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There are numerous ways to classify shock, but the most common and most clinically applicable is one based on the initiating mechanism. All states are characterised by systemic tissue hypoperfusion, and di ff erent states may coexist within the same patient. Summary box 2.1 Classification of shock /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Haemorrhagic and hypovolaemic shock Hypovolaemic shock is due to a reduced circulating volume. Hypovolaemia may be due to haemorrhagic or non- haemorrhagic causes. Non-haemorrhagic causes include poor fluid intake (dehydration), excessive fluid loss due to vomiting, diarrhoea, urinary loss (e.g. diabetes), evaporation or 'third-spacing', where fluid is lost into the gastrointestinal tract and interstitial spaces, as for example in bowel obstruction or pancreatitis. Hypovolaemia is the most common form of shock, and to some degree is a component of all other forms of shock. Absolute or relative hypovolaemia must be excluded or treated in the management of the shocked state, regardless of cause. Cardiogenic shock Cardiogenic shock is due to primary failure of the heart to pump blood to the tissues. Causes of cardiogenic shock include myocardial infarction, cardiac dysrhythmias, valvular heart disease, blunt myocardial injury and cardiomyopathy . Cardiac insu ffi ciency may also be due to myocardial depression caused by endogenous factors (e.g. bacterial and humoral agents agents or drug abuse. Evidence of venous hypertension with pulmonary or systemic oedema may coexist with the classical signs of shock. - Obstructive shock In obstructive shock there is a reduction in preload owing to mechanical obstruction of cardiac filling. Common causes of obstructive shock include cardiac tamponade, tension pneumothorax, massive pulmonary embolus or air embolus. In each case, there is reduced filling of the left and/or right sides of the heart, leading to low cardiac output. Distributive shock Distributive shock describes the pattern of cardiovascular responses characterising a variety of conditions, including septic shock, anaphylaxis and spinal cord injury . Inadequate organ perfusion is accompanied by vascular dilatation with hypotension, low systemic vascular resistance, inadequate afterload and a resulting abnormally high cardiac output. In anaphylaxis, vasodilatation is due to histamine release, while in high spinal cord injury there is failure of sympathetic outflow and adequate vascular tone (neurogenic shock). The cause in sepsis is less clear but is related to the release of bac - terial products (endotoxin) and the activation of cellular and humoral components of the immune system. There is mal- distribution of blood flow at a microvascular level, with arte - riovenous shunting and dysfunction of cellular utilisation of oxygen. In the later phases of septic shock there is hypovolaemia from fluid loss into interstitial spaces and there may be con - comitant my ocardial depression, complicating the clinical pic - ture (Table 2.1). Endocrine shock Endocrine shock may present as a combination of hypovolae- mic, cardiogenic or distributive shock. Causes of endocrine shock include hypo- and hyperthyroidism and adrenal insuf - ficiency . Hypothyroidism causes a shock state similar to that of neurogenic shock due to disordered vascular and cardiac responsiveness to circulating catecholamines. Cardiac output falls as a result of low inotropy and bradycardia. There may also be an associated cardiomyopathy . Thyrotoxicosis may cause a high-output cardiac failure. Adrenal insu ffi ciency leads to shock due to hypovolae- mia and a poor

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Haemorrhagic/hypovolaemic shock Cardiogenic shock Obstructive shock Distributive shock Endocrine shock TABLE 2.1 Cardiovascular and metabolic characteristics of shock. Hypovolaemic Cardiac output Low Systemic vascular resistance High Venous pressure Low Mixed venous saturation Low Base de /f_i cit High Cardiogenic Obstructive Distributive Low Low High High High Low High High Low Low Low High High High High

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