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The patient with a heart murmur

Evaluation, assessment and dental considerations

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A heart murmur is a prevalent finding in dental patients. It is of major concern because certain dental procedures occasionally can induce severe cardiovascular complications. Murmurs may indicate existing heart disease that is a risk factor for infective endocarditis (IE) following a dental procedure, as well as more severe heart conditions. We provide an overview of the types of heart murmurs, how they are diagnosed, how to understand the major aspects of an echocardiogram and how to use this information to provide better patient care.

Understanding the medical evaluation and assessment of a heart murmur results in improved patient care.

GENESIS AND TYPES OF HEART MURMURS

Murmurs are defined as sounds heard in addition to the sequence of two to three heart sounds during each heart-beat.¹ The two normal heart sounds—the first heart sound (S_1) and the second heart sound (S_2)—are produced mainly by the closure of the atrioventricular (tricuspid and mitral) and semilunar (aortic and pulmonary) valves, respectively. Occasionally, an additional heart sound associated with the ventricles filling up with blood may be heard. It is referred to as S_3 when it occurs in early diastole and S_4 when heard in late diastole after the atrial contraction.

Heart murmurs can be systolic, diastolic or continuous (Box 1), and they can be reported with accompanying intensity grades (Box 2). When clinically insignificant, murmurs are referred to as being “innocent”; they are caused by increased flow or turbulence across anatomically normal valves. Some systolic murmurs

Background. Heart murmurs, a common finding in dental patients, are of major concern to dental professionals because certain dental procedures occasionally can induce severe cardiovascular complications. Murmurs may indicate existing heart disease that is a risk factor for infective endocarditis following a dental procedure, as well as more severe heart conditions such as congenital heart disease, atrial fibrillation or congestive heart failure.

Types of Studies Reviewed. This review article is based on data published in peer-reviewed journals, including practice guidelines published by major dental and medical professional organizations.

Results. Echocardiography is the primary means of evaluating heart murmurs, and all dental professionals should become familiar with major aspects of an echocardiogram. Understanding the medical evaluation and assessment of a heart murmur fosters better communication with other health care professionals and results in improved patient care.

Clinical Implications. Beyond the need to administer antibiotic prophylaxis, the dentist also needs to address the underlying causes of a patient's heart murmur. By providing dental care to such patients, oral health care providers become part of the patient's overall health care team.

Key Words. Heart murmur; echocardiography; dentistry.



may be clinically innocent, while all diastolic and continuous murmurs are abnormal.²

Pathological murmurs occur as a result of either diseased cardiac valves or abnormal communications between cardiac chambers, blood vessels or both. These lesions may be congenital or acquired, and they pose varying risks of developing IE in conjunction with dental procedures (Box 3, page 349). Heart murmurs caused by cardiac

BOX 1

TYPES OF HEART MURMURS.

SYSTOLIC MURMURS

- These murmurs begin at or after the first heart sound (S₁) and end at or before the second heart sound (S₂)
- Midsystolic murmur begins after S₁ and stops before S₂
- Pansystolic or holosystolic murmur begins after S₁ and stops at S₂
- Late systolic murmur usually starts in middle or late systole and stops at S₂

DIASTOLIC MURMURS

- These murmurs begin at or after S₂ and end before the next S₁
- Early diastolic murmur begins immediately after S₂ and disappears before the next S₁
- Middiastolic murmur begins shortly after S₂ and stops before the next S₁
- Late diastolic (presystolic) murmur begins in late diastole and continues until the next S₁

CONTINUOUS MURMURS

- These murmurs begin in systole and continue without interruption through S₂ into all or part of diastole

INNOCENT MURMURS

- These murmurs are clinically insignificant
- Some systolic murmurs may be innocent, while all diastolic and continuous murmurs are abnormal

BOX 2

INTENSITY GRADES OF HEART MURMURS.*

GRADE	DESCRIPTION
1	Very faint-sounding murmur
2	Quiet murmur that can be heard immediately after placement of the stethoscope
3	Moderately loud murmur
4	Loud murmur
5	Very loud murmur that occasionally can be heard when the stethoscope is placed only partially on the chest
6	Very loud murmur that can be heard when the stethoscope is off the chest entirely

* Murmurs typically are reported as a fraction of the scale being used. For example, a grade 4 murmur on a scale of 6 is reported as ⅔.

lesions often warrant other important considerations, which need to be addressed by the treating dental professional (Table, page 350).

Congenital lesions. A full description of all congenital cardiac lesions is beyond the scope of this article. It suffices to say that congenital lesions can be cyanotic or noncyanotic, with the former group conferring a high risk of developing IE after a dental procedure (Box 3).

In cyanotic lesions, a portion of nonoxygenated blood never reaches the lungs; instead, it is shunted to the systemic circulation. Examples include transposition of great arteries, tetralogy of Fallot and single ventricle. Certain people with congenital heart disease have a characteristic body habitus, such as patients with trisomy 21

(Down syndrome). They have an increased prevalence of atrial or ventricular septal defects and tetralogy of Fallot.³

Furthermore, congenital heart disease is associated with heritable connective tissue disorders such as osteogenesis imperfecta,⁴ Ehlers-Danlos syndrome, Marfan syndrome⁵ and Stickler’s syndrome.⁶

Because patients with congenital lesions often are at a high risk of developing IE, dentists should familiarize themselves with such conditions and make sure that affected patients are given appropriate antibiotic prophylaxis before undergoing dental procedures.⁷

As shown in Box 3, some noncyanotic lesions confer a moderate risk of developing IE after dental procedures (for example, primum atrial septal defect [ASD], ventricular septal defect and patent ductus arteriosus), while others confer a low risk (for example, secundum ASD).⁸

Acquired lesions. Worldwide, the most common cause of clinically

significant heart murmurs is rheumatic heart disease (RHD). In the United States and Canada, however, RHD is an uncommon finding except among recent immigrants. The most common causes of clinically significant murmurs in this country are degenerative valvular disorders such as senile calcific aortic stenosis (AS) and mitral valve prolapse (MVP).^{9,10} Less common causes of acquired valvular disease are systemic lupus erythematosus (SLE)¹¹ and certain medications used in weight loss programs.¹²

Rheumatic heart disease. RHD is triggered by group A streptococcal pharyngitis. The microorganism induces an autoimmune reaction that may lead to valvular scarring and calcifications, especially involving the mitral valve. The most

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BOX 3

RISK OF INFECTIVE ENDOCARDITIS AND RECOMMENDATIONS FOR ANTIBIOTIC PROPHYLAXIS.**HIGH RISK: ANTIBIOTIC PROPHYLAXIS RECOMMENDED**

- Complex cyanotic lesions
 - Single ventricle
 - Transposition of great arteries
 - Tetralogy of Fallot
 - Systemic-to-pulmonary circulation shunts
- Prosthetic heart valves
- Previous infective endocarditis

MODERATE RISK: ANTIBIOTIC PROPHYLAXIS RECOMMENDED

- Certain congenital conditions
 - Mitral valve prolapse with regurgitation
 - Primum atrial septal defect
 - Ventricular septal defect
 - Patent ductus arteriosus
 - Bicuspid aortic valve
 - Coarctation of aorta
 - Hypertrophic obstructive cardiomyopathy
- Libman-Sacks nonbacterial endocarditis
- Acquired valvular disease
 - Rheumatic heart disease
 - Other acquired conditions (for example, radiation- or drug-induced valvulopathy)

LOW RISK: ANTIBIOTIC PROPHYLAXIS USUALLY NOT RECOMMENDED

- Certain congenital conditions
 - Mitral valve prolapse without regurgitation
 - Secundum atrial septal defect
 - Primum atrial septal defect, ventricular septal defect or patent ductus arteriosus more than six months after repair
- Nonvalvular heart surgery
 - Coronary bypass grafting
 - Pacemakers and defibrillators

common forms of RHD are mitral stenosis and aortic insufficiency, both of which produce diastolic murmurs.

Aortic stenosis. Owing to aging, an initially normal trileaflet aortic valve may undergo calcific degeneration. Subsequent narrowing of the aortic valve orifice results in the clinical manifestations of AS in the elderly.¹³ When calcific aortic stenosis is encountered in middle-aged people, the clinician should suspect a diagnosis of congenitally bicuspid aortic valve (BAV). BAV is the most common form of congenital heart disease, occurring in 1 to 2 percent of all live births.⁸

Mitral valve prolapse. Myxomatous degeneration of valve leaflets, typically those of the mitral valve, results in leaflet redundancy and systolic billowing of one or both leaflets into the left atrium (compare the appearance of a normal mitral valve in Figure 1 [page 351] with that of a valve affected by myxomatous degeneration leading to mitral valve prolapse in Figure 2 [page

351]). Sometimes, this is accompanied by regurgitation. Although the prognosis for MVP is good in general, it is the valvular disease that necessitates the most valve surgery in the United States. In cases in which MVP is associated with regurgitation, a murmur often will be heard preceded by a systolic click.^{14,15} Antibiotic prophylaxis is warranted for patients with MVP only when mitral regurgitation is present (Box 3).

Systemic lupus erythematosus. Verrucous valvular lesions in SLE can be found in up to 60 percent of affected patients. Libman-Sacks endocarditis in patients with SLE refers to valvular thickening, followed by vegetations (that is, thrombi adherent to a diseased valve and composed of platelets, fibrin and sometimes microorganisms) and valvular insufficiency. Initially, the damage is caused by immune complex deposition on the valve.¹⁶ Patients with SLE who have Libman-Sacks endocarditis are at moderate risk of developing IE. According to the guidelines of

TABLE

HEART MURMUR TYPES AND CONDITIONS.		
TYPE OF HEART MURMUR	ASSOCIATED VALVULAR ABNORMALITY	POSSIBLE ASSOCIATED DISEASES AND CONDITIONS
Early Systolic	MR*	Atrial fibrillation, LVH [†] , SLE [‡] , CTD [§]
	TR [¶]	Atrial fibrillation, JVD [#] , CTD
	VSD**	Atrial fibrillation, left-to-right shunt, CHF ^{††}
	AS	Atrial fibrillation, LVH
Midsystolic	AS ^{‡‡}	Atrial fibrillation, LVH
	PS ^{§§}	Right-sided CHF
Late Systolic	Mitral valve prolapse	CTD, Stickler's syndrome, trisomy 21 syndrome
Holosystolic	MR	Atrial fibrillation, LVH, SLE, CTD
	TR	Atrial fibrillation, JVD, CTD
	VSD	Atrial fibrillation, left-to-right shunt, CHF
Early Diastolic	AI ^{¶¶} or PI ^{##}	CHF
Middiastolic	Rheumatic MS ^{***}	Atrial fibrillation
Late Diastolic	Rheumatic MS	Atrial fibrillation
Continuous	Aortopulmonary and arteriovenous connections (for example, patent ductus arteriosus)	CHF, LVH
Innocent	Mammary souffle, Still's murmur, venous hum	None

* MR: Mitral regurgitation.
 † LVH: Left ventricular hypertrophy.
 ‡ SLE: Systemic lupus erythematosus.
 § CTD: Connective tissue disorders.
 ¶ TR: Tricuspid regurgitation.
 # JVD: Jugular venous distention.
 ** VSD: Ventricular septal defect.
 †† CHF: Congestive heart failure.
 ‡‡ AS: Aortic stenosis.
 §§ PS: Pulmonic stenosis.
 ¶¶ AI: Aortic insufficiency.
 ## PI: Pulmonic insufficiency.
 *** MS: Mitral stenosis.

the American College of Cardiologists/American Heart Association, these patients should receive prophylactic antibiotics.¹⁷

The prevalence of heart murmurs associated with SLE ranges from 18 to 50 percent.¹⁸ However, the exact risk of contracting IE among patients with SLE is unknown.¹¹

Diet medications. Dexfenfluramine and fenfluramine-phentermine used for weight reduction have been implicated in the development of valvulopathy and valvular regurgitation.¹⁹ According to the U.S. Food and Drug Administration criteria, valvulopathy may be considered to be due to diet medication if the patient develops at least mild aortic regurgitation, moderate

mitral regurgitation or both after initiating the diet drug therapeutic regimen.²⁰

In the largest study to date, mild or greater aortic regurgitation was present in 8.8 percent of treated patients and in 3.6 percent of control patients. Moderate or greater mitral regurgitation was present in 2.6 percent of treated patients and in 1.5 percent of control patients. The authors of that study concluded that valvulopathy developed primarily in patients who had taken fenfluramine-phentermine for more than six months, and it resulted predominantly in mild aortic regurgitation.²¹ Valvular regurgitation did not progress in the three to five months after discontinuation of dexfenfluramine therapy, at least

not in patients who had been treated with the drug for less than three months.²²

Radiation therapy. Clinicians may treat patients with heart defects that have been acquired as a result of undergoing radiation therapy to the mediastinal region for Hodgkin's and non-Hodgkin's lymphomas or cancers of the breast, lung and esophagus.

Radiation injury leads to fibrotic changes with or without calcifications of the heart valves. The pathophysiology of these changes is not well-understood. However, it is known that radiation may induce valvular regurgitation, stenosis or both, which may be progressive in nature. The prevalence of valvular fibrosis is 70 to 80 percent in patients who have received cumulative radiation doses of more than 35 gray.²³

A recent study of asymptomatic patients who had undergone mediastinal irradiation attempted to estimate the clinical significance of valvular pathology.²⁴ For patients who received radiation treatment within the 10 years preceding the study, 13 patients needed to be screened to detect one candidate with valvular lesions severe enough to require endocarditis prophylaxis. Among those who were treated at least 20 years before the study (when radiation dosages were much larger than those used currently), only 1.6 patients needed to be screened to detect one candidate with significant valvular disease. Echocardiography, therefore, is strongly recommended for patients with a history of mediastinal irradiation to screen for potential valvular heart disease.

In addition to developing valvular defects, these patients may develop pericardial effusion and thickening, and they are at an increased risk of developing premature coronary artery disease.

Endocrine conditions. Patients with active or treated acromegaly have a high prevalence of

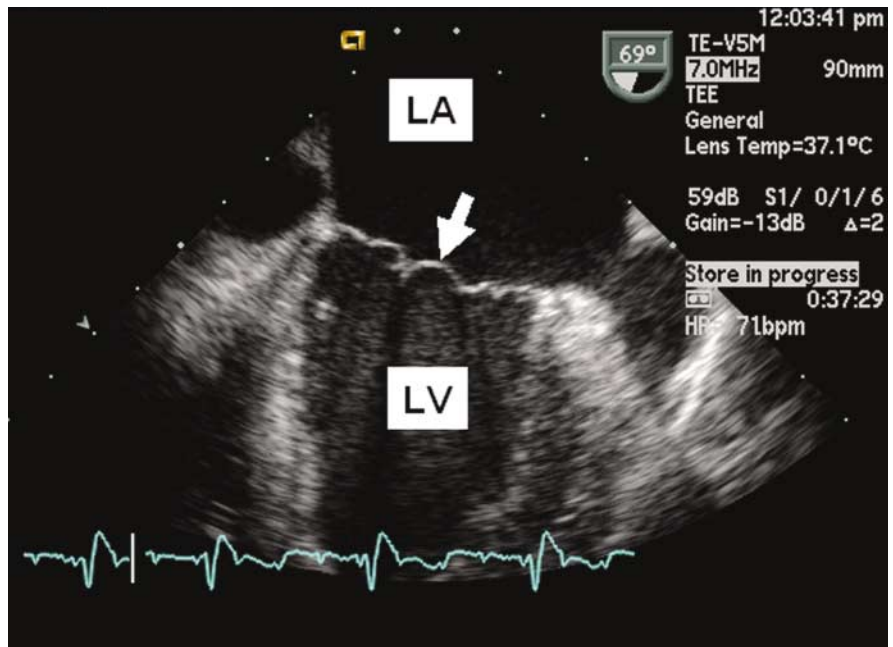


Figure 1. Mitral valve leaflets (arrow) are seen as a thin undulating membrane separating the left atrium (LA) from the left ventricle (LV) during systole.

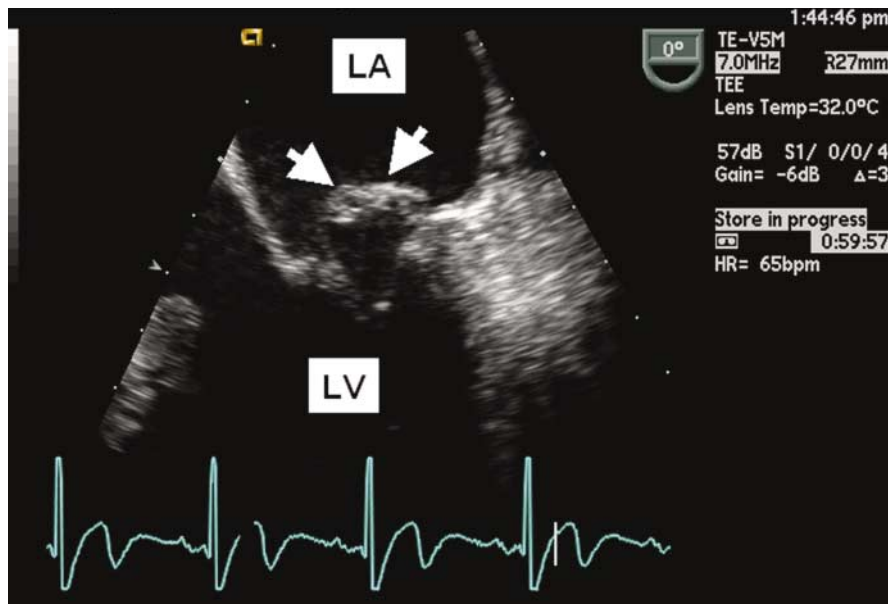


Figure 2. Mitral valve prolapse is characterized by excessive billowing of one or more segments of a mitral leaflet (arrows) into the left atrium (LA) and away from the left ventricle (LV) during systole.

mitral and aortic valve abnormalities characterized by fibrosis and/or calcifications of valve annulus and leaflets, which may lead to valvular regurgitation or stenosis.²⁵

All of the above-mentioned forms of acquired valvular disease confer a moderate risk of developing IE after dental procedures. Some forms of acquired valvular disease, however, carry a much

higher risk. These include a history of IE and valve surgery (Box 3).

DETECTION OF A HEART MURMUR

Using a stethoscope, clinicians first detect murmurs via auscultation, and they often characterize them further through ultrasound imaging of the heart (that is, echocardiography).

Auscultation. The most common abnormal auscultatory finding on cardiac examination is a systolic murmur, which occurs in 80 to 96 percent of children and in 15 to 44 percent of adults.²⁶ These murmurs can be functional (innocent) or pathological.

Attenhofer Jost and colleagues²⁶ suggested that all patients who have a systolic murmur associated with dyspnea, chest pain or lower-extremity edema should be suspected of having a pathological murmur. These cardiac signs and symptoms can be determined easily when the oral health care provider reviews systems with the patient. Dentists should refer patients with symptomatic murmurs to a cardiologist for further evaluation.

In one study, Roldan and colleagues²⁷ reported that auscultation had a sensitivity of 70 percent and a specificity of 98 percent for detecting valvular heart disease in subjects without symptoms, in comparison with echocardiography. A well-trained and experienced cardiologist can differentiate a functional (innocent) murmur from a pathological one in almost all cases.²⁸ However, according to a study by Gaskin and colleagues,²⁹ auscultation skills are suboptimal among modern medical trainees, who were able to correctly diagnose a murmur in only 33 percent of cases.

The current American College of Cardiologists/American Heart Association guidelines for the treatment of patients with valvular heart disease state that cardiac auscultation remains the most widely used technique of screening for heart disease, and that a careful observer will be able to deduce the correct origin and significance of a heart murmur.⁹ The physician should use echocardiography to confirm an impression or diagnosis of structural heart disease.

Echocardiography. Echocardiography is a diagnostic test that uses ultrasound waves to make images of the heart chambers, valves and surrounding structures. The purpose of echocar-

diography with regard to heart murmurs is to evaluate the primary lesion in terms of cause and severity, define the lesion's hemodynamic significance, detect coexisting abnormalities, evaluate the size and function of cardiac chambers and establish a reference point for future comparisons.

The standard echocardiography is called transthoracic echocardiography (TTE). In this procedure, the clinician performs cardiac imaging after placing an ultrasound probe on various points on the external chest and abdominal walls.

During transesophageal echocardiography (TEE), the heart and large vessels are imaged after a small ultrasound probe is introduced into the esophagus and stomach (Figure 1). It is considered an invasive procedure and should be used

only when information beyond that obtained from conventional TTE is needed.

Overall, TTE has a lower sensitivity (60 to 70 percent) than does TEE (75 to 95 percent) in detecting infective endocarditis, particularly that of left-sided (mitral and aortic) valves.³⁰ However, for the detection of infective endocarditis of the tricuspid

valve, TTE often is as sensitive as TEE.³¹

Both TTE and TEE are capable of M-mode, two-dimensional and Doppler imaging (Figure 3). Once echocardiographic images are acquired and stored on a videotape or computer disk, the cardiologist analyzes the examination findings and writes a report. The major components of this report are discussed below.

In view of the current literature, echocardiography is the procedure of choice for evaluating a heart murmur, but this technology should not be used as a "fishing net." Echocardiography probably is not a cost-effective method of screening asymptomatic patients for valvular heart disease. It should be reserved for dental patients with a moderate-to-severe risk of developing IE.³² When the clinician suspects IE, a positive echocardiographic finding (Figure 4) is one of several major Duke criteria for the establishment of an IE diagnosis.³³

Echocardiographic report. The echocardiographic report has many components. The normal values may vary from one medical center to another, but the various components of a standard report are similar. As with all labora-

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The most common abnormal auscultatory finding on cardiac examination is a systolic murmur.

tory reports, the initial review should make sure that the patient's name on the report is correct. The date and type of study (TTE or TEE) follow, along with general information about the patient (for example, age, sex, location of care).

The information regarding chamber sizes and wall thickness often is obtained via M-mode echocardiography. A descriptive section of the report follows the numeric data in which specific cardiac pathology (if present) is mentioned, and its hemodynamic significance is discussed based on the data from all echocardiographic imaging modalities.

One of the most important parameters of cardiac function is the left ventricular ejection function. An ejection fraction below 50 percent suggests left ventricular systolic dysfunction (that is, decreased left ventricular contractility).

As for valvular function, it is important for dentists to note that a mild degree of mitral, tricuspid and pulmonic regurgitation is present in a significant percentage of healthy patients.³⁴ This finding is considered to be physiologic, is not associated with audible murmurs and, in principle, does not require any specific medical intervention.

GENERAL MEDICAL TREATMENT OF PATIENTS WITH MURMURS

The clinician's approach to treating patients with heart murmurs depends on many variables, including the characteristics of the murmur itself (such as the intensity and type of murmur) and the presence or absence of cardiac symptoms.

For example, an extensive work-up is not necessary for patients with grade 1 and 2 midsystolic murmurs if they are children or young adults in whom the cardiac examination results are normal, and who do not have other signs and

symptoms associated with cardiac disease.⁹

However, if patients have signs and symptoms indicating the presence of IE, thromboembolism, congestive heart failure, myocardial infarction or syncope, echocardiography is indicated.

The classic symptoms of cardiac disease include chest pain or chest discomfort, dyspnea, palpitations and syncope.^{9,26} In the review of systems, clinicians must ask these key questions, the answers to which will give them clues regarding

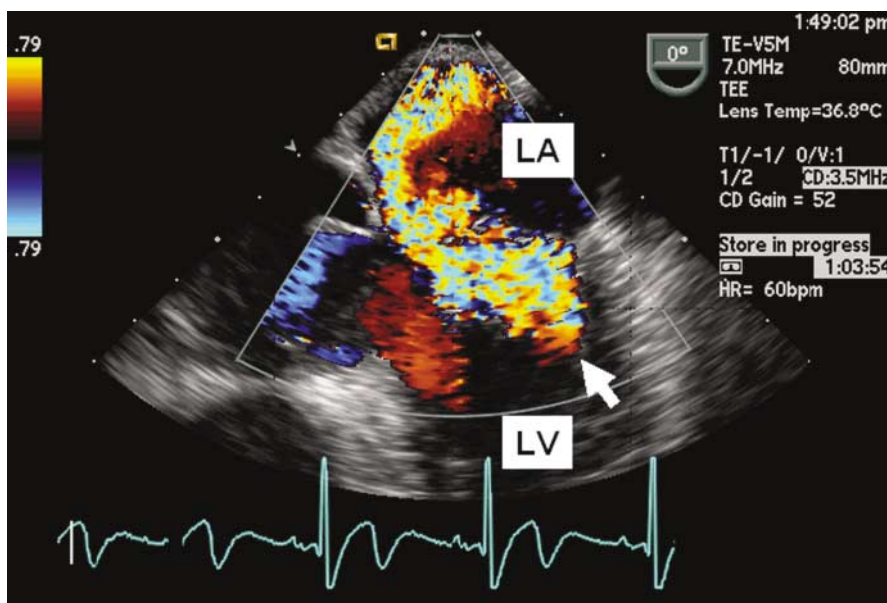


Figure 3. During transesophageal echocardiography, color Doppler imaging is used to visualize a highly mosaic jet of mitral regurgitation (arrow), which begins in the left ventricle (LV) and empties into the left atrium (LA) during systole.

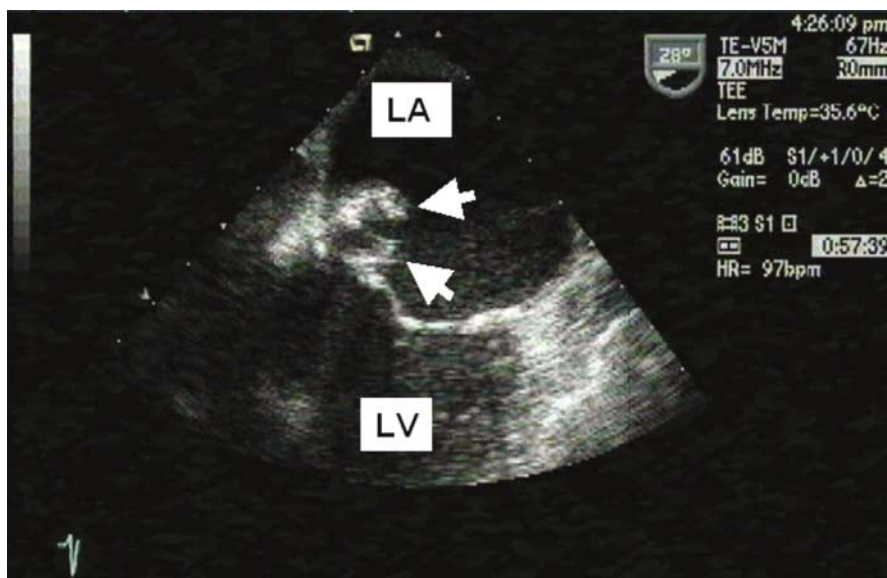


Figure 4. A large *Staphylococcus aureus* vegetation (arrows) is seen on the left atrial (LA) side of the mitral valve. LV: Left ventricle.

whether the murmur is innocent or clinically significant.

As noted above, mild regurgitation of the mitral, tricuspid or pulmonic valves frequently is detected in asymptomatic patients, often in the absence of an audible murmur on auscultation. In principle, such patients do not require routine antibiotic prophylaxis before dental and other bacteremia-inducing medical procedures.³⁴

In the work-up of a patient with a pathological heart murmur, echocardiography is mandatory to confirm the diagnosis, establish the severity of the lesion and assess ventricular function and size.

All health professionals, including dental professionals, should educate their patients who have clinically significant heart murmurs about antibiotic prophylaxis, while cardiologists and other physicians should counsel patients about functional limitation, exercise and work restrictions, and pregnancy issues.

DENTAL TREATMENT CONSIDERATIONS

When treating patients with murmurs, dental professionals should take into account the fact that many patients (particularly the elderly) are unaware of their murmurs or are unwilling to report them.³⁵ This is a growing concern because older patients, particularly men, have an increased prevalence of valvular heart disease.³⁶

A consultation with the patient's cardiologist or primary care physician should include inquiries about the type of murmur, its cause and plans for medical treatment. If echocardiography has been performed, the patient's dentist should request a copy of the report. Details regarding valvular pathology, its hemodynamic significance and the presence or absence of any associated cardiac disease usually are stated in the report's conclusions. Although the dentist is not expected to establish a cardiology diagnosis or provide medical recommendations, the information in the report broadens his or her understanding of a patient with a heart murmur.

It is beyond the scope of this article to discuss each and every condition associated with a heart murmur, and we address only the most common ones below. Readers are advised to seek additional information when treating patients with

heart conditions that are not addressed in this article.³⁷

When treating all patients with medically complex conditions, dental professionals need to address the following four concerns:

- hemostasis;
- the patient's susceptibility to developing IE;
- drug actions and interactions;
- the patient's ability to tolerate the stress and trauma associated with dental procedures.³⁸

Hemostasis. Worldwide, probably the most common cause of atrial fibrillation (AF) is rheumatic mitral valve disease. In the industrialized world, however, nonvalvular causes such as age, hypertension, diabetes mellitus and congestive heart failure are much more common. AF

may be associated with both systolic and diastolic murmurs (Table). Because the condition puts the patient at risk of developing thromboembolic events, anticoagulation with warfarin (rather than antiplatelet therapy with aspirin) is the preferred form of treatment.³⁹ An international normalized ratio (INR) of less than 3.5 or a prothrombin time (PT) of less than 20 seconds is recommended for routine

dental care.⁴⁰

The anticoagulation effect of warfarin can be increased with numerous medications used in dentistry. Although the risk of bleeding may increase after drug interactions, this might not be reflected by the INR or PT. These interactions are complex and may vary depending on the dosage, duration, intermittent versus long-term use and other factors.

Medications of concern include long-term use of acetaminophen; antibiotics such as metronidazole, erythromycin, cephalosporins, tetracycline and penicillin; and antifungal agents such as fluconazole and ketoconazole. An additive anticoagulant effect also is seen when aspirin-containing medications or nonsteroidal anti-inflammatory drugs are used simultaneously with warfarin.

Patient's susceptibility to IE. The American Heart Association has put forth recommendations regarding antibiotic prophylaxis for patients with specific heart conditions.⁷ The most common valvular conditions in a dental patient are aortic stenosis and mitral valve prolapse. Box 3 lists the specifics of antibiotic prophylaxis.

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.....

Drug actions and interactions. Apart from anticoagulation medications, digitalis glycosides (which may be used to treat patients with AF and congestive heart failure) are of particular concern because they have a narrow therapeutic range and exhibit numerous drug interactions. Antimicrobial agents used in dentistry, such as tetracycline, clarithromycin, erythromycin and ketoconazole, have been associated with increasing serum digoxin concentrations.

Tolerance to stress of dental procedure. Patients with heart murmurs may have accompanying symptoms of dyspnea, fatigue, orthopnea (labored breathing that occurs when lying supine and is relieved by sitting up), paroxysmal nocturnal dyspnea, palpitations and chest pains. Patients complaining of orthopnea need to be placed in a more upright position during dental treatment. The use of a rubber dam may be contraindicated in such patients, because it may restrict their ability to obtain an adequate volume of air. Appointments should be short and preferably in the late morning or early afternoon.

Anxiety can precipitate cardiac complications. The administration of nitrous oxide with adequate oxygen is safe and recommended for severely apprehensive patients.⁴¹ Adequate pain control also is important because endogenous catecholamine release increases with pain. The use of local anesthetic with epinephrine is not contraindicated in patients with AF. Although clinicians should evaluate each case separately, an evidence-based review of epinephrine did not indicate any contraindications for its use in patients with hypertension.⁴²

Beyond the provision of oral care, dental professionals are able to monitor certain cardiovascular conditions such as hypertension and, in collaboration with cardiologists and other medical professionals, screen for possible changes in the cardiac status of patients with heart murmurs.⁴³

CONCLUSION

Treating dental patients with heart murmurs can be challenging. Such patients may have multiple health problems that need to be addressed for dental professionals to provide safe and appropriate dental care. Dental clinicians should pay special attention to the patient's medical history and review of systems.

Dental clinicians are not expected to establish a cardiology diagnosis or provide medical recommendations to patients. Such decisions should be

left to a cardiologist or another physician. We have provided in-depth background information to broaden clinicians' understanding of heart murmurs in their patients. By delivering dental care to this patient population, oral health care providers become part of their patients' overall health care team. ■

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1. Braunwald E. Heart disease: A textbook of cardiovascular medicine. 6th ed. Philadelphia: Saunders; 2001:63.
2. Bickley LS, Hoekelman RA, Bates B. Bate's guide to physical examination and history taking. 7th ed. Philadelphia: Lippincott; 1999:277-332.
3. Marino B, Vairo U, Corno A, et al. Atrioventricular canal in Down syndrome: prevalence of associated cardiac malformations compared with patients without Down syndrome. *Am J Dis Child* 1990; 144:1120-2.
4. O'Connell AC, Marini JC. Evaluation of oral problems in an osteogenesis imperfecta population. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1999;87(2):189-96.
5. De Coster PJ, Martens LC, De Paepe A. Oral manifestation of patients with Marfan syndrome: a case-control study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2002;93:564-72.
6. Webb AC, Markus AF. The diagnosis and consequences of Stickler syndrome. *Br J Oral Maxillofac Surg* 2002;40(1):49-51.
7. Dajani AS, Taubert KA, Wilson W, et al. Prevention of bacterial endocarditis: recommendations by the American Heart Association. *JAMA* 1997;277:1794-801.
8. Brickner ME, Hillis LD, Lange RA. Congenital heart disease in adults: second of two parts. *N Engl J Med* 2000;342:334-42.
9. Bonow RO, Carabello B, de Leon AC, et al. ACC/AHA guidelines for the management of patients with valvular heart disease. Executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Patients with Valvular Heart Disease). *J Heart Valve Dis* 1998;7:672-707.
10. Shipton B, Wahba H. Valvular heart disease: review and update. *Am Fam Physician* 2001;63:2201-8.
11. Miller CS, Egan RM, Falace DA, Rayens MK, Moore CR. Prevalence of infective endocarditis in patients with systemic lupus erythematosus. *JADA* 1999;130:387-92.
12. Barasch A, Safford MM. Diet medications and valvular heart disease: the current evidence. *Spec Care Dentist* 2002;22(3):108-14.
13. Saric M, Kronzon I. Aortic stenosis in the elderly. *Am J Geriatr Cardiol* 2000;9:321-9, 345.
14. Bouknight DP, O'Rourke RA. Current management of mitral valve prolapse. *Am Fam Physician* 2000;61:3343-50, 53-4.
15. Lavie CJ. Echocardiography for mitral valve prolapse? *Postgrad Med* 1999;105(1):34, 37.
16. Moder KG, Miller TD, Tazelaar HD. Cardiac involvement in systemic lupus erythematosus. *Mayo Clin Proc* 1999;74:275-84.
17. Cheitlin MD, Alpert JS, Armstrong WF, et al. ACC/AHA Guidelines for the clinical application of echocardiography. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Clinical Application of Echocardiography). Developed in collaboration with the American Society of Echocardiography. *Circulation* 1997;95:1686-744.

18. Jensen-Urstad K, Svenungsson E, de Faire U, et al. Cardiac abnormalities are frequent in systemic lupus erythematosus patients with manifest arterial disease. *Lupus* 2002;11:744-52.
19. Connolly HM, Crary JL, McGoon MD, et al. Valvular heart disease associated with fenfluramine-phentermine [published correction appears in *N Engl J Med* 1997;337:1783]. *N Engl J Med* 1997;337:581-8.
20. Cardiac valvulopathy associated with exposure to fenfluramine or dexfenfluramine: U.S. Department of Health and Human Services interim public health recommendations, November 1997. *MMWR Morb Mortal Wkly Rep* 1997;46:1061-6.
21. Jollis JG, Landolfo CK, Kisslo J, Constantine GD, Davis KD, Ryan T. Fenfluramine and phentermine and cardiovascular findings: effect of treatment duration on prevalence of valve abnormalities. *Circulation* 2000;101:2071-7.
22. Weissman NJ, Tighe JF Jr, Gottdiener JS, Gwynne JT. Prevalence of valvular-regurgitation associated with dexfenfluramine three to five months after discontinuation of treatment. *J Am Coll Cardiol* 1999;34:2088-95.
23. Adams MJ, Hardenbergh PH, Constine LS, Lipshultz SE. Radiation-associated cardiovascular disease. *Crit Rev Oncol Hematol* 2003;45(1):55-75.
24. Heidenreich PA, Hancock SL, Lee BK, Mariscal CS, Schnittger I. Asymptomatic cardiac disease following mediastinal irradiation. *J Am Coll Cardiol* 2003;42:743-9.
25. Colao A, Spinelli L, Marzullo P, et al. High prevalence of cardiac valve disease in acromegaly: an observational, analytical, case-control study. *J Clin Endocrinol Metab* 2003;88:3196-201.
26. Attenhofer Jost CH, Turina J, Mayer K, et al. Echocardiography in the evaluation of systolic murmurs of unknown cause. *Am J Med* 2000;108:614-20.
27. Roldan CA, Shively BK, Crawford MH. Value of cardiovascular physical examination for detecting valvular heart disease in asymptomatic subjects. *Am J Cardiol* 1996;77:1327-31.
28. Bloch A, Crittin J, Jaussi A. Should functional cardiac murmurs be diagnosed by auscultation or by echocardiography? *Clin Cardiol* 2001;24:767-9.
29. Gaskin PR, Owens SE, Talner NS, Sanders SP, Li JS. Clinical auscultation skills in pediatric residents. *Pediatrics* 2000;105:1184-7.
30. Murtagh B, Frazier OH, Letsou GV. Diagnosis and management of bacterial endocarditis in 2003. *Curr Opin Cardiol* 2003;18(2):106-10.
31. San Roman JA, Vilacosta I, Zamorano JL, Almeria C, Sanchez-Harguindey L. Transesophageal echocardiography in right-sided endocarditis. *J Am Coll Cardiol* 1993;21:1226-30.
32. Kuruppu JC, Corretti M, Mackowiak P, Roghmann MC. Overuse of transthoracic echocardiography in the diagnosis of native valve endocarditis. *Arch Intern Med* 2002;162:1715-20.
33. Li JS, Sexton DJ, Mick N, et al. Proposed modifications to the Duke criteria for the diagnosis of infective endocarditis. *Clin Infect Dis* 2000;30:633-8.
34. Singh JP, Evans JC, Levy D, et al. Prevalence and clinical determinants of mitral, tricuspid, and aortic regurgitation (the Framingham Heart Study). *Am J Cardiol* 1999;83:897-902. Erratum in: *Am J Cardiol* 1999;84:1143.
35. Randall CW, Kressin NR, Garcia RI, et al. Heart murmurs: are older male dental patients aware of their existence? *JADA* 2001;132(2):171-6.
36. Freeman RV, Otto CM. Management of asymptomatic valvular aortic stenosis. *Indian Heart J* 2002;54(1):31-8.
37. Cecil RL, Goldman L, Bennett JC. Cecil textbook of medicine. 21st ed. Philadelphia: Saunders; 2000:160-378.
38. Glick M, Siegel MA, Brightman VJ. Evaluation of the dental patient: diagnosis and medical risk assessment. In: Burket LW, Greenberg MS, Glick M, eds. *Burket's oral medicine*. 10th ed. Hamilton, Ontario: B.C. Decker; 2003:27.
39. Fuster V, Ryden LE, Asinger RW, American College of Cardiology/American Heart Association/European Society of Cardiology Board, et al. ACC/AHA/ESC guidelines for the management of patients with atrial fibrillation: executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines and Policy Conferences (Committee to Develop Guidelines for the Management of Patients With Atrial Fibrillation): developed in collaboration with the North American Society of Pacing and Electrophysiology. *J Am Coll Cardiol* 2001;38:1231-66.
40. Tyler MT, Lozada-Nur F, Glick M, eds. *Clinician's guide to treatment of medically compromised dental patients*. 2nd ed. Baltimore: American Academy of Oral Medicine; 2001:4.
41. Hallonsten AL, Koch G, Schroder U. Nitrous oxide-oxygen sedation in dental care. *Community Dent Oral Epidemiol* 1983;11:347-55.
42. Bader JD, Bonito AJ, Shugars DA. A systematic review of cardiovascular effects of epinephrine on hypertensive dental patients. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2002;93:647-53.
43. Glick M. Screening for traditional risk factors for cardiovascular disease: a review for oral health care providers. *JADA* 2002;133:291-300.