Hydralazine can be administered orally to patients with chronic cardiac failure, but in intensive care practice it is usually given as an intravenous bolus (5–10 mg) to control acute increases in blood pressure, particularly after cardiac surgery.

**CALCIUM ANTAGONISTS**

The calcium channel blocker nifedipine can be administered orally or sublingually to control hypertension. It is a particularly potent peripheral smooth-muscle relaxant that causes a reduction in systemic vascular resistance and an increase in cardiac output. Most of the increased flow is distributed to musculoskeletal beds, with lesser increases in hepatic, splanchnic and renal flow.

**ANGIOTENSIN-CONVERTING ENZYME INHIBITORS**

ACE inhibitors such as captopril and enalapril reduce systemic vascular resistance and generally increase renal blood flow by reducing angiotensin-induced arteriolar tone. This effect is, however, more pronounced in the efferent vessels, and GFR may therefore remain unchanged or fall despite the increased flow. Nevertheless, sodium excretion usually increases, largely due to a reduction in aldosterone release. ACE inhibitors can be useful when weaning patients with cardiac failure from intravenous vasodilators.

**Note:** when used in patients with respiratory failure most inotropes and vasoactive agents increase venous admixture; this may be due simply to passive opening of pulmonary vessels by the increased flow and pressure or to a specific reversal of the hypoxic vasoconstrictor response.

**Mechanical circulatory assistance**

**INTRA-AORTIC BALLOON COUNTERPULSATION**

Various techniques for mechanically supporting the failing myocardium have been described; of these, intra-aortic balloon counterpulsation (IABCP) has proved most practical and is now widely used.

A catheter with an inflatable sausage-shaped balloon is inserted via a femoral artery using a percutaneous Seldinger technique and passed into the aorta until its tip lies just distal to the left subclavian artery (Fig. 5.17). If the Seldinger

![Fig. 5.17 Intra-aortic balloon counterpulsation (IABCP). (a) Rapid deflation of the balloon occurs at the onset of systole and causes a reduction in afterload. Early in diastole the balloon is rapidly inflated to increase the pressure in the aortic root and enhance coronary blood flow. (b) An intra-aortic balloon pump and console.](image-url)
technique fails, an open surgical approach can be used. Fluoroscopy makes insertion safer and allows accurate positioning of the catheter tip. In the absence of fluoroscopy the position of the catheter must be verified by chest X-ray. Occasionally, the balloon is inserted via the ascending aorta in those undergoing cardiac surgery, whilst in other cases the axillary or subclavian arteries are used.

Early in diastole, the balloon is rapidly inflated so that the pressure in the aortic root rises and coronary blood flow is increased. Rapid deflation of the balloon is timed to occur at the onset of systole (usually triggered by the R wave on the ECG) and this leads to a reduction in afterload and left ventricular wall tension. Preload, pulmonary artery pressure and pulmonary vascular resistance may also be reduced. Heart rate is usually unchanged. The reduction in left ventricular work reduces myocardial oxygen requirements and this, combined with the increased coronary blood flow, may result in a reversal of ischaemic changes and limitation of infarct size. IABCP may not, however, increase coronary blood flow distal to significant stenoses. Improved myocardial performance, together with the reduction in afterload, can lead to an increased cardiac output. Inflation and deflation of the balloon must be precisely timed to achieve effective counterpulsation (Fig. 5.18).

The only absolute contraindications to IABCP are aortic aneurysms (or other severe disease of the descending aorta) and significant aortic regurgitation. Relative contraindications include arrhythmias and extreme tachycardias, both of which limit the ability of the device to trigger balloon deflation accurately, and severe peripheral vascular disease.

IABCP is associated with significant morbidity. Complications include:

- failure to pass the balloon (usually in those with severe atheromatous disease);
- arterial dissection;
- limb ischaemia;
- thrombosis;
- embolism;
- infection;
- a distally positioned balloon may intermittently occlude the renal vasculature;
- balloon rupture is rare.

In order to minimize the risk of thrombosis on the surface of the balloon the patient should be heparinized to achieve a partial thromboplastin time of about 1 ½ times normal and the balloon should be kept in motion. Because there is a tendency to develop thrombocytopenia, the platelet count should be carefully monitored.

IABCP has proved most useful for weaning patients from cardiopulmonary bypass and for those who develop myocardial ischaemia in the perioperative period. It may also be used to support patients in cardiogenic shock who have surgically correctable lesions, such as ischaemic ventricular septal defects or mitral regurgitation, while they are being prepared for surgery. In patients with severe ischaemia (e.g. those with unstable angina) IABCP may relieve pain and possibly prevent infarction while preparations are made for coronary artery bypass grafting or percutaneous transluminal coronary angioplasty (PTCA) (see Chapter 9). The use of IABCP for the treatment of cardiogenic shock complicating myocardial infarction without a surgically correctable lesion has been less successful, although some feel that this may be due partly to delay in instituting IABCP and extension of the infarct caused by the prior use of inotropes. Although there are no PRCT data to indicate that IABCP can improve outcome in cardiogenic shock, it has been suggested that the technique might be beneficial when used in conjunction with revascularization, either surgically or by coronary angioplasty (Hasdai et al., 2000).

IABCP may also be indicated in patients with a failing transplanted heart and in those with viral myocarditis. IABCP is occasionally used prophylactically in patients undergoing high-risk cardiac surgery or PTCA. There is only limited experience with the use of IABCP in myocardial failure complicating septic shock, but it seems that the technique may be life-saving in those with reversible global myo-

Fig. 5.18 (a) The effect of intra-aortic balloon counterpulsation (IABCP) on the arterial pressure trace. Note the increased diastolic pressure, the slight fall in systolic pressure (due to afterload reduction) and reduced end-diastolic pressure (due to rapid balloon deflation). (b) It is important that the balloon inflates immediately the aortic valve closes (i.e. on the dicrotic notch). In this case the balloon is inflating too early. This impedes left ventricular ejection. (c) In this case balloon inflation is delayed.
cardial dysfunction complicating anaphylaxis (Raper and Fisher, 1988).

OTHER VENTRICULAR ASSIST DEVICES
Centrifugal pumps can be used to bypass and ‘off-load’ the right or, more often, the left ventricle while maintaining adequate systemic blood flow. Such devices are usually used as a means of discontinuing cardiopulmonary bypass in the hope that ventricular function will improve significantly over the ensuing 48–72 hours. They may also be used as a ‘bridge’ to cardiac transplantation. Alternative devices include mechanical hearts, used to support potential cardiac transplant recipients and arteriovenous pumps.

HAEMATOLOGICAL PROBLEMS
The most commonly encountered haematological problem in shock is a coagulation defect, usually due to massive blood transfusion and/or consumption coagulopathy (DIC), the latter being particularly common in sepsis (see above). This can usually be corrected with transfusions of fresh frozen plasma, occasionally combined with platelets, or cryoprecipitate. DIC should be prevented or reversed by aggressive treatment of the underlying condition. Heparinization has been recommended, but this is potentially extremely dangerous, particularly in trauma victims and postoperative patients, and is rarely indicated. Later, coagulation may be impaired secondary to the development of renal and/or hepatic failure.

RENAL FUNCTION
Prevention of acute intrinsic renal failure is best achieved by restoring cardiac output, blood pressure and renal blood flow as rapidly as possible, combined with early and aggressive management of oliguria (see Chapter 13).

MULTIPLE-ORGAN FAILURE
Although it is now usually possible to support patients through the early stages of shock, trauma or other life-threatening illnesses, a disappointingly high proportion subsequently develop progressive failure of several vital organs (Barton and Cerra, 1989; DeCamp and Demling, 1988). Mortality is high and correlates with the number of organs which fail; those with prolonged three-system failure have a mortality rate exceeding 50%, whilst more than 90% of those with prolonged six-system failure die. Sequential MOF is now the commonest mode of death in intensive care patients, accounting for as many as 75% of all deaths in surgical ICUs (Barton and Cerra, 1989). Moreover, these patients spend long periods in intensive care and consume a large proportion of available resources.

DEFINITION
Currently there is no consensus on the definition of the MOF syndrome and criteria for failure of individual organs differ. Because the extent of organ dysfunction can vary both between patients and within the same patient over time, the term multiple-organ dysfunction syndrome (MODS) has been suggested to indicate the wide range of severity and dynamic nature of this disorder (Bone et al., 1992; Members of the American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference, 1992). Organ dysfunction is defined as ‘the presence of altered organ function in an acutely ill patient such that homeostasis cannot be maintained without intervention’. Transient impairment of organ function rapidly responsive to short-term measures should probably be excluded from this definition.

AETIOLOGY AND CAUSES (see also pp. 96–99)
There are two distinct, but not mutually exclusive, pathways by which MODS may develop (Fig. 5.19).

- In primary MODS there is a direct insult to an individual organ (e.g. pulmonary aspiration, lung contusion or renal damage due to rhabdomyolysis), which fails early as the result of an inflammatory response that is confined, at least in the early stages, to the affected organ.
- The aetiology of secondary MODS is complex but, in general terms, organ damage is thought to be precipitated by systemic dissemination of a poorly controlled inflammatory/anti-inflammatory response associated with haemodynamic disturbance, microcirculatory abnormalities and defective oxygen utilization, as described earlier in this chapter. However, the precise mechanisms responsible for organ damage have yet to be determined. Secondary MODS can therefore be viewed as a complication of SIRS (see above) and can be considered as representing the more severe end of the spectrum of illness of patients with SIRS/sepsis/septic shock. Secondary MODS usually evolves after a latent period from the initiating event, which is most commonly infectious (usually bacterial, but sometimes viral, fungal or parasitic), but may be non-infectious (e.g. the presence of extensive wounds or necrotic tissue). Secondary MODS may therefore follow sepsis, trauma, shock, major surgery and many other serious illnesses (e.g. pancreatitis, perforation of the gastrointestinal tract, pneumonia), or may arise in response to SIRS precipitated by primary MODS.

![Fig. 5.19 Pathways for the development of multiple-organ dysfunction syndrome (MODS). SIRS, systemic inflammatory response syndrome.](image-url)


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