

CONGESTIVE HEART FAILURE IN CHILDREN

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OBJECTIVES:

At the end of the study, the student should be able to

1. Define heart failure and discuss its pathophysiology.
2. Recognize heart failure given signs and symptoms of a child with CHF.
3. Give the classification and stage of heart failure of a child with CHF.
4. Discuss the laboratory work-up that will help in the diagnosis of heart failure, its etiology, and initial management.
5. Enumerate possible etiology of heart failure based on the clinical manifestations and onset of heart failure.
6. Discuss the initial management of a child with heart failure.

Definition and Pathophysiology

- Heart failure is a clinical syndrome that arise from inadequate oxygen delivery by the heart or circulatory system to meet the demands of the body, and/or accommodate venous return from the body
- It is brought about by both
 - ❖ **Mechanical effect** of cardiac disease
 - ❖ **Maladaptive** Compensatory Mechanisms

HEART FAILURE Pathophysiology

NEUROHORMONAL PARADIGM

CLINICAL SYNDROME

- Heart Disease
- Reduced Cardiac output
- Increased Venous Pressures
- Cardiac remodelling

$$BP = CO \times PVR$$

HR

STROKE
VOLUME

In Heart failure, **MALADAPTIVE regulatory mechanisms** to increase **Cardiac Output** contribute to the signs and symptoms of Heart Failure

PRELOAD
CONTRACTILITY
AFTERLOAD

Heart Failure: pathophysiology

INADEQUATE CARDIAC OUTPUT AND OXYGEN DELIVERY

Myocardial dysfunction
(myocarditis, cardiomyopathy)

Volume overload (CHD with
increased pulmonary blood flow,
valvar insufficiency)

Arrhythmia (e.g. SVT, VT)

PHYSIOLOGIC COMPENSATIONS

NEUROHORMONAL ACTIVATION

- Sympathetic nervous system
- Renin-angiotensin-aldosterone system
- Arginine vasopressin (AVP/ADH)

MANIFESTATIONS

EASY FATIGABILITY

SALT AND WATER RETENTION

(pulmonary and systemic congestion)

INCREASED CARDIAC RATE

VASOCONSTRICTION

(pallor, cold extremities, narrow pulse pressure)

LEFT VENTRICULAR REMODELLING

(chronic; LV structural and functional deterioration)

COMPENSATORY MECHANISMS And Overview of Clinical Manifestations

- INCREASED PRELOAD

- pulmonary congestion
 - Tachypnea, dyspnea, orthopnea
- Systemic congestion
 - Hepatomegaly, bipedal edema
- Pallor

- INCREASED SYMPATHETIC STIMULATION

- Tachycardia
- Diaphoresis
- Arrhythmia

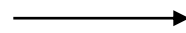
- INCREASED AFTERLOAD

- Vasoconstriction
- Cold extremities
- pallor

HALLMARK OF HEART FAILURE

❖ Decreased oxygen delivery

EASY FATIGABILITY



SHOCK

INTERMITTENCY OF
FEEDING

GETS TIRED FASTER THAN
PLAYMATES/PEERS

ALTERED SENSORIUM
DECREASED URINE OUTPUT

TACHYCARDIA
COLD EXTREMITIES
HYPOTENSION

An Infant with Heart Failure: History

A 6 month-old male was brought to the Emergency room for **difficulty of breathing**.

The patient was born full term with good suck and activity. The baby was doing well until **2 months old** when he was noted to **have intermittency of feeding, diaphoresis while feeding, and poor weight gain**.

There were previous hospital admissions for **Respiratory Tract Infection** 1 week PTC, the patient started to have fever, cough, and **progressive dyspnea** prompting consult

A Older Child with Heart Failure: History

- An 10 year old female consulted at the clinic for **cough and pallor**
- 1 month PTC, the patient was noted to have **easy fatigability** associated with cough and moderate grade fever temporarily relieved by Paracetamol. A consult was done and the patient was suspected to have a cardiac disease. They were advised to seek consult with a pediatric cardiologist. However, no consult was done due to financial constraints. 1-2 weeks PTC, the patient started to have **low grade fever, paroxysmal nocturnal dyspnea and 2-pillow orthopnea**. Persistence and progression of symptoms prompted consult.

HISTORY

NEWBORNS/INFANTS

- Birth and Maternal History** (look for risk factors for cardiac disease like maternal intake of teratogenic drugs, or maternal infection at birth)
- Intermittent feeding**
 - Feeding may be labored or impossible due to rapid breathing
 - Slow or poor feeding
 - Easy fatigability
- **Diaphoresis**
 - especially on feeding
 - even on cool and temperate environments
- **Poor weight gain** or weight loss
- **Repeated respiratory tract infection**/chronic cough
- Agitation & unusual irritability

HISTORY

CHILDREN AND YOUNG ADULTS

- Exercise Intolerance (easy fatigability)
- Tachypnea, shortness of breath, dyspnea
- Abdominal pain or distention
- Orthopnea & paroxysmal nocturnal dyspnea - older children
- Chronic cough
- Puffy eyelids (more seen in renal diseases), swollen feet (dependent edema)

An Infant with Heart Failure: Physical Examination

On Physical examination:

- An irritable, tachypneic, tachycardic, diaphoretic child, weight for length z-score <2
- (+) SC/IC retractions, (+) precordial bulge, and Harrison's groove; dynamic precordium with left ventricular heave; distinct heart sounds, tachycardic, regular rhythm, grade 3/6 holosystolic murmur at the left lower sternal border
- Slight hepatomegaly, full equal pulses

A Older Child with Heart Failure: Physical Examination

- Ambulatory, in mild cardiorespiratory distress
- **HR= 120/minute RR =34/min BP= 90/50 Wt= 21 kg T= 37.7 C**
- **Sallow**, pink conjunctivae, no alar flaring, no periorbital edema
- (-) neck vein engorgement
- Equal chest expansion, No SC IC retractions, **(+) occasional rales on lung bases**
- No precordial bulge, Adynamic precordium, (-) thrill, **AB/PMI 5th IC LAAL**, distinct heart sounds, tachycardic, regular rhythm, **grade 3/6 holosystolic murmur at the apex with radiation to axilla**
- **(+) hepatomegaly, 1-2 cm below the right costal margin**
- Full equal pulses, no edema

PHYSICAL EXAMINATION

GENERAL SURVEY

- ❖ Failure to thrive (thin, cachectic- verify with anthropometrics)
- ❖ Anxious (as opposed to a calm child who smiles)
- ❖ Irritable (inadequate cerebral perfusion, maybe hypoxemic)
- ❖ Lacks color (pale looking due to vasoconstriction or anemia)
- ❖ Diaphoretic (hyperadrenergic state)
- ❖ Distress, cardiorespiratory
(tachypneic, tachycardic- in some, pulsations are visible)

PHYSICAL EXAMINATION

ANTHROPOMETRICS AND VITAL SIGNS

a. Weight for length/height z score < 2

b. Sinus tachycardia

- Sustained CR > 160/min – infants
> 100-125/min – older children

Think about Supraventricular tachycardia if > 220 in infants and > 180 for older children

b. BP- Hypotension

c. RR- Tachypnea

PHYSICAL EXAMINATION

CARDIAC EXAMINATION:

- Harrison's groove (lateral chest indentation over insertion of diaphragm due to chronic poor lung compliance)
- Dynamic precordium
- Displaced apex beat, diffuse PMI (found in more than one interspace)
- Prominent 3rd (other) HS (gallop rhythm)
- Tachycardia
- Heart murmur- **may or may not be present**

PHYSICAL EXAMINATION

LUNG EXAMINATION- signs of pulmonary congestion

- ❖ Compromised oxygenation and ventilation
- Tachypnea and **retractions** (*usually* subcostal if mild; intercostal, moderate; suprasternal or with neck muscles if severe)
- Labored respiratory effort
- Nasal flaring
- Grunting
- Hacking cough – bronchial mucosal edema
- Rales- unusual in infants; may suggest concurrent pneumonia
- Wheezing- not all wheezing is asthma, may be a sign of heart failure (bronchiolar edema)

PHYSICAL EXAMINATION

SIGNS OF SYSTEMIC CONGESTION

- Hepatomegaly (Question: When do you say that a child has hepatomegaly?)
- Neck vein distention/engorgement- hard to appreciate in small children
- Facial edema/Puffy eyelids-unusual
- **Sacral or scalp edema** (dependent edema)

PHYSICAL EXAMINATION

EXTREMITIES

- Cool extremities (vasoconstricted)
- ↓ peripheral pulse
- ↑ capillary refill time
- Bipedal/peripheral edema – extremely rare in infants
 - ❖ **Sacral or scalp edema** (dependent edema in infants)

Heart Failure FUNCTIONAL Classification:

Guides initial management and evaluation of treatment

(How?)

CLASS	NYHA	ROSS
I	Without limitation of physical activity. No symptoms with ordinary physical activity.	Asymptomatic
II	With slight limitation of physical activity. Symptoms with ordinary physical activity.	Mild tachypnea or diaphoresis with feeding in infants
III	With marked limitation of physical activity. Symptoms with less than ordinary physical activity.	Marked tachypnea or diaphoresis with feeding in infants Prolonged feeding times with growth failure
IV	Cannot carry on any physical activity without discomfort. Symptoms at rest.	Symptoms such as tachypnea, retractions, grunting, or diaphoresis at rest

STAGES of heart failure in infants and children and recommended therapy

Stage	Definition	Example
A	Patients with increased risk of developing HF, but with normal cardiac function and chamber size	Congenital heart disease FMHx of cardiomyopathy Doxorubicin exposure
B	Patients with abnormal cardiac morphology or function, with no symptoms of HF, past or present	Single ventricle LV dysfunction Repaired CHD
C	Patients with structural or functional heart disease, and past or current symptoms of HF	Repaired and unrepaired CHDs Cardiomyopathy
D	Patients with end-stage <i>or severe</i> HF requiring specialized interventions	As in Stage C

Question: At what stage is a patient with CHF FC III secondary to a Large VSD in?

Congestive Heart Failure in Children: Evaluation

- Medical history
- Physical examination

LABORATORY WORK-UP

- Chest X-ray – Usually with cardiomegaly and increased pulmonary vascular markings (pulmonary congestion or edema)
- ECG- usually with sinus tachycardia; can suggest possible causes (What findings in the ECG can suggest the possible etiology of heart failure?)
- Echocardiography – usually shows cardiac pathology or the absence of it

OTHER TESTS to help in the Management and Diagnosis (How will these help?)

- ABG
- CBC; RBS ; electrolytes
- Inflammatory markers, and Cardiac enzymes as necessary (If the Troponin T is elevated, what is the possible etiology of CHF?)

BIOMARKER OF HEART FAILURE

Natriuretic peptides: BNP/NT-proBNP

- elevated as a result of ventricular dilation
- useful in distinguishing patients with congestive heart failure from those with a primary respiratory process.
- BNP levels of more than **100 pg/mL** are associated with congestive heart failure in adults and children. Normal levels may be slightly higher in neonates.

Causes of Heart Failure : Onset At Birth (do not depend on pulmonary pressure, but on a **markedly decreased cardiac output**)

Structural abnormalities

- Hypoplastic left heart syndrome
- Volume overload lesions
 - Tricuspid regurgitation
 - Pulmonary regurgitation
 - Systemic arteriovenous fistula

Heart rate abnormalities

- Supraventricular tachycardia
- Congenital complete heart block

Myocarditis

Causes of CHF : onset at 1st Week of Life (markedly decreased cardiac output and may be dependent on pulmonary pressure for shunting; can be a PDA-dependent lesion)
(Question: What is a PDA-dependent congenital heart disease?)

Structural abnormalities

- **Hypoplastic left heart syndrome**
- **TAPVR (w/ obstruction)**
- **Critical AS, Coarctation of aorta, Interrupted aortic arch**
- **Critical PS**
- **TGA**
- **PDA (premature infants)**

Causes of CHF : 1st 2 Months of Life

Structural abnormalities

LEFT TO RIGHT SHUNTS

- Atrial level shunt (e.g. TAPVR)
- **Ventricular level shunt (e.g. VSD)**
- **Aortic level shunt (e.g. PDA)**
- Left sided obstructive lesions (as above)
- Others: Anomalous origin of LCA from PA

6-month old infant
had symptoms at 2
months of age

When the pulmonary pressure decreases to adult level around 6-8 weeks of life, an increase in L to R shunting takes place, causing the onset, if not increase, in heart failure signs and symptoms.

Causes of CHF : 1st 2 Months of Life

Heart muscle abnormalities

- Cardiomyopathy/ EFE
- Myocarditis
- Pompe's disease

Causes of CHF: **Older** Children

- ***Acquired heart diseases***
- rheumatic fever & heart disease (5-15 YO) (**How old is our 2nd patient?**); myocarditis ; cardiomyopathy; pericarditis ; cor pulmonale; infective endocarditis
- **Congenital heart disease**
- unoperated end stage; pulmonary hypertension ; post-operative heart failure
- **Others:**
- dysrhythmias ; systemic hypertension ; anemia

MANAGEMENT OF HEART FAILURE IN CHILDREN

Regardless of underlying cause, TREATMENT of CHF remains grounded on the same principles.

Treatment Goals

1. ELIMINATE the cause, if possible
 - Surgical or cardiac catheterization intervention
 - Cardiac transplantation
2. OPTIMIZE Cardiac Output
 - Decrease Preload (because of volume overload)
 - Augment Contractility, especially in acute heart failure
 - Decrease Afterload (to decrease the workload of the heart while maintaining adequate organ perfusion and good blood pressure)
3. ENHANCE Oxygen Delivery
4. ALIGN metabolic demands of the body
5. PREVENT Left ventricular remodelling and deterioration of cardiac function

TREATMENT

❖ NON-PHARMACOLOGIC

❖ PHARMACOLOGIC

TREATMENT: NON-PHARMACOLOGIC

Decrease metabolic demands of the body

- Thermoregulation
- Eradicate infection
- Bed rest
- Appropriate fluid and electrolyte therapy
- Nutritional support
- High back rest (cardiac chair)

HEART FAILURE: Pharmacologic Treatment

1. Decrease Preload

- DIURETICS
 - **Furosemide**
(usually at 1-2 mg/kg/day Once a day to every 6 hours)
 - Spironolactone
- Venous vasodilator like nitroglycerin (also an arterial vasodilator)

2. Decrease Afterload

- ACE INHIBITORS (Captopril, Enalapril)
- Milrinone (both inotropic and vasodilatory, for acute heart failure)
- Arterial vasodilator like nitroglycerin (also a venous vasodilator)

HEART FAILURE: Pharmacologic Treatment

3. Augment contractility if necessary (severe heart failure)

- BETA-AGONIST
 - Dobutamine
 - Dopamine
 - Epinephrine
 - Milrinone (both inotropic and vasodilatory, for acute heart failure)
 - DIGOXIN / LANOXIN
-
- ❖ Decrease catecholamine/sympathetic effects – Chronic, stable, heart failure
 - ❑ BETA BLOCKERS (Carvedilol)

HEART FAILURE: Pharmacologic Treatment

4. Prevent progressive cell death and structural deterioration

- ACE INHIBITORS/ARBs, BETA

BLOCKERS, ALDOSTERONE ANTAGONIST

Question: What are three (3) neurohormones/hormones that promote LV remodeling that can lead to further deterioration of ventricular function?

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God is around us, above us, beneath us and within us.

He is with us.

But we are not conscious of Him for our nature is different.

But as we **accept the grace of God**
through faith in Christ Jesus our Savior and Lord,
who became man and died on the cross to **atone** for our sins,
we become conscious of God
and rejoice in **His presence** and **love**.

(Insights from The Attributes of God by AW Tozer)

*YOU
ARE
LOVED...*

1 John 4:10