Heart function and Heart failure in children

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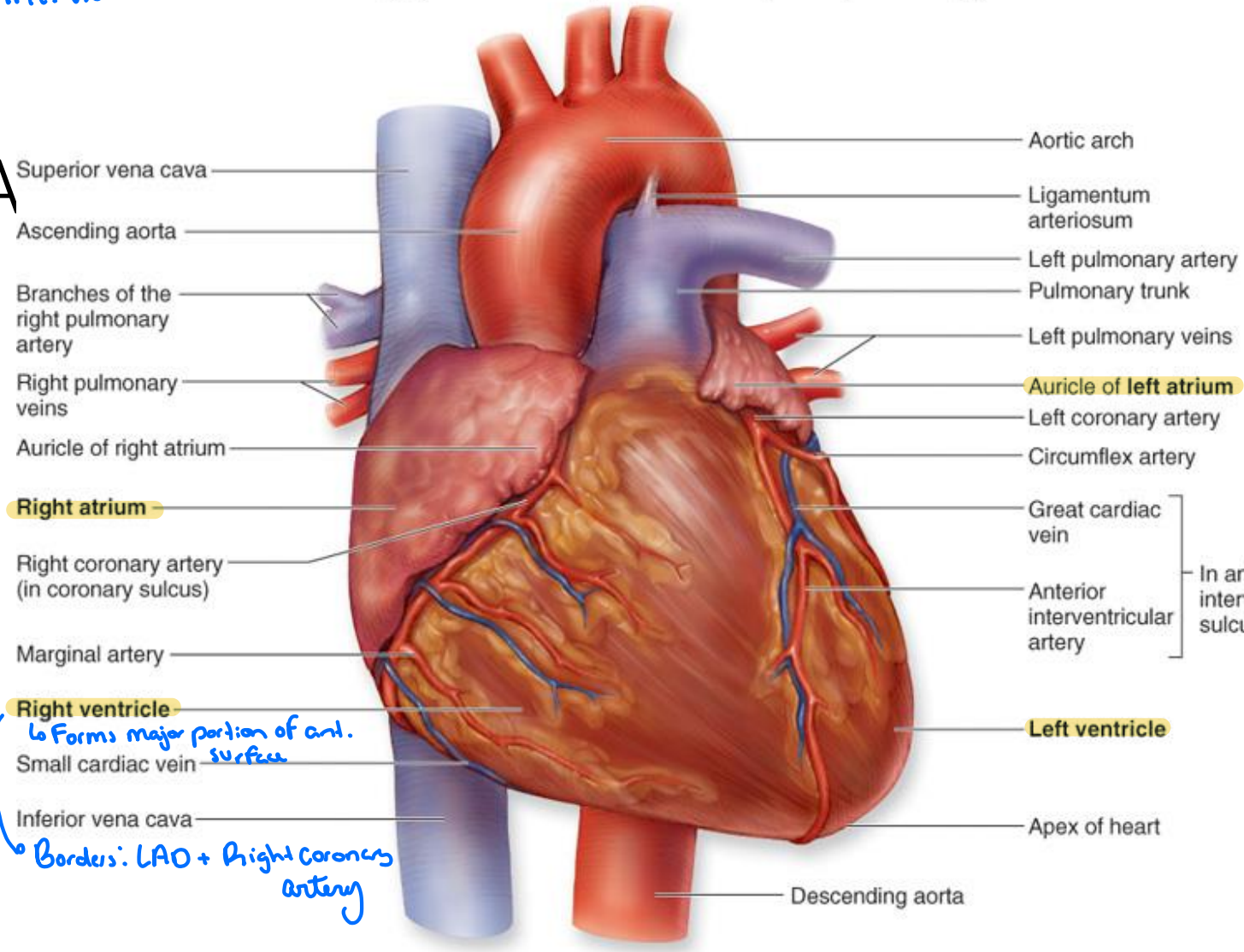
Outline

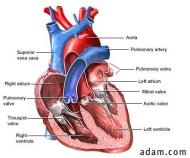
- Introduction
  - Anatomy revision
  - Physiology and cardiac output
  - Cardiac cycle revision
- Heart failure
  - Definition
  - Pathophysiology
  - Clinical picture and diagnosis
  - Etiology
  - Management guide

Ant. view

A



The aorta and pul. artery cross each other (which is why we hear the aorta on the right even tho it extends from the LV and vice versa for the pul. artery)



Ascending aorta
Superior vena cava
Right pulmonary artery
Right pulmonary veins
Right auricle
Interatrial septum
Right atrium
Opening for inferior vena cava
Right atrioventricular valve
Chordae tendineae
Papillary muscle
Right ventricle
Inferior vena cava

Aortic arch
Ligamentum arteriosum
Left pulmonary artery
Pulmonary trunk
Left pulmonary veins
Left atrium
Aortic semilunar valve
Left atrioventricular valve
Pulmonary semilunar valve
Trabeculae carneae
Interventricular septum
Left ventricle
Descending aorta

The coronary sinus drains the coronary veins into the RA

The pul. veins drain into the LA

Thin wall, rough border, thick trabeculae

Thick walled, bullet shape, smooth wall, fine trabeculations

The doctor mentioned right-sided & left-sided aortic arch if you want to look it up

You can't see the RV on AP X-ray

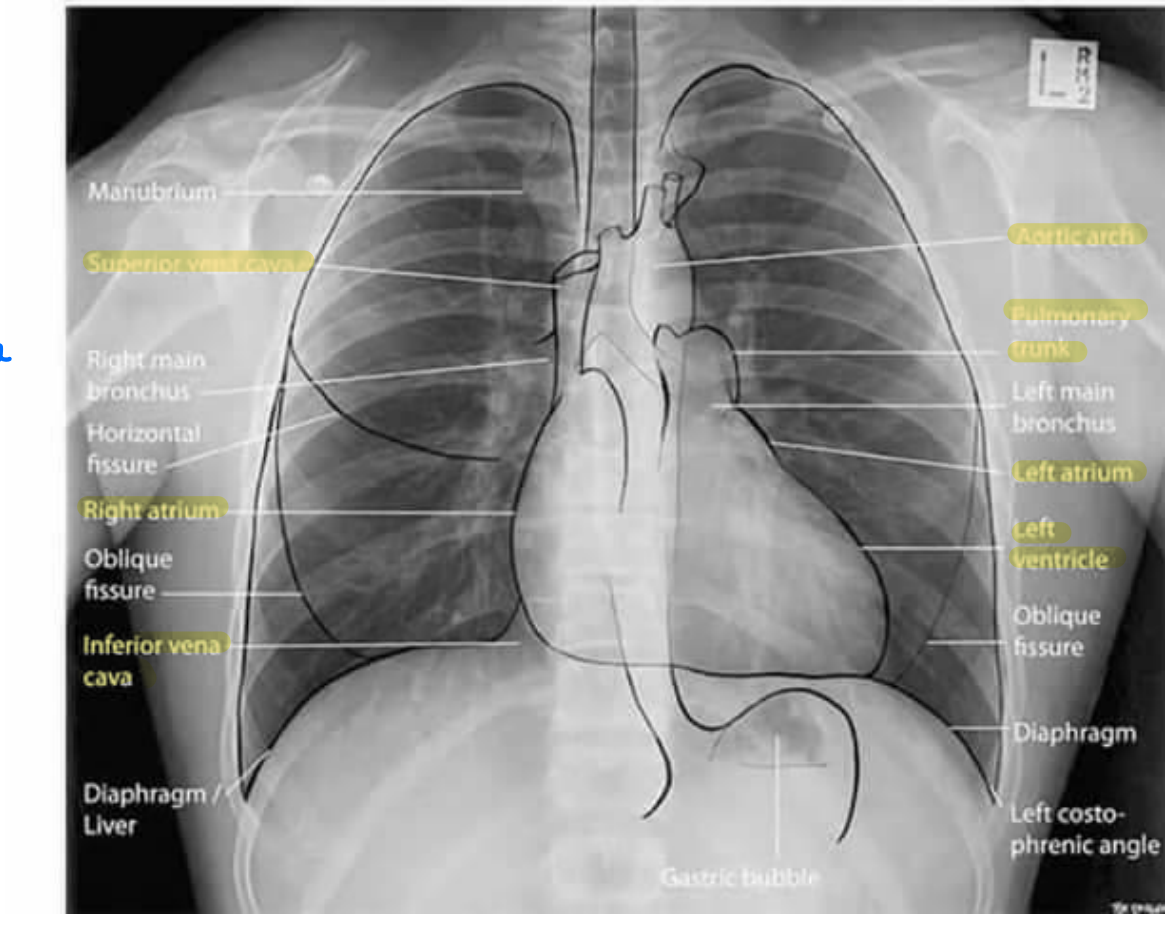
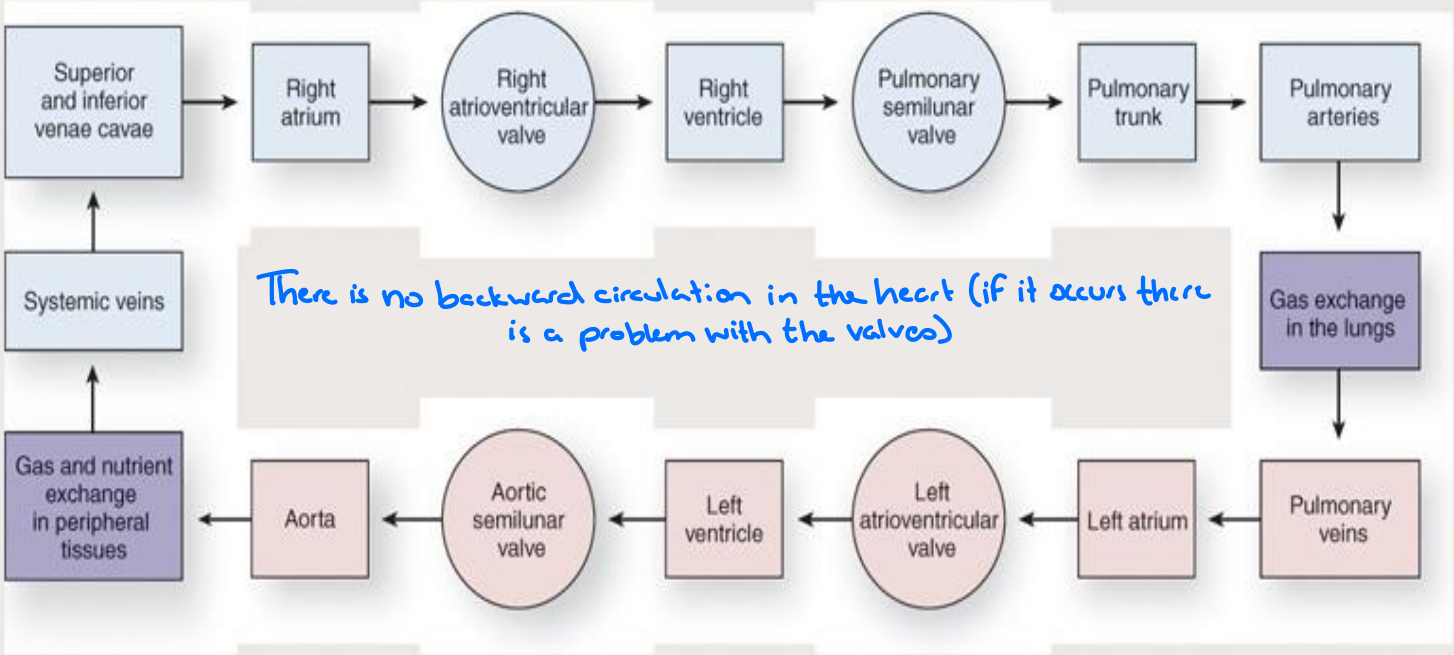
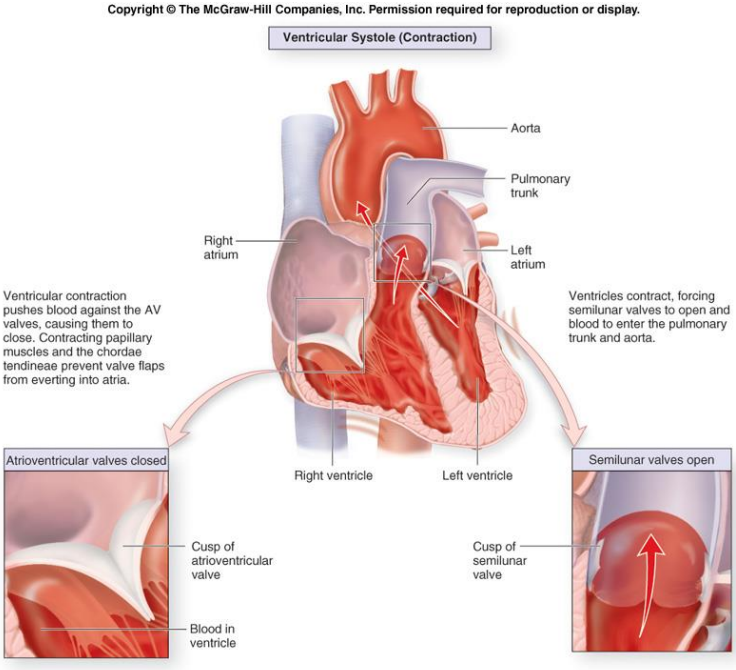


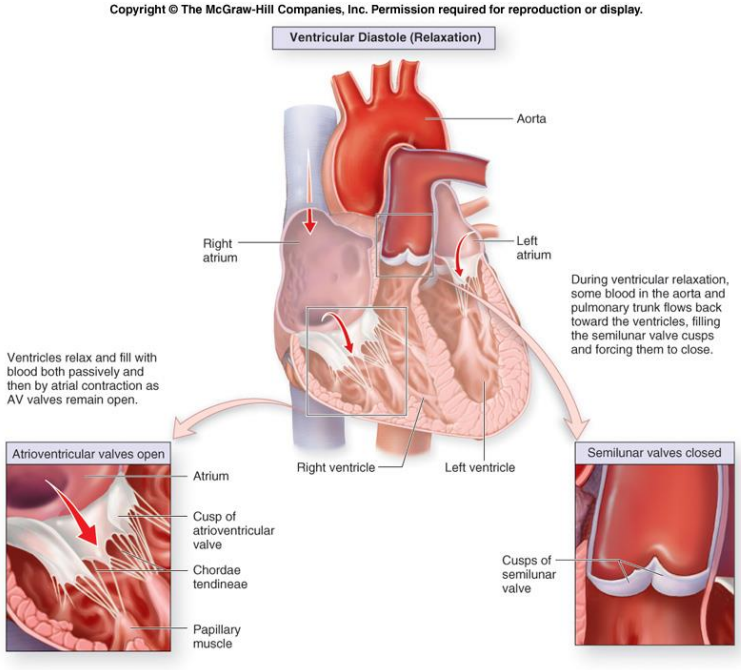
Table 22.3 Blood Flow Through the Heart



Chamber of the Heart	Receives Blood From	Sends Blood To	Valves Through Which Blood Flows
Right atrium	Superior vena cava, inferior vena cava, coronary sinus	Right ventricle	Right AV valve
Right ventricle	Right atrium	Pulmonary trunk (blood enters pulmonary circuit of vessels)	Pulmonary semilunar valve
Left atrium	Pulmonary veins	Left ventricle	Left AV valve
Left ventricle	Left atrium	Aorta (blood enters systemic circuit of vessels)	Aortic semilunar valve

Ventricular systole and diastole





There should be no shunting blt the systemic and pul. circulations. The circuits are in series, not parallel

Cardiac function

- Pumping of oxygenated blood to the systemic organs through systemic circulation, and pumping of de-oxygenated blood to the pulmonary circulation
- Blood flow to the systemic circulation is measured as liters/minute and is called (Cardiac output)
- Cardiac output is regulated by tissue demand for oxygen
- Cardiac output is a result of : stroke volume X heart rate per minute
- Stroke volume is the volume of blood ejected to the systemic circulation in one beat
- In normal heart

stroke volume = end diastolic volume - end systolic volume

SV is what reaches the Systemic Circulation, not necessarily what's pumped by the LV (as in a VSD blood goes to the Rt side or w/ MR effective CO is smaller than what the LV is pumping)

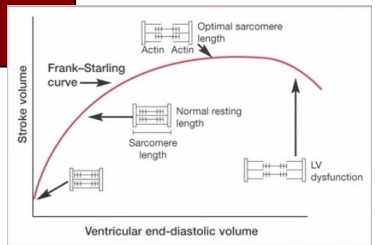
Cardiac output

Heart rate	X	Stroke Volume
Regulated by sympathetic and Parasympathetic nervous system Symp➔ ↑ Parasymp➔ ↓		Determined by: •Preload (volume) •Afterload (resistence) •Caontractility (↑ by SNS)

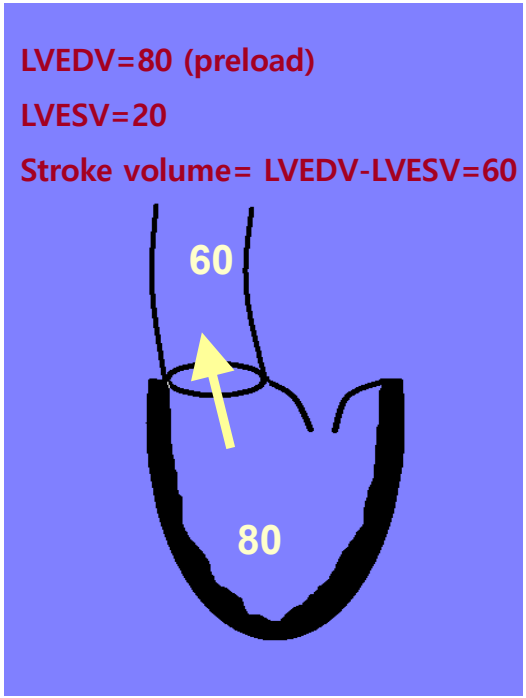
↑ preload = ↑ SV

↑ afterload = ↓ SV

Preload can't indefinitely ↑ SV (Frank-Starling law)



Stroke volume



Preload: volume of blood in LV at end diastole

(venous return)

Afterload: resistence against which the LV is pumping

Contractility: degree of shortening of the muscle when it contracts (strength of muscle)

In HF we like to give vasodilators (↓ afterload) to help ease the work of pumping of the LV

(but we have to be careful bc too much would ↓ BP)

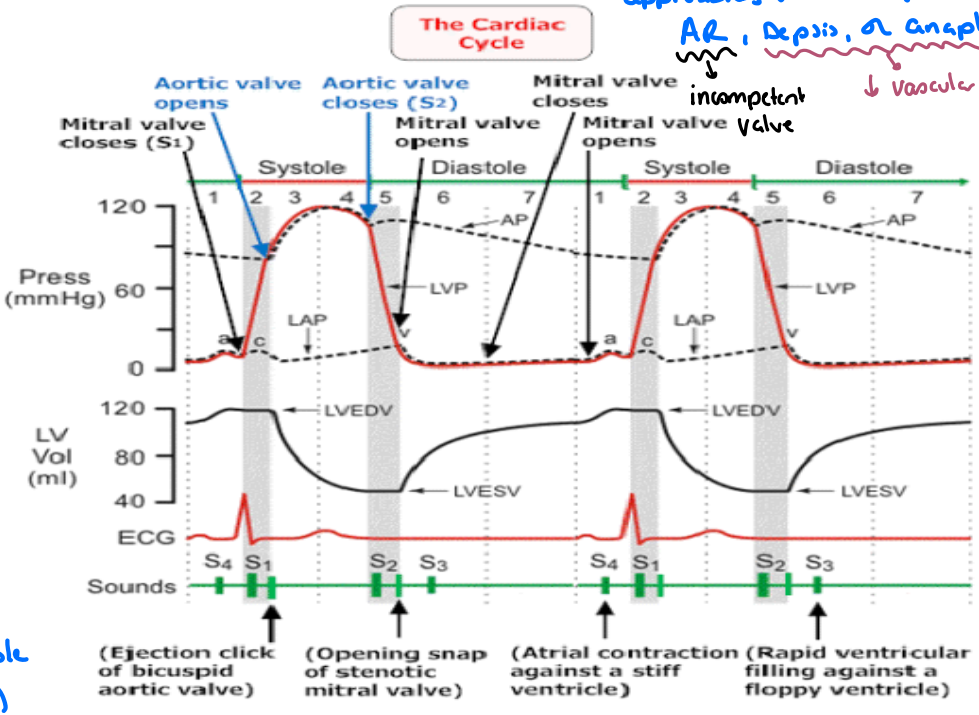
Card

The aortic valve + vascular tone is what maintains diastolic aortic pressure (dotted line) of 80

Widened pulse pressure (where the aortic diastolic pressure approaches ventricular pressure) could be due to AR, Sepsis, or anaphylaxis

AR: incompetent valve

Sepsis, anaphylaxis: ↓ vascular tone



S1 = beginning of Systole (ventricular contraction)

S2 = beginning of diastole

1 -> Atrial Contraction --> Mitral Valve Opens
2 -> Isovolumic Ventricular Contraction --> Mitral Valve Closes
---> Aortic Valve Opens
3 -> Rapid Ejection
4 -> Reduced Ejection
5 -> Isovolumic Ventricular Relaxation --> Aortic Valve Closes
---> Mitral Valve Opens
6 -> Rapid Ventricular Filling
7 -> Diastasis ---> Mitral Valve Closes
1 -> Atrial Contraction --> Mitral Valve Opens

Heart Failure

- Simply stated

Heart failure is the failure of the heart to produce cardiac output that meets the metabolic demand

Etiology and clinical presentation differs significantly between children and adults.

mostly due to congenital / structural causes

mostly due to IHD and cardiomyopathy

- In high output HF (like from hyperthyroidism) the problem is from abnormally high metabolic demand

Pathophysiology

What happens when supply becomes less than demand

Compensation mechanisms start to operate

Renin-Angiotensin-Aldosterone

Main effects:

- Fluid retention (aldosterone)
- vasoconstriction (angiotensin)

to ↑ preload

over-compensation can end up as a burden to the heart which is why we use ACEI / ARBs to treat HF

Autonomic nervous system

Quick Response

Main effects:

- Tachycardia
- Increased myocardial contractility
- vasoconstriction

Clinical presentations (SIGNS & SYMPTOMS)

S&S related to Fluid retention ➔congestion	S&S related to symp. stimulation	S&S related to low tissue perfusion
Pulmonary congestion: tachypnea, dyspnea, respiratory distress	**Tachycardia/ palpitation**	**Decreased capillary refil time**
Systemic congestion: edema, hepatomegaly, increased venous pressure	**Diaphoresis (infants)**	**Exercise intolerance/fatigue (in infants this results in poor feeding and poor weight gain)**
	Irritability (infants)	**Low urine output**
	S&S of vasoconstriction (cold extremities, poor pulses)	**Altered level of consciousness**
		S&S of metabolic acidosis

AND: S&S of the etiology of heart failure

Diagnosis of heart failure

Mainly depends on the clinical features

Other helpful investigations:

ECG: if there is suspicion of arrhythmogenic cause, or secondary rhythm disturbance. Also helps in some structural heart disease

CXR: May show cardiomegaly (not specific for etiology), shows the degree of pulmonary adema

Echocardiography: Very helpful in determining etiology of HF

Other investigations helps determining severity/complications/etiology:

1)

2)

3)

Etiology of HF in children

CHD with increased pulmonary blood flow

- VSD (holosystolic murmur)
- PDA (machinery murmur, wide pulse pressure)
- AV canal defect (Down syndrome)
- Truncus arteriosus (mild desaturation, possible ejection click, wide pulse pressure)

CHD with flow obstruction

- Aortic stenosis (click, radiation to the neck)
- Coarctation of aorta (high blood pressure, poor femoral pulses)

Poor myocardial contractility

- Dilated cardiomyopathy (family history)
- Myocarditis (hx of viral infection)
- **Sepsis:** resulting in septic shock/ organ damage
- **Rare-** coronary artery anomalies

HF due to dysrrhythmia

- SVT (HR >220)
- Bradycardia (complete heart block), congenital CHB presents earlier

High output failure: High demands
Examples: thyrotoxicosis, Severe anemia, extensive AV malformation

Management guide for HF in children

Nutritional support	Increase caloric intake (fortified formulas, more frequent feeds, NG feeding if needed)
Medications	Diuretics: (examples: furosemide, spironolactone) decrease the congestion, improves respiratory distress Afterload reduction (Examples: ACE, ARB): improve tissue perfusion, in some cases may manipulate the shunts (in CHD) Inotropic support: Examples are sympathomimetics, phosphodiesterase inhibitors, digoxin Beta-blockers: for long term use
Treating the etiology	CHD➔ repair Arrhtyhmia ➔ Rhythm control, pacemakers.. Devices and transplant for refractory cases

END

Additional reading: Pediatric Heart Failure: A Practical Guide to Diagnosis and Management - Pediatrics & Neonatology (pediatr-neonatol.com)