

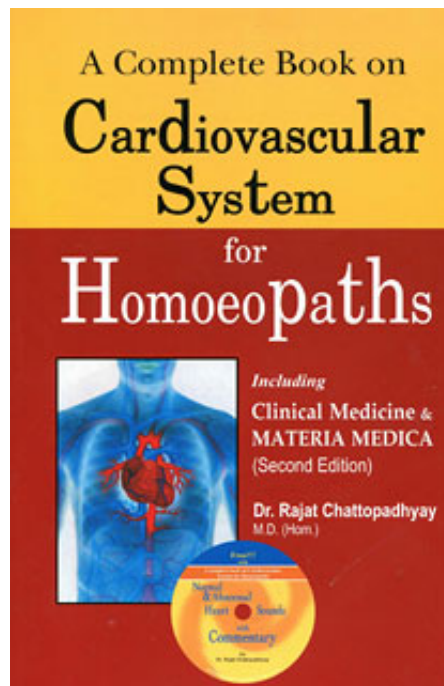
Dr. R. Chattopadhyay  
A Complete Book on Cardiovascular System for  
Homoeopaths

Reading excerpt

[A Complete Book on Cardiovascular System for Homoeopaths](#)

of [Dr. R. Chattopadhyay](#)

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# Cardiac Failure

Cardiac failure or heart failure is an imprecise term used to describe the state that develops when the heart cannot maintain an adequate cardiac output or can do so only at the expense of an elevated filling pressure.

Almost all forms of heart disease may lead to heart failure and it is important to appreciate that, like anaemia the term refers to a clinical syndrome rather than a specific diagnosis.

## **Aetiology**

*Myocardial Dysfunction:* A kinetic or Dyskinetic segments contract poorly and may impede the function of the normal segment by distorting their contraction and relaxation patterns.

*Example:*

Myocarditis/cardiac myopathy.

Myocardial infarction.

- *Ventricular Volume Overload:* As a compensatory mechanism dilatation and hypertrophy allow the ventricle to generate a high stroke volume, but secondary changes in the myocardium eventually lead to impaired contractibility and worsening heart failure.

*Example:*

Mitral regurgitation.

Aortic regurgitation.

Atrial septal defect.

Ventricular septal defect.

- *Ventricular Outflow Obstruction:* Though initially ventricular hypertrophy allow the ventricle to maintain normal output, but increased obstruction lead to failure.

*Example:*

Systemic hypertension. Aortic Stenosis. Pulmonary hypertension. Pulmonary valve stenosis. Chronic lung diseases (C.O.P.D.)

- *Compromised Ventricular Filling:* Due to less inflow of blood into the ventricle, it pumps vigorously to maintain the normal output.

*Example:*

Mitral stenosis. Tricuspid stenosis. Constrictive pericarditis. Pericardial tamponade. Restrictive cardiomyopathy.

- *Arrhythmias:* They exert a deleterious effect for a variety of reasons. Either ventricle gets less time for filling or there is reduced output because of ventricular bradycardia or dissociation between atrial and ventricular contractions results in the loss of atrial booster pump mechanism.

### **Pathophysiology**

When the heart fails, a number of adaptations occur both in the heart and systemically.

#### ***Local changes***

- **Chamber enlargement:** If the stroke volume of either ventricle is reduced by depressed contractility or excessive afterload, end-diastolic volume and pressure in that chamber will rise. If the condition is chronic, ventricular dilatation will occur.
- **Myocardial hypertrophy:** Increased activity of heart muscles to maintain the stroke volume causes muscular hypertrophy.

- **Increased heart rate:** Reduced cardiac output if associated with reduced arterial pressure or perfusion of kidneys, will activate sympathetic nervous systems which will increase the heart rate remarkably.

### *Systemic changes*

- **Activation of sympathetic nervous system:** Reduced cardiac output increases sympathetic activity which in turn stimulate myocardial contractility, heart rate and venous tone. It also increases peripheral vascular resistance to maintain perfusion to vital organs, but when it is excessive it may itself reduce renal and other tissue blood flow.
- **Action of the Renin-angiotensin—Aldosterone system:** Lower cardiac output also reduces the renal blood flow and glomerular filtration rate. It activate the renin - angiotensin-aldosterone system, leading to further increase in peripheral vascular resistance and left ventricular after load.
- **Release or antidiuretic hormones:** Heart failure is associated with increased circulation levels of arginine-vasopressin, which also serves as a vasoconstrictor and inhibitor of water excretion.
- **Release of atrial natriuretic peptide:** Release of atrial natriuretic peptide is increased in heart failure owing to the elevated atrial pressures, but there is evidence of resistance to its natriuretic and vasodilating effects.

### Clinical features

## LEFT HEART FAILURE

### *Symptoms*

- Languor & lassitude.
- Flashes of light before eyes.
- Dizziness especially on suddenly assuming an erect position.
- Tendency to syncope.
- Shortness of breath, chiefly exertional dyspnoea at first, then progressing to orthopnoea , paroxysmal nocturnal dyspnoea and rest dyspnoea.
- Chronic non- productive cough often worse in recumbent position.
- Haemoptysis may be present at later stage.

### *Signs*

- Cyanosis is present and central in type.
- Tachycardia with pulsus alternans are usually present.

- On palpation apex beat is shifted laterally and downwards.
- Apex beat is heaving.
- Short accentuated first sounds often with tic-tac rhythm.
- S<sub>3</sub> & S<sub>4</sub> may be audible giving rise to gallop rhythm.
- Murmur may be present in mitral and aortic area if valvular disease for heart failure is present.
- Basal crepitations in the lungs are present.
- Later on bilateral coarse moist sounds all over chest develop due to pulmonary oedema.

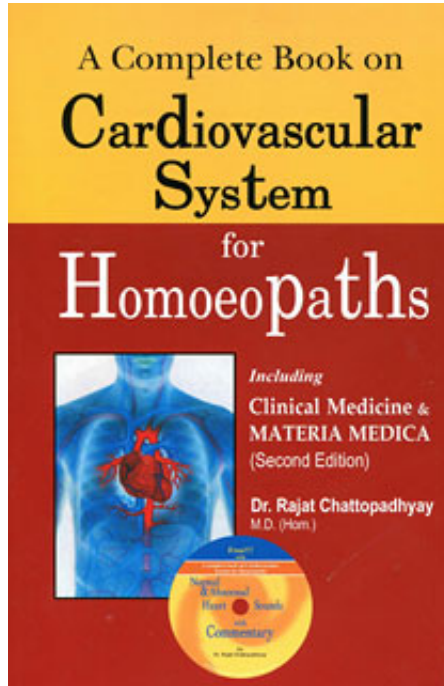
### **RIGHT HEART FAILURE**

#### *Symptoms*

- Fatigue & exhaustion.
- Pain in the right hypochondria due to passive congestion of liver.
- Anorexia and nausea due to oedema of the gut or impaired gastrointestinal perfusion.
- Oliguria is present; sometimes nocturia due to reabsorption of oedema fluid during night's rest in recumbent position.

#### *Signs*

- Cyanosis is present which is partly central and partly peripheral.
- Oedema on dependent parts of the body and is pitting in type. Afterwards generalized oedema with collection of fluids in the serous cavities.
- Apex of heart is displaced more laterally than downwards.
- Pulse is rapid, feeble and may be irregular.
- Often signs of tricuspid regurgitation.
- Gallop rhythm may be distinctly heard over the left lower sternal region or epigastric region.
- Jugular venous distension with or without V waves of tricuspid regurgitation.
- There may be evidences of emphysema or fibrosis of lungs as a causal factors.
- Tender, smooth hepatic enlargement is present.
- Ascites may be seen in some cases.



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