HEART FAILURE

By:

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6th stage
Heart Failure :
Definition:

HF results when cardiac output is insufficient to meet the metabolic demands of the body.

Over time, decreased cardiac output leads to a cascade of compensatory responses that are aimed directly or indirectly at restoring normal perfusion to the body’s organs and tissues.
Prevalence:

The overall incidence and prevalence of pediatric HF is unknown, largely because there is:

1. No accepted universal classification applied to its many forms.
2. The largest HF burden comes from children born with congenital malformations. It has been estimated that 15% to 25% of children who have structural heart disease develop HF.
3. Although cardiomyopathy is relatively rare, approximately 40% of patients who experience cardiomyopathy develop heart failure of such severity that it leads to transplantation or death.
Pathophysiology:

Unmet tissue demands for cardiac output result in:

1. Activation of the renin-aldosterone angiotensin System.

2. The sympathetic nervous system, Stimulation of the sympathetic nervous system and increased release of catecholamines cause tachycardia, enhanced myocardial contractility

3. Cytokine-induced inflammation
Although decreased cardiac output stimulates deleterious neuroendocrine mechanisms, endogenous mechanisms defend the heart from progressive HF.

- These mechanisms include stimulation of insulin-like growth factor and growth hormone and increased concentrations appear to be protective, growth hormone deficiency and low insulin-like growth factor concentrations have been associated with poor HF outcomes in adults.

- Secretion of atrial and brain natriuretic peptides (ANP and BNP). For example, ANP and BNP are hormones secreted by the heart in response to volume and pressure overload that increase vasodilation and diuresis acutely and chronically prevent inflammation, cardiac fibrosis, and hypertrophy.
\[ \downarrow \text{Cardiac Output} \]

- ↑Sympathetic tone
- ↑Angiotensin
- ↑Mineralocorticoid
- ↑Inflammation/Cachexia

Cardiac Remodeling

- ↑ANP/BNP
- ↑IGF-1
- ↑GH
Signs and Symptoms:
Clinical Manifestations:

- In neonates, the earliest clinical manifestations may be subtle.

- Most commonly, infants have feeding difficulties due to dyspnea, increased fatigability, and secretion of anorexic hormones that limit the volume of feedings. Ultimately, affected babies fail to thrive.

- Physical findings in infants who have HF include mild-to-severe retractions, tachypnea or dyspnea with grunting (a form of positive end-expiratory pressure), tachycardia, a gallop rhythm (S3, S4), and hepatomegaly.
Signs and Symptoms:

- Shortness of breath
- Swelling of feet & legs
- Chronic lack of energy
- Difficulty sleeping at night due to breathing problems
- Swollen or tender abdomen with loss of appetite
- Cough with frothy sputum
- Increased urination at night
- Confusion and/or impaired memory
Modified Ross Heart Failure Classification for Children

- **Class I**: Asymptomatic

- **Class II**: Mild tachypnea or diaphoresis with feeding in infants. Dyspnea on exertion in older children.

- **Class III**: Marked tachypnea or diaphoresis with feeding in infants. Marked dyspnea on exertion. Prolonged feeding times with growth failure.

- **Class IV**: Symptoms such as tachypnea, retractions, grunting, or diaphoresis at rest.
Causes
Common Causes of HF:

Heart failure can result from cardiac and noncardiac causes.

Cardiac causes include those associated with
1. congenital
2. structural malformations

(Table 1) and those involving no structural anomalies (Table 2)
Table 1. Cardiac Malformations Leading to Heart Failure

Shunt Lesions

- Ventricular septal defect
- Patent ductus arteriosus
- Aortopulmonary window
- Atrioventricular septal defect
- Single ventricle without pulmonary stenosis
- Atrial septal defect (rare)

Total/Partial Anomalous Pulmonary Venous Connection

Valvular Regurgitation

- Mitral regurgitation
- Aortic regurgitation

Inflow Obstruction

- Cor triatriatum
- Pulmonary vein stenosis
- Mitral stenosis

Outflow Obstruction

- Aortic valve stenosis/subaortic stenosis/supravalvular

- aortic stenosis
- Aortic coarctation
Table 2. Sources of Heart Failure With a Structurally Normal Heart

<table>
<thead>
<tr>
<th>Primary Cardiac</th>
<th>Noncardiac</th>
</tr>
</thead>
<tbody>
<tr>
<td>● Cardiomyopathy</td>
<td>● Anemia</td>
</tr>
<tr>
<td>● Myocarditis</td>
<td>● Sepsis</td>
</tr>
<tr>
<td>● Myocardial infarction</td>
<td>● Hypoglycemia</td>
</tr>
<tr>
<td>● Acquired valve disorders</td>
<td>● Diabetic ketoacidosis</td>
</tr>
<tr>
<td>● Hypertension</td>
<td>● Hypothyroidism</td>
</tr>
<tr>
<td>● Kawasaki syndrome</td>
<td>● Other endocrinopathies</td>
</tr>
<tr>
<td>● Arrhythmia (bradycardia or tachycardia)</td>
<td>● Arteriovenous fistula</td>
</tr>
<tr>
<td></td>
<td>● Renal failure</td>
</tr>
<tr>
<td></td>
<td>● Muscular dystrophies</td>
</tr>
</tbody>
</table>
Cardiac Malformations Associated With Excessive Preload (Volume Loading)

Among cardiac malformations, those that cause left-to-right systolic shunts at the ventricular level are associated most commonly with HF. In this situation, a volume load on the left side of the heart causes preload stress.

The combined cardiac output is increased due to the volume of “ineffective” pulmonary blood flow that recirculates through the lungs.
Cardiac Malformations Associated With Excessive Afterload

Left heart obstructive lesions such as mitral stenosis (rare), aortic stenosis, and coarctation of the aorta (common) induce acute HF or lethal arrhythmias when they cause severe afterload stress.
Disorders of Contractility:

1. Cardiomyopathy (dilated, hypertrophic, constrictive, or restrictive)

2. Dilated cardiomyopathy is characterized by enlarged ventricular chambers and impaired systolic and diastolic function.

3. Restrictive cardiomyopathy often is idiopathic but can be caused by infiltrative or storage diseases.

4. Hypertrophic cardiomyopathy, such as idiopathic hypertrophic subaortic stenosis, seldom is associated with pediatric HF.
Arrhythmias Associated With Pediatric HF

Arrhythmias cause HF when the heart rate is too fast or too slow to meet tissue metabolic demands.

During tachycardia-related diseases, diastolic filling time shortens to the point that cardiac output is decreased.

With chronic bradycardias, the left ventricle enlarges to accommodate larger stroke volumes.
Investigation:
Investigation:

- Pulse oximetry is helpful in identifying cyanosis in infants who have HF caused by increased pulmonary blood flow (left-to-right shunts).

- The 12-lead electrocardiogram may be helpful in assessing the cause of heart failure but does not establish the diagnosis so ECG is essential to assess arrhythmia induced HF.

- The chest radiograph may demonstrate cardiac enlargement, increased pulmonary blood flow, venous congestion, or pulmonary edema.

- Echocardiography is essential for identifying causes of HF such as structural heart disease, ventricular dysfunction.
X- Ray:
Echo:

Normal Patient  Patient with Amyloid Deposits in Heart
HF Biomarkers:

HF biomarkers have been identified that aid in assessing the severity of HF and predicting the course of the disease.

- BNP measurement is a readily available test that can distinguish between primary respiratory disease and cardiac-induced tachypnea.

- C-reactive protein TNF-alpha are both sensitive markers of systemic inflammation that correlate positively with a worse HF outcome in adult studies.
Serum B-type natriuretic peptide (BNP) :

- A cardiac neurohormone released in response to increased ventricular wall tension, is elevated in adult patients with congestive heart failure.

- In children, BNP may be elevated in patients with heart failure due to systolic dysfunction (cardiomyopathy) as well as in children with volume overload (left-to-right shunts such as ventricular septal defect).
Treatment:
TREATMENT:

The underlying cause of cardiac failure must be removed or alleviated if possible.

- General Measures
- Diet
- Digitalis
- Diuretics
- Afterload-Reducing Agents
- ACE Inhibitors
- α- and β-Adrenergic Agonists
- Phosphodiesterase Inhibitors
- Chronic Treatment with β-Blockers
General Measures:

Strict bed rest is rarely necessary except in extreme cases.

Some older patients feel better sleeping in a semi-upright position, using several pillows (orthopnea).

For infants with heart failure, an infant chair may be advisable.
Diet:

- Infants with heart failure may fail to thrive because of increased metabolic requirements and decreased caloric intake.

- Severely ill infants may lack sufficient strength for effective sucking because of extreme fatigue, rapid respirations, and generalized weakness so nasogastric feedings may be helpful.

- The use of low sodium formulas in the routine management of infants with heart failure is not recommended because these preparations are often poorly tolerated and may exacerbate diuretic-induced hyponatremia.
Digitalis:

✓ Digoxin, once the mainstay of heart failure management in both children and adults

✓ Digoxin should be discontinued if a new rhythm disturbance is noted

✓ Prolongation of the P-R interval is not necessarily an indication to withhold digitalis, but a delay in administering the next dose or a reduction in the dosage should be considered
Digoxin Toxicity:

- Serum digoxin determination is helpful when digitalis toxicity is suspected, although it may be less reliable in infants.

- ST segment or T-wave changes are commonly noted with digitalis administration and should not affect the digitalization regimen.

- Hypokalemia and hypercalcemia exacerbate digitalis toxicity. Because hypokalemia is relatively common in patients receiving diuretics, potassium levels should be monitored closely in those receiving a potassium-wasting diuretic (e.g., furosemide) in combination with digitalis.
Diuretics:

A. Furosemide is the most commonly used diuretic in patients with heart failure. It inhibits the reabsorption of sodium and chloride in the distal tubules and the loop of Henle.

B. Spironolactone is an inhibitor of aldosterone and enhances potassium retention, often eliminating the need for oral potassium supplementation, which is frequently poorly tolerated.
Afterload-Reducing Agents and ACE Inhibitors:

- This group of drugs reduces ventricular afterload by decreasing peripheral vascular resistance and thereby improving myocardial performance. They are most often used in conjunction with other anticongestive drugs such as digoxin and diuretics.

- Blood pressure must be continuously monitored because sudden hypotension can occur.
α- and β-Adrenergic Agonists:

- Dopamine is a predominantly β-adrenergic receptor agonist, but it has α-adrenergic effects at higher doses. Dopamine results in increased contractility with little peripheral vasoconstrictive effect.

- Dobutamine, a derivative of dopamine, is useful in treating low cardiac output. It causes direct inotropic effects with a moderate reduction in peripheral vascular resistance.

- Dobutamine can be used as an adjunct to dopamine therapy to avoid the vasoconstrictive effects of high-dose dopamine.

- Epinephrine is a mixed α- and β-adrenergic receptor agonist that is usually reserved for patients with cardiogenic shock and low arterial blood pressure.
Phosphodiesterase Inhibitors:

- Milrinone is useful in treating patients with low cardiac output who are refractory to standard therapy and has been shown to be highly effective in managing low-output state in children after open heart surgery.

- Milrinone has both positive inotropic effects on the heart and significant peripheral vasodilatory effects.
Chronic Treatment with β-Blockers:

The agents most often used are:

A. metoprolol, a $\beta_1$-adrenergic receptor selective antagonist

B. carvedilol, an agent with both $\alpha$- and $\beta$-adrenergic receptor blocking β blockers

both used for the chronic treatment of patients with heart failure and should not be administered when patients are still in the acute phase of heart failure
Prognosis:
Prognosis:

- The outcome for patients experiencing HF depends largely on its cause.

- When noncardiac disorders are responsible, the improvement in HF is related to successful treatment of the systemic disease.

- For many cardiac malformations (preloading and afterloading conditions), surgical correction can be curative.
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Learn and Live

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Dedicated to the health of all children

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