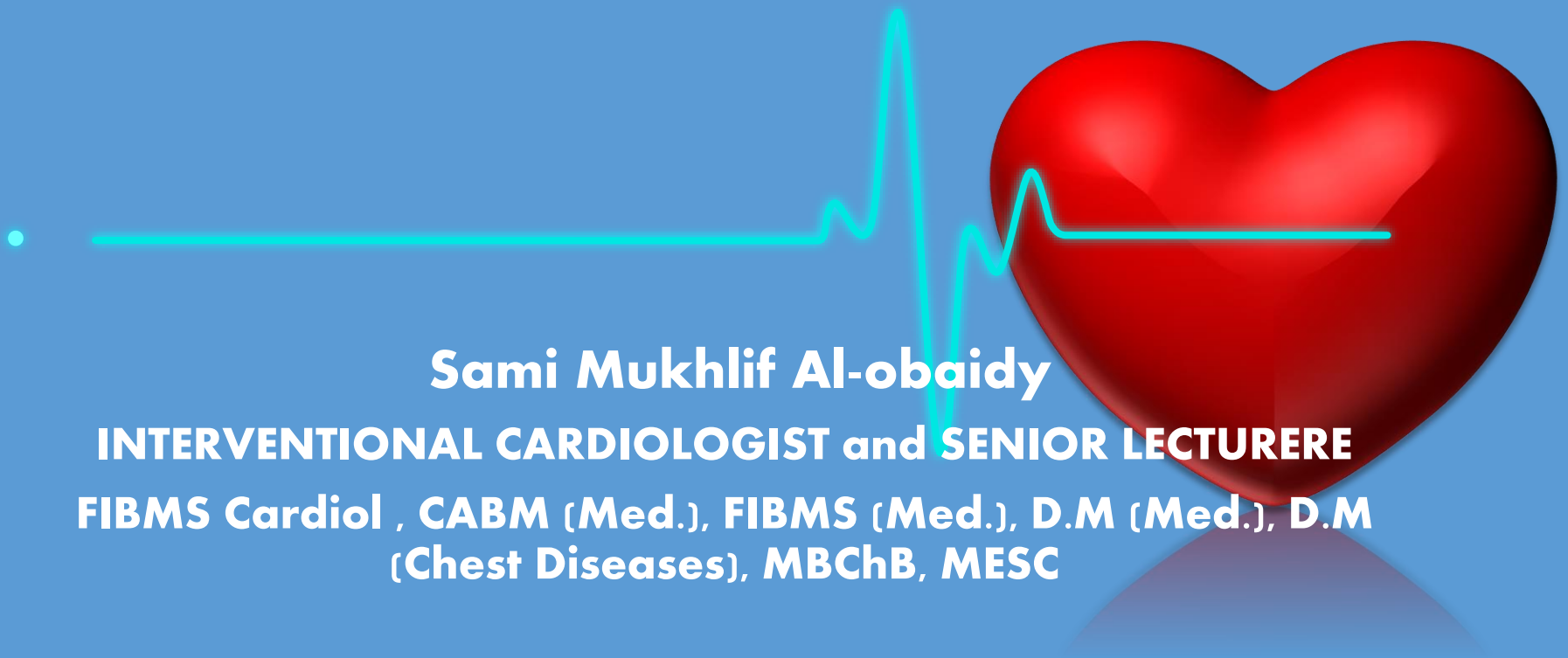
HEART FAILURE



Sami Mukhlif Al-obaidy

INTERVENTIONAL CARDIOLOGIST and SENIOR LECTURERE

FIBMS Cardiol , CABM (Med.), FIBMS (Med.), D.M (Med.), D.M (Chest Diseases), MBChB, MESC

Heart failure describes the clinical syndrome that develops when the heart cannot maintain adequate output, or can do so only at the expense of elevated ventricular filling pressure.

In mild to moderate forms of heart failure, symptoms occur only when the metabolic demand increases during exercise or some other form of stress.

In severe heart failure, symptoms may be present at rest. In clinical practice, heart failure may be diagnosed when a patient with significant heart disease develops the signs or symptoms of a low cardiac output, pulmonary congestion or systemic venous congestion at rest or on exercise.

TYPES OF HEART FAILURE

Left heart failure

This is characterised by a reduction in left ventricular output and an increase in left atrial and pulmonary venous pressure. If left heart failure occurs suddenly – for example, as the result of an acute MI – the rapid increase in left atrial pressure causes pulmonary oedema.

If the rise in atrial pressure is more gradual, as occurs with mitral stenosis, there is reflex pulmonary vasoconstriction, which protects the patient from pulmonary oedema.

However, the resulting increase in pulmonary vascular resistance causes pulmonary hypertension, which in turn impairs right ventricular function.

Right heart failure

This is characterised by a reduction in right ventricular output and an increase in right atrial and systemic venous pressure. The most common causes are chronic lung disease, pulmonary embolism and pulmonary valvular stenosis. The term ‘cor pulmonale’ is used to describe right heart failure that is secondary to chronic lung disease.

Biventricular heart failure

In biventricular failure, both sides of the heart are affected. This may occur because the disease process, such as dilated cardiomyopathy or ischaemic heart disease, affects both ventricles or because disease of the left heart leads to chronic elevation of the left atrial pressure, pulmonary hypertension and right heart failure.

Pathogenesis

Heart failure occurs when cardiac output fails to meet the demands of the circulation. Cardiac output is determined by preload (the volume and pressure of blood in the ventricles at the end of diastole), afterload (the volume and pressure of blood in the ventricles during systole) and myocardial contractility, forming the basis of Starling's Law.

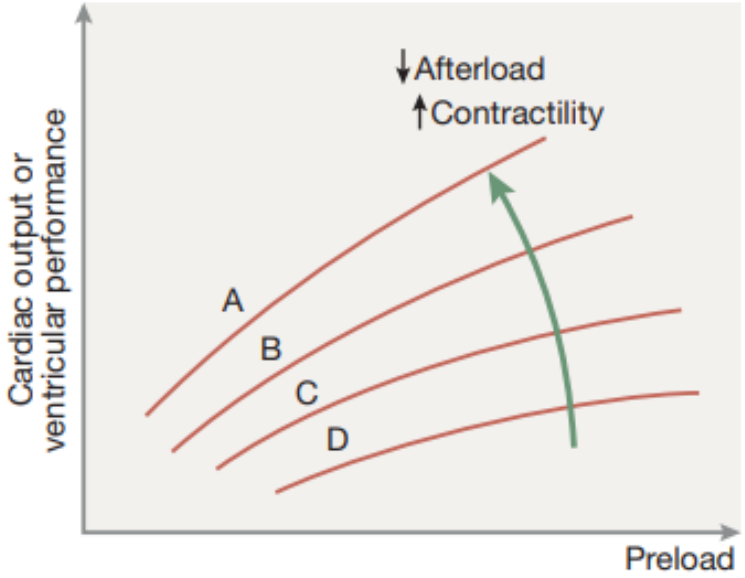


Fig. 16.24 Starling's Law. Normal (A), mild (B), moderate (C) and severe (D) heart failure. Ventricular performance is related to the degree of myocardial stretching. An increase in preload (end-diastolic volume, end-diastolic pressure, filling pressure or atrial pressure) will therefore enhance function; however, overstretching causes marked deterioration. In heart failure, the curve moves to the right and becomes flatter. An increase in myocardial contractility or a reduction in afterload will shift the curve upwards and to the left (green arrow).

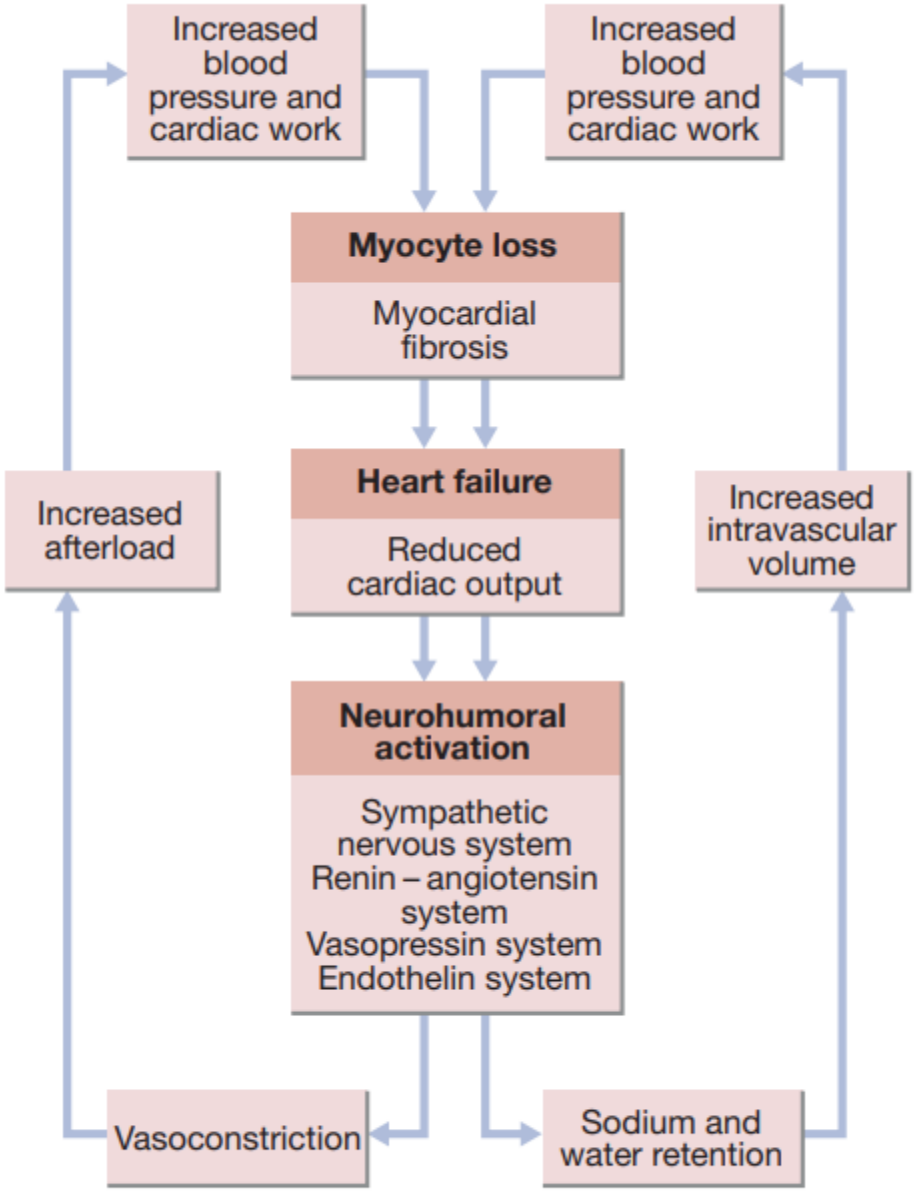


Fig. 16.25 Neurohumoral activation and compensatory mechanisms in heart failure. There is a vicious circle in progressive heart failure.

16.12 Mechanisms of heart failure

Cause	Examples	Features
Reduced ventricular contractility	Myocardial infarction (segmental dysfunction)	In coronary artery disease, 'akinetic' or 'dyskinetic' segments contract poorly and may impede the function of normal segments by distorting their contraction and relaxation patterns
	Myocarditis/cardiomyopathy (global dysfunction)	Progressive ventricular dilatation
Ventricular outflow obstruction (pressure overload)	Hypertension, aortic stenosis (left heart failure) Pulmonary hypertension, pulmonary valve stenosis (right heart failure)	Initially, concentric ventricular hypertrophy allows the ventricle to maintain a normal output by generating a high systolic pressure. Later, secondary changes in the myocardium and increasing obstruction lead to failure with ventricular dilatation and rapid clinical deterioration
Ventricular inflow obstruction	Mitral stenosis, tricuspid stenosis	Small, vigorous ventricle; dilated, hypertrophied atrium. Atrial fibrillation is common and often causes marked deterioration because ventricular filling depends heavily on atrial contraction
Ventricular volume overload	Left ventricular volume overload (mitral or aortic regurgitation) Ventricular septal defect Right ventricular volume overload (atrial septal defect) Increased metabolic demand (high output)	Dilatation and hypertrophy allow the ventricle to generate a high stroke volume and help to maintain a normal cardiac output. However, secondary changes in the myocardium lead to impaired contractility and worsening heart failure
Arrhythmia	Atrial fibrillation	Tachycardia does not allow for adequate filling of the heart, resulting in reduced cardiac output and back pressure
	Tachycardia	Prolonged tachycardia causes myocardial fatigue
	Complete heart block	Bradycardia limits cardiac output, even if stroke volume is normal
Diastolic dysfunction	Constrictive pericarditis	Marked fluid retention and peripheral oedema, ascites, pleural effusions and elevated jugular veins
	Restrictive cardiomyopathy	Bi-atrial enlargement (restrictive filling pattern and high atrial pressures). Atrial fibrillation may cause deterioration
	Left ventricular hypertrophy and fibrosis	Good systolic function but poor diastolic filling
	Cardiac tamponade	Hypotension, elevated jugular veins, pulsus paradoxus, poor urine output

CLINICAL ASSESSMENT:

Heart failure may develop suddenly, as in MI, or gradually, as in valvular heart disease.

When there is gradual impairment of cardiac function, several compensatory changes take place.

The term compensated heart failure is sometimes used to describe the condition of those with impaired cardiac function, in whom adaptive changes have prevented the development of overt heart failure. However, a minor event, such as an intercurrent infection or development of atrial fibrillation, may precipitate acute heart failure in these circumstances.

Similarly, acute heart failure sometimes supervenes as the result of a decompensating episode, on a background of chronic heart failure; this is called acute-on-chronic heart failure.

16.13 Factors that may precipitate or aggravate heart failure in pre-existing heart disease

- Myocardial ischaemia or infarction
- Intercurrent illness
- Arrhythmia
- Inappropriate reduction of therapy
- Administration of a drug with negative inotropic (β-blocker) or fluid-retaining properties (non-steroidal anti-inflammatory drugs, glucocorticoids)
- Pulmonary embolism
- Conditions associated with increased metabolic demand (pregnancy, thyrotoxicosis, anaemia)
- Intravenous fluid overload

Acute left heart failure

Acute left heart failure presents with a sudden onset of dyspnoea at rest that rapidly progresses to acute respiratory distress, orthopnoea and prostration. Often there is a clear precipitating factor, such as an acute MI, which may be apparent from the history.

The patient appears agitated, pale and clammy. The peripheries are cool to the touch and the pulse is rapid, but in some cases there may be an inappropriate bradycardia that may contribute to the acute episode of heart failure.

The BP is usually high because of SNS activation, but may be normal or low if the patient is in cardiogenic shock.

The jugular venous pressure (JVP) is usually elevated, particularly with associated fluid overload or right heart failure.

In acute heart failure, there has been no time for ventricular dilatation and the apex is not displaced.

A ‘gallop’ rhythm, with a third heart sound, is heard quite early in the development of acute left-sided heart failure. A new systolic murmur may signify acute mitral regurgitation or ventricular septal rupture.

Chest examination may reveal crepitations at the lung bases if there is pulmonary oedema, or crepitations throughout the lungs if this is severe.

There may be an expiratory wheeze. Patients with acute-on-chronic heart failure may have additional features of chronic heart failure.

Chronic left heart failure

- Patients with chronic heart failure commonly follow a relapsing and remitting course, with periods of stability and episodes of decompensation, leading to worsening symptoms that may necessitate hospitalisation.
- The clinical picture depends on the nature of the underlying heart disease, the type of heart failure that it has evoked, and the changes in the SNS and RAAS that have developed.
- Low cardiac output causes fatigue and a poor effort tolerance; the peripheries are cold and the BP is low.
- To maintain perfusion of vital organs, blood flow is diverted away from skeletal muscle and this may contribute to fatigue and weakness. Poor renal perfusion leads to oliguria and uraemia.
- Pulmonary oedema due to left heart failure presents with dyspnoea and inspiratory crepitations over the lung bases.
- In contrast, right heart failure produces a high JVP with hepatic congestion and dependent peripheral oedema.
- In ambulant patients the oedema affects the ankles, whereas in bed-bound patients it collects around the thighs and sacrum. Ascites or pleural effusion may occur.
- Chronic heart failure is sometimes associated with marked weight loss (cardiac cachexia), caused by a combination of anorexia and impaired absorption due to gastrointestinal congestion, poor tissue perfusion due to a low cardiac output, and skeletal muscle atrophy due to immobility.

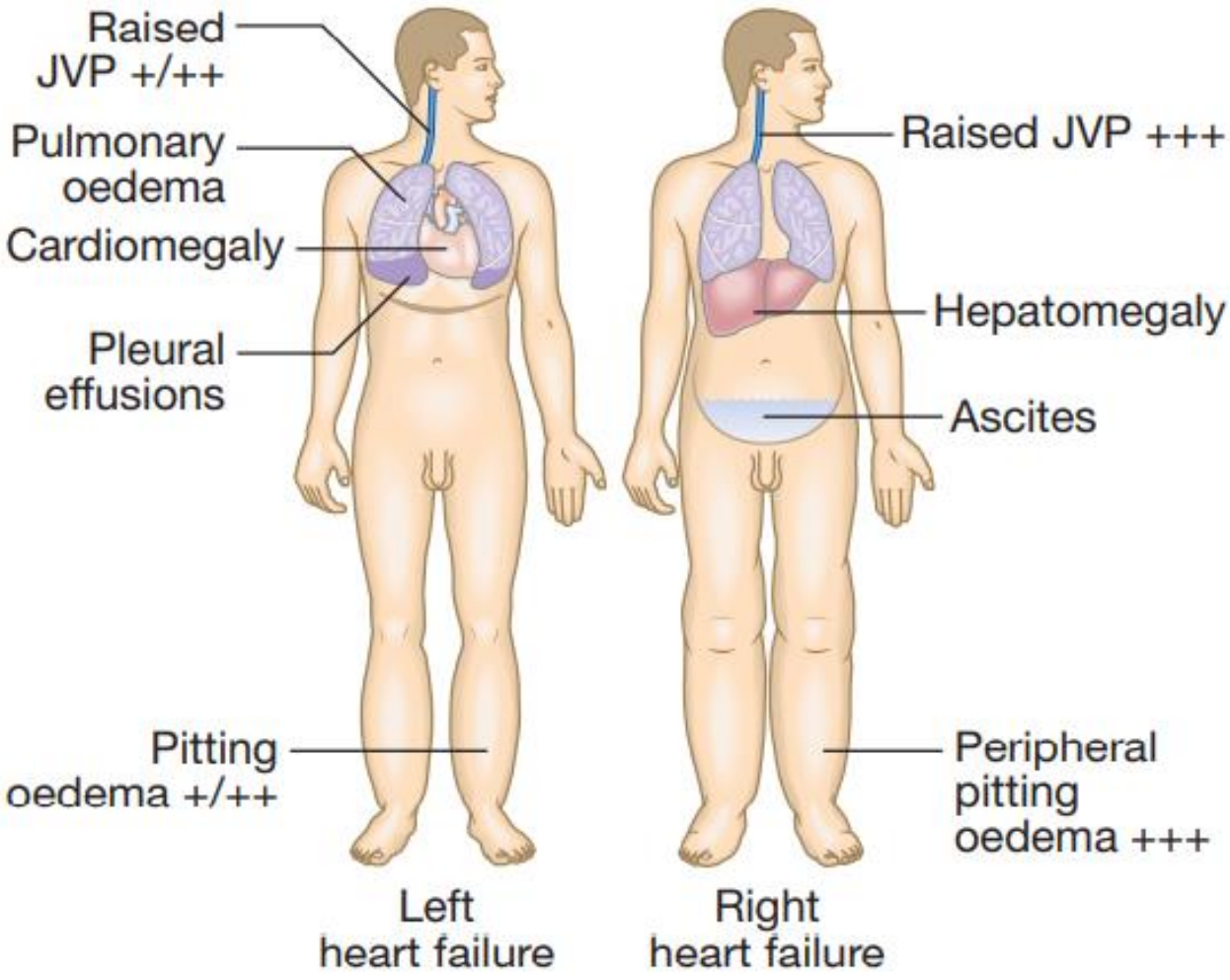


Fig. 16.26 **Clinical features of left and right heart failure.**
(JVP = jugular venous pressure)

Complications of Heart Failure

- Renal failure is caused by poor renal perfusion due to low cardiac output and may be exacerbated by diuretic therapy, ACE inhibitors and angiotensin receptor blockers (ARBs).
- Hypokalaemia may be the result of treatment with potassium-losing diuretics or hyperaldosteronism caused by activation of the RAS and impaired aldosterone metabolism due to hepatic congestion. Most of the body's potassium is intracellular and there may be substantial depletion of potassium stores, even when the plasma concentration is in the reference range.
- Hyperkalaemia may be due to the effects of drugs that promote renal resorption of potassium, in particular the combination of ACE inhibitors, ARBs and mineralocorticoid receptor antagonists.
- These effects are amplified if there is renal dysfunction due to low cardiac output or atherosclerotic renal vascular disease.
- Hyponatraemia is a feature of severe heart failure and is a poor prognostic sign. It may be caused by diuretic therapy, inappropriate water retention due to high vasopressin secretion, or failure of the cell membrane ion pump.
- Impaired liver function is caused by hepatic venous congestion and poor arterial perfusion, which frequently cause mild jaundice and abnormal liver function tests; reduced synthesis of clotting factors can make anticoagulant control difficult.
- Thromboembolism. Deep vein thrombosis and pulmonary embolism may occur due to the effects of a low cardiac output and enforced immobility. Systemic emboli occur in patients with atrial fibrillation or flutter, or with intracardiac thrombus complicating conditions such as mitral stenosis, MI or left ventricular aneurysm.
- Atrial and ventricular arrhythmias are very common and may be related to electrolyte changes such as hypokalaemia and hypomagnesaemia, the underlying cardiac disease, and the pro-arrhythmic effects of sympathetic activation. **Atrial fibrillation occurs in approximately 20% of patients** with heart failure and causes further impairment of cardiac function. Ventricular ectopic beats and runs of non-sustained ventricular tachycardia are common findings in patients with heart failure and are associated with an adverse prognosis.
- Sudden death occurs in up to 50% of patients with heart failure and is most probably due to ventricular fibrillation.

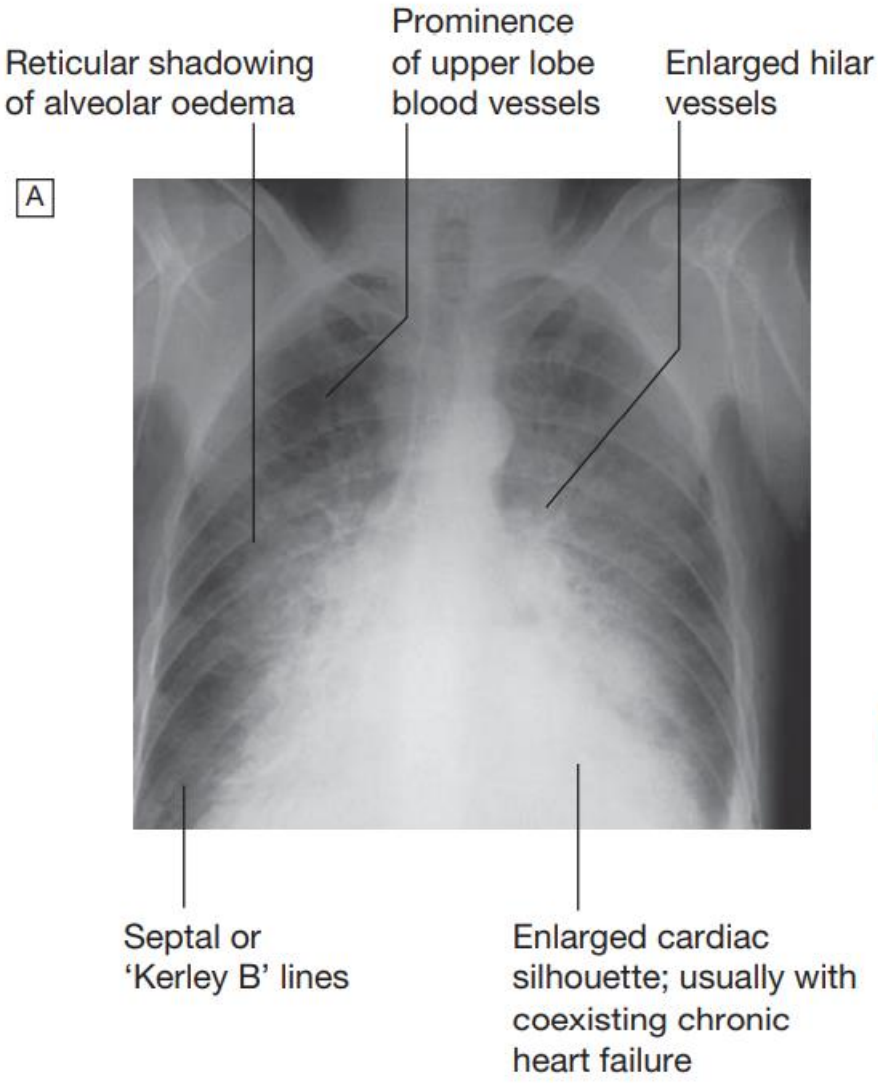
Investigations:

CXR:

should be performed in all cases.

This may show abnormal distension of the upper lobe pulmonary veins with the patient in the erect position.

Vascularity of the lung fields becomes more prominent and the right and left pulmonary arteries dilate. Subsequently, interstitial oedema causes thickened interlobular septa and dilated lymphatics. These are evident as horizontal lines in the costophrenic angles (septal or ‘Kerley B’ lines). More advanced changes due to alveolar oedema cause a hazy opacification spreading from the hilar regions, and pleural effusions.



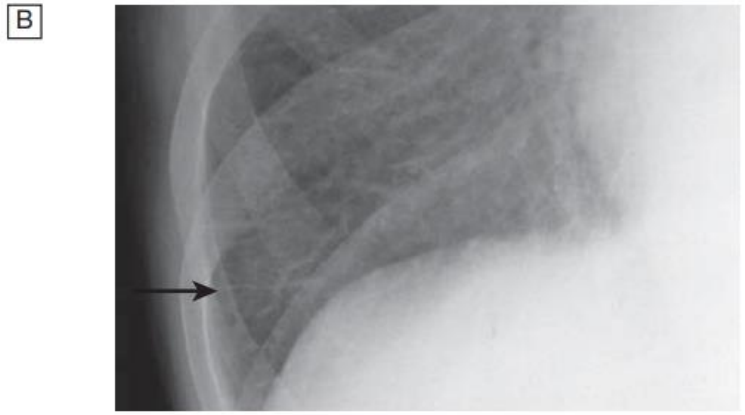


Fig. 16.27 Radiological features of heart failure. A Chest X-ray of a patient with pulmonary oedema. B Enlargement of lung base showing septal or 'Kerley B' lines (arrow).

Echocardiography

is very useful and should be considered in all patients with heart failure in order to:

- determine the **aetiology**
- detect hitherto unsuspected **valvular heart disease**, such as occult mitral stenosis, and other conditions that may be amenable to specific remedies
- identify patients who will benefit from long-term drug therapy.

Serum urea, creatinine and electrolytes, haemoglobin and thyroid function may help to establish the nature and severity of the underlying heart disease and detect any complications.

BNP is elevated in heart failure and is a prognostic marker, as well as being useful in differentiating heart failure from other causes of breathlessness or peripheral oedema.

Management of Acute Heart Failure:

Acute heart failure with pulmonary oedema is a medical emergency that should be treated urgently. The patient should initially be kept rested, with continuous monitoring of cardiac rhythm, BP and pulse oximetry.

Intravenous opiates can be of value in distressed patients but must be used sparingly, as they may cause respiratory depression and exacerbation of hypoxaemia and hypercapnia.

16.15 Management of acute pulmonary oedema

Action	Effect
Sit the patient up	Reduces preload
Give high-flow oxygen	Corrects hypoxia
Ensure continuous positive airway pressure (CPAP) of 5–10 mmHg by tight-fitting mask	Reduces preload and pulmonary capillary hydraulic gradient
Administer nitrates:* IV glyceryl trinitrate (10–200 μg/min) Buccal glyceryl trinitrate 2–5 mg	Reduces preload and afterload
Administer a loop diuretic: Furosemide (50–100 mg IV)	Combats fluid overload

*The dose of nitrate should be titrated upwards every 10 mins until there is an improvement or systolic blood pressure is <110 mmHg.

If these measures prove ineffective, inotropic agents such as dobutamine (2.5–10 μg/kg/min) may be required to augment cardiac output, particularly in hypotensive patients.

Insertion of an intra-aortic balloon pump may be beneficial in patients with acute cardiogenic pulmonary oedema and shock.

Following management of the acute episode, additional measures must be instituted to control heart failure in the longer term.

Management of Chronic Heart Failure

16.16 General measures for the management of heart failure

Education

- Explanation of nature of disease, treatment and self-help strategies

Diet

- Good general nutrition and weight reduction for the obese
- Avoidance of high-salt foods and added salt, especially for patients with severe congestive heart failure

Alcohol

- Moderation or elimination of alcohol consumption; alcohol-induced cardiomyopathy requires abstinence

Smoking

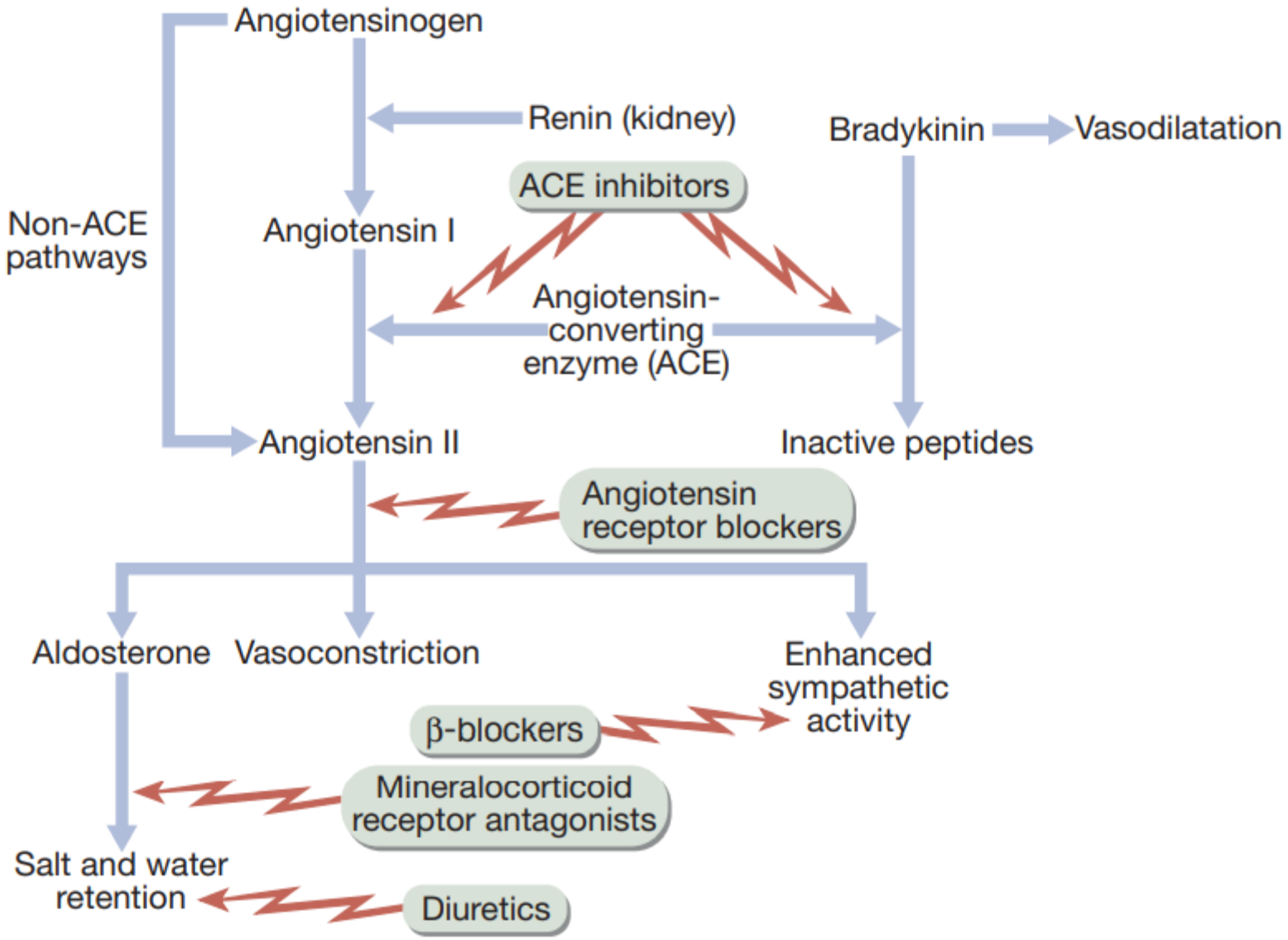
- Cessation

Exercise

- Regular moderate aerobic exercise within limits of symptoms

Vaccination

- Consideration of influenza and pneumococcal vaccination



Neurohumoral activation and sites of action of drugs used in the treatment of heart failure.

Diuretics promote urinary sodium and water excretion, leading to a reduction in blood plasma volume, which in turn reduces preload and improves pulmonary and systemic venous congestion.

They may also reduce afterload and ventricular volume, leading to a fall in ventricular wall tension and increased cardiac efficiency. Although a fall in preload (ventricular filling pressure) normally reduces cardiac output, patients with heart failure are beyond the apex of the Starling curve, so there may be a substantial and beneficial fall in filling pressure with either no change or an improvement in cardiac output.

Nevertheless, the dose of diuretics needs to be titrated carefully so as to avoid excessive volume depletion, which can cause a fall in cardiac output with hypotension, lethargy and renal failure. This is especially likely in patients with a marked diastolic component to their heart failure.

Oedema may persist, despite oral loop diuretic therapy, in some patients with severe chronic heart failure, particularly if there is renal impairment. Under these circumstances an intravenous infusion of furosemide (5–10 mg/hr) may initiate a diuresis. Combining a loop diuretic with a thiazide diuretic such as bendroflumethiazide (5 mg daily) may also prove effective but care must be taken to avoid an excessive diuresis.

Mineralocorticoid receptor antagonists, such as spironolactone and eplerenone, are potassium-sparing diuretics that are of particular benefit in patients with heart failure with severe left ventricular systolic dysfunction.

They have been shown to improve long-term clinical outcome in individuals with severe heart failure or heart failure following acute MI but may cause hyperkalaemia, particularly when used with an ACE inhibitor.

Angiotensin-converting enzyme inhibitors ACE inhibitors play a central role in the management of heart failure since they interrupt the vicious circle of neurohumoral activation that is characteristic of the disease by preventing the conversion of angiotensin I to angiotensin II. This, in turn, reduces peripheral vasoconstriction, activation of the sympathetic nervous system, and salt and water retention due to aldosterone release, as well as preventing the activation of the renin–angiotensin system caused by diuretic therapy.

In moderate and severe heart failure, ACE inhibitors can produce a substantial improvement in effort tolerance and in mortality. They can also improve outcome and prevent the onset of overt heart failure in patients with poor residual left ventricular function following MI.

Adverse effects of ACE inhibitors include symptomatic hypotension and impairment of renal function, especially in patients with bilateral renal artery stenosis or those with pre-existing renal disease.

An increase in serum potassium concentration may also occur, which can be beneficial in offsetting the hypokalaemia associated with loop diuretic therapy. Short-acting ACE inhibitors can cause marked falls in BP, particularly in the elderly or when started in the presence of hypotension, hypovolaemia or hyponatraemia. In stable patients without hypotension (systolic BP over 100 mmHg), ACE inhibitors can usually be safely started in the community.

In other patients, however, it is usually advisable to withhold diuretics for 24 hours before starting treatment with a small dose of a long-acting agent, preferably given at night.

Renal function and serum potassium must be monitored and should be checked 1–2 weeks after starting therapy.

Angiotensin receptor blockers ARBs act by blocking the action of angiotensin II on the heart, peripheral vasculature and kidney.

In heart failure, they produce beneficial haemodynamic changes that are similar to the effects of ACE inhibitors but are generally better tolerated. They have comparable effects on mortality and are a useful alternative for patients who cannot tolerate ACE inhibitors. Like ACE inhibitors they should be started at a low dose and titrated upwards, depending on response.

Unfortunately, they share all the more serious adverse effects of ACE inhibitors, including renal dysfunction and hyperkalaemia. ARBs are normally used as an alternative to ACE inhibitors.

Neprilysin inhibitors The only drug currently in this class is sacubitril, a small-molecule inhibitor of neprilysin, which is responsible for the breakdown of the endogenous diuretics ANP and BNP. Used in combination with the ARB valsartan (sacubitril–valsartan), it has been shown to produce additional symptomatic and mortality benefit over ACE inhibition and is now recommended in the management of resistant heart failure.

Vasodilators These drugs are valuable in chronic heart failure, when ACE inhibitors or ARBs are contraindicated. Venodilators, such as nitrates, reduce preload, and arterial dilators, such as hydralazine, reduce afterload. Their use is limited by pharmacological tolerance and hypotension.

Beta-adrenoceptor blockers Beta-blockade helps to counteract the deleterious effects of enhanced sympathetic stimulation and reduces the risk of arrhythmias and sudden death. When initiated in standard doses β-blockers may precipitate acute-on-chronic heart failure, but when given in small incremental doses they can increase ejection fraction, improve symptoms, reduce the frequency of hospitalisation and reduce mortality in patients with chronic heart failure. A typical regimen is bisoprolol, starting at a dose of 1.25 mg daily and increased gradually over a 12-week period to a target maintenance dose of 10 mg daily. Beta-blockers are more effective at reducing mortality than ACE inhibitors.

Ivabradine acts on the If inward current in the SA node, resulting in reduction of heart rate. It reduces hospital admission and mortality rates in patients with heart failure due to moderate or severe left ventricular systolic impairment, ivabradine is best suited to patients who cannot take β-blockers or whose heart rate remains high despite β-blockade. **It is ineffective in patients with atrial fibrillation.**

Digoxin can be used to provide rate control in patients with heart failure and atrial fibrillation. In patients with severe heart failure (NYHA class III–IV), digoxin reduces the likelihood of hospitalisation for heart failure, although it has no effect on long-term survival.

Amiodarone This is a potent anti-arrhythmic drug that has little negative inotropic effect and may be valuable in patients with poor left ventricular function. It is effective only in the treatment of symptomatic arrhythmias and should not be used as a preventative agent in asymptomatic patients. Amiodarone is used for prevention of symptomatic atrial arrhythmias and

of ventricular arrhythmias when other pharmacological options have been exhausted.

16.18 Dosages of ACE inhibitors, β-blockers and angiotensin receptor blockers in heart failure

	Starting dose	Target dose
ACE inhibitors		
Enalapril	2.5 mg twice daily	10 mg twice daily
Lisinopril	2.5 mg daily	20 mg daily
Ramipril	1.25 mg daily	10 mg daily
Angiotensin receptor blockers		
Losartan	25 mg daily	100 mg daily
Candesartan	4 mg daily	32 mg daily
Valsartan	40 mg daily	160 mg daily
β-blockers		
Bisoprolol	1.25 mg daily	10 mg daily
Metoprolol	25 mg twice daily	100 mg twice daily
Carvedilol	3.125 mg twice daily	25 mg twice daily

Non-pharmacological treatments

ICD

CRT

CORONARY REVASCULARISATION

CARDIAC TRANSPLANTATION

LVAD