PEDIATRICS

DOSSIER

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CARDIOLOGY 📘

🔷 29. ACYANOTIC CONGENITAL HEART DISEASES

Key Concept : Left-to-Right Shunts

- Acyanotic at presentation ; cyanosis may develop years later (Eisenmenger syndrome)
- Frequency : VSD > ASD > PDA
- Blood is shunted left → right because left-sided pressures are higher
- Effect : increased pulmonary blood flow → pulmonary hypertension → right heart overload → Eisenmenger (shunt reversal → late cyanosis)

Ventricular Septal Defect (VSD)

- Pathophysiology : Left → right shunt at ventricular level → increased pulmonary blood flow → increased left atrial and left ventricular volume → volume overload on LV

Presentation :

- Small VSD : asymptomatic , loud holosystolic murmur (smaller VSD = louder murmur) (past question)
- Large VSD : tachypnea , poor feeding , failure to thrive , sweating with feeds , FTT — symptoms start 4–8 weeks after birth (past question) when pulmonary vascular resistance falls
- Holosystolic murmur at left sternal border ; displaced apical impulse ; loud S2
- Hepatomegaly , cardiomegaly on CXR , increased pulmonary vascular markings

Clues :

- Most common cause of CHD presenting with HF in infants
- VSD becoming smaller → murmur gets LOUDER (past question)
- Wide pulse pressure = NOT a finding in VSD (past question : wide pulse pressure = PDA)
- ASD has fixed split S2 ; VSD does NOT have fixed split S2
- CXR : cardiomegaly + increased pulmonary vascular markings (past question)
- ECG : LVH (±RVH if large) ; left axis deviation in tricuspid atresia (NOT VSD)
- VSD does NOT cause left atrial dilatation on ECG (past question)

Atrial Septal Defect (ASD)

- Types : Ostium secundum (most common , usually isolated) ; Ostium primum (associated with Down syndrome and mitral regurgitation)

Presentation :

- Often asymptomatic ; may present with exercise intolerance in older children
- Fixed wide splitting of S2 (past question) — hallmark of ASD

- Mid-systolic ejection murmur at left upper sternal border
- Right ventricular heave ; right atrial and right ventricular dilatation
- CXR : cardiomegaly , increased pulmonary markings , prominent RA/RV
- ECG : RVH ; right axis deviation ; RSR' pattern in V1

What ASD does NOT cause :

- ASD does NOT cause gallop rhythm (past question : "ASD and gallop rhythm" = WRONG)
- ASD does NOT cause LV dilatation — it causes RA and RV dilatation (past question : "ASD and LV dilatation" = WRONG)
- Eisenmenger is NOT a complication in late childhood — it occurs much later in adulthood (past question)

Patent Ductus Arteriosus (PDA)

- Physiology : In fetal life , PDA shunts blood from PA to aorta (right → left) ; after birth , shunt reverses (left → right) as PVR drops
- Can be caused by : prematurity , congenital rubella

Presentation :

- Continuous "machinery" murmur at left subclavicular area (past question)
- Wide pulse pressure — e.g. BP 100/40 (past question : "seen in PDA" = wide pulse pressure)
- Bounding pulses (past question)
- Left ventricular volume overload ; displaced apical impulse ; tachycardia
- CXR : cardiomegaly , increased pulmonary vascular markings
- Signs start at 4–8 weeks after birth (same as large VSD)

What PDA does NOT cause :

- PDA does NOT cause right atrial hypertrophy (past question : "wrong about PDA" = RA hypertrophy)
- PDA does NOT cause left axis deviation on ECG

Management :

- Indomethacin closes PDA (inhibits prostaglandin synthesis) (past question)
- PGE1 keeps PDA open → used for ductal-dependent lesions (past question)
- Surgical/catheter-based closure for significant PDA

Coarctation of the Aorta

Presentation :

- Hypertension in upper limbs ; weak or absent femoral pulses ; radiofemoral delay (past question)

- Systolic murmur with inter-scapular radiation
- Neonatal CoA : shock (ductal-dependent) ; differential cyanosis (lower saturation in lower limbs)
- Older child : hypertension , LVH , headache
- CXR : notched ribs (from collateral intercostal artery erosion) + cardiomegaly (past question)
- ECG : LVH (past question)
- Wide mediastinum is NOT a finding in CoA (past question : “wide mediastinum in CoA” = WRONG — it is narrow in TGA)
- CoA does NOT produce wide pulse pressure , bounding pulses , or diastolic murmur (past question)

Aortic Stenosis (AS)

- Ejection systolic murmur at right upper sternal border , radiating to neck/suprasternal notch (past question : murmur at right upper sternal border = aortic stenosis)
- Ejection click may be present
- Causes LV pressure overload → LVH
- Causes LV dilatation if severe (past question)
- Critical AS in neonates : ductal-dependent ; present as shock when PDA closes
- Syncope during exercise is a red flag (past question)

Pulmonary Stenosis (PS)

- Ejection systolic murmur at left upper sternal border ; ejection click (past question : “opening systolic click on the right side” = aortic ; click at left upper sternal = pulmonary)
- Murmur radiates to back
- Causes RV pressure overload → RVH
- Cyanosis + systolic murmur over left sternal border → PS (past question)

Innocent Murmur (Functional Murmur)

- Soft (grade 1–2/6) , systolic , disappears when child stands up or during Valsalva (past question)
- No other cardiac findings , normal CXR and ECG
- Reassurance is appropriate (past question)
- A mid-systolic murmur that decreases upon standing → reassurance (past question)
- Differential in asymptomatic child with soft systolic murmur : innocent murmur , small ASD , mild PS , mild anemia — mild aortic regurgitation is NOT in the differential of a soft systolic murmur (past question)

- Diastolic murmur is ALWAYS pathological in children (past question : diastolic murmur in 6-month-old = significant pathology)

Eisenmenger Syndrome

- Uncorrected left-to-right shunt → pulmonary hypertension → RVH → shunt reversal (right → left) → late cyanosis , clubbing , polycythemia
- NOT a complication in late childhood for ASD (past question) — occurs in adulthood

Fetal Circulation

3 important shunts:
1. Umbilical vein → **ductus venosus** → IVC (bypassing hepatic circulation).
2. Most of the highly oxygenated blood from IVC → **foramen ovale** → LA.
3. Deoxygenated blood from SVC → RA → RV → main pulmonary artery → **ductus arteriosus** → descending aorta; shunt is due to ↑ fetal pulmonary artery resistance.

At birth, infant takes a breath → ↓ resistance in pulmonary vasculature → ↑ LA pressure vs RA pressure → foramen ovale closes (now called fossa ovalis); ↑ in O_2 (from respiration) and ↓ in prostaglandins (from placental separation) → closure of ductus arteriosus.

NSAIDs (eg, indomethacin, ibuprofen) or acetaminophen help close the patent ductus arteriosus (PDA) → ligamentum arteriosum (remnant of ductus arteriosus). "Endomethacin" ends the PDA.

Prostaglandins E_1 and E_2 kEEp PDA open.

- Foramen ovale : right atrium → left atrium (right → left) (past question)
- Ductus arteriosus : pulmonary artery → aorta (right → left) (past question : "blood moves from PA to aorta through DA" = CORRECT in fetal life)
- Umbilical vein carries the most oxygenated blood (past question)
- Umbilical arteries carry deoxygenated blood (2 arteries , 1 vein)
- Single umbilical artery → associated with congenital anomalies (past question)
- PVR is highest at birth and decreases to normal levels ; it does NOT increase from birth to 4–8 years (past question : "PVR lowest at birth and increases" = WRONG)

🔷 30. CYANOTIC CONGENITAL HEART DISEASE

The 5 T's

- 1. Tetralogy of Fallot (most common cyanotic CHD overall)
- 2. Transposition of great arteries (most common cyanotic CHD presenting at birth)
- 3. Tricuspid atresia
- 4. Truncus arteriosus
- 5. TAPVR (Total anomalous pulmonary venous return)
- + Ebstein anomaly

Cyanosis Definition

- Clinically evident when deoxygenated Hb ≥ 5 g/dL in systemic circulation (corresponds to SpO2 ≤ 80%)

Hyperoxia Test

- Give 100% O2 for 10 minutes
- If PaO2 fails to rise significantly → cardiac cause (past question : "absence of improvement with O2 = cardiac")
- If PaO2 rises to > 150–200 mmHg → pulmonary cause
- TGA does NOT respond to oxygen (past question)

Tetralogy of Fallot (TOF)

- 4 components : VSD + RV outflow obstruction (infundibular/pulmonary stenosis) + overriding aorta + RVH
- Most common cyanotic CHD overall (5% of all CHD) (past question)
- Caused by anterior malalignment of interventricular septum
- Pulmonary stenosis severity determines degree of cyanosis

Presentation :

- Harsh ejection systolic murmur at left sternal border (pulmonic area) (past question)
- Central cyanosis ; normal pulses (past question)
- Tet spells : hypercyanotic episodes triggered by crying , exercise , fever (past question)
- Squatting increases SVR → decreases right-to-left shunt → improves cyanosis (past question)
- Clubbing occurs LATER (not at 3 months) (past question : "clubbing in 3-month-old with TOF" = WRONG — too early)
- Normal respiratory rate usually
- CXR : Boot-shaped heart + decreased pulmonary vascular markings (past question)
- Cardiomegaly is NOT seen in TOF (past question : "cardiomegaly in TOF" = WRONG)
- TGA has narrow mediastinum , NOT TOF
- What increases cyanosis (worsens tet spells) : Exercise , fever , dehydration , high altitude , crying
- What decreases cyanosis (improves tet spells) : Squatting (past question : "squatting increases cyanosis" = WRONG)
- Worsening cyanosis in TOF: Murmur gets SOFTER as pulmonary obstruction worsens → less pulmonary flow → more right-to-left shunt"
- ECG : RVH ; right axis deviation
- Association : 22q11 deletion syndrome

Tet Spell Management :

- Calm the child ; knee-to-chest position (↑ SVR)
- Oxygen (blow-by) , IV fluids
- IV propranolol (slow HR) + phenylephrine (↑ SVR)

Transposition of Great Arteries (TGA)

- Most common cyanotic CHD presenting in the FIRST WEEK of life (past question)
- Aorta arises from RV ; pulmonary artery arises from LV → parallel circulations (not compatible with life without mixing)
- Mixing occurs at : PFO , ASD , VSD (35–40%) , PDA
- Requires PDA , VSD & PFO for survival ; balloon atrial septostomy to improve mixing

Presentation :

- Severe cyanosis within first 12 hours ; no respiratory distress (past question)
- No murmur or single S2 (past question : "cyanotic CHD without murmur = TGA")
- Does NOT respond to oxygen (past question)
- CXR : Egg-on-a-string / egg-on-its-side appearance + narrow mediastinum (past question : "TGA and wide mediastinum" = WRONG — it is NARROW)
- ECG : normal or RVH
- Management : PGE1 to keep PDA open + balloon atrial septostomy + arterial switch procedure (surgery)

Tricuspid Atresia

- Absence of tricuspid valve + hypoplastic RV
- Requires both ASD and VSD/PDA for viability (past question)
- Requires PFO/ASD for survival (past question)
- ECG : left axis deviation (past question — unique among cyanotic CHDs) + RA hypertrophy (tall P waves)
- Physical exam : NO right ventricular heave (past question : "tricuspid atresia and RV heave" = WRONG — RV is hypoplastic)

Truncus Arteriosus

- Single great vessel arising from both ventricles ; almost always has VSD
- Increased pulmonary blood flow → mild cyanosis + features of heart failure (not severe cyanosis)
- Wide pulse pressure ; possible ejection click
- CXR : increased pulmonary vascular markings , cardiomegaly

TAPVR (Total Anomalous Pulmonary Venous Return)

- Pulmonary veins drain into right heart (SVC , coronary sinus , etc) instead of left atrium
- Requires ASD/PDA for survival (right → left mixing to maintain cardiac output)
- Supracardiac TAPVR : snowman/figure-of-8 CXR appearance (past question)
- PGE1 does NOT help infracardiac obstructive TAPVR (it can worsen it) (past question)
- Mild cyanosis with increased PBF

Ebstein's Anomaly

- Downward displacement of tricuspid valve into RV → "atrialization" of RV → tricuspid regurgitation + right-sided heart failure
- Associated with : accessory conduction pathways (WPW) , ASD
- Can cause decreased PBF
- associated with lithium exposure in utero (past question)

PDA-Dependent Lesions (must give PGE1)

Lesion	Why PDA needed
Pulmonary atresia with intact septum	PDA provides only pulmonary blood flow
Tricuspid atresia with no VSD	PDA provides only pulmonary blood flow
Critical AS / HLHS / Critical CoA / Interrupted aortic arch	PDA provides only systemic blood flow
TGA	PDA improves mixing

- TOF does NOT require PDA for survival (past question)
- Truncus arteriosus does NOT require PDA (past question)
- VSD does NOT require PDA (past question)

Down Syndrome and CHD

- Most common cardiac anomaly = AVSD (AV canal defect) / ASD (past question : "TOF is most common in Down syndrome" = WRONG)

🔷 31. HEART FAILURE

Definition

- Failure of the heart to produce cardiac output sufficient to meet metabolic demand

Pathophysiology

- Compensatory mechanisms : tachycardia , ↑ contractility , vasoconstriction (RAA system + sympathetic) , water/Na retention
- Polycythemia is NOT a compensatory mechanism in HF (past question) — it is seen in chronic hypoxia/cyanosis

Clinical Features

System	Findings
Pulmonary congestion	Tachypnea , intercostal retractions , dyspnea , grunting
Systemic congestion	Hepatomegaly , edema , elevated JVP
Low output	Cool extremities , prolonged cap refill , pallor , oliguria , poor feeding , FTT
Sympathetic	Tachycardia , diaphoresis , irritability

- Hepatomegaly in HF = due to systemic venous congestion (past question : “hepatic inflammation” = WRONG)
- Retractions in VSD = due to pulmonary vascular congestion (past question)
- FTT in HF = due to increased metabolic demand + poor feeding (past question : “malabsorption” = WRONG)
- Cold extremities in HF = due to systemic vasoconstriction (past question : “vasodilation” = WRONG)
- Anemia worsens HF (↓ oxygen carrying capacity) (past question)
- Cardiac output increases with increasing preload ; cardiac output DECREASES with increasing afterload (past question : “CO increases with increasing afterload” = WRONG)

Common Causes of HF in Children

Time	Cause
1st week of life (ductal-dependent)	HLHS , critical AS , critical CoA , interrupted aortic arch
4–8 weeks	Large VSD , PDA , AVSD , truncus arteriosus
Any age	Myocarditis (Coxsackie B) , cardiomyopathy , arrhythmia

- Large ASD is least likely cause of HF in infants (past question : ASD rarely causes HF in infants)
- Pulmonary valve stenosis causes pressure overload but NOT volume overload → NOT a cause of HF with increased pulmonary markings (past question)

Investigations

- CXR : cardiomegaly + pulmonary vascular congestion (past question)
- Echo : most useful to determine etiology
- ECG : helps detect arrhythmia or structural clues
- Myocarditis : cardiomegaly + pulmonary edema on CXR ; preceded by viral illness

Management of HF

- Diuretics (furosemide) : decrease congestion , improve respiratory distress
- Furosemide side effects : hyponatremia , hypokalemia , hypochloremia , hypercalciuria (past question : metabolic acidosis is NOT a side effect of furosemide — it causes metabolic alkalosis)
- ACE inhibitors / ARBs : afterload reduction , improve tissue perfusion
- Inotropes : dopamine , dobutamine , epinephrine , milrinone (phosphodiesterase inhibitor)
- Propranolol (beta-blocker) does NOT improve cardiac contractility (past question) — it decreases it; used long-term for HF but not acutely
- Norepinephrine does NOT have positive inotropic effect (primarily vasopressor) (past question)
- Milrinone , dobutamine , dopamine , epinephrine = positive inotropes (past question)
- Beta-blockers : for long-term management only
- Nutritional support : fortified formula , NG feeding if needed

Cardiogenic Shock Management

- Oxygen + diuresis + inotropic support (past question : “correct management of viral myocarditis cardiogenic shock”)
- Avoid large fluid boluses (can worsen pulmonary edema)
- Dilated cardiomyopathy with shock : 10 ml/kg fluid bolus (careful) → inotropes (past question)

Marker of Worsening in Shock

- Decreased central venous O2 saturation (e.g. from 60% to 45%) = worse (past question)
- Decreased serum lactate = improvement ; increased pH = improvement

🔷 32. ARRHYTHMIAS

Normal Cardiac Physiology

- Heart rate decreases with age (past question : “increases” = WRONG)
- Blood pressure increases with age (past question)
- PVR is highest at birth then decreases (past question : “PVR lowest at birth” = WRONG)

ECG Key Points

- RVH : Tall R in V1 , deep S in V6 , T wave opposite of normal in V1 , pure R (or QR) in V1
- LVH : Tall R in V6 , deep S in V1 ; if T wave inverted in V6 → strain pattern ; deep Q in V6 → septal hypertrophy
- Long QT : QTc = QT / √R-R ; normal QTc ≤ 450 ms ; Long QT → syncope , VT/VF

SVT (Supraventricular Tachycardia)

- Usually narrow complex ; rate outside normal sinus range
- Most common arrhythmia in children requiring treatment
- Signs of hemodynamic compromise (low BP , cold extremities) = low cardiac output (past question : SVT + low BP + cold extremities = low cardiac output)
- HR > 220 = SVT (in infants) (past question)

Management :

- Stable SVT involving AV node (AVNRT , WPW) : adenosine to terminate
- Unstable SVT : synchronized cardioversion
- WPW : short PR interval + delta wave ; avoid digoxin and verapamil

Sudden Cardiac Death — Causes to Consider

- Aortic stenosis , long QT syndrome , hypertrophic obstructive cardiomyopathy (HOCM) , coronary anomalies
- Coarctation of the aorta does NOT cause sudden cardiac death (past question)
- SVT does NOT cause sudden cardiac death (past question)
- Mitral regurgitation alone does NOT cause SCD (past question)
- A diastolic murmur at right 2nd intercostal space = aortic regurgitation , NOT mitral stenosis (past question : “associated with mitral stenosis” = WRONG)

Syncope Red Flags Suggesting Cardiac Cause

- Syncope during exercise
- Retrosternal chest pain before fainting
- Abnormal blood pressure (high or low)

- RV heave on exam
- A murmur that disappears when standing = innocent murmur → reassurance (past question : "murmur disappears standing + syncope" = reassurance for the murmur itself ; but syncope during exercise needs workup)

🔷 33. SHOCK

Definition

- Significant systemic reduction in tissue perfusion → decreased oxygen delivery → cellular hypoxia

Key Concept

- Hypotension is a LATE sign in children — children compensate well (past question)
- Tachycardia is the EARLIEST sign of compensated shock
- Children rely on heart rate more than stroke volume (cannot modify stroke volume as well)

Types of Shock

Type	Mechanism	Skin	Pulses	Examples
Hypovolemic	↓ preload	Cool , pale	Weak	Gastroenteritis , bleeding , burns
Distributive (septic)	↓ SVR	Warm/flushed early	Bounding early	Sepsis , anaphylaxis
Cardiogenic	↓ contractility	Cool	Weak	Myocarditis , HLHS , arrhythmia
Obstructive	Mechanical obstruction	Cool	Weak	Tension pneumothorax , tamponade , PE

- Most common cause of shock in children = hypovolemic (from gastroenteritis)
- Diarrhea + shock → mechanism = decreased preload (past question)
- Mechanism of respiratory failure in reactive airway disease = V/Q mismatch (past question)
- Best indicator of oxygenation = SpO2 (past question)
- Worsening shock marker = decreased SvO2/ScvO2 (past question)

Hypotension Thresholds by Age

Age	Hypotension
Neonates (0–28 days)	< 60 mmHg systolic
Infants (1–12 months)	< 70 mmHg systolic
Children 1–10 years	< 70 + (2 × age in years) mmHg systolic
≥ 10 years	< 90 mmHg systolic

Management

- First : ABCDE ; secure IV/IO access
- Fluid bolus : 20 mL/kg NS or Ringer's lactate over 10–15 min ; up to 60 mL/kg in first hour
- Cardiogenic shock : start with 5–10 mL/kg (careful) , avoid overload
- Anaphylaxis : first treatment = IM epinephrine (past question)
- Ductal-dependent lesion (suspected in neonate) : start PGE1 (past question : "unstable infant with suspected cyanotic CHD = give PGE1")
- Hemorrhagic shock : Crystalloid boluses , If unresponsive to initial crystalloid bolus (or if severe bleeding is obvious), give 10 mL/kg of packed RBCs (O-negative if uncrossmatched) or Whole Blood.
- Septic shock : early antibiotics within 1 hour + vasopressors if needed
- Refractory shock : consider adrenal insufficiency → stress dose hydrocortisone ; check echo ; vasopressin if unresponsive

MCQ TRAP SUMMARY

Wrong Statement	Correct
PVR lowest at birth and increases	Highest at birth , then decreases
Tricuspid atresia has RV heave	No RV heave (hypoplastic RV)
TGA has wide mediastinum	Narrow mediastinum (egg-on-string)
Loud murmur in TGA	Usually no murmur
ASD has gallop rhythm	No gallop (gallop = VSD/HF)
ASD has LV dilatation	RA and RV dilatation
Eisenmenger in ASD = late childhood	Late adulthood
TOF has cardiomegaly	Boot-shaped heart , decreased PBF , NO cardiomegaly
Squatting worsens TOF cyanosis	Squatting improves cyanosis (↑ SVR)
Worsening murmur in TOF = VSD growing	Means pulmonary obstruction worsening
Clubbing in 3-month-old with TOF	Too early — clubbing appears later
CoA has wide mediastinum	Wide mediastinum = TGA ; CoA = notched ribs
PDA causes RA hypertrophy	NOT RA hypertrophy
Furosemide causes metabolic acidosis	Causes metabolic alkalosis (hypokalemic hypochloremic)
Propranolol improves contractility	Decreases contractility

Norepinephrine = positive inotrope	Primarily vasopressor ; not a classic inotrope
Polycythemia = compensatory mechanism in HF	NOT a mechanism in HF (seen in cyanotic CHD)
CO increases with increasing afterload	CO decreases with increasing afterload
Newborn cannot increase CO as well as older children	Newborn has limited ability to increase stroke volume , relies on HR
Foramen ovale : blood flows left → right in fetus	Right → left in fetal life
Umbilical arteries carry oxygenated blood	Umbilical vein carries most oxygenated blood
TOF needs PDA for survival	Does NOT need PDA
Down syndrome — TOF is most common CHD	AVSD (AV canal defect) is most common
Diastolic murmur in right 2nd ICS = mitral stenosis	Aortic regurgitation
Coarctation causes sudden cardiac death	Does NOT cause SCD
SVT causes sudden cardiac death	Does NOT cause SCD
Mid-systolic murmur disappearing on standing = pathological	Innocent murmur → reassurance

4. CARDIOLOGY

4.1 Resuscitation

- Neonatal resuscitation: PPV is first step when HR <60 bpm
- Chest compressions start when HR <60 after 30 seconds of adequate PPV
- Neonatal CPR ratio: 3:1 (3 compressions : 1 breath)
- Pulse check site in neonate: umbilical artery (NOT carotid or radial)
- IV route preferred for medications in neonatal resuscitation
  - ✗ **WRONG:** Umbilical artery is not the site for pulse in neonatal resuscitation → **CORRECT:** Umbilical artery IS the preferred site for neonatal pulse
- DO NOT use ambu bag in congenital diaphragmatic hernia (causes bowel herniation into chest to expand more)
- Cardiac arrest with asystole: resume chest compressions immediately (no shock for asystole/PEA)
- V-fib and pulseless V-tach: DEFIBRILLATE
- PEA (pulseless electrical activity): NO shock — CPR + treat cause
- Stop CPR to check rhythm (not to check pulse every 30 sec)
  - ✗ **WRONG:** Stop chest compressions every 30 seconds to check pulse → **CORRECT:** Do NOT stop every 30 sec — check rhythm as needed, resume immediately

4.2 Arrhythmias

- SVT: most common tachyarrhythmia in children
  - Narrow complex QRS, rate 250–300 bpm, no visible P waves
  - Infant: irritability, pallor, tachypnea, poor feeding, gallop rhythm
  - Treatment: if hemodynamically stable → IV adenosine; if unstable → synchronized cardioversion
  - NOT a cause of sudden cardiac death
- Long QT syndrome: associated with sudden cardiac death, syncope
  - **(past question)** 3-year-old, consanguineous parents, hearing loss, syncope, FHx sudden deaths → Long QT syndrome (Jervell-Lange Nielsen syndrome)
- HOCM: cause of sudden cardiac death
- Pulmonary stenosis: NOT associated with sudden cardiac death
  - ✗ **WRONG:** Pulmonic stenosis is associated with sudden cardiac death → **CORRECT:** Pulmonic stenosis is NOT associated with sudden cardiac death

4.3 Shock

- MCC of pediatric shock: hypovolemic shock
- MCC of shock requiring ICU in children: septic shock
- Decreased CO in hemorrhagic shock: decreased preload
- Shock can be diagnosed before hypotension develops; low BP is a LATE sign
  - ✗ **WRONG:** Decrease in BP is required to diagnose shock → **CORRECT:** Low BP is a LATE sign; shock diagnosed before BP drops
- Compensated shock signs: tachycardia, prolonged CRT, cool extremities, normal BP
- Septic shock treatment: antibiotics, fluid, vasopressors, nutritional support, mechanical ventilation
  - ✗ **WRONG:** Diuretics help in septic shock to increase urine output → **CORRECT:** Diuretics are NOT given in septic shock

4.4 Cardiomyopathy

- Dilated cardiomyopathy: systolic dysfunction (correct)
- Most important prognostic factor: improvement in ejection fraction (EF)
- Indicators of improvement: increased UOP, decreased CRT, decreased HR, improved BP, decreasing lactate
  - ✗ **WRONG:** Decrease of HCO3 from 24 to 18 indicates improvement in dilated cardiomyopathy → **CORRECT:** Decreasing HCO3 means worsening acidosis, NOT improvement
  - ✗ **WRONG:** UOP from 0.5 to 1.2 mL/kg/hr is NOT an indicator of improvement → **CORRECT:** Increased UOP IS an indicator of improvement

4.5 Pericarditis / Myocarditis

- Pericarditis: sharp midsternal pain, friction rub, low-grade fever → first step: ECG + CXR
- Myocarditis: after viral illness, gallop, cardiomegaly on CXR, tachycardia, respiratory distress

4.6 Rheumatic Fever

- Caused by Group A beta-hemolytic streptococcus (GABHS) pharyngitis
- Evidence of GABHS required for diagnosis: positive throat culture or elevated ASO titer
- Jones Criteria — Major: carditis, polyarthritis, chorea, erythema marginatum, subcutaneous nodules

    - ✗ **WRONG:** Fever is a major criterion of rheumatic fever → **CORRECT:** Fever is a MINOR criterion
    - ✗ **WRONG:** Janeway lesion is a major criterion of RF → **CORRECT:** Janeway lesions are in infective endocarditis, NOT RF
    - ✗ **WRONG:** Cervical lymphadenopathy is a criterion of RF → **CORRECT:** Not a Jones criterion
- Arthritis: migratory, self-limiting, resolves completely with no deformity
- Affected valve: mitral valve most common (MR first, then MS in chronic)
    - ✗ **WRONG:** Aortic valve is the most likely affected in rheumatic carditis → **CORRECT:** Mitral valve is most commonly affected
- Mitral stenosis murmur: apical diastolic with presystolic accentuation
- Prophylaxis: long-acting penicillin
    - No carditis: 5 years or until age 21
    - With carditis (no damage): 10 years or until age 21
    - With persistent valve disease: lifelong
    - ✗ **WRONG:** Prophylaxis for 5 years is correct when there is cardiac involvement → **CORRECT:** With cardiac involvement, prophylaxis should be for 10 years (not 5)

4.7 Murmurs

- Innocent murmur: systolic, grade 1–2/6, disappears with standing/Valsalva, no radiation
- NEVER innocent: diastolic murmur, grade 3+, radiating, thrill
    - ✗ **WRONG:** Diastolic murmur can be innocent → **CORRECT:** Diastolic murmurs are NEVER innocent
- Mitral stenosis: DIASTOLIC murmur at apex with presystolic accentuation
- Systolic murmur that INCREASES with Valsalva: HOCM
- Wide pulse pressure causes: AR, PDA, truncus arteriosus, large AVM
    - ✗ **WRONG:** Mitral stenosis causes wide pulse pressure → **CORRECT:** Mitral stenosis causes NARROW pulse pressure

4.8 Infective Endocarditis (IE)

- Staphylococcus aureus is the overall most common cause of infective endocarditis, while Streptococcus viridans is classically associated with subacute IE and dental procedures.
- Work-up: 3 blood cultures from different sites before starting antibiotics

4.9 Hypertension

- Definition in children/adolescents: average BP ≥95th percentile (not 140/90 as in adults)
- Single BP measurement 140/85 in 12-year-old: needs repeat measurement first
- MCC renovascular HTN in childhood: fibromuscular dysplasia
- HTN crisis: IV nitroprusside (drug of choice)
- HTN with proteinuria: ACE inhibitor first line
- BP cuff: large cuff → falsely LOWER readings
    - ✗ **WRONG:** Large cuff gives falsely higher readings → **CORRECT:** Large cuff gives falsely LOWER BP readings
- Causes of HTN in children: renal artery stenosis, pheochromocytoma, coarctation of aorta, PSGN
    - ✗ **WRONG:** MCD causes HTN → **CORRECT:** MCD does NOT cause HTN (normal BP in nephrotic syndrome usually)

4.10 Anaphylaxis

- First-line treatment: IM epinephrine (intramuscular, NOT IV)
- Also used: antihistamines, inhaled beta-agonists, corticosteroids
  - ✗ **WRONG:** Theophylline is used in anaphylaxis → **CORRECT:** Theophylline is NOT used in anaphylaxis

4.11 Congenital Rubella — Cardiac

- PDA most commonly associated with congenital rubella
- Cardiac defects occur with first trimester infection (not third trimester)
  - ✗ **WRONG:** Subclinical maternal rubella does not lead to congenital infection → **CORRECT:** Even subclinical infection CAN cause congenital rubella

⚡ HIGH-YIELD — CARDIOLOGY

- Neonatal pulse check: umbilical artery
- PEA: no shock; asystole: no shock — resume CPR
- SVT treatment: adenosine (stable) → cardioversion (unstable)
- Shock: low BP is LATE sign; diagnose before hypotension
- Rheumatic fever: mitral valve most affected; with carditis → 10 years prophylaxis
- Innocent murmur: NEVER diastolic
- HTN in children: defined as ≥95th percentile; single reading → repeat first
- Anaphylaxis: IM epinephrine (not IV)
- Large cuff → falsely LOW BP