causes renal ischemia with possible tubular or glomerular necrosis and renal vein thrombosis. Reduced blood flow to the lungs can interfere with surfactant secretion and result in acute respiratory distress syndrome, which is characterized by sudden pulmonary congestion and atelectasis with formation of a hyaline membrane. Gastrointestinal tract bleeding and perforation are always possibilities after splanchnic ischemia and necrosis of intestinal mucosa. Metabolic complications of shock may include hypoglycemia, hypocalcemia, and other electrolyte disturbances.

Diagnostic Evaluation

The etiology of shock can be discerned from the history and the physical examination. The severity of the shock is determined by measurements of vital signs, including CVP and capillary filling (Box 23-14). Shock can be regarded as a form of compensation for circulatory failure. Because of the progressive nature of shock, it can be divided into the following three stages or phases:

1. **Compensated shock:** Vital organ function is maintained by intrinsic compensatory mechanisms; blood flow is usually normal or increased but generally uneven or maldistributed in the microcirculation.

2. **Decompensated shock:** Efficiency of the cardiovascular system gradually diminishes until perfusion in the microcirculation becomes marginal despite compensatory adjustments. The outcomes of circulatory failure that progress beyond the limits of compensation are tissue hypoxia, metabolic acidosis, and eventual dysfunction of all organ systems.

3. **Irreversible, or terminal, shock:** Damage to vital organs, such as the heart or brain, is of such magnitude that the entire organism will be disrupted regardless of therapeutic intervention. Death occurs even if cardiovascular measurements return to normal levels with therapy.

Box 23-14

Clinical Manifestations of Shock