Table 40-1 Classification of Shock and Common Underlying Causes

TYPE	PRIMARY CIRCULATORY DERANGEMENT	COMMON CAUSES
Hypovolemic	Decreased circulating blood volume	Hemorrhage Diarrhea Diabetes insipidus, diabetes mellitus Burns Adrenogenital syndrome Capillary leak
Distributive	Vasodilation → venous pooling → decreased preload	Sepsis
	Maldistribution of regional blood flow	Anaphylaxis CNS/spinal injury Drug intoxication
Cardiogenic	Decreased myocardial contractility	Congenital heart disease Arrhythmia Hypoxic/ischemic injuries Cardiomyopathy Metabolic derangements Myocarditis Drug intoxication Kawasaki disease
Obstructive	Mechanical obstruction to ventricular filling or outflow	Cardiac tamponade Massive pulmonary embolus Tension pneumothorax Cardiac tumor
Dissociative	Oxygen not appropriately bound or released from hemoglobin	Carbon monoxide poisoning Methemoglobinemia

CNS, Central nervous system.

contractility. Neurohormonally mediated constriction of the arterioles and capacitance vessels maintains blood pressure, augments venous return to the heart to improve preload, and redistributes blood flow from nonvital to vital organs. If hypovolemic shock remains untreated, the increased heart rate may impair coronary blood flow and ventricular filling, while elevated systemic vascular resistance increases myocardial oxygen consumption, resulting in worsening myocardial function. Ultimately, intense systemic vasoconstriction and hypovolemia produce tissue ischemia, impairing cell metabolism and releasing potent vasoactive mediators from injured cells. Cytokines and other vasoactive peptides can change myocardial contractility and vascular tone and promote release of other inflammatory mediators that increase capillary permeability and impair organ function further.

DISTRIBUTIVE SHOCK

Abnormalities in the distribution of blood flow may result in profound inadequacies in tissue perfusion, even in the presence of a normal or high cardiac output. This maldistribution of flow usually results from abnormalities in vascular tone. Septic shock is the most common type of distributive shock in children. Other causes include anaphylaxis, neurologic injury, and drug-related causes (see Table 40-1).

Distributive shock may present with the **systemic inflammatory response syndrome** (SIRS), defined as two or more of the following: temperature greater than 38° C or less than 36° C; heart rate greater than 90 beats/min or more than two standard deviations above normal for age; tachypnea; or white blood count greater than 12,000 cells/mm^3, less than 4000 cells/mm^3, or greater than 10% immature forms.

CARDIOGENIC SHOCK

Cardiogenic shock is caused by an abnormality in myocardial function and is expressed as depressed myocardial contractility and cardiac output with poor tissue perfusion. Compensatory mechanisms may contribute to the progression of shock by depressing cardiac function further. Neurohormonal vasoconstrictor responses increase afterload and add to the work of the failing ventricle. Tachycardia may impair coronary blood flow, which decreases myocardial oxygen delivery. Increased central blood volume caused by sodium and water retention and by incomplete emptying of the ventricles during systole results in elevated left ventricular volume and pressure, which impair subendocardial blood flow. As compensatory mechanisms are overcome, the failing left ventricle produces increased ventricular end-diastolic volume and pressure, which leads to increased left atrial pressure, resulting in pulmonary edema. This sequence also contributes to right ventricular failure because of increased pulmonary artery pressure and increased right ventricular afterload.

Primary cardiogenic shock may occur in children who have congenital heart disease. Cardiogenic shock also may occur in previously healthy children secondary to viral myocarditis, dysrhythmias, or toxic or metabolic abnormalities or after hypoxic-ischemic injury (see Chapters 142, 145, and 147, as well as Table 40-1).

OBSTRUCTIVE SHOCK

Obstructive shock results from mechanical obstruction of ventricular outflow. Causes include congenital lesions such as coarctation of the aorta, interrupted aortic arch, and severe aortic valvular stenosis, along with acquired diseases (e.g., hypertrophic cardiomyopathy) (see Table 40-1). For neonates presenting in shock, obstructive lesions must be considered.

DISSOCIATIVE SHOCK

Dissociative shock refers to conditions in which tissue perfusion is normal, but cells are unable to use oxygen because the hemoglobin has an abnormal affinity for oxygen, preventing its release to the tissues (see Table 40-1).

CLINICAL MANIFESTATIONS

All forms of shock produce evidence that tissue perfusion and oxygenation are insufficient (increased heart rate, abnormal blood pressure, alterations of peripheral pulses). The etiology of shock may alter the initial presentation of these signs and symptoms.