Heart Failure

Heart failure (HF) is much like anemia in that it represents a symptom complex that may be caused by a number of specific diseases. HF represents the end stage of many of the cardiovascular diseases. The American College of Cardiology/ American Heart Association 2005 Guideline Update for the Diagnosis and Management of Chronic Heart Failure in the Adult1 defines HF as a complex clinical syndrome that may result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood. Patients with untreated or poorly managed HF are at high risk during dental treatment for complications such as cardiac arrest, cerebrovascular accident, and myocardial infarction. The dentist must be able to identify these patients on the basis of history and clinical findings, refer them for medical diagnosis and management, and work closely with the physician to develop a dental management plan that will be effective and safe for the patient.

GENERAL DESCRIPTION

Incidence and Prevalence

Approximately 5 million people in the United States have HF, and more than 550,000 patients each year are given this diagnosis for the first time. 1000 patient-years older than age 65 to 30 per 1000 patient-years older than age 80. The most common underlying causes of HF in the United States are coronary heart disease, hypertension, cardiomyopathy, and valvular heart disease, with coronary heart disease. The second most common cause of HF, accounting for about one fourth of all cases, is dilated cardiomyopathy (DCM). DCM is a syndrome characterized by cardiac enlargement with impaired systolic function of one or both ventricles, Known causes of cardiomyopathy include alcohol abuse, hereditary cardiomyopathy, and viral infection. longstanding history of hypertension. Valvular heart disease used to be a significant cause of HF; however, with rates of rheumatic heart disease and congenital heart disease

BOX 6-1

Most Common Causes of Heart Failure

- Coronary heart disease
- Hypertension
- Cardiomyopathy
- Valvular heart disease
- Myocarditis
- Infective endocarditis
- Congenital heart disease
- Pulmonary hypertension
- Pulmonary embolism
- Endocrine disease

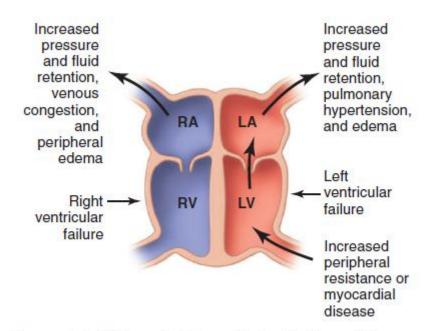


Figure 6-1. Effects of right- and left-sided heart failure.

CLINICAL PRESENTATION

Signs and Symptoms

The symptoms and signs of HF reflect respective ventricular dysfunction. Left ventricular failure produces pulmonary vascular congestion with resultant pulmonary edema and dyspnea. Dyspnea is the most common symptom of HF and usually is present only with exertion or physical activity. Dyspnea at rest is an indication of severe HF. Orthopnea is positional dyspnea, which is precipitated or worsened by a recumbent or semirecumbent position. Most patients with mild to moderate HF do not exhibit orthopnea when treated adequately. Paroxysmal nocturnal dyspnea (PND) is an attack of sudden, severe shortness of breath that awakens the patient from sleep, usually within 1 to 3 hours after the patient goes to bed, and resolves within 10 to 30 minutes after the patient arises, often gasping

for air. The occurrence of PND is uncommon. Both orthopnea and PND are relatively specific indicators of HF and are due to increased venous return encouraged by the recumbent position, with resultant increases in pulmonary venous pressure and alveolar edema. Central regulation of respiration also may be impaired in patients with advanced HF, resulting in alternating cycles of rapid, deep breathing (hyperventilation) and periods of central apnea, called *Cheyne-Stokes respiration*.10 PND is a common clinical feature associated with Cheyne-Stokes respiration in patients with HF. Exercise intolerance (e.g., inability to climb a flight of stairs) is one of the hallmark symptoms of HF and is due to a combination of dyspnea and reduced blood and oxygen supply to the skeletal muscles. Fatigue (especially muscle fatigue) is a common, nonspecific symptom of HF. The pulmonary examination of patients with HF is usually unremarkable. However, rales (or crackles), representing alveolar fluid, are a hallmark of HF when present.

BOX 6-2

Symptoms of Heart Failure

- Dyspnea (perceived shortness of breath)
- Fatigue and weakness
- Orthopnea (dyspnea in recumbent position)
- Paroxysmal nocturnal dyspnea (dyspnea that awakens patient from sleep)
- Acute pulmonary edema (cough or progressive dyspnea)
- Exercise intolerance (inability to climb a flight of stairs)
- · Fatigue (especially muscular)
- Dependent edema (swelling of feet and ankles after standing or walking)
- Report of weight gain or increased abdominal girth (fluid accumulation; ascites)
- · Right upper quadrant pain (liver congestion)
- Anorexia, nausea, vomiting, constipation (bowel edema)
- Hyperventilation followed by apnea during sleep (Cheyne-Stokes respiration)

]	BOX 6-3
Signs	of Heart Failure
Che alte	id, shallow breathing yne-Stokes respiration (hyperventilation rnating with apnea during sleep) piratory rales (crackles)
	irt murmur
• Gall	op rhythm
• Incr	eased venous pressure
• Enla	argement of cardiac silhouette on chest
	ograph
 Puls 	sus alternans
 Dist 	tended neck veins
 Large 	ge, tender liver
 Jaur 	ndice
Peri	pheral edema
Asc	ites
Cya	nosis
• Wei	ght gain
Club	obing of fingers

Chest radiographs may reveal enlargement and displacement of the cardiac silhouette or abnormalities of the pulmonary vasculature. Evidence of interstitial fluid or pleural effusion also may be seen. Right ventricular failure results in systemic venous congestion and peripheral edema.



Figure 6-3. Distended jugular vein in patient with heart failure.



Figure 6-4. Pitting edema in a patient with heart failure. A depression ("pit") remains in the edematous tissue for some minutes after firm fingertip pressure is applied. (From Forbes CD, Jackson WF. Color Atlas and Text of Clinical Medicine. Edinburgh, Mosby, 2004.)



Figure 6-6. Clubbing of the fingers in a patient with congestive heart failure.

Laboratory Findings

electrocardiography, Chest radiography, echocardiography, radionuclide angiography or ventriculography, exercise stress testing, ambulatory electrocardiography (Holter) monitoring. and cardiac catheterization. Measurement of plasma hormone levels of norepinephrine, plasma atrial natriuretic peptide, and plasma renin has possible prognostic value and may be helpful clinically. Routine testing may include complete blood count, renal function testing and electrolytes, liver function testing, blood glucose, lipids, and thyroid function testing.

MEDICAL MANAGEMENT

The medical management of HF is applied with a graduated approach, depending on the stage of the disease For stages A and B, management

begins with risk reduction and includes the identification and treatment of underlying medical problems such as hypertension, atherosclerotic disease, diabetes, obesity, and metabolic syndrome (abdominal obesity, elevated blood glucose, dyslipidemia, hypertension). In addition, behavioral modification is promoted for smoking cessation, weight loss for the obese patient, reduction of risk factors for cardiovascular disease, mild aerobic exercise, adequate rest, and avoidance of alcohol and illicit drugs. Drug therapy may be indicated for the treatment of patients with vascular disease or diabetes in stage A, as well as for those with ventricular dysfunction in stage B. In stage C, all measures from stage A and B apply, in addition to salt restriction and drug therapy. Drug therapy begins with diuretics to control fluid retention. Several types of diuretics, including loop diuretics, thiazide diuretics, and potassium-sparing diuretics, are used. Diuretics are used for three purposes: (1) they are the only drugs that can adequately control fluid retention, (2) they produce more rapid symptomatic relief than other drugs, and (3) they modulate other drugs used to treat HF.

BOX 6-4

NYHA Classification of Heart Failure

- Class I: No limitation of physical activity. No dyspnea, fatigue, or palpitations with ordinary physical activity.
- Class II: Slight limitation of physical activity. These patients have fatigue, palpitations, and dyspnea with ordinary physical activity but are comfortable at rest.
- Class III: Marked limitation of activity. Less than ordinary physical activity results in symptoms, but patients are comfortable at rest.
- Class IV: Symptoms are present at rest, and any physical exertion exacerbates the symptoms.

NYHA, New York Heart Association.

BOX 6-5 Medical Management of Patients With Heart Failure¹ STAGE A (PATIENTS AT HIGH RISK FOR HEART FAILURE [HF] BUT WITHOUT STRUCTURAL HEART DISEASE OR SYMPTOMS OF HF) Treat hypertension, encourage smoking cessation, treat lipid disorders, encourage regular exercise, discourage alcohol intake and illicit drug use, and control metabolic syndrome. Use angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs) in appropriate patients for treatment of vascular disease or diabetes. STAGE B (PATIENTS WITH STRUCTURAL HEART DISEASE BUT WITHOUT SIGNS OR SYMPTOMS OF HF) Provide all measures for stage A, plus ACE inhibitors (or ARBs) in appropriate patients · Beta-blockers in appropriate patients STAGE C (PATIENTS WITH STRUCTURAL HEART DISEASE WITH PRIOR OR CURRENT SYMPTOMS OF HF) Provide all measures for stages A and B, dietary salt restriction, plus Drugs for routine use: diuretics, ACE inhibitors, beta-blockers Drugs in selected patients: aldosterone antagonists, ARBs, digitalis, hydralazine/nitrates Devices in selected patients: biventricular pacing, implantable defibrillators STAGE D (PATIENTS WITH REFRACTORY HF REQUIRING SPECIAL INTERVENTIONS) · Provide appropriate measures from stages A, B, and C. • Use heart transplant, chronic inotropes, permanent mechanical support, experimental drugs, or surgery. Provide compassionate end-of-life care/hospice.

Although diuretics are effective in decreasing the signs and symptoms of fluid retention, they cannot maintain the clinical stability of patients with HF when used alone. Spironolactone, a potassium-sparing diuretic, also blocks the action of aldosterone (aldosterone antagonist) and, when used in patients with class IV symptoms, has been shown to reduce the risk of death by 25% to 30%. Other than spironolactone, diuretics do not influence the natural history of chronic HF. In addition to diuretics, drugs that modulate or decrease neurohormonal activity have become the foundation of treatment for HF. These drugs decrease themorbidity and mortality of HF by inhibiting the cardiotoxic effects of the neurohormonal system, thereby retarding the progression of HF. Several types of neurohormonal antagonists, including enzyme inhibitors angiotensin-converting (ACEIs), beta-adrenergic blockers, and angiotensin receptor blockers (ARBs), are used to treat HF. ACEIs are first-line drugs used to treat patients with HF; they have been shown to reduce risks of death and hospitalization by 20% to 30%. They are typically prescribed along with or following diuretic therapy, and they decrease the need for large doses of diuretics, as well as some of the adverse metabolic effects of diuretics.9 In addition to ACEIs, beta-adrenergic blockers are advocated; when used in combination with ACEIs, beta blockers appear to reduce the risk of death or hospitalization for heart failure by 30% to 40%. Digitalis glycosides have been used for many years for the treatment of patients with HF; digoxin is most commonly prescribed. However, with the advent of ACEIs, their use has declined. Digoxin has not been shown to decrease the risk of death or of hospitalization, as opposed to ACEIs and beta blockers, both of which do decrease these risks. Digoxin is, however, effective in alleviating symptoms; therefore, it is principally used

to treat residual symptoms not controlled by other drugs. Digoxin is the preferred agent in patients with HF who have atrial fibrillation and a rapid ventricular response. A significant problem with digitalis glycosides is their narrow therapeutic range and the resultant toxicity that easily may occur. Other drugs used to treat HF that is unresponsive to ACEIs include angiotensin receptor blockers and direct-acting vasodilators (hydralazine, isosorbide dinitrate). For all patients with HF, drugs that are known to worsen clinical status should be avoided. These include nonsteroidal antiinfl ammatory drugs (NSAIDs), most antiarrhythmic drugs, and calcium channel blockers. In selected patients, other nonpharmacologic measures, such as biventricular pacing or the use of an implantable defi brillator, may be indicated. If drug therapy is found to be inadequate to control the disorder in patients with severe, refractory HF (stage D), mechanical and surgical intervention may be provided. These measures may include intra-aortic balloon counterpulsation, a left ventricular assist device, and heart transplantation. The final measure is end of life hospice care.

BOX 6-6

Clinical Manifestations of Digitalis Toxicity

- Headache, nausea, vomiting
- Hypersalivation
- Altered vision and color perception
- Fatigue, malaise, drowsiness
- · Arrhythmias (tachycardias or bradycardias)

DENTAL MANAGEMENT

Medical Considerations

The risk of treating a patient with symptomatic HF is that symptoms could abruptly worsen and result in acute failure, a fatal arrhythmia, stroke, or myocardial infarction. Identification of patients with a history of HF, those with undiagnosed HF, or those prone to developing HF is the first step in risk assessment and in avoiding an untoward event. It should be recalled that HF is a symptom complex that is the end result of an underlying disease such as coronary heart disease, hypertension, or cardiomyopathy; therefore, the cause of HF must be identified and steps taken toward appropriate dental management. Identification is accomplished by obtaining a thorough medical history, including a pertinent review of systems, and measuring and evaluating vital signs (i.e., pulse rate and rhythm, blood pressure, respiratory rate). All medications that are being taken should be identified as well. In a review of systems, patients should be asked about the presence of signs or symptoms related to the cardiovascular and pulmonary systems. Patients who report shortness of breath, orthopnea, PND, fatigue, or exercise intolerance may have HF or another cardiovascular disease. A report of an inability to climb a flight of stairs without shortness of breath or fatigue may reflect poor functional capacity or diminished cardiopulmonary reserve, with increased risk for an adverse outcome. Patients with a previous history of HF and those who are asymptomatic have compensated HF (NYHA class I). Those who are symptomatic have decompensated HF (NYHA classes II, III, and IV). The dentist must make a determination of the risk involved in providing dental treatment to a patient with HF and must decide whether the benefits of treatment outweigh the risks. This often requires consultation with the physician. The American College of Cardiology and the American

Heart Association have published guidelines that can help the clinician to make this determination. These guidelines are intended for use by physicians who are evaluating patients with cardiovascular disease to determine whether they can safely undergo surgical procedures. They also can be applied to the provision of dental care and are of significant value to the dentist who must make a determination of risk. These guidelines suggest that patients with decompensated HF constitute a major risk for the occurrence of a serious event (acute MI, unstable angina, or sudden death) during treatment. Thus, patients with symptoms of HF (decompensated; NYHA class II, III, or IV) generally are not candidates for elective dental care, and treatment should be deferred until medical consultation can be obtained. Patients who have a history of HF but who are asymptomatic (compensated; NYHA class I) constitute an intermediate risk for occurrence of a serious event. With good functional capacity and reserve (i.e., can climb a flight of stairs), however, they generally can undergo any required treatment with little likelihood of problems. Thus, patients who are NYHA class I may receive routine outpatient dental care. Many patients who are NYHA class II and some who are class III also may undergo routine treatment in an outpatient setting after approval from the physician. Remaining NYHA class III patients and all class IV patients are best treated in a special care facility, such as a hospital dental clinic with continuous monitoring. Recommendations for management include short, stress-free appointments. Patients with HF may not tolerate a supine chair position because of pulmonary edema and will need a semisupine or upright chair position. For patients taking a digitalis glycoside (digoxin), epinephrine should be avoided, if possible, as the combination can potentially precipitate arrhythmias. If it is considered essential to use epinephrine, it should be used

cautiously. A maximum of 0.036 mg epinephrine (two cartridges of 2% lidocaine with 1 : 100,000 epinephrine) is recommended, with care taken to avoid inadvertent intravascular injection. Epinephrine-impregnated gingival retraction cord should be avoided. Patients should be observed for signs of digitalis toxicity, such as hypersalivation. If toxicity is suspected, patients should promptly be referred to their physician. For patients who are NYHA class III or IV, vasoconstrictors should be avoided; however, if their use is considered essential, it should be discussed with the physician. NSAIDs should be avoided because they can exacerbate symptoms of HF. Nitrous oxide plus oxygen sedation may be used if adequate O2 flow (at least 30%) is maintained.

BOX 6-7

Dental Management of the Patient With Heart Failure

- Evaluate patient for history, signs, or symptoms of heart failure (HF).
- For patients with symptoms of untreated or uncontrolled HF, defer elective dental care and refer to physician.
- · For patients diagnosed and treated for HF:
 - · Confirm status with patient or physician
 - New York Heart Association (NYHA) class I patients (asymptomatic)—Provide routine care.
 - NYHA class II (and some class III patients)— Obtain consultation with physician for medical clearance and provide routine care.
 - NYHA (some class III and class IV) patients— Obtain consultation with physician; consider treatment in a special care or hospital setting.
 - Identify underlying cardiovascular disease (i.e., coronary artery disease, hypertension, cardiomyopathy, valvular disease), and manage appropriately.
- Drug considerations:
 - For patients taking digitalis, avoid epinephrine; if considered essential, use cautiously (maximum 0.036 mg epinephrine or 0.20 mg levonordefrin); avoid gag reflex; avoid erythromycin and clarithromycin, which may increase the absorption of digitalis and lead to toxicity.
 - For patients with NYHA class III and IV congestive heart failure, avoid use of vasoconstrictors; if use is considered essential, discuss with physician.
 - Avoid epinephrine-impregnated retraction cord.
- See Table 6-1 for drug considerations and adverse effects.
- Schedule short, stress-free appointments.
- Use semisupine or upright chair position.
- Watch for orthostatic hypotension, make position or chair changes slowly, and assist patient into and out of chair.
- Avoid the use of nonsteroidal antiinflammatory drugs (NSAIDs).
- Watch for signs of digitalis toxicity (i.e., tachycardia, hypersalivation, visual disturbances, etc.).
- Nitrous oxide/oxygen sedation may be used with a minimum of 30% oxygen.

Treatment Planning Modifications

In general, patients with HF who are under good medical management can receive any indicated dental treatment as long as the dental management plan deals effectively with the problems presented by HF, its underlying cause, and the effects of medications. Patients with symptomatic HF present a definite challenge that mandates specific management considerations.

Oral Manifestations

No oral manifestations are related to HF per se; however, many of the drugs used to manage HF can cause dry mouth and oral lesions. Digitalis may exaggerate the patient's gag reflex.

Suggestive Reading

James W Little, Craig S Miller, Nelson L Rhodus. Dental management of medically compromised patient, 9th edition, Elsevier, 2018