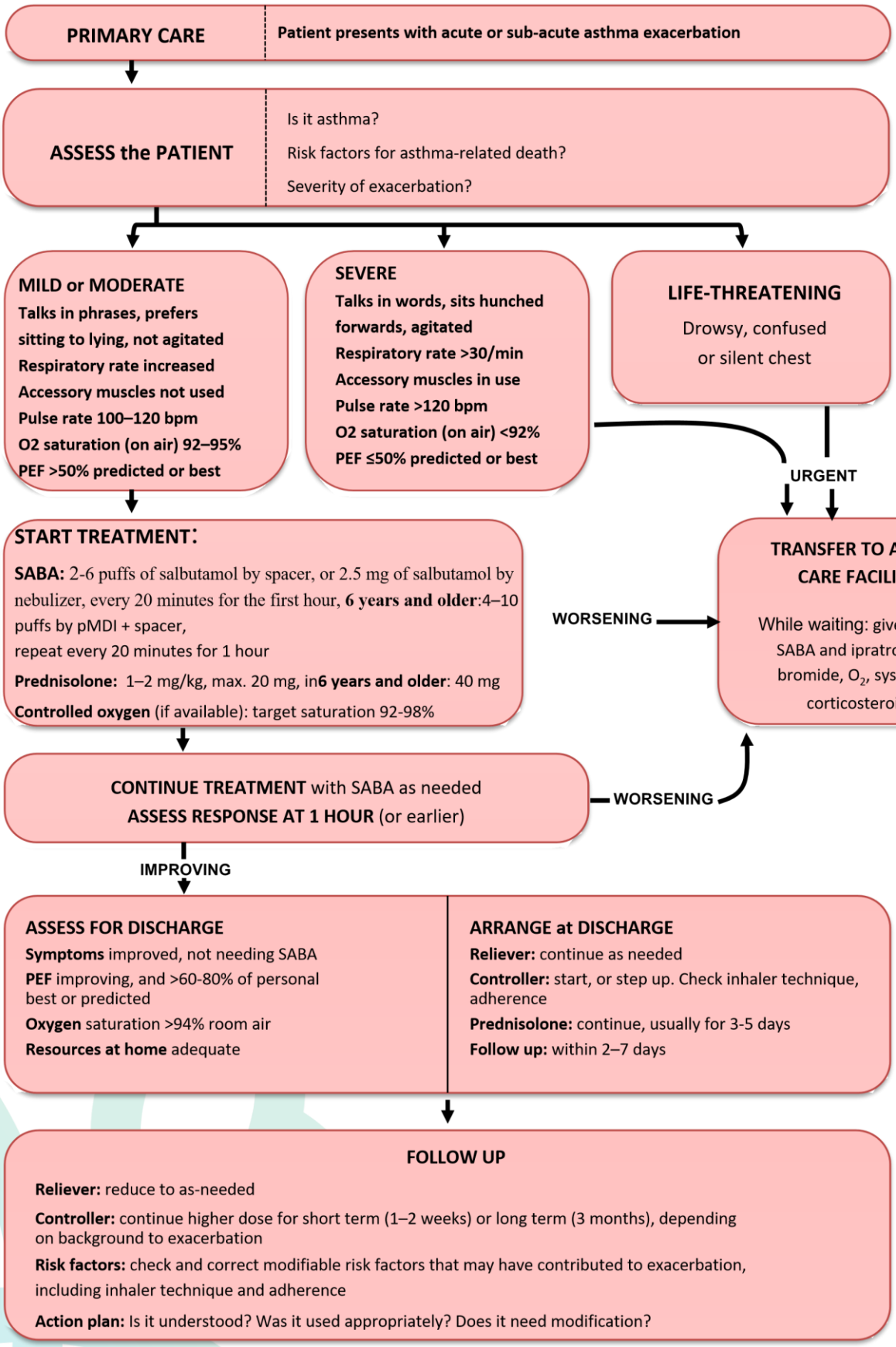
REFERENCE:

- Simons FER et al., for the WAO. J Allergy Clin Immunol 2011; 127: 587-93-e22 and WAO Journal 2011; 4: 13-36. Illustrator: J Schaffer

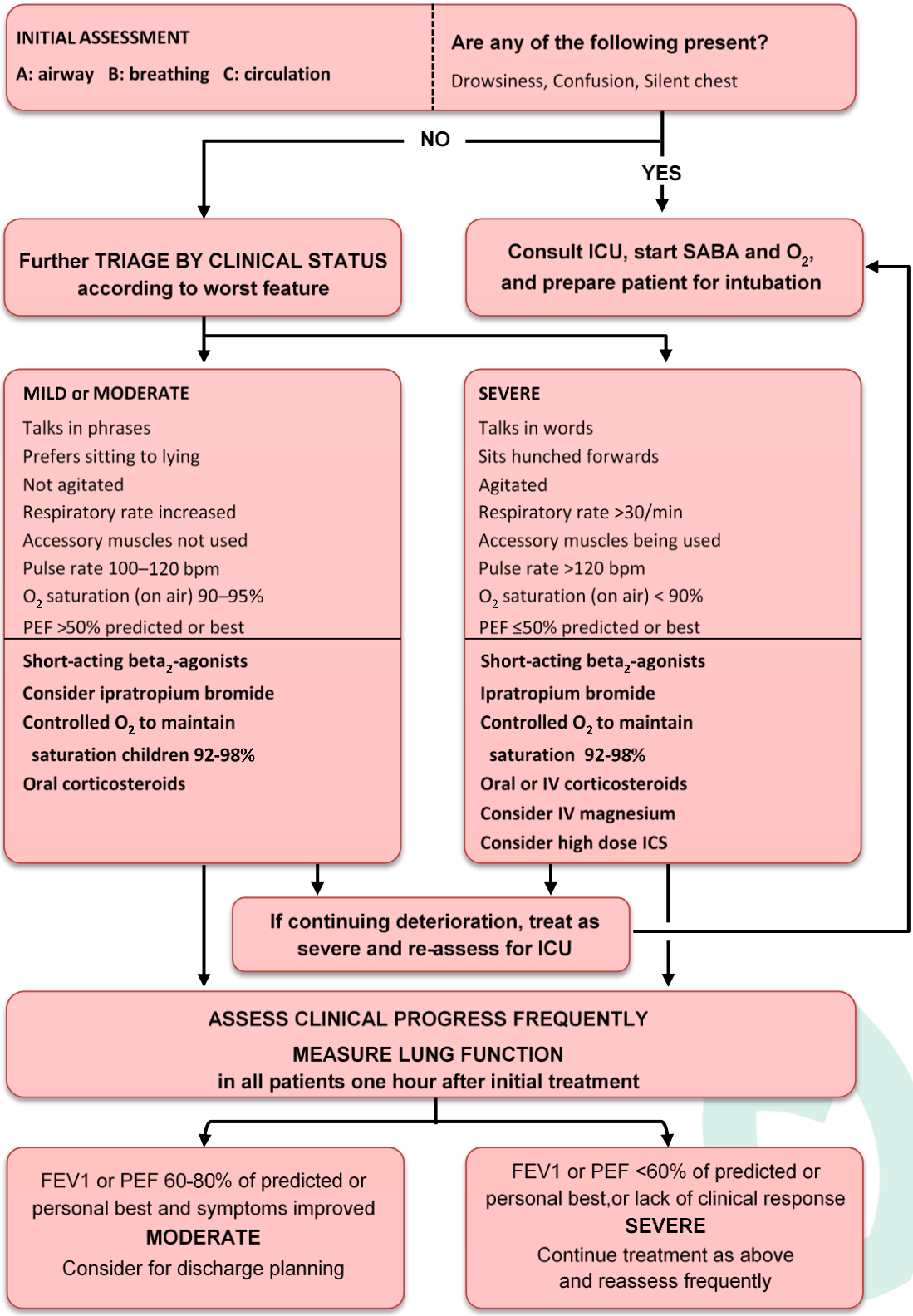
1	**Have a written emergency protocol** for recognition and treatment of anaphylaxis and rehearse it regularly.	
2	**Remove exposure to the trigger** if possible, eg. discontinue an intravenous diagnostic or therapeutic agent that seems to be triggering symptoms.	
3		**Assess the patient's circulation, airway, breathing, mental status, skin, and body weight** (mass).
		Promptly and simultaneously, perform steps 4, 5 and 6.
4		**Call for help:** resuscitation team (hospital) or emergency medical services (community) if available.
5		**Inject epinephrine** (adrenaline) intramuscularly in the mid-anterolateral aspect of the thigh, 0.01 mg/kg of a 1:1,000 (1 mg/mL) solution, maximum of 0.5 mg (adult) or 0.3 mg (child); **record the time of the dose and repeat it** in 5-15 minutes, if needed. Most patients respond to 1 or 2 doses.
6		**Place patient on the back** or in a position of comfort if there is respiratory distress and/or vomiting; **elevate the lower extremities;** fatality can occur within seconds if patient stands or sits suddenly.
7		**When indicated, give high-flow supplemental oxygen** (6-8 L/minute), by face mask or oropharyngeal airway.
8		**Establish intravenous access** using needles or catheters with wide-bore cannulae (14 - 16 gauge). **When indicated, give 1-2 litres of 0.9% (isotonic) saline rapidly** (e.g. 5-10 mL/kg in the first 5-10 minutes to an adult; 10 mL/kg to a child).
9		**When indicated at any time, perform cardiopulmonary resuscitation** with continuous chest compressions.
		In addition,
10		**At frequent, regular intervals, monitor patient's blood pressure, cardiac rate and function, respiratory status, and oxygenation** (monitor continuously, if possible).

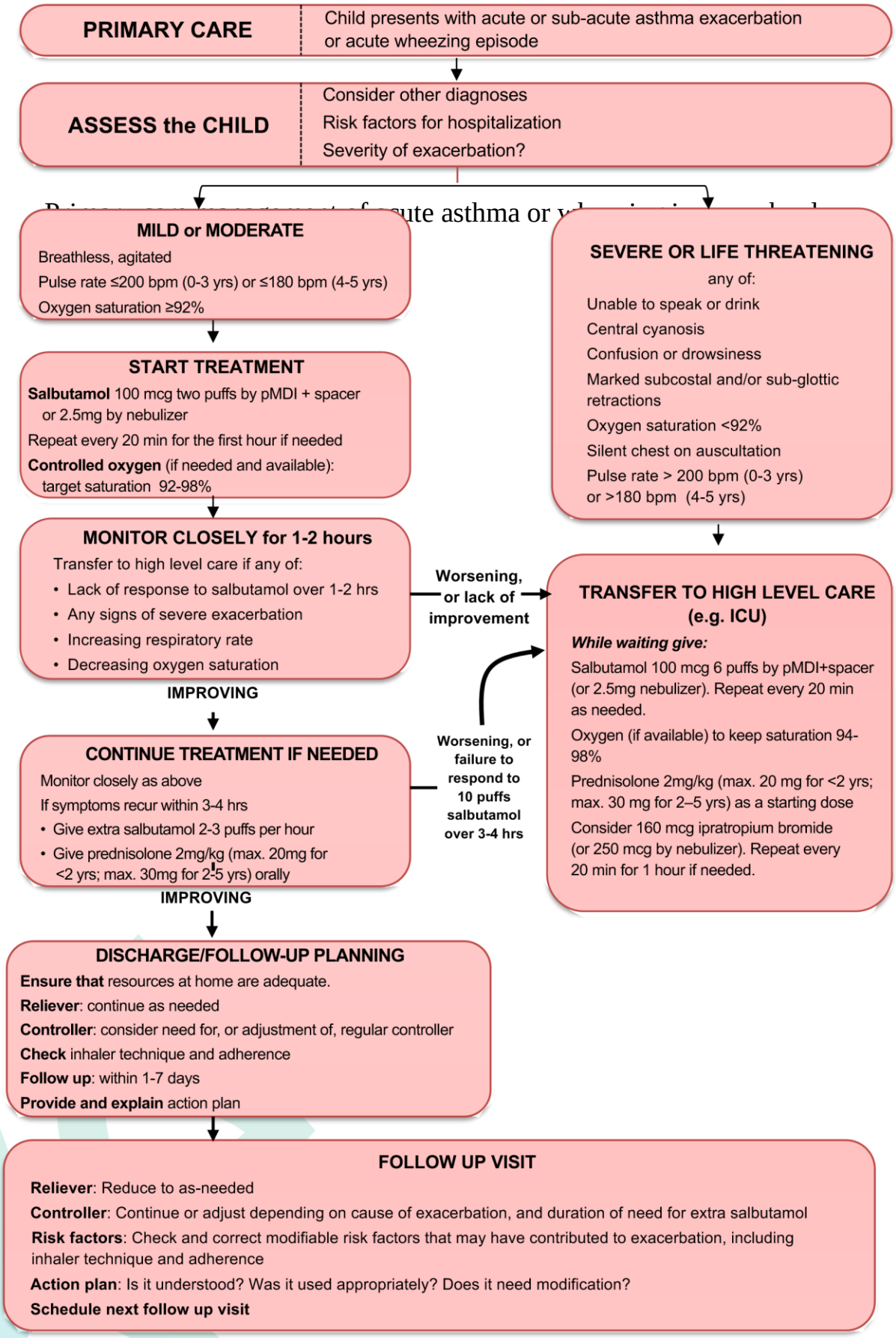

apls
Advanced Paediatric Life Support
Anaphylaxis Management
Symptoms and signs of anaphylaxis
Call for help
Remove trigger/causative agent
Assess ABC
Cardiac arrest
Respiratory arrest
Complete airway obstruction
No
Yes
Signs of upper airway obstruction (stridor/respiratory distress)
or
Lower airway obstruction (wheeze/respiratory distress)
or
Shock
Basic and advanced life support
Adrenaline IM 10 mcg/kg or IV if in cardiac arrest
Adrenaline IM 10 mcg/kg
Repeat in 3-5 mins if no improvement
0.9% saline 20 mL/kg for shock
Resolution
Observe
Monitoring and ABC reassessment
Consider oral antihistamine and oral prednisolone
No resolution
Persistent upper airway obstruction
• Nebulised adrenaline
• Consider adrenaline infusion
Persistent lower airway obstruction
• Nebulised adrenaline or salbutamol
• Consider adrenaline infusion
• Consider IV aminophylline
Persistent shock
• Further 0.9% saline
• Adrenaline infusion

Acute Severe Asthma



Managing exacerbations in acute care settings




PRIMARY CARE
Child presents with acute or sub-acute asthma exacerbation or acute wheezing episode
ASSESS the CHILD
Consider other diagnoses
Risk factors for hospitalization
Severity of exacerbation?
MILD or MODERATE
Breathless, agitated
Pulse rate ≤200 bpm (0-3 yrs) or ≤180 bpm (4-5 yrs)
Oxygen saturation ≥92%
SEVERE OR LIFE THREATENING
any of:
Unable to speak or drink
Central cyanosis
Confusion or drowsiness
Marked subcostal and/or sub-glottic retractions
Oxygen saturation <92%
Silent chest on auscultation
Pulse rate > 200 bpm (0-3 yrs) or >180 bpm (4-5 yrs)
START TREATMENT
Salbutamol 100 mcg two puffs by pMDI + spacer or 2.5mg by nebulizer
Repeat every 20 min for the first hour if needed
Controlled oxygen (if needed and available): target saturation 92-98%
MONITOR CLOSELY for 1-2 hours
Transfer to high level care if any of:
• Lack of response to salbutamol over 1-2 hrs
• Any signs of severe exacerbation
• Increasing respiratory rate
• Decreasing oxygen saturation
Worsening, or lack of improvement
TRANSFER TO HIGH LEVEL CARE (e.g. ICU)
While waiting give:
Salbutamol 100 mcg 6 puffs by pMDI+spacer (or 2.5mg nebulizer). Repeat every 20 min as needed.
Oxygen (if available) to keep saturation 94-98%
Prednisolone 2mg/kg (max. 20 mg for <2 yrs; max. 30 mg for 2–5 yrs) as a starting dose
Consider 160 mcg ipratropium bromide (or 250 mcg by nebulizer). Repeat every 20 min for 1 hour if needed.
IMPROVING
CONTINUE TREATMENT IF NEEDED
Monitor closely as above
If symptoms recur within 3-4 hrs
• Give extra salbutamol 2-3 puffs per hour
• Give prednisolone 2mg/kg (max. 20mg for <2 yrs; max. 30mg for 2-5 yrs) orally
Worsening, or failure to respond to 10 puffs salbutamol over 3-4 hrs
IMPROVING
DISCHARGE/FOLLOW-UP PLANNING
Ensure that resources at home are adequate.
Reliever: continue as needed
Controller: consider need for, or adjustment of, regular controller
Check inhaler technique and adherence
Follow up: within 1-7 days
Provide and explain action plan
FOLLOW UP VISIT
Reliever: Reduce to as-needed
Controller: Continue or adjust depending on cause of exacerbation, and duration of need for extra salbutamol
Risk factors: Check and correct modifiable risk factors that may have contributed to exacerbation, including inhaler technique and adherence
Action plan: Is it understood? Was it used appropriately? Does it need modification?
Schedule next follow up visit

Treatment of Acute Severe Asthma in PICU:

1. Monitoring:

- Noninvasive blood pressure and oxygen saturations (SpO2). Those with respiratory failure requiring mechanical ventilation should undergo the placement of central venous, arterial, and urinary bladder catheters.

2. Oxygen:

- Oxygen should be used as carrier gas for intermittent or continuous nebulization and to keep oxygen saturation above 92%.

3. Steroids:

- Methylprednisolone loading dose of 2 mg/kg followed by 0.5 to 1 mg/kg every 6 hours.

4. Bronchodilator:

- The usual dose for continuous albuterol nebulization ranges from 0.15 to 0.5 mg/kg/h.
- Nebulized ipratropium, in 0.25 to 0.50 mg doses, can be used every 20 minutes during the first hour, followed by the same dose range every 6 hours.

5. Magnesium Sulfate:

- The dose of magnesium sulfate is 25 to 50 mg/ kg/dose (maximum 2 g), infused for 20 to 30 minutes, and followed by continuous infusion dependent on the patient's weight.
- Children weighing < 30 kg: of 25 mg/kg/h
- Children weighing > 30 kg may receive 20 mg/kg/h,
- Infusion rates must not exceed 2 g/h in any patient. Titration to the desired clinical effect should be based on serum magnesium concentrations and tolerability.

Keep serum magnesium level below 6 mg /dl

- Observation of magnesium sulphate side effects:

Serum magnesium concentrations above 9 mg/dL causes:
Nausea, flushing, somnolence, vision changes, muscle weakness and hypotension

Serum magnesium concentrations above 12 mg/dL causes:
Respiratory depression and arrhythmias

6. Methylxanthines:

- The theophylline dose is 80% of the aminophylline dose.
- A loading intravenous dose, 5mg/kg of theophylline or 6 mg/kg of aminophylline, given during 20 minutes is needed to achieve a therapeutic concentration.
- After the loading dose, a continuous infusion should be started. Infants younger than 6 months are 0.5 mg/kg/h
- Infants 6 months to 1 year 0.85 to 1 mg/kg/h
- Children 1 to 9 years, 1 mg/ kg/h
- Children older than 9 years, 0.75 mg/kg/h.

Serum drug concentrations should be obtained:

- 30 to 60 minutes after the loading dose is finished
- At 12 hours after the beginning of the continuous infusion
- Every 12 to 24 hours or when toxicity is suspected.

7. Mechanical ventilation of asthmatic patients:

Bilevel Positive Airway Pressure

- Noninvasive positive pressure ventilation (NPPV) in addition to conventional therapy showed clinical improvement and correction of gas exchange abnormalities in children and adults with asthma. NPPV was well tolerated in children, including patients as young as 1 year
- Typically, recommended settings include an inspiratory positive airway pressure of 10 cm H2O, an expiratory positive airway pressure of 5 cm H2O, with or without a low back-up ventilation rate.

Criteria for selecting severe asthmatic patients for NPPV trial

- ✓ Tachypnea above normal limit of age
- ✓ Tachycardia above normal limit of age
- ✓ Use of accessory muscles of respiration
- ✓ Hypoxia with a Pa,O2/FI,O2 ratio >200 mmHg
- ✓ Hypercapnia with Pa, CO_2, 60 mmHg FEV1< 50% pred"

Absolute and relative contraindication for noninvasive positive pressure ventilation (NPPV) trial

Absolute contraindications:

- ✓ Need for immediate endotracheal intubation
- ✓ Decreased level of consciousness
- ✓ Excess respiratory secretions and risk of aspiration
- ✓ Past facial surgery precluding mask fitting

Relative contraindication:

- ✓ Hemodynamic instability
- ✓ Severe hypoxia and/or hypercapnia, Po2/Fio2 ratio of [200 mmHg, Pco2] 60 mmHg
- ✓ Poor patient cooperation
- ✓ Severe agitation
- ✓ Lack of trained or experienced staff

Invasive Mechanical Ventilation:

1. Criteria for intubation
2. Recommendations for intubation technique
3. Recommendations for appropriate ventilator settings
4. Management in the immediate postintubation period
5. Medical management of asthma in the ventilated patient 6- Prevention and treatment of complications.
6. Prevention and treatment of complications.

Step	Therapy	Comments
1	Albuterol, Ipratropium, Steroids	These medications should be ordered for all patients admitted to the PICU.
2	Continuous Albuterol	0.5-1 mg/kg/hr. If < 20 kg give 10-20 mg/hr; 20-30 kg give 10-30 mg/hr, > 30 kg give 15 -45 mg/hr
3	IV Magnesium	25 to 50 mg/kg/dose (max 2 g) infused over 20 to 30 min. Follow by continuous infusion of 15-25 mg/kg/hr. Mg level ≈ 4 mg/dL. Monitor for hypotension.
4	Heliox	Provide O_2 using non-rebreathing mask. May combine O_2 by nasal cannula if necessary to keep SaO_2 > 92%.
5	IV Terbutaline	Loading dose of 10 mcg/kg over 10 min followed by 0.4 mcg/kg/min. Increase by 0.4 mcg/kg/min every 15 min. Range 0.1 to 10 mcg/kg/min (average dose is 4 mcg/kg/min)
6	IV Theophylline	Loading dose of 5 mg/kg over 20 min followed by continuous infusion of 0.5-1 mg/kg/hr. Check serum theophylline concentration 30 min after the end of the loading dose. Target theophylline concentration is 10-20 mg/L
7	Non-Invasive Ventilation	Consider BiPAP to unload WOB. IPAP:10 EPAP:5
8	IV Ketamine	1 mg/kg/hr for sedation. Bronchodilatory properties. Increase airway secretions.
9	Intubation	Ketamine + Midazolam + Rocuronium
10	Ventilation	Try to avoid neuromuscular blockade. Permissive hypercapnia. PC/PRVC/PSV. Monitor peak to plateau pressure difference.

Criteria for Intubation:

Clinical:

- Cardiac arrest
- Respiratory arrest
- Progressive exhaustion
- Altered sensorium such as lethargy or agitation, interfering with oxygen delivery or anti-asthma therapy.

Laboratory:

- pH less than 7.2, carbon dioxide pressure increasing by more than 5 mm Hg/h or greater than 55 to 70 mm Hg, or oxygen pressure less than 60 mm Hg on 100% oxygen delivered through a mask.

- Failure to reverse severe respiratory acidosis despite intensive therap.

1. Recommendation for intubation:

- ✓ **Orotracheal intubation with sedation** and neuromuscular blockade are preferredfor asthmatic patients in critical respiratory distress.
- ✓ **Intubation medication**

- Atropine at a dose of 0.02 mg/kg IV (minimum 0.1 mg, maximum 0.5 mg child, 1 mg adolescent) used to attenuate the vagal reflexes that lead to laryngospasm and worsen bronchospasm

- Ketamine: 1.0 to 1.5 mg/kg I.V and 1.0 to 1.5 mg/kg succinylcholine I.V
    - It stimulates the release of catecholamines leading to bronchodilation; Side effects include hypersecretion, hypotension and hypertension, arrhythmias, and hallucinations

- Or propofol 2 mg/kg administered I.V over 2 minutes with succinylcholine, preferred in patients with hypertension, and succinylcholine should be avoided in patients with hyperkalemia.

2. Mechanical ventilation recommendations:

- Low rate
- Low PEEP
- Prolonged expiratory time
- Allow hypercapnia: till pH as low as 7.15 and a Pa CO2 of up to 80 mmHg

All these settings to avoid hyperinflation and auto-PEEP

1. Deal with expected complications:

- **Hypotension:**

  ☞ **Look for:**

  - ✓ Pneumothorax
  - ✓ Hypovolemia
  - ✓ High auto PEEP

- **Cardiac arrest:**

  ☞ **Look for:**

  - ✓ Hypoxia
  - ✓ Exclude right mainstem intubation (21 cm at incisors)
  - ✓ Exclude pneumothorax and place pleural drain
  - ✓ Exclude pneumonia and other lung disease
  - ✓ Pneumothorax

Mechanical ventilation algorithm

Atropine

Used to attenuate the vagal reflexes that lead to laryngospasm and worsen bronchospasm

(0.02 mg/kg IV minimum 0.1 mg, maximum 0.5 mg child, 1 mg adolescent)

↓

Ketamine

Stimulates the release of catecholamines leading to bronchodilation; Side effects include hypersecretion, hypotension and

(Dose: 1.0 to 1.5 mg/kg I.V)

↓

Intubation

(Orotracheal is preferable)

↓

Mechanical ventilation settings

Low rate

Low PEEP

Prolonged expiratory time

Allow hypercapnia

(Till pH as low as 7.15 and a Pa CO2 of up to 80 mmHg)

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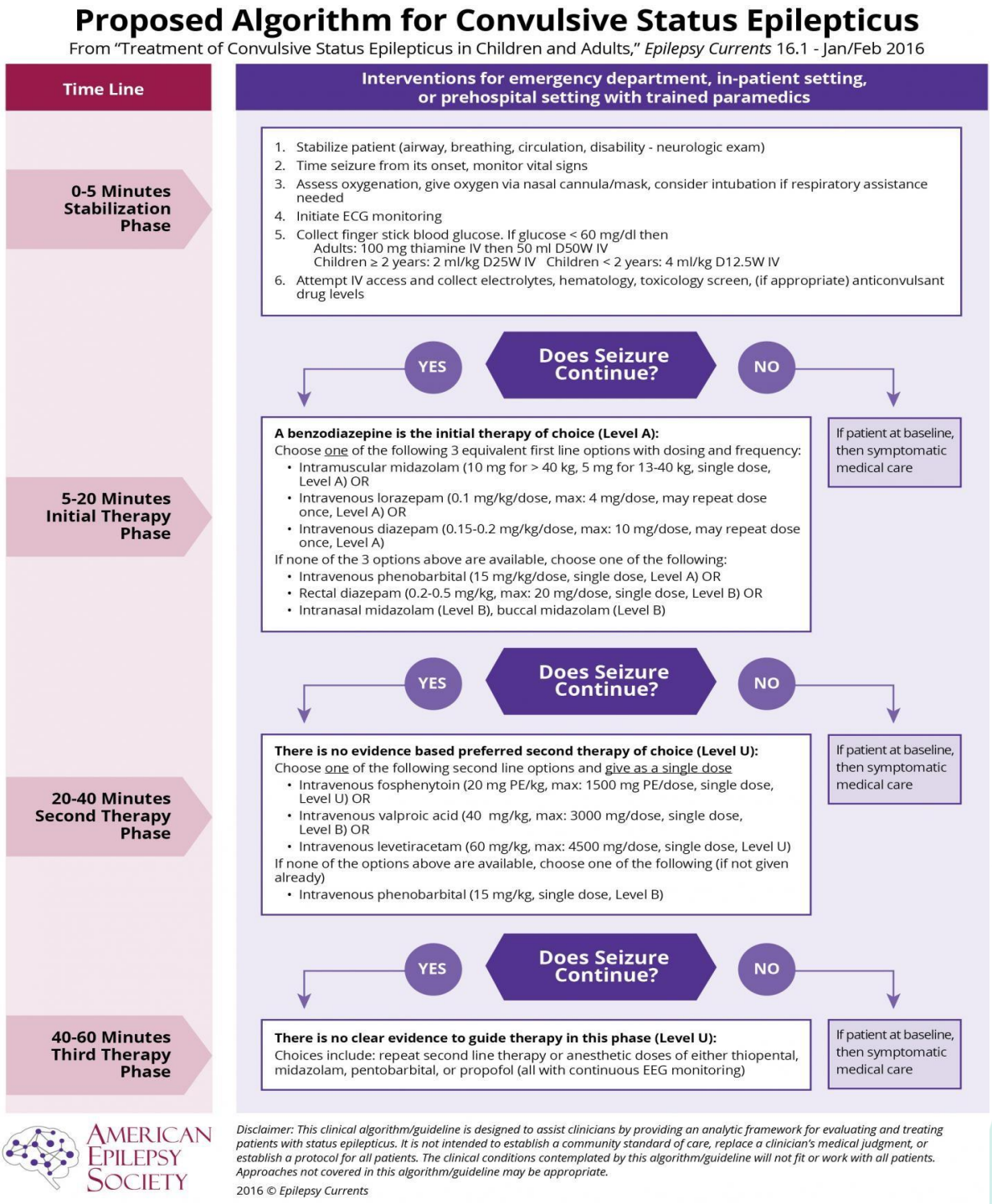
Status Epilepticus

Definition:

5 min or more of (i) continuous clinical and/or electrographic seizure activity **or** (ii) recurrent seizure activity without recovery (returning to baseline) between seizures.



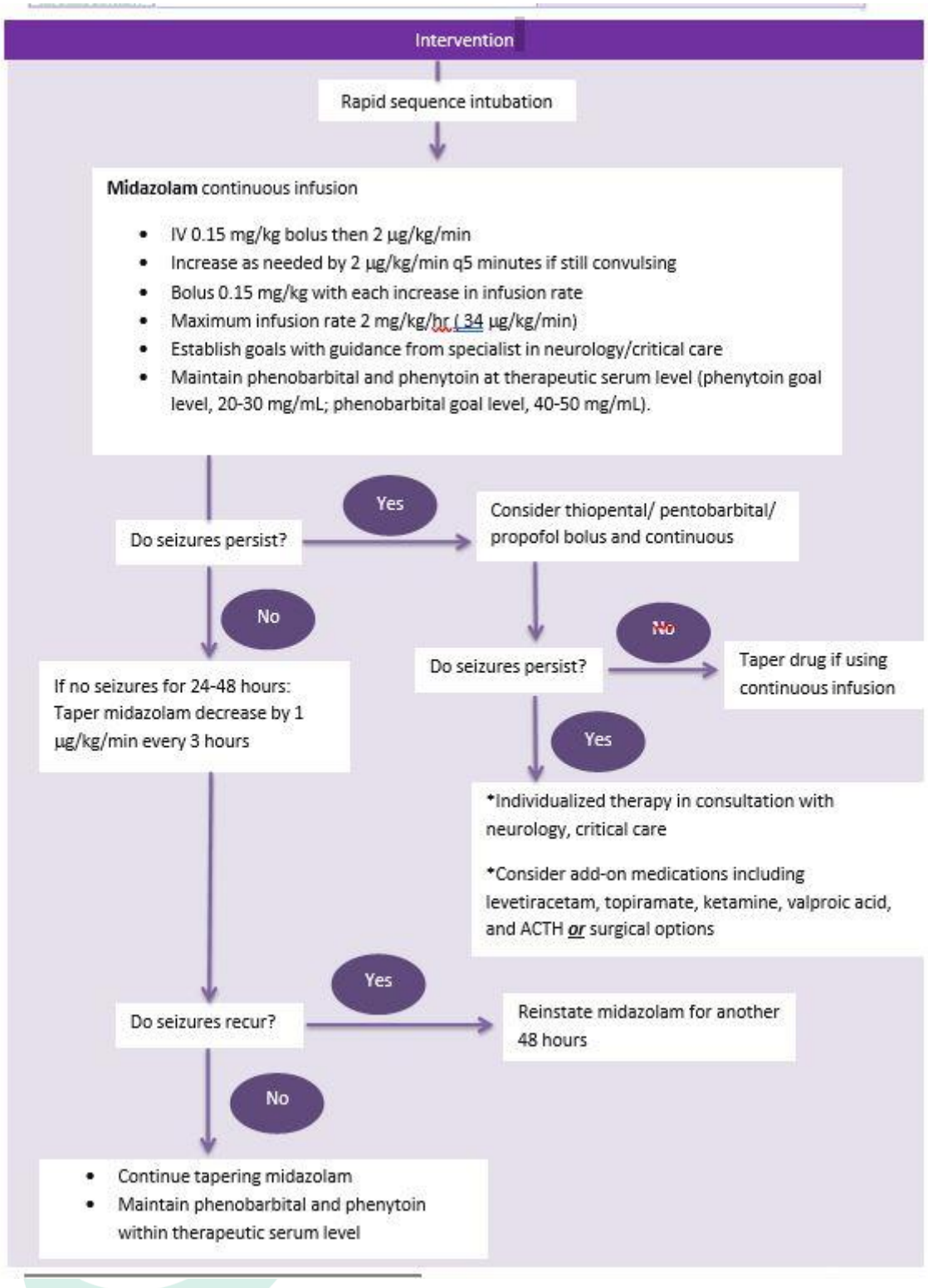

Intervention
Rapid sequence intubation
Midazolam continuous infusion
IV 0.15 mg/kg bolus then 2 µg/kg/min
Increase as needed by 2 µg/kg/min q5 minutes if still convulsing
Bolus 0.15 mg/kg with each increase in infusion rate
Maximum infusion rate 2 mg/kg/hr (34 µg/kg/min)
Establish goals with guidance from specialist in neurology/critical care
Maintain phenobarbital and phenytoin at therapeutic serum level (phenytoin goal level, 20-30 mg/mL; phenobarbital goal level, 40-50 mg/mL).
Do seizures persist?
Yes
Consider thiopental/ pentobarbital/ propofol bolus and continuous
No
If no seizures for 24-48 hours: Taper midazolam decrease by 1 µg/kg/min every 3 hours
Do seizures persist?
No
Taper drug if using continuous infusion
Yes
*Individualized therapy in consultation with neurology, critical care
*Consider add-on medications including levetiracetam, topiramate, ketamine, valproic acid, and ACTH or surgical options
Do seizures recur?
Yes
Reinstate midazolam for another 48 hours
No
Continue tapering midazolam
Maintain phenobarbital and phenytoin within therapeutic serum level

Table (6): Intermittent drug dosing in SE *(Brophy et al., 2012)*

Drug	Initial dosing	Administration rates and alternative dosing recommendations	Serious adverse effects	Considerations
Diazepam	0.15 mg/kg IV up to 10 mg per dose, may repeat in 5 min	Up to 5 mg/min (IVP) Peds: 2–5 years, 0.5 mg/kg (PR); 6–11 years, 0.3 mg/kg (PR); greater than 12 years, 0.2 mg/kg (PR)	Hypotension Respiratory depression	Rapid redistribution (short duration), active metabolite, IV contains propylene glycol
Lorazepam	0.1 mg/kg IV up to 4 mg per dose, may repeat in 5–10 min	Up to 2 mg/min (IVP)	Hypotension Respiratory depression	Dilute 1:1 with saline IV contains propylene glycol
Midazolam	0.2 mg/kg IM up to maximum of 10 mg	Peds: 10 mg IM (>40 kg); 5 mg IM (13–40 kg); 0.2 mg/kg (intranasal); 0.5 mg/kg (buccal)	Respiratory depression Hypotension	Active metabolite, renal elimination, rapid redistribution (short duration)
Fosphenytoin	20 mg PE/kg IV, may give additional 5 mg/kg	Up to 150 mg PE/min; may give additional dose 10 min after loading infusion Peds: up to 3 mg/kg/min	Hypotension Arrhythmias	Compatible in saline, dextrose, and lactated ringers solutions
Lacosamide	200–400 mg IV	200 mg IV over 15 min No pediatric dosing established	PR prolongation Hypotension	Minimal drug interactions Limited experience in treatment of SE
Levetiracetam	1,000–3,000 mg IV Peds: 20–60 mg/kg IV	2–5 mg/kg/min IV		Minimal drug interactions Not hepatically metabolized
Phenobarbital	20 mg/kg IV, may give an additional 5–10 mg/kg	50–100 mg/min IV, may give additional dose 10 min after loading infusion	Hypotension Respiratory depression	IV contains propylene glycol
Phenytoin	20 mg/kg IV, may give an additional 5–10 mg/kg	Up to 50 mg/min IV; may give additional dose 10 min after loading infusion Peds: up to 1 mg/kg/min	Arrhythmias Hypotension Purple glove syndrome	Only compatible in saline IV contains propylene glycol
Topiramate	200–400 mg NG/PO	300–1,600 mg/day orally (divided 2–4 times daily) No pediatric dosing established	Metabolic acidosis	No IV formulation available
Valproate sodium	20–40 mg/kg IV, may give an additional 20 mg/kg	3–6 mg/kg/min, may give additional dose 10 min after loading infusion Peds: 1.5–3 mg/kg/min	Hyperammonemia Pancreatitis Thrombocytopenia Hepatotoxicity	Use with caution in patients with traumatic head injury; may be a preferred agent in patients with glioblastoma multiforme

IM intramuscular; *IV* intravenous; *IVP* intravenous push; *min* minute; *NG* nasogastric; *PE* phenytoin equivalents; *PEDs* pediatric; *PO* by mouth; *PR* rectal administration; *PRIS* propofol related infusion syndrome

Table (7): Pharmacological and non-pharmacological therapies for the treatment of RSE/SRSE *(A. Vasquez, et al., 2018)*

	Mechanism of action	dose	Adverse events	Clinical considerations	Level of evidence
Pharmacological therapies					
Benzodiazepines Midazolam	Positive allosteric modulation of GABA-A receptors, Increases frequency of Cl channel opening	Loading dose: 0.2 mg/kg; administer at an infusion rate of 2 mg/min Infusion rate: 0.05- 2 mg/kg/h Breakthrough SE: 0.1-0.2 mg/kg bolus, increase rate by 0.05-0.1 mg/kg/h. every 3-4 h	Hypotension, respiratory depression	Prolonged use may cause tachyphylaxis and drug accumulation	Class IIA, Level B Class IV
IV anesthetic agents Barbiturates Pentobarbital	Activation of GABA Receptors increase mean Cl channel opening duration, inhibition of NMDA receptors, alteration in conductance of Cl, K+ , Ca2+ ion channels. Same as Pentobarbital	Loading dose: 5- 15 mg/kg; infusion rate ≤ 50 mg/min Infusion rate: 0.5- 5 mg/kg/h Breakthrough SE: 5 mg/kg bolus, increase rate by 0.5- 1 mg/kg/h. every 12 h	Hypotension, cardiac and respiratory depression, paralytic ileus, infection	Long half-life (15-50 h) Requires mechanical ventilation. Can exacerbate porphyria Hepatic enzyme inducer Drug accumulation with prolonged use	Class IIB, Level B Class IV

	Mechanism of action	dose	Adverse events	Clinical considerations	Level of evidence
Pharmacological therapies					
Thiopental	Same as the mechanism described Above	2-7 mg/kg, infusion rate ≤ 50 mg/min Infusion/ maintenance rate: 0.5-5 mg/kg/h Breakthrough SE:1- 2 mg/kg bolus, titrate by 0.5-1 mg/ kg/h. every 12 h	Hypotension, cardiac and respiratory depression	Requires mechanical ventilation, titrate infusion rates to EEG burst-suppression	Class IV
Propofol	Chloride channel conductance, enhances GABA-A receptor	Initial loading dose: 1-2 mg/kg Initial infusion rate 20 mcg/kg/min titrated by 5-10 mcg/kg/min Use with caution with doses > 65 mcg/kg/min Breakthrough SE: Increase infusion rate by 5-10 mcg/ kg/min every 5 min	PRIS, hypotension, cardiac and respiratory depression	Requires mechanical ventilation Prolonged infusion of propofol is a relative contraindication in children (due to risk of PRIS) and in patients with metabolic acidosis, mitochondrial disorders or hypertriglyceridemia Reduces ICP Caution with concomitant use of steroid or catecholamine therapy	Class IIB, Level B Class IV

	Mechanism of action	Dose	Adverse effects	Clinical considerations	Level of evidence
Ketamine	Noncompetitive NMDA glutamate receptor antagonist reduces neuronal excitability	0.5-3 mg/kg Infusion rate: 1-10 mg/kg/h	Tachycardia, hypertension, ICP elevation	Relative contraindication in patients with ICP. Ketamine is an enzyme inducer and inhibitor (CYP2C9)	Class IV
Inhalational Anesthesia Isoflurane	Enhancement of GABA-A receptors, noncompetitive antagonist of NMDA receptor	Concentration 1-5% Titrate to achieve burst-suppression on EEG	Hypotension requiring use of vasopressors, atelectasis, paralytic ileus, infection, deep vein thrombosis	High seizure recurrence rate	Class IV
Immunomodulatory therapy IVIG	Alteration of IgG- specific receptors (FcgR) expression and function (decreases cytokine production), attenuation of complement mediated cell damage	1-2 g/kg divided over 3-5 days	Hypersensitivity reactions, transfusion related acute lung injury, thromboembolic events, renal dysfunction with concentrated solutions, aseptic meningitis	Immunomodulatory therapies may be considered in patients with cryptogenic, autoimmune etiologies of RSE/SRSE.	Class IV
Corticosteroids: Methyl Prednisolone	Inhibition of inflammation associated proteins (e.g. cytokines, chemokines) and immunosuppressive action	1 g/day for 3-5 days	Glucose intolerance, psychiatric disturbances, altered immune function, adrenal suppression		Class IV

	Mechanism of action	Dose	Adverse effects	Clinical considerations	Level of evidence
Non-pharmacological alternatives					
Ketogenic diet	Ketosis mediated decreased glycolysis, increase in free and polyunsaturated fattyacids, anti-inflammatory action, stabilization of neuronal membrane	4:1 (ratio fat to carbohydrates and proteins)	Hypoglycemia, hyperlipidemia, weight loss, acute pancreatitis, metabolic acidosis	Contraindicated inpyruvate carboxylase deficiency, disorders of fatty acid oxidation and metabolism, orporphyria	Class IV
Hypothermia	Reduction of Na+ exchange, decreased K+ conductance, regulation of glutamatergic synaptic transmission, disruption of synchronized discharges	32-35C x 24h Rewarming ≤ 0.5 C/ h	Deep venous thrombosis, infections, cardiac arrhythmias, electrolyte disturbances, acute intestinal ischemia, coagulation disorders	Requires EEG monitoring	Class IV
Electroconvulsive Therapy	Enhancement of GABA neurotransmission, increase of seizure threshold and reduction of neural metabolic activity	Variable protocols	May induce seizures and non-convulsive SE after treatment, amnesia, headache, cognitive impairment	Relative contraindication in patients with cardiovascular conditions Requires EEG monitoring	Class IV
Vagus nerve Stimulation	Modulation of the locus coeruleus, thalamus and limbic circuit through noradrenergic and serotoninergic projections, elevation of GABA levels in brainstem	Surgical implantation	Hoarseness, surgical infection, rarely asystole or bradycardia		Class IV

N.B:

- **If patient is less than 18 months give pyridoxine 100 mg i.v**
- **As regards thiopental infusion:**
  - If thiopental infusion is started stop midazolam infusion
  - Increase thiopental infusion by 1mg/kg/hour every 30 min and give2mg/kg bolus as needed if seizures is occurred on infusion
  - If convulsion is controlled Taper thiopental infusion after 24- 48hours by 25% decrease every 12 hours
- **As regard propofol infusion:**
  - When seizures have been controlled for 12 hours, the drug dosage should be slowly reduced over a further 12 hours. If seizures recur, the drug infusion should be given again for another 12 hours, and then withdrawal attempted again. This cycle may need to be repeated every 24 hours until seizure control is achieved.
- **When to do EEG:**
  - After clinical control of convulsion to diagnose subclinical status epilepticus
  - After midazolam or thiopental or propofol infusion to document burst suppression

Acknowledgment:

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Diabetic ketoacidosis

Definition:

Diabetic Ketoacidosis is one of two serious, acute life-threatening complications of Type I diabetes mellitus (IDDM), or Type II, insulin insufficient diabetes mellitus, the other being severe hypoglycemia.

The biochemical criteria:

Blood glucose	> 200 mg/dL
Venous pH	< 7.3
bicarbonate	< 15 mmol/L
Ketonemia or Ketonuria	

The severity of DKA is categorized by the degree of acidosis

Degree	pH	HCO3
Mild	< 7.3	<15 mmol/L
Moderate	< 7.2	<10 mmol/L
Severe	< 7.1	<5 mmol/L

Clinical Signs:

1. Dehydration (which may be difficult to detect)
2. Tachycardia
3. Tachypnea (which may be mistaken for pneumonia or asthma)
4. Deep, sighing (Kussmaul) respiration; breath has the smell of acetone
5. Nausea, vomiting (which may be mistaken for gastroenteritis)
6. Abdominal pain that may mimic an acute abdominal condition
7. Confusion, drowsiness, progressive reduction in level of consciousness and, eventually, loss of consciousness.

Management:

Acute management should follow the general guidelines for PALS with particular attention to the following aspects for the child who presents in DKA.

1. Immediately measure BG and urine ketone concentrations with bedside meters.
2. Perform a clinical evaluation to identify a possible infection
3. Weigh the patient
4. Assess severity of dehydration:

5% dehydration	Prolonged capillary refill time (normal capillary refill is ≤1.5–2 s) bnormal skin turgor (‘tenting’ or inelastic skin) bnormal respiratory pattern (hyperpnea). ry mucus membranes, sunken eyes, absent tears, weak pulses, and cool extremities.
≥10% dehydration	weak or impalpable peripheral pulses hypotension oliguria

5. Assess level of consciousness using Glasgow coma scale (GCS).
6. Additional measures is done of unconscious patient Secure the airway and empty the stomach by continuous nasogastric suction to prevent pulmonary aspiration
7. Give oxygen to patients with severe circulatory impairment or shock.
8. A cardiac monitor should be used for continuous electrocardiographic monitoring to assess T waves for evidence of hyper- or hypokalemia
9. Obtain a blood sample for laboratory measurement of:

- ✓ Serum or plasma glucose
- ✓ Electrolytes (including Na, K)
- ✓ Blood urea nitrogen, creatinine
- ✓ Serum osmolality
- ✓ Venous pH, pCO2

- ✓ Complete blood count. Note that an increased white blood cell count in response to stress is characteristic of DKA and is not indicative of infection.
- ✓ Albumin, calcium, phosphorus, magnesium concentrations.
- ✓ Urine analysis
- ✓ Cultures (blood, urinary, sputum) only if evidence of infection
- ✓ HbA1c to assess duration of hyperglycemia
- ✓ ECG is done if serum measurement of K is delayed

10. Give antibiotics to febrile patients after obtaining appropriate cultures of body fluids

11. Catheterization of the bladder usually is not necessary, but if the child is unconscious or unable to void on demand (e.g., infants and very ill young children) the bladder should be catheterized

Goals of therapy:

a. Correct dehydration

b. Correct acidosis and reverse ketosis

c. Restore BG to near normal

d. Monitor for complications of DKA and its treatment

e. Identify and treat any precipitating event

Calculations:

Anion gap = Na − (Cl + HCO3): normal is 12 ± 2 mmol/L.

- In DKA, the anion gap is typically 20–30 mmol/L; an anion gap >35 mmol/L suggests concomitant lactic acidosis.

Corrected sodium = measured Na + 2 [(plasma glucose − 100)/100] mg/dL. Patients with DKA are liable for hyponatremia due to:

Glucose largely restricted to the extracellular space, causes osmotic movement of water into the extracellular space thereby causing dilutional hyponatremia

the low sodium content of the elevated lipid fraction of the serum in DKA

1-Fluid therapy:

Fluid replacement should begin before starting insulin therapy.

I. Antishock therapy:

It is given as required, to restore peripheral circulation.

- Patients not in shock but with volume depletion 0: 20 ml /kg over 1: 2 hours may need to be repeated until tissue perfusion is adequate.
- Patient with DKA in shock: rapidly restore circulatory volume with isotonic saline in 20 mL/kg boluses infused as quickly as possible through a large bore cannula with reassessment after each bolus.

II. Deficit therapy:

- ✓ In moderate DKA 5- 7% (50:70 ml/ kg)
- ✓ In severe DKA 7-10% (70:100 ml/kg)

- Calculate the subsequent rate of fluid administration, including the provision of maintenance fluid requirements, aiming to replace the estimated fluid deficit evenly over 48 h.

- Maintenance fluid: to be calculated based on body weight.
- 100cc/kg for the first 10kg, 50cc/kg for the next 10kg, and 20 cc/kg thereafter

“The rate of fluid administration should seldom exceed 1.5–2 times the usual daily maintenance requirement.”

Table 1: showing maintenance volumes, also after subtraction of initial boluses given for the patient assuming it was 10-20 ml/

Body weight, kg	Maintenance mL/24 hr	DKA: give maintenance+ 5% of body weight/24 hr	
		mL/24hr	mL/hr
4	325	530	22
5	405	650	27
6	485	790	33
7	570	920	38
8	640	1040	43
9	710	1160	48
10	780	1280	53
11	840	1390	58
12	890	1490	62
13	940	1590	66
14	990	1690	70
15	1030	1780	74
16	1070	1870	78
17	1120	1970	82
18	1150	2050	85
19	1190	2140	89
20	1230	2230	93
22	1300	2400	100
24	1360	2560	107
26	1430	2730	114
28	1490	2890	120
30	1560	3060	128
32	1620	3220	134
34	1680	3360	140
36	1730	3460	144
38	1790	3580	149
40	1850	3700	154
45	1980	3960	165
50	2100	4200	175
55	2210	4420	184
60	2320	4640	193
65	2410	4820	201
70	2500	5000	208
75	2590	5180	216
80	2690	5380	224

Duration of IV fluid therapy:

Divide fluids over remainder of time for replacement: This is calculated based on serum osmolality (mosmol/kg H_2O):

$$\text{Calculate s-osmolality} = Na^{+}_{(meq/l)} \times 2 + \frac{glucose\,(mg/dl)}{18} + \frac{BUN\,(mg/dl}{2.8}$$

- If **s-osmolality 300 - ≤ 320** correct **over 24 hours**
- If **s-osmolality > 320 - < 340** correct **over 36 hours**.
- If s-osmolality **≥ 340 or initial sNa^{+} >150 meq/L** correct **over 48hours.**

2-Insulin therapy:

Insulin therapy: begin with 0.1 U/kg/h in patients above five years

0.05 U/Kg/h in patients below five years

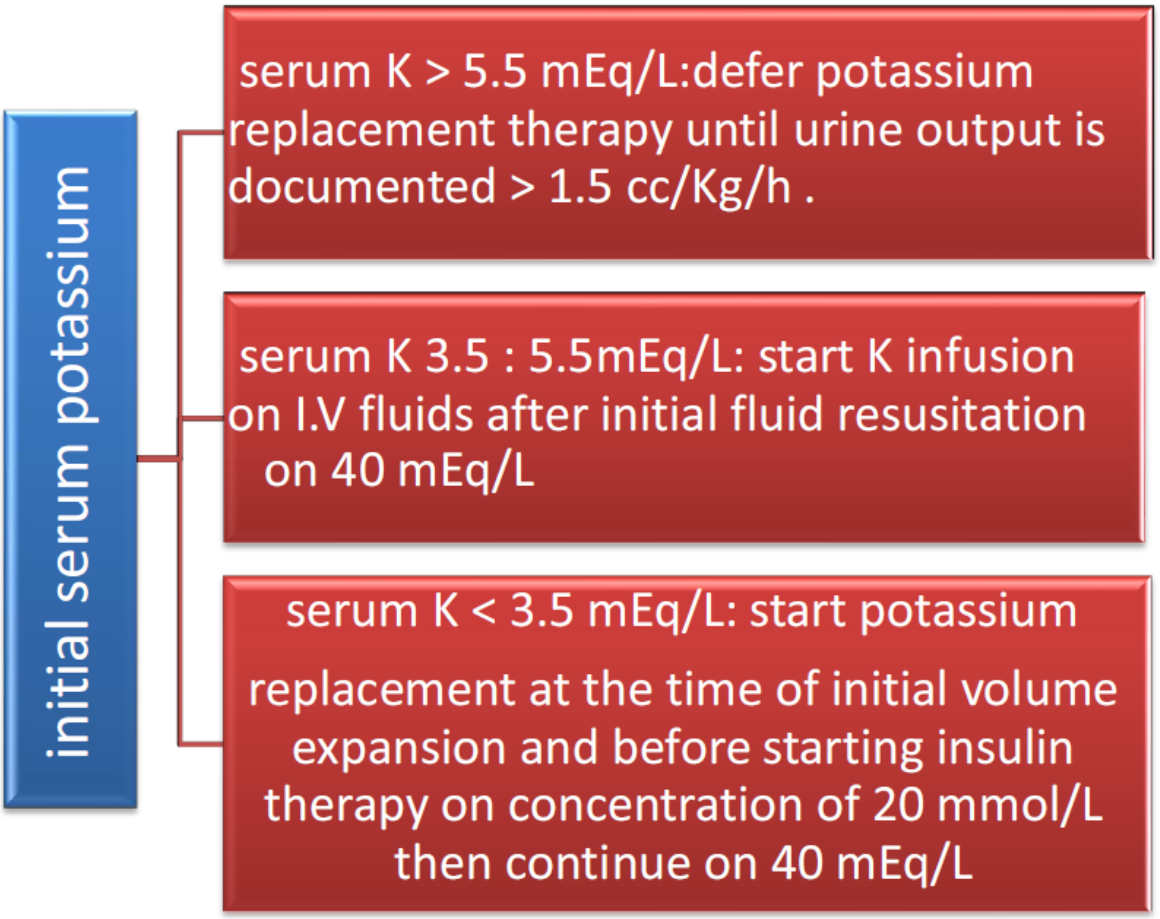
Insulin drip start 1–2 h AFTER starting fluid replacement therapy.

Dilute 50 units regular human insulin in 500 mL normal saline, (0.1 unit insulin =1 mL).

3-Potassium replacement:

If immediate serum potassium measurements are unavailable, an ECG may help to determine whether the child has hyper- or hypokalemia.

Signs of hypokalemia in ECG:	Prolongation of the PR interval T-wave flattening and inversion ST depression, prominent U waves apparent long QT interval (due to fusion of the T and U waves)
Signs of hyperkalemia in ECG:	Tall, peaked, and symmetrical T waves shortening of the QT interval



4. Bicarbonate Administration:

Bicarbonate administration is not recommended except for treatment of life threatening hyperkalemia.

Bicarbonate is NOT recommended and has potential hazards in patients with DKA:

1. HCO3 diffuses slowly through BBB

(HCO3 + H+ CO2 + H2O)
CO2 diffuses rapidly paradoxical CNS acidosis * ↑ risk of cerebral edema.

2. Alkalosis is associated with hypokalemia.
3. May ↑ s-Na+ in a patient with hyperosmolar dehydration.
4. $HCO3^-$ therapy shifts the OxyHb dissociation curve to the left (decreases O2 release to the tissues) ↑ tissue hypoxia.

Indication for bicarbonate therapy in patients with DKA:

(1) A patient with pH < 6.9 who is in shock with decreased cardiac contractility and peripheral vasodilatation with poor tissue perfusion.

(2) Patients with LIFE THREATENING Hyperkalemia.

In these cases only Give NaHCO3 1-2 meq/kg or 80 meq/m2 body surface area added to 0.45% saline over 1 hour (never by bolus).

Be careful about s-K+ and DO NOT stop K+ infusion while bicarbonate is being given.

Reassess after 1 hour of finishing bicarbonate infusion.

Rate of I.V fluid administration:

Rate of I.V fluid and insulin drips depend on RBS and rate of decent of RBS/ Hour which should not exceed 90 mg/dL.

RBS level	Type of I.V fluid	Rate of insulin drip
> 300 mg/dL	Saline 0.9 %	> 5 years 0.1 IU/kg/h < 5 years 0.05 IU/kg/h
140 :300 mg/dL **Or rate of decrease** **> 90 mg/dL**	Saline 0.9%: glu 5%	Same rate
80: 140 mg/dL **Or rate of decrease** **> 90 mg/dL**	Saline 0.9%: glu 10%	Same rate
> 80 mg/dL **Or rate of decrease** **> 90 mg/dL**	Saline 0.9%: glu 12.5%	Same rate
> 80 mg/dL **Or rate of decrease** **> 90 mg/dL**	Saline 0.9%: glu 12.5%	Decrease to half rate

If rate of blood sugar decrease less than 30 mg/dL or acidosis is not corrected so you should revaluate:

1. IV fluid calculations
2. Insulin delivery system & dose
3. Need for additional resuscitation
4. Consider sepsis

Clinical and biochemical monitoring

There should be documentation on a flow chart of hour-by-hour clinical observations, IV and oral medications, fluids, and laboratory results. Monitoring should include the
Following:

- Hourly (or more frequently as indicated) vital signs (heart rate, respiratory rate, blood pressure).

- Hourly (or more frequently as indicated) neurological observations (GCS) for warning signs and symptoms of cerebral edema that include:

1) Headache
2) Inappropriate slowing of heart rate
3) Recurrence of vomiting
4) Change in neurological status (restlessness, irritability, increased drowsiness, incontinence)
5) Specific neurologic signs (e.g., cranial nerve palsies, abnormal pupillary responses)
6) Rising blood pressure
7) Decreased oxygen saturation
8) Rapidly increasing serum sodium concentration suggesting loss of urinary free water as a manifestation of diabetes insipidus (from interruption of blood flow to the pituitary gland due to cerebral herniation)
9) Failure of measured serum sodium levels to rise or a further decline in serum sodium levels with therapy is thought to be a potentially ominous sign of impending cerebral edema. Too rapid and ongoing rise in serum sodium concentration may also indicate possible cerebral edema as a result of loss of free water in the urine from diabetes insipidus.

- Amount of administered insulin.
- Hourly (or more frequently as indicated) accurate fluid input (including all oral
- fluid) and output.
- Capillary blood glucose concentration should be measured hourly (but must be cross-checked against laboratory venous glucose, as capillary methods may be inaccurate in the presence of poor peripheral circulation and acidosis).
- Laboratory tests: serum electrolytes, glucose, blood urea nitrogen, calcium, magnesium, phosphorus, hematocrit, and blood gases should be repeated 2–4 h, or more frequently, as clinically indicated, in more severe cases.

Where Should the Patient be treated? Indications for ICU admission:

1. Patients with severe DKA: (pH < 7.1, shock, or with long duration of symptoms).
2. Patients with altered level of consciousness.
3. DKA in children below 5 years (are at increased risk of cerebral edema).
4. Patients with high BUN, possible oliguria & acute tubular necrosis (for need of a central venous catheter & dialysis).
5.If cerebral edema develops as a complication of treatment.

Introduction of oral fluids and shift to S.C. insulin

- Can introduce oral fluids after substantial clinical improvement (mild acidosis/ketosis may still be present).
- Plan to change to SC insulin when ketoacidosis has resolved (pH > 7.3, HCO3> 15, anion gap is normal) + oral fluids are tolerated.
- Best is to shift before a meal time.
- Start S.C. intermediate acting + short acting insulin.

To prevent rebound hyperglycemia, the first SC injection should be given 15–30 min (with rapid acting insulin) or 1–2 h (with regular insulin) before stopping the insulin infusion to allow sufficient time for the insulin to be absorbed. With intermediate or long-acting insulin, the overlap should be longer and the rate of IV insulin infusion gradually lowered.

Calculate insulin dose as:

0.7 U/kg/d in prepubertal children with long standing DM (may need IU/kg/d in new cases).

1.0 U/kg/d at mid puberty.

1.2 U/kg/d by the end of puberty.

Give the first dose of rapid-acting insulin analogue 15 minutes before stopping insulin infusion and of regular insulin 1 hour before stopping it.

Cerebral edema:

The incidence of clinically overt cerebral edema in national population studies is 0.5–0.9% and the mortality rate is 21–24%

Diagnosis is established by:

1. One diagnostic criterion ***or***
2. two major criteria ***or***
3. one major and two minor criteria

These have a sensitivity of 92% and a false positive rate of only 4%. Signs that occur before treatment should not be considered in the diagnosis of cerebral edema.

Diagnostic criteria	*Major criteria*	*Minor criteria*
• Abnormal motor or verbal response to pain • Decorticate or decerebrate posture • Cranial nerve palsy (especially III, IV, and VI) • Abnormal neurogenic respiratory pattern (e.g., grunting, tachypnea, Cheyne–Stokes respiration, apneusis)	• Altered mentation/fluctuating level of consciousness • Sustained heart rate deceleration (decrease more than 20 beats/min) not attributable to improved intravascular volume or sleep state • Age-inappropriate incontinence	• Vomiting • Headache • Lethargy or not easily arousable • Diastolic blood pressure >90mmHg • Age <5 yr

Treatment of cerebral edema:

Initiate treatment as soon as the condition is suspected.

I. Reduce the rate of fluid administration by one-third.

II. Give mannitol, 0.5–1 g/kg IV over 10–15 min, and repeat if there is no initial response in 30 min to 2 h.

III. Hypertonic saline (3%), suggested dose 2.5–5 mL/kg over 10–15 min, may be used as an alternative to mannitol, especially if there is no initial response to mannitol

IV. Hyperosmolar agents should be readily available at the bedside.

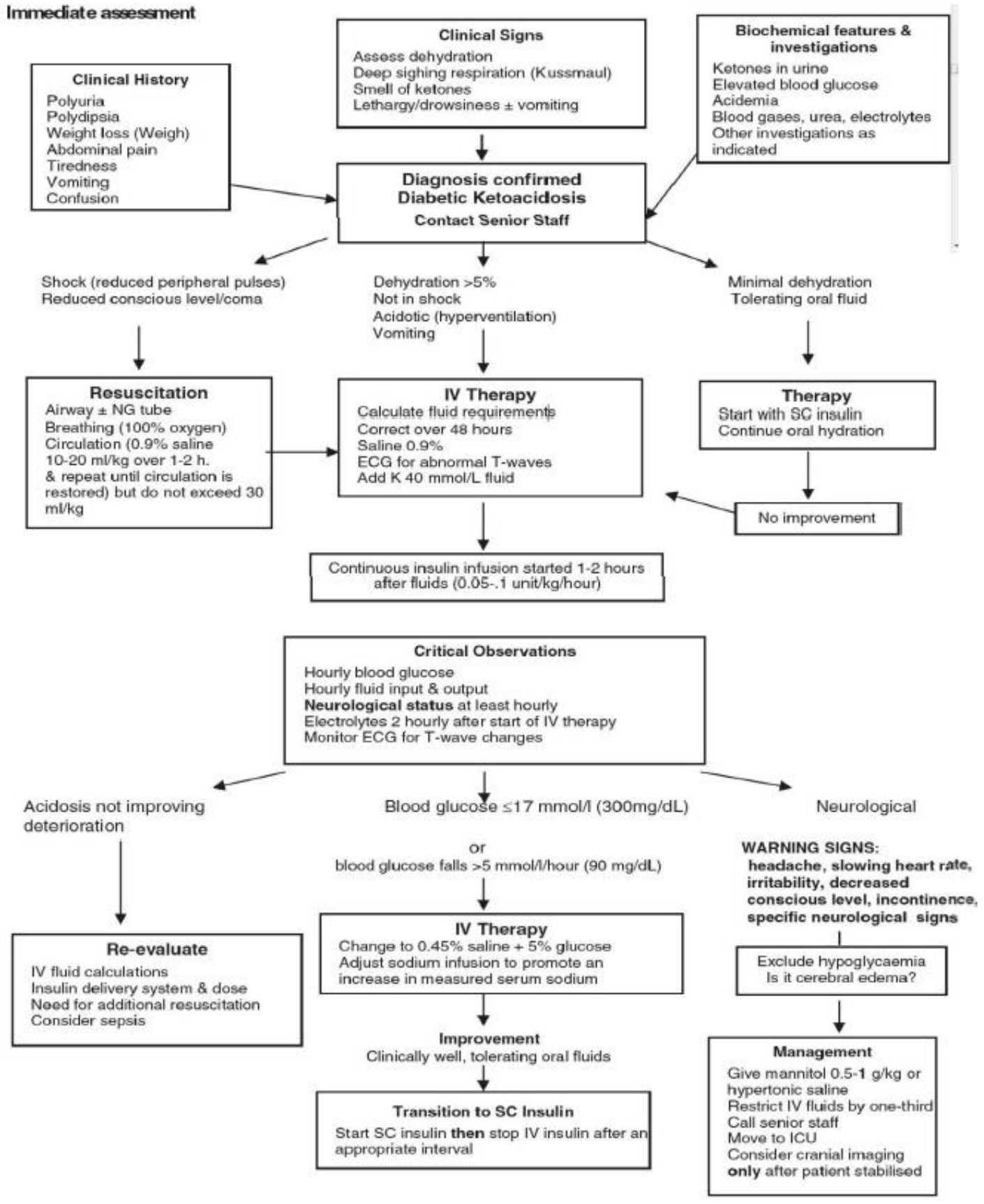
V. Elevate the head of the bed to 30.◦

VI. Intubation may be necessary for the patient with impending respiratory failure.

VII. After treatment for cerebral edema has been started cranial imaging may be considered as with any critically ill patient with encephalopathy or acute focal neurologic deficit. The primary concern is whether the patient has a lesion requiring emergency neurosurgery (e.g., intracranial hemorrhage) or a lesion that may necessitate anticoagulation (e.g cerebrovascular thrombosis)

References:

- **ISPAD Clinical Practice Consensus Guidelines 2018 Compendium**
- **Protocol of Management of Type 1 Diabetes Mellitus, the Diabetes Clinic Children's Hospital - Ain Shams Universit**


Immediate assessment
Clinical History
Polyuria
Polydipsia
Weight loss (Weigh)
Abdominal pain
Tiredness
Vomiting
Confusion
Clinical Signs
Assess dehydration
Deep sighing respiration (Kussmaul)
Smell of ketones
Lethargy/drowsiness ± vomiting
Biochemical features & investigations
Ketones in urine
Elevated blood glucose
Acidemia
Blood gases, urea, electrolytes
Other investigations as indicated
Diagnosis confirmed
Diabetic Ketoacidosis
Contact Senior Staff
Shock (reduced peripheral pulses)
Reduced conscious level/coma
Dehydration >5%
Not in shock
Acidotic (hyperventilation)
Vomiting
Minimal dehydration
Tolerating oral fluid
Resuscitation
Airway ± NG tube
Breathing (100% oxygen)
Circulation (0.9% saline
10-20 ml/kg over 1-2 h.
& repeat until circulation is restored) but do not exceed 30 ml/kg
IV Therapy
Calculate fluid requirements
Correct over 48 hours
Saline 0.9%
ECG for abnormal T-waves
Add K 40 mmol/L fluid
Therapy
Start with SC insulin
Continue oral hydration
No improvement
Continuous insulin infusion started 1-2 hours after fluids (0.05-.1 unit/kg/hour)
Critical Observations
Hourly blood glucose
Hourly fluid input & output
Neurological status at least hourly
Electrolytes 2 hourly after start of IV therapy
Monitor ECG for T-wave changes
Acidosis not improving deterioration
Blood glucose ≤17 mmol/l (300mg/dL)
or
blood glucose falls >5 mmol/l/hour (90 mg/dL)
Neurological
WARNING SIGNS:
headache, slowing heart rate, irritability, decreased conscious level, incontinence, specific neurological signs
Re-evaluate
IV fluid calculations
Insulin delivery system & dose
Need for additional resuscitation
Consider sepsis
IV Therapy
Change to 0.45% saline + 5% glucose
Adjust sodium infusion to promote an increase in measured serum sodium
Exclude hypoglycaemia
Is it cerebral edema?
Improvement
Clinically well, tolerating oral fluids
Transition to SC Insulin
Start SC insulin then stop IV insulin after an appropriate interval
Management
Give mannitol 0.5-1 g/kg or hypertonic saline
Restrict IV fluids by one-third
Call senior staff
Move to ICU
Consider cranial imaging only after patient stabilised

Hypoglycemia

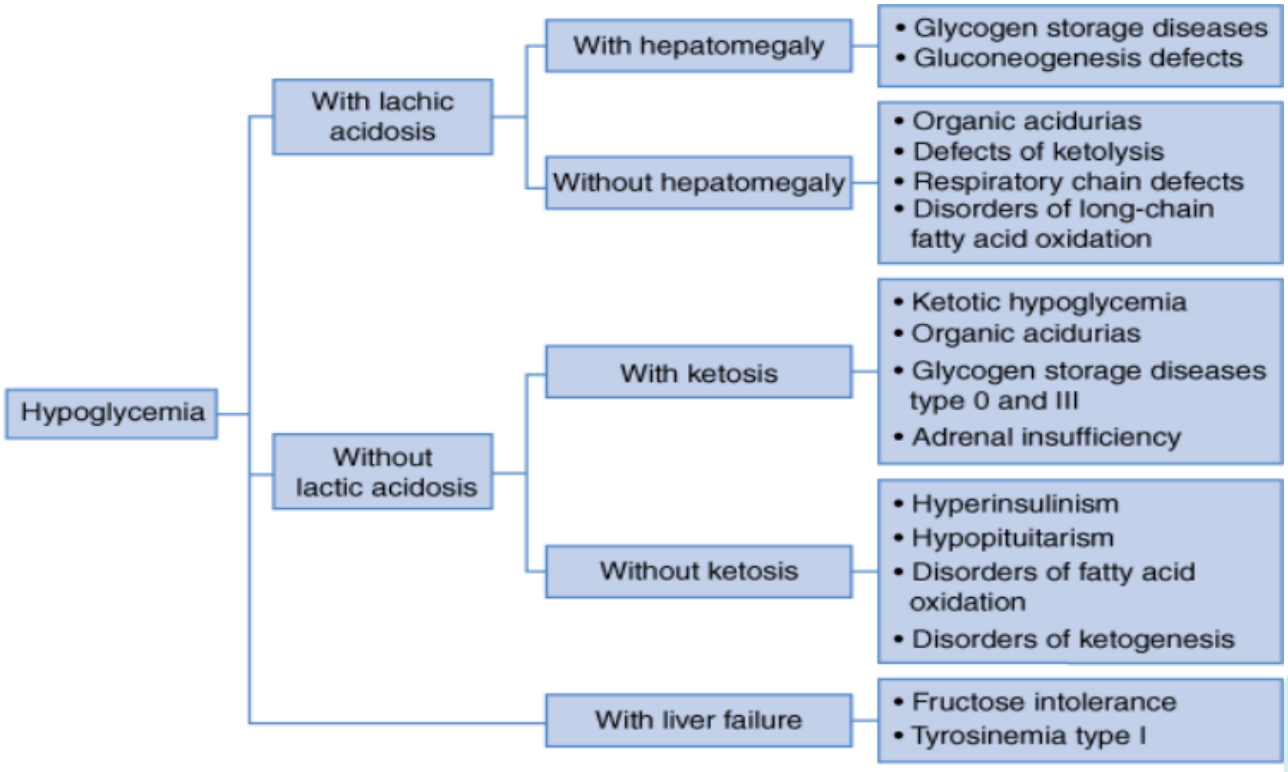
Diagnosis:

In addition to the measured glucose concentration thresholds listed below, symptomatic hypoglycemia is defined by the presence of clinical signs such as:

- Tachycardia
- Sweating
- Altered level of consciousness (agitation, lethargy, or seizures)

Age	Consensus Definition of Hypoglycemia
Preterm neonates **Term neonates**	Less than 45 mg/dL
Infants **Children** **Adolescents**	Less than 60 mg/dL

Approach for Diagnosis



Source: Lowry AW, Bhakta KY, Nag PK: *Texas Children's Hospital Handbook of Pediatrics and Neonatology*: www.accesspediatrics.com

Management of Hypoglycemia:

A. Emergency Treatment:

If...	Intervene by Administering...
The glucose concentration is low in a responsive child	Oral glucose (eg, juice or other glucose-containing fluid) as long as the child is not in shock
The glucose concentration is low and the child is unresponsive or in shock	IV/IO dextrose (dextrose is the same as glucose)

If hypoglycemia is treated with IV dextrose (0.5 to 1 g/kg), administer one of the following:

- ✓ D25W (25% dextrose in water): 2 to 4 mL/kg
- ✓ D10W (10% dextrose in water): 5 to 10 mL/kg

B. Continuous Treatment for persistent Hypoglycemia:

Agent	Dosing	Administration	Side Effects[a]
Dextrose	4-8 mg/kg/min[5] (max: 20-30 mg/kg/min[20])	Continuous infusion	
Diazoxide	10-15 mg/kg/day [25,36-38]	Orally (once every 8 hr)	Hirsutism, heart failure[39,40] fluid retention, nausea, vomiting
Glucagon	Bolus: 200 mcg/kg[25] 1 mg/day[26,27]	Intermittent infusion Continuous infusion	Hyponatremia[28], thrombocytopenia[28]
Glucocorticoids			Growth suppression, hypertension
Dexamethasone	0.25 m/kg[28] 1-2.5 mg/kg/dose[30,31]	Intravenous (once every 12 hr) Intravenous (once every 6 hr)	
Hydrocortisone	50 mg/m^2/day		
Nifedipine	Initial: 0.25-0.3 mg/kg/day Final: 0.5-0.8 mg/kg/day[24,47-48]	Orally (once every 8 hr)	None reported
Octreotide	7-12 mcg/kg/day[42] (max: 40 mcg/kg/day[42-44])	Subcutaneous (every 4-6 hr) May be given continuously IV	Cholelithiasis[43]

hr, hours; IV, intravenous; max, maximum

[a]Adverse events listed are not all inclusive. Those reported have been described in patients receiving these agents for the treatment of hypoglycemia.

Adrenal Crisis

Definition

it is a physiological event caused by an acute relative insufficiency of adrenal hormones.

Etiology

- May be precipitated by physiological stress in a susceptible patient.
- Should be considered in patients with congenital adrenal hyperplasia.
- Hypopituitarism on replacement therapy.
- Those previously or currently on prolonged steroid therapy.

Assessment:

A) History and physical examination – look for:

- **Glucocorticoid deficiency**:

➢ Weakness, anorexia, nausea and/or vomiting, hypoglycemia, hypotension (particularly postural) and shock.

- **Mineralocorticoid deficiency**:

➢ Dehydration, hyperkaliemia, hyponatremia, acidosis and prerenal renal failure.

B) Investigations

- Immediate blood glucose.
- Serum glucose, urea, sodium and potassium.
- Arterial or capillary acid base.

"Where the underlying diagnosis not known, collect at least 2 ml of clotted blood for later analysis (cortisol and 17 hydroxyprogesterone)."

Management:

Susceptible patients who present with vomiting but who are not otherwise unwell should be considered to have incipient adrenal crisis. To attempt to prevent this from developing further:

- Administer I.M hydrocortisone 2 mg/kg.
- Give oral fluids and observe for 4–6 hours before considering discharge.
- Discuss with appropriate consultant.

For all other children:

1. Give intravenous fluids.

Shock or severe dehydration:

- Normal saline 20 ml/kg I.V. bolus. Repeat until circulation is restored.
- Administer remaining deficit plus maintenance fluid volume as normal saline in
- 5% dextrose evenly over 24 hours.
- Check electrolytes and glucose frequently.
- After the first few hours, if serum sodium is greater than 130 mmol/L, change to half normal saline.
- 10% dextrose may be needed to maintain normoglycaemia.

Moderate dehydration:

- Normal saline 10 ml/kg i.v. bolus. Repeat until circulation is restored.
- Administer remaining deficit plus maintenance fluid volume as normal saline in
- 5% dextrose evenly over 24 hours.

Mild or no dehydration:

- No bolus.
- 1.5 times maintenance fluid volume administered evenly over 24 hours.

2. Give hydrocortisone

Administer hydrocortisone intravenously. If I.V access is difficult, give I.M while establishing intravenous line.

- 10 mg for infants
- 25 mg for toddlers
- 50 mg for older children
- 100 mg for adolescents

Should be administered as a bolus and a similar total amount given in divided doses at 6 hr intervals for the 1st 24 hr. These doses may be reduced during the next 24 hr if progress is satisfactory

When stable reduce I.V. hydrocortisone dose, then switch to triple dose oral hydrocortisone therapy, gradually reducing to maintenance levels (10–15 mg/m2/day).

Equivalent doses (20-25% of the hydrocortisone dose) of prednisone or prednisolone may be divided and given twice daily.

In patients with mineralocorticoid deficiency, start fludrocortisone at maintenance doses (0.05-0.2 mg daily) as soon as the patient is able to tolerate oral fluids.

3. Treat hypoglycemia

- Hypoglycemia is common in infants and small children.
- Treat with I.V. bolus of 5 ml/kg 10% dextrose in a neonate or infant and 2 ml/kg of 25% dextrose in an older child or adolescent.
- Maintenance fluids should contain 5–10% dextrose.

4. Treat hyperkalemia

- Hyperkalemia usually normalizes with fluid and electrolyte replacement.
- If potassium is above 6 mmol/L perform an ECG and apply cardiac monitor as arrhythmias and cardiac arrest may occur.
- If potassium is above 7 mmol/L and hyperkalemic ECG changes are present:
  (eg. peaked T waves, wide QRS complex)

Give:

➢ 10 % calcium gluconate 0.5 ml/kg I.V over 3–5 mins.
➢ Commence infusion of insulin 0.1 units/kg/hr I.V together with an infusion of 50% dextrose 2 ml/kg/hr.

- If the potassium is above 7 mmol/L with a normal ECG :

Give sodium bicarbonate 1–2 mmol/kg I.V over 20 mins, with an infusion of 10% dextrose at 5 ml/kg/hr.

5. Identify and treat potential precipitating causes such as sepsis.

6. Admit to appropriate inpatient facility.

Prevention

Prevention of a crisis if possible, is essential and may involve:

- Anticipating problems in susceptible patients.
- Giving triple normal oral maintenance steroid dose for 2–3 days during stress (eg. fever, fracture, laceration requiring suture).
- Giving intramuscular hydrocortisone when absorption of oral medication is doubtful eg. In vomiting or severe diarrhea.
- Increasing parenteral hydrocortisone (1–2 mg/kg) before anesthesia, with or without an increased dose postoperatively.

Acute kidney Injury (AKI)

Background

AKI =

- A recent **increase of >1.5x in creatinine from a previous baseline** or a value of > **1.5 x upper limit of the reference interval for age.**
- **Usually associated with a fall in urine output <0.5ml/kg/hour for 8 hours.**
- Creatinine results should be interpreted in the context of age, body and muscle mass, and ethnicity.

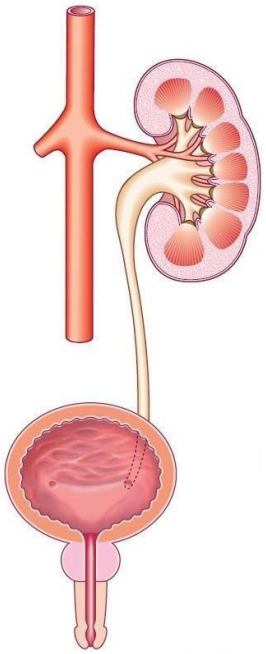
Creatinine Reference Ranges

Neonates (premature)	0.33-0.98 mg/L	29 - 87 µmol/L
Neonates (full-term)	0.31-0.87 mg/L	27 - 77 µmol/L
to 12 months	0.16-0.38 mg/L	14 - 34 µmol/L
1to <3 years	0.17-0.35 mg/L	15 - 31 µmol/L
to <5 years	0.26-0.42 mg/L	23 - 37 µmol/L
5 to <7 years	0.32-0.59 mg/L	28 - 52 µmol/L
7 to <9 years	0.4-0.6 mg/L	35 - 53 µmol/L
9 to <11 years	0.38-0.74 mg/L	34 - 65 µmol/L
11 to <13 years	0.52-0.79 mg/L	46 - 70 µmol/L
13 to <15 years	0.57-0.87 mg/L	50 - 77 µmol/L
15 years and over		
Male:	0.7 – 1.2 mg/L	62 - 106 µmol/L
Female:	0.5 – 0.9 mg/L	44 - 80 µmol/L

AKI Warning Score

AKI stage	Creatinine change from baseline/ upper limit *or* eGFR (mL/min/1.73m^2)	Urine output
1	**>1.5-2x *or* eGFR < 75**	**<0.5mL/kg for 8 hours**
2	**2-3x *or* eGFR < 50**	
3	**>3x *or* eGFR < 35**	

Assessment



Causes of acute kidney injury.
Source : Davidsons Essentials of Medicine,

Causes	Considerations in the history
Pre-renal • Hypovolemia • Impaired Cardiac output • Renal vessel occlusion • Hepatorenal syndrome	• Signs and symptoms of hypovolemia e.g. vomiting or diarrhea, decreased UO, dizziness, lethargy • renal artery stenosis • Past history: biliary atresia, cardiac disease
Intrinsic renal disease • Glomerulonephritis • Involvement of renal microvasculature- HUS, HSP • Interstitial nephritis • Drugs • Acute Tubular necrosis • Tumour lysis syndrome	• Recent viral illness • Change in urine color e.g. red or “coca cola” colored • History of transplant or nephrotoxic drugs
Post-renal or obstructive • Posterior urethral valves • Bilateral ureteric obstruction (trauma, calculi) • Urethral obstruction (trauma, calculus)	• Abdominal pain Reduced UO • History of trauma • History of kidney stones • Frequent UTI’s

GFR Calculation

In children, eGFR is calculated using the following formula, in which:
k = 30 µmol/L for children <1 year
k = 40 µmol/L for children >1 year

$$\text{eGFR (mL/min/1.73m}^2) = \frac{k \times \text{height(cm)}}{\text{Serum creatinine (µmol/L)}}$$

N.B
(1mg/dl = 88 µmol/L)

Management

High risk groups

- Nephro-urological, cardiac, liver disease
- Malignancy, bone marrow transplant
- Dependence on others for access to fluids
- Medication: ACE-I ARB, NSAID, diuretics, calcineurin inhibitors

1. Risk Assessment

- Clinical history including infective symptoms, Input output, Hx of transplant, renal disease, nephrotoxic drugs
- Fluid assessment - peripheral circulation, edema, UO, mucous membranes
- Signs of cardiac failure
- Neurological exam -?electrolyte derangement
- Full set of observations
  - BP (changes are late & measurements in isolation not usually helpful)

High risk scenarios

- History of reduced UO
- Sepsis
- Hypoperfusion or dehydration
- Nephrotoxic drug / toxin exposure
- Renal disease or urinary tract obstruction
- Major surgery

2. Initial management

- Stop nephrotoxic drugs and consider alternatives in acute illness
- Monitor PEWS, weight and Input / output closely
- Check renal function early
- Optimize fluid balance
- Educate parents on risks of dehydration-give quantitative targets (100:50:20 rule)

Urine output <0.5ml/kg/hr for 8 hour OR High risk scenario OR Concerns in high risk group

3. Investigations

→ U&E, FBC, bone profile, blood gas (bicarbonate)

→ Urinalysis, urine microscopy

Subsequent findings

sCr between ULRI and 1.5x ULRI

→ Repeat U&E immediately then as advised by senior clinician.

AKI stage 1

→ If clinically relevant investigations as per AKI 2/3

AKI stage 2 or 3

→ C3/4, ASOT, Immunoglobulins, ANA, ANCA, anti-GBM, CK, LDH, blood film
→ Urinary tract USS
→ CXR and echocardiography
→ Catheterization if obstructed

Pediatric nephrology referral to the Renal Team

- **Immediate referral for any stage AKI where**
  - K^+ >6.5mmol/L
  - Oligo/anuria and plasma Na^+ <125 mmol/L
  - Pulmonary edema of hypertension unresponsive to diuretics
  - Plasma urea >40mmol/L unresponsive to fluid challenge
  - Persistent or worsening metabolic acidosis
- AKI stage 2 or 3 and consider for stage 1
- **Any AKI**
  - in CKD patient or patient with a renal transplant
  - Suspected intrinsic renal disease (e.g. nephritis / HUS)

If any concerns outside of this list, please discuss with ECHL

Fluid management in AKI

Clinical Assessment of Fluid Status

- Fluid assessment- peripheral circulation, edema, urine output
- Signs of cardiac failure- raised JVP, hepatomegaly, peripheral pitting edema, bilateral lung crepitations.
- Blood pressure (changes are late and measurements in isolation not usually helpful)
- Low BP with cool peripheries -> intravascular depletion and shock
- High BP with warm peripheries -> fluid overload

	Hypovolemia	Euvolemia	Overloaded
Initial management	**1. Fluid challenge 10 mL/kg0.9% sodium chloride STAT** **2.** Initial fluid replacement = **insensible losses (400ml/m²/day) + UO (last24h)**	• Fluid challenge 10 mL/kg 0.9% sodium chloride **over 1 hour**	• Furosemide IV infusion 3-5 mg/kg up to 4 times a day (max dose 250mg;max 1g/day) may be considered but this should be a decision **by a senior clinician**
Ongoing management	• Replace UO and insensiblelosses for at least 24-48h ▪ Beware of increased losses in fever, profuseD&V, hyperventilating • Beware of polyuric recoveryphase	Replace ongoing fluid losses (insensible at 400ml/m²/d, urine, GI losses) Insensible losses are higher in febrile children and lower in ventilated children	Fluid restrict to 50-75%of UO

"If renal function continues to improve, set a fluid target.

Ongoing management "Monitor, Maintain, Minimize"

Monitor

Strict and Accurate Input / Output

- ✓ At least **daily weights**
- ✓ Always plot height and weight on a growth chart
- ✓ Ideally at the same time each day, especially for small children
- ✓ **Blood pressure at least four hourly**

Nutrition

- ✓ Children with AKI are in a catabolic state and therefore need monitoring to ensure meeting adequate calorie requirement

Investigations

- ✓ **Bloods:** daily U&E. Management of electrolyte abnormalities especially

Hyperkalemia

- ✓ Urinalysis at least daily

Maintain

- ✓ Ensure adequate circulatory volume – address hypoperfusion urgently with fluid boluses (10 mL/kg) and inotropic support once the volume is restored.

Minimize

- ✓ Further harm should be reduced by stopping nephrotoxic drugs and restarting when appropriate with dose adjustments
- ✓ Intravenous contrast should also be avoided

ABBREVIATIONS	
ACE-I	Angiotensin-Converting Enzyme Inhibitor
AKI	Acute Kidney Injury
ANA	Antinuclear Antibody
ANCA	Antineutrophil Cytoplasmic Antibodies
anti-GBM	Anti–Glomerular Basement Membrane
ARB	Angiotensin II Receptor Blocker
ASOT	Antistreptolysin O Titer
C3/4	Complement 3/4
CK	Creatine Kinase
CKD	Chronic Kidney Disease
D&V	Diarrhea and Vomiting
GI	Gastrointestinal
HSP	Henoch-Schönlein Purpura
HUS	Hemolytic Uremic Syndrome
Hx	History
JVP	Jugular Venous Pulse
LDH	Lactate Dehydrogenase
NSAID	Non-Steroidal Anti-Inflammatory Drugs
PEWS	Pediatric Early Warning Signs
sCr	Serum Creatinine
STAT	To Be Performed Immediately
U & E	Urea and Electrolytes (Sodium “Salt”, Potassium, And Magnesium)
ULRI	Upper Limit Of The Reference Interval
UO	Urine Output
USS	Ultrasound Scan

References

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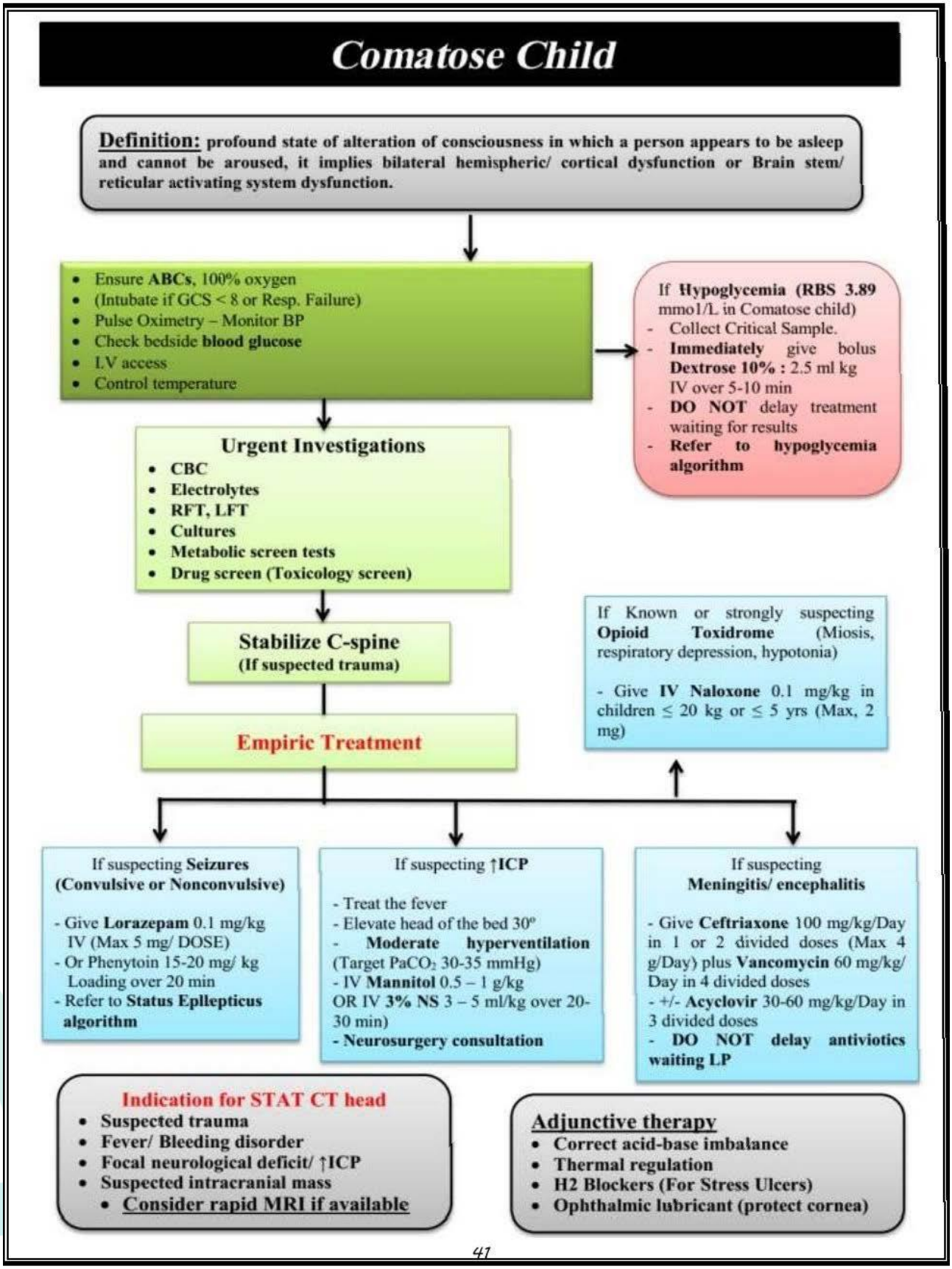
Comatose Child



Cerebral Oedema

Scope

Applicable to cerebral oedema in infants, children and adolescents

This protocol is NOT intended to address the management of specific etiologies of cerebral oedema/ increased intracranial pressure

Disclaimer

Protocols and guidelines outline the recommended or suggested clinical practice; however, they cannot replace sound clinical judgment by appropriately trained and licensed physicians. The physician is ultimately responsible for management of individual patients under their care.

Recognition

Cerebral oedema needs a high index of suspicion and must be actively checked for in any acute neurological condition and in case of neurological deterioration in any patient.

The most common underlying conditions are:

- ✓ Traumatic brain injury
- ✓ Hypoxic/ ischemic brain injury
- ✓ Intracerebral & subarachnoid hemorrhage
- ✓ Cerebrovascular stroke
- ✓ Hypertensive encephalopathy
- ✓ CNS infectious and inflammatory conditions
- ✓ Intracranial space-occupying lesions
- ✓ Hyponatremic encephalopathy
- ✓ Hepatic encephalopathy
- ✓ In-born errors of metabolism during acute crisis
- ✓ During treatment of DKA and severe hypernatremia
- ✓ Dialysis disequilibrium syndrome

The most important clinical manifestations include:

- ✓ Altered consciousness; irritability or deteriorating consciousness
- ✓ Sluggish pupillary reaction and hyperreflexia
- ✓ Focal neurological abnormalities
- ✓ Bradycardia and hypertension
- ✓ Abnormal respiration

NB. **CT findings are NOT essential before starting therapy. CT may be needed to evaluate etiology. Assess patient stability and initiate indicated emergency measures before transfer for imaging.**

NB. Increased ICP may also be evaluated through the presence of papilloedema and CSF pressure measurement.

Treatment of Cerebral Oedema

- **Ventilation, Oxygenation and Circulation**
- **Control of cerebral metabolism**
- **Fluid and hyperosmolar therapy**
- **Dexamethasone**
- **Specific treatment of the underlying cause**
- **CSF diversion procedures and decompressive craniotomy**

Ventilation, Oxygenation and Circulation

Ensure/ secure adequate airway and breathing

- Intubation may be necessary for GCS<8, inability to maintain airway or hypoventilation
- Consider cervical spine immobilization in trauma victims

Maintain normal ventilation and oxygenation

- Oxygen, keeping patients with borderline oxygen or CO2 is NOT sufficient
- In patients with acute severe increase in ICP, a BRIEF period of MILD hyperventilation (PaCO2 30mmHg) can be implemented.

"Avoid prolonged or excessive hyperventilation as it can exaggerate cerebral ischemia"

Maintain adequate B.P. for cerebral perfusion

- Use volume and vasoactive drugs as necessary
- Hypotension can exacerbate cerebral ischemia

Ensure adequate cerebral venous drainage:

- Elevate the head of the bed 30 degrees in neutral position if not contraindicated
- Avoid neck compression
- Wide, bilateral and unnecessary jugular venous lines are not recommended

Control of Cerebral Metabolism

Increased cerebral energy requirement in the context of compromised perfusion can increase cerebral damage:

- Control of any clinical or subclinical (bedside EEG) seizure activity
- Even if this would mean the need for intubation and MV in an ICU setting
- Active normothermia : aggressive management of fever or hyperthermia
- Monitor and correct hypoglycemia

Fluid and Hyperosmolar Therapy

In absence of (or after correcting) hypovolemia or hypotension, a restricted fluid management is necessary:

- Provide 50-70% of normal maintenance requirements
- If correcting dehydration as in hypernatremia or DKA, reduce the fluid rate by 1/3 provided there is no hypovolemia or hypotension

Avoid rapid reduction or subnormal serum sodium levels, modify sodium content of fluids

Avoid rapid reduction of blood glucose or urea levels

Hyperosmolar Therapy

IV hypertonic saline (5 mL/Kg of 2.7% NaCl over 30 min & may be repeated once)

- Treatment may be repeated after several hours as clinically indicated
- Increasing serum Na beyond 155-160mmol/L is not recommended
- Continuous hypertonic saline at a low rate may be used to maintain serum Na >145mmol/L but is not routine

IV mannitol (0.5-1g/Kg, 20% better than 10%, over 20min)

- Mannitol is effective even in those who cannot receive hypertonic saline
- Mannitol has a higher nephrotoxic potential than hypertonic saline
- Hypertonic saline improves systemic and cerebral perfusion compared to mannitol
- Both may be given in the same patient
- Repeated regular (eg q6h) doses of mannitol are not recommended

Dexamethasone:

- The main value is in cases of inflammatory vasogenic cerebral oedema; as in cases with vasculitis, cerebral infarctions, infectious, inflammatory and neoplastic lesions
- In bacterial meningitis, it is initiated with the first dose of antibiotics
- It is not recommended in cases of intracerebral hemorrhage
- Dose: 0.15 mg/kg/6h; a higher initial dose (0.5 mg/Kg) may be used

Specific Treatment of The Underlying Cause

CSF Diversion Procedures and Decompressive Craniotomy

- These should be considered case by case; weighing risks and benefits
- They non-specifically reduce ICP but do not address cerebral oedema itself
- CSF diversion is the specific treatment of acute hydrocephalus

Blood Product Prescription

Key Points

- All blood transfusion activity must occur in compliance with the relevant hospital procedures andguidelines.
- All patients should have consent for blood product administration recorded in the medical record prior totransfusion.
- A blood transfusion should only be given when the expected benefits to the patient are likely to outweighthe potential hazards.
- A blood product transfusion may be required to treat acute blood loss associated with surgery or trauma,or when the body cannot make enough blood cells in the case of bone marrow failure, cancer or bone marrow suppression.

Background

This guideline is adapted from the National Blood Authority (NBA) Patient Blood Management Guidelines: Module6 Neonatal and Pediatrics (2016) as well as the British Society for Hematology Guidelines on transfusion for fetuses, neonates and older children (2016). Local procedures or guidelines may vary.

Indications for Red Blood Cell (RBC) transfusion

Table 1: Indications for red blood cell transfusion

Hb	Indication
Hb <7 g/dL	Red Blood Cell (RBC) transfusion is often indicated, however lower thresholds may be acceptablein patients without symptoms (symptoms may include – tachycardia, flow murmur, lethargy, dizziness, shortness of breath, and cardiac failure) and where specific therapy (e.g. iron) is available.
Hb 7-9 g/dL	RBC transfusion may be indicated, depending on the clinical setting e.g. presence of bleeding orhemolysis and clinical signs and symptoms of anemia.
Hb >9 g/dL	RBC transfusion is often unnecessary and may be inappropriate
Transfusion may be indicated at higher thresholds for specific situations: Children with cyanotic congenital heart disease or on Extra Corporeal Life Support (ECLS) Children with Hemoglobinopathies (thalassemia or sickle cell disease) or congenital anemia on a chronictransfusion program	

Indications for Platelet Transfusion

Table 2: Indications for platelet transfusion in infants and children	
Platelet count (x10^9/L)	**Indication to trigger platelet transfusions in infants and children**
<10	Clinically stable pediatric patients receiving chemotherapy for leukemia or posthematopoietic stem cell transplantation (HSCT) (prophylactic).* Clinically stable patients with solid tumors (prophylactic).* Critically ill patients with no bleeding. * Transfusions at higher levels may be required for bladder, brain or necrotic tumors.
<20	Chemotherapy, HSCT & risk factors (e.g. fever, sepsis, minor bleeding, mucositis, disseminated intravascular coagulopathy (DIC) without bleeding) Critically ill patients with risk factors for bleeding (e.g. sepsis, renal failure, medications)Nasogastric tube insertion Intramuscular injections e.g. Erwinia asparaginase Insertion of a non-tunneled central venous line
<30	Lumbar puncture (LP) and ongoing chemotherapy-induced thrombocytopenia Central nervous system (CNS) tumor and: A VP shunt or Ommaya reservoir Has a **gross total resection** and is receiving chemotherapy and/or radiation Has **residual tumor** and is receiving chemotherapy and/or radiation
<50	LP and new disease induced thrombocytopenia Patient undergoing invasive procedure (including tunneled central venous line insertion) Moderate active bleeding (including bleeding associated with DIC) CNS tumour and: A past history of intracranial hemorrhage Is receiving an anti- angiogenesis agent such as bevacizumab
<75	Major hemorrhage due to trauma or significant post-operative bleeding
<100	Patient undergoing high risk invasive procedure (e.g. neurosurgery/ophthalmology) ECLS (lower platelets may be acceptable in stable patients)

Platelet transfusion is NOT indicated for the following

- Stable patients with chronic, stable, severe thrombocytopenia due to: Alloimmunization
- Immune thrombocytopenia (ITP)
- Thrombotic thrombocytopenic purpura (TTP) Aplastic anemia or myelodysplastic syndrome (MDS)
- These patients should be observed without prophylactic platelet transfusions and should receive platelet transfusions only with clinically significant bleeding
- Bone marrow aspirate and trephine biopsy Intravenous cannula insertion

Indications for Fresh Frozen Plasma (FFP)

FFP is appropriate for the following

- Acute bleeding in the setting of significant coagulopathy.
- Warfarin reversal, in the presence of significant or life-threatening bleeding or prior to emergency surgical procedures
- Given in addition to vitamin K

Note:

- ✓ Vitamin-K dependent clotting factor concentrates (e.g. prothrombinex) may be given instead of FFP for bleeding secondary to warfarin or emergency warfarin reversal.
- ✓ Liver disease, with clinically significant bleeding in the context of coagulopathy post liver transplantation. Acute disseminated intravascular coagulopathy (DIC) with bleeding and significant coagulopathy
- ✓ During massive transfusion or cardiac bypass for the treatment of bleeding Plasma exchange for the treatment of TTP
- ✓ Specific factor deficiencies where a factor concentrate is not available

FFP is NOT indicated for the following

- ✓ The correction of minor coagulation abnormalities (minor prolongation of the INR/APTT) in the non- bleeding child
- ✓ Liver disease when there are minor coagulation abnormalities and no-bleeding
- ✓ For reversal of a INR <2.0 in patients undergoing minor procedures

Indications for Cryoprecipitate

- ✓ Active bleeding and fibrinogen level <1.5 g/L
- ✓ During massive transfusion or cardiac bypass, for the treatment bleeding when the fibrinogen level <1.5 g/L or there is hyperfibrinolysis
- ✓ Acquired fibrinogen deficiency or acute DIC when there is significant bleeding and the fibrinogen <1.0 g/L
- ✓ Prior to an invasive procedure when the fibrinogen <1.0 g/L and there is a risk of significant bleeding associated with the surgery or it is at a critical site (e.g. neurosurgery or eye surgery)

Cryoprecipitate is NOT indicated for the following

- ✓ Non-bleeding children with mildly reduced fibrinogen levels
- ✓ Liver disease when there are minor coagulation abnormalities and no active bleeding

Management - Transfusion volumes and rates

In children less than 20 kg, transfusion volumes should be calculated based on weight and prescribed in mLs In children greater than 20 kg, calculate and prescribe the transfusion volume with consideration to pack sizes for RBC transfusion, prescribe a single unit followed by clinical reassessment to determine additional transfusion requirements for platelet transfusions, the usual platelet dose in an adult is one adult unitAll transfusions must be completed within 4 hours of spiking a pack

Table 3: Transfusion Volumes and Rates				
Blood product	**RBC**	**Platelets**	**FFP**	**Cryoprecipitate**
The formula for	**Children <20 kg:**	**Children <20 kg:**	10 – 20 mL/kg	5 – 10 mL/kg
Calculating Transfusion Volume (mL)	mL = wt (kg) x Hb (g/L) rise (desired Hb – actual Hb) x 0.5	Pooled platelets 10 mL/kg Apheresis platelets 5 – 10 mL/kg		
	e.g.10 kg child requiring Hb to rise from 60 to 80g/L:	**Children >20 kg:** 1 unit for >20 kg child		
	10 x 20 x 0.5 = 100mL			
	Children >20 kg: 1 unit for >20 kg child			
Typical unit Volume	Red cell unit ~ 258 ml Pediatric red cells (pedipak) ~ 61 mL	Pooled platelet ~ 334 mL Apheresis platelet ~181 mL	FFP ~284 mL Pediatric FFP (pedipak) ~69 mL	Cryoprecipitate ~ 36 mL
Transfusion Rate	5 mL/kg/hr. Commence at a slower rate (e.g. half the prescribed rate) for the first 15 minutes (Usual maximum rate 150 ml/hr.)	10 – 20 mL/kg/hr. Faster infusion rates (e.g. given over 30 minutes) may result in a transfusion reaction	10 – 20 mL/kg/hr.	10 – 20 mL/kg/hr.

Blood product modifications

- Request the appropriate blood component and special requirements:
- **Irradiated blood products** should be given to:
  - ✓ All immunocompromised patients, including all immunology patients, oncology patients, children with cardiac disease, and directed blood donations to prevent graft-versus-host disease.

CMV negative products:

- Leucocyte-depleted blood products are considered an acceptable alternative to CMV seronegative products.
- CMV-negative products are only indicated for exchange transfusion, pregnant adolescents and patients with Severe Combined Immunodeficiency Disease (SCID) who are CMV negative.

Phenotype matched red blood cells:

- Indicated for chronically transfused patients or patients with red cell alloantibodies

Cryodepleted FFP

- May be requested for patients with Thrombotic Thrombocytopenic Purpura (TTP), although FFP may be as effective.

IgA deficient products

- Patients with IgA deficiency who have developed an anti-IgA antibody

HLA matched

- For children with immunological refractory thrombocytopenia

Clinical Practice Guidelines on Prevention and Management of Pain

Recommendations	Strength of Recommendation	Quality of Evidence
Analgesia 1) We suggest that, in critically ill pediatric patients 6 yr. old and older who are capable of communicating, pain assessment via self-report be routinely performed using the Visual Analog Scale, Numeric Rating Scale, Oucher Scale, or Wong-Baker Faces pain scale.	Conditional	Low
2) We recommend the use of either the Faces, Legs, Activity, Cry, and Consolability or COMFORT-B scales for assessing pain in non-communicative critically ill pediatric patients.	Strong	Moderate
3) We recommend the use of observational pain assessment tools rather than vital signs alone for assessment of postoperative pain in critically ill pediatric patients.	Strong	Moderate
4) We suggest the use of observational pain assessment tools rather than vital signs alone for assessment of procedure-related pain in critically ill pediatric patients.	Conditional	Low
5) We recommend that IV opioids be used as the primary analgesic for treating moderate to severe pain in critically ill pediatric patients.	Strong	Moderate
6) We recommend the addition of an adjunct NSAID (IV or oral) to improve early postoperative analgesia in critically ill pediatric patients.	Strong	Moderate
7) We suggest the addition of an adjunct NSAID agent (IV or oral) to decrease opioid requirements in the immediate postoperative period in critically ill pediatric patients.	Conditional	Low
8) We suggest the addition of adjunct acetaminophen (IV or oral) to improve early post-operative analgesia in critically ill pediatric patients.	Conditional	Low
9) We suggest the addition of adjunct acetaminophen (IV or oral) to decrease opioid requirements intermediate postoperative period in critically ill pediatric patients.	Conditional	Low
10) We recommend that music therapy be offered to augment analgesia in critically ill postoperative pediatric patients.	Strong	Moderate
11) We recommend that nonnutritive sucking with oral sucrose be offered to neonates and young infants prior to performing invasive procedures.	Strong	High

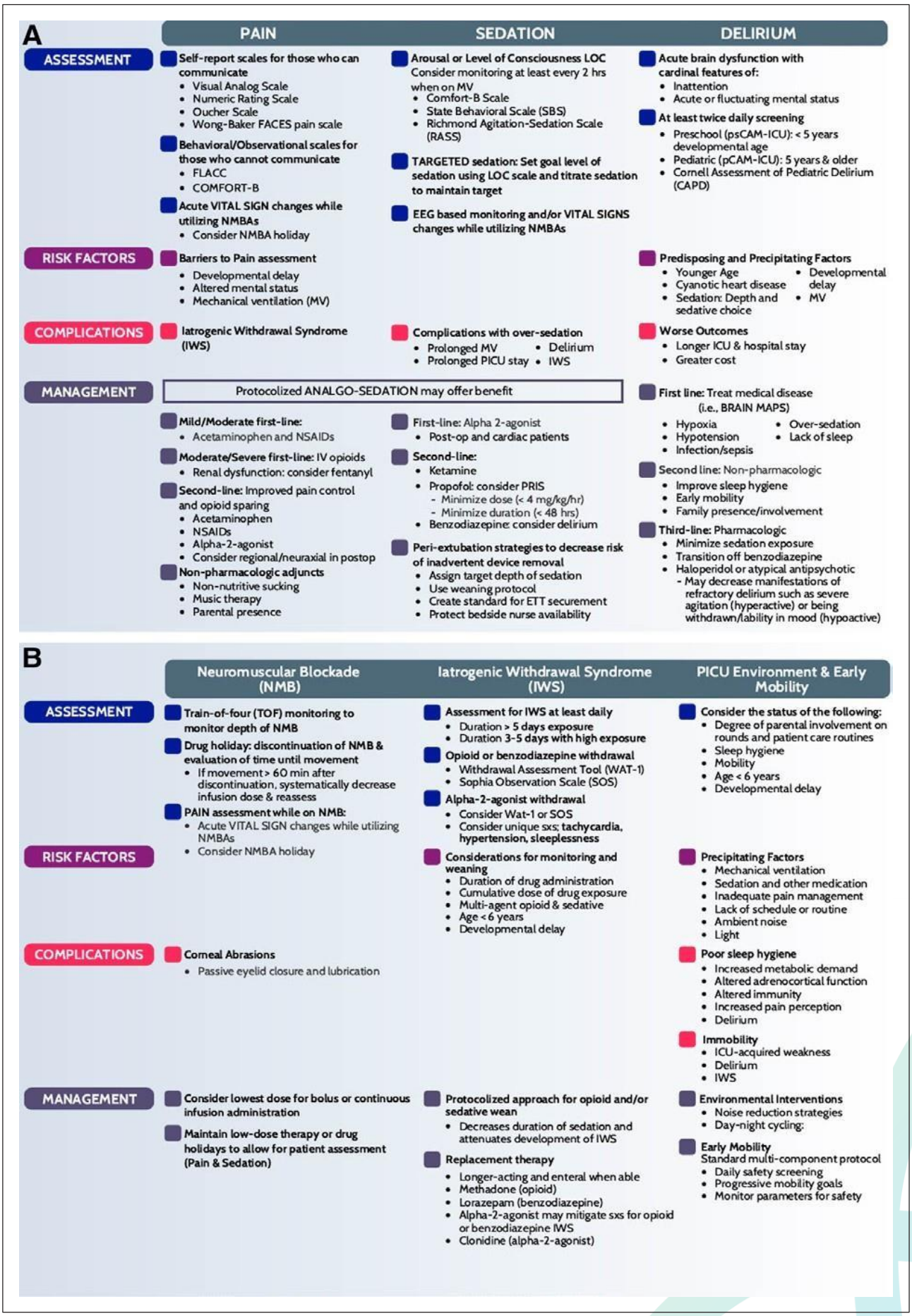
Recommendations	Strength of Recommendation	Quality of Evidence
Sedation	Strong	Moderate
1) We recommend the use of the COMFORT-B Scale or the State Behavioral Scale, to assess level of sedation in mechanically ventilated pediatric patients.	Conditional	Low
2) We suggest the use of the Richmond Agitation-Sedation Scale to assess the level of sedation in mechanically ventilated pediatric patients.	Conditional	Low
3) We suggest that all pediatric patients requiring MV are assigned a target depth of sedation using a validated sedation assessment tool at least once daily.	Conditional	Low
4) We suggest the use of protocolized sedation in all critically ill pediatric patients requiring sedation and/or analgesia during MV.	Conditional	Low
5) The addition of daily sedation interruption to sedation protocolization is not suggested due to lack of improvement in outcomes.		
6) During the periextubation period when sedation is typically lightened, we suggest the following bundle strategies to decrease the risk of inadvertent device removal: a) Assign a target depth of sedation at an increasing frequency to adapt to changes in patient clinical status and communication strategies to reach the titration goal. b) Consider a sedation weaning protocol. c) Consider unit standards for securement of endotracheal tubes and safety plan. d) Restrict nursing workload to facilitate frequent patient monitoring, and decrease sedation requirements, and risk of self-harm.		
7) We suggest the use of alpha$_2$-agonists as the primary sedative class in critically ill pediatric patients requiring MV.		

Recommendations	Strength of Recommendation	Quality of Evidence
8) We recommend that dexmedetomidine be considered as a primary agent for sedation in critically ill pediatric postoperative cardiac surgical patients with expected early extubation.	Strong	Moderate
9) We suggest the use of dexmedetomidine for sedation in critically ill pediatric postoperative cardiac surgical patients to decrease the risk of tachyarrhythmias.	Conditional	Low
10) We suggest that continuous protocol sedation at doses less than 4 mg/kg/hr. (67 µg/ kg/min) and administered for less than 48hr may be a safe sedation alternative to minimize the risk of protocol-related infusion syndrome development.	Conditional	Low
11) Short-term (< 48 hr.) continuous protocol sedation may be a useful adjunct during the periextubation period to facilitate the weaning of other analog-sedative agents prior to extubation.	Good practice	
12) We suggest consideration of adjunct sedation with ketamine in patients who are not otherwise at an optimal sedation depth.	Conditional	Low
13) During the periextubation period when sedation is typically lightened, we suggest the following bundle strategies to decrease risk of inadvertent device removal: a) Assign a target depth of sedation at increasing frequency to adapt to changes in- patient clinical status and communicate strategies to reach titration goal. b) Consider a sedation weaning protocol. c) Consider unit standards for securement of endotracheal tubes and safety plan. d) Restrict nursing workload to facilitate frequent patient monitoring, decrease sedation requirements, and risk of self-harm.	Conditional	Low

Recommendations	Strength of Recommendation	Quality of Evidence
Neuromuscular blockade		
1) We suggest that train-of-four monitoring be used in concert with clinical assessment to determine the depth of neuromuscular blockade.	Conditional	Low
2) We suggest using the lowest dose of NMBAs required to achieve desired clinical effects and manage undesired breakthrough movement.	Conditional	Low
3) Electroencephalogram-based monitoring may be a useful adjunct for the assessment of sedation depth in critically ill pediatric patients receiving NMBAs.	Good practice	
4) We suggest that sedation and analgesia should be adequate to prevent awareness prior to and throughout NMBA use.	Conditional	Low
5) We recommend routine use of passive eyelid closure and eye lubrication for the prevention of corneal abrasions in critically ill pediatric patients receiving NMBAs.	Strong	Moderate
ICU delirium		
1) We recommend the use of the preschool and pediatric Confusion Assessment Methods for the ICU or the Cornell Assessment for Pediatric Delirium as the most valid and reliable delirium monitoring tools in critically ill pediatric patients.	Strong	High
2) We recommend routine screening for ICU delirium using a validated tool in critically ill pediatric patients upon admission through ICU discharge or transfer.	Strong	High
3) Given low patient risk, and possible patient benefit to reduce the incidence and/or decrease duration or severity of delirium we suggest the following non-pharmacologic strategies: optimization of sleep hygiene, use of interdisciplinary rounds, family engagement on rounds, and family involvement with direct-patient care.	Conditional	Low
4) We suggest performing EM, when feasible, to reduce the development of delirium.	Conditional	Low
5) We recommend minimizing benzodiazepine-based sedation when feasible in critically ill pediatric patients to decrease incidence and/or duration or severity of delirium.	Strong	Moderate

Recommendations	Strength of Recommendation	Quality of Evidence
6) We suggest strategies to minimize overall sedation exposure whenever feasible to reduce coma and the incidence and/or severity of delirium in critically ill children.	Conditional	Low
7) We do not suggest routine use of haloperidol or atypical antipsychotics for the prevention of or decrease in duration of delirium in critically ill pediatric patients.	Conditional	Low
8) We suggest that in critically ill pediatric patients with refractory delirium, haloperidol or atypical antipsychotics be considered for management of severe delirium manifestations, with consideration of possible adverse drug effects.	Conditional	Moderate
9) We recommend a baseline electrocardiogram followed by routine electrolyte and QTc interval monitoring for patients receiving haloperidol or atypical antipsychotics	Strong	Moderate
iatrogenic withdrawal syndrome (iws) 1) We recommend use of either the Withdrawal Assessment Tool-1or Sophia Observation Scale for the assessment of IWS due to opioid or benzodiazepine withdrawal in critically ill pediatric patients.	Strong	Moderate
2) We suggest routine IWS screening after a shorter duration (3–5 d) when higher opioid or benzodiazepine doses are used.	Conditional	Moderate
3) Until a validated screening tool is developed, monitoring for IWS from alpha2-agonists should be performed using a combination of associated symptoms (unexplained hypertension or tachycardia) with adjunct use of a validated benzodiazepine or opioid screening tool.	Good practice	
4) We suggest that opioid-related IWS be treated with opioid replacement therapy to attenuate symptoms, irrespective of preceding dose and /or duration or opioid exposure.	Conditional	Low
5) Benzodiazepine-related IWS should be treated with benzodiazepine replacement therapy to attenuate symptoms, irrespective of preceding dose and/or duration of benzodiazepine exposure.	Good practice	
6) Alpha2-agonist–related IWS should be treated with IV and/or or enteral alpha2-agonist replacement therapy to attenuate symptoms, irrespective of preceding dose and/or duration of alpha2-agonist exposure.	Good practice	
7) We suggest use of a standardized protocol for sedation/analgesia weaning to decrease duration of sedation taper and attenuate emergence of IWS.	Conditional	Low

Recommendations	Strength of Recommendation	Quality of Evidence
Optimizing Environment 1) We suggest facilitation of parental or caregiver presence in the PICU during routine care and interventional procedures to a) provide comfort to the child, b) decrease pa-rental levels of stress and anxiety and c) increase level of satisfaction of care.	Conditional	Low
2) We suggest offering patients the use of noise reducing devices such as ear plugs or headphones to reduce the impact of non-modifiable ambient noise (conditional, low-level evidence).	Conditional	Low
3) We suggest that PICU teams make environmental and/orbehavioral changes to reduce excessive noise and therefore improve sleep hygiene and comfort, in critically ill pediatric patients.	Conditional	Low
4) We suggest performing EM to minimize the effects of immobility in critically ill pediatric patients.	Conditional	Low
5) We suggest the use of a standardized EM protocol that outlines readiness criteria, contraindications, developmentally appropriate mobility activities and goals, and safety thresholds guided by the multidisciplinary team and family decision-making.	Conditional	Low


A
PAIN
SEDATION
DELIRIUM
ASSESSMENT
Self-report scales for those who can communicate
Visual Analog Scale
Numeric Rating Scale
Oucher Scale
Wong-Baker FACES pain scale
Behavioral/Observational scales for those who cannot communicate
FLACC
COMFORT-B
Acute VITAL SIGN changes while utilizing NMBAs
Consider NMBA holiday
Arousal or Level of Consciousness LOC
Consider monitoring at least every 2 hrs when on MV
Comfort-B Scale
State Behavioral Scale (SBS)
Richmond Agitation-Sedation Scale (RASS)
TARGETED sedation: Set goal level of sedation using LOC scale and titrate sedation to maintain target
EEG based monitoring and/or VITAL SIGNS changes while utilizing NMBAs
Acute brain dysfunction with cardinal features of:
Inattention
Acute or fluctuating mental status
At least twice daily screening
Preschool (psCAM-ICU): < 5 years developmental age
Pediatric (pCAM-ICU): 5 years & older
Cornell Assessment of Pediatric Delirium (CAPD)
RISK FACTORS
Barriers to Pain assessment
Developmental delay
Altered mental status
Mechanical ventilation (MV)
Predisposing and Precipitating Factors
Younger Age
Cyanotic heart disease
Sedation: Depth and sedative choice
Developmental delay
MV
COMPLICATIONS
Iatrogenic Withdrawal Syndrome (IWS)
Complications with over-sedation
Prolonged MV
Prolonged PICU stay
Delirium
IWS
Worse Outcomes
Longer ICU & hospital stay
Greater cost
MANAGEMENT
Protocolized ANALGO-SEDATION may offer benefit
Mild/Moderate first-line:
Acetaminophen and NSAIDs
Moderate/Severe first-line: IV opioids
Renal dysfunction: consider fentanyl
Second-line: Improved pain control and opioid sparing
Acetaminophen
NSAIDs
Alpha-2-agonist
Consider regional/neuraxial in postop
Non-pharmacologic adjuncts
Non-nutritive sucking
Music therapy
Parental presence
First-line: Alpha 2-agonist
Post-op and cardiac patients
Second-line:
Ketamine
Propofol: consider PRIS
- Minimize dose (< 4 mg/kg/hr)
- Minimize duration (< 48 hrs)
Benzodiazepine: consider delirium
Peri-extubation strategies to decrease risk of inadvertent device removal
Assign target depth of sedation
Use weaning protocol
Create standard for ETT securement
Protect bedside nurse availability
First line: Treat medical disease (i.e., BRAIN MAPS)
Hypoxia
Hypotension
Infection/sepsis
Over-sedation
Lack of sleep
Second line: Non-pharmacologic
Improve sleep hygiene
Early mobility
Family presence/involvement
Third-line: Pharmacologic
Minimize sedation exposure
Transition off benzodiazepine
Haloperidol or atypical antipsychotic
- May decrease manifestations of refractory delirium such as severe agitation (hyperactive) or being withdrawn/lability in mood (hypoactive)
B
Neuromuscular Blockade (NMB)
Iatrogenic Withdrawal Syndrome (IWS)
PICU Environment & Early Mobility
ASSESSMENT
Train-of-four (TOF) monitoring to monitor depth of NMB
Drug holiday: discontinuation of NMB & evaluation of time until movement
If movement > 60 min after discontinuation, systematically decrease infusion dose & reassess
PAIN assessment while on NMB:
Acute VITAL SIGN changes while utilizing NMBAs
Consider NMBA holiday
Assessment for IWS at least daily
Duration > 5 days exposure
Duration 3-5 days with high exposure
Opioid or benzodiazepine withdrawal
Withdrawal Assessment Tool (WAT-1)
Sophia Observation Scale (SOS)
Alpha-2-agonist withdrawal
Consider Wat-1 or SOS
Consider unique sxs: tachycardia, hypertension, sleeplessness
Consider the status of the following:
Degree of parental involvement on rounds and patient care routines
Sleep hygiene
Mobility
Age < 6 years
Developmental delay
RISK FACTORS
Considerations for monitoring and weaning
Duration of drug administration
Cumulative dose of drug exposure
Multi-agent opioid & sedative
Age < 6 years
Developmental delay
Precipitating Factors
Mechanical ventilation
Sedation and other medication
Inadequate pain management
Lack of schedule or routine
Ambient noise
Light
COMPLICATIONS
Corneal Abrasions
Passive eyelid closure and lubrication
Poor sleep hygiene
Increased metabolic demand
Altered adrenocortical function
Altered immunity
Increased pain perception
Delirium
Immobility
ICU-acquired weakness
Delirium
IWS
MANAGEMENT
Consider lowest dose for bolus or continuous infusion administration
Maintain low-dose therapy or drug holidays to allow for patient assessment (Pain & Sedation)
Protocolized approach for opioid and/or sedative wean
Decreases duration of sedation and attenuates development of IWS
Replacement therapy
Longer-acting and enteral when able
Methadone (opioid)
Lorazepam (benzodiazepine)
Alpha-2-agonist may mitigate sxs for opioid or benzodiazepine IWS
Clonidine (alpha-2-agonist)
Environmental Interventions
Noise reduction strategies
Day-night cycling:
Early Mobility
Standard multi-component protocol
Daily safety screening
Progressive mobility goals
Monitor parameters for safety

Schematic summary of the key Pain, Agitation, Neuromuscular Blockade, and Delirium in critically ill pediatric patients with consideration of the PICU Environment and Early Mobility (PANDEM) recommendations and representation of the interplay between sedative and analgesic choice on unintended but related outcomes.

BRAIN MAPS = Bring oxygen, Remove/Reduce deliriogenic drugs, patient Atmosphere, Immobilization, New organ dysfunction, Metabolic disturbances, Awake, Pain, Sedation
CAPD = Cornell Assessment of Pediatric Delirium
COMFORT-B = COMFORT-Behavior
EEG = electroencephalogram
ETT = endotracheal tube
FLACC = Faces, Legs, Activity, Cry, and Consolability
IWS = iatrogenic withdrawal syndrome
MV = mechanical ventilation
NMBA = neuromuscular blocking agent
NSAID = nonsteroidal anti-inflammatory drug
pCAM-ICU = pediatric Confusion Assessment Method for the ICU
PRIS = propofol-related infusion syndrome
psCAM-ICU = preschool Confusion Assessment Method for the ICU
RASS = Richmond Agitation-Sedation Scale
SBS = State Behavioral Scale
SOS= Sophia Observation Scale
TOF=train-of-four
WAT-I=WithdrawalAssessmentTool-1

Basic Mechanical Ventilation

Scope:

MV is a respiratory support modality. Patients requiring MV are likely to require other therapies; for the underlying disease, for respiratory care (eg physiotherapy, suction, nebulized medications) and possibly for other system support. This protocol addresses the MV part; however all aspects of patient care are equally important. MV is not a contraindication for enteral feeding.

This protocol addresses mechanical ventilation of pediatric patients; beyond the neonatal period, in the pediatric critical care setting.

Disclaimer:

Protocols and guidelines outline the recommended or suggested clinical practice; however, they cannot replace sound clinical judgment by appropriately trained and licensed physicians. The physician is ultimately responsible for management of individual patients under their care.

Concept Definitions

Ventilation: gas exchange between alveolar and surrounding gas. The volume inspired (normally = expired) each breath is the tidal volume (Vt). Minute volume is the amount of ventilation per minute and equals tidal volume x respiratory rate.

RR: respiratory rate is the number of breaths per minute

Cycle: in the context of MV, a respiratory cycle is a breath (inspiration & expiration)

Cycling: in the context of MV, cycling is the end of inspiration. Cycling occurs when the ventilator ends inspiration allowing passive expiration by the patient

Ti: inspiratory time is the time interval from the beginning of inspiration till the beginning of expiration

Rise time: the time interval between the beginning of inspiration and reaching the plateau; during which pressure is rising. It can be expressed in seconds or a percentage of inspiratory time

Oxygenation: (arterial oxygenation) the amount of oxygen reaching arterial blood, measured as partial pressure (PaO2; normally 80-100mmHg) or as oxygen saturation (SaO2 or SpO2; normally 95-97%); which is the percentage of oxyhemoglobin. Arterial oxygenation depends on ventilation, FiO2 and pulmonary gas exchange.

FiO2: The percentage of oxygen in inspired gas. Room air has 21% oxygen.

Compliance: change in volume for change in pressure ($\Delta V/\Delta P$). Low compliance means more change in pressure is required to achieve a volume change (stiff lungs; as in pneumonia, collapse, ARDS)

Resistance: airway resistance is the force opposing air flow into or outside the lungs. Bronchospasm increases airway resistance and this increases the effort necessary to move air into/ outside the alveoli

Work of Breathing: the energy used (essentially the amount of effort used) in breathing; aiming to achieve normal ventilation.

PIP: the peak inspiratory pressure; the highest pressure reached during inspiration

PEEP: the positive end-expiratory pressure; during expiration, an amount of +ve pressure is maintained and it does not drop to zero. The main purpose is to prevent alveolar collapse during expiration.

MAP: mean airway pressure. The average pressure throughout the respiratory cycle (inspiration & expiration)

P plateau: the plateau pressure is the pressure at the plateau phase towards the end of inspiration, measured in an inspiratory pause when there is no air flow. It is correlated to the alveolar peak pressure; by excluding the pressure gradient needed to overcome airway resistance.

AP: the pressure difference between plateau pressure and PEEP

Indications

- Post CPR management
- Severe hypoxemia (PaO2< 50-55mmHg) despite oxygen therapy. An SpO2<85% by pulse oximeter is highly suggestive of PaO2<50-55mmHg
- Severe hypoventilation (PaCO2 >60mmHg with respiratory acidosis)
- Sustained or frequent apnea with desaturation
- Unacceptable work of breathing (most common indication in PICU)
- Excessively slow, shallow or irregular breathing
- Severe or increasing respiratory distress (impending exhaustion)
- Airway compromise requiring intubation
- Severe refractory shock
- CNS: deep coma with respiratory weakness or airway compromise, severe cerebral oedema especially with (even mild) hypoventilation, refractory status epilepticus needing anesthetic drugs
- Surgery under general anesthesia with neuromuscular blockade (most common indication)

Objectives of MV

- Normal/ acceptable ventilation
- Normal/ acceptable oxygenation
- Acceptable work of breathing. Eliminating patient effort is not the objective, but increased WOB on MV is not acceptable either.
- Lung protection. Avoidance of ventilator-induced lung injury

Initiation

Airway Establishment

- Non-invasive ventilation through a face mask may be used for those with adequate airway and adequate circulation. Response should be assessed after 1hr and invasive MV initiated if response is inadequate.
- ET intubation is the most common approach. Bag-mask ventilation is effective until preparations are made for intubation and ventilation.
- Tracheostomy should be considered when anticipated duration of MV exceeds 2-4weeks

Primary settings

1. Mode:

There are different modes of MV and they are all acceptable provided they are appropriate to the ventilator specs, user experience and patient's condition

CMV: Only acceptable when the patient either

(a) Can provide no significant work of breathing due to disease condition or necessary sedation/ neuromuscular blockade; or
(b) Has severe ARDS or very severe bronchial obstruction and cannot be adequately ventilated except after eliminating patient efforts by neuromuscular blockade. This should be employed for the shortest time necessary

SIMV: The strategy is to share the effort between the ventilator and the patient based on rate. Reducing set ventilator rate can increase patient's spontaneous rate and vice versa. The total rate (ventilator + patient) should be appropriate for age and condition. Weaning depends on reducing set rate.

Assist-Control: The strategy is to assist every breath initiated by the patient. Ventilatory support is based on patient's rate, the set rate should be below the patient's respiratory rate and acts as a backup. It is important that the given pressure/volume per breath is sufficient to achieve an acceptable minute volume; without discrepancy between set Ti and patient Ti and without excessively high respiratory rate. Improved lung condition is associated with lower PIP achieving normal Vt. Further weaning requires shift to PSV or SIMV.

Pressure Support: The strategy is also to assist every breath initiated by the patient, who also determines the inspiratory time. This is pressure controlled. Reducing pressure support (ΔP above PEEP) increases patient work and vice versa. PS can be applied alone for patients who have a good respiratory drive, or combined with SIMV to assist breaths above the set rate.

2. Primary Control Variable:

- Ventilators give a breath based on delivering a certain tidal volume (volume controlled) or on increasing pressure to a certain value (pressure controlled) for the duration of inspiration, followed by a passive expiration.
- Normal Vt is 6 -8mL/Kg. VC may use constant or decelerating (recommended) flow. The resulting PIP will depend on lung mechanics and is measured
- PC depends on raising pressure to a certain PIP (in some ventilators ΔP above PEEP). The Vt delivered depends on lung mechanics and is measured
- Both VC and PC aim to provide an appropriate Vt
- There are modes that attempt to combine both methods (Dual modes eg PRVC, VG, VC+, etc). The principle is setting a target Vt and adjusting pressure based on actual measured Vt; with many variations. Proportional assist ventilation depends on giving a variable support depending on patient's effort aiming to achieve normal ventilation without increasing patient WOB. These modes can be helpful and improve patient synchronization when used appropriately. Each device has its own principles and users should be aware of these before using such modes.

3. PIP or Vt:

- Other than dual modes, you will set one and monitor the other
- The plateau pressure (pressure at the end of inspiration) should not exceed 28-30cmH2O to avoid lung injury. Patients with near normal compliance can achieve normal Vt with MUCH lower pressures; while those with severe ARDS may not be able to achieve normal Vt at this pressure and a lower Vt (4-5mL/kg) may be accepted. Both high volume and high pressure should be avoided.
- Note: Plateau pressure is normally close to PIP but can be considerably less in presence of airway obstruction. Some ventilators set ΔP not PIP (so you need to add PEEP to get PIP)
- All modern ventilators can measure both delivered Vt and peak/plateau P.

4. Inspiratory Time Ti:

Other than PS and similar modes, you set the Ti after which the ventilator allows the patient to expire. Setting must consider:

(a) For an I/E ratio near 2 (initially used), the Ti should be 20/ total (patient + ventilator) rate

(b) Without changing rate, a longer Ti leads to a higher MAP in PC (& you can then lower PIP if tolerated) and a lower PIP for a given Vt in volume control (both are favorable). A too short Ti can lead to inadequate Vt or excessive PIP.

(c) Increasing Ti is limited by the need for adequate time for expiration to avoid air trapping. While stiff lungs can usually tolerate I/E ratio 1:1, those with obstructive pathology need great caution

(d) A set Ti greatly different from the patient's own Ti can lead to asynchrony

5. Ventilator Rate:

In CMV and SIMV, this is the rate per min. the ventilator should provide. Initially, a normal rate for age should be set.

6. PEEP:

This prevents end-expiratory collapse and optimizes tidal breaths to a favorable segment of lung compliance. A starting PEEP can be 5cmH2O and note:

- Those with stiff lungs may need considerably higher PEEP to maintain lung recruitment
- The best PEEP is that which improves oxygenation and Vt at a relatively low ΔP
- Excessive PEEP can lead to alveolar overstretch, increasing an air leak, reduced venous return and increased intracranial pressure

7. FiO2 :

- Initially set 100% if the patient is unstable, cyanosed or severely hypoxic with lung pathology; otherwise set 40-50%. Adjust based on pulse-oximetry to the FiO2 achieving acceptable oxygenation (saturation around 94%).
- Neonates and especially preterms are more susceptible to the adverse effects of excessive oxygen
- Those with brain injury, refractory shock, pulmonary hypertensive crisis & CO poisoning require high FiO2 and maintaining a higher degree of arterial oxygenation
- The maximum safe duration by FiO2 is as follows:
  - ✓ 100% 3-4h
  - ✓ 80% 24h
  - ✓ 60% 3-4days
  - ✓ 40% 3-4 weeks

8.Flow or Rise Time:

Changing these settings alter how rapidly the peak pressure is attained during inspiration.

9.Trigger Sensitivity:

Correct setting is associated with:

(a) The ventilator recognizing most patient efforts (no missed triggers); and

(b) There are no false triggers

NB:

- **Humidifier must be used with appropriate temperature, necessary bacterial filters must be installed, flow sensor must be installed and calibrated for any mode other than CMV/IMV, alarm limits must be checked and backup power must be operational (ventilator battery or UPS).**
- **Interrupting the ventilator circuit should be minimized. ET suction is a sterile procedure and should be done when necessary. Unnecessary injections of saline into the ETT should be avoided.**

Initial Settings Summary	
Mode & control variable	CMV only when patient not breathing or with neuromuscular blockade SIMV, with or without PS, or AC PC and VC (with decelerating flow) both acceptable Dual modes when applicable & user experienced in them
PIP or Vt	Initial PIP: 15 for normal lungs, 20 for pathological lungs Check chest expansion/air entry/ expired Vt and adjust PIP up or down targeting a normal Vt (while beside patient) Neonates & preterms need lower pressures Generally will not exceed 30. In severe obstruction Pplateau should not exceed 30 (PIP might be higher) VC: set a normal Vt 6-8mL/min. Check resulting airway pressure and if it exceeds above limits, reduce Vt to 4-6mL/min. to achieve safe pressure.
Ti Rate	Start at an I/E of 1:2 (Ti= 20/ total rate). At age-appropriate rates: Infants: Ti 0.5 at a rate of 40 Small children: Ti 0.6-0.7 at a rate of 30 Older children: Ti 1.0 at a rate of 20 Longer Ti may be helpful with stiff lungs I/E may reach 1:1 with a longer Ti or high RR but ensure complete expiration before next cycle (auscultation, vent. Curves). Not recommended with bronchial obstruction.
PEEP	Generally 5. Those with stiff lungs may need considerably higher
FiO2	Start 100% if cyanosed, severely hypoxic or otherwise unstable Else, start around 50% Adjust targeting SpO2 ≥92%; not necessarily close to 100% (there are some exceptions needing higher SpO2)
Flow or rise time	Mostly don't change ventilator preset
Trigger sensitivity	If patient initiating breaths, adjust to avoid missed efforts and false triggers

Monitoring and Adjustment

Monitoring of MV

- Patient monitoring is essential; including continuous ECG, pulse oximetry and non-invasive blood pressure. Useful data can be obtained by clinical examination, from ventilator measurements and graphics, blood gases and imaging studies.
- The patient should be monitored for the objectives of MV.

Note:

- **Ventilation and oxygenation can be assessed from blood gases; however, pulse oximetry can be used to assess oxygenation, the ventilator can usually measure minute volume and capnography may be used for end-tidal CO2.**
- **Pulmonary gas exchange is assessed using oxygen-derived pulmonary indices:**

  (1) Oxygenation index (OI) = FiO2 (%) x MAP / PaO2
  Higher value ➔ *more severe pathology*
  Above 20 ➔ *severe lung pathology*
  Below 5 ➔ *necessary to consider extubation*
  Cannot apply in patients without positive pressure support (MV or CPAP)
  (2) Oxygen saturation index. Uses SpO2 instead of PaO2. Values are somewhat lower than OI
  (3) Arterial/ inspired oxygen ratio= PaO2/ FiO2(fraction 0.21-1.00)
  Can be applied with or without positive pressure support
  Normally >300. Lower values ➔ *more severe pathology*
  <100 ➔ *severe ARDS*

- **The ventilator can usually measure compliance and resistance**
- **Work of breathing is generally assessed clinically. It is also important to identify patient-ventilator asynchrony (ventilator graphics can help) and determine the appropriate level of sedation (patient calm and synchronized but not oversedated)**
- **NB Proper settings and synchronization can be associated with less need for sedation in many patients. No patient may receive neuromuscular blockade without deep sedation.**
- **Patients should be monitored for ventilator induced lung injury, including ventilator parameters that denote overdistension, barotrauma or atelectrauma, as well as the development of VAP.**

Adjustment of Settings

Ventilation:

- Increasing tidal volume increases ventilation, but avoid overdistension & observe ΔP limits
- Increasing rate increases ventilation, but observe for Te adequacy

Oxygenation:

- Ventilation affects oxygenation
- Increasing FiO2 increases oxygenation
- Increasing MAP (PEEP, I/E ratio, lastly PIP) increases oxygenation
- A successful lung recruitment improves oxygenation

WOB:

- Patient work of breathing can be decreased by increasing ventilation provided by the ventilator, as well as by improving patient-ventilator synchronization
- Where possible and tolerated, reducing ventilation provided by the ventilator, with equivalent increase in patient contribution, corresponds to lowering the level of support

NB:

- **It is acceptable to increase settings during a procedure or in response to deterioration (particularly FiO2 and ventilator rate). However, it is mandatory to identify & correct the cause of deterioration. After the reason is over, aim to return to the preceding settings relatively rapidly.**

Acute deterioration on MV:

- The most common causes
  - ✓ Equipment failure: inlet gases, ventilator, circuit leak or obstruction, etc
  - ✓ ETT blockage (secretions, blood) or displacement
  - ✓ Patient (most notably pneumothorax; also bronchospasm, asynchrony, pulmonary hemorrhage, pulmonary oedema, pulmonary embolism and collapse. Some of these may cause less sudden deterioration
- If the cause is not immediately obvious, a trial of bag & tube ventilation will immediately by-pass ventilator, circuit or source gas failure. Further, it will enable assessment of resistance, chest rise and auscultation.
- A blocked tube should be removed and patient ventilated by bag and mask until reintubation is done.
- An obvious tension pneumothorax with failure of ventilation should be immediately drained (using needle thoracentesis if appropriate) without unnecessary delays. A chest tube will then be required (needle will not be enough during on-going MV)

Beyond adjustment of settings:

- If the patient's condition is deteriorating/ not responsive despite increasing/high settings in absence of a rapidly correctable factor, also consider

Lung Recruitment Maneuver

More conservative goals:

- Permissive hypercapnia or hypoxemia. Accepting lower than usual goals of ventilation & oxygenation (eg PaCO2 60-65 provided pH>7.15, SpO2 86-88%) may be appropriate in those with severe lung pathology so long they do not lead to a decompensation.

Note:

- **Permissive hypercapnia requires heavy sedation & frequently paralysis. These goals are NOT TO BE USED in those with CNS injury**

Using a different mode:

- As CMV, HFV, ECMO

WEANING

The following general principles apply:

- Once the patient is ventilated and stable, assess readiness for weaning (decreasing support) at least daily. Based on

  (a) Improvement of the underlying cause

  (b) Condition of other systems eg hemodynamics, metabolic, neurological

  (c) Absence of neuromuscular blockade and absence of heavy sedation. It is helpful to interrupt sedation for 1-3hrs daily to judge consciousness and respiratory drive

  (d) Patient's ability to tolerate a reduction in support (eg rate in SIMV, ΔP in PS) without excessive RR or effort
- When settings are low enough, a spontaneous breathing trial (without ventilator support) can be used to judge the patient's ability to breathe spontaneously. Judge based on ventilation, oxygenation and WOB. Trials should not be prolonged (1-2h are usually enough)
- FiO2 <40%, PEEP ≤5, PS ≤10, OI<5
- Patients on long-term MV are more difficult to wean so a more gradual process is needed
- Any correctable factors should be addressed before extubation (volume, BP, temp, electrolytes, P, Mg, hemoglobin, etc)
- Be prepared to manage laryngeal oedema and to reintubate if necessary. Those on MV for significant periods may be weaned to mask CPAP

Poisoning Protocol

Important Toxicology mnemonics

Miosis causes: COPS
- Cholinergic, Clonidine
- Opiates, Organophosphates
- Phenothizine, Pilocarpine
- Sedatives, Barbiturates

Mydriasis causes: AAAS
- Antihistamines
- Antidepressant
- Anticholinergic
- sympathomimetics (Amphetamines, Cocaine, PCP)

Diaphoretic skin causes: SOAP
- Symathomimetics
- Organophosphates
- Aspirin
- PCP

Red skin causes
- Carbon monoxide
- Boric acid

Bkue skin causes
- Cyanosis
- Methemoglobinemia

↓HR, ↓BP causes
- B-Blockers
- Calcium channel blockers
- Digoxin
- Narcotics

Seizure toxins: OTIS CAMPBELL
- Organophosphates
- TCA
- INH
- Sympathomimetic
- Camphor
- Amphetamines
- Methylzanthines
- PCP, Propnolol, Phenol
- Bzp withdrawal
- Lithium, Lead
- Lidocaine, Lindane

Coma Toxins: LLETHARGIC
- Lwad, Lithium
- Ethanol, Ethylene glycols
- TCAs, Toluene
- Heroin, Heavy metals, Hypoglycemics
- Antipsychotics, Antihistamine
- Respiridone
- GHBB
- INH, Insulin
- Carbon monoxide, Cyanide, Clonidine

↑Anion Gap causes: MUDPILE CATS
- Methanol
- Uremia
- DKA
- Paraldehyde, Phenoformin
- Iron, INH
- Lactic acidosis
- Ethylene glycol
- ASA, Alcohol
- Toluene
- Solvents

"One Pill Can Kill"
- Cardiovascular drugs (-blockers, Ca-Channel antagonist)
- Antidepressants
- Antipsychotics
- Anticonvulsants
- Antiarrhythmic
- Salicylates
- Oral hypoglycemic agents
- Opioids

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Acute Acetaminophen (paracetamol) poisoning

Toxic Single dose ≥ 150mg/kg or 7.5 g

Symptoms (Started in stage II-24 to 72 hrs after overdose):
- Right upper quadrant pain, hepatic tenderness +/- Hepatomegaly
- Hepatotoxicity (↑Liver enzymes,↑PT, INR)
- Nephrotoxicity (oliguria, ↑urea, ↑creatinin) in severe cases
- ↑Amylase, ↑Lipase +/- clinical pancreatitis

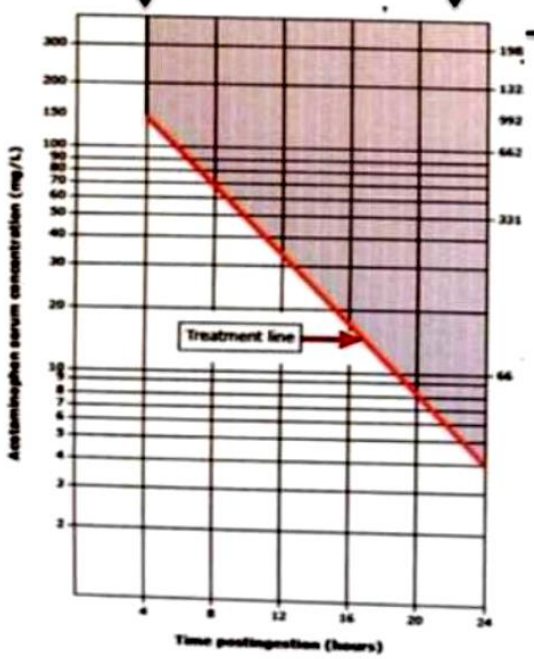
ABC

Check if child has **any risks for hepatotoxicity:**
- Fasting or malnutrition
- Co-ingestion of other drugs (especially trimethoprim/ Sulfa, or rifampin)
- Underlying medical condition or liver disease

Within 4 hours

If > 4 hours Take urgent Paracetamol level

- Activated Charcol (PO) 1g/kg (max. 50 g/Dose) unless contraindicated
Gastric lavage NOT recommended



If toxic range, start antidote: N-acetyleysteine (NAC)

Oral NAC 72 hrs course **(US regimen)**
- Loading NAC orally: 140mg/kg followed by 17 doses of 70 mg/kg/dose every 4hrs total 1330 mg/kg)

To help tolerating, dilute it to 5% solution in cola or juice, covering the cup & drink with straw or may use ondasetrone

IV NAC: 21 hrs course (UK regimen)
(Dilute with D5% 1/2 NS)
- **≤ 20 kg**

Loading: 150 mg/kg dilute in 3 ml/kg over 1 hr
2nd dose: 50mg/kg dilute in 7 ml/kg over 4 hrs
3rd dose: 100 mg/kg in 14 ml/kg diluent over 16 hours
- **20-40 kg**

Loading: 150 mg/kg 100 ml diluent over 1 hr
2nd dose: 50 mg/kg in 250 ml diluent over 4 hrs
3rd dose: 100 mg/kg in 500 ml diluent over 16 hours
- **>40 kg (refer to adult dose)**

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Hydrocarbon Ingestion

Found in:
- **Petroleum distillates** (Kerosene, gasoline, mineral seal oils and naphtha: e.g. furniture polish, lighter fluids, lamp oil)
- **Turpentine** (e.g. pine oil) (↑Aspiration Risk)
- **Aromatic hydrocarbons** (Benzene, Toluene, xylene)
- **Chloroform** e.g. glue, nail polish, paint) (↑ Systemic Risk)

Signs & Symptoms:
Vitals: Fever
Respiratory: Choking, Cough, Wheeze, ↓SpO2, cyanosis, Respiratory compromise & Chemical pneumonitis
CNS: CNS depression, seizure, euphoria, disorientation, hallucinations & come
Cardiac: Arrhythmia 2° to ↑ sensitization to endogenous (+ exogenous) catecholamines leading to VF
GI: Irritation of esophagus → intestines causing GI bleeds & haematemesis.

Investigations
- CBC (↑WBCs)
- RFT, Mg, K, glucose, LFT (↑ALT, AST)
- BGA (metabolic alkalosis, hypoxia, later acidosis)
- Urine analysis
- ECG (arrhythmia, VF)
- Serial CXR (at presentation & ≥ 4 hrs post-exposure

Immediate admission if:
- **Symptomatic**
- **Suicidal intent**
- **Massive ingestion**

AVOID activated charcoal and AVOID induce emesis (Risk of aspiration)

- Ensure **ABCs, 100% Oxygen**
- **Salbutamol** if clinically indicated
- Continuous **cardiac monitor**
- **NPO & IV fluids** maintenance
- **External Decontamination** - remove contaminated clothes, cleanse affected hair and skin, copious water irrigation of eyes as indicated

IF **Respiratory failure/ CNS depression:**
- Intubation & ventilation

IF **Seizures**
- Give **Lorazepam** 0.1 mg/kg IV (Ma. 5 mg/ DOSE)

IF **Arrhythmia/ VF**
- Correct electrolytes (Mg, K)
- IF **VF:** refer to VF algorithm (CPR/ Defibrillator), **Lidocaine** 1 mg/kg infusion rate 20-50 mcg/min or use **–β-blocker**

IF **Pneumonitis**
- **Antibiotics** if fever or leukocytosis 48 hrs post-exposure or ↑ CXR infiltrates or positive sputum/ treacheal bacterial culure
- **Antibiotics** should **NOT** be used prophylactically

ONLY if high risk of systemic toxicity, or large amounts ingested, within the past 60 mins, consider Nasogastric lavage under **balloon cuffed ETT** whilst patient lying on left lateral position (↓risk of aspiration during procedure)

AVOID Epinephrine sensitization to catecholamine arrhythmias →

N.B: Corticosteroid should **NOT** be used (has been associated with ↑morbidity)

Observe for 6 hours in the ED & repeat CXRI IF:
- Asymptomatic & normal CXR: **Discharge,** appropriate instructions to return if fever, tachypnea or cough develops.
- Asymptomatic & abnormal CXR: **OPD follow-up after 24 hours.**

Symptomatic or cannot guarantee close follow-up: **Admit to ward for observation & supportive management.**

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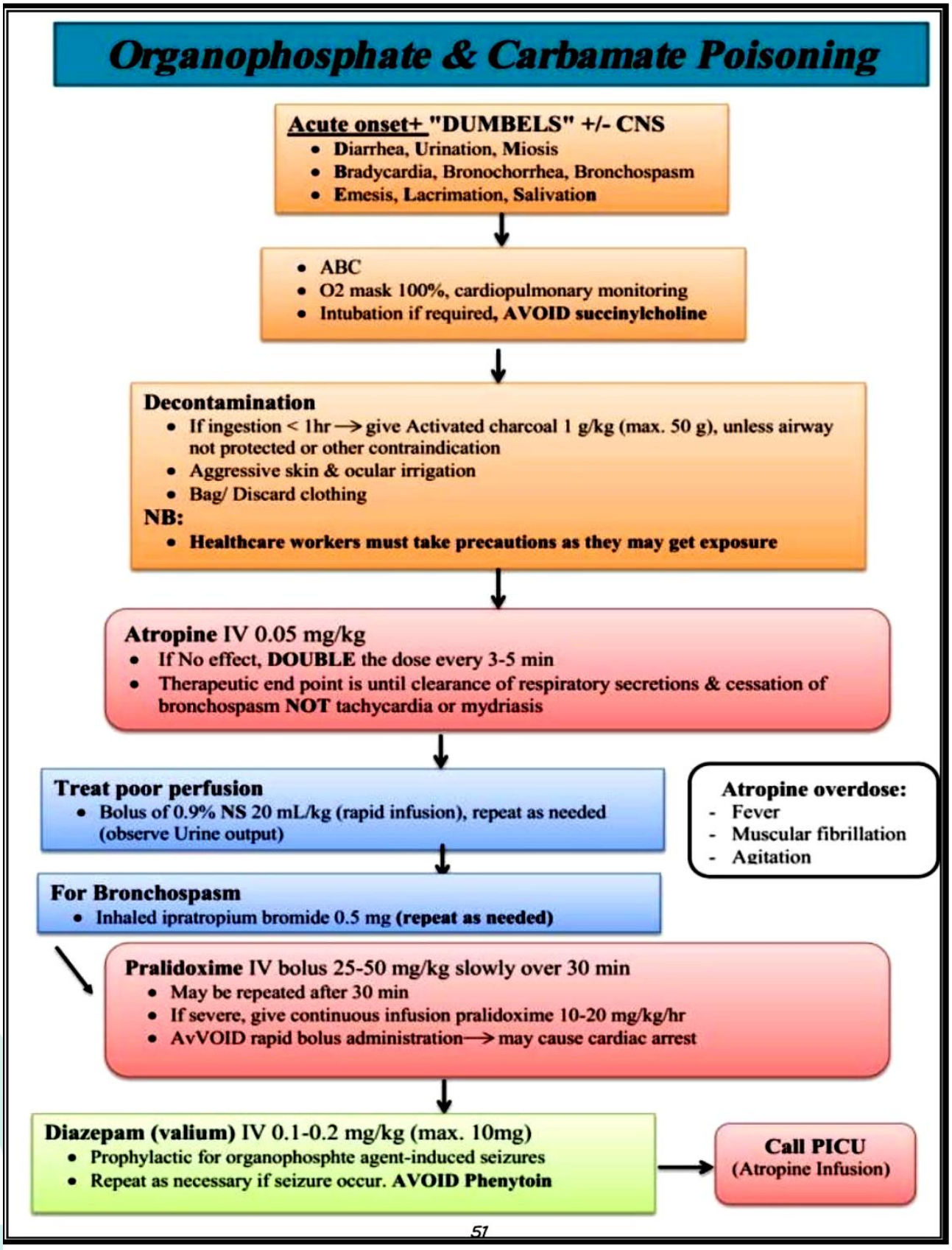
Organophosphate & Carbamate Poisoning
Acute onset+ "DUMBELS" +/- CNS
Diarrhea, Urination, Miosis
Bradycardia, Bronochorrhea, Bronchospasm
Emesis, Lacrimation, Salivation
ABC
O2 mask 100%, cardiopulmonary monitoring
Intubation if required, AVOID succinylcholine
Decontamination
If ingestion < 1hr → give Activated charcoal 1 g/kg (max. 50 g), unless airway not protected or other contraindication
Aggressive skin & ocular irrigation
Bag/ Discard clothing
NB:
Healthcare workers must take precautions as they may get exposure
Atropine IV 0.05 mg/kg
If No effect, DOUBLE the dose every 3-5 min
Therapeutic end point is until clearance of respiratory secretions & cessation of bronchospasm NOT tachycardia or mydriasis
Treat poor perfusion
Bolus of 0.9% NS 20 mL/kg (rapid infusion), repeat as needed (observe Urine output)
Atropine overdose:
- Fever
- Muscular fibrillation
- Agitation
For Bronchospasm
Inhaled ipratropium bromide 0.5 mg (repeat as needed)
Pralidoxime IV bolus 25-50 mg/kg slowly over 30 min
May be repeated after 30 min
If severe, give continuous infusion pralidoxime 10-20 mg/kg/hr
AvVOID rapid bolus administration → may cause cardiac arrest
Diazepam (valium) IV 0.1-0.2 mg/kg (max. 10mg)
Prophylactic for organophosphte agent-induced seizures
Repeat as necessary if seizure occur. AVOID Phenytoin
Call PICU
(Atropine Infusion)
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PICU

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