

**box 54-2****Response to Shock States  
in the Older Patient**

As an individual ages, normal physiological changes may limit the ability of the body to respond efficiently to shock states. The nurse should be aware of physiological changes of aging and monitor closely for changes in the older patient's baseline assessment(s). The patient's medical history indicates other chronic disease states that further compromise normal physiological changes seen with aging.

**Cardiovascular system:** Increased dysrhythmias, increased atrial size and irritability, left ventricular myocardial thickening leading to decreased compliance and lower ejection fraction; thickened heart valves that interfere with forward flow; decreased response to sympathetic nervous system; decreased sensitivity of baroreceptors; generalized stiffening of arterial vessels, including aorta

**Pulmonary system:** Decreased tidal volume and respiratory muscle strength, decreased alveolar surface area, increased dead space at end-expiration, decreased elastic recoil of lungs, increased resting respiratory rate, increased risk of infection as a result of decreased number of cilia, blunted response to hypoxemia, decreased gag and cough reflex leading to increased risk for infection, aspiration

**Hematologic system:** Decreased ability of bone marrow to produce cells (red blood cells, white blood cells, platelets), increased anemia, decreased immune function (decreased production of T and B lymphocytes) leading to increased infections, lower baseline temperature, gradual changes in temperature in the elderly versus spikes (101.3°F [38.5°C]), increased risk of adverse drug reactions

diagnosed by the presence of systemic and pulmonary hemodynamic alterations, which result from inadequate cardiac output and tissue perfusion. Typically, this occurs when greater than 40% of ventricular mass is damaged. The most common cause of cardiogenic shock is an extensive left ventricular myocardial infarction. The reported incidence of cardiogenic shock due to myocardial infarction ranges from 15% to 20% and carries a mortality rate of 75% of 85%.<sup>3</sup> It is believed that this rate has decreased since the introduction of rapid invasive monitoring and revascularization procedures.<sup>4</sup> Although cardiogenic shock may develop within a few hours after the onset of myocardial infarction symptoms, it often occurs after hospitalization. Other causes of cardiogenic shock include papillary muscle rupture, ventricular septal rupture, cardiomyopathy, acute myocarditis, valvular disease, and dysrhythmias.

Box 54-3 shows independent predictors for development of cardiogenic shock. Patients with all five risk factors have a greater than 50% chance for developing cardiogenic shock. Research indicates that mortality rates for patients who present with cardiogenic shock compared with those in whom it develops in the hospital are similarly high. Identifying patients at risk for development of car-

**box 54-3 Risk Factors for Inpatient  
Development of Cardiogenic Shock**

- Increased age (elderly)
- Left ventricular ejection fraction <35% on hospital admission
- Large myocardial infarction
- History of diabetes mellitus
- Previous myocardial infarction

diogenic shock and formulating strategies for prevention are extremely important.

**Pathophysiology**

Cardiogenic shock is caused by loss of ventricular contractile force, which results in decreased stroke volume and decreased cardiac output. Neuroendocrine compensatory mechanisms, which are discussed in detail in the section on hypovolemic shock, are activated to increase preload through retention of sodium and water. Vasoconstriction also increases afterload (SVR). Ventricular filling pressures increase because of the increased preload, but lack of contractility prevents complete ejection. The ventricle becomes distended, further impairing effective contraction, and cardiac output continues to decrease. Compensatory mechanisms continue the vicious cycle of elevated ventricular filling pressures and SVR in combination with an inability of the heart to eject an adequate volume of blood into circulation. Blood pools in the pulmonary circulation, resulting in pulmonary congestion. Pulmonary capillaries are under increased pressure and leak fluid into the interstitium and alveoli, preventing pulmonary diffusion of oxygen and reducing oxygen tension in the blood (PaO<sub>2</sub>; Fig. 54-4).

The body's cells become ischemic because of the decreased cardiac output, adding to an already tenuous state of myocardial functioning by further stimulating compensatory mechanisms to increase perfusion to the cells. Increased sympathetic stimulation increases the heart rate even more, further escalating myocardial oxygen demands and compounding the crisis. Associated hypotension prevents adequate oxygenation of myocardial tissue, exacerbating the anaerobic metabolism of the myocardial tissue and further decreasing the contractile state of the heart. These stressors placed on the failing heart may result in extension of a myocardial infarction.

**Assessment**

Patients admitted with the diagnosis of myocardial infarction require close monitoring. Assessment parameters are similar to the signs and symptoms of congestive heart failure but are more extreme. Assessment findings should be followed over time to allow the nurse to perceive the subtle changes that signal the beginning of cardiogenic shock.

**HISTORY**

A thorough history provides the information necessary to predict patients at risk for development of cardiogenic