Acute heart failure: a practical guide to management

David R Murdoch, John JV McMurray

Acute heart failure is a common cause of referral and admission to hospital. It requires prompt recognition and treatment. This article will address the practical issues regarding diagnosis, investigation, treatment and prevention of recurrence.

Acute heart failure is a common and potentially fatal acute medical emergency. It is a frightening condition, for both the patient and attending physician, requiring prompt diagnosis and administration of often life-saving treatment. Unfortunately, although the condition is common, there are few randomized clinical trials to guide therapy and much is, therefore, empirical. This article will examine the acute emergency treatment of left ventricular failure (more appropriately termed 'acute heart failure', as not all cases are caused by left ventricular dysfunction), and its subsequent management.

AETIOLOGY

In the western world, the majority of cases of acute heart failure are caused by left ventricular systolic dysfunction (LVSD), itself a consequence of ischaemic heart disease. Many patients with acute heart failure will have chronic heart failure which has decompensated. Patients with acute myocardial infarction, uncontrolled atrial fibrillation or severe valvular disease also commonly present with acute heart failure. A minority of patients with acute heart failure will have other causes (*Table 1*).

Acute pulmonary oedema may also occur with a normal heart as a consequence of severe anaemia, severe renal or hepatic dysfunction, severe hypoalbuminaemia, or following overzealous fluid or blood administration.

DIAGNOSIS

Clinical features

Acute heart failure may either arise de novo (e.g. acute myocardial infarction, uncontrolled atrial fibrillation) or as an acute on chronic problem. There are three broad clinical presentations of acute heart failure:

1. Marked peripheral oedema, often with renal impairment, usually complicating chronic heart failure
2. Predominantly pulmonary oedema (may be associated with peripheral oedema or hypotension)
3. Hypotension and evidence of central and peripheral hypoperfusion ('shock') — often occurs in the context of acute myocardial infarction and is usually associated with pulmonary oedema.

Signs of fluid retention, such as a raised jugular pressure and 'pitting' peripheral oedema, are often present in patients with heart failure; however, it is important to appreciate that neither are a prerequisite to nor confirm a diagnosis of acute heart failure. Examination should include a search for possible precipitating causes or factors which will influence management in other ways (*Table 2*), or possible alternative diagnoses which can mimic

Dr David R Murdoch is Specialist Registrar in Cardiology and Honorary Clinical Teacher and **Professor John JV McMurray** is Professor and Consultant Cardiologist in the Department of Cardiology, Western Infirmary, Glasgow G11 6NT

Correspondence to: Dr DR Murdoch

TABLE 1. Causes of acute heart failure

Acute myocardial infarction	Left ventricular systolic dysfunction
	Papillary muscle rupture
	Ventricular septal perforation
Decompensated chronic heart failure	
Arrhythmia (commonly recent onset uncontrolled atrial fibrillation)	
Severe obstructive or regurgitant valvular disease (may be clinically silent)	
Alcoholic cardiomyopathy	
'Idiopathic' dilated cardiomyopathy	
Viral myocarditis/pericarditis	
Severe, reversible, acute myocardial ischaemia	
Haemochromatosis	
Hypertrophic cardiomyopathy	
Cardiac amyloidosis	

acute heart failure such as chronic obstructive pulmonary disease, lower respiratory tract infection, pulmonary fibrosis or lymphangitis carcinomatosa. Alternatively, mild ankle oedema may be attributable to orthostatic oedema, venous varicosity or hypoalbuminaemia rather than heart failure.

INVESTIGATION

Basic investigation

Often treatment in acute severe left heart failure with pulmonary oedema must begin immediately, i.e. on the basis of a rapid clinical history and examination. A 12-lead electrocardiogram (ECG) and chest X-ray are, nevertheless, extremely valuable. As most cases of acute heart failure are the result of ischaemic heart disease, in the main, patients usually have a history of prior or current myocardial infarction and will have significant ECG abnormalities. Significant impairment of left ventricular function has been shown to be rare in the presence of a normal ECG (Davie et al, 1996), and should stimulate a search for alternative causes, particularly valvular disease, or alternative diagnoses. An ECG is also imperative to exclude acute myocardial infarction or arrhythmia-induced heart failure. Haemoglobin, urea and creatinine concentrations should be measured and cardiac enzyme analysis performed to exclude covert myocardial infarction.

TABLE 2.
Possible precipitants and other factors which may affect management

Unknown valvular disease
Myocardial infarction or ischaemia
Arrhythmias
Pericardial effusion
Pulmonary embolism
Infection
Anaemia
Drug induced

TABLE 3.
Therapy for acute heart failure

Pharmacological	Specific	Oxygen
		Nitrates
	Diuretics	Inotropes
		Dopamine
	Other	Digoxin
		Amiodarone
Non-pharmacological	Venesection	
	Continuous positive airways pressure	
	Ventilation	
	Intra-aortic balloon counterpulsation/mechanical assist	
	Surgery — including transplantation	
	Haemofiltration	
	Electrical cardioversion	
	Pacing	

Echocardiography

Echocardiography is invaluable in the assessment of possible new cases of heart failure. It should be considered in all patients as it allows simultaneous non-invasive assessment of left ventricular and valvular structure and function.

Other investigations

Plasma ferritin, thyroid function tests, glucose and liver function tests should be performed, later, in all new cases of possible heart failure without an obviously ischaemic cause. In selected patients, such as those with valvular disease or severe myocardial ischaemia, cardiac catheterization and/or transoesophageal echocardiography may be necessary if 'revascularization' or valve repair or replacement is being considered. Where the diagnosis is in doubt, and especially if significant LVSD has not been demonstrated by echocardiography, further investigations, e.g. pulmonary function tests, may be necessary.

IMMEDIATE MANAGEMENT

Aims and principles

The main aims of acute management of the patient with acute heart failure are:

1. Relieve anxiety and pain
2. Ensure adequate oxygenation (avoid hypoxaemia)
3. Maintain coronary, renal, cerebral and peripheral perfusion
4. Clear pulmonary oedema
5. Clear peripheral oedema
6. Re-establish stable cardiac status
7. Prevent recurrence.

Treatment options in acute heart failure are summarized in *Table 3* and a treatment algorithm according to presentation is shown in *Figure 1*.

General measures

Where the diagnosis is clear, severe left heart failure complicated by pulmonary oedema requires aggressive therapy. In all cases, patients should be sat upright and given unrestricted high concentration oxygen therapy (up to 100%). There is no rationale for restricting oxygen while arterial blood gas estimation is sought, even in those with

a history of chronic obstructive pulmonary disease, as hypoventilation is unlikely to occur in the acutely ill, well-observed patient. Intravenous (IV) fluids should be stopped and oral fluids restricted until clinical improvement occurs. 'Standard' pharmacological treatment for most patients should include IV opiates, IV loop diuretic and nitrate therapy given either intravenously or by the sublingual route depending upon circumstances.

Opiates: An opiate, usually diamorphine (2.5–10 mg in increments depending upon body size), should be given intravenously. This alleviates anxiety, and reduces the feelings of breathlessness and/or associated chest pain. It also reduces sympathetic outflow by central inhibition, thereby reducing counterproductive peripheral vasoconstriction. The antiemetic drug metoclopramide (normal dose 10mg IV) is given concomitantly to prevent vomiting. Cyclizine (and the combination agent cyclizine and morphine which is commonly administered in the community) should be avoided as this induces further peripheral vasoconstriction (Tan et al, 1988). Intramuscular injections should be avoided as absorption may be delayed, they interfere with cardiac enzyme analysis, and may cause a painful haematoma if thrombolysis is indicated.

Diuretics: An IV loop diuretic such as frusemide (50–100 mg slowly, or more in renal dysfunction) should also be administered. The mechanism of effect of IV diuretic in acute heart failure has long been debated. On balance, current evidence suggests that IV frusemide has an initially direct venodilator effect in humans, reducing preload, and contributing to an immediate clinical effect independent of its effects on urinary flow (Johnston et al, 1983; Pickkers et al, 1996; Biddle and Yu, 1979; Dikshit et al, 1973).

Nitrates: Nitrate therapy (glyceride trinitrate (GTN) or isosorbide dinitrate (ISDN)) appears to be an ideal treatment for acute heart failure and should be considered as routinely as frusemide. Nitrates reduce left ventricular filling pressure and relieve pulmonary oedema by venodilation. In addition, they improve myocardial ischaemia and chest pain by reducing myocardial oxygen demand through reductions in preload and afterload, and increase myocardial perfusion by direct coronary vasodilation.

Although the rationale for nitrate therapy is clear, there have been only two randomized trials of its effectiveness (Cotter et al, 1998; Nelson et al, 1983). Cotter et al (1998) showed that high dose ISDN given by intermittent bolus injection (3 mg every 5 minutes) was superior to high dose frusemide (80 mg bolus every 15 minutes) plus low dose infusion of ISDN in patients with acute heart failure with reduced requirement for mechanical ventilation (7/52 (13%) *vs* 21/52 (40%), $P<0.005$) or progression to myocardial infarction (9/52 (17%) *vs* 19/52 (37%)). In a previous study Nelson and colleagues (1983) demonstrated the haemodynamic profile of ISDN, given by infusion, to be preferable to IV frusemide when given to patients with heart failure following acute myocardial infarction.

How to give nitrates: In the community, ambulance or immediate arrival to the accident and emergency department, nitrates are most easily given sublingually (GTN spray 400 µg (2 puffs) every 5–10 minutes), or buccally (ISDN 1 or 3 mg) while monitoring blood pressure. In hospital the IV route is preferable for ease of control (i.e. GTN or ISDN 1–6 mg/hour), giving the maximum tolerated dose to control hypertension but avoid hypotension (systolic blood pressure <90–100 mmHg depending upon baseline). Nitrates should not be used in severe aortic stenosis as such patients may experience a profound hypotensive response as a result of vasodilation in the presence of a relatively fixed cardiac output.

MONITORING TREATMENT

In the majority of cases, patients with acute heart failure will improve rapidly with standard treatment and this will be obvious on clinical assessment. The following indices are useful in monitoring therapy.

- Oxygen saturation: by pulse oximetry >90% (preferably using the ear lobe as peripheral perfusion is often reduced making finger measurements inaccurate)
- Arterial pressure: every 10 minutes — keep systolic pressure >90 mmHg. May require alteration in nitrates or institution of inotropic therapy
- Heart rate/rhythm: preferably sinus rhythm and control of tachycardia (e.g. poorly controlled atrial fibrillation)
- Urinary flow: unless symptoms settle rapidly, urinary catheterization is valuable in monitoring — aim for a minimum of 30 ml/hour

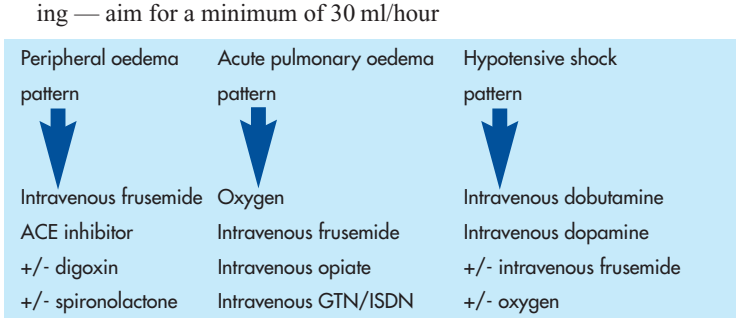

Figure 1. Standard management of 'acute heart failure' according to presentation.
ACE = angiotensin-converting enzyme; GTN = glyceride trinitrate; ISDN = isosorbide dinitrate.

- Chest X-ray: on admission, after 48–72 hours and thereafter according to response
- Pulmonary artery flotation catheter: to be considered where hypotension appears to complicate acute heart failure with a clear chest.

SPECIAL SITUATIONS

Mechanical complications of acute myocardial infarction

Potentially remediable mechanical complications such as papillary muscle rupture and ventricular septal perforation should always be sought in the patient developing acute heart failure after myocardial infarction. Echocardiography here is mandatory as these problems may be clinically silent. Such cases are best managed in a regional cardiothoracic centre where facilities such as intra-aortic balloon counterpulsation (IABP) and immediate cardiac surgical facilities are available.

Severe aortic stenosis

Acute left heart failure is nowadays not an uncommon presentation of severe aortic stenosis. Often a murmur is not heard or is very soft. Echocardiography may be required to make the diagnosis. Such patients are best managed with opiates and diuretics. Cardiac catheterization may be indicated as aortic valve replacement is usually well tolerated in this group, even in the very elderly (75–85 years) who otherwise have a very poor prognosis.

Severe mitral regurgitation

Occurring as a result of prosthetic valvular dysfunction, degenerative valvular disease or as a consequence of bacterial endocarditis, severe mitral regurgitation is best stabilized with vasodilators such as GTN and/or sodium nitroprusside with a view to valve replacement. Where there is likely to be a delay in valve replacement, or in those not suitable for surgery, angiotensin-converting enzyme inhibitors alleviate symptoms, especially in those with concomitant left ventricular dysfunction. In carefully selected patients the use of an IABP can be very valuable in this setting as a bridge to surgery.

Arrhythmia

Whenever possible sinus rhythm should be attained. Acute heart failure occurring in the context of ventricular tachycardia requires either immediate DC cardioversion or ventricular overdrive pacing. Development of new atrial fibrillation frequently leads to acute decompensation in those with chronic heart failure. Severely compromised patients should undergo immediate DC cardioversion. In less severe cases, and where the duration of atrial fibrillation is clearly <48 hours, IV heparin should be started immediately and an attempt to restore sinus rhythm with IV amiodarone (300 mg in 100 ml 5% glucose over 1 hour, then 900 mg in 1000 ml over 23 hours, in a large peripheral vein) should be considered. This appears safe even in those with pulmonary oedema and will control the ventricular rate quickly even in those who stay in atrial fibrillation.

In those who remain in atrial fibrillation beyond 24 hours, DC cardioversion should be considered early if the patient has received heparin within 48 hours of onset of the arrhythmia, or otherwise as a deferred procedure following at least a month's treatment with warfarin, at a dose to attain an international normalized ratio of 2.0 to 3.0. In the mean time the ventricular rate should be controlled with digoxin or a β-blocker for those without significant LVSD.

Renal dysfunction

Transient renal dysfunction, or worsening of previous chronic renal failure, is a common accompaniment to the treatment of acute heart failure. Where this limits diuretic therapy and pulmonary oedema or severe peripheral oedema persist, consideration should be given to haemofiltration or dialysis.

WHAT TO DO IF ALL ELSE FAILS?

Maintained blood pressure

In the absence of complicating factors (e.g. obstructive valvular disease, complications of a recent myocardial infarction, marked hypotension (systolic blood pressure <90 mmHg)), venesection still has a place in life-threatening pulmonary oedema. Remove no more than one unit of blood, using a venesection pack or a large IV cannula attached to a reversed blood giving set, while carefully monitoring blood pressure and clinical response.

Significant symptomatic hypotension

The occurrence of hypotension (systolic blood pressure <90 mmHg) complicating pulmonary oedema is an ominous clinical sign. When accompanied by cool underperfused skin and evidence of end-organ hypoperfusion (oliguria or anuria, confusion and agitation), mortality approaches 90% (Goldberg et al, 1999). Inotropic agents further increase myocardial oxygen demand, exacerbate myocardial ischaemia, and dopamine has been shown to worsen hypoxaemia (Tanaka et al, 1992). They should therefore only be used when absolutely essential.

Peripheral perfusion and urinary flow are of greater importance than blood pressure per se;

however, in severe hypotension, dobutamine (5 μg/kg/min and titrated upwards to achieve adequate vital organ perfusion) can be used in the absence of prominent tachycardia. Low dose dopamine (2.5 μg/kg/min) can improve urine output but does not improve renal function. Higher doses are rarely helpful as alpha-adrenergic effects exacerbate peripheral vasoconstriction. In clinical practice, a combination of both of these is often used. Recent unpublished data suggest that the calcium sensitiser, levosimendan, may increase survival compared to both placebo and dobutamine.

Resistant pulmonary oedema/hypoxaemia

Continuous positive airways pressure of 5–10 cmH_2O given by a tight-fitting facemask can be very effective in assisting clearance of pulmonary oedema and relieving hypoxaemia. Ventilation should be considered at an early stage if conservative measures are failing in those with potentially correctable mechanical causes, such as papillary muscle rupture.

Transplantation

Younger patients with intractable heart failure should be considered for urgent heart transplantation. Those without significant concomitant disease and preserved renal function stand the best chance of success and should be discussed with the regional transplant unit.

FURTHER MANAGEMENT

The morbidity and mortality of chronic heart failure is considerable with mortality approaching 50% at 12 months for those most severely affected patients with rest symptoms (New York Heart Association class IV). Prevention of re-occurrence with attention to risk factors and appropriate drug therapy aimed to improve symptoms and prognosis is therefore essential in all cases. A specialist should follow up all patients surviving acute heart failure for consideration of further investigation and drug therapy (e.g. angiotensin-converting enzyme inhibitors, spironolactone, β-blockers, statin therapy). Possible precipitants, such as non-steroidal anti-inflammatory drugs, diltiazem and verapamil, alcohol and chest infection (consider flu and pneumococcal vaccination), should be avoided. There is now accumulating evidence that follow-up of patients hospitalized with acute heart failure by specialist nurses reduces recurrence and readmission (Stewart et al, 1999).

CONCLUSIONS

Acute heart failure is a common medical emergency requiring rapid assessment and prompt treatment. Physicians should be aware of common causes and special situations which require specialist management. Optimization of drug therapy and avoidance of precipitants is essential to prevent re-occurrence and improve prognosis. HM

Conflict of interest: none.

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KEY POINTS

- Acute heart failure is a common medical emergency requiring prompt diagnosis and treatment.
- Most patients with acute heart failure will have ischaemic heart disease, and the majority of these a history of prior myocardial infarction.
- In severe pulmonary oedema, treatment must begin immediately on the basis of history and examination, otherwise the electrocardiogram and chest X-ray are invaluable guides to initial management.
- 'Standard' medical inpatient therapy for confirmed acute heart failure should now include nitrate therapy (preferably intravenous) in addition to a combination of oxygen and intravenous opiates and diuretics.
- Be aware of special situations that demand alternative or additional treatment, e.g. pulmonary embolism, severe aortic stenosis or complications of myocardial infarction.
- Monitor the effects of treatment with simple clinical measures including pulse, blood pressure, oxygen saturation and urinary flow.
- All patients should undergo echocardiography to confirm left ventricular systolic dysfunction and/or possible concomitant valvular disease.
- Prevention of recurrence is best achieved through a multidisciplinary approach of lifestyle changes, drug treatment and specialist medical and nursing input.