Hypovolemic Shock

Hypovolemic shock is distinguished from other causes of shock by history and the absence of signs of heart failure or sepsis. In addition to the signs of sympathoadrenal activity (tachycardia, vasoconstriction), clinical manifestations include signs of dehydration (dry mucous membranes, decreased urine output) or blood loss (pallor). Recovery depends on the degree of hypovolemia, the patient's preexisting status, and rapid diagnosis and treatment. The prognosis is good, with a low mortality in uncomplicated cases.

Distributive Shock

Patients with distributive shock usually have tachycardia and alterations of peripheral perfusion. In early stages, when cytokine release results in vasodilation, pulses may be bounding, and vital organ function may be maintained (an alert patient, with rapid capillary refill and some urine output in *warm shock*). As the disease progresses untreated, extremities become cool and mottled with a delayed capillary refill time. At this stage, the patient has hypotension and vasoconstriction. If the etiology of distributive shock is sepsis, the patient often has fever, lethargy, petechiae, or purpura, and he or she may have an identifiable source of infection.

Cardiogenic Shock

Cardiogenic shock results when the myocardium is unable to supply the cardiac output required to support tissue perfusion and organ function. Because of this self-perpetuating cycle, heart failure progressing to death may be rapid. Patients with cardiogenic shock have tachycardia and tachypnea. The liver is usually enlarged, a gallop is often present, and jugular venous distention may be noted. Because renal blood flow is poor, sodium and water are retained, resulting in oliguria and peripheral edema.

Obstructive Shock

Restriction of cardiac output results in an increase in heart rate and an alteration of stroke volume. The pulse pressure is narrow (making pulses harder to feel), and capillary refill is delayed. The liver is often enlarged, and jugular venous distention may be evident.

Dissociative Shock

The principal abnormality in dissociative shock is the inability to deliver oxygen to tissues. Symptoms include tachycardia, tachypnea, alterations in mental status, and ultimately cardiovascular collapse.

LABORATORY AND IMAGING STUDIES

Shock requires immediate resuscitation before obtaining laboratory or diagnostic studies. Following initial stabilization (including glucose administration if hypoglycemia is present), the type of shock dictates the necessary laboratory studies. All patients with shock may benefit from determination of a baseline arterial blood gas and blood lactate level to assess the impairment of tissue oxygenation. Measurement of **mixed venous oxygen saturation** aids in the assessment of the adequacy of oxygen delivery. In contrast to other forms of shock, patients with sepsis often have high mixed venous saturation values because of impairment of mitochondrial function and inability of tissues to extract oxygen. A complete blood count can potentially assess intravascular blood volume after equilibration following a hemorrhage. Electrolyte measurements in patients with hypovolemic shock may identify abnormalities from losses. Patients presenting in distributive shock require appropriate bacterial and viral cultures to identify a cause of infection. If cardiogenic or obstructive shock is suspected, an echocardiogram assists with the diagnosis and, in the case of tamponade, assists with placement of a pericardial drain to relieve the fluid. Patients with dissociative shock require detection of the causative agent (carbon monoxide, methemoglobin). The management of shock also requires monitoring of arterial blood gases for oxygenation, ventilation (CO_2), and acidosis, and frequently assessing the levels of serum electrolytes, calcium, magnesium, phosphorus, and blood urea nitrogen (BUN).

DIFFERENTIAL DIAGNOSIS

SEE TABLE 40-1.

TREATMENT

General Principles

The key to therapy is the recognition of shock in its early, partially compensated state, when many of the hemodynamic and metabolic alterations may be reversible. Initial therapy for shock follows the ABCs of resuscitation. Later therapy can then be directed at the underlying cause. Therapy should minimize cardiopulmonary work, while ensuring cardiac output, blood pressure, and gas exchange. Intubation, combined with mechanical ventilation with oxygen supplementation, improves oxygenation and decreases or eliminates the work of breathing but may impede venous return if distending airway pressures (positive end-expiratory pressure [PEEP] or peak inspiratory pressure) are excessive. Blood pressure support is crucial because the vasodilation in sepsis may reduce perfusion despite supranormal cardiac output.

Monitoring a child in shock requires maintaining access to the arterial and central venous circulation to record pressure measurements, perform blood sampling, and measure systemic blood pressure continuously. These measurements facilitate the estimation of preload and afterload. Regional monitoring with near infrared spectroscopy may allow early, noninvasive detection of alterations in perfusion.

Organ-Directed Therapeutics

Fluid Resuscitation

Alterations in preload have a dramatic effect on cardiac output. In hypovolemic and distributive shock, decreased preload significantly impairs cardiac output. In these cases, early and aggressive fluid resuscitation is important and greatly affects outcome. In cardiogenic shock, an elevated preload contributes to pulmonary edema.

Selection of fluids for resuscitation and ongoing use is dictated by clinical circumstances. Crystalloid volume expanders generally are recommended as initial choices because they are effective and inexpensive. Most acutely ill children with