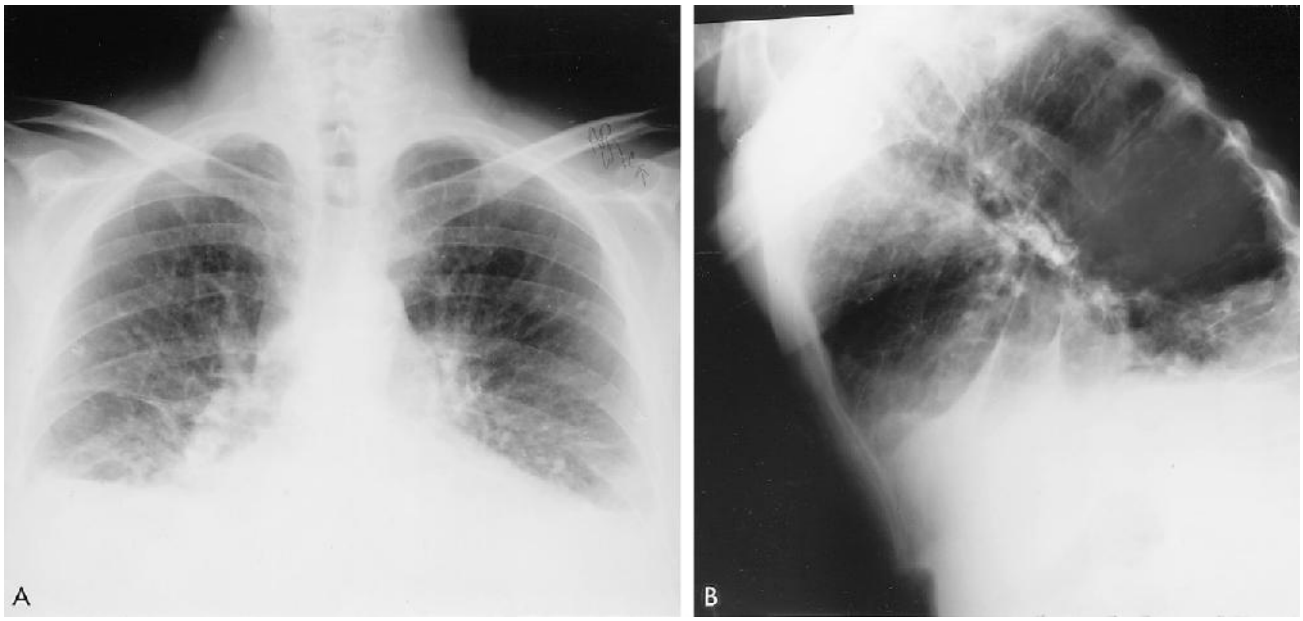

FIGURE 5-5 **A,** Posteroanterior chest radiograph showing cardiomegaly. **B,** Lateral chest radiograph showing pulmonary vascular congestion that is typical of pulmonary edema.

ventricular hypertrophy, and ventricular function, have been used in clinical trials to assess the efficacy of therapies.

Laboratory Evaluation

Initial laboratory evaluation includes a complete blood count (CBC) to assess for anemia and a basic chemistry panel for electrolyte abnormalities. The serum sodium level may be impaired, and there may be evidence of renal dysfunction due to decreased cardiac output and renal artery vasoconstriction or elevated venous pressures reflected in the renal veins (i.e., cardiorenal syndrome). Patients should be evaluated for hyperthyroidism or hypothyroidism and for hemochromatosis (i.e., with a serum ferritin level) because it is a reversible cause of HF. Patients should be tested for human immunodeficiency virus (HIV) infection. Laboratory tests for other modifiable risk factors include a fasting lipid panel and a blood glucose level. Liver function enzymes may be elevated in patients with HF and hepatic congestion, which can result from volume overload and significant LV dysfunction and may be seen in cases of right ventricular HF or severe tricuspid regurgitation.

Tests for plasma natriuretic peptide levels (BNP or NT-proBNP) were initially developed to evaluate patients with acute dyspnea when the diagnosis of HF was in doubt. When results are normal, this test has strong discriminatory power to eliminate HF as the cause of dyspnea. The Valsartan Heart Failure Trial (Val-HeFT) established that serial measurements of natriuretic peptide levels correlate with prognosis.

Acute Treatment

After the clinical diagnosis of HF is established, a model proposed by Stevenson and colleagues (Fig. 5-7) focuses on assessing volume status and perfusion and then further characterizes the patient according to volume overload/congestion-related and perfusion/output-related presentations. Using the history and physical examination findings, a physician can make astute clinical decisions based on one of four profiles for patients with HF.

In patients with acute onset of pulmonary edema, initial management should be directed at improving oxygenation and providing hemodynamic stability. Patients commonly have marked elevation of blood pressure, myocardial ischemia, and worsening mitral regurgitation. Standard therapy includes supplemental oxygen and an intravenous loop diuretic.

Nitroglycerin helps to reduce preload through venodilation and may provide symptomatic relief for patients with ischemic and nonischemic ventricular dysfunction. For patients with hypertensive urgency, severe hypertension, or decompensated HF related to aortic or mitral regurgitation, an arterial vasodilator such as nitroprusside may be helpful in reducing afterload. Evaluation of the patient's response to treatment requires serial assessment of blood pressure, heart rate, end-organ perfusion, and oxygen saturation. For severely decompensated patients with refractory hypoxia or respiratory acidosis, mechanical ventilation or continuous positive airway pressure (CPAP) therapy may be necessary.

Pulmonary artery catheterization may be helpful in documenting filling pressures and the cardiac index and in hemodynamically guiding the response to therapy. Although invasive monitoring has not been associated with improved outcomes, it is impossible to adjust these studies for disease severity. In patients with refractory pulmonary edema or a markedly impaired cardiac index, inotropic agents or short-term mechanical circulatory support (e.g., intra-aortic balloon pump) may become necessary.

Treatment of Heart Failure

Treatment of HF is directed at relieving the patient's symptoms, mitigating the underlying or precipitating causes (Table 5-3), and slowing disease progression. Patients should be educated about the importance of adherence to medical therapy and restriction of dietary sodium and fluid. Rhythm disturbances such as atrial