long-term complications in survivors are neuromuscular and psychosocial (level A evidence).

SHOCK

Shock is systemic organ hypoperfusion, usually caused by hypotension, that leads to cell injury and death. Four classifications are provided: cardiogenic shock (i.e., decreased cardiac output as a result of dysfunction), hypovolemic shock (i.e., decreased intravascular volume), septic or redistributive shock (i.e., decreased systemic vascular resistance), and obstructive shock (i.e., decreased cardiac output as a result of obstruction to flow). Anaphylactic shock caused by allergic reaction to a drug or a related insult is not discussed in this textbook.

For a deeper discussion on this topic, please see Chapter 106, "Approach to the Patient with Shock," 107, "Cardiogenic Shock," and 108, "Shock Syndromes Related to Sepsis," in Goldman-Cecil Medicine, *25th Edition.*

When encountering a patient in shock, the strategy is to gain vascular access quickly and to replace volume aggressively while making a careful assessment of the situation. This strategy is particularly appropriate when shock is thought to result from hypovolemia or sepsis. In the case of cardiogenic shock, strategies designed to improve cardiac function should be implemented, including administration of inotropic drugs or, in severe and unresponsive cases, cardiac bypass or cardiac-assist devices. In the case of severe hypovolemia, administration of saline is usually sufficient. Fluid replacement, antibiotic therapy, and drainage of infected spaces are paramount in treating sepsis.

Obstructive shock is the result of obstruction to blood flow, as observed in massive pulmonary embolism or saddle embolus lodged at the bifurcation of the right and left pulmonary arteries. In this setting, it is important to relieve the obstruction mechanically or through other methods (e.g., thrombolysis) or support the patient's circulation until the obstruction subsides.

The management of shock entails monitoring of blood pressure and organ perfusion. A central venous line facilitates the delivery of fluids and assessment of central volume status, and an arterial line allows accurate monitoring of blood pressure. Although the placement of a pulmonary artery catheter (i.e., Swan-Ganz catheter) showed favorable results in initial trails for a selected group of patients, recent studies have shown no beneficial effects (e.g., FACTT study, level I evidence). The catheter allows direct assessment of pressures in the right atrium, right ventricle, and pulmonary artery; measurement of the pulmonary capillary wedge pressure; and assessment of cardiac output.

Concerns have been raised about the true usefulness and benefit-risk ratio associated with catheter placement. Pulmonary artery catheters have been shown to increase complications, primarily nonlethal cardiac dysrhythmias, and to be poor predictors of fluid responsiveness in sepsis. The central venous oxygen saturation ($Scvo_2$) measured from a central venous catheter was shown to be similar to the mixed venous oxygen saturation ($S\bar{v}o_2$). Significant expertise is required for the insertion of these catheters and for adequate interpretation of the data generated.

For a deeper discussion on this topic, please see Chapters 106, "Approach to the Patient with Shock," and 107, "Cardiogenic Shock," in Goldman-Cecil Medicine, *25th Edition.*

SYSTEMIC INFLAMMATORY RESPONSE SYNDROME

The systemic inflammatory response syndrome (SIRS) is a constellation of clinical signs and symptoms triggered by the host response to diverse insults. The most common cause of SIRS is infection, which is called *sepsis*, but SIRS also can be triggered by noninfectious disorders such as pancreatitis and drug intoxication.

The diagnosis of SIRS requires at least two of the following criteria: temperature higher than 38° C or less than 36° C; tachycardia greater than 90 beats per minute; tachypnea greater than 20 breaths per minute; $Paco_2$ less than 32 mm Hg; and white blood cell count greater than 12,000 cells/μL or less than 4000 cells/μL. This systemic response may result in dysfunction of many organs, including the lung, liver, kidneys, heart, and central nervous system, which is called *multiple-organ dysfunction syndrome* or *multiple-organ system failure*. The prognosis worsens as more organs become involved, with mortality rates ranging from 30% for less severe cases to more than 90% for five or more failing organs. Treatment includes aggressive fluid replacement or vasopressors to improve blood pressure and prompt administration of antibiotics after blood and other cultures have been obtained.

For a deeper discussion on this topic, please see Chapter 108, "Shock Syndromes Related to Sepsis," in Goldman-Cecil Medicine, *25th Edition.*

NOXIOUS GASES, FUMES, AND SMOKE INHALATION

Inhalation of certain gases and fumes may cause asphyxia or cellular and metabolic injury (Table 22-2). Carbon monoxide poisoning is a common and frequently unsuspected cause of inhalational injury. It results in tissue hypoxia by competitively displacing oxygen from hemoglobin. Affinity of carbon monoxide for hemoglobin is about 250 times greater than that of oxygen.

The correlation between carbon monoxide levels and symptoms is weak, but patients with levels greater than 30% are usually

TABLE 22-2 TOXIC GASES AND FUMES

INJURIES	AGENTS	OCCUPATIONAL EXPOSURES
Simple asphyxia	Carbon dioxide	Mining, foundries
	Nitrogen	Mining, diving
	Methane	Mining
Cellular hypoxia and oxygen transport	Carbon monoxide	Mining, combustion in closed spaces
	Cyanide	Smoke inhalation
	Hydrogen sulfide	Petroleum refining
Direct tissue injury	Ammonia	Fertilizer, cleaning agents
	Chlorine	Bleaches, swimming pools
	Nitrogen dioxide	Farming, fertilizer, combustion in closed spaces
	Phosgene	Welding, paint removal
	Cadmium, mercury	Welding