TABLE 324-2 NORMAL HEMODYNAMIC PARAMETERS

Parameter	Calculation	Normal Values
Cardiac output (CO)	SV × HR	4–8 L/min
Cardiac index (CI)	CO/BSA	2.6–4.2 (L/min)/m^2
Stroke volume (SV)	CO/HR	50–100 mL/beat
Systemic vascular resistance (SVR)	([MAP – RAP]/CO) × 80	700–1600 dynes·s/cm^5
Pulmonary vascular resistance (PVR)	([PAP_m – PCWP]/CO) × 80	20–130 dynes·s/cm^5
Left ventricular stroke work (LVSW)	SV(MAP – PCWP) × 0.0136	60–80 g-m/beat
Right ventricular stroke work (RVSW)	SV(PAP_m – RAP)	10–15 g-m/beat

Abbreviations: BSA, body surface area; HR, heart rate; MAP, mean arterial pressure; PAP_m, pulmonary artery pressure—mean; PCWP, pulmonary capillary wedge pressure; RAP, right atrial pressure.

oxygen consumption, and oxygen-extraction ratio (Table 324-3). The hemodynamic patterns associated with the various forms of shock are shown in Table 324-4.

In resuscitation from shock, it is critical to restore tissue perfusion and optimize oxygen delivery, hemodynamics, and cardiac function rapidly. A reasonable goal of therapy is to achieve a normal mixed venous oxygen-saturation and arteriovenous oxygen-extraction ratio. To enhance oxygen delivery, red cell mass, arterial oxygen saturation, and cardiac output may be augmented singly or simultaneously. An increase in oxygen delivery not accompanied by an increase in oxygen consumption implies that oxygen availability is adequate and that oxygen consumption is not flow dependent. Conversely, an elevation of oxygen consumption with increased delivery implies that the oxygen supply was inadequate. However, cautious interpretation is required due to the link among increased oxygen delivery, cardiac work, and oxygen consumption. A reduction in systemic vascular resistance accompanying an increase in cardiac output indicates that compensatory vasoconstriction is reversing due to improved tissue perfusion. The determination of stepwise expansion of blood volume on cardiac performance allows identification of the optimum preload (Starling's law). An algorithm for the resuscitation of the patient in shock is shown in Fig. 324-3.

TABLE 324-3 OXYGEN TRANSPORT CALCULATIONS

Parameter	Calculation	Normal Values
Oxygen-carrying capacity of hemoglobin		1.39 mL/g
Plasma O_2 concentration		Po_2 × 0.0031
Arterial O_2 concentration (Cao_2)	1.39 Sao_2 + 0.0031 Pao_2	20 vol%
Venous O_2 concentration (Cvo_2)	1.39 Svo_2 + 0.0031 Pvo_2	15.5 vol%
Arteriovenous O_2 difference (Cao_2 – Cvo_2)	1.39 (Sao_2 – Svo_2) + 0.0031 (Pao_2 – Pvo_2)	3.5 vol%
Oxygen delivery (Do_2)	Cao_2 × CO (L/min) × 10 (dL/L) 1.39 Sao_2 × CO × 10	800–1600 mL/min
Oxygen uptake (Vo_2)	(Cao_2 – Cvo_2) × CO × 10 1.39 (Sao_2 – Svo_2) × CO × 10	150–400 mL/min
Oxygen delivery index (Do_2I)	Do_2/BSA	520–720 (mL/min)/m^2
Oxygen uptake index (Vo_2I)	Vo_2/BSA	115–165 (mL/min)/m^2
Oxygen extraction ratio (O_2ER)	[1 – ($\dot{V}o_2$/$\dot{D}o_2$)] × 100	22–32%

Abbreviations: BSA, body surface area; CO, cardiac output; Po_2, partial pressure of oxygen; Pao_2, partial pressure of oxygen in arterial blood; Pvo_2, partial pressure of oxygen in venous blood; Sao_2, saturation of hemoglobin with oxygen in arterial blood; Svo_2, saturation of hemoglobin with oxygen in venous blood.

TABLE 324-4 PHYSIOLOGIC CHARACTERISTICS OF THE VARIOUS FORMS OF SHOCK

Type of Shock	CVP and PCWP	Cardiac Output	Systemic Vascular Resistance	Venous O_2 Saturation
Hypovolemic	↓	↓	↑	↓
Cardiogenic	↑	↓	↑	↓
Septic				
Hyperdynamic	↓↑	↑	↓	↑
Hypodynamic	↓↑	↓	↑	↓↑
Traumatic	↓	↓↑	↑↓	↓
Neurogenic	↓	↓	↓	↓
Hypoadrenal	↓	↓	=↓	↓

Abbreviations: CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure.

SPECIFIC FORMS OF SHOCK

HYPOVOLEMIC SHOCK

This most common form of shock results either from the loss of red blood cell mass and plasma from hemorrhage or from the loss of plasma volume alone due to extravascular fluid sequestration or GI, urinary, and insensible losses. The signs and symptoms of nonhemorrhagic hypovolemic shock are the same as those of hemorrhagic shock, although they may have a more insidious onset. The normal physiologic response to hypovolemia is to maintain perfusion of the brain and heart while attempting to restore an effective circulating blood volume. There is an increase in sympathetic activity, hyperventilation, collapse of venous capacitance vessels, release of stress hormones, and an attempt to replace the loss of intravascular volume through the recruitment of interstitial and intracellular fluid and by reduction of urine output.

Mild hypovolemia (≤20% of the blood volume) generates mild tachycardia but relatively few external signs, especially in a supine young patient (Table 324-5). With moderate hypovolemia (~20–40% of the blood volume), the patient becomes increasingly anxious and tachycardic; although normal blood pressure may be maintained in the supine position, there may be significant postural hypotension and tachycardia. If hypovolemia is severe (≥40% of the blood volume), the classic signs of shock appear; the blood pressure declines and becomes unstable even in the supine position, and the patient develops marked tachycardia, oliguria, and agitation or confusion. Perfusion of the central nervous system is well maintained until shock becomes severe. Hence, mental obtundation is an ominous clinical sign. The transition from mild to severe hypovolemic shock can be insidious or extremely rapid. If severe shock is not reversed rapidly, especially in elderly patients and those with comorbid illnesses, death is imminent. A very narrow time frame separates the derangements found in severe shock that can be reversed with aggressive resuscitation from those of progressive decompensation and irreversible cell injury.

Diagnosis Hypovolemic shock is readily diagnosed when there are signs of hemodynamic instability and the source of volume loss is obvious. The diagnosis is more difficult when the source of blood loss is occult, as into the GI tract, or when plasma volume alone is depleted. Even after acute hemorrhage, hemoglobin and hematocrit values do not change until compensatory fluid shifts have occurred or exogenous fluid is administered. Thus, an initial normal hematocrit does not disprove the presence of significant blood loss. Plasma losses cause hemoconcentration, and free water loss leads to hypernatremia. These findings should suggest the presence of hypovolemia.

It is essential to distinguish between hypovolemic and cardiogenic shock (Chap. 326) because, although both may respond to volume initially, definitive therapy differs significantly. Both forms are associated with a reduced cardiac output and a compensatory sympathetic mediated response characterized by tachycardia and elevated systemic vascular resistance. However, the findings in cardiogenic shock of jugular venous distention, rales, and an S_3 gallop distinguish it from hypovolemic shock and signify that ongoing volume expansion is undesirable and may cause further organ dysfunction.