CASE STUDY

3 CONGESTIVE HEART FAILURE

For the Patient Case for this case study, see the printed book.

DISEASE SUMMARY

Definition

Congestive heart failure (CHF) is not a specific disease but a common, progressive pathophysiologic syndrome that describes several types of cardiac dysfunction that cause poor perfusion of tissues with vital blood-borne nutrients and oxygen. Sluggish blood flow (*congestion*) and tissue swelling (*edema*) are also major components of CHF. CHF is also known in the medical literature as *cardiac failure*, *ventricular failure*, or simply *heart failure*.

Prevalence

More than 3 million people in the United States now have CHF and the prevalence is increasing. Nearly 400,000 new cases are diagnosed annually, and approximately 1 million hospital admissions for advanced CHF occur every year. The frequency of CHF increases with age and more than 75% of cases are observed in patients age 65 and older. *CHF is currently the most common cause for hospital admission among the elderly*.

The incidence of CHF is highest among African-Americans, Hispanics, Native Americans, and recent immigrants from non-industrialized nations, Russia, and the former Soviet republics. African-Americans are 1.5 times more likely to die from CHF than are Caucasians.

Prevalence is greater in males than females for persons aged 40–75 years. However, no gender predilection exists for individuals older than 75 years.

Significance

Depending on the stage to which heart failure has progressed, mortality is significant and may exceed 50% within 5 years of diagnosis. Shortness of breath (*dyspnea*), fatigue, exercise intolerance, abdominal pain, nausea with loss of appetite, and significant swelling of the feet and ankles are especially bothersome. The accumulation of fluid within the lungs (*pulmonary edema*) is a serious and life-threatening complication of this condition.

CHF is the fastest growing clinical cardiac disease entity in the United States. An estimated $23 billion is spent on inpatient management and another $40 billion on outpatient management in the United States every year.

Causes and Risk Factors

CHF is a potential complication of many disease states. In the United States, heart attacks (myocardial infarctions) that result from coronary artery atherosclerosis are the most common cause. Cell death in the heart from reduced blood flow results in poor contractility of heart muscle. High blood pressure (*systemic hypertension*) is also an important national cause of CHF. Cardiomyopathies (a heterogeneous group of progressive, degenerative cardiac disorders), viral infections of the heart (myocarditis), and valvular heart disease are other significant causes of CHF today.

Pathophysiology

Cardiac function is determined by four major parameters:

1. contractile state of heart muscle
2. preload (i.e., end-diastolic volume and the resultant length of cardiac myofibers prior to onset of contraction)
3. afterload (resistance to ventricular ejection of blood)
4. heart rate

A significant alteration in any of these determinants can result in CHF. In most cases, the primary cause is a decrease in myocardial contractility, resulting from either a focal loss of functional muscle (e.g., myocardial infarction) or processes that adversely and diffusely affect the myocardium (e.g., cardiomyopathies). However, the heart may also fail when afterload is excessive (due to systemic hypertension or aortic stenosis) or preload is elevated. Intravascular fluid volume overload increases preload, stretches individual myofibers, and dilates the chambers of the heart. Cardiac function may also fail when heart rate is too fast or too slow. While the normal heart can tolerate major variations in preload, afterload, and heart rate, the diseased heart compensates poorly with such changes. *Systolic dysfunction* causes CHF when the heart is not contracting properly (e.g., with loss of functional muscle as a result of a heart attack). *Diastolic dysfunction* results in CHF when the heart is not relaxing adequately (e.g., as a result of "stiffening" of cardiac muscle with aging or cardiac hypertrophy from elevated ventricular afterload).

Based on cause, there are two major pathophysiologic types of heart failure: high- and low-output cardiac failure. Although uncommon, *high-output heart failure* is characterized by cardiac hyperfunction that is inadequate to meet the metabolic or oxygen demands of the body. Causes of high-output failure in which elevated heart rate is common include hyperthyroid disease and severe anemia. With hyperthyroidism, excessive secretions of the thyroid hormones T_3 and T_4 result in a hypermetabolic state. With anemia, the oxygen demands of tissues are not met. The strain of increased cardiac performance in the presence of increased metabolic demands or decreased oxygen delivery depletes cardiac reserves and low-output failure eventually ensues.

The much more common *low-output heart failure* results when afterload or preload is excessively elevated or when cardiac contractility is diminished. Low ventricular output ultimately results in stagnant blood flow (congestion), an increase in the intravascular hydrostatic pressure from increased blood volume, and tissue edema. Specific sites of congestion and edema (and, therefore, also the patient's specific clinical manifestations) are dependent on the cardiac location of decreased function (i.e., left ventricle, right ventricle, or both ventricles).

Diagnosis: Clinical Manifestations and Laboratory Tests

CHF may result from decreased right ventricular function (*right-sided failure*) or left ventricular function (*left-sided failure*). Since the cardiovascular system is a closed system, both ventricles typically fail with time and *bilateral heart failure* ensues. Patients with *left heart failure* have manifestations of low cardiac output and elevated pulmonary venous pressure. These manifestations include:

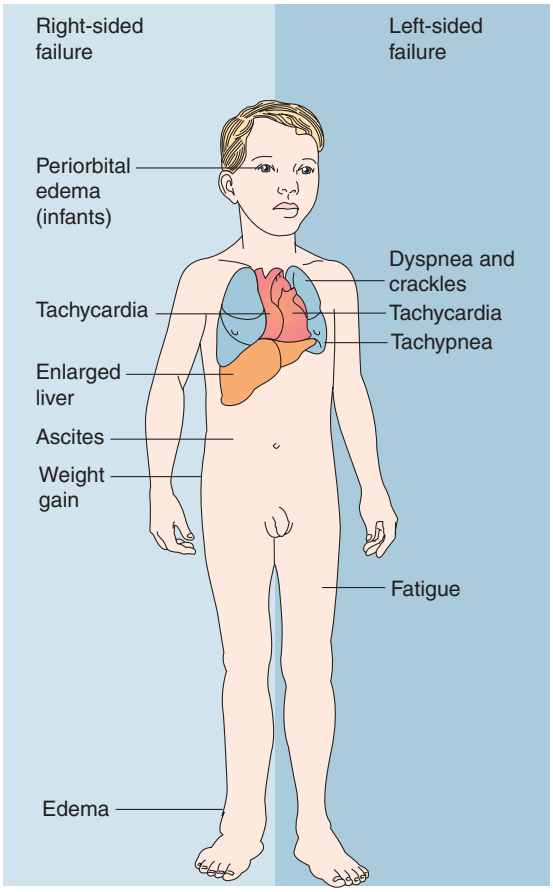
1. decreased renal blood flow and urine output with azotemia (i.e., elevated concentrations of blood urea nitrogen [BUN] and serum creatinine)

2. confusion, lethargy, and more serious alterations of consciousness from decreased blood flow and oxygen delivery to the brain and increased serum ammonia levels due to reduced liver function
3. fatigue and weakness from decreased oxygen delivery to skeletal muscle
4. dyspnea as fluid accumulates in the terminal airways (alveoli) of the lungs (pulmonary edema)

Dyspnea develops more rapidly when the patient is lying down (*orthopnea)* because decreased venous pressure in the legs increases total blood volume, which elevates preload. Patients often wake up at night feeling that they are suffocating, a condition known as *paroxysmal nocturnal dyspnea*. This increase in total blood volume in the supine position also explains why nocturia (increased frequency of urination during nighttime hours) is associated with CHF.

Signs of fluid accumulation predominate in *right heart failure* with the ambulatory patient developing pitting edema of the feet and ankles and the bedridden patient developing edema in the buttocks. Right ventricular failure is also characterized by an enlarged liver (*hepatomegaly*) and spleen (*splenomegaly*) from congestion. Congestion may also lead to abdominal pain, distension of the jugular veins (JVD) from increased jugular venous pressure, and, occasionally, ascites. Hepatic congestion causes right upper quadrant pain, while splenic congestion causes left lower quadrant pain. Most patients with CHF demonstrate signs of both left- and right-sided CHF. Disease Summary Figure 3.1 distinguishes between the clinical manifestations of right and left heart failure.

Major signs of compensated heart failure are associated with hyperactivity of the sympathetic nervous system (SNS). These signs include tachycardia and increased contractility (i.e., a "racing and pounding heart" known as *palpitations*), elevated blood pressure,



DISEASE SUMMARY FIGURE 3.1
Clinical manifestations of right- and left-sided heart failure. (Reprinted with permission from Pillitteri A. Maternal & Child Health Nursing. 4th Ed. Philadelphia: Lippincott Williams & Wilkins, 2003.)

diaphoresis (i.e., excessive sweating), and coolness and pallor of the skin. Cardiac hypertrophy with or without structural valvular changes may cause murmurs that can be heard with auscultation. Other signs of compensation result from low cardiac output and a reduction in both renal blood flow and glomerular filtration rate. Serum renin, angiotensin II and aldosterone concentrations are elevated, resulting in fluid retention, cardiac dilation (*cardiomegaly*), and elevated blood pressure. Compensation in the early stages of CHF helps to maintain blood pressure, contractility, and cardiac output. However, SNS and renin-angiotensin-aldosterone (RAA) hyperactivity ultimately cause decompensated heart failure (i.e., total loss of cardiac reserve) through persistent elevation of preload and afterload. At this point in the clinical course of CHF, the prognosis becomes poor.

Other signs of heart failure include:

- "crackles" (also known as *rales*) with auscultation of the lungs, especially at the base of each lung (suggests pulmonary edema)
- abnormal heart sounds, such as a diminished S_1 (suggesting impaired contractility) or an additional heart sound (S_3 or S_4)
- positive hepatojugular reflux (HJR), which occurs when jugular veins dilate in response to sustained, moderate pressure applied to the liver by the primary care provider
- elevated serum concentration of brain natriuretic peptide (BNP), which is released from ventricular cells when ventricular diastolic pressures are elevated
- dilation of the heart (cardiomegaly) and significant shadows in the lungs (alveolar fluid) with chest x-ray
- enlargement of the heart and decreased ventricular wall motion with echocardiography
- decreased ventricular ejection fraction (EF) with radionuclide imaging (When both stroke volume [SV] and end diastolic volume [EDV] are known, EF can be calculated by the equation $EF = SV/EDV \times 100\%$. An EF that is $<40\%$ is consistent with CHF.)

Appropriate Therapy

Depending on the patient's specific circumstances, therapy for CHF can be non-pharmacologic or pharmacologic. Non-pharmacologic approaches include salt restriction, home monitoring of weight as an index of fluid retention and progressing CHF, aerobic exercise for stable patients to improve activity tolerance, and coronary revascularization (i.e., bypass surgery) if CHF developed from coronary artery disease. Since many patients with CHF have asynchronous and inefficient cardiac contractions, resynchronization of heart function with a pacemaker is a promising non-pharmacologic approach that improves both EF and exercise tolerance.

Implantation of a defibrillator is the approach of choice in patients who have an associated arrhythmia, a reasonable life expectancy, and CHF that is stabilized. Implantable defibrillators have reduced mortality from CHF-associated arrhythmia by one third.

Because of the poor prognosis in patients with advanced or decompensated heart failure, cardiac transplantation is a last-resort therapeutic option. Post-transplant 5-year survival rates now exceed 70%. Infections and cancers from cyclosporine immunosuppressive therapy are major complications, and the high cost and few number of donor hearts are limiting factors for this type of therapy.

Effective pharmacologic treatments for CHF include the use of *diuretics, inhibitors of the RAA system, beta blockers, digitalis glycosides, vasodilators,* and *anticoagulants. A combination of a diuretic and an angiotensin-converting enzyme (ACE) inhibitor is the initial treatment in most symptomatic patients.*

Diuretics are the most effective means of providing symptomatic relief to patients with CHF. When fluid retention is mild, thiazide diuretics (e.g., hydrochlorothiazide) may be sufficient. Thiazide diuretics inhibit sodium resorption in the nephron, causing water and sodium loss in the urine (*natriuresis*). Patients with more severe heart failure should be treated with a loop diuretic (e.g., furosemide), which promotes sodium loss in the urine by inhibiting chloride resorption. Although rarely adequate when used alone, potassium-sparing diuretics are often useful in combination with a loop or thiazide diuretic to treat CHF in that they minimize life-threatening hypokalemia induced by more potent agents. Spironolactone promotes potassium retention and sodium excretion by inhibiting aldosterone action in the kidney (and may also prevent serious abnormal structural changes in the heart referred to as *myocardial remodeling*). Triamterene and amiloride reduce potassium excretion.

Since the RAA system is activated early in the course of CHF and promotes harmful increases in blood pressure, *inhibitors of the RAA system* should also be considered part of initial therapy. ACE inhibitors (e.g., captopril and enalapril) inhibit conversion of angiotensin I to angiotensin II, thus preventing angiotensin II-induced vasoconstriction and the secretion of aldosterone. Aldosterone causes sodium and water retention by the kidneys. ACE inhibitors also prevent the metabolism of the vasodilator bradykinin, which, in turn, stimulates synthesis of vasodilating prostaglandins and nitric oxide—all of which help to maintain a lower blood pressure. These drugs significantly reduce mortality rate, prevent hospitalization, and improve exercise tolerance in patients with CHF. The most common side effects of ACE inhibitors are dizziness and cough, although cough with newer generations of these drugs is uncommon. Angiotensin receptor blockers (e.g., valsartan) can be used in ACE inhibitor-intolerant patients.

The use of *beta blockers* (such as carvedilol and metoprolol) together with diuretics and ACE inhibitors significantly reduces mortality and hospital admissions in CHF patients. Although the mechanism of action is unknown, these drugs probably benefit patients by preventing progressive myocardial injury from chronic elevations in catecholamines and SNS hyperactivity. With 6 months of use, beta blockers are known to significantly increase EF and reduce the probability of cardiac hypertrophy and dilation and should be used in all stable patients who do not have contraindicating circumstances. Furthermore, patients with moderate to severe CHF have a high frequency of arrhythmia. Because many anti-arrhythmic drugs adversely affect cardiac function, beta blockers are first-line therapy for CHF patients with arrhythmia.

The *digitalis glycosides* (primarily digoxin) bind to the Na^+-K^+ ATPase on the membrane of the cardiocyte, inhibit the Na^+ pump, and increase intracellular Na^+. This facilitates Na^+-K^+ exchange with a resultant increase in cytosolic calcium, an increase in contractile protein cross-bridge formation, and a positive inotropic effect (i.e., increased force of contraction). As research studies have shown that a digoxin-associated reduction in death rate from progressive CHF is offset by an increase in mortality from arrhythmic events, digoxin is recommended only for patients taking diuretics and ACE inhibitors who remain symptomatic.

Vasodilators, which dilate arteriolar smooth muscle and decrease peripheral vascular resistance, reduce left ventricular afterload in patients with CHF. Intravenous nitrates (such as sodium nitroprusside) are used for severely decompensated CHF, especially when accompanied by hypertension. Although oral isosorbide and nitroglycerin ointment are effective for long-term therapy, development of tolerance is not uncommon. Nesiritide, a recombinant form of human brain natriuretic peptide, is a potent vasodilator that reduces preload and improves cardiac output. Because sustained hypotension is the primary side effect of the drug, nesiritide is best reserved for patients who remain symptomatic following treatment with diuretics and intravenous nitrates. Hydralazine is a potent arteriolar dilator but, as a single agent, has not been shown to improve symptoms of CHF. However, the combination of hydralazine and isosorbide nitrate has improved survival rates in heart failure patients.

Patients with CHF who have developed atrial fibrillation or who have suffered large, recent (3–6 months) myocardial infarctions will benefit from anticoagulant therapy (e.g., heparin, warfarin, or dicumarol).

Serious Complications and Prognosis

More than one third of patients hospitalized for CHF are re-admitted within 6 months, and the 5-year survival rate is approximately 50%. Major complications include pulmonary edema often followed closely by bacterial pneumonia, ventricular arrhythmia, cardiac enlargement with developing intracardiac thrombi that embolize, and decompensated and refractory CHF. Although the overall prognosis remains poor, probability for survival has improved during the past two decades, in part as a result of more widespread use of ACE inhibitors and beta blockers.

Suggested Readings

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