Congestive heart failure is a syndrome complex resulting from the inability of the heart to function as an effective output pump. This causes a lack of perfusion in the systemic circulation, making it unable to meet the metabolic demands of vital organs and peripheral tissue beds. The variable clinical presentation of this disorder depends on several factors, including the degree of end-stage pump failure, as well as predominant involvement of either or both sides of the heart. Although the majority of patients may exhibit features of biventricular (left and right) failure, isolated left heart failure results in inadequate left ventricular emptying during systole, causing primarily pulmonary symptoms (pulmonary hypertension and pulmonary edema). Isolated right heart failure results in poor right ventricular emptying, causing systemic symptoms. The management of this disease syndrome is aimed at the identification and correction of the underlying etiologic disorder, with concurrent amelioration of the resultant presenting symptoms. It is also important to identify the precipitating cause of the current heart failure episode. Despite advances in current therapy for this disorder, congestive heart failure continues to be associated with a high incidence of patient morbidity and mortality.

ETIOLOGY

The most common causes of congestive heart failure include underlying cardiac disease states, predominantly coronary artery disease and ischemic or hypertensive heart disease. By definition, myocardial infarction results in the death (necrosis) of myocardial tissue, which is necessary for the heart to function effectively. A variety of other predisposing cardiac conditions may lead to congestive heart failure, including congenital heart disease and cardiomyopathies (dilated, restrictive, and hypertrophic). Dilated cardiomyopathies are most likely idiopathic, but may result from toxic effects of alcohol or medications (eg, doxorubicin), infectious processes (eg, viral and parasitic), or collagen-vascular diseases. Restrictive cardiomyopathies may result from sarcoidosis, hemochromatosis, glycogen-storage diseases, or amyloidosis, among others. Hypertrophic cardiomyopathies result from a variety of causes, including an autosomal dominant genetic component, and are characterized by massive ventricular hypertrophy. Additional reasons for the progression to congestive heart failure include valvular heart disease (eg, aortic stenosis), pericarditis, pulmonary hypertension (eg, hypertension caused by pulmonary embolism and chronic obstructive pulmonary disease) and high-output states, such as hyperthyroidism, pheochromocytoma, atrioventricular shunts, and chronic anemias.

The most common precipitating factors of congestive heart failure include poor patient compliance with medical therapy, poor dietary control (eg, increased sodium intake), acute increase in cardiac metabolic demands (eg, trauma, sepsis, malignancy, pregnancy, thyrotoxicosis), acute dysrhythmias, silent myocardial infarction (eg, patients who are hospitalized or who have diabetes), systemic infections (eg, sepsis or infective endocarditis), pulmonary embolism, and the progression or acute exacerbation of underlying disease states, mentioned above.

DIAGNOSIS

The diagnosis of congestive heart failure depends on the patient’s history, physical examination findings,
current medications, laboratory evaluation, and radiographic examination. The constellation of signs and symptoms varies considerably in patients with congestive heart failure, depending on patient age, underlying etiologic disorder, and the onset and time course of the acute exacerbation episode. The recognition of congestive heart failure requires the presence of clinical features of compromised cardiac output, which includes fatigue, weakness, exercise intolerance, and decreased peripheral perfusion.

Additional signs and symptoms of congestive heart failure include features of pulmonary or systemic vascular congestion, such as dyspnea at rest or on exertion, S_3 (third heart sound, ventricular gallop), jugular venous distension, orthopnea, paroxysmal nocturnal dyspnea, peripheral pitting edema (caused by venous congestion), tachypnea, tachycardia, pulmonary basal rales, dullness to percussion with pleural effusions, cardiomegaly, pulsus alternans, ascites, hepatomegaly, splenomegaly, fluid retention, weight gain, cyanosis, and cool extremities.

Laboratory evaluation may show polycythemia, thrombocytopenia, leukopenia, elevated prothrombin and partial thromboplastin times (and international normalized ratio), abnormal liver function tests (elevated transaminases, increased total bilirubin), elevated blood urea nitrogen, and serum creatinine (caused by decreased renal perfusion), proteinuria, and hyponatremia (resulting from activation of the renin-angiotensin-aldosterone system caused by renal hypoperfusion). Arterial blood gas determination generally shows a respiratory alkalosis caused by tachypnea. Electrocardiographic changes include a variety of possible dysrhythmias, including conduction blocks and evidence of increased heart size.

A chest radiograph may indicate signs of increased cardiac silhouette ( cardiomegaly), pulmonary vascular redistribution with cephalization of vessels into the peripheral lung fields, blunting of the costophrenic angles (pleural effusion), and possibly Kerley B lines (lymphatic obstruction) with pulmonary edema. An echocardiogram may show evidence of wall motion abnormalities, valvular dysfunction, congenital heart defects, and pericardial disease. Swan-Ganz pulmonary artery catheterization usually shows elevated pulmonary capillary wedge pressures.

**TREATMENT**

The management of congestive heart failure is aimed at the identification and correction of both the underlying disorder and the precipitating factors. Specific goals include a reduction of cardiac workload and myocardial oxygen consumption, improvement in myocardial contractility, control of sodium and fluid retention, and increasing peripheral tissue perfusion and oxygenation. Treatment modalities include a reduction in physical activities, deep venous thrombosis prophylaxis with antiembolic support stockings or subcutaneous heparin administration, stress reduction, control of hypertension, dietary sodium restriction, fluid restriction, weight loss, smoking cessation, and supplemental portable oxygen. In some severe cases of congestive heart failure, mechanical fluid removal may be necessary. This includes dialysis, phlebotomy with rotating tourniquets, thoracentesis, or paracentesis. End-stage cardiac failure might be managed with mechanical circulatory assistance or cardiac transplantation. In general, medications useful in the treatment of congestive heart failure include diuretics, cardiac glycosides, vasodilators, angiotensin-converting enzyme inhibitors, phosphodiesterase inhibitors, and sympathomimetic amines.

Diuretic therapy is the standard in management of congestive heart failure, acting to reduce excess fluid accumulation and to improve hemodynamic parameters. Diuretics act to reduce preload, this is, the volume of fluid that is presented to an already failing heart, which then allows the heart to handle the decreased workload. Frequent complications of diuretic therapy include hypokalemia, hyponatremia, elevated serum urea nitrogen and creatinine, and volume depletion. Acute volume depletion can result in severe hypotension and further compromised cardiac output; therefore, the patient’s intake, output, and weight should be closely monitored. Hypokalemia may be particularly problematic in patients taking digitalis preparations or in those predisposed to cardiac dysrhythmias, and consideration should be given toward the use of potassium-sparing agents. Patients who are taking angiotensin-converting enzyme inhibitors with a potassium-sparing agent must be monitored closely for hyperkalemia. There are several types of diuretics, including thiazides (hydrochlorothiazide), loop diuretics (furosemide, bumetanide, ethacrynic acid), and potassium-sparing agents (spironolactone, triamterene, amiloride).

Digitalis glycosides (digoxin) act as positive inotropic agents to increase myocardial contractility and cardiac output through reversible inhibition of sodium-potassium adenosine triphosphatase activity, resulting in sodium and calcium inflow in the sarcoplasmic reticulum. Digoxin also acts as a negative chronotrope to decrease heart rate through inhibition at the SA node, which allows increased cardiac filling time during diastole. The dosage of digoxin varies with the severity of the illness, but usually involves a loading dose, followed by a maintenance dose, which is monitored with serum digoxin levels. Digoxin has many clinically significant drug interactions, including erythromycin and tetracycline, which increase digoxin levels caused...
by decreased metabolism by gut flora. Digitalis toxicity is an important clinical problem that may occur in 5% to 15% of patients on digoxin therapy. The signs and symptoms of digoxin toxicity are dose-related and include a variety of dysrhythmias and a variety of gastrointestinal and psychiatric symptoms that may worsen hypokalemia, including mental status changes, agitation, lethargy, visual disturbances, anorexia, nausea, vomiting, and diarrhea. Treatment of digoxin toxicity includes discontinuation of the drug, correction of precipitating factors (eg hypokalemia, hypoxemia, volume depletion), correction of serum potassium levels, treatment of dysrhythmias, and administration of digoxin-specific Fab antibody fragments to reverse an acute, life-threatening digoxin intoxication.

Sympathomimetic agents, such as dopamine and dobutamine, may be used in an acute hospital setting to increase myocardial contractility (positive inotropy) through beta-adrenergic stimulation. The beta_1_ effects are primarily chronotropic (increased heart rate) and inotropic (increased force of contraction), whereas the beta_2_ effects result in peripheral vasodilation and bronchodilation. At low doses, dopamine causes renal and mesenteric vasodilation, which may improve renal blood flow. At higher doses, dopamine stimulates alpha-adrenergic receptors, causing peripheral vasoconstriction, which increases systemic vascular resistance and afterload, worsening conditions in a patient with a low cardiac output. Dobutamine mainly functions to stimulate beta_1_ receptors. Additionally, phosphodiesterase inhibitors, such as amrinone, act through elevation of cyclic adenosine monophosphate, resulting in increased myocardial contractility and peripheral vasodilation.

Finally, vasodilator therapy may be useful for patients whose symptoms persist despite diuretic and digitalis regimens. Predominantly, venous vasodilators, such as nitroglycerin, nitroprusside, and nitrate preparations (isosorbide), function to reduce preload (resulting in decreased pulmonary congestion), whereas arterial vasodilators, such as angiotensin-converting enzyme inhibitors (captopril, enalapril), hydralazine, minoxidil, and some calcium-channel blockers, reduce cardiac afterload. Angiotensin-converting enzyme inhibitors prevent the conversion of angiotensin I to angiotensin II and thereby decrease systemic vascular resistance and improve renal blood flow. Again, caution must be exercised when instituting any vasodilator therapy to prevent an acute hypotensive event.

**DENTAL MANAGEMENT CONSIDERATIONS**

Because of the relatively frequent occurrence of congestive heart failure in the population, the general dental practitioner is exposed to this disease process. He should begin therapy by determining the patient’s current levels of control and compliance by consultation with the primary care physician or cardiologist. The dentist should have an understanding of the cause of the individual patient’s heart failure and current medications, and most importantly, he should be aware of any recent changes in signs and symptoms or therapy. Patients should avoid excessive fluid intake, excessive sodium in their diets, and smoking. The dental practitioner should realize that digitalis preparations may cause nausea and vomiting if doses are too high (digitalis toxicity), and that patients should be instructed to avoid a heavy meal before their dental appointment. An upright or semierect sitting position should be used, the dentist should avoid stimulating a gag reflex, and a rubber dam should be used cautiously. The dentist should use methods of stress reduction for the patient during the dental appointment, including the avoidance of excessive epinephrine and the use of profound local anesthesia.

Consideration should be given toward monitoring vital signs during dental treatment and toward using supplemental oxygen. In the event of an emergency situation, provisions should be in place for immediate transportation to an emergency care facility. For patients in whom a significant amount of bleeding may be expected (patients undergoing deep scaling and root planing or dental extractions), consideration should be given toward monitoring the prothrombin time (international normalized ratio) in a patient on coumadin therapy, and making the necessary adjustments in medication with the physician’s input. Also, subacute bacterial endocarditis prophylaxis should be provided if indicated (eg, if rheumatic heart disease is the cause of the heart failure). For the majority of patients with congestive heart failure that is adequately controlled, there are no contraindications to ambulatory dental treatment.

**SUGGESTED READING**


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