Heart failure - acute or decompensated

Clinical presentation
Consider differential diagnoses
Consider morphine for severe dyspnoea, agitation or pain

Clinical assessment
Is patient hypoxic or hypotensive?

Yes
Patient stable

No

Initial investigation
Consider differential diagnoses

Systolic BP less than 90mmHg
Not well perfused
Administer inotropic agent
Advanced investigation and intervention as required

Well perfused
Administer IV diuretics if fluid overloaded
Further investigation and intervention as required
Considerations for discharge when stable

Systolic BP more than 90mmHg
Administer IV diuretics if fluid overloaded
Third-line agents if unresponsive

Preserved LV function

Complete the venous thromboembolism (VTE) risk assessment
Go to VTE risk assessment

Information resources for patients and carers
Heart failure - acute or decompensated

Quick info:

Scope:
- this page covers the in-hospital assessment, investigation and management of acute heart failure (HF) or decompensated chronic HF

Out of scope:
- management of other associated or precipitating co-morbidity, eg acute coronary syndromes (ACS), arrhythmia or pulmonary embolism (see appropriate pathways)

Definition:
- acute HF is the term used to describe the potentially life threatening scenario of sudden onset of pulmonary or peripheral congestion (eg pulmonary oedema or peripheral oedema) occurring with or without hypoperfusion
- it may occur in patients without prior HF (eg following a new myocardial infarction [MI]) or in patients with known chronic HF that has become decompensated (unstable)
- it may occur as a result of:
  - increased preload (ventricular pressure at the end of diastole), as may occur with valve regurgitation or volume overload
  - increased afterload (ventricular pressure during systole), such as from valvular stenosis, pulmonary embolism or systemic hypertension;
  - high output states, eg anaemia, thyrotoxicosis
- several distinct clinical presentations of acute HF are recognised depending on the predominant features, these are principally determined by the level of peripheral perfusion, haemodynamic stability and presence of pulmonary congestion, if any

Classifications of acute HF:
- the Killip classification defines the extent of cardiac dysfunction:
  - stage I – no signs of HF
  - stage II – clinical signs of HF, eg rales in lower lung fields, third heart sound and pulmonary hypertension
  - stage III – severe HF with evident pulmonary oedema and rales throughout the lungs
  - stage IV – cardiogenic shock, ie blood pressure (BP) less than 90mmHg, fast, thready pulse and signs of hypoperfusion (cool, clammy skin, cyanosis, altered consciousness, oliguria)
- the Forrester classification is a graphical representation of acute HF defining four groups according to levels of pulmonary congestion (using the measure of pulmonary capillary wedge pressure [PCWP]) and hypoperfusion (using cardiac index [CI] – the individualised measure of cardiac output):
  - normal – CI more than 2.5L/minute/m² and PCWP of 15mmHg or less
  - pulmonary oedema – normal CI but PCWP more than 18mmHg
  - hypovolaemic shock (low cardiac output syndrome) – CI less than 2L/minute/m² but normal PCWP
  - cardiogenic shock – CI less than 2L/minute/m² and PCWP more than 18mmHg
- the ‘clinical severity’ classification would regard the above patients in the Forrester classification as being respectively:
  - class I – warm and dry
  - class II – warm and wet
  - class III – cold and dry
  - class IV – cold and wet

Aetiology:
- the majority of hospitalisations for acute HF occur in patients with a history of heart disease – common causes and precipitants are:
  - acute coronary syndromes (ACS):
    - ischaemic heart disease (IHD) is the precipitant of acute HF in 60-70% of cases
    - of patients with ACS, 30-35% demonstrate some degree of cardiac dysfunction
  - arrhythmia, eg new onset atrial fibrillation (AF) or other supraventricular or ventricular tachyarrhythmia
  - hypertensive crisis
  - valvular condition, eg aortic stenosis or worsening regurgitation
• cardiac tamponade
• aortic dissection
• high output syndromes, eg thyrotoxicosis or anaemia
• other precipitating factors include:
  • non-compliance with medication for chronic HF
  • systemic infection
  • following surgery
  • impaired renal function
  • drug or alcohol abuse

Incidence:
• HF is the leading cause of hospitalisation (US data) in people over age 65 years

Prognosis:
• mortality rates are high in patients with acute HF, particularly when associated with ACS
• ACS associated with acute HF has a 12 month mortality of 30%
• a randomised trial found that for all causes of acute HF, the 60 day mortality is 9.6% and re-hospitalisation within 60 days was 35.2%
• for documented cases of pulmonary oedema, the inpatient mortality was 12% and 40% at 1 year

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4 Clinical presentation

Quick info:
Several distinct clinical presentations of acute heart failure (HF) are recognised depending on the predominant features, these are principally determined by the level of peripheral perfusion, haemodynamic stability and presence of pulmonary congestion, if any:

- acute HF with pulmonary oedema:
  - severe respiratory distress and orthopnoea
  - reduced oxygen saturation (typically less than 90%)
  - widespread crackles across lung fields
  - chest X-ray appearance of pulmonary oedema (peri hilar 'bat wing' shadowing, Kerley B lines, pulmonary venous congestion and upper lobe diversion)
  - frequently tachycardia with low to normal blood pressure (BP)
  - reduced left ventricular (LV) function (as assessed haemodynamically by cardiac index [CI – in L/minute/m$^2$] and raised pulmonary capillary wedge pressure [PCWP])

- acute HF hypertensive crisis:
  - congestive signs and symptoms associated with high BP
  - usually tachycardic
  - relatively preserved CI and elevated PCWP
  - chest X-ray appearance of pulmonary oedema

- grades of low cardiac output, from mild disturbances through to cardiogenic shock (parameters for shock given in brackets):
  - signs of hypoperfusion with or without congestion
  - low BP (less than 90mmHg is taken as indicative of cardiogenic shock) or low mean arterial pressure (a drop of more than 30mmHg)
  - tachycardia (more than 90 beats/minute), thready pulse
  - reduced urine output (less than 0.5mL/kg/hour)
  - reduced CI (less than 1.8L/minute/m$^2$) and elevated PCWP (more than 18mmHg)

- high output failure:
  - signs of congestion
  - tachycardia, bounding pulse
  - warm perfused peripheries
  - typically normal BP
  - elevated CI and normal to low PCWP

- right sided HF:
  - predominant signs of systemic congestion, ie raised jugular venous pressure (JVP), hepatomegaly and oedema
  - reduced BP and typically reduced heart rate
  - low CI and low PCWP
  - acute decompensated HF:
    - milder signs and symptoms not fulfilling diagnostic criteria for any of the other syndromes
    - minimal change in CI and normal to mild elevation of PCWP

The European Society of Cardiology defines a range of presentations as:
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- forward HF (signs and symptoms of reduced cardiac output) – severity may vary from mild to cardiogenic shock:
  - fatigue and lethargy
  - peripheral hypoperfusion, eg pallor, cold, clammy skin
  - altered mental state
  - low BP and elevated thready pulse
  - reduced urine output
  - examination may reveal new murmurs, muffled heart sounds or paradoxical pulse suggestive of cause
  - may be variable congestion
- left backward HF (pulmonary congestion) – varying in severity from mild to acute pulmonary oedema:
  - dyspnoea – on exertion only through to tachypnoea at rest and orthopnoea
  - dry cough, sometimes with pink frothy sputum
  - may be cyanosed with peripheral hypoperfusion
  - BP may be normal to high
  - auscultation reveals crepitations which may be throughout the lung fields if severe
  - chest X-ray appearance of pulmonary oedema
- right backward HF – pulmonary and right sided dysfunction:
  - fatigue and lethargy
  - elevated jugular venous pressure (JVP)
  - peripheral pitting oedema, with ascities, sacral and scrotal oedema if severe
  - tender hepatomegaly due to hepatic venous congestion
  - may be reduced urine output

References:

5 Clinical assessment

Quick info:
A full examination may not be possible in the acute situation – make a rapid assessment including the following:

- general appearance:
  - respiratory rate at rest
  - agitation
  - pale, clammy or cyanosed
  - conscious level
- pulse rate, rhythm, volume and character:
  - arrhythmias
  - bounding suggesting high output failure
  - thready pulse of low output or hypovolaemic shock
  - paradoxical pulse (suggesting cardiac tamponade)
  - characteristics of valve abnormalities, eg slow rising pulse of aortic stenosis or collapsing pulse of regurgitation
- blood pressure (BP)
- temperature
- elevated jugular venous pressure (JVP) or distended neck veins
- carotid bruits
- palpate for the apex, presence of heaves or thrills
- heart auscultation:

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- third heart sound (gallop rhythm)
- murmurs
- chest auscultation:
  - wheeze
  - inspiratory crepitations – basal or distributed throughout lung fields
- abdominal palpation:
  - ascities
  - tender hepatomegaly
- peripheral pitting oedema

Initiate all investigations urgently (electrocardiogram [ECG], bloods and chest X-ray – covered in nodes below), concurrently to starting treatment.

References:

7 Yes
Quick info:
The initial priorities of treatment are:
- to stabilise the patient's airway, breathing and circulation
- relieve dyspnoea, pain or agitation
- to obtain haemodynamic stability
- investigation should be carried out concurrently to detect underlying causes

10 Initial investigation
Quick info:
Apply an electrocardiogram (ECG) monitor as soon as possible and continuously monitor:
- assess for abnormal rhythms (atrial fibrillation [AF] or flutter) and other tachyarrhythmia (supraventricular or ventricular tachycardia):
  - if present, manage the arrhythmia, eg acute AF with signs of haemodynamic compromise is likely to require electronic cardioversion
  - management of peri-arrest arrhythmias is beyond the scope of this pathway, see 'Atrial fibrillation' and other 'Peri-arrest arrhythmia' pathways as appropriate for further information
- assess for any signs of ischaemia or infarction and proceed as appropriate if an acute coronary syndrome (ACS)
- the ECG may suggest left ventricular (LV) or right ventricular (RV) hypertrophy or other signs consistent with heart failure (HF)

Order a chest X-ray:
- consider either a portable antero-posterior (AP) chest X-ray, or accompanied transfer to X-ray for a departmental X-ray, if the patient is in a stable condition
- may demonstrate:
  - pulmonary oedema – perihilar 'bat wing' shadowing, Kerley B lines, pulmonary venous congestion and upper lobe diversion
  - cardiomegaly (portable AP film will be unreliable for this)
  - pleural effusions
  - suggestion of other diagnoses, eg consolidation

Take an arterial blood gas:
- continue to monitor oxygen saturation whilst the patient is on oxygen therapy
Consider additional tests as indicated:
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- full blood count (FBC)
- liver function tests (LFTs)
- magnesium and calcium
- C reactive protein (CRP)
- urinalysis

B-type natriuretic peptide (BNP):
- BNP (also NT-proBNP) is a cardiac neurohormone released from ventricular cardiomyocytes in response to ventricular dilatation and volume overload
- it is rarely normal in a patient with chronic heart failure (HF) and values increase with severity of HF
- the value of BNP in the acute setting is debatable

References:

11 Consider differential diagnoses

Quick info:
Differential diagnoses (may be associated with acute heart failure [HF] or as an alternative diagnosis) include:
- acute coronary syndrome (ACS; 'Acute coronary syndrome' pathway)
- pneumonia or exacerbation of chronic obstructive pulmonary disorder (COPD)
- pulmonary embolism (see suspected PE page)
- supraventricular or ventricular tachycardias
- asthma
- pericarditis
- cardiac tamponade
- acute anaemia
- pneumothorax
- sepsis

Reference:

12 Consider morphine for severe dyspnoea, agitation or pain

Quick info:
- consider intravenous (IV) morphine as soon as IV access is obtained
- it causes venodilation and depresses heart rate, relieves dyspnoea, agitation, restlessness, and pain
- although morphine has consistently been used as a first-line treatment for pulmonary oedema or cardiogenic shock, there is limited evidence for its long-term effects or outcomes

References:
17 Administer IV diuretics if fluid overloaded

Quick info:
- Diuretics are indicated as first-line agents in any patient with acute heart failure (HF) and signs of pulmonary or systemic congestion.
- They are the most effective drugs for obtaining rapid symptomatic relief.
- Loop diuretics are the most effective, they increase sodium and free water excretion, maintaining this effect even when there is slight renal impairment.
- They reduce preload on the heart, cause vasodilatation and reduce pulmonary capillary wedge pressure (PCWP).
- Careful diuretic administration does not normally cause an adverse effect on blood pressure (BP) – conversely reduced preload frequently causes an increase in stroke volume and hence cardiac output due to reduced left ventricular (LV) dilatation.
- Administer at low dose initially as high dose can cause vasoconstriction (particularly in patients with acute coronary syndromes); vasodilators would be considered the mainstay of treatment in these cases.
- Administer orally or intravenously depending on clinical severity:
  - Oral administration has a peak effect at 1-2 hours, conversely intravenous (IV) administration may exert an effect within 15 minutes.
  - All severe cases will require IV administration.
- Several small studies have demonstrated adverse effects of bolus diuretic administration including greater fluctuations in intravascular volume, longer hospitalisation and increased mortality:
  - IV infusion is therefore considered preferable in both safety and efficacy.
- Consider initially 40-100mg of frusemide (depending on severity) followed by an infusion at a rate of 5-40mg/hour; titrate according to response.
- Monitor response (jugular venous pressure [JVP], pulmonary or peripheral oedema), heart rate, blood pressure (BP) and urine output – the aim is relief of congestion without adversely affecting haemodynamic stability.
- Resistance to diuretic therapy is recognised, particularly in patients with chronic HF on long-term therapy – causes include:
  - Intravascular volume depletion and decreased renal perfusion.
  - Decreased tubular secretion.
  - Enhanced activation of the sympathetic and renin-angiotensin systems.
  - Hypertrophy of the distal nephron.
  - Rebound uptake of sodium with volume loss.
- If congestion is refractory to loop diuretics, combination with a thiazide or spironolactone (select if low potassium) is considered to be preferable to higher doses of loop diuretic; however, likewise consider assessing the response of co-administration with vasodilators or inotropes.
- During continued management of acute HF with diuretics:
  - Monitor symptomatic response, daily weight, fluid intake and output, urea, creatinine, electrolytes and magnesium.
  - Reduce dose when fluid balance is controlled.
- Haemodialysis and ultrafiltration are sometimes considered as a final resort if fluid overload fails to be controlled by diuretic therapy.

References:

18 Administer vasodilators

Quick info:
in the absence of hypotension, nitrates should be considered as first-line agents to provide symptomatic relief in conjunction with diuretics
they cause venodilation at low dose and arteriodilation with increased dose
a balanced dose therefore has effects of reduction in both preload and afterload, in addition to coronary dilation effect in relieving ischaemia
two studies have demonstrated that administration of maximum tolerated nitrate infusion in addition to low dose loop diuretic, improved outcomes more than administering a higher dose of diuretic alone
administer nitrate at the optimal dose necessary to give increased cardiac index (CI) and a decrease in pulmonary capillary wedge pressure (PCWP)
administration may be sublingual or by aerosol spray, however in more severe cases intravenous (IV) administration will be necessary – consider:

2 puffs glyceryl trinitrate spray (0.4mg) every 5 minutes or buccal isosorbide dinitrate
IV infusion of glyceryl trinitrate (20-200micrograms/minute) or isosorbide dinitrate (1-10mg/hour)
titrater the dose carefully with monitoring of haemodynamic stability, maintaining systolic blood pressure (BP) above 100mgHg
approximately 20% of patients have nitrate tolerance and failure to respond to increased nitrate dose should indicate need to consider alternative therapy
effectiveness declines after 16-24 hours due to the development of tolerance therefore prolonged administration is not beneficial
hypotension and headache are common adverse effects
nitrates should not be used in patients with:
mitral or aortic stenosis
cardiac tamponade
constrictive pericarditis or hypertrophic obstructive cardiomyopathy

References:

19 Administer inotropic agent

Quick info:
Inotropic agent:

if the patient's clinical status is one of low cardiac output syndrome to cardiogenic shock (hypovolaemia and pulmonary oedema), intravenous (IV) inotropes should be considered as first-line therapy in addition to IV diuretic administration
they may also be considered in patients with pulmonary oedema without low output state who do not respond to initial therapy
however, note that although indicated for patients with severe refractory acute heart failure (HF) with reduced cardiac index (CI) and increased pulmonary capillary wedge pressure (PCWP), they are associated with several risks and adverse outcomes including increased mortality
inotropes cause increased force and rate of myocardial contraction and cause pulmonary and systemic vasodilatation
they increase oxygen demand, calcium loading and may precipitate tachyarrhythmia and ischaemia
different inotropes that may be used are the sympathomimetics dopamine and dobutamine or the phosphodiesterase inhibitors milrinone and enoximone:
both types act in slightly different ways – the sympathomimetics stimulate myocardial beta receptors and are therefore adversely affected by administration of beta blockers, they also have little effect on pulmonary vasculature
administration:
milrinone may be given as a bolus IV dose or infuse at a rate of 0.375-0.75micrograms/kg/minute
dobutamine and dopamine cannot be given as bolus doses; infuse at a rate of 2-20micrograms/kg/minute or 3-5micrograms/kg/minute respectively (dopamine at lower dose may also be used to improve renal perfusion)
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• monitoring of blood pressure (BP) for hypotension and electrocardiogram (ECG) for signs of arrhythmia or ischaemia is essential during administration – discontinue if they develop
• there is limited evidence available for use of inotropes and their effects have not been adequately assessed
• trials have demonstrated milrinone to be associated with increased in hospital and 60 day mortality and increased arrhythmias
• consider IV inotropes for short-term use only in carefully selected patients, ie those with cardiogenic shock who are unsuitable for vasodilator therapy, or those with pulmonary oedema that is refractory to other therapy

References:

20 Administer IV diuretics if fluid overload

Quick info:
Diuretics:
• are indicated as first-line agents in any patient with acute heart failure (HF) and signs of pulmonary or systemic congestion
• are the most effective drugs for obtaining rapid symptomatic relief
• loop diuretics are the most effective, they increase sodium and free water excretion, maintaining this effect even when there is slight renal impairment:
  • they reduce preload on the heart, cause vasodilatation and reduce pulmonary capillary wedge pressure (PCWP)
  • careful diuretic administration does not normally cause an adverse effect on blood pressure (BP); conversely reduced preload frequently causes an increase in stroke volume and hence cardiac output due to reduced left ventricular (LV) dilatation
  • initiate at a low dose as higher doses can cause vasoconstriction (particularly in patients with acute coronary syndromes [ACS]; vasodilators would be considered the mainstay of treatment in these cases)
• administer orally or intravenously depending on clinical severity:
  • oral administration has a peak effect at 1-2 hours, conversely intravenous (IV) administration may exert an effect within 15 minutes
  • all severe cases will require IV administration
• several small studies have demonstrated adverse effects of bolus diuretic administration including greater fluctuations in intravascular volume, longer hospitalisation and increased mortality:
  • IV infusion is therefore considered preferable in both safety and efficacy
• consider initially 40-100mg of frusemide (depending on severity) followed by an infusion at a rate of 5-40mg/hour – titrate according to response
• monitor response (jugular venous pressure [JVP], pulmonary and peripheral oedema), heart rate, blood pressure (BP) and urine output – the aim is relief of congestion without adversely affecting haemodynamic stability
• resistance to diuretic therapy is recognised, particularly in patients with chronic HF on long-term therapy – causes include:
  • intravascular volume depletion and decreased renal perfusion
  • decreased tubular secretion
  • enhanced activation of the sympathetic and renin-angiotensin systems
  • hypertrophy of the distal nephron
  • rebound uptake of sodium with volume loss
• if congestion is refractory to loop diuretics, combination with a thiazide or spironolactone (select if low potassium) is considered to be preferable to higher doses of loop diuretic:
  • however, likewise consider assessing the response of co-administration with vasodilators or inotropes
• during continued management of acute HF with diuretics:
  • monitor:
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- symptomatic response
- daily weight
- fluid intake and output
- urea
- creatinine
- electrolytes and magnesium
- reduce dose when fluid balance is controlled

- haemodialysis and ultrafiltration are sometimes considered as a final resort if fluid overload fails to be controlled by diuretic therapy

References:

21 Third-line agents if unresponsive

Quick info:
Alternative vasodilators may be considered if there is no response to nitrates and diuretics:
- sodium nitroprusside:
  - another alternative vasodilator with considerable venous and arterial effects and that reduces pulmonary capillary wedge pressure (PCWP)
  - it may be particularly indicated for patients with hypertensive acute heart failure (HF)
  - it is administered by intravenous (IV) infusion with careful haemodynamic monitoring
  - adverse effects include:
    - hypotension
    - renal impairment
    - toxicity from its cyanide metabolites
  - there is limited evidence for its use and its safety profile has not been adequately assessed – use with particular caution in patients with acute coronary syndromes (ACS)
  - inotropic agents:
    - if the patient's clinical status is one of low cardiac output syndrome to cardiogenic shock (hypovolaemia and pulmonary oedema), IV inotropes should be considered as first-line therapy in addition to IV diuretic administration
    - IV vasodilators are contraindicated in such patients, absolutely if systolic blood pressure (BP) is below 90mmHg
    - they may also be considered in patients with pulmonary oedema without low output state who do no respond to initial therapy
    - although indicated for patients with severe refractory acute HF with reduced cardiac index (CI) and increased PCWP, inotropes are associated with several risks and adverse outcomes
    - inotropes cause increased force and rate of myocardial contraction and cause pulmonary and systemic vasodilatation
    - they increase oxygen demand, calcium loading and may precipitate tachyarrhythmia and ischaemia
    - different inotropes that may be used are the sympathomimetics dopamine and dobutamine or the phosphodiesterase inhibitors milrinone and enoximone:
      - both types act in slightly different ways – the sympathomimetics stimulate myocardial beta receptors and are therefore adversely affected by administration of beta blockers – they also have little effect on pulmonary vasculature
      - administration:
        - milrinone may be given as a bolus IV dose or infuse at a rate of 0.375-0.75micrograms/kg/minute
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- dobutamine and dopamine cannot be given as bolus doses; infuse at a rate of 2-20 micrograms/kg/minute or 3-5 micrograms/kg/minute respectively (dopamine at lower dose may also be used to improve renal perfusion)
- monitoring of BP for hypotension and electrocardiogram (ECG) for signs of arrhythmia or ischaemia is essential during administration – discontinue if they develop
- there is limited evidence available for use of inotropes and their effects have not been adequately assessed
- trials have demonstrated milrinone to be associated with increased hospital and 60 day mortality and increased arrhythmias
- consider IV inotropes for short-term use only in carefully selected patients, ie those with cardiogenic shock who are unsuitable for vasodilator therapy, or those with pulmonary oedema that is refractory to other therapy
- nesiritide:
  - a relatively new vasodilating agent for acute HF that may be unfamiliar in clinical practice
  - it is a B-type natriuretic peptide (BNP) – similar to that produced endogenously by the ventricle, that reduces PCWP and causes vasodilatation with decrease in preload and afterload, but without considerable inotropic effect
  - arrhythmias and tachycardia are therefore less commonly associated than inotropic agents
  - it has been demonstrated to have comparable efficacy to nitrates – it is also considered safer than dobutamine
  - administer as a bolus or IV infusion with monitoring – hypotension is a predominant adverse effect
  - there is limited evidence to support its use and further trials are needed into its long-term outcomes:
    - analysis of five randomised controlled trials found a significant increase in impaired renal function when nesiritide was used in acute HF patients when compared to placebo, however the cause of this effect was not identified
    - three other studies found a significant increase in 30 day mortality with use of nesiritide compared to standard diuretic and nitrate therapy

References:

22 Advanced investigation and intervention as required

Quick info:
Care of people with acute heart failure (HF) is highly individualised, it depends on:
- clinical severity
- haemodynamic stability
- response to treatment
- associated cardiac morbidity
- non-cardiac co-morbidity

Patients who are considered to be unstable include those with:
- continued haemodynamic instability
- electrocardiogram (ECG) or troponins demonstrating signs of ischaemia or infarction
- the need for continuous vasodilator or inotropic infusion
- severe electrolyte imbalance
- non-sustained ventricular tachycardia (VT)
- end organ hypoperfusion, eg altered mental state, renal failure

Transfer patients to intensive care unit (ICU) as indicated by clinical severity and consider invasive haemodynamic monitoring for those:
- with cardiogenic shock or sustained low cardiac output
- refractory to treatment
• where accurate measures of haemodynamic status and pulmonary capillary wedge pressure (PCWP) are required to monitor response to therapy and guide further treatment

• modalities include:
  • arterial line – useful for repeated blood gas measurements and monitoring arterial blood pressure (BP)
  • central venous line:
    • inserted into the superior vena cava or right atrium, this monitors central venous pressure (note that this will be affected by tricuspid regurgitation and is a poor indicator of left atrial filling pressure)
    • separate ports can also be used for drug and fluid administration
    • for high impact interventions to reduce healthcare associated infections – see ‘Central venous catheter care bundle’
  • pulmonary artery catheter:
    • measures pressures in the pulmonary artery, superior vena cava, right atrium and right ventricle
    • it can be used to determine PCWP, cardiac output and hence the individualised measure of cardiac index (CI) – it is therefore an accurate measure to guide therapy
    • it is indicated in all patients with cardiogenic shock and those with low cardiac output or pulmonary oedema that is not responding to therapy, when measures of these direct parameters are necessary
    • note that inaccurate measures will be obtained in patients valve stenosis or regurgitation
  • a recent study did not show a survival benefit when patients with acute HF who were invasively monitored were followed up over a 12 month period
  • current advice is that invasive monitoring is not required routinely in the management of all acute HF patients
  • transfer to a tertiary care centre may be required as indicated

Further investigations:

• all patients with acute HF or chronic HF require echocardiological confirmation of the underlying structural or functional abnormality:
  • this should be considered as a matter of urgency in unstable cases, particularly in patients with suspected acute coronary syndromes (ACS)
  • patients with ACS should be considered for angiography at an early stage with a view to revascularisation, as this has been demonstrated to improve outcomes
  • if the patient is hypoperfused but there are no signs of pulmonary congestion – low BP, decreased CI but low PCWP, ie not in cardiogenic shock – assess response to a crystalloid fluid challenge
  • if there is no haemodynamic response to diuretics and inotropes and BP remains low, consider adrenaline by IV bolus or infusion
  • noradrenaline may be considered alternatively as indicated – it increases systemic vascular resistance and infusion may therefore be of use in certain situations, eg septic shock
  • if cardiac output becomes profoundly low, commence advanced life support as indicated
  • where appropriate, consider primary percutaneous coronary intervention (PCI) or thrombolysis

Monitoring:

• all patients should receive continued observation, monitoring of haemodynamic status and blood test results as indicated – this will include frequent assessment of:
  • pulse rate and rhythm, BP, oxygen saturation and ECG
  • fluid intake and output
  • signs and symptoms of acute HF
  • adverse effects
  • daily weight
  • daily urea, electrolytes and creatinine:
    • hyponatraemia occurs frequently as a result of volume overload and may respond to fluid restriction
    • other serology as indicated, eg full blood count (FBC), international normalised ratio (INR), cardiac enzymes
    • assess and manage other specific precipitants and co-morbidity, eg arrhythmias, valvular pathology and other non-cardiac causes

References:


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23 Further investigation and intervention as required

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    - separate ports can also be used for drug and fluid administration
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    - it can be used to determine PCWP, cardiac output and hence the individualised measure of cardiac index (CI) – it is therefore an accurate measure to guide therapy
    - it is indicated in all patients with cardiogenic shock and those with low cardiac output or pulmonary oedema that is not responding to therapy, when measures of these direct parameters are necessary
    - note that inaccurate measures will be obtained in patients valve stenosis or regurgitation
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- current advice is that invasive monitoring is not required routinely in the management of all acute HF patients
- transfer to a tertiary care centre may be required as indicated

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- patients with ACS should be considered for angiography at an early stage with a view to revascularisation, as this has been demonstrated to improve outcomes
- if the patient is hypoperfused but there are no signs of pulmonary congestion – low BP, decreased CI but low PCWP, ie not in cardiogenic shock – assess response to a crystalloid fluid challenge
- if there is no haemodynamic response to diuretics and inotropes and BP remains low, consider adrenaline by IV bolus or infusion
- noradrenaline may be considered alternatively as indicated – it increases systemic vascular resistance and infusion may therefore be of use in certain situations, eg septic shock
- if cardiac output becomes profoundly low, commence advanced life support as indicated
- where appropriate, consider primary percutaneous coronary intervention (PCI) or thrombolysis

Monitoring:
- all patients should receive continued observation, monitoring of haemodynamic status and blood test results as indicated – this will include frequent assessment of:
  - pulse rate and rhythm, BP, oxygen saturation and ECG
  - fluid intake and output
  - signs and symptoms of acute HF
  - adverse effects
  - daily weight
  - daily urea, electrolytes and creatinine:
    - hyponatraemia occurs frequently as a result of volume overload and may respond to fluid restriction
    - other serology as indicated, eg full blood count (FBC), international normalised ratio (INR), cardiac enzymes
  - assess and manage other specific precipitants and co-morbidity, eg arrhythmias, valvular pathology and other non-cardiac causes

References:

24 Considerations for discharge when stable

Quick info:
- the aims of treatment in acute heart failure (HF) are to:
  - improve clinical signs and symptoms
  - improve and maintain haemodynamic stability with reduction in pulmonary capillary wedge pressure (PCWP) and improved cardiac output
  - correct serological imbalance, eg correct electrolytes, improve renal function, decrease plasma B-type natriuretic peptide (BNP)
  - identify the underlying structural or functional aetiology of HF and other co-morbidity
  - to commence the patient on a suitable therapeutic regimen with appropriate monitoring and follow-up in place
  - to ensure the patient is fully educated and informed of their condition, and is supported by a multidisciplinary discharge care plan
  - overall to reduce duration of hospitalisation, try to prevent frequent re-admission and aim to improve long-term survival
  - patients with acute or decompensated HF are at a high risk of re-admission once discharged, therefore optimal pre-discharge care and follow-up arrangements are essential
  - prior to considering discharge patients should:
    - have optimal fluid balance achieved and control of symptoms
    - have been successfully commenced and stabilised on continued oral therapy (an angiotensin converting enzyme [ACE] inhibitor should be commenced for HF, in addition to diuretics, following recovery from the acute phase)
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- have had precipitating factors identified and a management plan for co-morbidity decided with referral where indicated
- have been ambulant in hospital and assessment made of their functional or social needs
- have full follow-up arranged, eg specialist nurse home visit, GP, next clinic appointment
- not have received IV vasodilator or inotropic therapy within the last 24 hours
- chronic HF often follows an unstable course with periods of stability compounded by decompensation, sometimes rapid and severe, that may considerably alter treatment plan and prognosis
- approximately half of all deaths related to HF occur suddenly
- prognostic information when considering the variable aetiology, co-morbidity and unstable course of HF is therefore difficult to provide, however strong indicators of adverse outcome include:
  - increased age
  - those with New York Heart Association (NYHA) class III or IV HF
  - persistent hypotension
  - high levels of BNP
  - low left ventricular ejection fraction (LVEF)
  - impairment of right ventricular function

Follow-up should be managed by a multidisciplinary team.

References:

25 Preserved LV function

Quick info:
Management of heart failure with preserved LV function consists of diuretics to remove fluid retention and treatment of the underlying pathology.
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Key Dates
Published: 21-Feb-2011, by Tameside & Glossop
Valid until: 20-Jul-2011

References
This is a list of all the references that have passed critical appraisal for use in the pathway Heart failure

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