The body attempts to counter these effects by secreting atrial natriuretic peptide and brain natriuretic peptide (BNP) from the myocardium. Endogenous natriuretic peptides promote salt and water excretion by the kidneys and cause arterial vasodilation, but they are relatively ineffective at reversing the changes associated with stimulating the sympathetic nervous system and the RAAS.

Clinical Presentation and Diagnosis

The approach to the patient with suspected HF starts with the history, physical examination, and testing to help establish the diagnosis. The history should assess for NYHA functional class, including symptoms of fatigue, weakness, dyspnea, orthopnea, edema, abdominal distention, and chest discomfort. The examiner should also assess for comorbidities, including hypertension, diabetes mellitus, dyslipidemia, obesity, and sleep-disordered breathing.

The medical history should inquire about exposure to cardiotoxic agents, including anthracycline-based chemotherapy. The social history evaluates past and current use of tobacco products, alcohol, and illicit drugs. The family history assesses for sudden cardiac death, coronary artery disease, and cardiomyopathy. For patients with an idiopathic dilated cardiomyopathy, a three-generation history should be obtained to establish a familial component.

The physical examination starts by assessing vital signs. Worrisome vital signs for significant cardiac dysfunction include faint pulses, a narrow pulse pressure due to peripheral vasoconstriction and low stroke volume, and resting tachycardia. Assessment of the peripheral pulse includes evaluating the patient for pulsus alternans, which is defined as beat-to-beat variation in the amplitude of the peripheral pulse, and it is pathognomonic for severe LV dysfunction.

Most symptoms of HF are related to elevated filling pressures. Dyspnea (in men) and fatigue (in women) are some of the most common symptoms of HF. They may have an acute onset resulting in pulmonary edema, or they may be chronic and progressive and occur at rest. Dyspnea on exertion has a sensitivity of 84% to 100% but a specificity of 17% to 34%. Dyspnea from HF is often exacerbated in the supine position (i.e., orthopnea), and it is caused by increased distribution of blood to the pulmonary circulation when lying flat. Patients with HF tend to use increased numbers of pillows to overcome orthopnea. Orthopnea has a sensitivity of 22% to 50% and a specificity of 74% to 77% for HF Episodes of paroxysmal nocturnal dyspnea (PND) awaken patients from sleep and are likely caused by central redistribution of edema, leading to a sudden rise in intracardiac pressures. The sensitivity of PND for the diagnosis of HF is 39% to 41%, and the specificity ranges from 80% to 84%. Patients with stage D HF may exhibit Cheyne-Stokes respirations, which is associated with a poor prognosis.

Evaluation of volume status includes assessment of serial weights, jugular venous pressure, pulmonary congestion, and peripheral edema. The jugular venous pressure is best assessed using the right internal jugular vein with the patient lying at a 30- to 45-degree angle. Patients with markedly elevated venous pressures may need to be positioned at a higher angle. The jugular venous pressure is an estimate of the central venous pressure (CVP) (i.e., right atrial pressure) and therefore of volume status. A normal CVP is in the range of 5 to 9 cm H_2O. An abnormally elevated CVP may be seen in hypervolemia, pericardial constriction, or pulmonary hypertension.

Evaluating the abdominal jugular reflux (i.e., hepatojugular reflux) involves gently compressing the abdomen or right upper quadrant for 15 to 30 seconds and assessing jugular venous distention. This method assesses volume status and right ventricular dysfunction and compliance. An abnormal abdominal jugular reflux is defined as a sustained increase in jugular venous pressure of more than 4 cm H_2O.

On lung auscultation, crackles may be heard. Crackles are a specific finding for HF, but they are not detected in approximately 60% of patients with chronic HF. Before auscultation, the precordium should be examined and the point of maximal impulse (PMI) evaluated. An abnormal PMI is defined as displacement below the fifth intercostal space and lateral to the midclavicular line. It offers the clinician an assessment of heart size and function if it is sustained for more than one third of systole or is palpable over two intercostal spaces.

On auscultation of the heart, abnormal findings include an early diastolic third heart sound (S_3). A third heart sound is compatible with elevated atrial pressures and increased ventricular chamber stiffness. The sound results from rapid deceleration of the passive component of blood flow from the atrium into the noncompliant ventricle. An S_3 sound can be generated from the left or right ventricle; the latter changes in intensity with respiration. A fourth heart sound (S_4) results from an exaggerated atrial contribution to LV filling, but it is not specific for HF. Patients may also have an accentuated P_2 pulmonic valve component of S_2 if pulmonary hypertension also exists. Poor prognostic signs on physical examination include elevated jugular venous pressures and an S_3 sound.

Peripheral edema usually involves the lower extremities, but edema can involve the thighs and abdomen. Abdominal ascites may develop, particularly in the setting of worsening right ventricular failure and severe tricuspid regurgitation. Lower extremity edema can occur in many other disease states, including nephrotic syndrome, cirrhosis, venous stasis, and lymphedema, and it is not specific for HF.

The murmurs of mitral and tricuspid regurgitation are common in patients with HF. They may become worse during an acute decompensation.

Diagnostic Testing

The electrocardiogram in patients with congestive HF usually is nonspecific, but it may reveal changes suggesting a prior MI, conduction system disease, and chamber enlargement. The chest radiograph may show cardiomegaly and signs of pulmonary congestion (Fig. 5-5). Treatment of HF improves the vascular congestion seen on the chest radiograph, but radiographic changes may lag 24 to 48 hours behind clinical improvement.

Transthoracic echocardiography (TTE) is recommended for all patients with suspected HF. A noninvasive echocardiogram can assess ventricular chamber sizes, ventricular wall thickness, systolic function, diastolic function, and valvular stenosis or regurgitation. It can provide an estimation of left and right atrial pressures and quantification of stroke volume and cardiac output (Fig. 5-6). These measurements, including chamber size,