



Pediatric Cardiology Protocol of EHA



First Edition 2024



Egyptian Clinical Practice Protocol
in
Pediatric Cardiology
for
Egypt Healthcare Authority
First Edition
2024

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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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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 Cardiology is to unify and standardize the delivery of healthcare to all pediatric patients at all health facilities.

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.

Standard textbooks, journals, and online resources currently available create excellent repositories of detailed information about the etiology, pathogenesis, clinical picture, diagnosis, and treatment of a condition. However, for a busy clinician looking for the best way to manage a sick patient, a standardized path for effective management of the patient may be impossible to discern. So, it would be a lot easier if we all managed simple things in a uniform way using the best available evidence and resources.

For the management of pediatric cardiology patients, 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 where high-grade evidence is not available.

In this protocol, we offer comprehensive reviews of selected topics and comprehensive advice about management approaches based,

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, nurse 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.

***Members of the Working Group
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In Pediatric Cardiology***

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List of Abbreviations

<i>ECG</i>	<i>Electro Cardio Gram</i>
<i>VF</i>	<i>Ventricular fibrillation</i>
<i>TGA</i>	<i>Transportation of the great vessels</i>
<i>TOF</i>	<i>Tetralogy of Fallot</i>
<i>TAPVC</i>	<i>Total anomalous pulmonary venous connection</i>
<i>COA</i>	<i>Coarctation of aorta</i>
<i>PDA</i>	<i>Patent ductus arteriosus</i>
<i>AR</i>	<i>Aortic regurge</i>
<i>AV</i>	<i>Arterio-venous fistulas</i>
<i>KD</i>	<i>Kawasaki disease</i>
<i>FBC</i>	<i>full blood count</i>
<i>UEC</i>	<i>Urea ,electrolytes, creatinine</i>
<i>LFT</i>	<i>Liver function test</i>

Approach to a Child with Suspected Arrhythmia

Symptoms:

- Any suspected cardiac symptom particularly palpitations and chest pain are an indication for rhythm evaluation.

Signs:

- A regular pulse with a rate normal for age is essential in any clinical exam, cardiac or otherwise.
- In the presence of any rhythm disturbance **a reference of pediatric heart rate and normal of ECG is essential** in evaluation, a 12-lead is the standard. (Vitals are shown in table p.6)

N.B:

1. **A monitor rhythm strip is not a replacement for an ECG.**
2. **During the evaluation of any cardiac disease, a 12 leads ECG is essential. If the cardiac disease is chronic, a Holter is needed as well.**
3. **While asystole represents 80% of pediatric arrests, an ECG monitor is needed in every resuscitation to detect the common asystole, less common ventricular fibrillation (19%) or the uncommon electro-mechanical dissociation (1%).**
4. **Whenever in doubt, consult a specialist or a senior person.**
5. **Children have been known to fully recover after prolonged CPR if effectively done, do not lose hope early.**



PEDIATRIC VITAL SIGNS REFERENCE CHART



Heart Rate (beats/min)			Respiratory Rate (breaths/min)	
Age	Awake	Asleep	Age	Normal
Neonate (<28 d)	100-205	90-160	Infant (<1 y)	30-53
Infant (1-12 mos)	100-190			
Toddler (1-2 y)	98-140	80-120	Toddler (1-2 y)	22-37
Preschool (3-5 y)	80-120	65-100	Preschool (3-5 y)	20-28
School-age (6-11 y)	75-118	58-90	School-age (6-11 y)	18-25
Adolescent (12-15 y)	60-100	50-90	Adolescent (12-15 y)	12-20

Reference: PALS Guidelines, 2015

Blood Pressure (mmHg)				
Age		Systolic	Diastolic	Systolic Hypotension
Birth (12 h)	<1 kg	39-59	16-36	<40-50
	3 kg	60-76	31-45	<50
Neonate (96 h)		67-84	35-53	<60
Infant (1-12 mos)		72-104	37-56	<70
Toddler (1-2 y)		86-106	42-63	<70 + (age in years × 2)
Preschool (3-5 y)		89-112	46-72	
School-age (6-9 y)		97-115	57-76	
Preadolescent (10-11 y)		102-120	61-80	<90
Adolescent (12-15 y)		110-131	64-83	

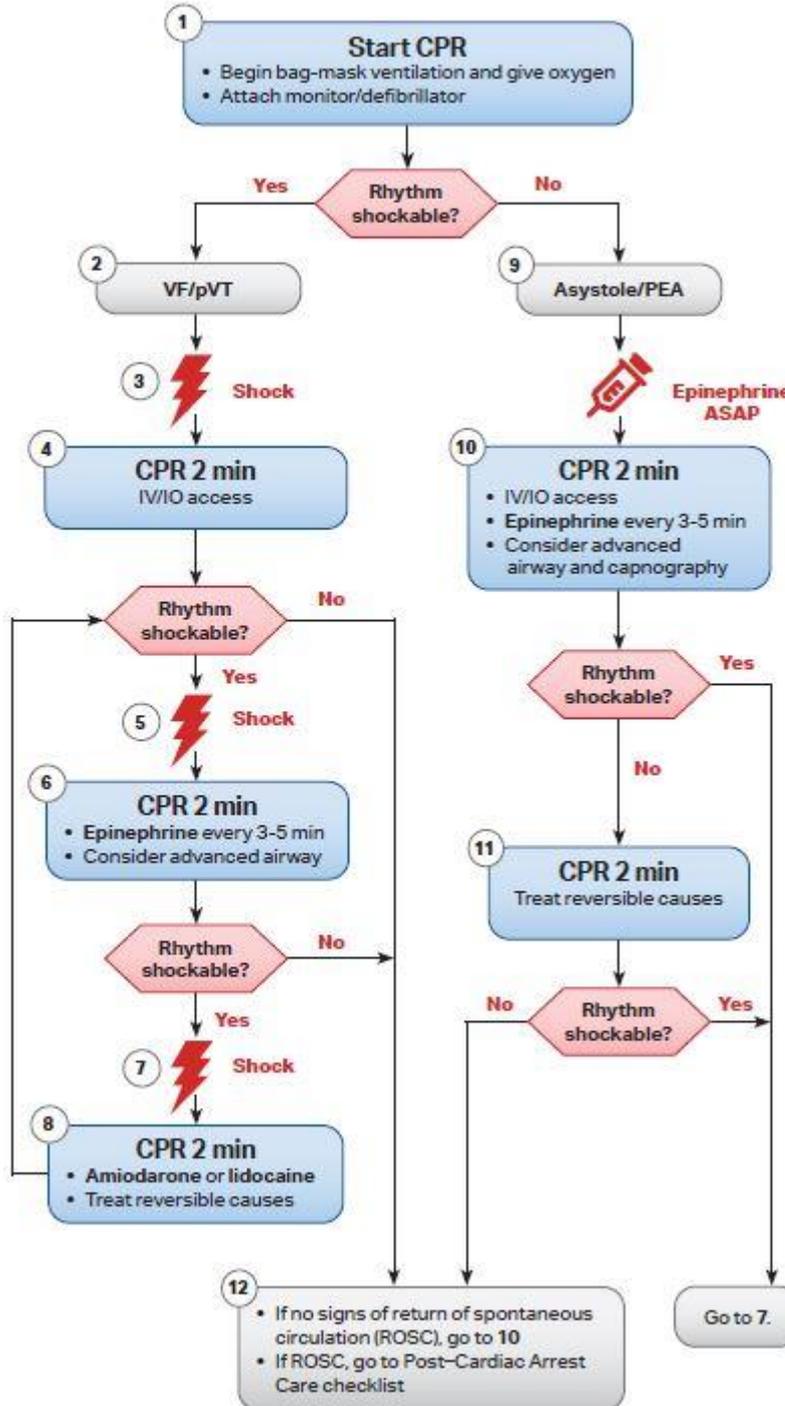
Reference: PALS Guidelines, 2015
 For diagnosis of hypertension, refer to the 2017 AAP guidelines Table 4 & 5:
<http://pediatrics.aappublications.org/content/early/2017/08/21/peds.2017-1904>

Temperature (°C)		Oxygen Saturation (SpO ₂)
Method	Normal	
Rectal	36.6-38.0	SpO ₂ is lower in the immediate newborn period. Beyond this period, a SpO ₂ of <90-92% may suggest a respiratory condition or cyanotic heart disease .
Tympanic	35.8-38.0	
Oral	35.5-37.5	
Axillary	36.5-37.5	
Ranges do not vary with age. Screening: axillary, temporal, tympanic (↓ accuracy) Definitive: rectal & oral (↑ reflection of core temp.) <i>Reference: CPS Position Statement on Temperature Measurement in Pediatrics (2015)</i>		

Dr. Chris Novak & Dr. Peter Gill for www.pedscases.com
 (Edited March 2020 by Richard He)

Pediatric Advanced Life Support

Pediatric Cardiac Arrest Algorithm



CPR Quality

- Push hard (≥1/3 of anteroposterior diameter of chest) and fast (100-120/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Change compressor every 2 minutes, or sooner if fatigued
- If no advanced airway, 15:2 compression-ventilation ratio
- If advanced airway, provide continuous compressions and give a breath every 2-3 seconds

Shock Energy for Defibrillation

- First shock 2 J/kg
- Second shock 4 J/kg
- Subsequent shocks ≥4 J/kg, maximum 10 J/kg or adult dose

Drug Therapy

- **Epinephrine IV/IO dose:** 0.01 mg/kg (0.1 mL/kg of the 0.1 mg/mL concentration). Max dose 1 mg. Repeat every 3-5 minutes. If no IV/IO access, may give endotracheal dose: 0.1 mg/kg (0.1 mL/kg of the 1 mg/mL concentration).
- **Amiodarone IV/IO dose:** 5 mg/kg bolus during cardiac arrest. May repeat up to 3 total doses for refractory VF/pulseless VT
- **Lidocaine IV/IO dose:** Initial: 1 mg/kg loading dose

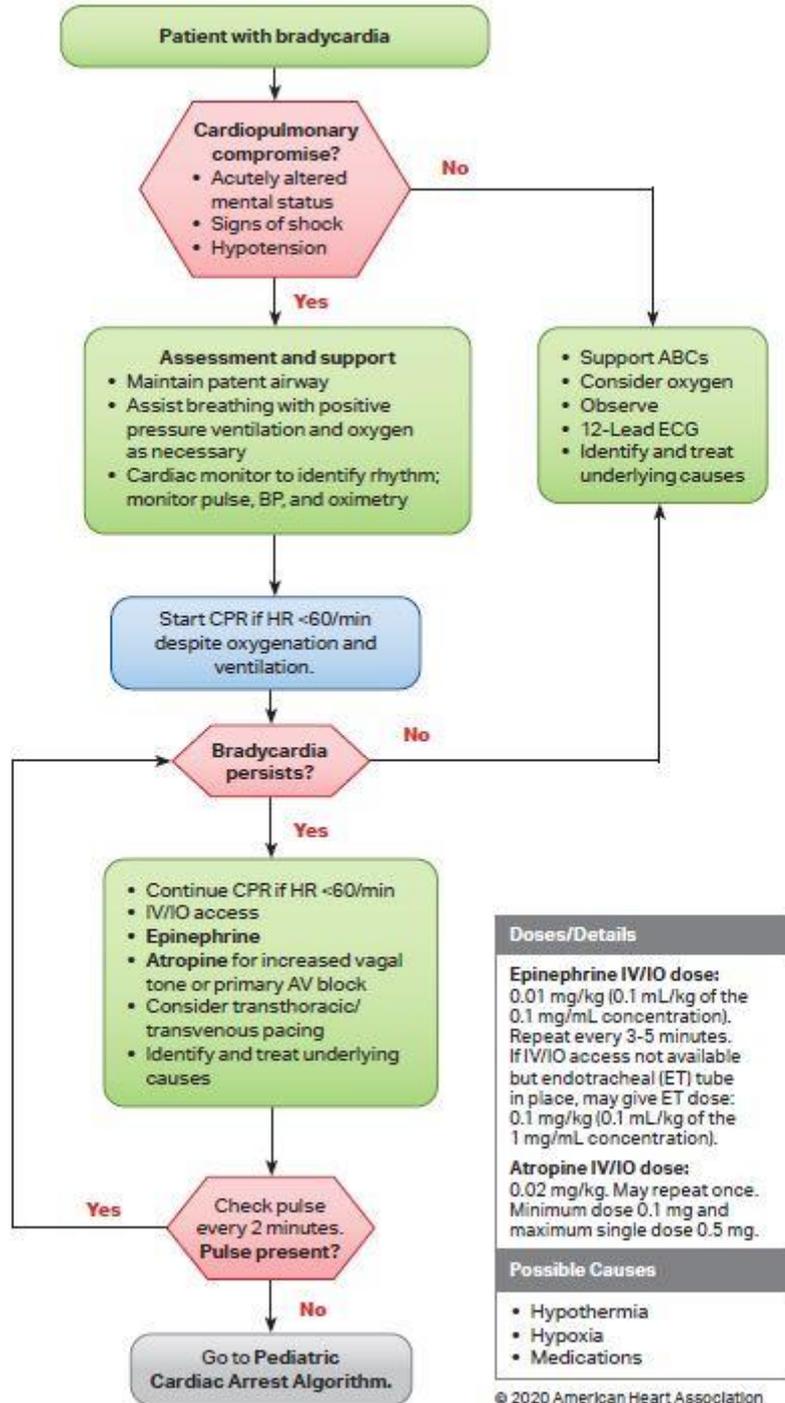
Advanced Airway

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement

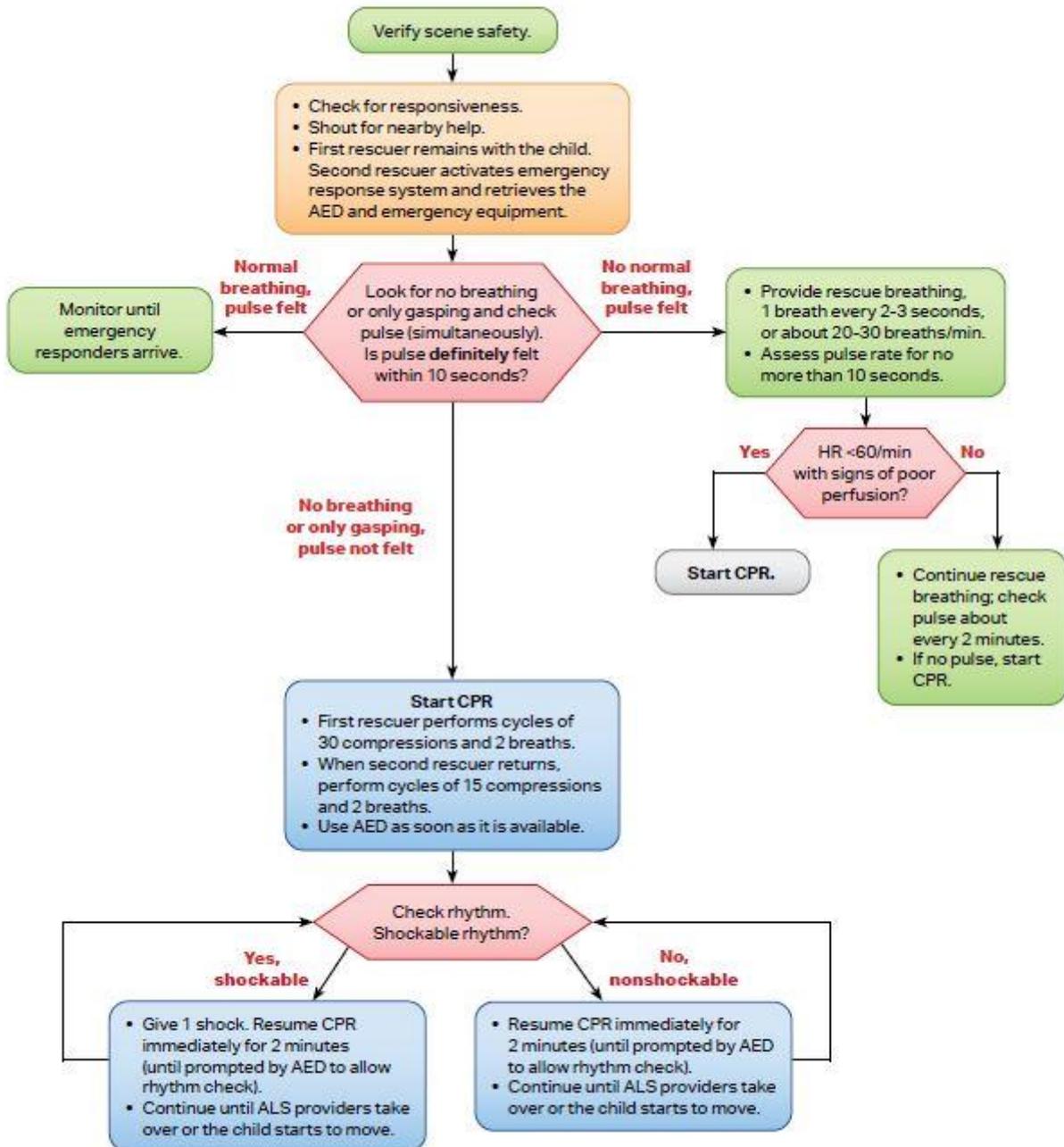
Reversible Causes

- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypoglycemia
- Hypo-/hyperkalemia
- Hypothermia
- Tension pneumothorax
- Tamponade, cardiac
- Toxins
- Thrombosis, pulmonary
- Thrombosis, coronary

Pediatric Bradycardia With a Pulse Algorithm

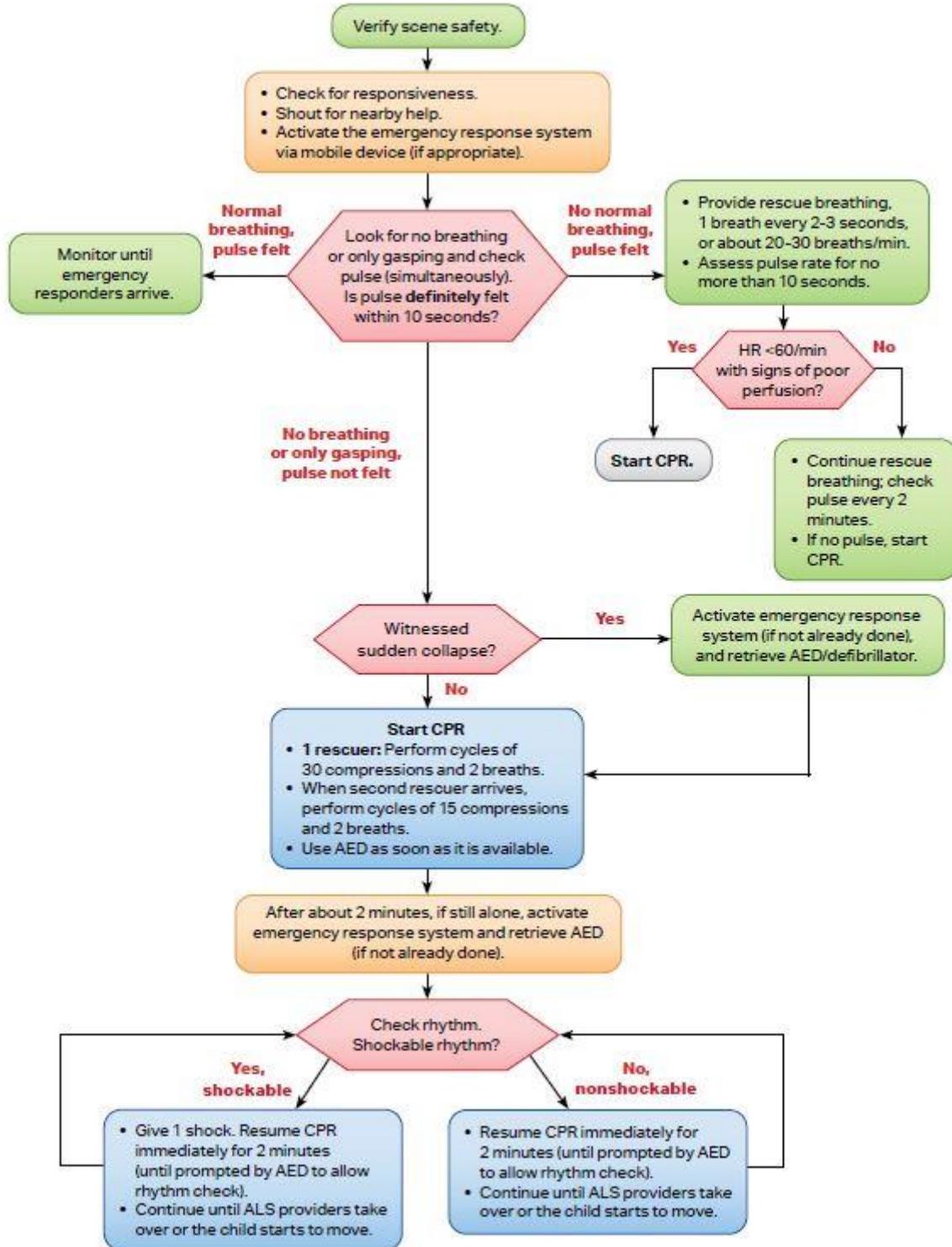


Pediatric Basic Life Support Algorithm for Healthcare Providers—2 or More Rescuers

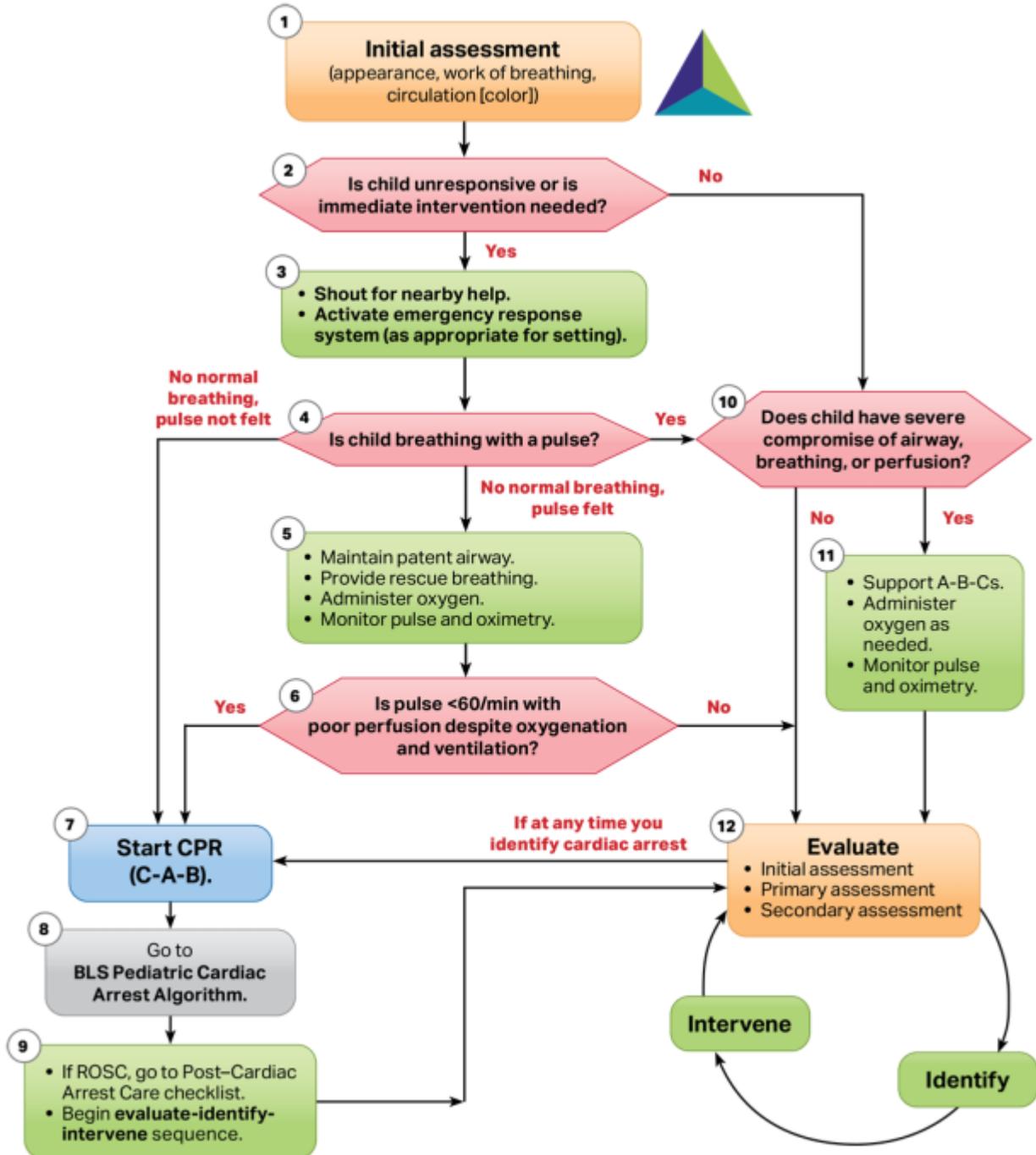


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Pediatric Basic Life Support Algorithm for Healthcare Providers—Single Rescuer



PALS Systematic Approach Algorithm



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Syncope in Pediatrics

Causes:

- ✓ *Neurally mediated: most common, benign (70%)*
- ✓ *Cardiac: arrhythmia or structural heart disease*
- ✓ *Neurological: seizures, head injury*
- ✓ *Orthostatic hypotension*
- ✓ *Metabolic: dehydration, hypoglycemia*

Red Flags for Cardiac Syncope:

- ✓ *Lack of a prodrome*
- ✓ *Palpitation or chest pain*
- ✓ *Exercise induced syncope*
- ✓ *Past cardiac history*
- ✓ *Family history of early cardiac death, arrhythmia or sudden death*

Work Out:

1. History and Physical Examination:

History and Physical Findings	RED LIGHT Recommend Urgent Referral to Syncope Specialist	YELLOW LIGHT Consider Further Investigations or Semiurgent Referral to Syncope Specialist	GREEN LIGHT Reassuring for Nonserious Cause; No Further Investigation Required
Hydration status and timing of most recent meal			Missed meals, poor fluid intake
Environmental conditions	Syncope triggered by loud noise-look for long QT and refer if ECG positive		Painful stimulus, sight of blood, very warm environment
Activity preceding the syncopal event	Midexertional syncope (consider cardiac causes) Syncope while swimming (might be associated with LQTS)		Postexertional syncope Prolonged standing
Use of drugs and medications		Medications that may prolong QT (refer if ECG abnormal)	No medications
Prodrome	No prodrome (concerning for arrhythmia)	Short or atypical prodrome	Warmth, nausea, light-headedness, a visual grey-out or tunneling of vision
Other symptoms		Acute chest pain followed by syncope Palpitations just before syncope	
Position of child preceding event		Supine (consider seizure)	Prolonged or recent standing Position change from seated or lying to standing
Duration of loss of consciousness		Prolonged > 5 minutes (consider seizure or somatization)	Short; < 1-2 minutes
Movement during event	Tonic-clonic movements or motor activity preceding LOC (consider seizure)	Exaggerated or flailing movements (consider somatization)	Myoclonic jerks after loss of consciousness
History (previous syncopal events, cardiac disease, diabetes, seizures, and psychiatric or psychological problems)	Arrhythmia, structural heart disease Seizures	Diabetes Psychiatric disorder or medications Significant comorbidities	No relevant medical history Previous events consistent with vasovagal syncope or breath-holding spells
Family history (structural cardiac disease, arrhythmias, sudden death, migraines, or seizures)	Sudden death Arrhythmias	Seizures Structural heart disease	Vasovagal syncope Migraines
Focused cardiac and neurologic examinations	Pathologic murmur Sternotomy scar Persistent neurologic deficits (consider stroke, seizure, migraine)		Normal examination Typical flow murmur

2. Blood Glucose

3. CBC

4. ECG

	ECG Findings
RED LIGHT	<ul style="list-style-type: none"> • Abnormal QT interval* <ul style="list-style-type: none"> ○ Long QT interval (QTc > 470 ms) ○ Short QT interval (QTc ≤ 330 ms) • Type 1 Brugada pattern • Delta wave (ventricular pre-excitation or Wolff-Parkinson-White syndrome) • Signs of myocardial ischemia (ST-T wave changes, Q waves > 1 mm wide) • PVCs, polymorphic • Third-degree AV block
YELLOW LIGHT	<ul style="list-style-type: none"> • Left ventricular hypertrophy (including left axis deviation, tall R wave in V₆, tall S wave in V₁, deep Q waves in II, III, and aVF and ST-T wave changes) • PVCs, monomorphic • Second-degree AV block • Heart rate < 40 bpm in normally nourished, nonathletic individual
GREEN LIGHT	<ul style="list-style-type: none"> • Sinus arrhythmia • Wandering atrial pacemaker; atrial or junctional rhythm • First-degree AV block • Negative T waves in right precordial leads • Early repolarization • Incomplete right bundle branch block

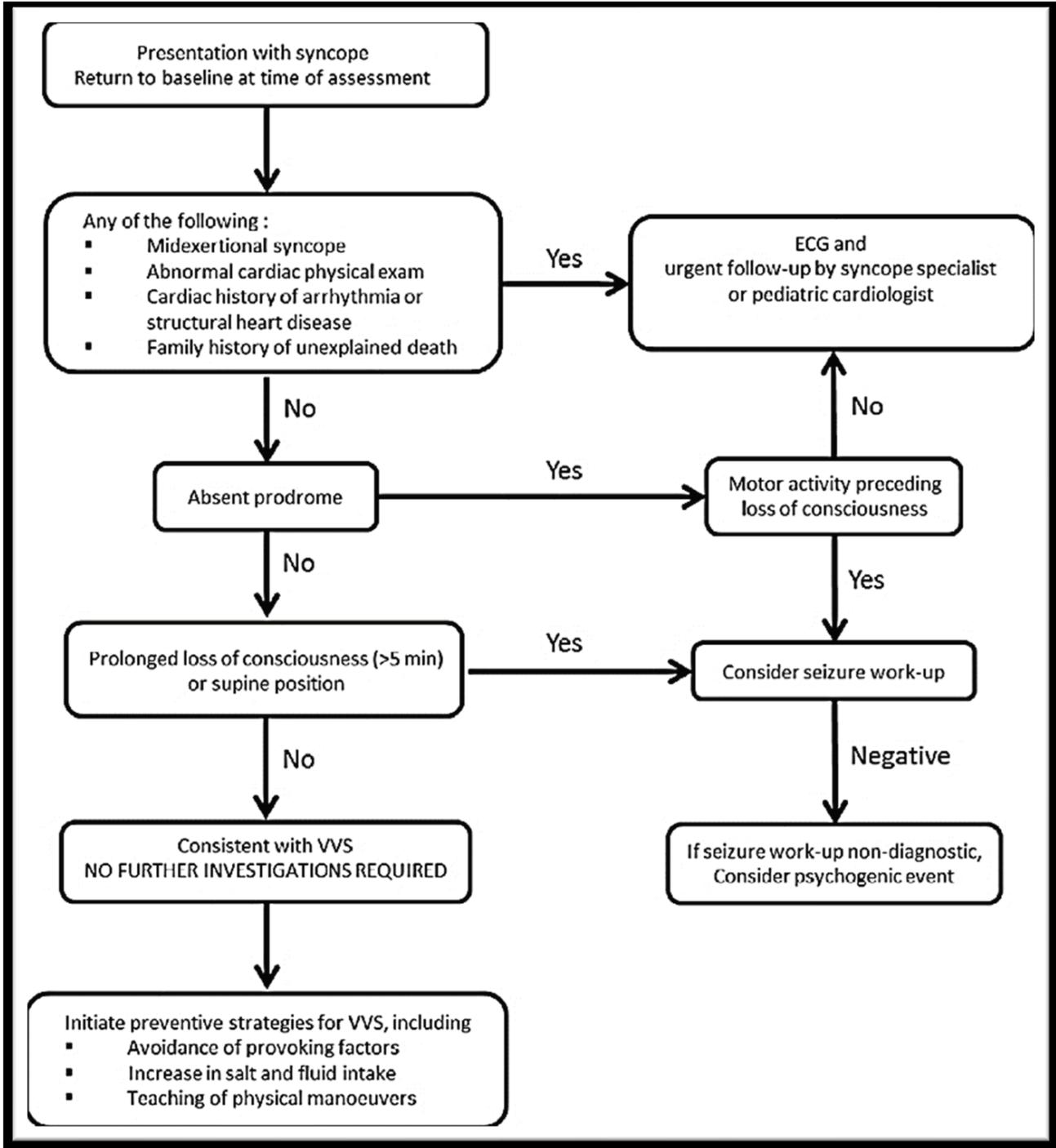
5. Echocardiography

6. If arrhythmia is suspected 24-hour Holter

7. If history is not conclusive and neurocardiogenic syncope is suspected, do tilt table testing

“Data collected from history, examination and investigations will be interpreted through the following chart”

Chart for interpretation of data of patient with syncope



“Neurocardiogenic syncope is the most common form, and it is benign”

Treatment:

- The patient is advised to avoid dehydration, long periods of standing and irregular mealtimes. Other simple measures include water and table salt intake to increase plasma volume.
- Some people have been able to abort an episode of vasovagal syncope by immediately engaging in muscle-tensing exercises. These exercises apparently reduce blood vessel dilation and increase the amount of blood being returned to the heart. Examples include:
 - Crossing your leg while tensing the legs, abdomen, and buttocks
 - Tensing the arms with clenched fists
 - Tensing your leg muscles
 - Squeezing a rubber ball
- When syncope persists despite behavioral changes, medical therapy can be used and usually includes:
 - Beta-blocker or
 - Fludrocortisone
 - Serotonin re uptake inhibitor (paroxetine)

“Patients with cardiac or neurological syncope should be referred to a specialist”



References:

1. 2018 ESC Guidelines for the diagnosis and management of syncope.
2. Michele Brignole, Angel Moya, Frederik J de Lange, Jean-Claude Deharo, Perry M Elliott, Alessandra Fanciulli, Artur Fedorowski, Raffaello Furlan, Rose Anne Kenny, Alfonso Martín ... Show more Author Notes.
3. European Heart Journal, Volume 39, Issue 21, 01 June 2018, Pages 1883–1948, <https://doi.org/10.1093/eurheartj/ehy037>- Published: 19 March 2018.

An Approach to A Cyanotic Neonate

Cyanosis Definition:

Blue coloration of the skin and mucous membranes due to the presence of deoxygenated hemoglobin occurs when the oxygen saturation of arterial blood falls below 85-90% (5g/dl deoxyhemoglobin).

- **Central Cyanosis:** Affects “central” parts of the body (mouth, tongue, head and torso).
- **Peripheral Cyanosis:** Appears at the hands, fingertips, toes +/- circumoral and periorbital areas, it is rarely life-threatening.
- **Differential Cyanosis:** It is cyanosis at both lower extremities with a pink right upper extremity. (**Lundsgaard and Van Slyk - 1928**)
- **Causes:**

Respiratory Causes:

- ✓ Aspiration
- ✓ Pierre robin syndrome
- ✓ Choanal atresia
- ✓ Hyaline membrane disease
- ✓ Pulmonary edema, pneumonia, pneumothorax, pleural effusion,
- ✓ Congenital Diaphragmatic Hernia

Cardiac Causes:

- ✓ Congenital heart disease presenting since birth e.g. (TGA)+ others
Lundsgaard and Van Slyk

CNS:

- ✓ Central nervous system dysfunction
- ✓ Asphyxia

Miscellaneous:

- ✓ Sepsis
- ✓ Hypocalcemia, hypoglycemia, Hypomagnesemia, Hypothermia

Cyanosis with Normal Po₂:

- ✓ Methemoglobinemia

Cardiac Causes of Cyanosis in the Neonatal Period:

Newborn period	1st week
<ul style="list-style-type: none">• TGA• Tricuspid atresia• TAPVC• Truncus Arteriosus• TOF (severe type)• Pulmonary Atresia with Hypoplastic RV• Hypoplastic RV	<ul style="list-style-type: none">• Pul. Atresia• Tricuspid atresia• TGA
	After 1 month
	<ul style="list-style-type: none">• TOF• TGA• TAPVC

Source:

- *Diagnostic Approach, Dr. D. Muthukumar MD, Medical College, Webpage: <https://slideplayer.com/slide/10500980/>*

Cardiac Evaluation:

Work Up

- ✓ Full history
- ✓ CLINICAL EXAMINATION (blood pressures measurement in all four limbs)
- ✓ PULSE OXIMETER
- ✓ Blood gases analysis & Hyperoxia test
- ✓ Full laboratory tests
- ✓ EEG & X-RAY CHEST.
- ✓ Echocardiography
- ✓ Cardiac catheterization in selected cases
- ✓ Ct angiography in selected cases(**Expert Opinion**).

Important Points During Examination:

- ✓ Inspection; Features denoting certain Syndrome: i.e., Di George Syndrome.
- ✓ Cyanosis & differential cyanosis, pallor, tachypnea, dyspnea, other congenital malformations
- ✓ Palpation: hyperactive precordium is characteristic of high-volume overload as large Lt to Rt shunts or severe valve regurgitation.
- ✓ Thrill is often of real diagnostic value.
- ✓ Peripheral pulses; Strong arm pulses and week leg pulses suggest COA.
- ✓ Bounding pulses are found in aortic run-off lesions eg: PDA, AR, AV fistulas.
- ✓ Weak, thread pulses indicate circulatory shock.
- ✓ Blood pressure: Upper & lower limbs.
- ✓ Auscultation: For heart sounds, audible murmurs
- ✓ Cover the genitalia with a sheet and slightly abduct the thigh. Press deeply, below the inguinal ligament and about midway between symphysis pubis and anterior superior iliac spine. Use two hands one on top of the other to feel the femoral pulse. Note the adequacy of the pulse volume.
- ✓ Pulse Oximetry Screening to Detect Critical Congenital Heart Disease (CHD)&hyperoxia test(**Expert Opinion**).



Hyperoxia Test:

- ✓ It differentiates between cardiac cyanosis and other causes of cyanosis as pulmonary or CNS causes
- ✓ Method: breathing 100% oxygen to the neonate increases arterial blood PO₂ above 100mmhg if cyanosis is due to non-cardiac cause
- ✓ If there is RT to LT shunt arterial PO₂ does not exceed 10 to 30mmhg.

CCHD Protocol:

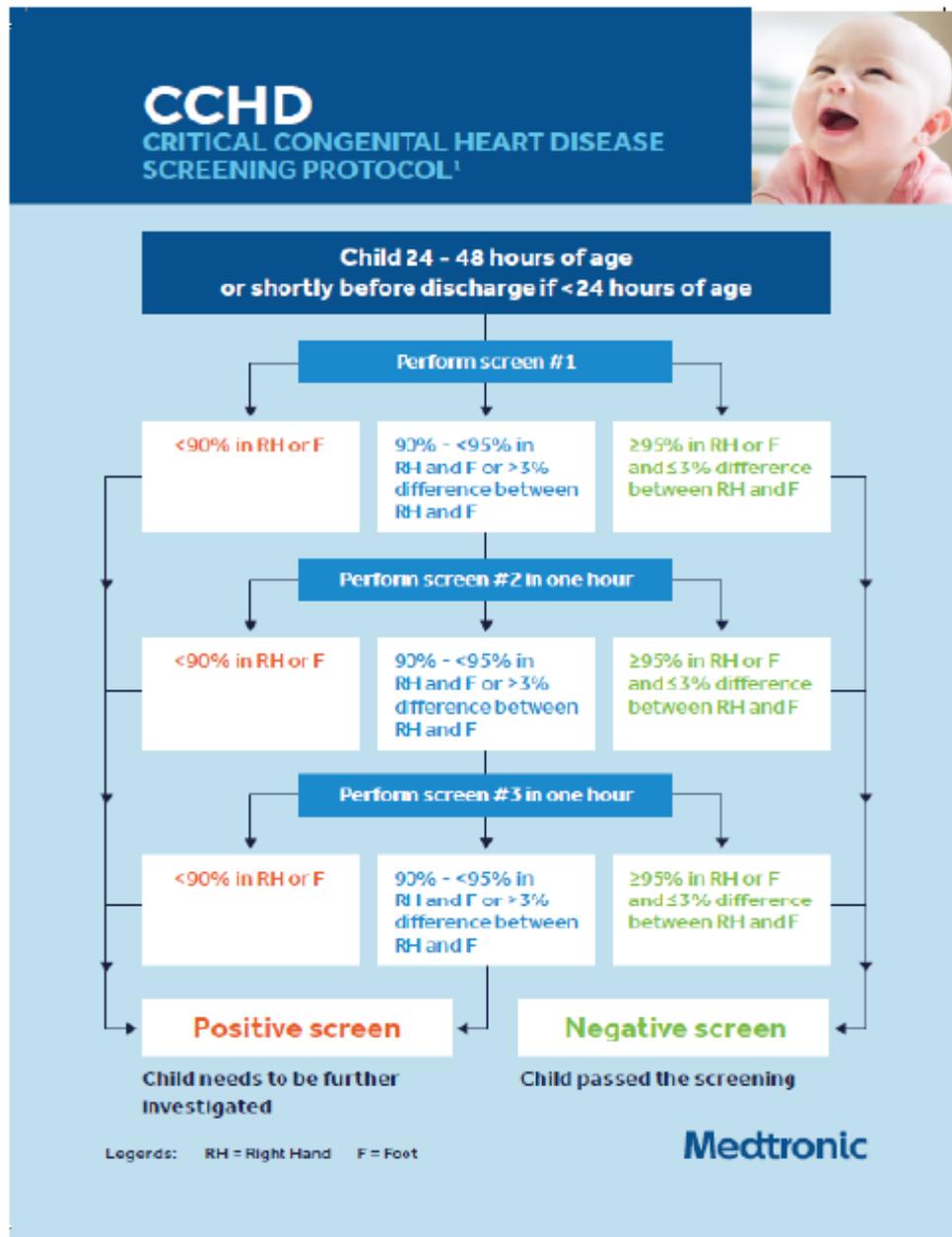
CCHD Screening Protocol

➤ Screening Recommendations:

- Right hand and one foot (parallel or in sequence)

➤ Positive Screening:

- <90%
- <95% in both extremities on 3 measurements, separated by 1 hour
- >3% difference in SpO₂ between right hand and foot on 3 measurements, separated by 1 hour



Source:

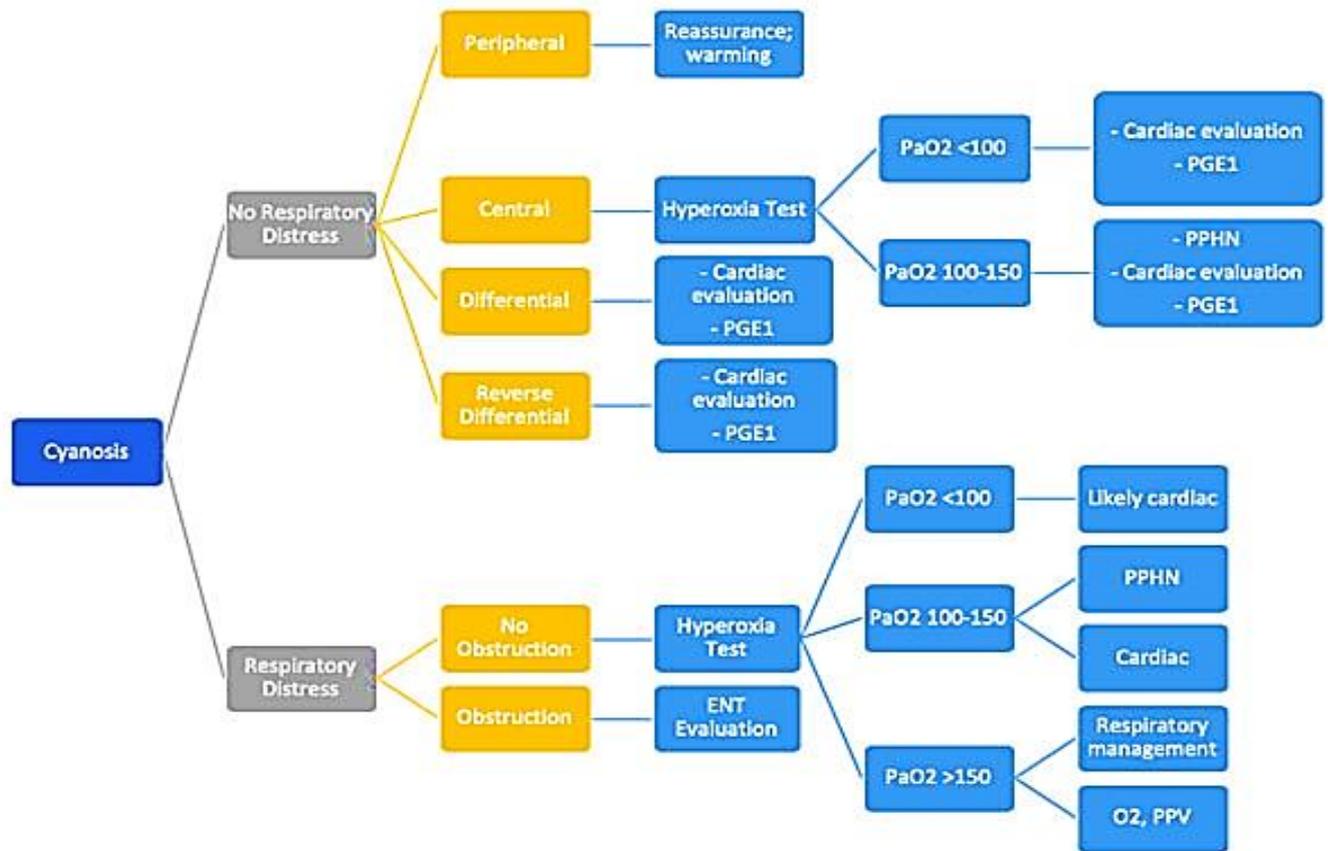
- *Dr. KEMPER'S PROTOCOL USED IN THE US WHERE THE SCREENING HAS BEEN MADE MANDATORY IN SEPT.2011*

Approach to A Cyanotic Neonate		
Low PaO ₂ , SaO ₂		
Cardiac	Symptoms & Signs	Pulmonary or Other
increased cyanosis	CRYING	decreased cyanosis
tachypnea, slow, deep	RESPIRATORY DISTRESS	retractions, grunting, tachypnea, apnea
normal or decreased	PaCO₂	increased
minimal response	FIO₂	responsive (usually)
murmur, weak pulses	CARDIAC EXAM	normal
can be abnormal	ECG	normal
Abnormal	ECHO	normal, pulmonary hypertension
heart abnormal or normal size situs inversus (complex) reduced pulmonary blood flow	CHEST X-RAY	lung disease

Source:

- *Lundsgaard C, Van Slyke DD. Cyanosis. Medicine. 1923;2:1-76.*

Algorithm for Management of Neonatal Cyanosis:



Source:

- Dasgupta, S., Bhargava, V., Huff, M., Jiwani, A. K., & Aly, A. M. (2016).
- Evaluation of The Cyanotic Newborn: Part I—A Neonatologist's Perspective. *NeoReviews*, 17(10), e598-e604

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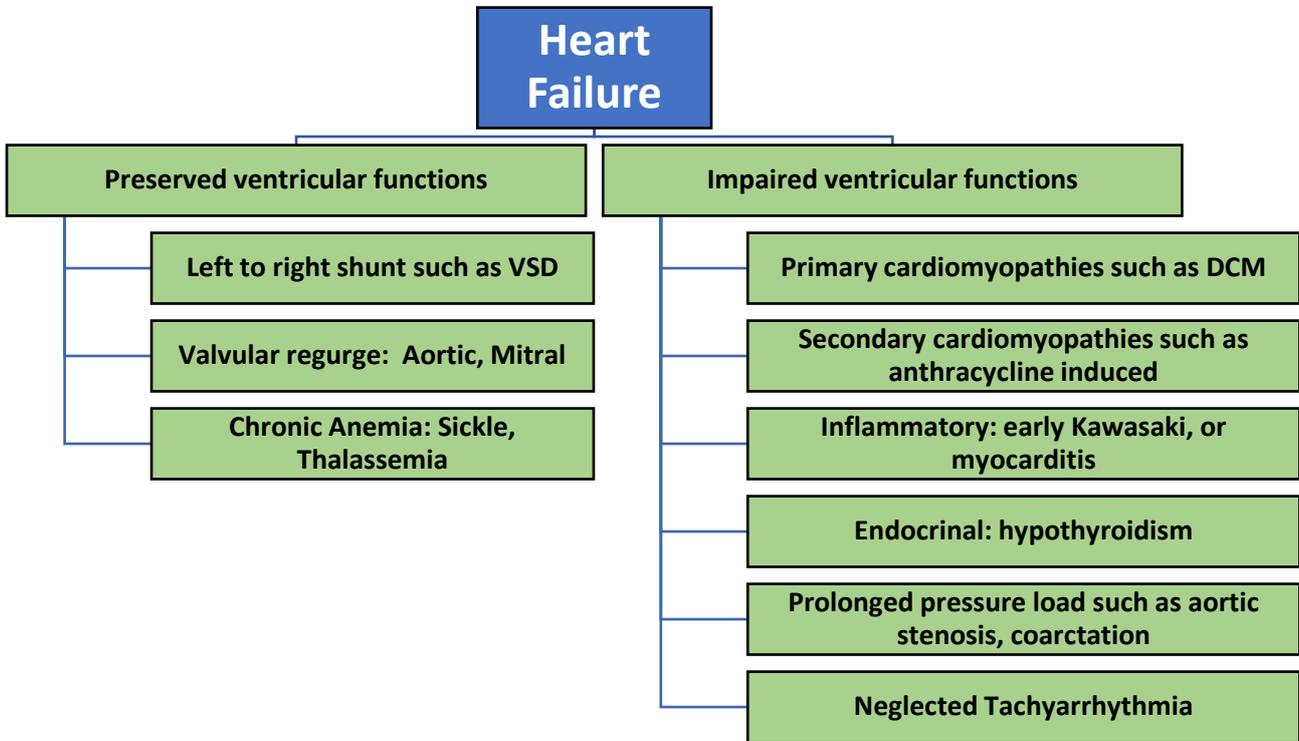
1. Dasgupta, S., Bhargava, V., Huff, M., Jiwani, A. K., & Aly, A. M. (2016).
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5. Dr. KEMPER'S PROTOCOL USED IN THE US WHERE THE SCREENING HAS BEEN MADE MANDATORY IN SEPT.2011
6. Lundsgaard C, Van Slyke DD. Cyanosis. *Medicine*. 1923;2:1–76.

Pediatric Heart Failure

Definition:

- Heart failure is a clinical syndrome based on the presence of clinical manifestations suggestive of inability of the heart to meet the metabolic needs of the body

Causes, when to suspect and severity classification:



Symptoms:

- ✓ Dyspnoea on exertion (poor feeding in infants)
- ✓ Sweating
- ✓ Failure to thrive
- ✓ Recurrent chest infections

Signs:

- ✓ Tachypnoea
- ✓ Tachycardia
- ✓ Weak and thready pulses
- ✓ Gallop rhythm
- ✓ Murmur
- ✓ Edema
- ✓ Cardiomegaly – very useful sign
- ✓ Tender hepatomegaly
- ✓ Basal crackles
- ✓ Cold and wet skin
- ✓ Some children present in extremis (cardiogenic shock)

Clinical Pearl:

- **New-onset heart failure may be less overtly symptomatic in older children. Symptoms of abdominal pain and nausea and anorexia can be present, sometimes diverting attention from the real cause.**

Classifications: Modified Ross Classification

	Infant	Child
Stage I	Asymptomatic with underlying lesion	
Stage II	Mild tachypnoea and diaphoresis on feeding No growth failure	Mild Dyspnea on exertion
Stage III	Marked tachypnoea and diaphoresis on feeding Growth failure	Marked dyspnea on exertion
Stage IV	Diaphoresis at rest	Dyspnea at rest

Management:

- Once suspected refer to tertiary care for X ray, Echocardiography
- Management according to previously mentioned staging:

Stage I	ACE I
Stage II	Add loop diuretics and aldosterone antagonist
Stage III	Refer to Pediatric Cardiology tertiary center of care: The cardiologist might recommend use of advanced treatments such as Ivabradine/Entresto or Carvedilol
Stage IV	ICU admission in tertiary care center with Pediatric Cardiology supervision to start IV inotropes+/- mechanical support

References:

- Masarone, Daniele et al. 2017. “Pediatric Heart Failure: A Practical Guide to Diagnosis and Management.” *Pediatrics & Neonatology* 58(4): 303–12. <https://linkinghub.elsevier.com/retrieve/pii/S1875957217300505>.

Guidelines of Hypertension (HTN)

1) General Considerations:

History:

- ✓ Headache/vomiting
- ✓ Blurred vision
- ✓ Change in mental state
- ✓ Seizures
- ✓ Chest pain/palpitations
- ✓ Shortness of breath
- ✓ Cardiac failure
- ✓ Past history of Acute Kidney Injury (AKI)

Examination:

- Confirm hypertension (See measuring blood pressure section below)
- Vitals: tachycardia, four limb BP for upper and lower limb discrepancy
- Height and weight: obesity, growth retardation
- Signs of end organ damage
 - Fundoscopy: hypertensive retinopathy
 - Cardiovascular: apical heave, hepatomegaly, oedema
 - Chronic renal failure: palpable kidneys
 - Focal neurology (eg facial nerve palsies)
- Signs of underlying cause
 - General appearance: Cushingoid, proptosis, goiter, webbed neck (Turner syndrome), elfin facies (William's syndrome)
 - Skin: Cafe-au-lait spots, neurofibromas, acanthosis nigricans, hirsutism, striae, acne, rash (vasculitis)
 - Cardiovascular: murmurs +/- radiation, apical heave, reduced femoral pulses, oedema, hepatomegaly (CCF)
 - Abdomen: masses, palpable kidneys, flank bruits
 - Genitourinary: ambiguous/virilized genitalia (eg CAH)

2) Ranges of BP to suspect HTN and determination of severity:

- Ensure the correct cuff size is selected for each patient, favoring a larger rather than smaller cuff (smaller cuff creates artificial hypertension)
 - ✓ BP cuff width should be 40% of the length of the arm measure from the shoulder tip to the elbow
 - ✓ Abnormal oscillatory BP measurement needs checking with a manual BP from the child's arm
- The table below identifies BP levels requiring further evaluation, starting with repeating the BP manually ensuring accurate measurement

*Screening BP Values Requiring Further Evaluation				
Age (years)	Blood pressure (mmHg)			
	Boys		Girls	
	Systolic	Diastolic	Systolic	Diastolic
1	98	52	98	54
2	100	55	101	58
3	101	58	102	60
4	102	60	103	62
5	103	63	104	64
6	105	66	105	67
7	106	68	106	68
8	107	69	107	69
9	107	70	108	71
10	108	72	109	72
11	110	74	111	74
12	113	75	114	75
≥13	120	80	120	80

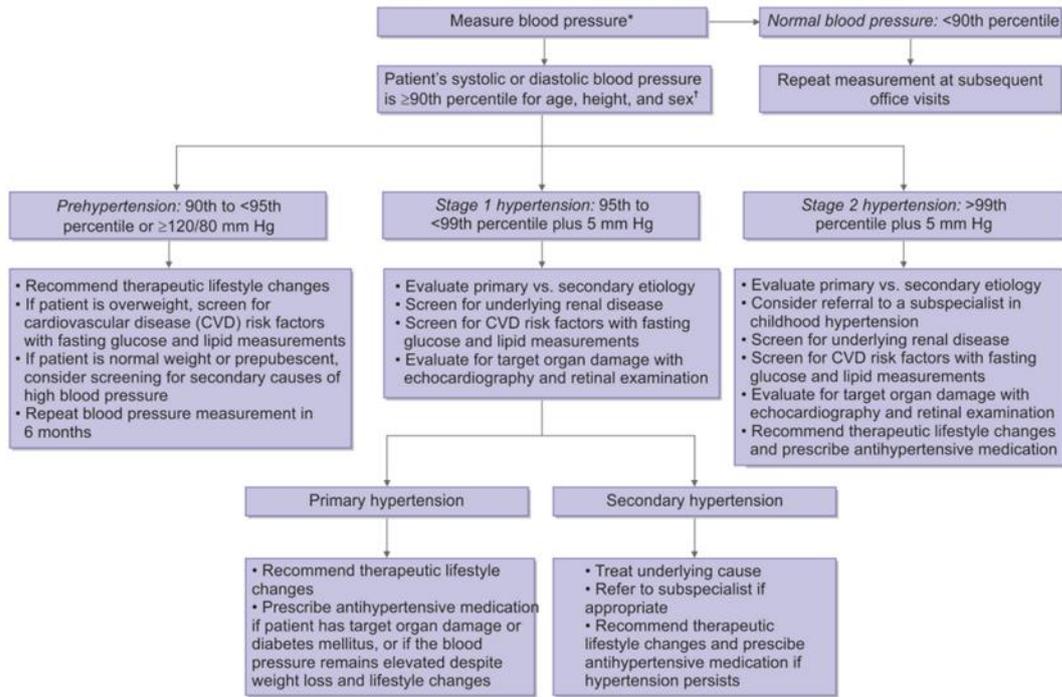
*** 90th centile for a child at average height**

Blood Pressure Classification in Children and Adolescents		
	For children aged 1 to 13 years	For children aged 13-17 years
Normal Blood Pressure	<90 th centile	<120/<80 mmHg
Elevated Blood Pressure	≥90 th centile to <95 th centile or 120/80 mmHg to <95 th centile (whichever is lower)	120/<80 to 129/<80 mmHg
Stage 1 Hypertension	≥95 th centile to <95 th centile + 12 mmHg or 130/80 to 139/89 mmHg (whichever is lower)	130/80 to 139/89 mmHg
Stage 2 Hypertension	≥95 th centile + 12 mmHg, or ≥140/90 mmHg (whichever is lower)	≥140/90 mmHg
Severe Hypertension		
Hypertensive Urgency	>95 th centile + 30 mmHg without symptoms/signs of target end organ damage (See Examination)	>180/120 without symptoms/signs of target end organ damage (See Examination)
Hypertensive Emergency	>95 th centile + 30 mmHg associated with encephalopathy, eg headache vomiting, vision changes and neurological symptoms (facial nerve palsy, lethargy, seizures, coma) +/- target-end organ damage	>180/120 associated with encephalopathy, eg headache vomiting, vision changes and neurological symptoms (facial nerve palsy, lethargy, seizures, coma) +/- target-end organ damage

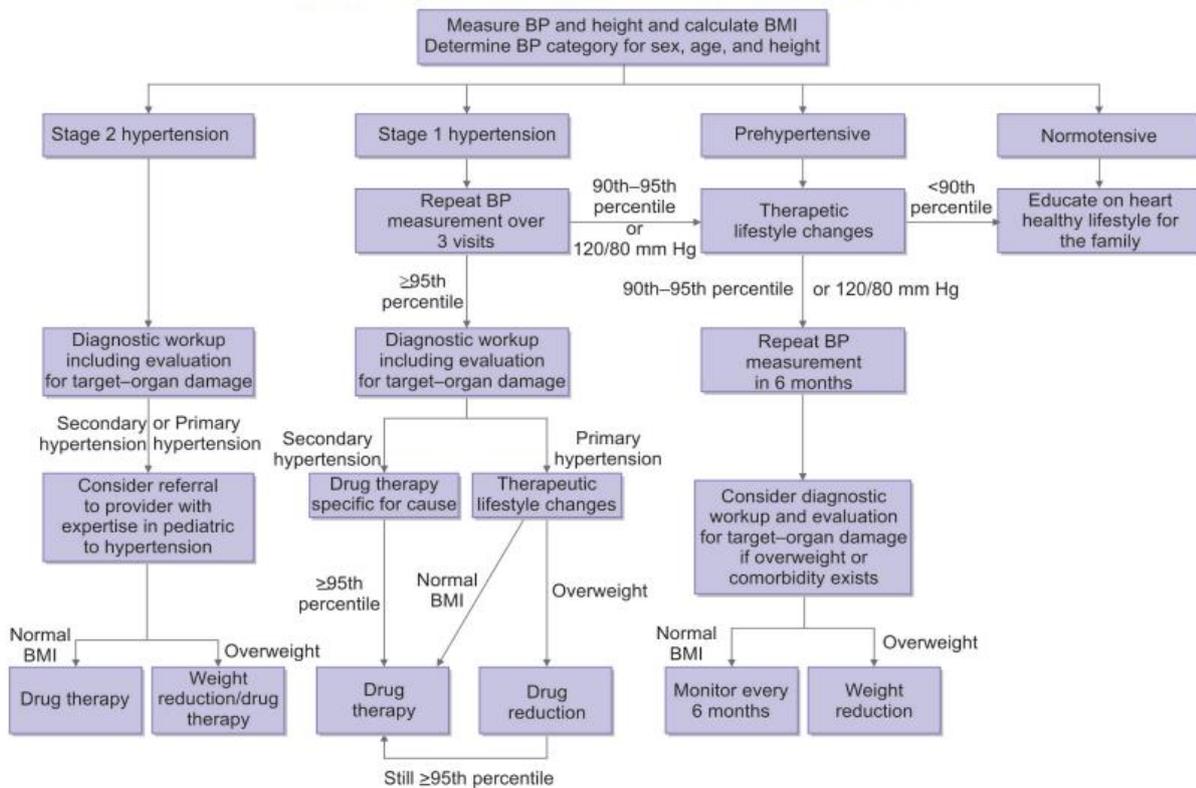
*** Use this AAP Pediatric Hypertension Guidelines - MDCalc for plotting the exact centile**

3) Management

Flowchart 1: Algorithm for the management of high blood pressure in children and adolescents.²⁰



Flowchart 1: Management algorithm for hypertension in pediatrics.²⁵



Medical Management:

- Should be commenced if:
 - ✓ Conservative measures have failed
 - ✓ Symptomatic hypertension develops
 - ✓ Stage 2 hypertension with no modifiable risk factors
 - ✓ Hypertension in setting of chronic kidney disease/diabetes
- Medical management should only be commenced in consultation with a general or renal pediatrician
 - ✓ Long-acting calcium channel blockers such as amlodipine or captopril +/- frusemide are recommended as first line therapy.

Consider consultation with local pediatric team when:

- Red flags (see history section above) or ongoing concerns are present
- Hypertensive urgency or hypertensive emergency

References:

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2. Flynn, J.T et al. Clinical practice guideline for screening and management of high blood pressure in children and adolescents. *Pediatrics*. 2017 vol 140 (3)

Signs and Symptoms	Diagnosis	Prognosis/Management
<p>2-Respiratory: 7-20%</p> <p>Wheeze ± dyspnea</p> <p>Exercise-induced asthma can often cause chest pain with exercise even in the absence of wheeze</p> <p>Sharp, sudden onset chest pain with significant dyspnoea</p> <ul style="list-style-type: none"> • Pain diffuse on the affected side with radiation to ipsilateral shoulder 	<p>a-Asthma/wheeze</p> <p>b.Pneumothorax</p>	<p>Trial of bronchodilator²</p> <p>CXR</p> <p>Conservative/interventional management</p>
<p>3-GIT: 3-6%</p> <p>Retrosternal burning</p> <ul style="list-style-type: none"> • Pain associated with posture, eating • Epigastric tenderness • Associated dysphagia suggests oesophageal origin 	<p>Gastro-oesophageal reflux,oesophagitis, gastritis</p>	<p>Trial of reflux treatment</p>
<p>4-Non –organic 1-9%</p> <p>Pain often fleeting or vague or localised over precordium +/-or left arm</p> <ul style="list-style-type: none"> • History of stressful events • Other recurrent somatic complaints, including headache or abdominal or extremity pain 	<p>Psychogenic</p>	<p>Reassurance ± psychological support</p>

Signs and Symptoms	Diagnosis	Prognosis/Management
5- Idiopathic: 12-52%		Reassurance
6- Miscellaneous: 4-11% Acutely painful vesicular rash Pain may precede rash	Herpes Zoster	Analgesic
7-Cardiac 0.6-1% • Sharp (anterior/precordial) • Exacerbated by leaning forward • ± systemic upset Palpitations • Dyspnoea Syncopal episodes (especially on exercise) • ± abnormal cardiac examination findings • ± family history of hereditary heart disease Central crushing chest pain ± radiating to jaw and arm • Associated sweating, nausea and pallor	a-Pericarditis b-Arrhythmia c-HOCM/aortic stenosis/long QT, d-Myocardial ischaemia	ECG to assess for widespread ST elevation Inflammatory markers/ Cardiology referral ECG (± prolonged ECG monitoring such as Holter Monitor) ± cardiology referral ECG reviewing QTc and PR intervals, delta waves, T wave changes Cardiology referral ECG for signs of ischaemia Cardiology referral

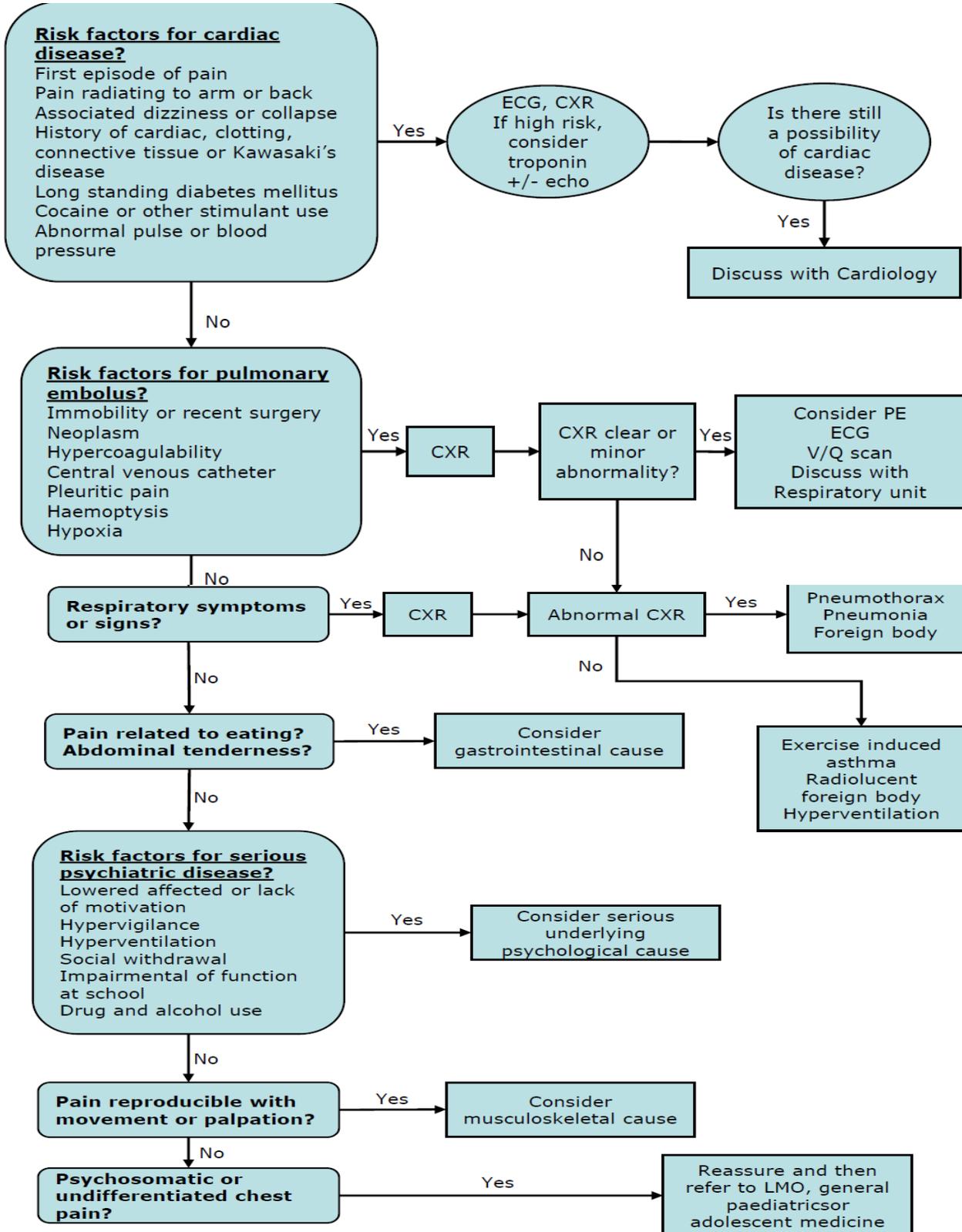
Cardiac Red flag:

1. Personal past or current history of acquired or congenital cardiac disease
2. Exertional syncope
3. Exertional cardiac-type chest pain
4. Hypercoagulable or hypercholesterolaemic state
5. Family history of sudden death under 35 years of age young onset ischaemic heart disease, inherited arrhythmias such as long QT syndrome or Brugada
6. Implantable cardioverter defibrillators in situ
7. Connective tissue disorders
8. History of cocaine/amphetamine use

The most important step in initial assessment is identifying signs of cardiorespiratory distress:

- ✓ Dyspnoea, tachypnoea, increased work of breathing
- ✓ Hypoxia
- ✓ Abnormal pulse or blood pressure
- ✓ Poor perfusion
- ✓ Distended neck veins, muffled heart sounds
- ✓ Depressed mental state

Pediatric Chest Pain Flowchart



References:

1. Collins SA, et al. Arch Dis Child Educ Pract Ed 2014;99:122–126. doi:10.1136/archdischild-2013-303919
2. Gal Garbut et al. Paediatric Chest Pain. Paediatrics in Review AAP. 2020 <https://publications.aap.org/pediatricsinreview/articleabstract/41/9/469/958/Pediatric-Chest-Pain>
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Guidelines on Fever with Rash/Kawasaki Recognition

Background:

- Kawasaki Disease (KD) is the second most common vasculitis in childhood after Henoch Schönlein purpura, and is the most common cause of acquired heart disease in children in high-income countries. Lack of appropriate treatment leads to coronary artery aneurysms (CAA) in approximately 25% of cases.
- Worldwide distribution, although more common in Asian children.
- Approximately 75% of cases occur under 5 years of age.
- Less common in children <6 months and >5 years; however, these children are more likely to develop CAA.
- Can present without all diagnostic criteria (see flowchart below) which can present a significant diagnostic challenge.

Assessment:

History:

- Physical findings can present sequentially over a number of days; thus, history should include asking about diagnostic features that may have resolved by the time of presentation.

Examination:

Kawasaki disease: Diagnostic Criteria

Fever persisting for 5 days, PLUS 4 of the 5 following criteria:

- A diagnosis earlier than 5 days can be made with a typical presentation in consultation with an experienced clinician
- KD can be diagnosed with less than four of the following features if coronary artery abnormalities are present

Criterion	Features
<p>Conjunctival Injection</p> 	<p>Bilateral, non-exudative, painless. Often with limbic sparing (zone around the iris is clear)</p>
<p>Rash</p> 	<p>Erythematous polymorphous rash occurs in the first few days, involving trunk and extremities Variable presentations, most commonly maculopapular, erythema multiforme-like or scarlatiniform Bullous, vesicular, or petechial rashes are not typical in KD</p>
<p>Oral Changes</p>  	<p>Strawberry Tongue Erythema, dryness, cracking and bleeding of the lips DiPuse oropharyngeal erythema Exudates are not typical of KD</p>
<p>Extremity Changes</p>  	<p>Hyperaemia and painful oedema of hands and feet that progresses to desquamation from the second week of illness</p>
<p>Lymphadenopathy</p> 	<p>Cervical, most commonly unilateral, tender. At least one node >1.5cm. Less common feature and seen in older children</p>

Common findings in addition to the diagnostic criteria include:

- ✓ Neurological: irritability, aseptic meningitis
- ✓ GIT symptoms: abdominal pain, vomiting, diarrhea, gallbladder hydrops
- ✓ Arthralgia / arthritis
- ✓ Dysuria
- ✓ Inflammation at recent (within 6 months) BCG vaccination site

"KD is a medium vessel vasculopathy; any organ system can be affected"

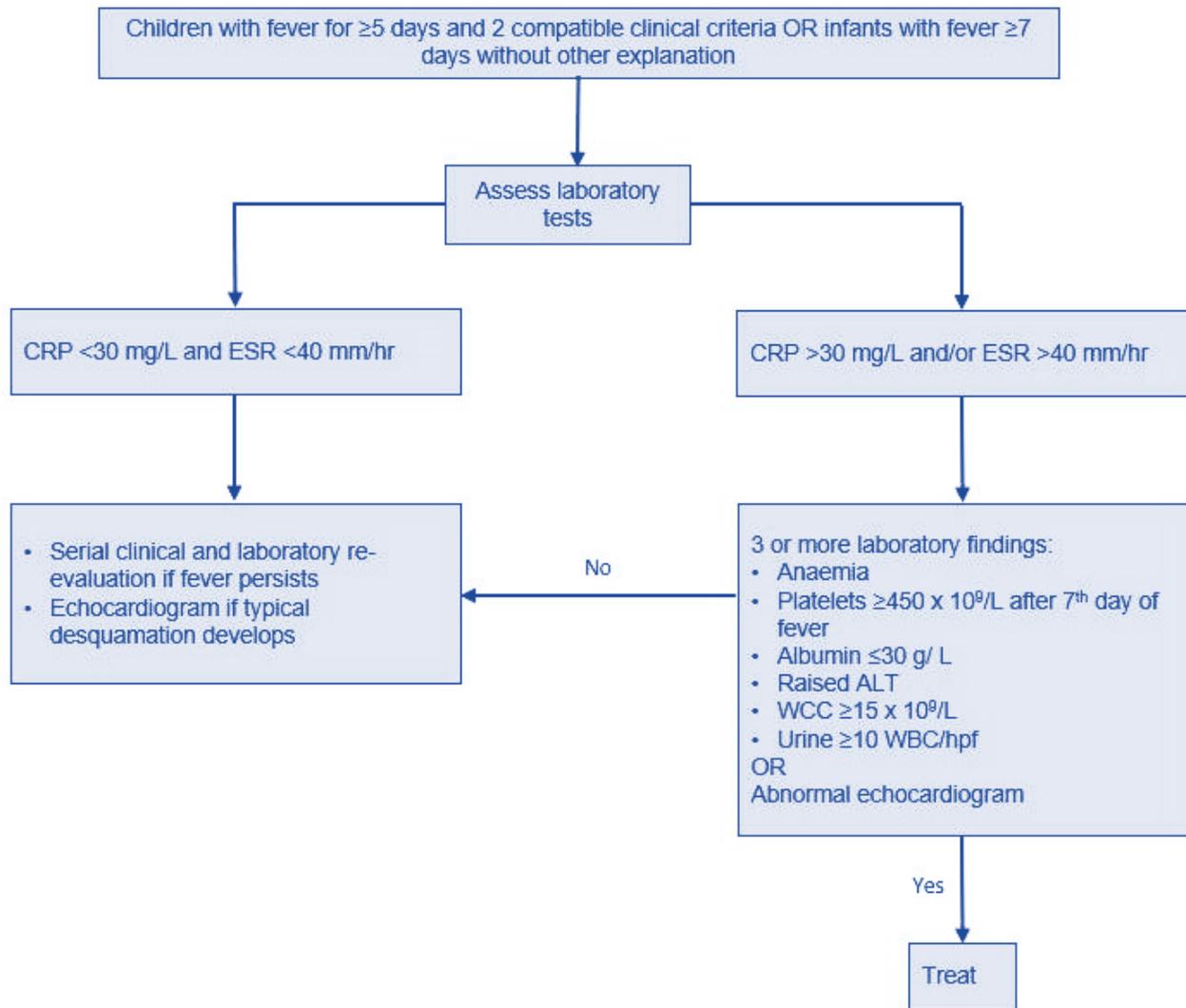
Incomplete Kawasaki Disease:

Consider in a child with a clinical presentation suggestive of KD but not meeting the full diagnostic criteria:

- Requires abnormal investigation results to support the diagnosis (see flowchart)
- Infants and adolescents often present with an incomplete picture and are at a higher risk for cardiac complications

Consider incomplete KD in:

- A child with fever for at least 5 days combined with 2 or 3 of the principal clinical features OR
- An infant with one/more of the following features:
 - ✓ Fever *7 days +/- irritability without other explanation
 - ✓ Prolonged fever and unexplained aseptic meningitis
- A child or infant with prolonged fever and:
 - ✓ Shock
 - ✓ Cervical adenitis not responsive to oral antibiotics
- Incomplete KD can present a significant diagnostic dilemma, however once the diagnosis is made, the treatment for KD and incomplete KD is identical



Incomplete Kawasaki

Source:

- Adapted from the American Heart Association (2017).

Differential Diagnosis:

- ✓ Group A streptococcal infections: tonsillitis, scarlet fever, acute rheumatic fever
- ✓ Viral infections including EBV, CMV, Adenovirus, HHV-6, SARS-CoV-2
- ✓ Systemic juvenile idiopathic arthritis (JIA)
- ✓ Sepsis
- ✓ Toxic shock syndrome (staphylococcal or streptococcal)
- ✓ Stevens-Johnson syndrome
- ✓ Drug reaction
- ✓ Malignancy

Management:
Investigations:

- There is no diagnostic test for KD. Laboratory tests provide support for diagnosis, assessment of severity, and monitoring of disease and treatment.
- Echocardiogram: discuss with cardiology specific timing of initial and follow-up studies, Suggested schedule:
 - ✓ At presentation (this should not delay initiation of treatment)
 - ✓ 2 weeks
 - ✓ 6 weeks

“Coronary artery lesions should be managed in consultation with pediatric cardiology and haematology services”

- In all patients consider:
 - ✓ FBC, CRP, ESR, UEC, LFT (note ESR result unreliable after IVIg administration)
 - ✓ Blood culture
 - ✓ ASOT
 - ✓ Serum to store (prior to IVIg administration)
 - ✓ Urinalysis and culture (sterile pyuria)
 - ✓ COVID-19 swab
 - ✓ ECG

**“Common abnormalities include elevation of ESR, CRP and neutrophils
Thrombocytosis is common in the second week of illness”**

Treatment:

1. Intravenous Immunoglobulin (IVIg): 2 g/kg as a single IV infusion on diagnosis

- IVIg should be given within the first 10 days of illness but should also be given to children diagnosed after 10 days if there is evidence of ongoing fever and/or inflammation
- A second dose of 2 g/kg IVIg should be given to children who do not respond to the first dose, as demonstrated by persistent or recurrent fevers 36 hours after the end of the first IVIg infusion. Seek specialist advice
- The National Blood Authority and BloodSTAR coordinate and authorise the use of blood products. IVIg is a product that must be ordered via their website (<https://www.blood.gov.au/bloodstar>)
- Haemolytic anaemia is an uncommon but recognised adverse effect of IVIg infusion, particularly for children receiving multiple doses. It typically occurs up to a week after IVIg administration
- Post IVIg vaccination: live vaccines (eg measles and varicella) should be deferred after IVIg administration, see the National Immunisation Handbook (<https://immunisationhandbook.health.gov.au/resources/handbook-tables/table-recommended-intervals-between-immunoglobulins-or-blood-products-and>). If the child is at high risk of measles, vaccinate and re-vaccinate after the appropriate period

2. Aspirin:

- Acute phase 50 mg/kg/day taken with IVIg
- Then continue with 3-5 mg/kg orally as a daily dose until normal echo on follow up (minimum 6 weeks)
- There is minimal risk of Reye syndrome with low-dose aspirin
- Avoid non-steroid anti-inflammatory medications whilst on aspirin

3. Corticosteroids:

- Evidence for indication and optimal dose/duration of adjunctive steroids in the primary treatment of KD is limited
- Corticosteroid use in KD should be considered in consultation with specialist advice
- Consider use in high-risk groups and in recurrent KD

4. Additional Treatments:

- A number of therapies are available for consideration in patients who are not responsive to initial IVIg. These options should only be used in consultation with KD specialists and include biological medicines such as infliximab

Consider Discharge When:

- Afebrile and well at least 36 hours after treatment
- Children are on a daily dose of aspirin (see treatment point 2 above)
- A follow-up plan is in place including general pediatric review and repeat echocardiograms planned with pediatric cardiology

References:

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Recurrent Chest Infections

1) General Considerations:

- Recurrent lower respiratory tract infection can be regarded as ≥ 3 annual episodes of documented bronchitis, bronchiolitis, or pneumonia and may merit further investigation for an underlying cause

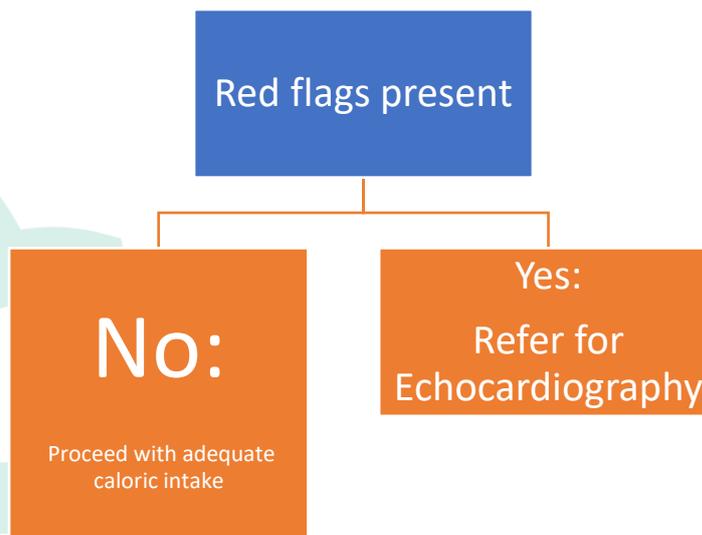
2) Red flags suggesting an underlying cardiac disease and prompting Echocardiography:

- Family history or features suggestive of a genetic disorder
- Signs of chronic illness, such as lethargy and recent weight loss
- Abnormal physical signs when the child is well, including digital clubbing and abnormal auscultatory signs
- Wet cough for >8 weeks continuously, no matter how well the child seems
- Failure to thrive

3) Common confounding differential diagnosis:

- ✓ Persistent bacterial bronchitis
- ✓ Tuberculosis
- ✓ Pertussis
- ✓ Asthma
- ✓ Foreign body

4) Algorithm for management:



Source:

- Couriel J. Assessment of the child with recurrent chest infections. *Br Med Bull.* 2002;61:115-32. doi: 10.1093/bmb/61.1.115. PMID: 11997302.

Failure to Thrive (Slow Weight Gain)

1) General Considerations:

Background:

Slow weight gain describes a child or infant whose current weight, or rate of weight gain is significantly below that expected for age and sex, or if weight has dropped ≥ 2 major percentile lines.

Growth Charts:

- < 2 years of age: WHO growth standards. Correct for prematurity (< 37 weeks) until 2 years old
- ≥ 2 years of age: CDC growth reference charts
- Use specific growth charts (eg Down, Turner syndrome) where appropriate

Source:

- *Growth charts for Down syndrome and Turner syndrome are available at:*
 - ✓ http://www.rch.org.au/genmed/clinical_resources/Growth_Resources/
 - ✓ <https://www.magicfoundation.org/Growth-Charts/>
 - ✓ <https://www.cdc.gov/ncbddd/birthdefects/downsyndrome/growth-charts.html>

Average Growth:

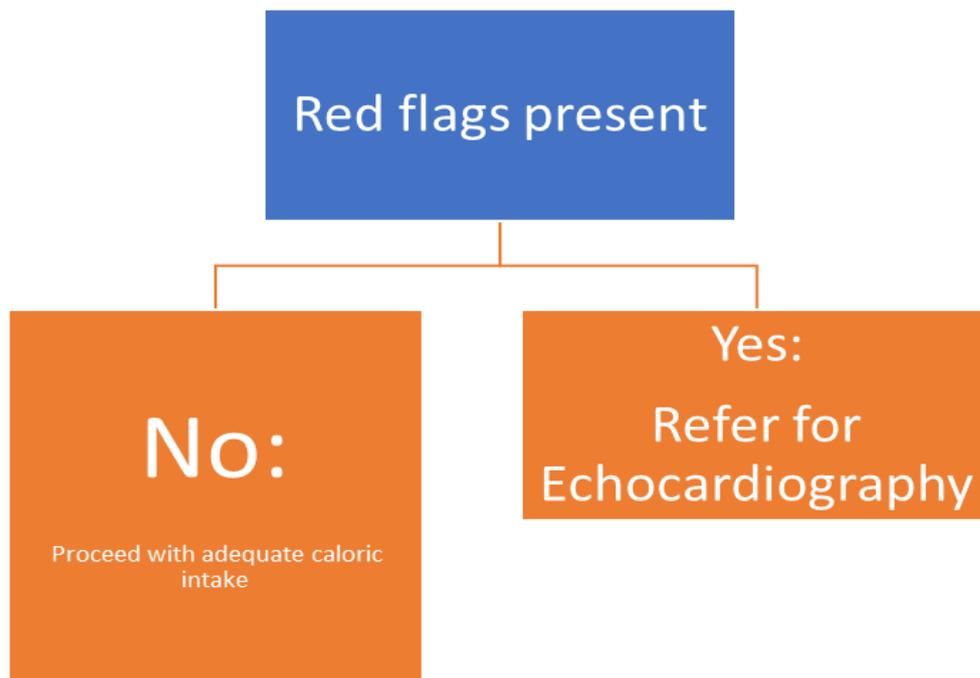
- Although the use of a growth chart is the most accurate indication of overall growth the use of average weekly weight gain for children who are followed up at frequent intervals may be required the rate of weight gain per week is variable.
- The table below is a guide to the expected average weight gain per week (it is not the minimally acceptable weight gain)

0 to 3 months	150–200 g/week
3 to 6 months	100–150 g/week
6 to 12 months	70–90 g/week

2) Red Flag Signs and Symptoms Suggesting cardiac Causes of Failure to Thrive:

- ✓ Murmur
- ✓ Edema
- ✓ Jugular venous distention
- ✓ Dysmorphic features
- ✓ Failure to gain weight despite reinstatement of adequate caloric intake
- ✓ Recurrent or severe respiratory infection
- ✓ Dysphagia (vascular ring)
- ✓ Cyanosis

3) Algorithm of Management:



Consider consultation with local pediatric team and transfer when:

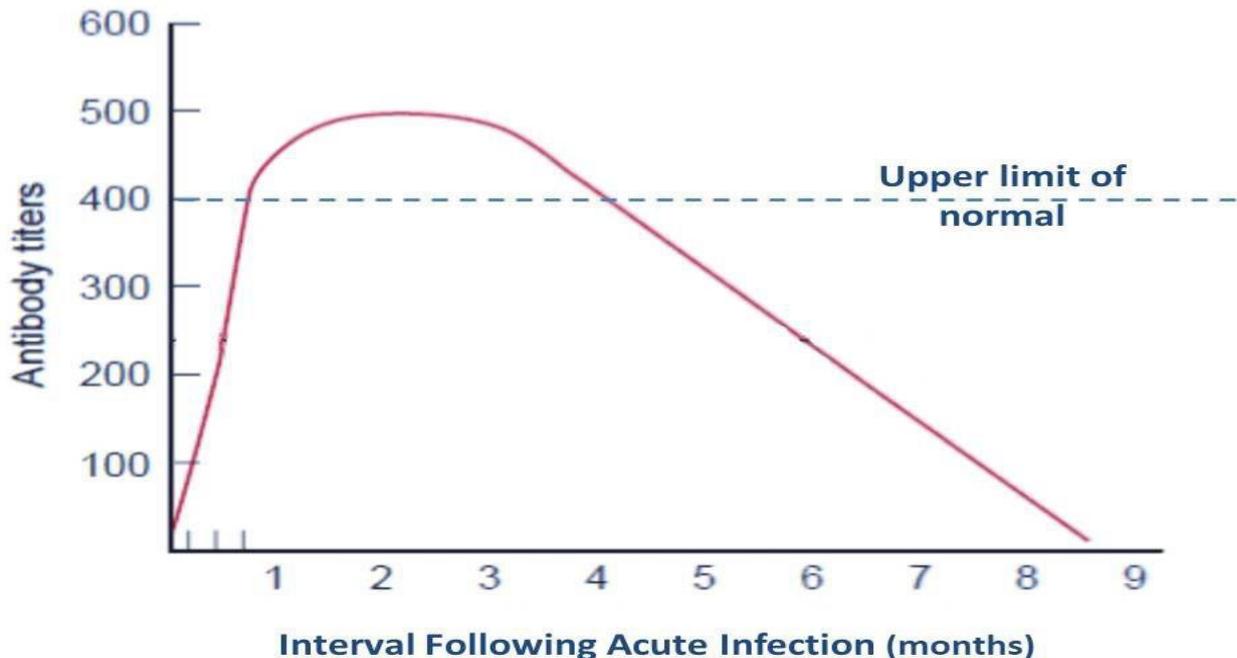
- ✓ Significant malnutrition, illness or dehydration
- ✓ Failed outpatient management
- ✓ Concern about potential child abuse or neglect
- ✓ Significant mental health concern in parent
- ✓ For further assessment of feeding technique, parent–child interaction and involvement of a multidisciplinary team
- ✓ Severe malnutrition, underlying cause or contributing factors requiring specialist input
- ✓ Child requiring care beyond comfort level of local services

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Diagnostic Utility of Anti-Streptolysin O Titer in Pediatric Cardiology Practice

I. Pattern of rise of Antistreptolysin O (ASO) antibodies following pharyngeal infection:



- Antistreptolysin O (ASO) antibodies are produced about a week to a month after an initial clinical or subclinical streptococcal pharyngeal, but not, skin infection. The amount of ASO antibody titer peaks at about 4 to 6 weeks after the illness and then tapers off but may remain detectable for several months after the streptococcal infection has resolved.

II. Indications of ASO Titer testing:

1. Children seen with symptoms or signs suggestive of either of rheumatic fever (RF), acute glomerulonephritis or poststreptococcal reactive arthritis (PSRA) in order to document a recent streptococcal infection with group A beta hemolytic streptococci had occurred to establish a causal relationship.
2. Children with recurrent sore throat to document that the recurrence is primarily related to repeated streptococcal infection with group A (GAS) rather recurrent viral pharyngitis, particularly if throat culture is positive that may represent a carrier state rather a concurrent GAS infection
3. Children with past the history of rheumatic fever or rheumatic heart disease (RHD) and under prophylactic therapy with benzathine penicillin G (BPG) for secondary prevention. An elevated or rising titer is a proof of recent or recurrent subclinical streptococcal pharyngitis and subsequently failed secondary prevention and liability for future rheumatic fever recurrence

III. ASO level in Egyptian children:

1. In healthy adults a titer more than 200 IU /mL is considered an elevated titer
2. The ULN (80th percentile) of ASO titer in asymptomatic healthy Egyptian children is 400 IU/mL owing to repeated subclinical streptococcal infection in our community, a level more than this 400 IU/mL is considered elevated titer.
3. The ULN (80th percentile) of ASO titer in children with history of recurrent streptococcal pharyngitis may reach up to 1600 IU/mL and is related to repeated infections that results in sustained or continuously rising titer.

IV. Clinical scenarios, interpretation and action plan in children seen with elevated ASO titer more than 400 IU/ ML:

- The management of children presenting with elevated ASO titer is related to the clinical scenarios and the indications for ASO titer testing as requested by the pediatrician.

Clinical scenario	Interpretation	Action Plan
Children presenting without clinical symptoms or signs suggestive of major manifestation of rheumatic fever eg: vague extremity pain, growing pain, chest pain of musculoskeletal cause or innocent murmur that is made louder by fever	A higher titer is not related to the current minor illness and simply reflect, a recent or recurrent uncomplicated streptococcal pharyngitis that is totally unrelated to the current minor problems.	Parents has to be assured for this benign phenomenon and BPG should not be prescribed or given in a clear-cut message

Note:

- **The pediatrician should NOT order for ASO titer in these previous scenarios**

Clinical Scenario	Interpretation	Action Plan
Children presenting with migratory polyarthritits or carditis	A higher titer supports the diagnosis of RF by strict application of updated criteria for diagnosis of RF, 2015	Patients should be treated as the initial attack of rheumatic fever and managed accordingly

Note:

- *Evidence of a preceding streptococcal infection will reduce, but not eliminate, the possibility of other diseases presenting with polyarthritits as JIA, SLE, serum sickness or leukemia. Such diseases need to be in mind before a definitive diagnosis of RF is made. Also, revision of the initial diagnosis as RF is justified if the therapeutic response to acetyl salicylic acid is absent or delayed*

Clinical Scenario	Interpretation	Action Plan
Children presenting with arthritis of acute onset, symmetric or asymmetric, non-migratory affecting any joint, persistent or recurrent with poor response to salicylate or NSAIDs together with lack of other major manifestations of RF.	A high ASO titer support the diagnosis of Post-streptococcal reactive arthritis (PSRA)	Patient should be treated as PSRA with anti-inflammatory therapy and secondary prevention is justified for 2 years

Clinical Scenario	Interpretation	Action Plan
<p>Children presenting with history of recurrent sore throat that might reflect either recurrent streptococcal pharyngitis or recurrent viral pharyngitis</p>	<p>A high ASO titer confirm the diagnosis of recurrent streptococcal pharyngitis</p>	<p>Tonsillectomy is recommended (by applying the current indications for tonsillectomy in children with recurrent tonsillitis) oral penicillin short term 3-6 months of BPG every two weeks if the frequency of recurrence is less than required for tonsillectomy.</p>

Clinical Scenario	Interpretation	Action Plan
<p>Children with history of RF (arthritis or chorea) or established RHD and assumed to be under secondary prevention program with BPG prophylaxis.</p>	<p>A rising titer confirms the diagnosis of recurrent subclinical streptococcal pharyngitis and possible prophylaxis failure.</p>	<p>Every effort must be done to ensure good compliance with BPG by revision of the dose and the frequency of injections or encouraging more compliance eg by adding lidocaine to BPG prior to IM injection.</p>

V. Clinical scenarios in which the diagnosis of the initial attack of rheumatic fever is definite

- Definite initial episode of ARF is established by: 2 major manifestations + evidence of preceding group A Streptococcal infection, or 1 major + 2 minor manifestations + evidence of preceding group A Streptococcal infection

Major Manifestations:

- ✓ Carditis (including subclinical evidence of rheumatic valvulitis on echocardiogram): new murmur suggestive of mitral and /or aortic regurgitation with or without cardiac enlargement, heart failure or pericarditis
- ✓ Polyarthritits: always migratory affecting large joints
- ✓ Sydenham chorea
- ✓ Erythema marginatum
- ✓ Subcutaneous nodules

Minor Manifestations:

- ✓ Fever ≥ 38.5 °C
- ✓ Polyarthralgia
- ✓ ESR ≥ 60 mm/h or CRP ≥ 6 mg/dL
- ✓ Prolonged P-R interval on ECG

VI. Clinical scenarios in which the diagnosis of the initial attack of rheumatic fever is probable:

- Children with age between 5:15 years presenting with clinical presentation in which ARF is considered a likely diagnosis but it falls short in meeting the criteria as:
 - A. Recent history (maximum 4 weeks) with overt joint pain in large joints of migratory nature without objective findings (migratory polyarthralgia) or
 - B. Recent history of clinical evidence of mono-arthritis in a large joint after exclusion of trauma or septic arthritis. These patients have to be managed as follows:

I. Action Plan(A-G):

- ✓ Avoid premature administration of anti-inflammatory therapy and even withhold it if prescribed in sub therapeutic dose. paracetamol is a good choice to control pain
- ✓ Begin antibiotic therapy for possible previous GAS infection for 10 days to abate the immune response to possible streptococcal antigen.
- ✓ Close bed side clinical assessment for evolution of typical migratory joint involvement or clinical carditis (major criteria)
- ✓ Exclude subclinical carditis by color Doppler echocardiography initially and within the ongoing two weeks (major criteria).
- ✓ Establish a recent evidence of GAS infection by rising titer of ASO so that such rise is causally related rather a mere coincidence
- ✓ Fulfill the requirement of strong positive acute phase reactants by elevated ESR =>60mm/h or CRP=> 6mg/dl
- ✓ Gather all physical and laboratory findings to rule out other possible causes of joint diseases as juvenile idiopathic arthritis (JIA), reactive arthritis secondary to recent viral infection, SLE, serum sickness or possible malignancy

II. Establishment of the diagnosis of probable rheumatic fever:

- **Mono-arthritis or migratory polyarthralgia** may occur with increasing frequency in rheumatic fever, so **a probable rheumatic etiology** of such pure joint involvement is established and managed accordingly if associated with the combination of the following **three requirements**:
 1. A significant acute phase response (ESR:60mm /H, or CRP =>6mg/dl),
 2. Recent evidence of GAS infection by rising titer of ASO (two samples at least one week apart), so that such rise is causally related,
 3. Other possible or overt causes of joint involvement as JIA, viral, autoimmune septic arthritis are ruled out.

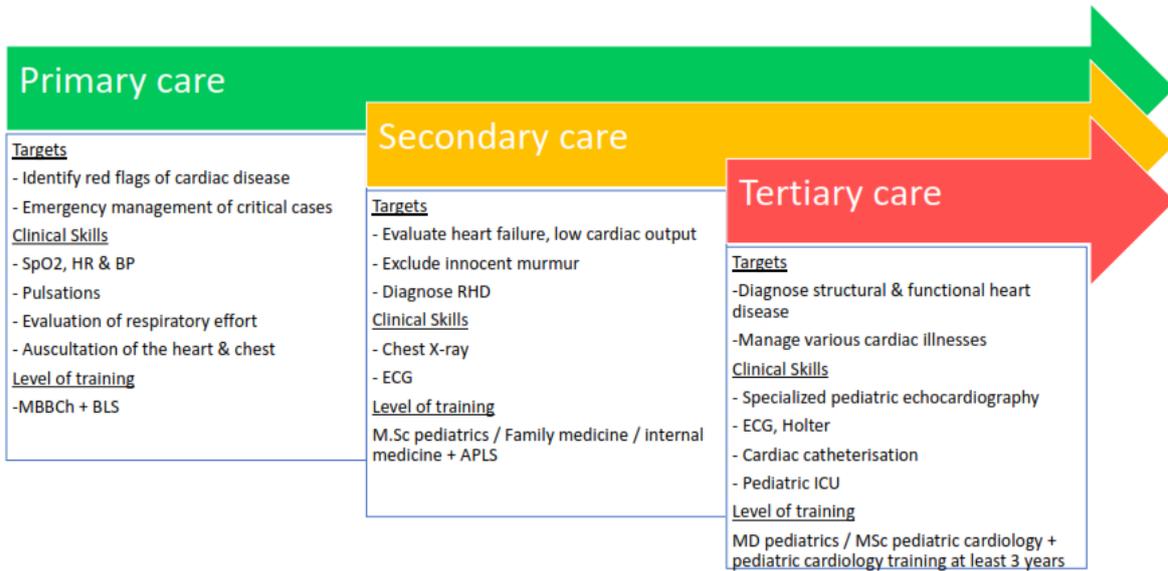
VII. Major differentiating points between growing pains and subacute rheumatic arthralgia:

	Joint Pains of Subacute Rheumatic Fever	Growing Pains
Timing	During entire day, disappears on getting warm in bed, worse on first getting out of bed in the morning	At end of day or soon after falling asleep, free of pain in morning
Location	In joints. Pain on motion. Child points out pain in joints. Involves joints in upper extremities. May cause limping.	In muscles of thigh or legs. No pain on motion. Child vague in pointing out site of pain. Pain in upper extremities unusual

References:

1. Ayoub EM, Ahmed S: Update on complications of group A streptococcal infections. *Curr Probl Pediatr*. 1997, 27 (3): 90-101.
2. Gewitz MH, Baltimore RS, Tani LY, et al. Revision of the Jones Criteria for the diagnosis of acute rheumatic fever in the era of doppler echocardiography: a scientific statement from the american heart association. *Circulation* 2015; 131:1806–18.
3. Kotby AA, Habeeb NM, El Elarab SE. Antistreptolysin O titer in health and disease: levels and significance. *Pediatr Rep* 2012; 4:68–70.
4. Riise OR, Lee A, Cvancarova M, Handeland KS, Wathne KO, Nakstad B: Recent-onset childhood arthritis--association with *Streptococcus pyogenes* in a population-based study. *Rheumatology*. 2008, Oxford, 47 (7): 1006-11. [10.1093/rheumatology/ken122](https://doi.org/10.1093/rheumatology/ken122).
5. Shet A, Kaplan EL. Clinical use and interpretation of group A streptococcal antibody tests: a practical approach for the pediatrician or primary care physician. *Pediatr Infect Dis J* 2002; 21:420–426.

Cardiac Murmurs



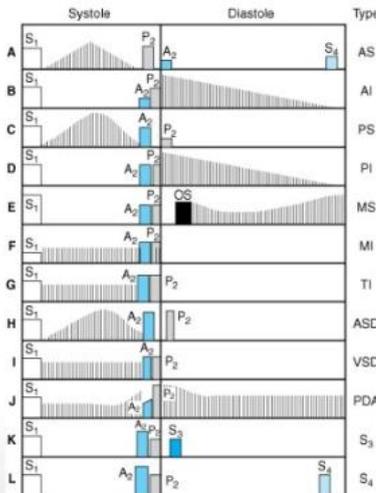
Red Flags of Cardiac Illness:

- Symptoms of age-related exertional dyspnea / exercise intolerance; recurrent chest infections; chronic cough; syncope; cyanosis; palpitations; chest pains; developmental delay
- Signs of abnormal growth; dysmorphism; delayed capillary refill; weak femoral pulse; displaced apical impulse; parasternal heave/ thrill; abnormal S2; hepatomegaly; edema
- History of antenatal maternal illness or drug exposure; familial sudden unexplained death, congenital heart disease or rheumatic heart disease

Reference:

- Frank JE & Jacobe JM. Evaluation and Management of Heart Murmurs in Children. Am Fam Physician. 2011 Oct 1;84(7):793-800.

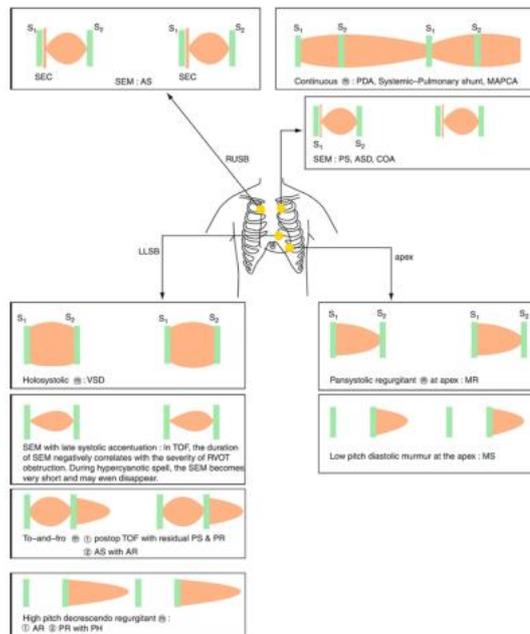
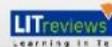
Auscultation: Character, Intensity



Grade	Description	Thrill
1	Very faint, not heard in all positions	No
2	Soft, heard in all positions	No
3	Easily heard, prominent	No
4	Loud, with palpable thrill	Yes
5	Loud, heard with edge of stethoscope partly off chest	Yes
6	Very loud, heard with stethoscope 5-10mm off chest	Yes

AS= aortic stenosis
 AI= aortic incompetence
 PS= pulmonary stenosis
 PI= pulmonary incompetence
 MS= mitral stenosis
 MI= mitral incompetence
 TI= tricuspid incompetence
 ASD= atrial septal defect
 VSD= ventricular septal defect
 PDA= patent ductus arteriosus
 S3= third heart sound
 S4= fourth heart sound

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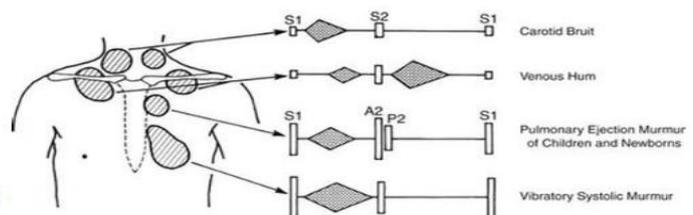


SEM= systolic ejection murmur
 AS= aortic stenosis
 AR= aortic regurgitation
 PS= pulmonary stenosis
 PR= pulmonary regurgitation
 MS= mitral stenosis
 MR= mitral regurgitation
 ASD= atrial septal defect
 VSD= ventricular septal defect
 PDA= patent ductus arteriosus
 MAPCA= major aorto-pulmonary collateral artery
 COA= coarctation of the Aorta
 RVOT= right ventricular outflow tract
 TOF= tetralogy of Fallot
 PH= pulmonary hypertension

Park, I.S., Kim, S.I. (2019). Introduction. In: Park, I. (eds) An Illustrated Guide to Congenital Heart Disease. Springer, Singapore. https://doi.org/10.1007/978-981-13-6978-0_1

Innocent murmur

- Asymptomatic – absence of red flags
- Soft (<III/VI)
- Systolic
- Localized
- Musical / buzzing
- Vary with posture / respiration



Myung K. Park, Introduction, Editor(s): Myung K. Park, Park's Pediatric Cardiology for Practitioners (Sixth Edition), Mosby, 2014: P63

Frank JE & Jacobs JM. Evaluation and Management of Heart Murmurs in Children. Am Fam Physician. 2011 Oct 1;84(7):793-800.

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