

# CHAPTER 9

## CIRCULATORY FAILURE

### SHOCK

Shock is a wonderful word! It means such different things to medical and lay people. How often we hear news reports that someone has suffered from shock after witnessing some tragic event. No doubt that person is surprised and possibly emotionally disturbed but they are not in a state of **circulatory collapse**, which is what a doctor regards as shock.

The 'shocked' patient is desperately ill and requires intensive treatment both to correct the condition that has produced the circulatory collapse and also to cope with the widespread ischaemic damage resulting from shock. By definition, the patient will have hypoperfusion of many tissues. Blood pressure may be low but need not be, as the patient may either have compensated by increasing peripheral vasoconstriction to keep the pressure normal or may have had a high blood pressure which has now dropped. There may be pallor, cold extremities, sweating and a tachycardia; the first two signs due to poor perfusion and the other two resulting from the attempt to compensate, which includes the release of adrenaline.

What has happened to precipitate this disastrous state? Well, logically, there will be a sudden generalized poor perfusion if the pump fails or if there is insufficient blood, so-called **cardiogenic** and **hypovolaemic** shock (Fig 9.1). Abrupt heart failure may result from myocardial infarction, arrhythmias and cardiac tamponade while hypovolaemic shock follows fluid loss due to haemorrhage, severe burns, diarrhoea or vomiting. Shock following pulmonary embolism mimics cardiogenic shock but the heart is normal and the reduced output is because the left atrial filling has dropped (Fig 9.2). A rather special but clinically very important form of shock is '**septic shock**' due to overwhelming infection, especially those caused by Gram-negative bacteria which have endotoxic lipopolysaccharides (see page 33).



### Key facts

Important mediators of vascular dilatation and increased permeability in septic shock

- Histamine
- Thromboxane
- Serotonin
- Prostaglandins
- Leukotrienes
- TNF- $\alpha$
- IL-1 $\alpha$
- C3a and C5a

Here the pathogenesis is complicated because of the varied effects of the bacterial products on endothelial cells, platelets and leucocytes which leads to a veritable web of interactions resulting in disseminated intravascular coagulation (DIC) and reduced blood volume because of vasodilatation and increased vascular permeability. Similar mechanisms probably operate in **anaphylactic shock** and **neurogenic shock**.

Let us concentrate first on cardiogenic shock as this follows naturally from the previous chapter on atherosclerosis and hypertension.

### CLINICAL CASE: MYOCARDIAL INFARCTION

Let us consider the clinical aspects. A typical scenario may be as follows. A 63-year-old man presented to the Accident and Emergency department complaining of chest pain. He had had central chest pain for 1–2 hours and the pain radiated down the left arm and into his