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Valvular Heart Disease and Heart Failure: Dental Management Considerations

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Valvular heart disease as a sequela of rheumatic fever has steadily declined in developed countries, but remains a significant problem elsewhere. Valvular disease is now more often due to degenerative disease, ischemia, calcification, or other functional causes. The prevalence of at least moderate calcific aortic valve stenosis is around 5% in the elderly, and the prevalence of at least moderate mitral valve regurgitation is around 11% [1,2]. According to the American Heart Association, approximately 5 million people are diagnosed with valvular heart disease in the United States each year. Many of these will present to the general dentist for routine dental care. The dentist must then understand the implications of this disease process to treat these patients safely and effectively. Of course patients with pathologic valve disease are managed in close consultation with their physicians. Even so, a dentist with knowledge of the disease process, as well as its diagnosis and treatment, has greater confidence when treating these patients. Patients with aortic stenosis carry the greatest risk of perioperative morbidity, though other decompensated valvular conditions also have implications when planning and performing procedures. Appropriate preoperative evaluation allows morbidity to be minimized.

There are three main concerns when providing dental care for patients with valvular heart disease:

- The risk of infective endocarditis

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- The risk of bleeding in anticoagulated patients
- The risk of exacerbating any coexisting heart failure

Valvular heart disease can be classified as primary or secondary. Primary valve disease comprises conditions in which structural abnormalities of the valves lead to abnormal function. Secondary valve disease occurs when the valve is structurally normal but is functionally impaired due to other secondary cardiovascular disorders, such as aortic root dilatation, ischemic mitral regurgitation, and cardiomyopathies.

One must first have an understanding of normal flow across the four heart valves before a discussion of pathologic valve disease can be undertaken. Blood flows from the right and left atria into the ventricles through the tricuspid and mitral valves, respectively. When the ventricles are full, the pressure gradient is such that these valves close. This closure generates the first heart sound on auscultation and prevents blood flowing back into the atria as the ventricles contract. As the ventricles contract, the pulmonic and aortic valves open and blood is pumped from the right ventricle to the lungs via the pulmonary artery, and from the left ventricle to the systemic circulation via the aorta. When ventricular contraction ends, the pulmonic and aortic valves close, thereby generating the second heart sound and preventing backflow of blood into the ventricles.

Primary valve disease may affect any of the four heart valves and may result in narrowing (stenosis) or regurgitation (incompetence) with the net effect being hemodynamic instability of varying severity. In essence, stenotic valves increase the work the heart has to do to push blood through the valve. Incompetent valves allow retrograde blood flow and overload of the chamber proximal to the valve, again making the heart work harder to ensure adequate forward flow. Both of these processes may precipitate heart failure.

Causes of valvular heart disease

In general, primary valve disease is either congenital or acquired.

Congenital valve disease most often affects the aortic or pulmonic valves. A bicuspid aortic valve is the most common congenital valve defect. The absence of the third cusp may result in stenosis, regurgitation, and, later, calcification.

Acquired valve disease involves structural changes to normal valves due to a variety of diseases or infections, including rheumatic fever and endocarditis. In addition, certain drugs and toxins may cause valvular disease. The anorectic drugs fenfluramine and phentermine have been shown to be associated with valvular heart disease [3], and were withdrawn from the market in 1997 as a result.

Secondary valve disease may be due to ischemia, myocardial infarction, cardiomyopathy, syphilis, aortic aneurysm, connective tissue diseases, tumors, drugs, or radiation.

Diagnosis

Cardiac auscultation to detect murmurs is the most widely used method to screen for valvular heart disease. Although heart murmurs may have no pathological significance, they may indicate the presence of valvular, congenital, or other structural abnormalities of the heart. Murmurs are due to three factors: (1) high blood flow through normal or abnormal orifices, (2) forward flow through a narrowed or irregular orifice, or (3) backward or regurgitant flow through an incompetent valve, septal defect, or patent ductus arteriosus. Regardless of etiology, the murmur is generated by turbulent blood flow. Murmurs are assessed based on when they occur in the cardiac cycle, configuration (crescendo, decrescendo, crescendo–decrescendo, or plateau), location and radiation, pitch and intensity (grades 1–6), and duration. Most systolic murmurs do not signify cardiac disease and many are related to physiological increases in blood-flow velocity [4]. In other instances, a systolic murmur may indicate cardiac disease (eg, aortic stenosis) that may be physiologically important even when asymptomatic. Conversely, virtually all diastolic murmurs, like most continuous murmurs, indicate underlying pathological conditions and require further workup [4].

An important consideration in a patient with a murmur is the presence or absence of symptoms. For example, a history of syncope, angina pectoris, or heart failure in a patient with a midsystolic murmur should trigger a more aggressive workup than in a patient with a similar murmur but no symptoms.

Evaluation

Heart murmurs are evaluated according to:

- The lesion's etiology and severity
- The hemodynamics
- Coexisting abnormalities and secondary lesions
- Cardiac chamber size and function

Echocardiography provides specific and quantitative information and may be the only test needed. Echocardiography provides images of cardiac and valvular structure and function. In addition, it measures the velocity of blood flow across valves. Two-dimensional echocardiography may indicate abnormal valve motion or structure while Doppler echocardiography indicates the direction and velocity of blood flow. The velocity reflects the pressure gradient across stenotic and regurgitant valves. However, such testing is not necessary for all patients, especially those with asymptomatic grade 1 or 2 midsystolic murmurs. If the diagnosis is still not clear after transthoracic echocardiography, transesophageal echocardiography may be needed. The sensitivity of these studies is such that tricuspid and pulmonary regurgitation may be detected in a significant number of healthy young patients,

and mitral regurgitation may be detected in a smaller number of healthy young patients [5–7].

ECG and chest radiography provide useful negative information, such as the absence of ventricular hypertrophy, atrial abnormality, dysrhythmias, ischemia and prior myocardial infarction, pulmonary vascular redistribution indicative of heart failure, and valvular calcifications.

Cardiac catheterization may provide information about the severity of valvular obstruction, regurgitation, or intracardiac shunting, but is not necessary in most patients.

Radionucleotide coronary angiography and magnetic resonance imaging may be helpful in assessing left ventricular function when the echocardiogram is inconclusive.

Specific valvular disorders

Aortic stenosis

The major causes of aortic stenosis are congenital bicuspid aortic valves, rheumatic valvular disease, and age-related calcification. Severe or symptomatic aortic stenosis poses the greatest risk for surgery in general [8] and to dental procedures as well. An assessment of aortic stenosis severity is based upon measurements of valve area, the pressure gradient across the valve, and the presence of symptoms. The valve area must be reduced by 75% before significant hemodynamic changes occur. The area of the normal adult aortic valve is 3 to 4 cm^2. Therefore, an area <1 cm^2 is considered severe stenosis, an area of 1 to 1.5 cm^2 is moderate stenosis, and an area of >1.5 cm^2 is considered mild stenosis [9]. Although this information is important, symptoms are the primary determinant for aortic valve replacement. Therefore, the presence of angina, heart failure, shortness of breath, and syncope are extremely relevant in this group of patients. Symptoms often appear late in the disease process. Without surgical intervention, life expectancy is 5 years after the onset of angina and 2 years after the onset of cardiac failure.

The ventricle becomes hypertrophied in response to the increased load and becomes susceptible to ischemia because of increased intraventricular pressure, increased muscle mass, and decreased coronary perfusion pressure. As a result, patients with aortic stenosis are at risk of perioperative ischemia, myocardial infarction, and death. During periods of exercise or stress, tachycardia or increased peripheral vascular resistance could result in an exaggerated pressure gradient and a marked increase in left ventricular systolic pressure, resulting in acute pulmonary edema or cardiac ischemia. Therefore, elective procedures should be postponed in severe or symptomatic aortic stenosis until the valve has been replaced. Patients who are not suitable candidates for valve replacement may be candidates for balloon aortic valvuloplasty. Although this procedure is efficacious in adolescents and young adults, it is only a temporary measure in older adults due to re-stenosis and

clinical deterioration occurring within 6 to 12 months [10,11]. In summary, most patients with mild or moderate asymptomatic aortic stenosis tolerate dental procedures well, but require antibiotic prophylaxis for endocarditis, which will be discussed in detail later in this article.

Mitral stenosis

Although rheumatic heart disease is becoming increasingly rare, it is still the major cause of mitral stenosis. The normal mitral valve area is 4 to 5 cm^2, and it must be narrowed to <2.5 cm^2 before symptoms develop. A valve area >1.5 cm^2 usually does not result in symptoms at rest [12]. The severity of mitral valve stenosis may be classified as follows:

Mild: valve area >1.5 cm^2
Moderate: valve area 1 to 1.5 cm^2
Severe: valve area <1 cm^2

As the mitral valve narrows, the left atrium enlarges over time, and many patients develop atrial fibrillation as a result, which in turn increases the risk of systemic embolization and stroke from atrial thrombus. These patients might be anticoagulated, further complicating their dental management. Additionally, increased atrial pressure is transmitted back to the pulmonary veins, resulting in pulmonary edema and ultimately pulmonary hypertension. Tachycardia is poorly tolerated since it reduces the diastolic filling time, causing further pulmonary vein congestion. Furthermore, a supine position may exacerbate pulmonary edema and shortness of breath.

The surgical treatment options for mitral stenosis are not limited to valve replacement, but also include valve repair procedures, such as valvotomy or balloon valvuloplasty, since the re-stenosis rates are lower than those in aortic stenosis [13]. In summary, the supine position or any stimulus causing tachycardia can suddenly precipitate pulmonary edema and cause shortness of breath. Atrial fibrillation is also a factor to bear in mind, not only in terms of its acute onset, but also in terms of anticoagulation therapy. However, if the mitral stenosis is mild, there is minimal impact to dental care other than the need for antibiotic prophylaxis for the prevention of endocarditis.

Mitral regurgitation

Etiologies for mitral regurgitation include rheumatic heart disease, mitral valve prolapse, ischemic heart disease, endocarditis, collagen vascular diseases, Marfan syndrome, and papillary muscle rupture after myocardial infarction. Mitral regurgitation allows blood to flow back into the left atrium during ventricular contraction. This regurgitant volume increases the left atrial pressure and may be transmitted back to the pulmonary veins. If the onset of mitral regurgitation is acute, as in the setting of myocardial infarction, this increased atrial pressure results in pulmonary edema. Conversely, if the mitral regurgitation is chronic, the left atrium slowly enlarges

without the increase in pressure and without signs of pulmonary edema. As is the case with mitral stenosis, patients with mitral regurgitation are predisposed to atrial fibrillation from atrial enlargement. Also, a compensatory eccentric enlargement of the left ventricle maintains a normal forward flow stroke volume. Over many years, this enlargement will result in ventricular dysfunction. The ejection fraction may be maintained in the low normal range (0.5–0.6) by these compensatory mechanisms, despite significant muscle dysfunction [14]. The goal in managing mitral regurgitation is to time the intervention before irreversible left ventricular dysfunction develops. To maintain forward flow, vasodilators (eg, calcium channel blockers and angiotensin converting enzyme inhibitors) are sometimes used, along with diuretics for symptom control, but definitive treatment is mitral valve repair or replacement. In summary, these patients are susceptible to exacerbation of pulmonary edema and acute shortness of breath by any increase in systemic vascular resistance. They are also at risk for atrial fibrillation. Dentists should consider these factors, as well as the need for antibiotic prophylaxis, while providing dental treatment.

Aortic regurgitation

Aortic regurgitation may result from rheumatic heart disease, endocarditis, trauma, collagen vascular diseases, and processes that dilate the aortic root (eg, aneurysm, Marfan syndrome, and syphilis). Aortic regurgitation allows blood flow back into the left ventricle. If this is an acute onset, it results in an increased left ventricular and atrial pressure that backs up to cause pulmonary edema. If this is a chronic process, the compensatory mechanisms (ie, increased compliance and ventricular hypertrophy) handle the ventricular volume overload. Thus, many patients are asymptomatic. In managing these patients, the goal is to maintain a normal or slightly elevated heart rate to minimize regurgitation and maintain aortic diastolic and coronary artery perfusion pressure. It is also prudent to avoid vasoconstricting drugs because they may cause an increase in systemic vascular resistance and a worsening of the regurgitation across the valve. In addition to diuretics for symptom control, vasodilators (eg, calcium channel blockers and angiotensin converting enzyme inhibitors) are sometimes used in an effort to maintain forward flow by reducing afterload. However, once symptoms develop, the definitive treatment is valve repair or replacement.

Prosthetic valves

The progression of valvular heart disease often results in the need for replacement of the diseased valve. Replacement valves can be classified as biological or mechanical. Biological valves, also known as bio-prosthetic valves, are either homografts (from cadavers) or xenografts (from pigs or cows), and are sewn onto a supporting frame. Most mechanical valves use either a tilting

disc, bileaflet valves, or a ball valve mechanism. The dentist treating patients with prosthetic valves must consider (1) the need for antibiotic prophylaxis against infective endocarditis and (2) the management of periprocedural anticoagulation, since thromboembolism is a devastating and often catastrophic complication of valve replacement surgery. The mechanical valves are more durable but require lifelong anticoagulation to reduce the risk of thrombosis and thromboembolism. Even after anticoagulation with warfarin, the risk of thromboemboli from mechanical valves is approximately 1% to 2% [15–17], and the risk is considerably higher without anticoagulation [18]. Biological valves, conversely, appear to be at lower risk of thromboembolic events. Without anticoagulation, the risk of thromboemboli associated with biological valves is approximately 0.7% [15–17]. Studies show that the risk of thromboemboli is greater with low-flow valves (eg mitral and tricuspid) for both mechanical and biological valves [15,18–20]. With either mechanical or biological prostheses, the risk is probably greatest in the first few months after placement [21], before the valve is endothelialized. In light of this risk, patients with mechanical valves are anticoagulated lifelong, while patients with biological valves are usually only anticoagulated with warfarin for 3 months after placement and thereafter with aspirin, unless there are associated risk factors, such as atrial fibrillation, left ventricle dysfunction, previous thromboembolism, or hypercoagulable states, in which cases warfarin anticoagulation should continue [12]. Those patients with low-flow valves often require a higher level of therapeutic anticoagulation.

Patients undergoing dental procedures that cause significant bleeding require careful anticoagulation management. The 5th Consensus Conference on Anticoagulation recommends that for procedures associated with significant bleeding the international normalized ratio (INR) be reduced to low or to the subtherapeutic range and that the normal dose of oral anticoagulation be resumed immediately after the procedure [22]. Patients at high risk of thromboemboli when anticoagulation is decreased include those with a mechanical valve in the mitral position, a Bjork–Shiley valve, recent thromboemboli (within 1 year), or three or more of the following risk factors: atrial fibrillation, previous thromboemboli (at any time), hypercoagulable condition, mechanical prosthesis, and left ventricular ejection fraction of $<30\%$. These high-risk patients should be managed with perioperative heparin [12]. Most dental procedures can be safely performed with an INR of up to 4.0 without having to stop anticoagulation [23]. The therapeutic INR for some valvular prostheses may be as high as 3.5 [12]. The risk of lowering the INR must be weighed against the risk of thromboembolism, and this decision must be made only after consultation and liaison with the patient's physician. If anticoagulation is to be stopped, the current guidelines are [12]:

- Stop warfarin 72 hours before the procedure and restart immediately after the procedure or after active bleeding has been controlled.

- Stop aspirin 1 week before the procedure and restart the day after the procedure or after active bleeding has been controlled.

The risk of stopping anticoagulation for 2 to 3 days in patients other than those in the high-risk group mentioned above is negligible [24]. Fortunately, many dental procedures can proceed without the need to manipulate the INR. Even after dental extraction or oral surgical procedures, bleeding can be controlled with local measures [25,26]. Such local measures include suturing, gelatin sponges, oxidized cellulose, topical thrombin, or antifibrinolytic mouthwashes, such as tranexamic acid. Regardless of whether the oral anticoagulation is stopped or continued, the INR must be assessed just before any procedures that may cause bleeding.

Infective endocarditis

Infective endocarditis is an infection of the mural, septal, or valvular endocardium. It may be caused by numerous organisms, including bacteria and fungi. More virulent organisms, such as *Staphylococcus aureus*, produce a rapid and destructive infection, while viridans strains of streptococci, enterococci, other Gram-positive and Gram-negative bacilli, and fungi tend to produce a subacute infection. Most patients in whom infective endocarditis develops have pre-existing valvular disease. The exceptions are intravenous-drug abusers and those with hospital-acquired infections in whom pre-existing valve disease is often absent.

Native valve endocarditis is usually caused by viridans streptococci, group D streptococci, *S aureus*, enterococci or HACEK organisms (ie, *Haemophilus aphrophilus, Haemophilus parainfluenzae, Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens* and *Kingella kingae*). Streptococci formerly accounted for approximately 60% of native valve endocarditis, but the number caused by *S aureus* has been increasing. In intravenous-drug abusers, *S aureus* accounts for over 60% of all cases of endocarditis and for 80% to 90% of cases involving the tricuspid valve. The remainder are caused by enterococci and streptococci, with a small number being caused by Gram-negative aerobic bacilli and fungi.

The microbiology of prosthetic valve endocarditis is distinctive. Early infections (within the first 2 months after valve placement) are commonly caused by staphylococci, Gram-negative organisms, and fungi. In late prosthetic valve endocarditis, infections are mostly caused by streptococci.

Incidence

The estimated frequency of infective endocarditis varies from 1 to 5 cases per 100,000 population per year [27–29]. In those under the age of 30, the ratio of men to women affected is 1:1, but in those aged over 35 the ratio

is 2:1 [30], and in the elderly the ratio is 5:1 [31]. This male predominance could be due to a higher incidence of intravenous drug abuse and congenital heart disease in males.

The mitral valve is affected in 28% to 45% of patients, the aortic valve in 5% to 36%, both valves in up to 35%, and the tricuspid valve in $<10\%$ [32]. These wide ranges stem from the different socioeconomic factors and intravenous-drug abuse rates among the populations studied. In recent years, the decline of rheumatic fever and the increase in degenerative valve disease has resulted in a greater proportion of patients with aortic valve endocarditis.

The risk of prosthetic valve endocarditis is approximately 2% per year for aortic valve prostheses and 0.5% per year for mitral valve prostheses [33]. While the risk of a normally functioning prosthesis being infected after a dental procedure is probably no greater than for a diseased or damaged native valve, the morbidity and mortality of an infected prosthetic valve is much greater, approaching 50% [33].

Pathogenesis and complications

Studies suggest that endothelial damage leads to platelet and fibrin deposition forming a vegetation. In the presence of bacteremia, the organisms colonize the vegetation, resulting in an infective vegetation. The course of infective endocarditis is determined by the degree of damage to the heart, which valve is affected, the presence of metastatic foci, and whether embolization occurs. Local destruction can result in valvular regurgitation as well as myocardial and aortic wall abscesses creating aneurysms. Peripheral embolization results in cerebral, myocardial, pulmonary, splenic or renal infarcts, and septic foci.

Antibiotic prophylaxis for infective (bacterial) endocarditis

Infective endocarditis carries significant morbidity and mortality. Antibiotic prophylaxis for infective bacterial endocarditis is based on the premise that the insulting bacteremia will be reduced or eliminated if antibiotics are administered before any procedure that may cause a bacteremia.

Several issues must be considered when recommending antibiotic prophylaxis for endocarditis. Evidence supporting antibiotic prophylaxis for endocarditis is based on clinical experience documenting endocarditis after bacteremia, and the fact that bacteremia consisting of organisms known to cause endocarditis follows various procedures, including dental procedures [34,35]. In addition, both animal models and human clinical studies show benefit from antibiotic prophylaxis against endocarditis [36,37]. However, the following evidence raises questions about the value of prophylaxis:

- Lack of any sufficiently sized, controlled human clinical trials to support the translation of animal study results to humans.

- Clinical reports of failure of antibiotic prophylaxis against endocarditis [38], and studies that appear to show that prophylaxis is not protective [37].
- Only circumstantial evidence that dental or other procedures cause endocarditis. The incidence of bacteremia is as high as 88% in periodontal surgery and 100% after dental extraction [35], yet the incidence of endocarditis is low.
- In specific circumstances, such as prophylaxis for all cases of mitral valve prolapse, the risk of death from penicillin prophylaxis is estimated to be greater than the risk of infective endocarditis [39–41].

Nevertheless, recommendations for prophylaxis have been in place in most countries for many years, and have been periodically updated based on new scientific information. The key principles of antibiotic prophylaxis against endocarditis include identification of patients at risk, identification of procedures that pose a risk, and the use of antibiotics with a spectrum of activity appropriate for the organisms known to cause bacteremia and endocarditis. Guidelines from the American Heart Association [42] identify patients at risk and stratify them into high-, moderate-, and low-risk groups based upon the cardiac defect involved (Box 1).

These guidelines also include the dental procedures for which antibiotic prophylaxis is recommended and those for which it is not necessary (Box 2). In 1997 the American Heart Association revised the guidelines on appropriate antibiotic prophylaxis (Table 1). One significant change was the elimination of the second dose of amoxicillin previously advocated. This second dose is now considered unnecessary because of the prolonged serum levels, which exceed the minimal inhibitory concentration of most streptococci, and the prolonged effect against such strains (6–14 hours) [43]. Clindamycin is still recommended as an alternative in patients allergic to penicillin. First-generation cephalosporins (cephalexin or cefadroxil) and certain macrolides (azithromycin or clarithromycin) are also acceptable alternative agents. When parenteral administration is needed in an individual allergic to penicillin, clindamycin is recommended and cefazolin may be used as an alternative. Earlier guidelines listed erythromycin as an alternate agent for the penicillin-allergic patient. Erythromycin is no longer included because of gastrointestinal upset and complicated pharmacokinetics of the various formulations.

Although the goal is to identify patients at risk for infective endocarditis and to administer antibiotic prophylaxis before treatment, situations may arise where the need for prophylaxis may only become apparent during treatment. This may be the case where the dentist encounters unexpected bleeding or provokes bacteremia in an at-risk patient. In such cases, antibiotic prophylaxis should be administered as soon as possible. Animal studies have demonstrated that antibiotics administered up to 2 hours after the unexpected bacteremia will provide effective prophylaxis [42,44].

Box 1. Cardiac conditions associated with endocarditis

Endocarditis prophylaxis recommended
High-risk category
- Prosthetic cardiac valves, including bioprosthetic and homograft valves
- Previous bacterial endocarditis
- Complex cyanotic congenital heart disease (eg, single ventricle states, transposition of the great arteries, tetralogy of Fallot)
- Surgically constructed systemic pulmonary shunts or conduits

Moderate-risk category
- Most other congenital cardiac malformations (other than above and below)
- Acquired valvular dysfunction (eg, rheumatic heart disease)
- Hypertrophic cardiomyopathy
- Mitral valve prolapse with valvular regurgitation and/or thickened leaflets

Endocarditis prophylaxis not recommended
Negligible-risk category (no greater risk than the general population)
- Isolated secundum atrial septal defect
- Surgical repair of atrial septal defect, ventricular septal defect, or patent ductus arteriosus (without residua beyond 6 months)
- Previous coronary artery bypass graft surgery
- Mitral valve prolapse without valvular regurgitation
- Physiologic, functional, or innocent heart murmurs
- Previous Kawasaki disease without valvular dysfunction
- Previous rheumatic fever without valvular dysfunction
- Cardiac pacemakers (intravascular and epicardial) and implanted defibrillators

Adapted from Dajani AS, Taubert K, Wilson W, et al. Prevention of bacterial endocarditis. Recommendations by the American Heart Association. JAMA 1997; 277(22):1795; with permission.

If a patient is already taking an antibiotic normally used for endocarditis prophylaxis, it is prudent to select a drug from a different class rather than to increase the dose of the current antibiotic. If the patient is taking penicillin, the dentist should select clindamycin, azithromycin, or clarithromycin

Box 2. Dental procedures and endocarditis prophylaxis

Endocarditis prophylaxis recommended for patients with high- and moderate-risk cardiac conditions

- Dental extractions
- Periodontal procedures, including surgery, scaling and root planing, probing, and recall maintenance
- Dental implant placement and reimplantation of avulsed teeth
- Endodontic (root canal) instrumentation or surgery only beyond the apex
- Subgingival placement of antibiotic fibers or strips
- Initial placement of orthodontic bands but not brackets
- Intraligamentary local anesthetic injections
- Prophylactic cleaning of teeth or implants where bleeding is anticipated

Endocarditis prophylaxis not recommended

- Restorative dentistry, operative and prosthodontic, and including restoration of decayed teeth (filling cavities) and replacement of missing teeth, with or without retraction cord (Clinical judgment may indicate antibiotic use in selected circumstances where significant bleeding may occur.)
- Local anesthetic injections (nonintraligamentary)
- Intracanal endodontic treatment; post placement and buildup
- Placement of rubber dams
- Postoperative suture removal
- Placement of removable prosthodontic or orthodontic appliances
- Taking of oral impressions
- Fluoride treatments
- Taking of oral radiographs
- Orthodontic appliance adjustment
- Shedding of primary teeth

Adapted from Dajani AS, et al. Prevention of bacterial endocarditis. Recommendations by the American Heart Association. JAMA 1997;277(22):1797; with permission.

for prophylaxis. Cephalosporins should be avoided because of cross- resistance. If possible, the procedure could be delayed until at least 9 to 14 days after completion of the antibiotic [44,45]. This will allow the usual oral flora to be re-established.

Chlorhexidine hydrochloride and povidone-iodine mouth rinses may also reduce the incidence and magnitude of bacteremia before dental treatment.

Table 1
Prophylactic regimens for dental, oral, respiratory tract, or esophageal procedures

Situation	Agent	Regimen
Standard general prophylaxis	Amoxicillin	Adults: 2.0 g; children: 50 mg/kg[a] orally 1 h before procedure
Unable to take medications	Ampicillin	Adults: 2.0 g intramuscularly or intravenously; children: 50 mg/kg[a] intramuscularly or intravenously within 30 min before procedure
Allergic to penicillin	Clindamycin	Adults: 600 mg; children: 20 mg/kg[a] orally 1 h before procedure
	Cephalexin[b] or cefadroxil[b]	Adults: 2.0 g; children: 15 mg/kg[a] orally 1 h before procedure
	Azithromycin or clarithromycin	Adults: 500 mg; children: 15 mg/kg[a] orally 1 h before procedure
Allergic to penicillin and unable to take oral medications	Clindamycin	Adults: 600 mg; children: 20 mg/kg[a] intravenously within 30 min before procedure
	Cefazolin[b]	Adults: 1.0 g; children: 25 mg/kg[a] intramuscularly or intravenously within 30 min before procedure

[a] Total children's dose should not exceed adult dose.

[b] Cephalosporins should not be used in individuals with immediate-type hypersensitivity reaction (eg, urticaria, angioedema, or anaphylaxis) to penicillins.

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Moreover, patients at risk for infective endocarditis, including patients with prosthetic heart valves and valvular disease, should be encouraged to maintain meticulous oral hygiene, since the presence of gingivitis, periodontal disease, and infections may result in bacteremia and infective endocarditis.

Heart failure

Heart failure is the inability of the heart to pump blood at a rate required by the metabolizing tissues, or when the heart can do so only with an elevated pressure. Heart failure occurs most frequently in the elderly population. Approximately 5 million patients in the United States have heart failure, with 500,000 new cases each year [46]. The prevalence of heart failure rises from $<1\%$ in individuals under 60 years of age to 6% to 10% in those over 65 years [47]. Heart failure has multiple etiologies, including ischemic heart disease, myocardial infarction, chronic hypertension,

cardiomyopathies (dilated, hypertrophic, alcoholic, and idiopathic), valve dysfunction, cardiac dysrhythmias, conduction defects, pericardial disease, and infection (viral myocarditis and HIV). Heart failure carries a poor prognosis, with an age-adjusted 5-year mortality rate of 59% for men and 45% for women [48]. Once symptoms develop, the prognosis is significantly worse with a 1-year mortality rate of around 45% [49].

Over the past several decades, our understanding of the pathophysiology of cardiac failure has evolved, and no single conceptual paradigm has withstood the test of time. Initial knowledge and management was based upon the ''cardio-renal model'' in which heart failure was viewed as a problem of excessive retention of salt and water caused by abnormalities in renal blood flow. Subsequent hemodynamic measurements demonstrated that heart failure was associated with reduced cardiac output and excessive peripheral vasoconstriction, and this led to the development of the ''cardiocirculatory'' or ''hemodynamic model'' for heart failure. These early models adequately describe salt retention and water retention, and account for the hemodynamic changes seen in cardiac failure. Furthermore, they provide a basis for treatment using diuretics, inotropes, and vasodilators. However, neither model is able to fully account for the relentless progression of heart failure, nor have they led to improved survival for patients with moderate-to-severe heart failure. It is now accepted that heart failure cannot be defined in simple hemodynamic terms, and really represents a summation of multiple anatomical, functional, and biological alterations that coexist and interact in a complex manner. So, although these early explanations are certainly part of the story, more recent models, such as the ''neurohormonal model,'' offer a new rationale and present novel treatment strategies.

Based on the hemodynamic model, the systolic function of the heart is governed by four major factors: the contractile state of the myocardium, the preload of the ventricle (the end diastolic volume and the resultant myocardial fiber length before contraction), the afterload applied to the ventricle (the resistance to left ventricular ejection), and heart rate. Cardiac failure may result from alterations in any of these factors. In most cases, the primary factor is reduced myocardial contractility caused by either functional muscle loss (eg, following myocardial infarction) or by processes affecting the myocardium more diffusely, such as cardiomyopathies. However, failure may also be due to excessive preload, as in valvular regurgitation, or excessive afterload, as in aortic stenosis or severe hypertension. Pump function is also affected by tachycardia and bradycardia. Whereas the normal heart can tolerate wide variations in preload, afterload, and heart rate, the diseased heart does not have this tolerance. In addition, 20% to 40% of heart failure may be due to diastolic dysfunction when filling of the ventricles is affected. This may result from impaired relaxation or reduced compliance of the ventricles. In these situations, the cardiac output is reduced despite normal systolic function and ejection fraction. Moreover, many of the changes that occur in the cardiovascular system as a result of aging affect diastolic

function more than systolic function [50]. Although the mortality of diastolic dysfunction is less than that for systolic dysfunction, diastolic dysfunction is associated with significant morbidity due to dyspnea and fatigue [51]. In practice, the diagnosis of diastolic heart failure is made in the presence of symptoms consistent with heart failure but a normal ejection fraction and no valvular abnormalities.

Although these models offer explanations for the basic hemodynamic alterations seen in heart failure, a complex blend of structural, functional, and biological changes are thought to account for the progressive nature of cardiac failure, as well as for the efficacy or failure of therapies [52]. For example, the rationale for the use of beta-blockers in a patient with a poorly contracting heart is based on a paradigm broader than the treatment of congestion with diuretics or inotropes alone. It is instead founded on an understanding of the role of the sympathetic nervous system in promoting the release of renin and other vasoactive substances that trigger vasoconstriction, tachycardia, and deleterious changes in myocytes leading to ventricular dilatation and the progression of cardiac dysfunction. Thus, the earlier hemodynamic model of heart failure considered the effect of an altered load on the failing ventricle and advocated the use vasodilators and inotropic agents. By comparison, the recent neurohormonal model recognizes the importance of biologically active substances, such as norepinephrine, angiotensin II, endothelin, aldosterone, and tumor necrosis factor. This paradigm shift has guided efforts to antagonize the effects of these circulating biologically active substances.

To summarize, cardiac failure is currently viewed as a progressive condition precipitated by a primary event and followed by a relentless progression of dysfunction. This primary event may have an abrupt onset, such as myocardial infarction, or an insidious onset, as is the case in hereditary cardiomyopathies. Regardless of what initiated heart failure, a complex interaction of several structural, functional, and biological factors determines the progression of dysfunction, resulting in a decline in pumping capacity of the heart.

Goldman and colleagues [8] identified congestive heart failure as a significant risk factor for postoperative morbidity and mortality. Although this study included patients undergoing general anesthesia, the data emphasize the importance of cardiac failure to the dental practitioner.

Classification

The executive summary from the American College of Cardiology and the American Heart Association [46] puts forward a new classification that considers the progression of disease by identifying four stages of heart failure:

- Stage A—patients at high risk of heart failure but without any structural disorder.
- Stage B—patients with a structural disorder of the heart but without symptoms of heart failure.

Stage C—patients with present or past symptoms of heart failure and a structural disorder.
Stage D—patients with end-stage disease requiring specialized treatments, such as mechanical circulatory support, continuous inotropic infusion, or cardiac transplantation.

This new classification recognizes the early risk factors and structural abnormalities that may precede the diagnosis of heart failure. Furthermore, it offers a staging system that is linked to therapeutic strategies appropriate for each stage (Fig. 1). The traditional definition of heart failure, described in the New York Heart Association (NYHA) functional classification, focuses on stages C and D and is based upon severity of symptoms and functional limitations, which may fluctuate throughout the course of the disease process. The NYHA classification defines four classes:

Class I—asymptomatic patients.
Class II—patients symptomatic with moderate exertion.
Class III—patients symptomatic with mild exertion.
Class IV—patients symptomatic at rest.

This new classification is an adjunct to the NYHA classification and offers a similar thought process to that already established in the treatment of cancer, including the screening of patients at risk.

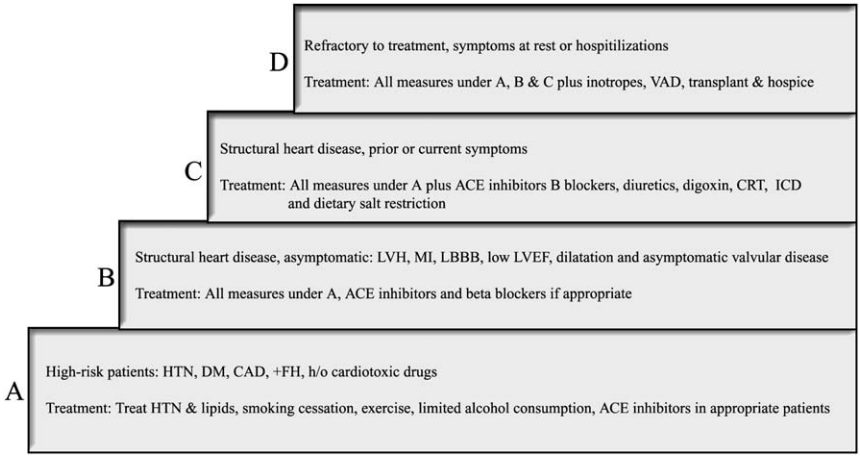

Fig. 1. Classification of American College of Cardiology and American Heart Association for stages of heart failure. ACE, angiotensin-converting enzyme; CAD, coronary artery disease; CRT, cardiac resynchronization therapy; DM, diabetes mellitus; FH, family history; h/o, history of; HTN, hypertension; ICD, implantable cardioverter-defibrillator; LBBB, left bundle branch block; LVEF, left ventricular ejection fraction; LVH, left ventricular hypertrophy; MI, myocardial infarction; VAD, ventricular assist device. (*Adapted from* Jessup M, Brozena S, Medical progress in heart failure. N Engl J Med 2003;348:2013.)

Symptoms and signs

Although nonspecific, the common symptoms of heart failure include:

- Shortness of breath (exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea, or dyspnea at rest)
- Fluid retention and edema
- Abdominal pain due to liver congestion
- Normal vital signs but the presence of tachycardia, hypotension, and reduced pulse pressure
- Jugular venous distension (used to assess right atrial pressure)
- Cold extremities and cyanosis
- Wheezing, rales, or rhonchi on chest auscultation
- Additional heart sounds (third heart sound or gallop rhythm)

Clinical assessment

A patient presenting for dental treatment with symptoms of breathlessness, fatigue, and edema should raise the index of suspicion for the presence of cardiac failure, and prompt consultation with the patient's physician. The most useful tests in the workup of a patient with suspected heart failure are the two-dimensional echocardiogram and Doppler flow studies. An echocardiogram will quantitate the size and function of the ventricles as well as the ejection fraction, and identify pericardial effusions and valvular abnormalities. The level of brain natriuretic peptide, which is elevated in the presence of cardiac failure, may help distinguish heart failure from other causes of these nonspecific symptoms, although it does not distinguish between systolic and diastolic dysfunction [46]. Other tests include radionuclide ventriculography, which provides accurate assessment of myocardial function and wall motion. Coronary artery disease should also be excluded in every patient presenting with heart failure.

Medical treatment

It is beyond the scope of this chapter to discuss in detail the evidence for the various medical and surgical treatments for heart failure. However, the dental practitioner should be aware of the range of treatment options and the drugs used. Fig. 1 illustrates the new classification and staging system and the appropriate therapies for each stage of heart failure.

Dental treatment

Signs and symptoms consistent with heart failure should be further investigated and consultation with a physician or cardiologist should be sought. Dental treatment for patients in stage A may proceed unless hypertension or diabetes is poorly controlled. For patients in stages B, C, and D, the dentist

should consult with a cardiologist who can perform diagnostic testing and workup, review medications, evaluate for cardiac resynchronization therapy or an implantable defibrillator, and, in general, improve the patient's condition as much as possible before dental treatment.

Left ventricular ejection fraction provides a quantitative marker of systolic ventricular dysfunction. Pasternack and colleagues [53] reported that patients undergoing surgery with an ejection fraction $>55\%$ were at low risk of myocardial complications postoperatively, while those with an ejection fraction of $<35\%$ had a 75% incidence of postoperative myocardial infarction. However, others report no such correlation between ejection fraction and postoperative cardiac complications [54].

The dental management of a patient with poorly compensated heart failure may be complicated by shortness of breath and precipitation of pulmonary edema when the patient is placed in the supine position. Any history of exertional dyspnea, orthopnea, or paroxysmal nocturnal dyspnea provides a useful indicator of this and should alert the dental practitioner. Local anesthetic with epinephrine may be used, although any intravascular injection resulting in tachycardia may cause decompensation and acute pulmonary edema with sudden onset of shortness of breath. Restricting the total dose of epinephrine to a maximum of 0.04 mg (two standard cartridges with 1:100,000 epinephrine) is recommended in patients with ischemic cardiac disease. If patients in stages A, B, or C of cardiac failure have been optimized from a medical standpoint, dental treatment may proceed relatively unimpeded. Those with active symptoms and in stage D should be managed with caution in a hospital setting.

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