Congestive Cardiac Failure (CCF) in Pediatrics

Concise, exam-oriented notes for MBBS Final Year

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1. Introduction

Definition: Inability of the heart to meet metabolic demands, causing inadequate perfusion and fluid accumulation in lungs/tissues.
Epidemiology: Infants—most commonly congenital heart disease; older children—myocarditis/cardiomyopathy more frequent.

Hallmark	Tachypnea + hepatomegaly ± feeding difficulty in infants; exercise intolerance in older children.
Edema	Peripheral edema uncommon in infants; hepatomegaly is a more reliable sign.
Classification	Use Ross classification in infants/children (functional class I–IV).

2. Etiology

- Congenital: VSD, PDA, AVSD, large ASD, coarctation of aorta, aortic/pulmonary stenosis, total anomalous pulmonary venous connection (TAPVC).
- Acquired: Myocarditis, dilated/hypertrophic cardiomyopathy, rheumatic heart disease, severe anemia (e.g., thalassemia), arrhythmias (SVT), Kawasaki disease/coronary anomalies, endocrine (thyrotoxicosis), high-output states (AV malformation, beriberi).

Common Causes by Age

Age Group	Typical Causes
Neonate/Infant	Large L→R shunts (VSD, PDA, AVSD), critical obstructive lesions (coarctation), myocarditis; duct-dependent lesions when PDA closes.
Toddler/Child	Unrepaired CHD, myocarditis/cardiomyopathy, SVT, severe anemia/nutritional deficiencies.
Adolescent	Cardiomyopathy (familial/idiopathic), rheumatic valve disease, coronary issues (Kawasaki sequelae), endocrine causes.

3. Pathophysiology

- Volume overload: L→R shunts (VSD/PDA/AVSD) → chamber dilation → pulmonary overcirculation.
- Pressure overload: Outflow obstruction (coarctation, AS/PS) → concentric hypertrophy.
- Decreased contractility: Myocarditis/cardiomyopathy → systolic dysfunction.
- Neurohormonal activation: RAAS, sympathetic & ADH activation → fluid retention, vasoconstriction, ventricular remodeling.

4. Clinical Features

Symptoms:
- Infants: Poor feeding, tachypnea, diaphoresis during feeds, failure to thrive, irritability.
- Older children: Fatigue, exertional dyspnea, orthopnea/PND, decreased exercise tolerance.

Signs:
- Tachycardia, tachypnea, subcostal/intercostal retractions.
- Hepatomegaly (reliable in infants), basal crackles/wheeze, gallop rhythm (S3 ± S4).
- Peripheral edema, ascites, elevated JVP (more in older children).

5. Diagnosis
- Clinical: History + exam remain central.
- Chest X-ray: Cardiomegaly; pulmonary edema/plethora (Kerley B lines in older children).
- ECG: Chamber enlargement, ischemia/myocarditis patterns, arrhythmias (SVT).
- Echocardiography: Structure (CHD), function (EF/FS), pulmonary pressures.
- Blood tests: BNP/NT-proBNP ↑; CBC (anemia), electrolytes, renal/liver function; inflammatory markers if myocarditis suspected.

6. Management
General measures: Oxygen, upright positioning, cautious fluids, salt restriction, high-calorie feeds; treat precipitating causes (infection, anemia, arrhythmia).
Acute decompensation (hospital/PICU): IV loop diuretics; consider inotropes/vasodilators (dobutamine/milrinone) with monitoring; manage arrhythmias; prostaglandin E_1 in duct-dependent shock (neonates).

Core Drugs – Quick Reference

Drug	Purpose	Key Notes
Furosemide	Diuresis ↓ preload	Monitor electrolytes & renal function; consider spironolactone add-on.
ACE inhibitors (Captopril/Enalapril)	Afterload reduction	Titrate to effect; watch for hypotension, renal dysfunction, hyperkalemia.
Spironolactone	K^+-sparing diuretic; neurohormonal block	Adjunct with loop diuretics; monitor K^+.
Digoxin	↑ Contractility; rate control	Use in systolic dysfunction/AF; dose carefully; check renal function.
Carvedilol (selected cases)	Neurohormonal modulation	Initiate when stable; specialist guidance in pediatrics.
Milrinone/Dobutamine (acute)	Inotropy/afterload ↓	PICU use with hemodynamic monitoring.

Definitive therapy: Correct structural lesions (VSD/AVSD closure, PDA device/ligation, relieve coarctation), treat myocarditis/cardiomyopathy per etiology, control arrhythmias, address anemia/endocrine causes.

7. Complications

- Pulmonary hypertension; recurrent respiratory infections.
- Failure to thrive; cachexia.
- Renal/hepatic dysfunction; hyponatremia.
- Thromboembolism/arrhythmic events (cardiomyopathy).

8. Prognosis

Depends on cause & timing of intervention. Correctable CHD has excellent outcomes post-repair; primary cardiomyopathies vary—prognosis improves with optimized therapy and follow-up.

Exam Triggers	Infant with tachypnea + hepatomegaly + poor feeding • CXR cardiomegaly • Echo confirms function & lesions • Start loop diuretic + ACEi • Fix the underlying defect