

Heart Failure [372–456]

1. Introduction

- Heart failure is not a specific disease entity, rather it is a clinical syndrome characterised by a number of clinical signs, symptoms and diagnostic findings. It occurs as a consequence of diastolic or systolic dysfunction resulting in the inability of the heart to provide adequate cardiac output for the body's metabolic needs.
- Acute Heart Failure (AHF) represents new or worsening signs or symptoms consistent with an underlying deterioration in ventricular function. It is characterised by dyspnoea resulting from acutely elevated cardiac filling pressures often, though not always, leading to rapid accumulation of fluid within the lung's interstitial and alveolar spaces (cardiogenic pulmonary oedema).

2. Incidence

- The overall prevalence of heart failure varies between 1 and 2%; it is the leading reason for hospital admission in patients over 65 years of age in westernised countries. Of heart failure cases, 80% are diagnosed following admission to hospital as an acute emergency.

3. Severity and Outcome

- The 60-day mortality following hospital admission because of an exacerbation of heart failure varies with estimates between 8–20%. Approximately 30% of heart failure patients will be re-admitted to hospital each year as a result of an acute exacerbation.
- The overall prehospital mortality rate for cases of acute cardiogenic pulmonary oedema has been reported at 8%.

4. Pathophysiology

- The pathophysiology underlying the worsening ventricular function is often coronary artery disease. Diseased coronary arteries become inelastic and intravascular pressures rise as a consequence – coronary artery perfusion will reduce unless mean arterial pressures are raised to ensure adequate coronary artery blood flow.
- As mean arterial pressures rise so too does cardiac afterload that will lead to an increased end diastolic volume unless the force of ventricular contraction is raised. These combined processes initiate a spiral of increasing mean arterial pressures, increasing afterload and increasing preload.

Risk Factors

- Advancing age
- History of CHF
- Hypertension

- Acute Heart Failure patients will often present with clinical signs of vascular congestion and the formation of oedema. The pathophysiology underlying the formation of oedema can vary depending on cause; however in Acute Heart Failure acute oedema occurs as a result of fluid shifts from the vascular compartment into the interstitial space.
- Left Ventricular Failure (LVF) may precipitate formation of pulmonary oedema as a result of reduced cardiac

output and increasing pulmonary hypertension. As pulmonary vascular pressures rise, so increasing amounts of fluid will shift from the pulmonary vascular compartment into the lung interstitial spaces and alveoli. Pulmonary oedema occurs as a result of the accumulation of this fluid in the alveoli which decreases gas exchange across the alveoli, resulting in decreased oxygenation of the blood and, in some cases, accumulation of carbon dioxide (CO_2).

- The pathophysiology of pulmonary oedema can be thought of in terms of three factors (Table 3.62):
 - i. flow
 - ii. fluid
 - iii. filter.

Table 3.62 – PULMONARY OEDEMA

i. Flow

The ability of the heart to eject the blood delivered to it depends on three factors:

1. the amount of blood returning to the heart (preload)
2. the co-ordinated contraction of the myocardium
3. the resistance against which it pumps (afterload).

Preload may also be increased by over-infusion of IV fluid or fluid retention. Coordinated contraction fails following heart muscle damage (Myocardial Infarction (MI), heart failure) or due to arrhythmias. Afterload increases with hypertension, atherosclerosis, aortic valve stenosis or peripheral vasoconstriction.

ii. Fluid

The blood passing through the lungs must have enough 'oncotic' pressure to 'hold on' to the fluid portion as it passes through the pulmonary capillaries. As albumin is a key determinant of oncotic pressure, low albumin states can also lead to the formation of pulmonary oedema, e.g. burns, liver failure, nephrotic syndrome.

iii. Filter

The capillaries through which the fluid passes may increase in permeability, e.g. acute lung injury (as in smoke inhalation), pneumonia or drowning.

- Right Ventricular Failure (RVF) may precipitate formation of peripheral oedema as a result of increasing right ventricular filling pressures, reduced right ventricular output and increasing systemic congestion. Peripheral oedema occurs as a result of the accumulation of fluid in the interstitial spaces and is most commonly noted in the lower legs and sacrum.
- The signs and symptoms of heart failure vary depending upon the extent of failure and underlying physiologic cause. However, three symptoms are common to nearly all forms: fatigue (including exercise intolerance), dyspnoea and congestion. It can be difficult to differentiate heart failure from other causes of breathlessness, such as exacerbation of Chronic Obstructive Pulmonary Disease (COPD), pulmonary embolism or pneumonia. Therefore, a thorough history and physical examination are required.

5. Assessment and Management

For the assessment and management of heart failure refer to Table 3.63.