BMJ Best Practice

Acute heart failure

Straight to the point of care

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Summary

Suspect acute heart failure in any patient with: breathlessness, ankle swelling, reduced exercise tolerance, fatigue, tiredness, increased time to recover after exercise, and nocturnal cough.

Urgently assess for any signs or symptoms related to the underlying cause of acute heart failure.

Arrange immediate bedside echocardiography (requires specialist expertise) and ECG for any patient who is haemodynamically unstable (low blood pressure or shock) or in respiratory failure with suspected acute heart failure as part of looking for life-threatening causes.

Urgently identify and treat any underlying precipitants/causes of acute heart failure that must be managed immediately to prevent further rapid deterioration (while recognising that any acute heart failure is potentially life-threatening).

Request urgent cardiology/critical care support for any patient with: respiratory distress/failure; reduced consciousness; use of accessory muscles for breathing, respiratory rate >25/minute; oxygen saturation (SpO_2) <90% despite supplemental oxygen; heart rate <40 or >130 beats per minute; systolic blood pressure persistently <90 mmHg (unless known to be usually hypotensive); signs or symptoms of hypoperfusion; haemodynamic instability; acute heart failure due to an acute coronary syndrome; persistent life-threatening arrhythmia.

For any patient with suspected heart failure always record and interpret a 12-lead ECG; monitor this continuously.

Also always order a chest x-ray, N-terminal-proB-type natriuretic peptide (NT-proBNP), or BNP as well other standard blood tests, and echocardiography to establish the presence or absence of cardiac abnormalities.

Determine acute drug treatment based on the patient's clinical presentation, including haemodynamic status and the presence of shock; drug treatment options include vasoactive drugs, diuretics, and vasodilators.

After stabilisation, start an oral diuretic if the patient has symptoms or signs of congestion, or switch from an intravenous to an oral diuretic once a patient who was started on an intravenous diuretic in the acute phase is euvolaemic.

Plan subsequent treatment based on measurement of the patient's left ventricular ejection fraction using echocardiography and their level of symptoms.

Ensure the patient has input from the heart failure specialist team within 24 hours of admission to hospital.

Definition

Heart failure is defined clinically as a syndrome in which patients have symptoms and signs resulting from an abnormality of cardiac structure and/or function.[1] Acute heart failure refers to rapid onset or worsening of symptoms and/or signs of heart failure, requiring urgent evaluation and treatment.[1] This topic does not cover children or pregnant women.

THEORY

Epidemiology

Approximately 900,000 people in the UK have heart failure; 5% of all adult emergency hospital admissions in the UK are caused or complicated by acute heart failure.[3] [4] Outside of the UK, prevalence of heart disease is about 1.3% in China, 6.7% in Malaysia, 1% in Japan, 4.5% in Singapore, 0.12% to 0.44% in India, 1% in South America, and 1% to 2% in Australia.[5]

The National Heart Failure Audit 2020/21, which included 82% of patients admitted to hospital with acute heart failure in England and Wales, showed a mean age of 77.8 years overall. There were more men at all ages, apart from the ≥85 years group in which women were a majority. Heart failure admissions dropped by 11% compared with 2019/20, reflecting the impact of the coronavirus disease 2019 (COVID-19) pandemic on hospitalisations.[6]

Risk factors

Strong

previous cardiovascular disease

Coronary heart disease is the most common cause of heart failure.[10]

older age

Prevalence of heart failure is ≥10% in people >70 years of age.[1]

prior episode of heart failure

In patients hospitalised for acute heart failure, around 75% have a history of prior heart failure.[11]

diabetes mellitus

Related directly to ischaemia and renal failure.

family history of ischaemic heart disease or cardiomyopathy

A risk factor for acute heart failure.[10]

excessive alcohol intake

Alcohol consumption is associated with a higher risk of heart failure.[1] [12]

smoking

The epidemiological associations of smoking with the development of cardiovascular disease suggest that smoking cessation would be beneficial.[1]

cardiac arrhythmias

Cardiac arrhythmias, including tachyarrhythmia and bradyarrhythmia, are risk factors for acute heart failure.

history of systemic conditions associated with heart failure

Conditions that are associated with heart failure include sarcoidosis and haemochromatosis.

previous chemotherapy

A risk factor for heart failure.

medication

Non-adherence to medication is a precipitating factor in patients with chronic heart failure. Drugs that may exacerbate heart failure include non-steroidal anti-inflammatory drugs, steroids, diltiazem, and verapamil.[10]

hypertension

Hypertension is associated with an increased risk of developing heart failure. Antihypertensive therapy markedly reduces the incidence of heart failure; however, alpha-adrenoceptor blockers are less effective than other antihypertensives in preventing heart failure.[1]

Weak

valvular heart disease

Both significant stenotic and regurgitate lesions can lead to heart failure.

Although rheumatic valvular disease is now rarely found in western countries, calcific valvular heart disease (in particular, aortic stenosis) is commonly encountered.

In patients with significant valvular disease, the heart failure will not improve until the underlying valvular disease has been corrected.

pericardial disease

A large pericardial effusion can present with symptoms or signs of acute heart failure.

Pericardial constriction, such as tuberculosis pericarditis or the effects of radiotherapy, can also present with acute heart failure.

myocarditis

There are many causes of myocarditis, of which a viral aetiology appears to be the most common. There is usually a prodrome of a non-specific illness characterised by fatigue, mild dyspnoea, and myalgias.

excessive salt intake

Noted in 22% of people with heart failure.[13]

excessive catecholamine stimulation

Can be caused by phaeochromocytoma or subarachnoid haemorrhage.[14]

abnormal thyroid function

Both hypothyroidism and thyrotoxicosis can be associated with heart failure.[15] [16]

Aetiology

Causes and precipitating factors are:

- Acute coronary syndrome (ACS)

- Hypertensive emergency
- Rapid arrhythmias or severe bradycardia/conduction disturbance
- An acute mechanical cause (e.g., myocardial rupture as a complication of ACS [such as free wall rupture], ventricular septal defect or acute mitral regurgitation, chest trauma)
- Acute pulmonary embolism
- Valve disease
- Myocarditis
- Decompensation of pre-existing chronic heart failure
- Cardiac tamponade
- Aortic dissection
- Postpartum cardiomyopathy
- Lack of adherence with medical treatment
- Volume overload
- Infections
- Severe brain insult
- After major surgery
- Reduction of renal function
- Drug abuse
- Phaeochromocytoma
- High output syndromes
    - Septicaemia
    - Thyrotoxic crisis
    - Anaemia
    - Shunt syndromes.

The most common concurrent conditions present in patients with acute heart failure are coronary artery disease, hypertension, diabetes mellitus, atrial fibrillation, and renal insufficiency.[7] [8]

Pathophysiology

During an episode of acute heart failure, the majority of patients will have evidence of volume overload with pulmonary and/or venous congestion. Haemodynamic measurements in these cases usually show increased right- and left-sided ventricular filling pressures with depressed cardiac index and cardiac output. However, if there is associated infection, the cardiac output may be normal or, in some cases, increased.

Activation of the sympathetic nervous system causes tachycardia, increased myocardial contractility, increased myocardial oxygen consumption, peripheral vasoconstriction, and activation of renin-angiotensin system with salt and water retention. There is also activation of vasoconstrictor neurohormones, which leads to sodium and fluid retention, increased myocardial wall stress, and decreased renal perfusion.[9]

If the condition is not treated effectively, the myocardium becomes unable to maintain a cardiac output sufficient to meet the demands of the peripheral circulation. In order for patients with acute heart failure to respond quickly to treatment, the increased myocardial stress must be reversed: for example, correction of acute severe hypertension. This is particularly important in acute heart failure caused by ischaemia, as a dysfunctional myocardium can return to normal when appropriately treated.

Classification

There have been several attempts to classify acute heart failure based on different criteria. The European Society of Cardiology supports a classification of acute heart failure based on clinical presentation. This allows the rapid identification and management of potentially reversible causes, precipitants, and coexisting life threatening conditions.[1]

Clinical presentation

The European Society of Cardiology describes four major clinical presentations of acute heart failure based on the presence of signs of congestion and/or peripheral hypoperfusion. While these presentations may overlap, each requires different treatment:[1]

- Acute decompensated heart failure
    - symptoms associated with peripheral fluid accumulation, increased intraventricular pressure
    - gradual onset (days)
    - normal or low systolic blood pressure
- Acute pulmonary oedema
    - symptoms associated with fluid redistribution to the lungs and acute respiratory failure
    - rapid onset (hours)
- Isolated right ventricular failure
    - symptoms from increased central venous pressure and often systemic hypoperfusion
    - gradual or rapid onset
    - low systolic blood pressure
- Cardiogenic shock
    - symptoms from systemic hypoperfusion (severe cardiac dysfunction)
    - gradual or rapid onset
    - low systolic blood pressure

Precipitants

An alternative approach is to classify patients according to the presence of factors leading to decompensation (which need to be treated urgently):[1]

- Acute coronary syndrome
- Hypertensive emergency
- Rapid arrhythmias or severe bradycardia/conduction disturbance
- Acute mechanical cause underlying acute heart failure
- Acute pulmonary embolism
- Infection (including myocarditis)

- Tamponade.

Types of heart failure

Traditionally heart failure is classified as:

- Systolic - associated with left ventricular dysfunction and characterised by cardiomegaly, third heart sound, and volume overload with pulmonary congestion. Left ventricular ejection fraction (LVEF) is decreased
- Diastolic - typically associated with normal cardiac size, hypertension, pulmonary congestion, and a fourth heart sound. LVEF is preserved.

Based on measurement of LVEF, heart failure is classified as:[1]

- Heart failure with reduced ejection fraction (HFrEF) - symptoms and signs and LVEF ≤40%
    - A subgroup of HFrEF can be further classified as heart failure with improved ejection fraction (HFimpEF) if a previous ejection fraction ≤40% has improved to >40% after follow-up measurement[2]
- Heart failure with mildly reduced ejection fraction (HFmrEF) - symptoms and signs and LVEF 41% to 49%
- Heart failure with preserved ejection fraction (HFpEF) - symptoms and signs and LVEF ≥50%
    - Evidence of structural heart disease may be used to further support the diagnosis of HFpEF.[2]

The diagnosis of HFmrEF and HFpEF requires evidence of spontaneous (at rest) or provokable (e.g., during exercise, fluid challenge) increased LV filling pressures.[1] [2] This may be fulfilled by elevated natriuretic peptides, non-invasive measures (e.g., echocardiographic diastolic parameters), or invasive haemodynamic measurement.[2]

Case history

Case history #1

A 70-year-old woman describes increasing exertional dyspnoea for the last 2 days and now has dyspnoea at rest. She has a history of hypertension for the last 5 years and a 35 pack-year smoking history, but no other established illnesses. Current medications are a diuretic daily for the last 3 years. She has been prescribed an ACE inhibitor but failed to collect the prescription. On examination her BP is 190/90 mmHg, and her heart rate is 104 bpm. There is an audible S4 and the jugular venous pressure is elevated 2 cm above normal. Lung examination reveals fine bibasal crepitations. Echocardiogram demonstrates normal biventricular size, a left ventricular ejection fraction of 60%, and no significant valvular disease.

Case history #2

A 73-year-old woman with a history of myocardial infarction presents to the accident and emergency department. She is breathless and finding it difficult to talk in full sentences. On examination she is centrally cyanosed with cool extremities. Her pulse is 110 bpm and systolic BP only just recordable at 80 mmHg. Jugular venous pressure is elevated 5 cm above normal, there is a gallop rhythm, and the cardiac apex beat is displaced. Respiratory rate is increased and she has widespread crackles and wheezes on chest examination. Echocardiogram shows an ejection fraction of 35%

Other presentations

Patients may present with predominant symptoms of the underlying condition such as chest pain with acute myocardial infarction, syncope with significant valvular stenosis, palpitations with arrhythmias, and viral prodrome with myocarditis.

Recommendations

Urgent

Arrange **immediate bedside echocardiography** (requires specialist expertise) and **ECG** for any patient who is haemodynamically unstable (low blood pressure or shock) or in respiratory failure with suspected acute heart failure as part of looking for **life-threatening causes**. Life-threatening causes include:[1] [4]

- Acute coronary syndrome (ACS)[4]
- Hypertensive emergency
- Rapid arrhythmias or severe bradycardia/conduction disturbance
- An acute mechanical cause (e.g., myocardial rupture as a complication of ACS, chest trauma)
- Acute pulmonary embolism
- Infection (including myocarditis)
- Tamponade.

Request **urgent critical care/cardiology support** for any patient with: [1]

- Respiratory distress/failure[22]
- Reduced consciousness
- Heart rate <40 or >130 bpm[22]
- Systolic blood pressure persistently <90 mmHg[22]
    - Unless known to be usually hypotensive (based on the opinion of our expert adviser)
- Signs or symptoms of hypoperfusion - see Shock
- Haemodynamic instability
- Acute heart failure due to ACS[4]
- Persistent life-threatening arrhythmia.

Request serum **brain natriuretic peptide (BNP) measurement** in the first set of blood tests for all patients with acute breathlessness who may have new acute heart failure.[4] [23]

- The UK National Institute for Health and Care Excellence recommends use of N-terminal pro-B-type natriuretic peptide (NT-proBNP).[24]
- BNP measurement is useful in differentiating acute heart failure from non-cardiac causes of acute dyspnoea.[1]

Organise **rapid transfer to hospital** (preferably to a site with a cardiology department and/or a coronary care/intensive care unit) for any patient in the community with suspected acute heart failure.[1]

- Acute heart failure is potentially life-threatening and requires urgent evaluation and treatment.

Key Recommendations

Assess for common symptoms and signs of acute heart failure. These include:[1]

- Dyspnoea
- Orthopnoea
- Paroxysmal nocturnal dyspnoea

- Ankle swelling
- Reduced exercise tolerance
- Fatigue
- Elevated jugular venous pressure
- Third heart sound (gallop rhythm)
- Pulmonary crepitations.

Ask about risk factors for heart failure. Established heart failure is unusual in a patient with no relevant medical history.[1]

Establish the patient's haemodynamic status as this will determine further management. Most patients present with either normal (90-140 mmHg) or hypertensive (>140 mmHg) systolic blood pressure (SBP).

- Hypotension (SBP <90 mmHg) is associated with poor prognosis, particularly when hypoperfusion is present.

Always order the following investigations for a patient with new suspected acute heart failure to establish the presence or absence of cardiac abnormalities:[1] [23]

- ECG
- Chest x-ray
- N-terminal-proB-type natriuretic peptide (NT-proBNP) or BNP and other standard blood tests
- Echocardiography.[6]

Once stabilised, use the patient's left ventricular ejection fraction to guide disease-modifying treatment. Address causative aetiology and relevant comorbidities.[1]

Full Recommendations

Clinical presentation

Suspect acute heart failure in any patient with:

- **Breathlessness** [1]
  - This may be acute but also includes orthopnoea and paroxysmal nocturnal dyspnoea[25]
- **Ankle swelling** [1]
  - This often reduces when the patient's legs have been elevated for a sustained period of time (e.g., in bed overnight)
- Reduced exercise tolerance[1]
- Fatigue, tiredness, increased time to recover after exercise[1]
- Less common symptoms, including: wheezing, dizziness, confusion (especially in older patients), loss of appetite, nocturnal ischaemic pain, nocturnal cough (frothy sputum suggests that it is alveolar in origin and not bronchial).[1] [25]

Urgently assess for any signs or symptoms related to the underlying cause of acute heart failure.[1] [25]

- It is important to screen for an underlying cause of heart failure as this may be treatable.[1]

- Underlying precipitants/causes of acute heart failure that must be managed immediately to prevent further rapid deterioration (while recognising that any acute heart failure is potentially life-threatening) include:[1]
    - Acute coronary syndrome (ACS).[4] See Unstable angina , Non-ST elevation myocardial infarction , and ST-elevation myocardial infarction
    - Hypertensive emergency. See Hypertensive emergencies
    - Rapid arrhythmias or severe bradycardia/conduction disturbance. See Assessment of tachycardia and Bradycardia
    - An acute mechanical cause (e.g., myocardial rupture as a complication of ACS [such as free wall rupture], ventricular septal defect or acute mitral regurgitation, chest trauma)
    - Acute pulmonary embolism. See Pulmonary embolism
    - Valve disease
    - Myocarditis. See Myocarditis
    - Tamponade.

Request **urgent critical care/cardiology support** for any patient with:[1]

- Respiratory distress/failure[22]
- Reduced consciousness
- Heart rate <40 or >130 bpm[22]
- Systolic blood pressure persistently <90 mmHg[22]
    - Unless known to be usually hypotensive (based on the opinion of our expert adviser)
- Signs or symptoms of hypoperfusion.[1] See Assessment of shock
- Haemodynamic instability[1]
- Acute heart failure due to ACS[4]
- Persistent life-threatening arrhythmia.

DIAGNOSIS

History

Check whether the patient has **previously been diagnosed** and treated for heart failure. If so, ask about:

- Current treatment for heart failure and the patient's adherence.

Ask about **risk factors for heart failure** if the patient has not previously been diagnosed, including:

- Previous cardiovascular disease; coronary heart disease is the most common cause of heart failure[10]
- Older age[1]
    - Prevalence of heart failure is ≥10% in people >70 years of age[1]
- Diabetes[10]
- Family history of ischaemic heart disease or cardiomyopathy[10]
- Excessive alcohol intake or smoking[10]
- Cardiac arrhythmias including tachyarrhythmia or bradyarrhythmia
- History of systemic conditions associated with heart failure (e.g., sarcoidosis and haemochromatosis)
- Previous chemotherapy.

Ask about **recent drug history**. Drugs that may exacerbate heart failure include non-steroidal anti-inflammatory drugs, steroids, diltiazem, and verapamil.[10]

Practical tip

Heart failure is unusual in patients with no relevant medical history.[1]

Physical examination

Assess the **degree of dyspnoea**, including:

- Respiratory rate[1]
- Breathlessness when lying flat[22]
- Effort of breathing[22]
- Degree of hypoxia.

Look for **signs of poor perfusion** such as:

- Cold extremities[1]
- Narrow pulse pressure[1]
- Confusion (especially in the elderly)[1]
- Oliguria[1]
- Dizziness[1]
- Central cyanosis
- Delayed capillary refill time.

Check for **signs of congestion** such as:

- Peripheral oedema[1] [4]
    - Leg oedema is usually bilateral and pitting
- Pulmonary crepitations
- Dullness to percussion and decreased air entry in lung bases
- Wheezing[26]
- Elevated jugular venous pressure
- Ascites.[1] [4]

Measure the patient's blood pressure; haemodynamic status will determine further management.

- Most patients present with either normal (90-140 mmHg) or hypertensive (>140 mmHg) systolic blood pressure (SBP).[1]
- Hypotension (SBP <90 mmHg) is not always associated with hypoperfusion, as BP may be preserved by compensatory vasoconstriction.[1] See Shock .

Listen to the patient's heart sounds. Signs of acute heart failure include:

- A displaced apex beat[1]
- A gallop rhythm (third heart sound).[1]

Also listen for potential valvular causes such as aortic stenosis or mitral regurgitation.

Practical tip

The sound of the crackles heard on chest auscultation in heart failure is described as 'wet' and sounding like Velcro. Crackles in heart failure are usually fine and quiet rather than the coarse sounds that are more commonly heard in lung disease. They can be mistaken for the bilateral crackles of lung fibrosis, but patients with fibrotic lungs are more likely to be hypoxic with exertional desaturation.
Always check above the level of the patient's earlobes for a raised jugular venous pressure because this is easily missed. However, a raised jugular venous pressure can be difficult to spot, even for a heart failure specialist.

Investigations

Always order

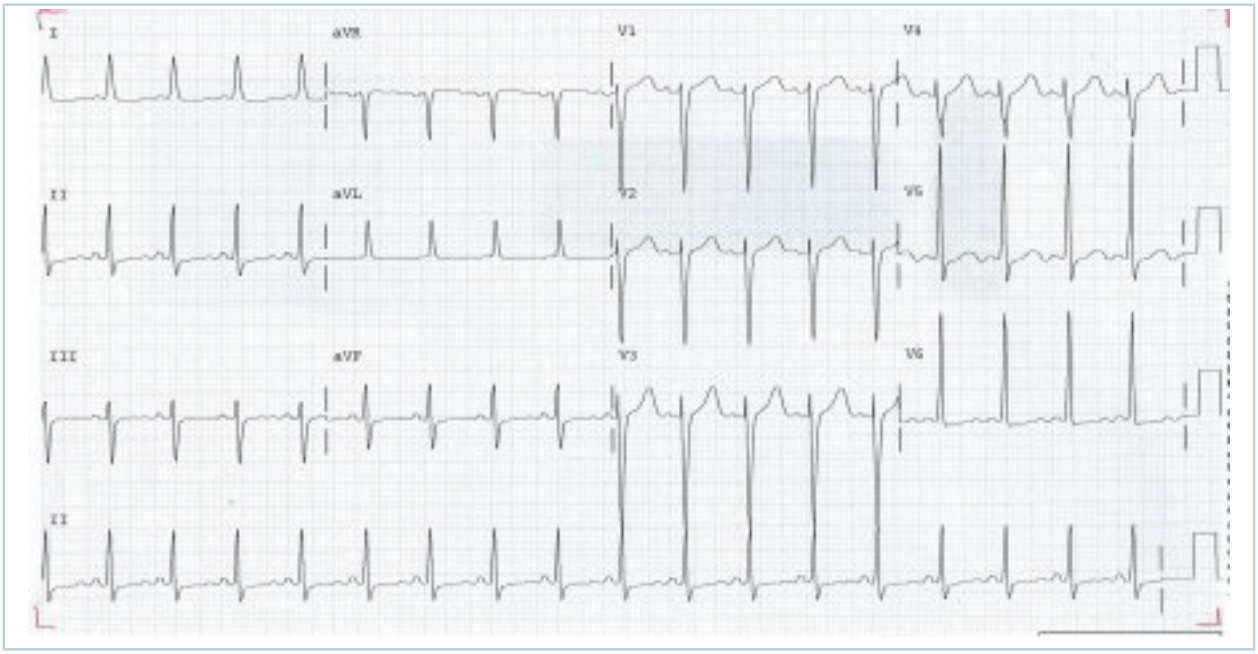
ECG

Record and interpret a **12-lead ECG** for all patients with suspected heart failure; **monitor this continuously**.[1]

- Arrange this immediately if the patient is haemodynamically unstable or in respiratory failure to look for any life-threatening cause of acute heart failure (e.g., acute coronary syndrome, particularly ST-elevation myocardial infarction). See ST-elevation myocardial infarction .[1]

Check heart rhythm, heart rate, QRS morphology, and QRS duration, as well as looking for specific abnormalities such as arrhythmias, atrioventricular block, evidence of a previous myocardial infarction (e.g., Q waves), and evidence of left ventricular hypertrophy.[1]

The **ECG is usually abnormal** in acute heart failure.[1]



ECG showing left ventricular hypertrophy with sinus tachycardia
From the private collections of Syed W. Yusuf, MBBS, MRCPI, and Daniel Lenihan, MD

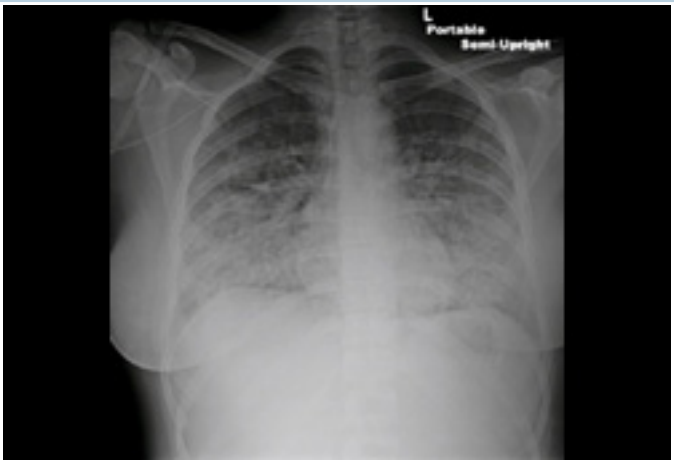
Chest x-ray

The UK National Institute for Health and Care Excellence recommends a chest x-ray for all patients with suspected heart failure.[23] Look for:[27]

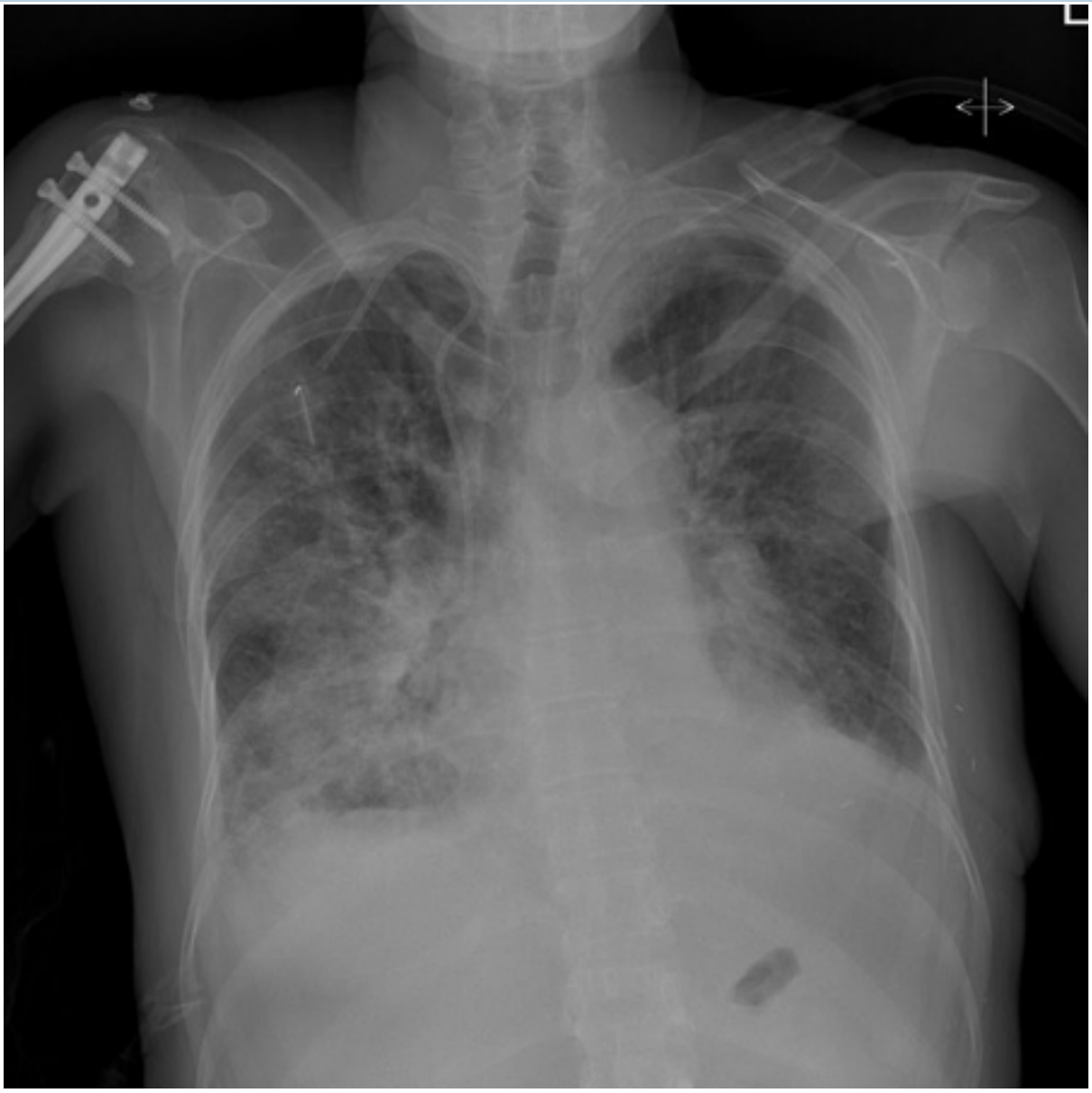
- Pulmonary congestion
- Pleural effusion
- Interstitial or alveolar fluid in horizontal fissure
- Cardiomegaly.

Practical tip

Be aware that significant left ventricular dysfunction may be present without cardiomegaly on chest x-ray.[27]



Chest x-ray showing acute pulmonary oedema with increased alveolar markings, fluid in the horizontal fissure, and blunting of the costophrenic angles
From the private collections of Syed W. Yusuf, MBBS, MRCPI, and Daniel Lenihan, MD



Chest x-ray showing acute pulmonary oedema with increased alveolar markings and bilateral pleural effusions
From the private collections of Syed W. Yusuf, MBBS, MRCPI, and Daniel Lenihan, MD

Blood tests

Natriuretic peptides

- Order **N-terminal pro-B-type natriuretic peptide (NT-proBNP)** if available. Brain natriuretic peptide (BNP) or mid-regional pro-atrial natriuretic peptide (MR-proANP) (in some countries) are alternatives.[1] [4] [23]
- Normal levels make the diagnosis of acute heart failure unlikely (normal levels: NT-proBNP <300 ng/L (<300 picograms [pg]/mL); BNP <100 ng/L (<100 pg/mL); MR-proANP <120 ng/L (<120 pg/mL).[1] [23] However, elevated levels of natriuretic peptides do not automatically confirm the diagnosis of acute heart failure as they may be associated with a wide variety of cardiac and non-cardiac causes. Low levels of natriuretic peptides can occur in end-stage heart failure, flash pulmonary oedema, or right-sided acute heart failure.[1]
- **Most older patients** presenting to hospital with acute breathlessness have an **elevated NT-proBNP** so separate 'rule in' diagnostic cut-offs are useful in this setting.
    - If NT-proBNP is significantly elevated (>1800 ng/L [>1800 pg/mL] in patients >75 years; see table below) acute heart failure is likely and should be confirmed by inpatient

echocardiography. If the NT-proBNP is intermediate (>300 ng/L [>300 pg/mL] but <1800 ng/L [<1800 pg/mL]), consider other possible causes of breathlessness, but if these are excluded and diagnostic suspicion of heart failure remains, request an echocardiogram.[23]

Age (years)	<50	50-75	>75
NT-proBNP level (ng/L or pg/mL) above which acute heart failure is likely [4]	>450	>900	>1800

Practical tip

Be aware that natriuretic peptides may be raised due to other causes (e.g., acute coronary syndrome, myocarditis, pulmonary embolism, older age, and renal or liver impairment), hence the need for practical 'rule in' cut-offs.[1]

Troponin

- Measure troponin in all patients with suspected acute heart failure.[22]
- Most patients with acute heart failure have an elevated troponin level. As well as diagnosis, troponin may be useful for prognosis; elevated levels are associated with poorer outcomes.[22]
- Be aware that **interpretation is not straightforward**; type 2 myocardial infarction and myocardial injury are common.

Full blood count

- Identify **anaemia**, which can worsen heart failure and also suggest an alternative cause of symptoms, and check iron status (transferrin, ferritin) before discharge.[1]

Electrolytes, urea, and creatinine

- Order as a baseline test to inform decisions on drug treatment that may affect renal function (e.g., diuretics, ACE inhibitors), and to exclude concurrent or causative renal failure.

Glucose

- Measure blood glucose in all patients with suspected acute heart failure to screen for diabetes.[22]
- In practice, also request HbA1c (based on the opinion of our expert).

Liver function tests

- Order these for any patient with suspected acute heart failure.
- Liver function tests are often elevated due to reduced cardiac output and increased venous congestion. Abnormal liver function tests are associated with a worse prognosis.[1]

Thyroid function tests

- Order thyroid-stimulating hormone in any patient with newly diagnosed acute heart failure. Both hypothyroidism and hyperthyroidism can cause acute heart failure.[1] See Overview of thyroid dysfunction.

C-reactive protein

- Consider ordering C-reactive protein (based on the opinion of our expert).
- Inflammation is associated with progression of chronic heart failure.
- Levels of high-sensitivity C-reactive protein are raised in patients with acute heart failure.[28]

D-dimer

- Indicated in patients with suspicion of acute pulmonary embolism.[22] See Pulmonary embolism .

Echocardiography

Arrange **immediate bedside echocardiography** for any patient with suspected acute heart failure who is haemodynamically unstable or in respiratory failure.[4] Specialist expertise is required.

If a patient is haemodynamically stable, arrange echocardiography within 48 hours.[6] [23]

Echocardiography is used to assess myocardial systolic and diastolic function of both left and right ventricles, assess valvular function, detect intracardiac shunts, and measure left ventricular ejection fraction (LVEF).[23]

- Evaluating the patient's LVEF has a key role in assessing the severity of any decrease in systolic function and is essential in determining your patient's long-term management.[1]

Practical tip

The diagnosis of heart failure with reduced ejection fraction (HFrEF) requires an LVEF ≤40%. Patients with heart failure with preserved ejection fraction (HFpEF) have clinical signs of heart failure with normal or near-normal LVEF, and evidence of structural and/or functional cardiac abnormalities (including raised natriuretic peptides) in the absence of significant valvular disease. There is an emerging group of patients with heart failure with mildly reduced ejection fraction (41% to 49%) (HFmrEF) and encouraging data with some therapies recommended for HFrEF. However, current guidelines do not offer strong recommendations regarding HFrEF therapies for these patients.[1]

Consider ordering

Venous or arterial blood gas

Perform an **arterial blood gas** (ABG) if an accurate measurement of arterial partial pressure of oxygen (PaO_2) and arterial partial pressure of carbon dioxide ($PaCO_2$) is needed.[1]

Consider measurement of blood pH and $PaCO_2$ even if the patient does not have cardiogenic shock, especially if respiratory failure is suspected.[1]

- Do not routinely perform an ABG. Venous blood gas may acceptably indicate pH and $PaCO_2$.

A blood gas may show:

- Hypoxaemia
  - PaO_2 <10.67 kPa (<80 mmHg) on arterial blood gas)
- Metabolic acidosis with raised lactate in a patient with hypoperfusion

- pH <7.35, lactate >2 mmol/L (>18 mg/dL)

- Type I or type II respiratory failure
  - Type I respiratory failure: PaO_2 <8 kPa (<60 mmHg)
  - Type II respiratory failure: $PaCO_2$ >6.65 kPa (>50 mmHg).

Blood tests screening for myocarditis

Consider blood tests screening for acute myocarditis if you suspect myocarditis as a cause of acute heart failure. These include screening for viruses that cause acute myocarditis, including coxsackievirus group B, HIV, cytomegalovirus, Ebstein-Barr virus, hepatitis, echovirus, adenovirus, enterovirus, human herpes virus 6, parvovirus B19, and influenza viruses. See Myocarditis .

Bedside thoracic ultrasound

Bedside thoracic ultrasound is useful in countries with no access to BNP/NT-proBNP testing for detecting signs of interstitial oedema and pleural effusion in heart failure if specialist expertise is available.[1] [4]

History and exam

Key diagnostic factors

breathlessness (common)

Assess the degree of dyspnoea, including:

- Respiratory rate[1]
- Breathlessness when lying flat[22]
- Effort of breathing[22]
- Degree of hypoxia.

Dyspnoea may be acute, but also includes orthopnoea and paroxysmal nocturnal dyspnoea.[25]

peripheral oedema (common)

Leg oedema is usually bilateral and pitting.

Ankle swelling often reduces when the patient's legs have been elevated for a sustained period of time (e.g., in bed overnight).

reduced exercise tolerance (common)

Due to poor cardiac functioning.

fatigue (common)

Fatigue, tiredness, and an increased time to recover after exercise are common signs of acute heart failure.

Due to poor cardiac functioning.

cold extremities (common)

A sign of poor perfusion. Other signs of poor perfusion include:

- Narrow pulse pressure[1]
- Altered mental status[1]
- Oliguria[1]
- Dizziness[1]
- Central cyanosis
- Delayed capillary refill time.

elevated jugular venous pressure (common)

A sign of congestion.

Always check above the level of the patient's earlobes for an elevated jugular venous pressure because this is easily missed. However, an elevated jugular venous pressure can be difficult to spot, even for a heart failure specialist.

risk factors (common)

Ask about risk factors for heart failure if the patient has not previously been diagnosed, including:

- Previous cardiovascular disease; coronary heart disease is the most common cause of heart failure[10]
- Older age[1]
    - Prevalence of heart failure is ≥10% in people >70 years of age[1]
- Diabetes[10]
- Family history of ischaemic heart disease or cardiomyopathy[10]
- Excessive alcohol intake or smoking[10]
- Cardiac arrhythmias including tachyarrhythmia or bradyarrhythmia
- History of systemic conditions associated with heart failure (e.g., sarcoidosis and haemochromatosis)
- Previous chemotherapy.

Ask about recent drug history. Drugs that may exacerbate heart failure include non-steroidal anti-inflammatory drugs, steroids, diltiazem, and verapamil.[10]

displaced apex beat (common)

A common sign of acute heart failure.

gallop rhythm (third heart sound) (common)

A common sign of acute heart failure.

Other diagnostic factors

nocturnal cough (uncommon)

Due to pulmonary congestion.

Frothy sputum suggests that it is alveolar in origin and not bronchial.

wheezing (uncommon)

A sign of congestion.

dizziness (uncommon)

A sign of poor perfusion. Other signs of poor perfusion include:

- Cold extremities
- Narrow pulse pressure[1]
- Altered mental status[1]
- Oliguria[1]
- Central cyanosis
- Delayed capillary refill time.

confusion (uncommon)

May be a sign of acute heart failure, especially in older people.

loss of appetite (uncommon)

A less common sign of acute heart failure.

nocturnal ischaemic pain (uncommon)

A less common sign of acute heart failure.

ascites (uncommon)

A less common sign of acute heart failure. Due to portal hypertension.

central cyanosis (uncommon)

A sign of poor perfusion. Other signs of poor perfusion include:

- Cold extremities
- Narrow pulse pressure[1]
- Altered mental status[1]
- Oliguria[1]
- Dizziness[1]
- Delayed capillary refill time.

narrow pulse pressure (uncommon)

A sign of poor perfusion. Other signs of poor perfusion include:

- Cold extremities
- Altered mental status[1]
- Oliguria[1]
- Dizziness[1]
- Central cyanosis

- Delayed capillary refill time.

altered mental status (uncommon)

A sign of poor perfusion. Other signs of poor perfusion include:

- Cold extremities
- Narrow pulse pressure[1]
- Oliguria[1]
- Dizziness[1]
- Central cyanosis
- Delayed capillary refill time.

oliguria (uncommon)

A sign of poor perfusion. Other signs of poor perfusion include:

- Cold extremities
- Narrow pulse pressure[1]
- Altered mental status[1]
- Dizziness[1]
- Central cyanosis
- Delayed capillary refill time.

delayed capillary refill time (uncommon)

A sign of poor perfusion. Other signs of poor perfusion include:

- Cold extremities
- Narrow pulse pressure[1]
- Altered mental status[1]
- Oliguria[1]
- Dizziness[1]
- Central cyanosis.

pulmonary crepitations (uncommon)

A sign of congestion.

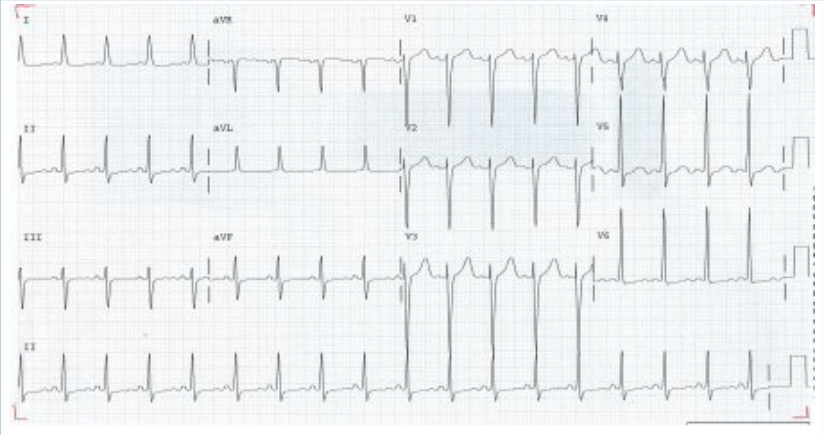
The sound of the crackles heard on chest auscultation in heart failure is described as ‘wet’ and sounding like Velcro. Crackles in heart failure are usually fine and quiet rather than the coarse sounds that are more commonly heard in lung disease. They can be mistaken for the bilateral crackles of lung fibrosis, but patients with fibrotic lungs are more likely to be hypoxic with exertional desaturation.

dullness to percussion/decreased air entry in lung bases (uncommon)

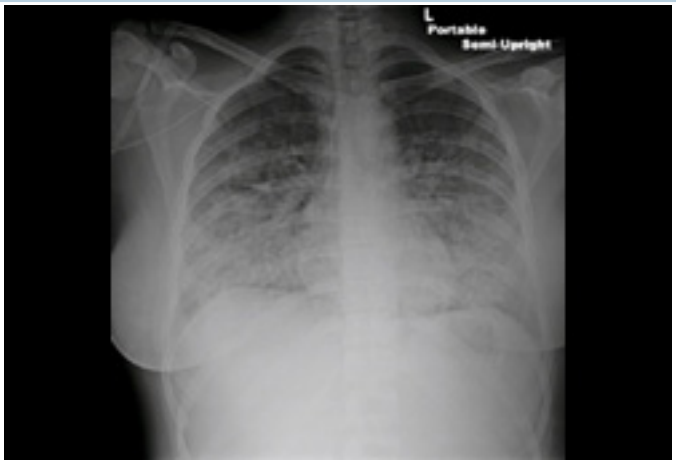
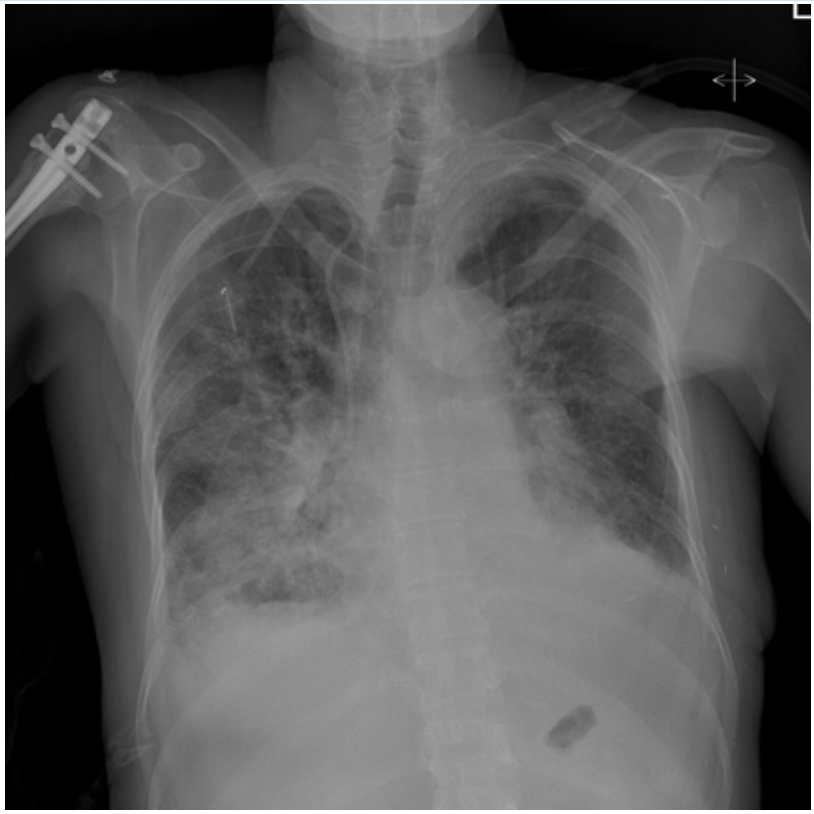
A sign of congestion.

Investigations

1st test to order

Test	Result
ECG Record and interpret a 12-lead ECG for any patient with suspected heart failure; monitor this continuously.[1] • Arrange this immediately if the patient is haemodynamically unstable or in respiratory failure to look for any life-threatening cause of acute heart failure such as acute coronary syndrome. See Non-ST-elevation myocardial infarction. [1] Check heart rhythm, heart rate, QRS morphology, and QRS duration, as well as looking for specific abnormalities such as arrhythmias, atrioventricular block, evidence of a previous myocardial infarction (e.g., Q waves) and evidence of left ventricular hypertrophy.[1] The ECG is usually abnormal in acute heart failure.[1]  *ECG showing left ventricular hypertrophy with sinus tachycardia* *From the private collections of Syed W. Yusuf, MBBS, MRCPI, and Daniel Lenihan, MD; used with permission*	arrhythmias, ischaemic ST- and T-wave changes
chest x-ray Look for:[23] [27] • Pulmonary congestion • Pleural effusion • Interstitial or alveolar oedema • Cardiomegaly. **Practical tip** Be aware that significant left ventricular dysfunction may be present without cardiomegaly on chest x-ray.[27]	pulmonary congestion, pleural effusion, pulmonary oedema, cardiomegaly

DIAGNOSIS

Test	Result
 *Chest x-ray showing acute pulmonary oedema with increased alveolar markings, fluid in the horizontal fissure, and blunting of the costophrenic angles* *From the private collections of Syed W. Yusuf, MBBS, MRCPI, and Daniel Lenihan, MD; used with permission*  *Chest x-ray showing acute pulmonary oedema with increased alveolar markings and bilateral pleural effusions* *From the private collections of Syed W. Yusuf, MBBS, MRCPI, and Daniel Lenihan, MD; used with permission*	
natriuretic peptides Order N-terminal pro-B-type natriuretic peptide (NT-proBNP) if available. Brain natriuretic peptide (BNP) or mid-regional pro-	NT-proBNP >300 ng/L (>300 picograms [pg]/mL), BNP

Test	Result
atrial natriuretic peptide (MR-proANP) (in some countries) are alternatives.[1] [4] [23] Normal levels make the diagnosis of acute heart failure unlikely.[1] [23] However, elevated levels of natriuretic peptides do not automatically confirm the diagnosis of acute heart failure as they may be associated with a wide variety of cardiac and non-cardiac causes. Low levels of natriuretic peptides can occur in end-stage heart failure, flash pulmonary oedema, or right-sided acute heart failure.[1] Most older patients presenting to hospital with acute breathlessness have an elevated NT-proBNP so separate 'rule in' diagnostic cut-offs are useful in this setting. • If NT-proBNP is significantly elevated (>1800 ng/L [>1800 pg/mL] in patients >75 years; see table below) acute heart failure is likely and should be confirmed by inpatient echocardiography. If the NT-proBNP is intermediate (>300 ng/L [>300 pg/mL] but <1800 ng/L [<1800 pg/mL]), consider other possible causes of breathlessness, but if these are excluded and diagnostic suspicion of heart failure remains, request an echocardiogram.[23]	>100 ng/L (>100 pg/mL), MR-proANP >120 ng/L (>120 pg/mL)

Age (years)	<50	50-75	>75
NT-proBNP level (ng/L or pg/mL) above which acute heart failure is likely [4]	>450	>900	>1800

Practical tip

Be aware that natriuretic peptides may be raised due to other causes (e.g., acute coronary syndrome, myocarditis, pulmonary embolism, older age, and renal or liver impairment), hence the need for practical 'rule in' cut-offs.[1]

troponin Measure troponin in all patients with suspected acute heart failure.[22] Troponin may be useful for prognosis; elevated levels are associated with poorer outcomes.[22] Be aware that interpretation is not straightforward; type 2 myocardial infarction and myocardial injury are common.	most patients with acute heart failure have an elevated troponin level

Test	Result
full blood count Order a full blood count to identify anaemia, which can worsen heart failure and also suggest an alternative cause of symptoms.[1]	may show anaemia
urea, electrolytes, and creatinine Order as a baseline test to inform decisions on drug treatment that may affect renal function (e.g., diuretics, ACE inhibitors), and to exclude concurrent or causative renal failure.	baseline levels
glucose and HbA1c Measure blood glucose in all patients with suspected acute heart failure to screen for diabetes.[22] In practice, also request HbA1c (based on the opinion of our expert).	may be elevated
liver function tests Order these for any patient with suspected acute heart failure. Liver function tests are often elevated due to reduced cardiac output and increased venous congestion. Abnormal liver function tests are associated with a worse prognosis.[1]	may be elevated
thyroid function tests Order thyroid-stimulating hormone in any patient with newly diagnosed acute heart failure. Both hypothyroidism and hyperthyroidism can cause acute heart failure.[1] See Overview of thyroid dysfunction .	may show hypothyroidism or hyperthyroidism
C-reactive protein Consider ordering C-reactive protein (based on the opinion of our expert). Inflammation is associated with progression of chronic heart failure. Levels of high-sensitivity C-reactive protein are raised in patients with acute heart failure.[28]	raised in acute heart failure
D-dimer Indicated in patients with suspicion of acute pulmonary embolism.[22]	raised in acute pulmonary embolism; see Pulmonary embolism
echocardiography Arrange immediate bedside echocardiography for any patient with suspected heart failure who is haemodynamically unstable or in respiratory failure.[4] Specialist expertise is required. If a patient is haemodynamically stable, arrange echocardiography within 48 hours.[6] [23] Echocardiography is used to assess myocardial systolic and diastolic function of both left and right ventricles, assess valvular function, detect intracardiac shunts, and measure left ventricular ejection fraction (LVEF).[23]	left ventricular systolic dysfunction, left ventricular diastolic dysfunction, constriction, left ventricular hypertrophy, valve disease, restrictive heart disease, right ventricular dysfunction, pulmonary hypertension, may detect the underlying cause

Test	Result
• Evaluating the patient's LVEF has a key role in assessing the severity of any decrease in systolic function and is essential in determining your patient's long-term management.[1] **Practical tip** The diagnosis of heart failure with reduced ejection fraction (HFrEF) requires an LVEF ≤40%. Patients with heart failure with preserved ejection fraction (HFpEF) have clinical signs of heart failure with normal or near-normal LVEF, and evidence of structural and/or functional cardiac abnormalities (including raised natriuretic peptides) in the absence of significant valvular disease. There is an emerging group of patients with heart failure with mildly reduced ejection fraction (41% to 49%) (HFmrEF) and encouraging data with some therapies recommended for HFrEF. Current guidelines do not offer any strong recommendations regarding HFrEF therapies for these patients.[1]	

Other tests to consider

Test	Result
venous or arterial blood gas Perform an arterial blood gas (ABG) if an accurate measurement of arterial partial pressure of oxygen (PaO_2) and arterial partial pressure of carbon dioxide ($PaCO_2$) is needed.[1] Consider measurement of blood pH and $PaCO_2$ even if the patient does not have cardiogenic shock, especially if respiratory failure is suspected.[1] • Do not routinely perform an ABG. Venous blood gas may acceptably indicate pH and $PaCO_2$. [1] A blood gas may show: • Hypoxaemia • Metabolic acidosis with raised lactate in a patient with hypoperfusion • Type I or type II respiratory failure.	hypoxaemia: PaO_2 <10.67 kPa (<80 mmHg) on arterial blood gas; metabolic acidosis with raised lactate: pH <7.35, lactate >2 mmol/L (>18 mg/dL); type I respiratory failure: PaO_2 <8 kPa (<60 mmHg); type II respiratory failure: $PaCO_2$ >6.65 kPa (>50 mmHg)
blood tests screening for myocarditis Consider blood tests screening for acute myocarditis if you suspect myocarditis as a cause of acute heart failure. These include screening for viruses that cause acute myocarditis, including coxsackievirus group B, HIV, cytomegalovirus, Ebstein-Barr virus, hepatitis, echovirus, adenovirus, enterovirus, human herpes virus 6, parvovirus B19, and influenza viruses. See Myocarditis .	may show presence of virus
bedside thoracic ultrasound Bedside thoracic ultrasound is useful in countries with no access to BNP/NT-proBNP testing for detecting signs of interstitial oedema and pleural effusion in heart failure if specialist expertise is available.[1] [4]	interstitial oedema, pleural effusion

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Pneumonia	• Fever, cough, productive sputum. • Focal signs of consolidation - increased vocal fremitus and bronchial breathing.	• WBC: elevated. • Blood cultures: positive for organism. • Chest x-ray: consolidation.
Pulmonary embolism	• Haemoptysis and sharp, pleuritic chest pain. • Risk factors of thromboembolism (TE) include personal history of TE, family history, recent trauma, prolonged immobilisation, smoker, or combined hormonal contraception use.	• CT pulmonary angiography: clot in pulmonary artery.
Asthma	• Wheezing on physical examination.	• Reduced peak flow. • Spirometry: obstructive pattern, reversibility with beta-agonist inhalers
Interstitial lung disease	• Progressively increasing dyspnoea. • Oxygen desaturation with exercise. • Fine bibasal crepitations with no other signs of heart failure.	• Chest x-ray: reticular infiltrate in the late stages of disease. • High-resolution CT scan: ground-glass appearance, reticular infiltrates, honeycombing, and architectural distortion. • Spirometry: restrictive pattern.
Acute respiratory distress syndrome	• Severe hypoxia, fine crepitations.	• Chest x-ray: diffuse infiltrates • Pulmonary artery wedge pressure: <18 mmHg

Criteria

New York Heart Association (NYHA) functional classification of heart failure based on severity of symptoms and physical activity[1]

- Class I: asymptomatic
  - No limitation of physical activity
  - Ordinary physical activity does not result in undue breathlessness, fatigue, or palpitations
- Class II: mild symptoms with moderate exertion
  - Slight limitation of physical activity

- Comfortable at rest
- Ordinary physical activity causes undue breathlessness, fatigue, or palpitations

- Class III: symptoms with minimal activity
  - Marked limitation of physical activity
  - Comfortable at rest
  - Less than ordinary physical activity causes undue breathlessness, fatigue, or palpitations

- Class IV: symptoms at rest
  - Unable to carry on any physical activity without discomfort
  - Discomfort increases if any physical activity undertaken

Recommendations

Urgent

Request **urgent cardiology/critical care support** for any patient with:[1]

- Respiratory distress/failure[22]
- Reduced consciousness
- Use of accessory muscles for breathing, respiratory rate >25/minute[22]
- Oxygen saturation (SpO_2) <90% despite supplemental oxygen
- Heart rate <40 or >130 bpm[22]
- Systolic blood pressure persistently <90 mmHg[22]
    - Unless known to be usually hypotensive (based on the opinion of our expert adviser)
- Signs or symptoms of hypoperfusion (see Shock)[1]
- Haemodynamic instability[1]
- Acute heart failure due to an acute coronary syndrome (ACS)[4]
- Persistent life-threatening arrhythmia.

Urgently identify and treat any underlying precipitants/causes of acute heart failure that must be managed immediately to prevent further rapid deterioration (while recognising that any acute heart failure is potentially life-threatening). Causes to consider include:[1]

- ACS. See Unstable angina , Non-ST elevation myocardial infarction , and ST-elevation myocardial infarction [4]
- Hypertensive emergency. See Hypertensive emergencies
- Rapid arrhythmias or severe bradycardia/conduction disturbance. See Assessment of tachycardia and Bradycardia
- An acute mechanical cause (e.g., myocardial rupture as a complication of ACS, chest trauma)
- Acute pulmonary embolism. See Pulmonary embolism
- Infections (including myocarditis)
- Tamponade.

> **Practical tip**
>
> You should initiate management of acute heart failure in tandem with investigation and treatment of underlying precipitants/causes. However, depending on the clinical setting and circumstances, addressing the underlying cause may take immediate priority: for example, if primary percutaneous coronary intervention is available and indicated for a patient with acute ST-elevation myocardial infarction, this should not be delayed by continued efforts to stabilise the patient.

Organise **rapid transfer to hospital** for any patient in the community with suspected acute heart failure.[1] Transfer to the most appropriate setting.[1]

Key Recommendations

Determine **acute drug treatment based on the patient's clinical presentation, including haemodynamic status** and the **presence of shock**; drug treatment options include vasoactive drugs, diuretics, and vasodilators.[1] [4] [23]

After stabilisation, start an **oral diuretic** if the patient has symptoms or signs of **congestion**, or **switch from an intravenous to an oral diuretic** once a patient who was started on an intravenous diuretic in the acute phase is **euvolaemic**.[1]

Plan subsequent treatment based on measurement of the patient's **left ventricular ejection fraction** (LVEF) using echocardiography and their **level of symptoms**.[1] [6]

- Start an **ACE inhibitor** (or an angiotensin-II receptor antagonist if unable to tolerate an ACE inhibitor) and a **beta-blocker** in patients with **reduced LVEF** (≤40%).[1] [4] If the patient is already taking a beta-blocker for a comorbidity (e.g., angina, hypertension), switch to a beta-blocker that is licensed for heart failure.[24]
- Start an **aldosterone antagonist** in addition to an ACE inhibitor (or angiotensin-II receptor antagonist) and a beta-blocker in patients with acute heart failure and reduced LVEF.[6] [23]
- **Sacubitril/valsartan** is recommended as a replacement for an ACE inhibitor in suitable patients with heart failure with reduced ejection fraction who remain symptomatic despite optimal treatment with an ACE inhibitor (or an angiotensin-II receptor antagonist), a beta-blocker, and an aldosterone antagonist.[1] Sacubitril/valsartan may also be considered as a first-line therapy instead of an ACE inhibitor.[1] Treatment with sacubitril/valsartan should be started by a heart failure specialist.[23]
- Aim to provide **symptomatic relief** (e.g., reducing symptoms of congestion with diuretics) and **improve general overall health and well-being** for any patient with **preserved LVEF** (>40%).[1]

Do not give oxygen routinely; it should be used only if the patient has oxygen saturations <90% or PaO_2 <8 kPa (<60 mmHg).[1]

Ensure the patient has **input from the heart failure specialist team** within 24 hours of admission to hospital.[29]

Full Recommendations

Treatment goals

The goals of initial treatment of the patient with acute heart failure are to:[1]

- Identify and treat any underlying cause
- Alleviate symptoms
- Improve congestion and organ perfusion
- Restore oxygenation
- Limit organ damage (cardiac, renal, hepatic, gut).

Subsequent treatment aims to:[1]

- Improve symptoms and quality of life

- Control symptoms and fully relieve congestion, prevent early readmission, and improve survival.

More info: Using the type of clinical presentation to guide management decisions

The European Society of Cardiology describes four major clinical presentations of acute heart failure. Although these presentations may overlap, each requires different treatment. This classification therefore offers a practical framework to help guide management decisions.[1]

Acute decompensated heart failure

- symptoms associated with peripheral fluid accumulation, increased intraventricular pressure
- gradual onset (days)
- normal or low systolic blood pressure
- **Treatment**: diuretics, vasoactive drugs if peripheral hypoperfusion/hypotension (an inotrope and/or a vasopressor), short-term mechanical ventilatory support or renal replacement therapy if needed.

Acute pulmonary oedema

- symptoms associated with fluid redistribution to the lungs and acute respiratory failure
- rapid onset (hours)
- normal to high systolic blood pressure
- **Treatment**: oxygen (given as continuous positive airway pressure, non-invasive positive pressure-ventilation, and/or high-flow nasal cannula), diuretics, vasodilators.

Isolated right ventricular failure

- symptoms from increased central venous pressure and often systemic hypoperfusion
- gradual or rapid onset
- low systolic blood pressure
- **Treatment**: diuretics for peripheral congestion, vasoactive drugs if peripheral hypoperfusion/hypotension (an inotrope and/or a vasopressor), short-term mechanical ventilatory support or renal replacement therapy if needed.

Cardiogenic shock

- symptoms from systemic hypoperfusion (severe cardiac dysfunction)
- gradual or rapid onset
- low systolic blood pressure
- **Treatment**: vasoactive drugs (an inotrope and/or a vasopressor), short-term mechanical ventilatory support, and/or renal replacement therapy, together with early identification and treatment of the underlying cause.

Initial supportive care

Request **urgent cardiology/critical care support** for any patient with:[1]

- Respiratory distress/failure[22]

- Reduced consciousness
- Use of accessory muscles for breathing, respiratory rate >25/minute[22]
- Oxygen saturation (SpO_2) <90% despite supplemental oxygen
- Heart rate <40 or >130 bpm[22]
- Systolic blood pressure persistently <90 mmHg[22]
    - Unless known to be usually hypotensive (based on the opinion of our expert adviser)
- Signs or symptoms of hypoperfusion (see Shock)[1]
- Haemodynamic instability
- Acute heart failure due to an acute coronary syndrome (ACS)[4]
- Persistent life-threatening arrhythmia.

Seek expert help on any use of intravenous fluids in patients with known underlying cardiac impairment such as heart failure.[30]

Monitor transcutaneous arterial oxygen saturation (SpO_2).[1]

- Give oxygen if the patient has oxygen saturations <90% or PaO_2 <8 kPa (<60 mmHg).[1]
- **Monitor** controlled oxygen therapy. An **upper SpO_2 limit of 96%** is reasonable when administering supplemental oxygen to most patients with acute illness who are **not at risk of hypercapnia**.
    - Evidence suggests that liberal use of supplemental oxygen (target SpO_2 >96%) in acutely ill adults is associated with higher mortality than more conservative oxygen therapy.[31]
    - A lower target SpO_2 of 88% to 92% is appropriate if the patient is at risk of hypercapnic respiratory failure.[32]
- Do not use oxygen routinely in non-hypoxaemic patients with acute heart failure because it causes vasoconstriction and a reduction in cardiac output.[1]

Consider **non-invasive positive pressure ventilation** (continuous positive airway pressure [CPAP], bilevel positive airway pressure [BiPAP]) in patients with respiratory distress (respiratory rate >25 breaths/minute, SpO_2 <90%); start as soon as possible to decrease respiratory distress and reduce the rate of mechanical endotracheal intubation. Use with caution in patients with hypotension, monitoring blood pressure regularly.[1]

Consider **invasive ventilation** if the patient has respiratory failure leading to hypoxaemia (PaO_2 <8 kPa [<60 mmHg]), hypercapnia ($PaCo_2$ >6.65 kPa [>50 mmHg]), and acidosis (pH <7.35) that cannot be managed non-invasively.[1]

Evidence: Target oxygen saturation in acutely ill adults

Too much supplemental oxygen increases mortality.

Evidence from a large systematic review and meta-analysis supports conservative/controlled oxygen therapy versus liberal oxygen therapy in acutely ill adults who are not at risk of hypercapnia.

- Guidelines differ in their recommendations on target oxygen saturation in acutely unwell adults who are receiving supplemental oxygen.
    - The 2017 British Thoracic Society (BTS) guideline recommends a target SpO_2 range of 94% to 98% for patients not at risk of hypercapnia, whereas the 2022 Thoracic Society of Australia and New Zealand (TSANZ) guideline recommends 92% to 96%.[32] [33]
    - The 2022 Global Initiative For Asthma (GINA) guidelines recommend a target SpO_2 range of 93% to 96% in the context of acute asthma exacerbations.[34]
- One systematic review including a meta-analysis of data from 25 randomised controlled trials, published in 2018, found that, in adults with acute illness, liberal oxygen therapy (broadly equivalent to a target saturation >96%) is associated with higher mortality than conservative oxygen therapy (broadly equivalent to a target saturation ≤96%).[31]
    - In-hospital mortality was 11 per 1000 higher for the liberal oxygen therapy versus conservative therapy group (95% CI 2 to 22 per 1000 more).
    - Mortality at 30 days was also higher in the group who had received liberal oxygen (RR 1.14, 95% CI 1.01 to 1.29).
    - The trials included adults with sepsis, critical illness, stroke, trauma, myocardial infarction, and cardiac arrest, and patients who had emergency surgery. Studies that were limited to people with chronic respiratory illness or psychiatric illness, and patients on extracorporeal life support, receiving hyperbaric oxygen therapy, or having elective surgery, were all excluded from the review.
- An upper SpO_2 limit of 96% is therefore reasonable when administering supplemental oxygen to medical patients with acute illness who are not at risk of hypercapnia. However, a higher target may be appropriate for some specific conditions (e.g., pneumothorax, carbon monoxide poisoning, cluster headache, and sickle cell crisis).[35]
- In 2019 the BTS reviewed its guidance in response to this systematic review and meta-analysis and decided an interim update was not required.[36]
    - The committee noted that the systematic review supported the use of controlled oxygen therapy to a target.
    - While the systematic review showed an association between higher oxygen saturations and higher mortality, the BTS committee felt the review was not definitive on what the optimal target range should be. The suggested range of 94% to 96% in the review was based on the lower 95% confidence interval and the median baseline SpO_2 from the liberal oxygen groups, along with the earlier 2015 TSANZ guideline recommendation.

- Subsequently, experience during the coronavirus disease 2019 (COVID-19) pandemic has also made clinicians more aware of the feasibility of permissive hypoxaemia.[37]
- Management of oxygen therapy in patients in intensive care is specialised and informed by further evidence (not covered in this summary) that is more specific to this setting.[38] [39] [40]

Urgently **identify and treat any underlying precipitants/causes of acute heart failure that must be managed immediately** to prevent further rapid deterioration (while recognising that any acute heart failure is potentially life-threatening). Causes to consider include:[1]

- ACS.[4] See Unstable angina , Non-ST elevation myocardial infarction , and ST-elevation myocardial infarction
- Hypertensive emergency. See Hypertensive emergencies
- Rapid arrhythmias or severe bradycardia/conduction disturbance (see Assessment of tachycardia and Bradycardia)
- An acute mechanical cause (e.g., myocardial rupture as a complication of ACS, acute valvular regurgitation, chest trauma)
- Acute pulmonary embolism. See Pulmonary embolism
- Infections (including myocarditis)
- Tamponade.

Practical tip

You should initiate management of acute heart failure in tandem with investigation and treatment of underlying precipitants/causes. However, depending on the clinical setting and circumstances, addressing the underlying cause may take immediate priority: for example, if primary percutaneous coronary intervention is available and indicated for a patient with acute ST-elevation myocardial infarction, this should not be delayed by continued efforts to stabilise the patient.

Organise **rapid transfer to hospital** for any patient in the community with suspected acute heart failure.[1] Transfer to the most appropriate setting.[1]

Ensure the patient has **input from the heart failure specialist team** within 24 hours of admission to hospital.[29]

Evidence: Specialist review

Access to a heart failure specialist during admission with acute heart failure improves prescription of disease-modifying heart failure treatment and reduces mortality rates both in hospital and post discharge.

The UK National Institute for Health and Care Excellence (NICE) guideline on acute heart failure recommends that "all people admitted to hospital with suspected acute heart failure have early and continuing input from a dedicated specialist heart failure team".[23]

- This is based on evidence of reduced mortality from six observational studies, of which data from the 2012 and 2013 National Institute for Cardiovascular Outcomes Research (NICOR) heart failure audits in England and Wales were felt to be the most relevant.
- 'Early' is defined as 24 hours in the linked NICE quality standard.[41]

The UK National Confidential Enquiry into Patient Outcome and Death (NCEPOD) in people who died following a hospital admission for acute heart failure found that specialist review could have been improved for 23.7% of the patients included.[4]

- The enquiry included all adult patients admitted as an emergency with a primary diagnosis of heart failure and who died in hospital (between 1 January 2016 and 31 December 2016); a subset of those who died within 7 days was used for more detailed analysis.
- At some point in their admission 197/585 patients (33.7%) were transferred to a specialist ward (cardiology, coronary care, or critical care).
- 199/603 patients (33.0%) were reviewed by a specialist heart failure team.
- 273/561 patients (48.7%) were reviewed by a cardiologist.
    - Cardiology review frequently resulted in changes to treatment (90/134; 67.2% of patients).
    - Where information on timing of cardiology review was available, 61 patients (37.7%) were reviewed within 12 hours of admission, 102 (63%) within 24 hours, and 136 (84%) within 48 hours.
        - 38/133 cardiology reviews (28.6%) assessed by NCEPOD peer reviewers were judged as not having taken place within an appropriate time frame.
- 52/218 patients (23.9%) who did not have any specialist review died within 24 hours of admission to hospital.
- Care was more likely to be rated as 'good' for those patients who had specialist review (53.8% vs. 12.4%).
    - People under the age of 80 and those with newly diagnosed heart failure were more likely to have specialist review.

The NICOR heart failure audit (England and Wales) has shown consistently that specialist review is associated with reduced inpatient mortality. Figures from the 2022 audit (based on 2020-2021 data) showed there is still room for improvement in specialist review of patients admitted with heart failure; this applies both during acute admission and post-discharge.[6]

- Approximately 88% of patients had a specialist review during hospital admission.
    - Overall, 53% saw a consultant cardiologist and 49% saw a specialist nurse.
    - Patients admitted to cardiology wards were more likely to see a specialist than those on general medical wards (99% vs. 74%).
    - There was huge variation, with only 65% of hospitals achieving review rates over 80%.
- The percentage of patients with heart failure with reduced ejection fraction being prescribed a combination of all three disease-modifying medicines (ACE inhibitors, beta-blockers, and aldosterone antagonists) at discharge was 52% irrespective of the ward setting and specialist review. This increased to 58% for patients managed on a cardiology ward and 55% for patients who had specialist review.
    - Over a 5-year period prescription rates improved for specialist review, while prescription rates were generally static for patients not undergoing specialist review.
- In-hospital mortality was 9.2% for all patients admitted to hospital.
    - Mortality was reduced for patients who were reviewed by a specialist or managed on a cardiology ward (7.9% and 6.0%, respectively).
    - Age-adjusted multivariable analyses showed that not being admitted to a cardiology ward (hazard ratio 1.75, P <0.001) was an independent predictor of increased mortality when other common markers of disease severity are included in the model.

Do not routinely offer opioids to a patient with acute heart failure.[23]

Acute drug treatment

Determine acute drug treatment based on the patient's haemodynamic status and the presence or absence of shock.[1] [4] [23]

Haemodynamically unstable: hypotensive (systolic blood pressure <90 mmHg) or other signs of cardiogenic shock

Get **urgent cardiology or critical care support**; treatment should be provided in a specialist environment.

- **Vasoactive drugs** (an inotrope and/or a vasopressor) should only be considered in patients with acute heart failure with potentially reversible cardiogenic shock or those who are potential candidates for a heart transplant. They should only be administered in a cardiac care unit or high-dependency unit or an alternative setting with at least level 2 care.[23] [42] Selection of appropriate vasoactive agents may vary according to clinician preference and local practice guidelines.
- **Short-term intravenous infusion of inotropic drugs** may be considered in this group of patients to increase cardiac output, increase blood pressure, improve peripheral perfusion, and maintain end-organ function.[1] This should be given in a specialist setting.
- Use of **short-term mechanical circulatory support devices** (e.g., intra-aortic balloon pumps, impella devices, short-term ventricular assist devices) may be considered by specialists.[43]

- For more information on the assessment and management of patients with cardiogenic shock, see Shock .

Haemodynamically unstable: hypertensive crisis

Consider giving a vasodilator intravenously if the patient has hypertension, in addition to usual care.[1] [23] This may also be used for relief of dyspnoea in this group of patients.[1] [4] [23]

- Monitor the patient's symptoms and blood pressure in a **critical care environment** to ensure systolic blood pressure remains >90 mmHg.[1] [23]
- Sodium nitroprusside may be given in clinical practice but the UK National Institute for Health and Care Excellence recommends that it **should not be given to patients with acute heart failure**.[23] However, it is approved for use in acute heart failure in the UK and it is suggested as an intravenous vasodilator option for acute heart failure by the European Society of Cardiology guidelines.[1] Monitor blood pressure (including intra-arterial blood pressure) and blood cyanide concentration.
- Although evidence for survival benefit from the use of vasodilators in patients with acute heart failure is lacking, they remain in widespread use for symptom relief and blood pressure control.[1] [23]

Evidence: Vasodilators

Guidelines recommend using vasodilators in selected patients with acute heart failure, but this is based on clinical experience and there is no evidence to support their use. Use of vasodilators is associated with an increased risk of adverse events: in particular, headache and hypotension.

Although vasodilators are commonly used in adults with acute heart failure, the UK National Institute for Health and Care Excellence (NICE) reviewed the evidence for their use in 2014 due to variation in practice both in the UK and across Europe. The reviewers identified five relevant randomised controlled trials (RCTs) (n=1369).[23]

- The interventions were: intravenous glyceryl trinitrate (two RCTs, n=529), oral isosorbide dinitrate (two RCTs, n=28), and intravenous sodium nitroprusside (one RCT, n=812). All were compared with placebo.
- Only the study with sodium nitroprusside reported mortality as an outcome.
    - For men with acute left ventricular failure and presumed myocardial infarction there was no difference in all-cause mortality at 48 hours, 21 days, or 13 weeks with sodium nitroprusside compared with placebo (n=812, very low-quality evidence assessed using GRADE).
- Haemodynamic outcomes were reported as favourable for all interventions (four studies); however, as it was unclear whether/how these relate to longer-term clinical benefit, they were not used by NICE to formulate its recommendations.
- There was no difference in global symptomatic improvement or patient-reported dyspnoea with glyceryl trinitrate compared with placebo (follow-up 3 hours, GRADE moderate to low).
- Two studies reported adverse events, of which headache and hypotension were considered the most important.
    - More people had headache with glyceryl trinitrate compared with placebo (follow-up 3 hours, risk ratio [RR] 5.63, 95% CI 1.69 to 18.78; GRADE moderate).[23] In the first 24 hours after administration headache occurred in 44 people (20%) and hypotension occurred in 27 people (13%), although only one person had severe hypotension.[44] Hypotension was not reported for the placebo group; therefore, NICE did not report this outcome.
    - With sodium nitroprusside, significantly more patients reached the hypotensive limit compared with placebo (RR 26.87, 95% CI 6.59 to 109.46; absolute effect 128 more per 1000 [from 28 more to 536 more], GRADE low). Headache and severe headache were also more common in the sodium nitroprusside group (GRADE low to very low).
- While there was limited evidence of any benefit, the guideline group noted that, based on its clinical experience, nitrates may help some patients: for example, those with myocardial ischaemia or severe hypertension.

Key evidence since the 2014 NICE guideline#evidence review

Two subsequent RCTs, the Goal-directed Afterload Reduction in Acute Congestive Cardiac Decompensation (GALACTIC) study (published 2019, n=788) and the Effect of an Emergency Department Care Bundle on 30-Day Hospital Discharge and Survival Among Elderly Patients With Acute Heart Failure (ELISABETH) trial (published 2020, n=503), compared interventions that included early use of vasodilation with usual care without early vasodilation.

- Neither study demonstrated a survival/hospitalisation benefit from early use of intravenous vasodilators compared with usual care (including high-dose diuretics).[45] [46]
- Consequently, the European Society of Cardiology did not issue any recommendation for a regimen favouring vasodilator-based treatment versus usual care in its 2021 guideline for the diagnosis and treatment of acute and chronic heart failure.[1]

Give an **intravenous loop diuretic to all patients** with acute heart failure including those with severe hypertension.[23]

- If the patient is **already on long-term diuretic therapy**, give an initial intravenous dose that is at least equal to the pre-existing oral dose (some experts recommend approximately twice the equivalent oral dose) unless you have significant concerns about the patient's adherence to their diuretic therapy before admission.[1] [23] [47]
- Give the diuretic as either intermittent boluses or a continuous infusion.[1] [23]
- Adjust the dose according to the patient's symptoms and clinical status.[1]
- Closely monitor the patient's weight, renal function, and urine output while they are taking diuretics.[1] [23]
- Discuss with the patient the best strategies of coping with an increased urine output.[23]

Practical tip

Avoid excessive diuresis; this is more dangerous than oedema.[1]

Consider adding a **thiazide-type diuretic** or an **aldosterone antagonist** if the patient has resistant oedema or symptoms or signs of congestion despite treatment with a loop diuretic.[1]

- Carefully monitor the patient for hypokalaemia or hyperkalaemia, renal impairment, and hypovolaemia.[1]

Haemodynamically stable: normal blood pressure

Give an **intravenous loop diuretic** to a haemodynamically stable patient if there are **symptoms or signs of congestion**.[1]

- If the patient is **already on long-term diuretic therapy**, give an initial intravenous dose that is at least equal to the pre-existing oral dose (some experts recommend approximately twice the equivalent oral dose) unless you have significant concerns about the patient's adherence to their diuretic therapy before admission.[1] [23]
- [1]Give the diuretic as either intermittent boluses or a continuous infusion.[23]
- Adjust the dose according to the patient's symptoms and clinical status.[1]
- Monitor the patient's weight, renal function, and urine output carefully while they are taking diuretics.[1] [23]

- Aim to achieve positive diuresis with a reduction of body weight by 0.75 to 1.0 kg/day.[1]

Practical tip

Avoid excessive diuresis; this is more dangerous than oedema.[1]

Consider adding an **aldosterone antagonist** or a **thiazide-type diuretic** if the patient has resistant oedema or symptoms or signs of congestion despite treatment with a loop diuretic.[1]

- Carefully monitor the patient for hypokalaemia or hyperkalaemia, renal impairment, and hypovolaemia.[1]

Do not give intravenous vasodilators routinely in patients with normal blood pressure. Consider them in specific circumstances: for example, for concomitant myocardial ischaemia or aortic/mitral regurgitation.[1] [4] [23]

- If vasodilators are given, monitor the patient's symptoms and blood pressure in a critical care environment to ensure systolic blood pressure remains >90 mmHg.[1] [23]

Evidence: Vasodilators

Guidelines recommend using vasodilators in selected patients with acute heart failure, but this is based on clinical experience and there is no evidence to support their use. Use of vasodilators is associated with an increased risk of adverse events: in particular, headache and hypotension.

Although vasodilators are commonly used in adults with acute heart failure, the UK National Institute for Health and Care Excellence (NICE) reviewed the evidence for their use in 2014 due to variation in practice both in the UK and across Europe. The reviewers identified five relevant randomised controlled trials (RCTs) (n=1369).[23]

- The interventions were: intravenous glyceryl trinitrate (two RCTs, n=529), oral isosorbide dinitrate (two RCTs, n=28), and intravenous sodium nitroprusside (one RCT, n=812). All were compared with placebo.
- Only the study with sodium nitroprusside reported mortality as an outcome.
    - For men with acute left ventricular failure and presumed myocardial infarction there was no difference in all-cause mortality at 48 hours, 21 days, or 13 weeks with sodium nitroprusside compared with placebo (n=812, very low-quality evidence assessed using GRADE).
- Haemodynamic outcomes were reported as favourable for all interventions (four studies); however, as it was unclear whether/how these relate to longer-term clinical benefit, they were not used by NICE to formulate its recommendations.
- There was no difference in global symptomatic improvement or patient-reported dyspnoea with glyceryl trinitrate compared with placebo (follow-up 3 hours, GRADE moderate to low).
- Two studies reported adverse events, of which headache and hypotension were considered the most important.
    - More people had headache with glyceryl trinitrate compared with placebo (follow-up 3 hours, risk ratio [RR] 5.63, 95% CI 1.69 to 18.78; GRADE moderate).[23] In the first 24 hours after administration headache occurred in 44 people (20%) and hypotension occurred in 27 people (13%), although only one person had severe hypotension.[44] Hypotension was not reported for the placebo group; therefore, NICE did not report this outcome.
    - With sodium nitroprusside, significantly more patients reached the hypotensive limit compared with placebo (RR 26.87, 95% CI 6.59 to 109.46; absolute effect 128 more per 1000 [from 28 more to 536 more], GRADE low). Headache and severe headache were also more common in the sodium nitroprusside group (GRADE low to very low).
- While there was limited evidence of any benefit, the guideline group noted that, based on its clinical experience, nitrates may help some patients: for example, those with myocardial ischaemia or severe hypertension.

Key evidence since the 2014 NICE guideline#evidence review

Two subsequent RCTs, the Goal-directed Afterload Reduction in Acute Congestive Cardiac Decompensation (GALACTIC) study (published 2019, n=788) and the Effect of an Emergency Department Care Bundle on 30-Day Hospital Discharge and Survival Among Elderly Patients With Acute Heart Failure (ELISABETH) trial (published 2020, n=503), compared interventions that included early use of vasodilation with usual care without early vasodilation.

- Neither study demonstrated a survival/hospitalisation benefit from early use of intravenous vasodilators compared with usual care (including high-dose diuretics).[45] [46]
- Consequently, the European Society of Cardiology did not issue any recommendation for a regimen favouring vasodilator-based treatment versus usual care in its 2021 guideline for the diagnosis and treatment of acute and chronic heart failure.[1]

Continue a beta-blocker if the patient is already taking this, unless they have:[23]

- Heart rate <50 bpm
- Second- or third-degree atrioventricular block
- Shock.

Treatment after stabilisation

Start an **oral diuretic** if the patient has symptoms or signs of **congestion**, or **switch from an intravenous to an oral diuretic** once a patient who was started on an intravenous diuretic in the acute phase is **euvolaemic**.[1]

- Most patients will require a loop diuretic due to severe symptoms of congestion and worsening renal function. Use a combination of a loop and a thiazide-type diuretic if the patient has resistant oedema.[1]
- Adjust the dose according to the patient's symptoms and clinical status.[1]
- Monitor the patient's weight, renal function, and urine output carefully while they are taking a diuretic.[1] [23]
    - Aim to achieve positive diuresis with a reduction of body weight by 0.75 to 1.0 kg/day.[1]

Practical tip

Avoid excessive diuresis; this is more dangerous than oedema.[1] In practice, convert the patient from an intravenous to an oral diuretic when there is significant reduction in peripheral oedema (i.e., oedema to ankles only).

Heart failure with reduced ejection fraction (LVEF ≤40%)

Start an **ACE inhibitor** (or an angiotensin-II receptor antagonist if unable to tolerate an ACE inhibitor) and a **beta-blocker**.[1] [4] [6] [23]

- In general, **start with low doses** and **titrate upwards** to maximally tolerated doses, taking into account any contraindications.[1]
- If the patient is already taking a beta-blocker for a comorbidity (e.g., angina, hypertension), switch to a beta-blocker that is licensed for heart failure.[24]

- Make sure the patient has remained stable for at least 48 hours after starting or restarting a beta-blocker before they are discharged.[23]

Give an **aldosterone antagonist** in addition to an ACE inhibitor (or an angiotensin-II receptor antagonist if unable to tolerate an ACE inhibitor) and a beta-blocker.[23]

Sacubitril/valsartan is recommended as a replacement for an ACE inhibitor in suitable patients with heart failure with reduced ejection fraction who remain symptomatic despite optimal treatment with an ACE inhibitor, a beta-blocker, and an aldosterone antagonist; however, it may also be considered for first-line therapy instead of an ACE inhibitor.[1]

- Treatment with sacubitril/valsartan should be started by a heart failure specialist.[24]

Start a **sodium-glucose co-transporter 2 (SGLT2) inhibitor** (dapagliflozin or empagliflozin) in patients with heart failure with reduced ejection fraction in addition to an ACE inhibitor (or angiotensin-II receptor antagonist), a beta-blocker, and an aldosterone antagonist, regardless of whether they have diabetes or not (unless contraindicated or not tolerated).[1]

Early involvement of the specialist heart failure team for all patients admitted with acute heart failure enables consideration of additional treatments to optimise outcomes.[6] For patients with heart failure with reduced ejection fraction these may include:[1] [24]

- Ivabradine
- Isosorbide dinitrate plus hydralazine
- Digoxin
- Cardiac resynchronisation therapy
- Implantable cardioverter defibrillator
- Transplantation or mechanical circulatory support device.

Heart failure with mildly reduced ejection fraction (LVEF 41% to 49%)

Formulate an individualised care plan for patients with mildly reduced ejection fraction (HFmrEF), which should depend on clinical characteristics, risk factors, patterns of cardiac remodelling, and the patient's comorbidities and prognosis.

- Offer symptomatic relief and measures to improve general overall health and well-being. This should include screening for and treating any comorbidities.
- Consider therapies recommended for patients with HFrEF (e.g., ACE inhibitor, angiotensin-II receptor antagonist, beta-blocker, aldosterone antagonist, sacubitril/valsartan).[1] Treatment with a SGLT2 inhibitor may also be beneficial for this group, regardless of the presence of diabetes.[48]

Heart failure with preserved ejection fraction (LVEF ≥50%)

Aim to provide symptomatic relief (e.g., by starting a diuretic) and improve general overall health by identifying and treating the underlying risk factors, aetiology, and coexisting comorbidities.[1] [2]

- These patients tend to be older with more comorbidities (cardiovascular and non-cardiovascular) compared with patients with HFrEF.[1]
- The European Society of Cardiology states that no specific treatment has been shown to convincingly reduce morbidity or mortality; however, one subsequent randomised controlled trial suggests that these patients may benefit from a SGLT2 inhibitor (empagliflozin), regardless of the presence of diabetes.[48]

Discharge

Consider discharging the patient if:

- They have an up-to-date echocardiogram (in practice, within the last year if considered unnecessary during this hospital visit)
- They have been reviewed by the heart failure specialist team[6] [29]
- They are stable and euvolaemic (persistent congestion before discharge is associated with a higher risk of readmission and mortality)[1]
- They have been established on recommended oral medication[1]
- Their condition has been stable for typically 48 hours after starting or restarting beta-blockers.[23]

Ensure the patient has the following before discharge:

- A follow-up appointment with a member of the multidisciplinary heart failure team within 2 weeks[4] [6] [23]
- Offer of referral to cardiac rehabilitation.[4] [24] Cardiac rehabilitation should be personalised and exercise-based. It should also address psychological and educational aspects.[4] [24]

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute (summary)		
haemodynamically unstable: hypotensive (systolic BP <90 mmHg) or other signs of cardiogenic shock		
	1st	**treatment of underlying cause**
	consider	**vasoactive drug**
	consider	**respiratory support**
	consider	**mechanical support device**
	plus	**referral to specialist**
haemodynamically unstable: hypertensive crisis		
	1st	**treatment of underlying cause**
	consider	**vasodilator**
	plus	**loop diuretic**
	consider	**aldosterone antagonist or thiazide-type diuretic**
	consider	**respiratory support**
	plus	**referral to specialist**
haemodynamically stable		
	1st	**treatment of underlying cause**
	plus	**loop diuretic**
	consider	**vasodilator**
	consider	**aldosterone antagonist or thiazide-type diuretic**
	consider	**continue beta-blocker**
	consider	**respiratory support**
	plus	**referral to specialist**

Ongoing		(summary)
acute episode stabilised: LVEF ≤40%		
	1st	**ACE inhibitor or angiotensin-II receptor antagonist or sacubitril/valsartan**
	plus	**beta-blocker**
	plus	**aldosterone antagonist**
	plus	**sodium-glucose co-transporter 2 (SGLT2) inhibitor**
	consider	**diuretic**
	plus	**referral to specialist**
	plus	**cardiac rehabilitation**
acute episode stabilised: LVEF 41% to 49%		
	1st	**referral to specialist**
	plus	**cardiac rehabilitation**
	consider	**diuretic**
acute episode stabilised: LVEF ≥50%		
	1st	**referral to specialist**
	plus	**cardiac rehabilitation**
	consider	**diuretic**

Treatment algorithm

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute

haemodynamically unstable: hypotensive (systolic BP <90 mmHg) or other signs of cardiogenic shock

1st **treatment of underlying cause**

» Urgently identify and treat any underlying precipitants/causes of acute heart failure that must be managed immediately to prevent further rapid deterioration (while recognising that any acute heart failure is potentially life-threatening). Causes to consider include:[1]

- Acute coronary syndrome (ACS).[4] See Unstable angina , Non-ST elevation myocardial infarction , and ST-elevation myocardial infarction
- Rapid arrhythmias or severe bradycardia/ conduction disturbance. See Assessment of tachycardia and Bradycardia
- An acute mechanical cause (e.g., myocardial rupture as a complication of ACS, acute valvular regurgitation, chest trauma)
- Acute pulmonary embolism. See Pulmonary embolism
- Infections (including myocarditis)
- Tamponade.

Practical tip

You should initiate management of acute heart failure in tandem with investigation and treatment of underlying precipitants/ causes. However, depending on the clinical setting and circumstances, addressing the underlying cause may take immediate priority: for example, if primary percutaneous coronary intervention (PCI) is available and indicated for a patient with acute ST-elevation myocardial infarction, this should not be delayed by continued efforts to stabilise the patient.

consider **vasoactive drug**

Treatment recommended for SOME patients in selected patient group

» **Vasoactive drugs** (an inotrope and/or a vasopressor) should be considered in patients

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with acute heart failure with potentially reversible cardiogenic shock or those who are potential candidates for a heart transplant. They should **only be administered in a cardiac care unit or high-dependency unit or an alternative setting with at least level 2 care**.[23] [42] Selection of appropriate vasoactive agents may vary according to clinician preference and local practice guidelines. For more information on the assessment and management of patients with cardiogenic shock, see Shock .

» **Short-term intravenous infusion of inotropic drugs** may be considered in patients with hypotension (systolic blood pressure <90 mmHg) and/or signs/symptoms of hypoperfusion despite adequate filling status, to increase cardiac output, increase blood pressure, improve peripheral perfusion, and maintain end-organ function. **This should be given in a specialist setting.** [1]

consider **respiratory support**

Treatment recommended for SOME patients in selected patient group

» Give **oxygen** if the patient has oxygen saturations <90% or PaO_2 <8 kPa (<60 mmHg). [1]

- **Monitor controlled oxygen therapy. An upper SpO_2 limit of 96%** is reasonable when administering supplemental oxygen to most patients with acute illness who are **not at risk of hypercapnia**.
- Evidence suggests that liberal use of supplemental oxygen (target SpO_2 >96%) in acutely ill adults is associated with higher mortality than more conservative oxygen therapy.[31]
- A lower target SpO_2 of 88% to 92% is appropriate if the patient is at risk of hypercapnic respiratory failure.[32]
- **Do not use oxygen routinely in non-hypoxaemic patients** with acute heart failure because it causes vasoconstriction and a reduction in cardiac output.[1]

» Consider **non-invasive positive pressure ventilation** (continuous positive airway pressure [CPAP], bilevel positive airway pressure [BiPAP]) in patients with respiratory distress (respiratory rate >25 breaths/minute, SpO_2 <90%); start as soon as possible to decrease respiratory distress and reduce the rate of mechanical endotracheal intubation. Use with

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caution in patients with hypotension, monitoring blood pressure regularly.[1]

» Consider **invasive ventilation** if the patient has respiratory failure leading to hypoxaemia (PaO_2 <8 kPa [<60 mmHg]), hypercapnia ($PaCo_2$ >6.65 kPa [>50 mmHg]), and acidosis (pH <7.35) that cannot be managed non-invasively.[1]

consider **mechanical support device**

Treatment recommended for SOME patients in selected patient group

» Use of short-term mechanical circulatory support devices (e.g., intra-aortic balloon pumps, impella devices, short-term ventricular assist devices) may be **considered by specialists**.[43]

plus **referral to specialist**

Treatment recommended for ALL patients in selected patient group

» Ensure the patient has input from the heart failure specialist team within 24 hours of admission to hospital.[29]

haemodynamically unstable: hypertensive crisis

1st **treatment of underlying cause**

» Urgently identify and treat any underlying precipitants/causes of acute heart failure that must be managed immediately to prevent further rapid deterioration (while recognising that any acute heart failure is potentially life-threatening) Causes to consider include:[1]

- Acute coronary syndrome (ACS).[4] See Unstable angina , Non-ST elevation myocardial infarction , and ST-elevation myocardial infarction
- Hypertensive emergency. See Hypertensive emergencies
- Rapid arrhythmias or severe bradycardia/ conduction disturbance. See Assessment of tachycardia and Bradycardia
- An acute mechanical cause (e.g., myocardial rupture as a complication of ACS, acute valvular regurgitation, chest trauma)
- Acute pulmonary embolism. See Pulmonary embolism.

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Practical tip

You should initiate management of acute heart failure in tandem with investigation and treatment of underlying precipitants/causes. However, depending on the clinical setting and circumstances, addressing the underlying cause may take immediate priority: for example, if primary percutaneous coronary intervention (PCI) is available and indicated for a patient with acute ST-elevation myocardial infarction, this should not be delayed by continued efforts to stabilise the patient.

consider **vasodilator**

Treatment recommended for SOME patients in selected patient group

Primary options

» glyceryl trinitrate: 10 micrograms/minute intravenous infusion initially, adjust dose according to response, maximum 400 micrograms/minute

OR

» isosorbide dinitrate: 2-10 mg/hour intravenous infusion initially, adjust dose according to response, maximum 20 mg/hour

Secondary options

» sodium nitroprusside: consult specialist for guidance on dose

» Consider giving a vasodilator intravenously if there is hypertension.[1] [23] This may also be used for relief of dyspnoea in this group of patients.[1] [23]

- Monitor the patient's symptoms and blood pressure in a **critical care environment** to ensure systolic blood pressure remains >90 mmHg.[1]
- Sodium nitroprusside may be given in clinical practice but the UK National Institute for Health and Care Excellence recommends that it **should not be given to patients with acute heart failure**.[23] However, it is approved for use in acute heart failure in the UK and it is suggested as an intravenous vasodilator option for acute heart failure by the European Society of Cardiology guidelines.[1] Monitor blood pressure

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(including intra-arterial blood pressure) and blood cyanide concentration.
- Although evidence for survival benefit from the use of vasodilators in patients with acute heart failure is lacking, they remain in widespread use for symptom relief and blood pressure control.[1] [23]

plus **loop diuretic**

Treatment recommended for ALL patients in selected patient group

Primary options

» furosemide: 20-50 mg intravenously initially, increase by 20 mg every 2 hours if required according to response, maximum 1500 mg/day
Doses greater than 50 mg should be given by intravenous infusion only.

» Give an intravenous loop diuretic to all patients with acute heart failure and severe hypertension.[23]

- If the patient is **already on long-term diuretic therapy**, give an initial intravenous dose that is at least equal to the pre-existing oral dose (some experts recommend approximately twice the equivalent oral dose) unless you have significant concerns about the patient's adherence to their diuretic therapy before admission.[1] [23]
- Give the diuretic as either intermittent boluses or a continuous infusion.[1] [23]
- Adjust the dose according to the patient's symptoms and clinical status.[1]
- Closely monitor the patient's weight, renal function, and urine output while they are taking diuretics.[1] [23]
- Discuss with the patient the best strategies of coping with an increased urine output.[23]

consider **aldosterone antagonist or thiazide-type diuretic**

Treatment recommended for SOME patients in selected patient group

Primary options

» spironolactone: 25 mg orally once daily initially, increase gradually according to response, maximum 50 mg/day

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Doses of up to 200 mg/day may be required in congestive heart failure.

OR

» eplerenone: 25 mg orally once daily initially, increase gradually according to response, maximum 50 mg/day

OR

» metolazone: 5-10 mg orally once daily initially, increase gradually according to response, maximum 80 mg/day

» Consider adding an aldosterone antagonist (e.g., spironolactone, eplerenone) or a thiazide-type diuretic (e.g., metolazone) if the patient has **resistant oedema** or **symptoms or signs of congestion** despite treatment with a loop diuretic.[1]

- Carefully monitor the patient for hypokalaemia or hyperkalaemia, renal impairment, and hypovolaemia.[1]

consider **respiratory support**

Treatment recommended for SOME patients in selected patient group

» Give **oxygen** if the patient has oxygen saturations <90% or PaO_2 <8 kPa (<60 mmHg).[1]

- **Monitor controlled oxygen therapy. An upper SpO_2 limit of 96%** is reasonable when administering supplemental oxygen to most patients with acute illness who are **not at risk of hypercapnia**.
- Evidence suggests that liberal use of supplemental oxygen (target SpO_2 >96%) in acutely ill adults is associated with higher mortality than more conservative oxygen therapy.[31]
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- **Do not use oxygen routinely in non-hypoxaemic patients** with acute heart failure because it causes vasoconstriction and a reduction in cardiac output.[1]

» Consider **non-invasive positive pressure ventilation** (continuous positive airway

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pressure [CPAP], bilevel positive airway pressure [BiPAP]) in patients with respiratory distress (respiratory rate >25 breaths/minute, SpO_2 <90%); start as soon as possible to decrease respiratory distress and reduce the rate of mechanical endotracheal intubation.[1]

» Consider **invasive ventilation** if the patient has respiratory failure leading to hypoxaemia (PaO_2 <8 kPa [<60 mmHg]), hypercapnia ($PaCo_2$ >6.65 kPa [>50 mmHg]), and acidosis (pH <7.35) that cannot be managed non-invasively.[1]

plus **referral to specialist**

Treatment recommended for ALL patients in selected patient group

» Ensure the patient has input from the heart failure specialist team within 24 hours of admission to hospital.[29]

haemodynamically stable

1st **treatment of underlying cause**

» Urgently identify and treat any underlying precipitants/causes of acute heart failure that must be managed immediately to prevent further rapid deterioration (while recognising that any acute heart failure is potentially life-threatening). Causes to consider include:[1]

- Acute coronary syndrome (ACS).[4] See Unstable angina , *Non-ST elevation myocardial infarction* , and *ST-elevation myocardial infarction*
- Hypertensive emergency. See Hypertensive emergencies
- Rapid arrhythmias or severe bradycardia/ conduction disturbance. See Assessment of tachycardia and Bradycardia
- An acute mechanical cause (e.g., myocardial rupture as a complication of ACS, acute valvular regurgitation, chest trauma)
- Acute pulmonary embolism. See Pulmonary embolism
- Infection (including myocarditis)
- Tamponade.

Acute

Practical tip

You should initiate management of acute heart failure in tandem with investigation and treatment of underlying precipitants/causes. However, depending on the clinical setting and circumstances, addressing the underlying cause may take immediate priority: for example, if primary percutaneous coronary intervention (PCI) is available and indicated for a patient with acute ST-elevation myocardial infarction, this should not be delayed by continued efforts to stabilise the patient.

plus **loop diuretic**

Treatment recommended for ALL patients in selected patient group

Primary options

» furosemide: 20-50 mg intravenously initially, increase by 20 mg every 2 hours if required according to response, maximum 1500 mg/day
Doses greater than 50 mg should be given by intravenous infusion only.

» Give an intravenous loop diuretic if there are **symptoms or signs of congestion**.[1]

- If the patient is **already on long-term diuretic therapy**, give an initial intravenous dose that is at least equal to the pre-existing oral dose (some experts recommend approximately twice the equivalent oral dose) unless you have significant concerns about the patient's adherence to their diuretic therapy before admission.[1] [23]
- Give the diuretic as either intermittent boluses or a continuous infusion.[1] [23]
- Adjust the dose according to the patient's symptoms and clinical status.[1]
- Monitor the patient's weight, renal function, and urine output carefully while they are taking diuretics.[1] [23]
    - Aim to achieve positive diuresis with a reduction of body weight by 0.75 to 1.0 kg/day.[1]

consider **vasodilator**

Treatment recommended for SOME patients in selected patient group

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Primary options

» glyceryl trinitrate: 10 micrograms/minute intravenous infusion initially, adjust dose according to response, maximum 400 micrograms/minute

OR

» isosorbide dinitrate: 2-10 mg/hour intravenous infusion initially, adjust dose according to response, maximum 20 mg/hour

Secondary options

» sodium nitroprusside: consult specialist for guidance on dose

» **Do not give intravenous vasodilators routinely** in patients with normal blood pressure. Consider them in specific circumstances: for example, for concomitant myocardial ischaemia or aortic/mitral regurgitation.[1] [4] [23]

- If vasodilators are given, monitor the patient's symptoms and blood pressure in a **critical care environment** to ensure systolic blood pressure remains >90 mmHg.[1] [23]
- Sodium nitroprusside may be given in clinical practice but the UK National Institute for Health and Care Excellence recommends that it **should not be given to patients with acute heart failure**.[23] However, it is approved for use in acute heart failure in the UK and it is suggested as an intravenous vasodilator option for acute heart failure by the European Society of Cardiology guidelines.[1] Monitor blood pressure (including intra-arterial blood pressure) and blood cyanide concentration.
- Although evidence for survival benefit from the use of vasodilators in patients with acute heart failure is lacking, they remain in widespread use for symptom relief and blood pressure control.[1] [23]

consider **aldosterone antagonist or thiazide-type diuretic**

Treatment recommended for SOME patients in selected patient group

Primary options

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» spironolactone: 25 mg orally once daily initially, increase gradually according to response, maximum 50 mg/day
Doses of up to 200 mg/day may be required in congestive heart failure.

OR

» eplerenone: 25 mg orally once daily initially, increase gradually according to response, maximum 50 mg/day

OR

» metolazone: 5-10 mg orally once daily initially, increase gradually according to response, maximum 80 mg/day

» Consider adding an aldosterone antagonist (e.g., spironolactone, eplerenone) or a thiazide-type diuretic (e.g., metolazone) if the patient has **resistant oedema** or **symptoms or signs of congestion** despite treatment with a loop diuretic.[1]

- Carefully monitor the patient for hypokalaemia or hyperkalaemia, renal impairment, and hypovolaemia.[1]

consider **continue beta-blocker**

Treatment recommended for SOME patients in selected patient group

» **Continue a beta-blocker** if the patient is already taking this, unless they have:[23]

- Heart rate <50 bpm
- Second- or third-degree atrioventricular block
- Shock.

consider **respiratory support**

Treatment recommended for SOME patients in selected patient group

» Give **oxygen** if the patient has oxygen saturations <90% or PaO_2 <8 kPa (<60 mmHg).[1]

- **Monitor controlled oxygen therapy. An upper SpO_2 limit of 96%** is reasonable when administering supplemental oxygen to most patients

MANAGEMENT

Acute

with acute illness who are **not at risk of hypercapnia**.

- Evidence suggests that liberal use of supplemental oxygen (target SpO_2 >96%) in acutely ill adults is associated with higher mortality than more conservative oxygen therapy.[31]
- A lower target SpO_2 of 88% to 92% is appropriate if the patient is at risk of hypercapnic respiratory failure.[32]
- **Do not use oxygen routinely in non-hypoxaemic patients** with acute heart failure because it causes vasoconstriction and a reduction in cardiac output.[1]

» Consider **non-invasive positive pressure ventilation** (continuous positive airway pressure [CPAP], bilevel positive airway pressure [BiPAP]) in patients with respiratory distress (respiratory rate >25 breaths/minute, SpO_2 <90%); start as soon as possible to decrease respiratory distress and reduce the rate of mechanical endotracheal intubation.[1]

» Consider **invasive ventilation** if the patient has respiratory failure leading to hypoxaemia (PaO_2 <8 kPa [<60 mmHg]), hypercapnia ($PaCo_2$ >6.65 kPa [>50 mmHg]), and acidosis (pH <7.35) that cannot be managed non-invasively.[1]

plus **referral to specialist**

Treatment recommended for ALL patients in selected patient group

» Ensure the patient has input from the heart failure specialist team within 24 hours of admission to hospital.[29]

Ongoing

acute episode stabilised: LVEF ≤40%

1st **ACE inhibitor or angiotensin-II receptor antagonist or sacubitril/valsartan**

Primary options

» lisinopril: 2.5 mg orally once daily initially, increase gradually according to response, maximum 35 mg/day

OR

» ramipril: 1.25 mg orally once daily initially, increase gradually according to response, maximum 10 mg/day given in 1-2 divided doses

OR

» enalapril: 2.5 mg orally once daily initially, increase gradually according to response, maximum 40 mg/day given in 2 divided doses

Secondary options

» candesartan: 4 mg orally once daily initially, increase gradually according to response, maximum 32 mg/day

OR

» losartan: 12.5 mg orally once daily initially, increase gradually according to response, maximum 150 mg/day

OR

» valsartan: 40 mg orally twice daily initially, increase gradually according to response, maximum 320 mg/day

Tertiary options

» sacubitril/valsartan: dose depends on whether patient is currently stabilised on an ACE inhibitor (or angiotensin-II receptor antagonist); consult specialist for guidance on dose

» Start the patient on an ACE inhibitor (or an angiotensin-II receptor antagonist if an ACE inhibitor is not tolerated).[4] [6] [23] In general, **start with low doses** and **titrate upwards** up to maximally tolerated doses after taking into account any contraindications.[1]

MANAGEMENT

Ongoing

» **Sacubitril/valsartan** is recommended as a replacement for an ACE inhibitor (or angiotensin-II receptor antagonist) in ambulatory patients with heart failure with reduced ejection fraction who remain symptomatic despite optimal treatment with an ACE inhibitor (or an angiotensin-II receptor antagonist), a beta-blocker, and an aldosterone antagonist; however, it may also be considered for first-line therapy instead of an ACE inhibitor.[1] Treatment with sacubitril/valsartan should be started by a heart failure specialist.[24]

plus **beta-blocker**

Treatment recommended for ALL patients in selected patient group

Primary options

» bisoprolol: 1.25 mg orally once daily initially for 1 week, increase gradually according to response, maximum 10 mg/day

OR

» carvedilol: 3.125 mg orally twice daily initially, increase gradually according to response, maximum 50 mg/day (body weight <85 kg) or 100 mg/day (body weight >85 kg)

OR

» nebivolol: 1.25 mg orally once daily initially, increase gradually according to response, maximum 10 mg/day

» Start a beta-blocker up to maximally tolerated doses, once the patient's condition has been stabilised.[6] [23]

» If the patient is already taking a beta-blocker for a comorbidity (e.g., angina, hypertension), switch to a beta-blocker that is licensed for heart failure.[24]

» In general, **start with low doses** and **titrate upwards**.[1]

» Make sure the patient has remained stable for at least 48 hours after starting or restarting a beta-blocker before they are discharged.[23]

plus **aldosterone antagonist**

Treatment recommended for ALL patients in selected patient group

Primary options

Ongoing

» spironolactone: 25 mg orally once daily initially, increase gradually according to response, maximum 50 mg/day
Doses of up to 200 mg/day may be required in congestive heart failure.

OR

» eplerenone: 25 mg orally once daily initially, increase gradually according to response, maximum 50 mg/day

» Give an aldosterone antagonist in addition to an ACE inhibitor and a beta-blocker in patients with acute heart failure and reduced left ventricular ejection fraction.[6] [23]

» In general, **start with low doses** and **titrate upwards** to maximally tolerated doses after taking into account any contraindications.[1] [4] [23]

plus **sodium-glucose co-transporter 2 (SGLT2) inhibitor**

Treatment recommended for ALL patients in selected patient group

Primary options

» dapagliflozin: 10 mg orally once daily

OR

» empagliflozin: 10 mg orally once daily

» Start an **SGLT2 inhibitor** (dapagliflozin or empagliflozin) in addition to other therapies, regardless of whether the patient has diabetes or not (unless contraindicated or not tolerated).[1]

consider **diuretic**

Treatment recommended for SOME patients in selected patient group

Primary options

» furosemide: 40 mg orally once daily initially, titrate gradually according to response, maximum 120 mg/day

OR

» torasemide: 5 mg orally once daily initially, titrate gradually according to response, maximum 40 mg/day

OR

MANAGEMENT

Ongoing

» furosemide: 40 mg orally once daily initially, titrate gradually according to response, maximum 120 mg/day

-or-

» torasemide: 5 mg orally once daily initially, titrate gradually according to response, maximum 40 mg/day

--AND--

» metolazone: 5-10 mg orally once daily initially, increase gradually according to response, maximum 80 mg/day

» Start an oral diuretic if the patient has **symptoms or signs of congestion**, or switch from an intravenous to an oral diuretic once a patient who was started on an intravenous diuretic in the acute phase is **euvolaemic**.[1]

- Most patients will require a loop diuretic (e.g., furosemide, torasemide) due to severe symptoms of congestion and worsening renal function. Use a combination of a loop and a thiazide-type diuretic if the patient has resistant oedema.[1]
- Adjust the dose according to the patient's symptoms and clinical status.[1]
- Monitor the patient's weight, renal function, and urine output carefully while they are taking a diuretic.[1] [23]
    - Aim to achieve positive diuresis with a reduction of body weight by 0.75 to 1.0 kg/day.[1]

Practical tip

Avoid excessive diuresis; this is more dangerous than oedema.[1]
In practice, convert the patient from an intravenous to an oral diuretic when there is significant reduction in peripheral oedema (i.e., oedema to ankles only).

plus **referral to specialist**

Treatment recommended for ALL patients in selected patient group

» Ensure the patient has input from the heart failure specialist team within 24 hours of admission to hospital.[29]

» **Early involvement of the specialist heart failure team** for all patients admitted with acute

MANAGEMENT

Ongoing

heart failure enables consideration of additional treatments to optimise outcomes.[6] For patients with heart failure with reduced ejection fraction these may include:[1] [24]

- Ivabradine
- Isosorbide dinitrate plus hydralazine
- Digoxin
- Cardiac resynchronisation therapy
- Implantable cardioverter defibrillator
- Transplantation or mechanical circulatory support device.

plus **cardiac rehabilitation**

Treatment recommended for ALL patients in selected patient group

» Offer the patient a referral to cardiac rehabilitation before they are discharged.[4] [24]

» Cardiac rehabilitation should be personalised and exercise-based. It should also address psychological and educational aspects.[4] [24]

acute episode stabilised: LVEF 41% to 49%

1st **referral to specialist**

» Ensure the patient has input from the heart failure specialist team within 24 hours of admission to hospital.[29]

» **Early involvement of the specialist heart failure team** for all patients admitted with acute heart failure enables consideration of additional treatments to optimise outcomes.[6]

» Patients with mildly reduced ejection fraction (HFmrEF) need an individualised care plan, based on clinical characteristics, risk factors, patterns of cardiac remodelling, and the patient's comorbidities and prognosis. This may include therapies usually recommended for patients with HFrEF (e.g., ACE inhibitor, beta blocker, aldosterone antagonist, sacubitril/valsartan).[1] Treatment with a SGLT2 inhibitor may also be beneficial for this group, regardless of the presence of diabetes.[48] See *acute episode stabilised: LVEF ≤40%* (above) for more information.

plus **cardiac rehabilitation**

Treatment recommended for ALL patients in selected patient group

MANAGEMENT

Ongoing

» Offer the patient a **referral to cardiac rehabilitation** before they are discharged.[4] [24]

» Cardiac rehabilitation should be personalised and exercise-based. It should also address psychological and educational aspects.[4] [24]

consider **diuretic**

Treatment recommended for SOME patients in selected patient group

Primary options

» furosemide: 40 mg orally once daily initially, titrate gradually according to response, maximum 120 mg/day

OR

» torasemide: 5 mg orally once daily initially, titrate gradually according to response, maximum 40 mg/day

OR

» furosemide: 40 mg orally once daily initially, titrate gradually according to response, maximum 120 mg/day
-or-
» torasemide: 5 mg orally once daily initially, titrate gradually according to response, maximum 40 mg/day
--AND--
» metolazone: 5-10 mg orally once daily initially, increase gradually according to response, maximum 80 mg/day

» Consider a **diuretic for relief of symptoms due to congestion** in patients with heart failure with mildly reduced ejection fraction.[2]

acute episode stabilised: LVEF ≥50%

1st **referral to specialist**

» Ensure the patient has input from the heart failure specialist team within 24 hours of admission to hospital.[29]

» **Early involvement of the specialist heart failure team** for all patients admitted with acute heart failure enables consideration of additional treatments to optimise outcomes.[6]

» The European Society of Cardiology states that no specific treatment has been shown to convincingly significantly reduce morbidity or

Ongoing

mortality. However, one subsequent randomised controlled trial suggests that these patients may benefit from a SGLT2 inhibitor (empagliflozin), regardless of the presence of diabetes.[48]

plus **cardiac rehabilitation**

Treatment recommended for ALL patients in selected patient group

» Offer the patient a **referral to cardiac rehabilitation** before they are discharged.[4] [24]

» Cardiac rehabilitation should be personalised and exercise-based. It should also address psychological and educational aspects.[4] [24]

consider **diuretic**

Treatment recommended for SOME patients in selected patient group

Primary options

» furosemide: 40 mg orally once daily initially, titrate gradually according to response, maximum 120 mg/day

OR

» torasemide: 5 mg orally once daily initially, titrate gradually according to response, maximum 40 mg/day

OR

» furosemide: 40 mg orally once daily initially, titrate gradually according to response, maximum 120 mg/day
-or-
» torasemide: 5 mg orally once daily initially, titrate gradually according to response, maximum 40 mg/day
--AND--
» metolazone: 5-10 mg orally once daily initially, increase gradually according to response, maximum 80 mg/day

» Consider a **diuretic for relief of symptoms due to congestion** in patients with heart failure with preserved ejection fraction.[2]

MANAGEMENT

Emerging

Tolvaptan

A vasopressin antagonist that blocks the action of arginine vasopressin at the V_2 receptor in renal tubules and promotes aquaresis.[1] Tolvaptan may be used to treat patients with volume overload and resistant hyponatraemia.[1]

Cinepazide

Cinepazide, a vasodilator, was associated with significantly improving symptoms with less adverse effects in patients with decompensated heart failure, compared with dobutamine.[49]

Vericiguat

The US Food and Drug Administration has approved vericiguat, an orally administered soluble guanylate cyclase stimulator, for treatment of chronic heart failure in patients who are hospitalised for heart failure or need outpatient intravenous diuretics. When compared with placebo, it demonstrated a reduced incidence of death from cardiovascular causes or hospitalisation for heart failure in this patient group.[50]

Other investigational medications

These include ularitide, tezosentan, istaroxime, perhexiline, relaxin, and cardiac myosin activators. These agents are investigational and not routinely used to treat acute heart failure.[51] [52] [53] [54] Adenosine A1- receptor antagonists (e.g., tonapofylline and rolofylline) have failed to show any clinical benefit in initial studies.[55] [56] When compared with placebo, rolofylline did not show any benefit in patients with acute heart failure and impaired renal function.[56] In a phase 2 trial of patients with acute heart failure (ejection fraction <40%), treatment with omecamtiv mecarbil (a selective small-molecule activator of cardiac myosin) did not improve the primary end point of dyspnoea, or any pre-specified secondary end point when compared with placebo.[57]

Primary prevention

Consider interventions aimed at modifying risk factors in order to delay or prevent the onset of acute heart failure, including:[1]

- Coronary artery disease: manage with aspirin, beta-blockers, statins, and ACE inhibitors, as needed
    - Optimising treatment of hypertension, smoking cessation, and lipid control provides substantial benefit in patients with coronary artery disease[1]
    - Optimal control of hypertension may require more than one antihypertensive medication.[17] Different antihypertensive drugs (diuretics, ACE inhibitors, angiotensin receptor blockers, beta-blockers, calcium channel blockers) have been shown to be effective, especially in older people, both with and without a history of myocardial infarction[1]
- Diabetes mellitus: in addition to metabolic control, ensure aggressive control of lipids and blood pressure[18] [19]
- Alcohol consumption and excessive salt and fluid intake: discourage in patients with known left ventricular dysfunction[20]
- Drugs that can cause or potentiate heart failure: avoid, if safe and possible to do so.[21]

Secondary prevention

All patients with heart failure are recommended to have pneumococcal vaccination and annual influenza vaccine.

Patient discussions

Advise the patient on measures to prevent further episodes, including:

- Restricting fluid intake
- Restricting salt intake
- Limiting alcohol intake
- Continuing medication as prescribed
- Checking of weight daily.

Provide the patient with a self-management plan that outlines when they should seek medical attention.[4]

Monitoring

Monitoring

During the acute phase all patients require cardiac monitoring. Other types of monitoring might also be indicated, depending on drugs used: for example, renal function, electrolytes, heart rate, blood pressure, and overall clinical status should be closely monitored during treatment with beta-blockers, aldosterone antagonists, or angiotensin-converting enzyme inhibitors; renal function, weight, and urine output should be closely monitored during diuretic therapy.[23]

After discharge from hospital, a follow-up clinical assessment should be undertaken by a member of the specialist heart failure team within 2 weeks.[23] Follow-up arrangements should be clearly documented.[4]

Once the acute phase is over and patients are stable and considered to have stable heart failure, they should be encouraged to do regular aerobic exercise, and it is recommended that they be enrolled in a multidisciplinary care management programme.[1]

FOLLOW UP

Complications

Complications	Timeframe	Likelihood
arrhythmias	**short term**	**high**
Acute heart failure is frequently precipitated by arrhythmias (in particular, atrial fibrillation), but acute heart failure may also cause arrhythmias.[60] [61]		
complications of glyceryl trinitrate	**short term**	**high**
Commonly causes headache and hypotension. The headache is usually mild to moderate in severity and either resolves or diminishes in intensity with continued nitrate therapy. If hypotension occurs then the infusion rate should be decreased. If hypotension persists then the infusion should be discontinued and restarted when the patient is haemodynamically stable.		
complications of treatment: nesiritide	**short term**	**high**
Causes headache and hypotension. If hypotension occurs, then the infusion rate should be decreased. If hypotension persists, then the infusion should be discontinued and restarted when the patient is haemodynamically stable.		
complications of treatment: diuretics	**short term**	**medium**
Over-diuresis leads to worsening of renal function, hypotension, and hypokalaemia, and also activation of neurohormones including renin-angiotensin system and the sympathetic system. It may potentiate the toxicity of other agents like digoxin, either by causing hypokalaemia or by decreasing the glomerular filtration. In cases of worsening renal impairment due to over-diuresis, the dose of diuretics should be decreased. In case of severe renal impairment the diuretic can be withheld and the patients assessed daily, with re-introduction of diuretic at lower doses.		
complications of treatment: inotropes	**short term**	**medium**
Dobutamine and milrinone can cause arrhythmias and worsening of coronary ischaemia. The occurrence of sustained arrhythmias should lead to discontinuation. In cases where these medications are absolutely needed, concomitant use of amiodarone may be advisable, although there are no large-scale data on the use of anti-arrhythmics in this setting. If the patient has symptomatic coronary ischaemia, these infusions should be discontinued.		

Prognosis

Acute heart failure carries an inpatient mortality of 11% overall; in England and Wales there is significant variation between acute hospitals (lowest 6%; highest 26%).[4]

Predictors of adverse outcomes include: hypotension, renal dysfunction, older age, male sex, ischaemic congestive heart failure (CHF), previous CHF, respiratory rate on admission >30/minute, anaemia, hyponatraemia, elevated troponin, elevated B-type natriuretic peptide, and other comorbidities such as cancer.[58]

The National Heart Failure Audit 2020/21, based on data acquired during the coronavirus disease 2019 (COVID-19) pandemic, showed that in-patient mortality (9.2%) and 1-year mortality (39%), was unchanged from 2019/20. Mortality was lower for patients admitted to cardiology (6.0%) compared with general medical (10.2%) wards and for those seen by a specialist (7.9%) compared with those who weren't (14.9%).[6]

One study found that among patients hospitalised with heart failure, patients across the ejection fraction spectrum have a similarly poor 5-year survival with an elevated risk for cardiovascular and heart failure admission.[59] All patients in this cohort, regardless of ejection fraction, had a remarkably high mortality rate at 5 years from index admission (75.4%).[59]

Diagnostic guidelines

United Kingdom

Acute heart failure: diagnosis and management

Published by: National Institute for Health and Care Excellence **Last published:** 2021

Europe

ESC guidelines for the diagnosis and treatment of acute and chronic heart failure

Published by: European Society of Cardiology **Last published:** 2021

North America

2022 AHA/ACC/HFSA guideline for the management of heart failure

Published by: American Heart Association; American College of Cardiology; Heart Failure Society of America **Last published:** 2022

2017 comprehensive update of the Canadian Cardiovascular Society guidelines for the management of heart failure

Published by: Canadian Cardiovascular Society **Last published:** 2017

Treatment guidelines

United Kingdom

Implantable cardioverter defibrillators and cardiac resynchronisation therapy for arrhythmias and heart failure

Published by: National Institute for Health and Care Excellence **Last published:** 2014

Europe

ESC guidelines for the diagnosis and treatment of acute and chronic heart failure

Published by: European Society of Cardiology **Last published:** 2021

North America

2022 AHA/ACC/HFSA guideline for the management of heart failure

Published by: American Heart Association; American College of Cardiology; Heart Failure Society of America **Last published:** 2022

Academy of Nutrition and Dietetics evidence-based practice guideline for the management of heart failure in adults

Published by: Academy of Nutrition and Dietetics **Last published:** 2018

2017 comprehensive update of the Canadian Cardiovascular Society guidelines for the management of heart failure

Published by: Canadian Cardiovascular Society **Last published:** 2017

2016 ACC/AHA/HFSA focused update on new pharmacological therapy for heart failure: an update of the 2013 ACCF/AHA guideline for the management of heart failure

Published by: American College of Cardiology; American Heart Association; Heart Failure Society of America **Last published:** 2016

Recommendations for the use of mechanical circulatory support: device strategies and patient selection

Published by: American Heart Association **Last published:** 2012

2012 ACCF/AHA/HRS focused update of the 2008 guidelines for device-based therapy of cardiac rhythm abnormalities

Published by: American College of Cardiology; American Heart Association; Heart Rhythm Society **Last published:** 2012

Oceania

Guidelines for the prevention, detection, and management of heart failure in Australia

Published by: National Heart Foundation of Australia; Cardiac Society of Australia and New Zealand **Last published:** 2018

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Images

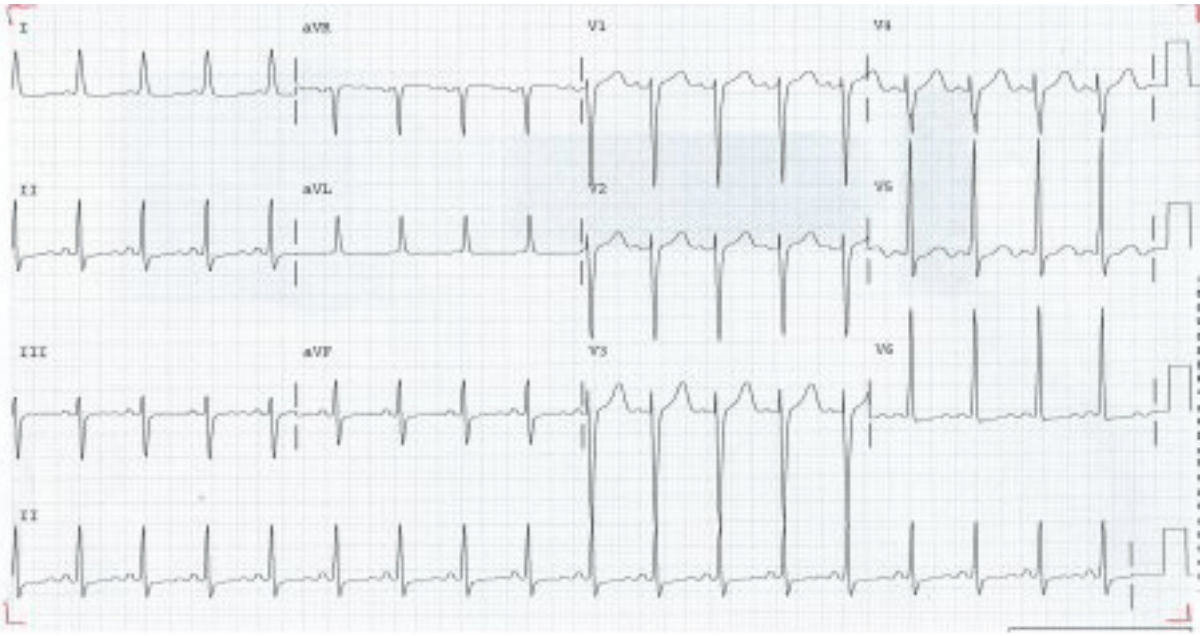

Figure 1: ECG showing left ventricular hypertrophy with sinus tachycardia

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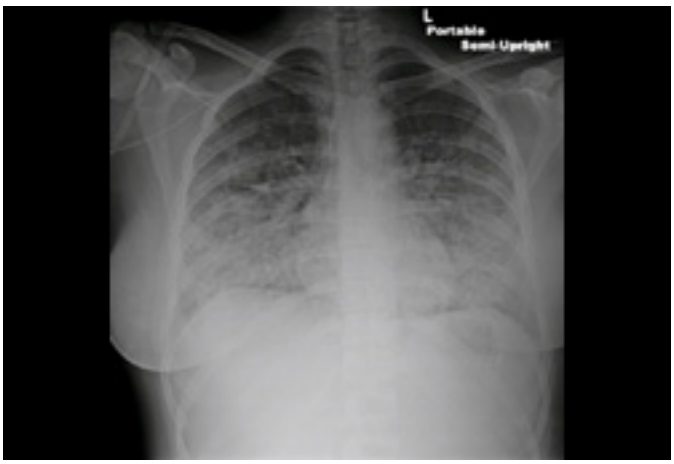

Figure 2: Chest x-ray showing acute pulmonary oedema with increased alveolar markings, fluid in the horizontal fissure, and blunting of the costophrenic angles

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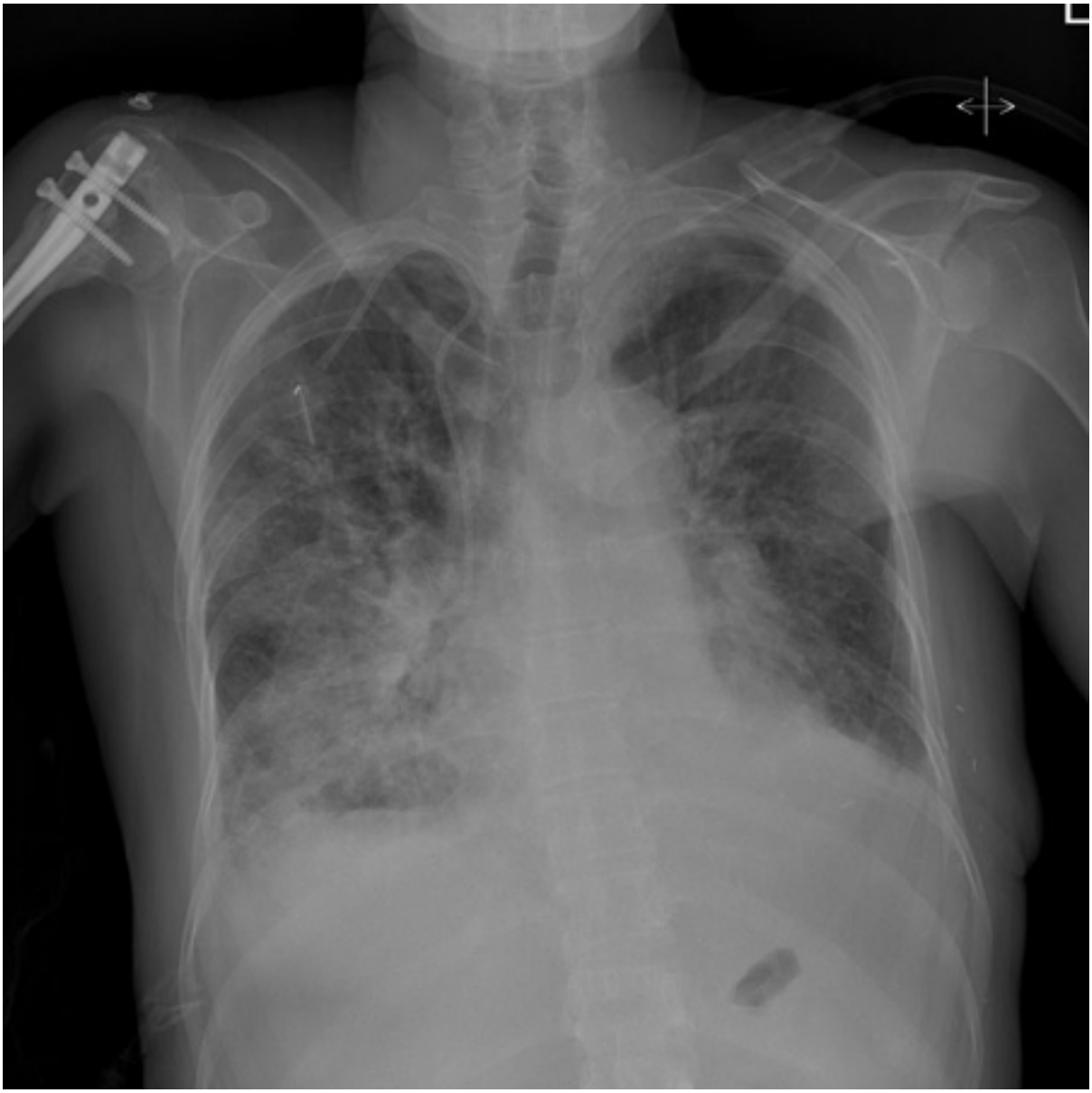

Figure 3: Chest x-ray showing acute pulmonary oedema with increased alveolar markings and bilateral pleural effusions

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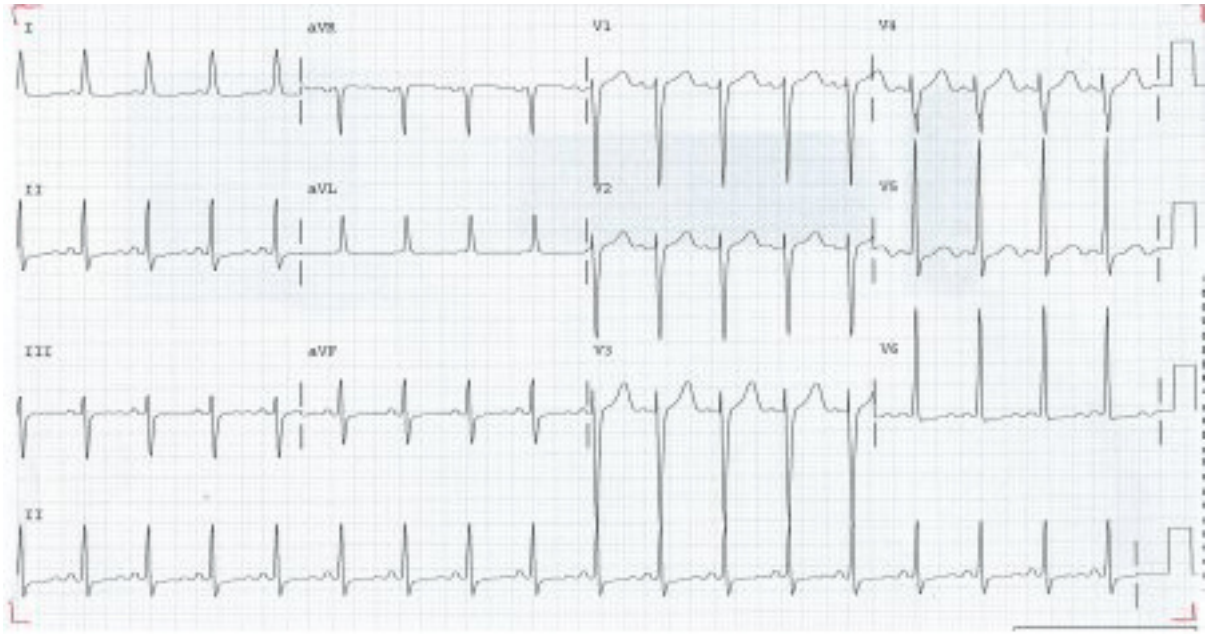

Figure 4: ECG showing left ventricular hypertrophy with sinus tachycardia

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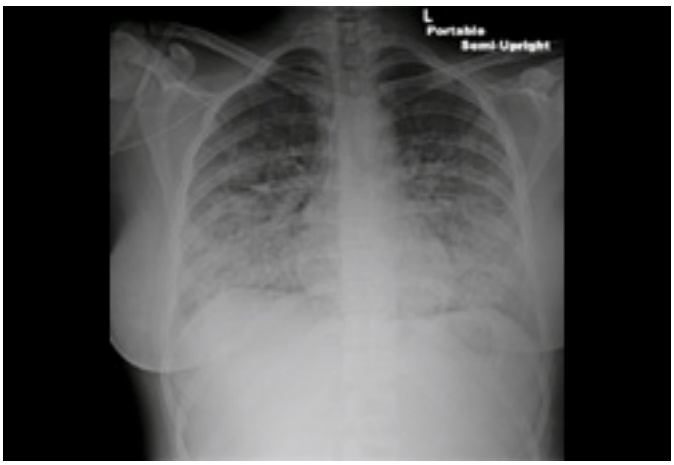

Figure 5: Chest x-ray showing acute pulmonary oedema with increased alveolar markings, fluid in the horizontal fissure, and blunting of the costophrenic angles

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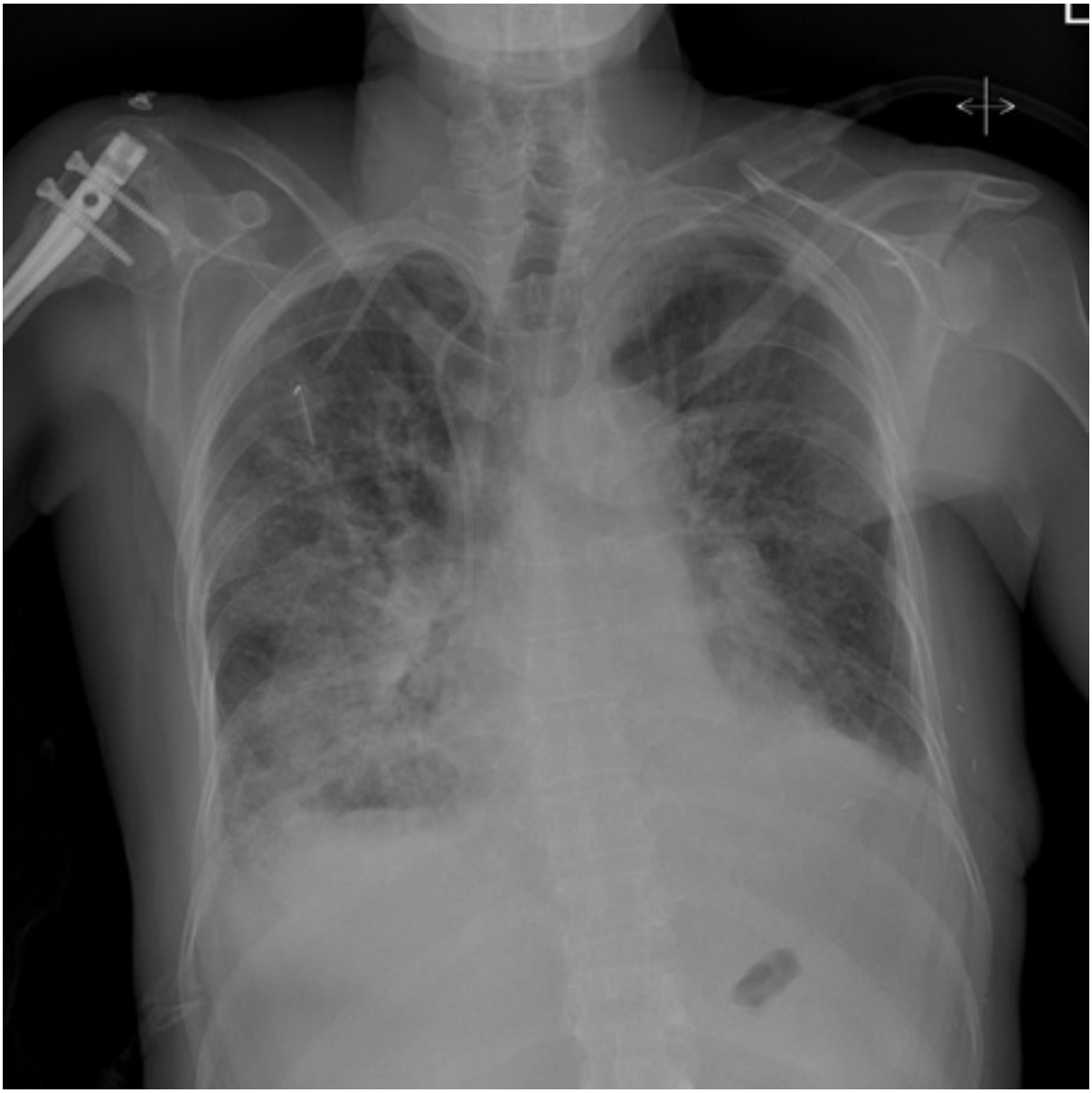

Figure 6: Chest x-ray showing acute pulmonary oedema with increased alveolar markings and bilateral pleural effusions

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