factors impair the pumping ability or increase the workload of the heart so an adequate cardiac output cannot be maintained.

Compensatory Mechanisms

As the heart fails, the low cardiac output and inadequately filled arteries activate the neurohormonal system by several feedback mechanisms. One mechanism is increased sympathetic activity and circulating catecholamines (neurohormones), which increases the force of myocardial contraction, increases heart rate, and causes vasoconstriction. The effects of the baroreceptors in the aortic arch and carotid sinus that normally inhibit undue sympathetic stimulation are blunted in clients with HF, and the effects of the high levels of circulating catecholamines are intensified. Endothelin, a neurohormone secreted primarily by endothelial cells, is the most potent endogenous vasoconstrictor and may exert direct toxic effects on the heart and result in myocardial cell proliferation.

Another mechanism is activation of the renin–angiotensin–aldosterone system. Renin is an enzyme produced in the kidney in response to impaired blood flow and tissue perfusion. When released into the bloodstream, renin stimulates the production of angiotensin II, a powerful vasoconstrictor. Arterial vasoconstriction impairs cardiac function by increasing the resistance (afterload) against which the ventricle ejects blood. This raises filling pressures inside the heart, increases stretch and stress on the myocardial wall, and predisposes to subendocardial ischemia. In addition, clients with severe HF have constricted arterioles in cerebral, myocardial, renal, hepatic, and mesenteric vascular beds. This results in increased organ hypoperfusion and dysfunction. Venous vasoconstriction limits venous capacitance, resulting in venous congestion and increased diastolic ventricular filling pressures (preload). Angiotensin II also promotes sodium and water retention by stimulating aldosterone release from the adrenal cortex and the release of vasopressin (antidiuretic hormone) from the posterior pituitary gland.

All of these mechanisms combine to increase blood volume and pressure in the heart chambers, stretch muscle fibers, and produce dilation, hypertrophy, and changes in the shape of the heart (a process called cardiac or ventricular remodeling) that make it contract less efficiently. Overall, the compensatory mechanisms increase preload (amount of venous blood returning to the heart), workload of the heart, afterload (amount of resistance in the aorta and peripheral blood vessels that the heart must overcome to pump effectively), and blood pressure. These compensatory mechanisms that initially preserve cardiac function result in progressive deterioration of myocardial function over time.

Signs and Symptoms

Clients with compensated HF usually have no symptoms at rest and no edema; dyspnea and fatigue occur only with activities involving moderate or higher levels of exertion. Symptoms that occur with minimal exertion or at rest and are accompanied by ankle edema and distention of the jugular vein (from congestion of veins and leakage of fluid into tissues) reflect decompensation. Acute, severe cardiac decompensation is manifested by pulmonary edema, a medical emergency that requires immediate treatment. Clients with chronic HF are often described according to the New York Heart Association classification categories that separate clients into four groups according to symptoms and activity tolerance (Box 51–1). These categories are often used to help evaluate results of therapy, and to indicate a client's functional status.

DRUG THERAPY

Several drugs are used to treat acute HF, and a combination of an angiotensin-converting enzyme (ACE) inhibitor or angiotensin receptor blocker (ARB) and a diuretic is first-line therapy for chronic failure. Increasingly, digoxin, a beta-adrenergic blocking agent, or spironolactone is being added to the ACE inhibitor or ARB and diuretic regimen.

Drug therapy of HF continues to evolve as the pathophysiologic mechanisms are better understood and research studies indicate more effective regimens. Combinations of drugs are commonly used in efforts to improve circulation, alter the compensatory mechanisms, and reverse heart damage. Most of the drugs used to treat HF are also used in other disorders and are discussed in other chapters; their effects in HF are described in Box 51–2. The primary focus of this chapter is inotropic agents, which include digoxin, a cardiac glycoside, and the phosphodiesterase inhibitors inamrinone and milrinone. These drugs are discussed in the following sections and in Drugs at a Glance: Drugs for Heart Failure.

BOX 51–1 NEW YORK HEART ASSOCIATION CLASSIFICATION OF PATIENTS WITH HEART DISEASE

Class I. No limitations of physical activity; ordinary physical activity does not cause dyspnea, fatigue, or palpitations.

Class II. Slight limitations of physical activity. Patients are comfortable at rest but have dyspnea, fatigue, palpitations, or chest pain (angina) with ordinary physical activity.

Class III. Marked limitations of physical activity. Patients are comfortable at rest but develop symptoms with less than ordinary physical activity.

Class IV. Patients are unable to perform any physical activity without discomfort. Symptoms of heart failure or angina are present even at rest. If any physical activity is undertaken, discomfort increases.