

MEDICAL MANAGEMENT UPDATE

Editor: Donald Falace

Dental management of the patient with cardiac arrhythmias: An update

Nelson L. Rhodus, DMD, MPH,^a and James W. Little, DDS, MS,^b Minneapolis, Minn UNIVERSITY OF MINNESOTA

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Cardiac arrhythmias are present in a significant percentage of the population seeking dental treatment.^{1,2} Cardiac arrhythmias may be disturbances of rhythm, rate, or conduction of the heart. They may be found in healthy individuals and in those with various forms of cardiovascular disease.^{1,3-7} Some of these arrhythmias are of little concern to the patient or dentist; however, some can produce symptoms, and some can be lifethreatening, including arrhythmias that occur secondary to anxiety (eg, those associated with dental care).⁸ Therefore, patients with significant arrhythmias must be identified before undergoing dental treatment.

Cardiac arrhythmias may be found in healthy individuals, in patients taking various medications, and in patients with certain cardiovascular conditions or with other systemic diseases.^{3,9-11} The most common causes are (1) primary cardiovascular disorders, (2) pulmonary disorders (eg, embolism or hypoxia), (3) autonomic disorders, (4) systemic disorders (eg, thyroid disease), (5) drug-related side effects, and (6) electrolyte imbalances.

The purpose of this article was to review the types of cardiac arrhythmias and their treatment, the importance and identification of the more significant arrhythmias in the dental patient, and the dental management considerations for patients with a cardiac arrhythmia.

^bProfessor Emeritus, Division of Oral Medicine, University of Minnesota, Minneapolis.

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TYPES AND SIGNIFICANCE OF ARRHYTHMIAS

Arrhythmias are classified on the basis of causing ectopic beats, slowing of the heart rate, speeding of the heart rate, and arresting the heart rate. They encompass a broad spectrum, ranging from incidental arrhythmias such as premature atrial beats to lethal ones including ventricular fibrillation. Some of the more common arrhythmias include premature atrial or ventricular beats, sinus bradycardia, and sinus tachycardia.^{3,11,12}

Isolated ectopic beats

Premature atrial beats. Premature impulses arising from ectopic foci anywhere in the atrium may result in premature atrial beats. They are common in conditions associated with dysfunction of the atria such as congestive heart failure.

Premature AV beats. Premature atrioventricular (AV) beats are less common than premature atrial or premature ventricular ectopic beats. Impulses can spread toward either the atria or the ventricles. When they are present, digitalis toxicity should be suspected.

Premature ventricular beats. Premature ventricular beats are the most common form of arrhythmia, regardless of whether heart disease exists. They also are common with digitalis toxicity and hypokalemia. Late premature ventricular beats can lead to ventricular tachycardia or fibrillation in the presence of ischemia. More than 6 late premature ventricular beats per minute may be an indication of cardiac instability.

Bradycardias

Sinus bradycardia. A sinus rate of less than 60 beats per minute is defined as *bradycardia*.² Bradycardia is a normal finding in young, healthy adults and in well-conditioned athletes and also can occur secondary to medication use. Medications with parasympathetic effects

^aProfessor and Director, Division of Oral Medicine, University of Minnesota, Minneapolis.

such as digoxin and phenothiazines may slow the heart rate. A sinus bradycardia that persists in the presence of congestive heart failure, pain, or exercise and after atropine administration is considered abnormal. Sinus bradycardia is a common finding early in myocardial infarction. It also may occur in infectious diseases, myxedema, obstructive jaundice, and hypothermia.

SA heart block. Sinoatrial (SA) heart block is relatively uncommon. Most cases are caused by rheumatic heart disease, myocardial infarction, acute infection, or drug toxicity (eg, digitalis, atropine, salicylates, and quinidine). The block may occur in stages or degrees: in first-degree block, an impulse takes undue time to enter the atrium; in second-degree block 1 or more impulses fail to emerge from the SA node; and in complete block, no impulses emerge from the SA node.

AV heart block. Rheumatic fever, ischemic heart disease, myocardial infarction, hyperthyroidism, and certain drugs (eg, digitalis, propranolol, potassium, quinidine) may cause AV heart block. AV block also occurs in degrees: the first-degree block features slow impulses with increased conduction time; in seconddegree block, some impulses fail to reach the ventricles; and no impulses reach the ventricles in thirddegree, or complete, heart block. Sarcoidosis, Hodgkin's disease, myeloma, and open-heart surgery (aortic valve, ventricular septal defects) also may result in complete heart block.

Tachycardias

Sinus tachycardia. A sinus rate greater than 100 beats per minute is defined as *sinus tachycardia.* The condition occurs most often as a physiologic response to exercise, anxiety, stress, or emotions. The pharma-cologic causes of sinus tachycardia include atropine, epinephrine, nicotine, and caffeine. Pathologic causes include fever, hypoxia, infection, anemia, and hyperthyroidism.

Atrial tachycardia. In atrial tachycardia, ectopic impulses may result in atrial rates of 150 to 200 beats per minute.² Atrial tachycardia also is seen in some cases of chronic obstructive lung disease, advanced pathosis of the atria, acute myocardial infarction, pneumonia, or drug intoxications (eg, alcohol or catechols)[•] AV block with ectopic atrial tachycardia can occur and usually indicates digitalis toxicity or hypokalemia.

Atrial flutter. A rapid, regular atrial rate of 220 to 360 beats per minute is defined as *atrial flutter*, which is rare in healthy individuals and most often associated with ischemic heart disease in people over 40 years of age.² Atrial flutter also is seen as a complication in patients with mitral stenosis or cor pulmonale and after open heart surgery. It may result when patients with

atrial fibrillation have been treated with quinidine or procainamide.

Atrial fibrillation. Atrial fibrillation is a common arrhythmia characterized by an extremely rapid atrial rate of 400 to 650 beats per minute with no discrete P waves on the electrocardiogram (ECG) tracing.^{1,2} The ventricular response is irregular, because only a portion of the impulses pass through the AV node. Atrial fibrillation may be found in healthy individuals, but the condition usually is associated with rheumatic heart disease, hypertension, ischemic heart disease, or thyrotoxicosis. Patients with atrial fibrillation are susceptible to thrombus formation.

Ventricular tachycardia. Three or more ectopic ventricular beats within a minute when the heart rate is 100 or more per minute is defined as *ventricular tachycardia.* This arrhythmia almost always occurs in diseased hearts. Certain drugs such as digitalis, sympathetic amines (epinephrine), potassium, quinidine, and procainamide may induce ventricular tachycardia. On rare occasions, it may be found in young, healthy adults. Ventricular tachycardia may be unsustained or sustained or may degenerate into ventricular fibrillation.

Wolff-Parkinson-White syndrome

Three events are involved in Wolff-Parkinson-White syndrome. First, an accessory AV pathway allows the normal conduction systems to be bypassed. Second, this accessory pathway allows rapid conduction and short refractoriness, with impulses being passed rapidly from atrium to ventricle. Third, the parallel-conduction system provides a route for reentrant tachyarrhythmias. This pattern constitutes a syndrome when patients display symptoms from paroxysmal supraventricular tachycardia. Such symptoms may include fatigue, dizziness, syncope, and angina. Paroxysmal atrial fibrillation and flutter also may occur, leading to ventricular fibrillation and death.²

Cardiac arrest

Ventricular fibrillation, ventricular asystole, and agonal rhythm are the 3 types of arrhythmias associated with cardiac arrest. All are lethal. Patients with these conditions require immediate treatment for survival.

Ventricular fibrillation. Ventricular fibrillation is represented as chaotic activity on the electrocardiogram, with the ventricles contracting rapidly but ineffectively. This usually is lethal unless therapy is administered rapidly. Coronary atherosclerosis is the most common form of heart disease predisposing to ventricular fibrillation. Other causes of this arrhythmia include rheumatic heart disease, anaphylaxis, blunt cardiac trauma, mitral valve prolapse, cardiac surgery, digitalis intoxication, and cardiac catheterization. *Ventricular asystole.* In ventricular asystole, cardiac standstill occurs when no impulses are conducted to the ventricles (ie, with the ECG registering a flat line) and no muscular activity takes place. The conditions causing ventricular fibrillation also can lead to ventricular asystole.

In a survey, Bialy et al^{13} reported that 10.6% of all hospital admissions included patients who were diagnosed with cardiac arrhythmia. The prevalence of atrial fibrillation in the United States is approximately 2.2 million people.¹⁴ Little et al^{1,12} found the prevalence of cardiac arrhythmias in a large population (>10,000) of general dental patients to be 17.2%, with greater than 4% of those serious, life-threatening cardiac arrhythmias. Cardiac arrhythmias may be associated with various systemic diseases. Pathologic sinus bradycardia may be found in patients with febrile illnesses, myxedema, obstructive jaundice, increased intracranial pressure, and myocardial infarction. Pathologic sinus tachycardia may be found in patients with fever, infection, hyperthyroidism, and anemia. Atrial extrasystoles may occur in patients with congestive heart failure, coronary insufficiency, and myocardial infarction. Supraventricular tachycardias have been reported⁸ in approximately 6% of individuals with mitral valve prolapse and may be found in patients with pneumonia or acute myocardial infarction. Atrial flutter may be found in patients with ischemic heart disease and complicates 2% to 5% of myocardial infarction cases.¹⁴ Atrial fibrillation may be associated with rheumatic mitral disease, hypertension, ischemic heart disease, or thyrotoxicosis.¹¹ Ventricular extrasystoles are the most common form of rhythm disturbance found in patients with ischemic heart disease and congestive heart failure. They also occur in approximately 45% of individuals with mitral valve prolapse.¹¹ Ventricular tachycardia almost always is associated with a diseased heart and has been reported in 6% of patients with mitral valve prolapse, in addition to 28% to 46% of monitored patients with myocardial infarction.¹⁴ Ventricular fibrillation is a terminal arrhythmia unless rapid and effective therapy is given. It may be precipitated by coronary atherosclerotic heart disease, cardiomyopathy of any origin, rheumatic heart disease, blunt cardiac trauma, mitral valve prolapse, cardiac surgery, and cardiac catheterization. 3,11,15

A major problem in patients with arterial hypertension is the presence of arrhythmias, especially if they have left ventricular hypertrophy.¹⁶ A recent PubMed search found that arterial hypertension is a major cause of nonrheumatic atrial fibrillation and other supraventricular arrhythmias.¹⁶ In addition, the prevalence of ventricular arrhythmias is increased in hypertensive patients without left ventricular hypertrophy. If left ventricular hypertrophy is present, the risk for ventricular tachycardias is increased 4-fold.¹⁶

COMPLICATIONS

Arrhythmias may be asymptomatic and cause no hemodynamic changes. However, some can affect cardiac output by (1) producing insufficient forward flow because of a slow cardiac rate; (2) reducing forward flow because of insufficient diastolic filling time, with a rapid cardiac rate; or (3) decreasing flow because of poor sequence in AV activation, with direct effects on ventricular function.^{3,17} The effect of an arrhythmia often is dependent on the physical condition of the patient. For example, a young, healthy person with paroxysmal atrial tachycardia may have minimal symptoms, whereas an elderly patient with heart disease with the same arrhythmia may develop shock, congestive heart failure, or myocardial ischemia.3,11,17-19 The presence of heart failure can have a dramatic impact on the risk for cardiac arrest. The presence of New York Heart Association class III or IV heart failure and a low ejection fraction is a predictor of arrhythmic death/cardiac arrest. For every 5% reduction in left ventricular ejection fraction, the risk of cardiac arrest/ arrhythmic death increases by 15%.²⁰ There is evidence that patients with certain types of cardiac arrhythmias (eg, atrial fibrillation) are more susceptible to ischemic events in the dental office when overly stressed or given excessive amounts of a local anesthetic containing a vasoconstrictor.²¹

CLINICAL FINDINGS

Cardiac arrhythmias may be detected as a change in the rate or rhythm, or both, of the pulse. A slow pulse may indicate a type of bradycardia, and a fast pulse may indicate a tachyarrhythmia. Electrocardiographic monitoring is needed to identify the true nature of many cardiac arrhythmias.^{3,11,22} The impact of an arrhythmia on the circulation is more important than the arrhythmia itself. Symptoms that may be attributable to an arrhythmia include fatigue, dizziness, syncope, and angina. The patient may report heart palpitations occurring on a regular or irregular basis.^{3,11} Stress testing with monitoring commonly is used to assess an individual's cardiac status.^{3,11}

MEDICAL MANAGEMENT

The medical management of cardiac arrhythmias includes medications, pacemakers, surgery, or cardioversion.²³⁻²⁵ Asymptomatic arrhythmias usually require no therapy. Symptomatic arrhythmias usually are treated first with medications. The molecular targets for optimal action of the drugs involve channels in the cellular membranes through which ions (Na⁺, Ca⁺⁺, and K⁺) are diffused rapidly. Various types of arrhythmias and the degrees of severity will be affected differently with

Drug	Channels	Receptor	Pump	Adverse reactions	Drug interactions
Lidocaine (Xylocaine)	Na			Seizures, drowsiness, and coma	Cimetidine: increases plasma lidocaine concentration
Mexiletine (Mexitil)	Na			Tremor, visual blurring, dizziness, and nausea	Dilantin, phenobarbital, rifampicin: reduce half-life of mexiletine
Procainamide (Procan SR)	Na and K			Arrhythmia aggravation, lupuslike syndrome, and agranulocytosis	Cimetidine: renal clearance of procainamide reduced
Disopyramide (Norpace)	Na and K	M ₂		Congestive heart failure, urinary retention, constipation, dry mouth, and esophageal reflux	Phenytoin, rifampin, and phenobarbital increase hepatic metabolism; avoid β-blockers and calcium channel blockers
Quinidine	Na and K	α and $M^{}_2$		Syncope, hypotension, diarrhea, vomiting, tinnitus, and thrombocytopenia	Cimetidine: inhibits metabolism; increased by phenytonin, phenobarbital, and rifampin; causes digoxin toxicity
Propafenone (Rythmol)	Na	β		Abnormal taste, nausea and vomiting, dizziness	Metoprolol inhibition; increases plasma level of warfarin; and cimetidine increases plasma levels
Flecainide (Tambocor)	Na and K			Induced proarrhythmic events, decreased left ventricular function and increased pacing thresholds	Cimetidine: increases half-life, amiodarone elevates plasma level of flecainide (reduce dosage)
Bretylium (Bretylol)	K	α and β		Nausea and vomiting, increased blood pressure, hypotension, and ventricular arrhythmias	Avoid tricyclic antidepressants
Amiodarone (Cordarone)	Na, K, and Ca	α,β and M_2		Hypotension, interstitual pneumonitis, and photosensitivity	Increases plasma level of warfarin and other antiarrhythmic drugs
Digoxin			Na/K ATPase	Cardiac arrhythmias, conduction disturbances, GI disturbances, and visual disturbances	Avoid potassium-depleting diuretics and avoid or use with care with other antiarrhythmic drugs

Table I. Classifi	cation of	antiarrhythmic	drugs c	on the	basis o	of their	differential	effects or	n channels,	receptors,	and
transmembrane	pumps ²⁶										

GI, Gastrointestinal; ATPase, adenosine triphosphatase.

different medications.^{26,27} Those who do not respond to medications may then be treated with an implanted pacemaker or cardioversion.^{17,28} If both medications and pacing fail to control the arrhythmia, surgical intervention may be attempted. Cardioversion is indicated for any tachyarrhythmias that compromise hemodynamics or life, or both. Cardiac arrest also is treated by cardioversion.^{3,7,28}

Antiarrhythmic drugs

With many antiarrhythmic drugs, the toxic/therapeutic ratio is very narrow; therefore, the dosage for a given patient must be individualized. Measurement of the plasma level of the medication is often an important part of therapy. Antiarrhythmic drugs were originally classified in terms of their electrophysiologic effects.²⁶ Class I ("local anesthetic" or "membrane-stabilizing" activity) drugs included quinidine, procainamide, disopyramide, lidocaine, mexiletine, tocainide, flecainide, and propafenone. Class II (β-adrenergic antagonism) drugs included propranolol, timolol, and metoprolol. Class III (prolong the duration of cardiac action potential and refractoriness) drugs included amiodarone, sotalol, bretylium, and ibutilide. Class IV action (calcium channel antagonism) drugs included veramil, bepridil, diltiazem, and nifedipine. A newer classification referred to as "The Sicilian Gambit" is based on the differential effects of antiarrhythmic drugs on channels, receptors, and transmembrane pumps.²⁶ Table I lists selective antiarrhythmic drugs and their side effects and drug interactions as used in the Sicilian Gambit classification system. Patients with arrhythmias medicated with digitalis are susceptible to digitalis toxicity, especially if they are elderly or have hypothyroidism, renal dysfunction, dehydration, hypokalemia, hypomagnesemia, or hypocalcemia.²⁹ Patients with atrial fibrillation often are prescribed warfarin sodium (Coumadin) to prevent atrial thrombosis.¹¹

Device			
location	Little or no risk	Potential risk	Risk
Home	CB radios, electric drills, electric blankets, electric shavers, heating pads, microwave ovens, TV transmitters, and remote-control TV changers	Patients with older pacemakers may be at risk with microwave ovens and high-powered TV sets.	_
Dental	Diagnostic radiation, dental drill, motor for chair, amalgamators, electric pulp testers, curing lights, and electric toothbrushes	Patients with older pacemakers may be at risk with pulp testers and dental motors.	Electrosurgery units, ultrasonic cleaners, and ultrasonic scaling devices
Medical	Diagnostic radiation, and electroconvulsive therapy	Extracorporeal shock-wave lithotripsy	Magnetic resonance imaging, therapeutic radiation, radiofrequency ablation, and transcutaneous electrical nerve stimulation
Others	-	Cellular telephones, antitheft systems, and metal detectors	Arc welders and radar transmitters

*All patients with a pacemaker should have identification that shows others that they have a pacemaker.

[†]The patient should always inform their physician or dentist before any testing or treatment with medical or electrical devices.

[‡]The dentist should consult with the patient's physician to determine which, if any, electrical devices may interfere with the function of the patient's pacemaker. *CB*, Citizens band; *TV*, television.

Pacemakers

More than 1 million persons in the United States have permanent pacemakers.^{17,30} In this country, approximately 115,000 pacemakers are inserted each year.³⁰ Pacemakers are useful in the management of several conduction system abnormalities including symptomatic sinus bradycardia, symptomatic AV block, and tachyarrhythmias refractory to drug therapy. The most common pacing system in use today is the demand ventricular pacemaker with a lithium-powered generator and transvenous leads.^{17,32-35} The newer units contain pacing circuits that allow for programming, memory, and telemetry.^{17,31,36} Some side effects can result from pacemakers. Infection at the generator site and thrombosis of the leads or electrodes are uncommon, but they can occur. Infective endocarditis may also occur but is rare. The American Heart Association (AHA) does not recommend antibiotic prophylaxis for patients with a cardiac pacemaker.³⁷

Skeletal muscle may be stimulated if insulation is lost around the lead or if the generator rotates. In rare cases, myocardial burning can occur. Some patients become depressed, and suicide attempts have been reported.^{17,32}

Electromagnetic interference from noncardiac electrical signals may temporarily interfere with the function of a pacemaker³³ (Table II). This occurs by a mimicking of the frequency of spontaneous heartbeats, which causes inappropriate pacemaker inhibition. Examples would be transmission from radar transmitters or arc welders. Other forms of electrical signals can potentially cause revision of the pacemaker mode to a fixed rate of transmission. These would include microwave ovens, diathermy and electrocautery units, and direct-contact pulse generators in boat or automobile motors. The newer pacemakers are better shielded to protect against electromagnetic interferences.¹⁷

Implantable cardioverter-defibrillator

Certain patients with ventricular fibrillation or unstable ventricular tachycardias are candidates for an automatic implantable cardioverter-defibrillator (AICD). The AICD is a self-contained diagnostic-therapeutic system that monitors the heart, and, when it detects fibrillation or tachycardia of the ventricle, it sends a correcting electric shock to restore normal rhythm.³⁴⁻³⁶ By 1996, more than 100,000 patients worldwide had had an AICD surgically implanted. The AICD is 99% reliable in detecting ventricular fibrillation and 98% reliable in detecting ventricular tachycardias. Its conversion effectiveness is excellent. Usually one 25-J discharge converts the arrhythmia.¹⁸ The AHA does not recommend antibiotic prophylaxis for patients with either a cardiac pacemaker or an AICD.³⁷

Cardioversion/defribrillation

Direct-current cardioversion to convert atrial and ventricular arrhythmias was first described in 1962.² Cardioversion can be effective for the treatment of reentrant arrhythmias such as atrial flutter, atrial fibrillation, ventricular tachycardia, and ventricular fibrillation. Tachyarrhythmias that result in hemodynamic collapse, prolonged angina pectoris, or pulmonary edema should be treated promptly with direct-current cardioversion. The countershock simultaneously depolarizes the entire myocardium, allowing synchronous repolarization and the resumption of sinus rhythm.^{15,21}

A defibrillator is an electrical device that sends a pulse of current through the heart to arrest several types of arrhythmias. The pulse is applied to electrodes placed on the thorax. One electrode is placed on the left chest over the region of the apex and the other on the right side of the chest just to the right of the sternum and below the clavicle. Usually, a damped sine wave defibrillator is used that can store 400 J of energy and deliver approximately 350 J into a 50- Ω resistor. Either multiple low-energy shocks (2 J/kg) or a single highenergy first shock (4 J/kg) is used. A dose concept should be developed so that the strength of the shock required is related to the size of the subject's heart. The practice of turning the output control to maximum and delivering a full jolt to all adults can be dangerous. Defibrillation usually is instantaneous, and cardiac pumping resumes within a few seconds. It may have to be repeated if defibrillation is unsuccessful (ie, regular heartbeat does not occur). Cardiopulmonary resuscitation must be used until defibrillation has been successful. When defibrillation is attempted, all rescue personnel-except the individual holding the electrodesmust stand clear of the patient.^{15,22}

Automated external defribrillator

Currently, there are several types of automated external defribrillator (AED) devices that can be purchased for use in the dental office. Some of these AED devices include the Heartstream FR-2 AED, the Laerdal AED, the Medtronic Physio-control AED, and the Survivalink AED. The use of AEDs is now being taught in cardiopulmonary resuscitation courses and is encouraged for public use by the AHA. AEDs may be used in the emergency management of sudden cardiac arrest, in addition to with arrhythmias such as atrial flutter, atrial fibrillation, ventricular tachycardia, and ventricular fibrillation. These devices are simple, easy to use, and inexpensive and may be very helpful in the management of serious cardiac arrhythmias in the dental office.³⁸

DENTAL MANAGEMENT

Medical considerations

Stress associated with dental treatment or excessive amounts of injected epinephrine may produce lifethreatening cardiac arrhythmias in susceptible dental patients.²¹ Patients may have their arrhythmia under control through the use of drugs or a pacemaker but may require special consideration when receiving dental treatment because of the potential increased risks for myocardial infarction, heart failure, and death.³⁹ Table III lists some clinical predictors of increased perioperative cardiovascular risk factors for myocardial infarction, heart failure, or death. Note that certain arrhythmias are included as major risk factors. Patients with
 Table III. Clinical predictors of increased perioperative

cardiovascular risk (eg, MI, heart failure, and death)*
Major
Unstable coronary syndromes
Acute or recent MI with evidence of important ischemic risk by
clinical symptoms or noninvasive study
Unstable or severe angina
Decompensated heart failure
Significant arrhythmias
High-grade atrioventricular block
Symptomatic (ventricular) arrhythmias in the presence of
underlying heart disease
Supraventricular arrhythmias with uncontrolled ventricular rate
Severe valvular disease
Intermediate
Mild angina pectoris
Previous MI by history or pathologic Q waves
Compensated or prior heart failure
Diabetes mellitus (particularly type 1)
Renal insufficiency
Minor
Advanced age
Abnormal ECG (eg, with left ventricular hypertrophy, left bundle-
branch block, or ST-T abnormalities)
Rhythm other than sinus (eg, atrial fibrillation)
Low functional capacity (eg, inability to climb 1 flight of stairs
with a bag of groceries)
History of stroke
Uncontrolled systemic hypertension

*From Circulation 2002;105:10 (with permission of the American Heart Association).

MI, Myocardial infarction; ECG, electrocardiogram.

these high-risk arrhythmias may not be candidates for elective dental care. The keys to the dental management of patients susceptible to developing a cardiac arrhythmia and those with an existing arrhythmia are in identification and prevention.

Prevention of medical complications

The identification of patients with an existing arrhythmia and those susceptible to developing an arrhythmia is most important. Specific arrhythmias may dictate specific management, but the general principles that the dentist should follow are outlined here. The dentist must obtain and evaluate a patient's medical history and obtain their vital signs. The presence of signs and/or symptoms of a cardiac arrhythmia may mandate further evaluation by a physician before dental treatment is performed. Patients who have an irregular cardiac rhythm with or without symptoms should be referred for a medical evaluation. Elderly patients with a regular heart rate that varies in intensity with respiration should be referred for an evaluation of possible sinus arrhythmias and sinus node disease.^{11,17} Patients with a history of significant heart disease, thyroid disease, or chronic pulmonary disease must be identified and their medical status determined. If their status is uncertain, a medical

Table IV. Estimated energy requirements for various activities*[†]

*Perioperative cardiac and long-term risks are increased in patients unable to meet a 4 MET level demand during most normal daily activities. [†]From Circulation 2002;105:10 (with permission of the AHA American Heart Association).

consultation should be obtained to accurately determine their current status and risk for developing a cardiac arrhythmia. In addition, patients taking antiarrhythmic medications and those with a pacemaker must be identified through their medical history and, again, their current status confirmed.

The severity of a given arrhythmia may depend on the health of the patient; the patient's age; and the presence of conditions such as severe hypertension, recent myocardial infarction, unstable angina, untreated hyperthyroidism, or congestive heart failure.¹² Several studies^{1,2,40} have documented the benefit of the use of a 3-lead ECG unit to screen dental patients for arrhythmias.

In 2002, the American College of Cardiology and the AHA published new guidelines for perioperative cardiovascular evaluation for patients undergoing noncardiac surgery.³⁹ These guidelines are intended for physicians who are involved in the preoperative, operative, and postoperative care of patients undergoing noncardiac surgery. Nonetheless, the guidelines can be useful to the dentist in the evaluation of the risk of cardiac arrhythmia, myocardial infarction, heart failure, and death occurring in patients being evaluated for dental procedures. Tables III, IV, and V can be used by the dentist to help determine the risks of patients with various types of cardiovascular disease developing serious complications during or after dental treatment. Table III stratifies medical conditions on the basis of risk. It should be noted that major risk conditions include certain arrhythmias such as symptomatic ventricular arrhythmias in patients with underlying heart disease,

Table V. Cardiac risk (combined incidence of cardiac death and nonfatal myocardial infarction) stratification for noncardiac surgical procedures^{$*^{\dagger}$}

High (ie, reported cardiac risk often greater than 5%)
Emergent major operations, particularly in the elderly
Aortic and other major vascular surgery
Peripheral vascular surgery
Anticipated prolonged surgical procedures associated with large
fluid shifts or blood loss, or both
Intermediate (ie, reported cardiac risk generally less than 5%)
Carotid endarterectomy
Head and neck surgery
Intraperitoneal and intrathoracic surgery
Orthopedic surgery
Prostate surgery
Low (ie, reported cardiac risk generally less than 1%)
Endoscopic procedures
Superficial procedures
Cataract surgery
Breast surgery

*Surgery-specific cardiac risk of noncardiac surgery is related to 2 important factors: the type of surgery itself and the degree of hemodynamic stress associated with the procedure.

[†]From Circulation 2002;105:10 (with permission of the American Heart Association).

high-grade atrioventricular block, and supraventricular arrhythmias with an uncontrolled ventricular rate.

A patient's ability to perform various physical activities also can be used to evaluate the risk for noncardiac surgery.³⁹ Table IV lists activities under the heading of metabolic equivalent (MET) levels. Patients with an MET level of 1 to 4 are at greatest risk; those with an MET level of 4 to 10 are at less risk; and those with an MET level of greater than 10 are at the least risk. The risk includes development of arrhythmias, myocardial infarction, congestive heart failure, and death. From the dentist's viewpoint, a patient who can't climb a flight of stairs or walk up a hill (a less than 4 MET level) may be at increased risk of adverse cardiovascular events during certain dental procedures and a medical consultation should be performed before starting any dental treatment. The cardiac risks for various noncardiac surgical procedures³⁹ are listed in Table V under the headings of high risk (ie, risk of arrhythmia, myocardial infarction, heart failure or death greater than 5%), intermediate risk (ie, cardiac risk generally less than 5%), and low risk (ie, cardiac risk less than 1%). Most dental treatment (not shown) would be included in the lowrisk group with a cardiac risk less than 1%, but some oral surgery or periodontal surgery may fall into the intermediate-risk category. The dentist can use the appropriate information from Tables III, IV, and V to serve as the basis for medical consultation and to help establish risk assessment for patients with arrhythmias.

The dentist can prevent many cardiac arrhythmiarelated medical emergencies by being aware of who are

Table VI. Dental management of the patient at risk for a cardiac arrhythmia

Medical consultation

- 1. Refer for diagnosis any patient with signs and symptoms suggestive of arrhythmia
- 2. Establish the type and severity of the arrhythmia
- 3. Establish current status for patient with arrhythmia
- 4. For patients with pacemakers, determine
 - Type of pacemaker being used
 - Type of arrhythmia being managed
 - Degree of shielding provided for the generator
 - Types of electrical equipment that should be avoided
- 5. For patients with atrial fibrillation, determine whether they are being treated with Coumadin and
 - Determine whether Coumadin is used to prevent atrial thrombosis
 - Determine INR if surgery is planned*
 - Determine whether dosage of Coumadin needs to be adjusted before surgery
- 6. Establish presence and current status of any condition that may cause arrhythmia
- Patient management
- 1. Reduce anxiety as much as possible
 - Prescribe premedication—a benzodiazepine Open and honest communication with the patient Short morning or early afternoon appointments Nitrous oxide–oxygen inhalation if needed IV sedation if needed—a benzodiazepine
- 2. Avoid excessive amounts of epinephrine
 - Use 1:100,000 epinephrine in the local anesthetic Use anesthetic without epinephrine in patients with severe arrhythmias, if treatment is necessary
 - High-grade atrioventricular block
 - Symptomatic (ventricular) arrhythmias in presence of underlying heart disease
 - Supraventricular arrhythmias with uncontrolled ventricular rate
 - In general, use no more than 3 cartridges of anesthetic Aspirate before injecting the local anesthetic Do not use epinephrine in gingival packing
 - Do not use epinephrine to control local bleeding
- 3. Manage underlying condition such as rheumatic heart disease (antibiotic prophylaxis to prevent BE)
- 4. Avoid use of general anesthesia in most dental practices
- 5. In general, avoid the use of the following devices:
 - Electrosurgery units Ultrasonic bath cleaners
 - Ultrasonic scalers
- 6. Be prepared to deal with life-threatening arrhythmia by Stopping the procedure
 - Evaluating vital signs—blood pressure, pulse rate and rhythm, and mental alertness
 - Calling for medial assistance if indicated
 - Administering oxygen
 - Placing patient in Trendelenburg's position to reduce effects of hypotension
 - Performing vagal maneuver (carotid massage) if hypotension and tachycardia are present
 - If indicated, initiating cardiopulmonary resuscitation
 - Using an automated external defribrillator if no pulse can be established

the high-risk patients and by taking appropriate precautions during dental treatment. These precautions include the following (Table VI):

- 1. Management considerations of coexisting conditions: Patients with underlying cardiac disease must be managed as indicated by the nature of the underlying cardiac problem (eg, those susceptible to endocarditis, ischemic heart disease, congestive heart failure, hypertrophic cardiomyopathy, and so on).⁴¹⁻⁴⁵
- 2. Reduce patient anxiety: Any increase in sympathetic tone can precipitate an arrhythmia.³⁹ Premedication with a short-acting benzodiazepine (ie, triazolam [0.25-0.5 mg] or oxazepam [Serax] 5 mg) the night before the appointment or 1 hour before the appointment, or both, may be helpful to reduce anxiety. Nitrous oxide–oxygen inhalation sedation can also be used during dental treatment. An open, honest approach with the patient (ie, explaining what will happen) is most important.
- 3. Minimize stressful situations: Patients with significant arrhythmias, coronary atherosclerotic heart disease, ischemic heart disease, or congestive heart failure should be managed with short morning appointments; furthermore, the session should be terminated if the patient becomes tired, to prevent or minimize acute exacerbation of conditions that might trigger significant arrhythmias.^{44,45} Complex dental procedures can be performed at several appointments to reduce the stress to the patient.
- 4. Avoid excessive amounts of vasoconstrictive agent and local anesthetic considerations: Excessive amounts of epinephrine can trigger arrhythmia or another adverse cardiovascular event.^{12,44,45} At the same time, however, vasoconstrictors in appropriate concentration in the local anesthetic are beneficial. The need to achieve profound local anesthia and hemostasis far outweighs the very slight risk of the use of these agents in small amounts (eg, 1:100,000 epinephrine). However, the use of more than 3 cartridges (5.4 mL) of anesthetic (which contains 0.06 mg of epinephrine) is not advised for any given appointment attributable to increasing risks.^{21,41}

In patients with severe arrhythmias (such as premature ventricular contractions or ventricular tachycardia) and in some patients with atrial fibrillation, recent myocardial infarction, unstable angina, or untreated hyperthyroidism, a local anesthetic without a vasoconstrictor should be used to prevent the vasoconstrictor from precipitating serious complications or cardiac arrest.^{21,41} Vasoconstrictors should not be used in gingival packing material for crown impressions or to control local bleeding. In addition, caution should be exercised when local anesthetics containing vasocon-

^{*}Most dental surgeries can be performed if INR is 3.5 or less. *INR*, International normalized ratio; *IV*, intravenous; *BE*, bacterial endocarditis.

strictors are used in patients taking digitalis because of an increased risk of precipitating an arrhythmia.⁴⁶

- 5. Avoid general anesthesia: Patients at risk for developing significant cardiac arrhythmias and those with significant arrhythmias should not be given general anesthesia in the dental office because of the increased risk of myocardial infarction, congestive heart failure, or death.^{12,44,45}
- 6. Use caution around electrical equipment: During the medical consultation for patients with a pacemaker, the risk for electromagnetic interference from electrical equipment used in the dental office should be discussed. The type of pacemaker should be identified and its susceptibility to electromagnetic interference determined. Miller et al47 discovered that the only devices causing significant electromagnetic interference with pacemakers in the dental office were electrosurgery units, ultrasonic bath cleaners, and ultrasonic scaling devices (Table II). Amalgamators, electric pulp testers, curing lights, handpieces, electric toothbrushes, microwave ovens, x-ray units, and sonic scalers did not cause any significant electromagnetic interference with pacemakers in the dental office. Internal shielding has been increased on the newer generators to minimize the adverse effects of electromagnetic interference.

Patients with new, well-shielded generators are at low risk for electromagnetic interference. However, patients with older pacemakers with poor shielding may be at higher risk for complications in pacing because of electromagnetic interference. Studies have identified certain types of equipment that may be safe, but pulp testers, motorized dental chairs, and belt-driven handpieces all may be capable of causing pacemaker malfunction in a patient with poor shielding in the pacemaker generator. Electrosurgery units, ultrasonic bath cleaners, and ultrasonic scaling devices can be of risk to all patients with pacemakers, and their use in or near these patients is contraindicated.¹²

7. Recognize anticoagulant therapy: Patients with certain arrhythmias (eg, atrial fibrillation) may be receiving anticoagulant therapy, and therefore the International Normalized Ratio (INR) level must be determined before performing surgical procedures. If the INR is 3.5 or less, most dental treatments (including minor oral surgery) can be safely performed.⁹ The dentist should consult with the patient's physician to determine the level of anticoagulation and the need to reduce the warfarin dosage before dental treatment. If the INR is greater than 3.5, the physician should consider reducing the warfarin dosage. If the warfarin dosage is reduced, it will take 3 to 4 days for the INR reduction to occur. On the day of surgery, the level of anticoagulation

must be determined. If it is within the desired range, then the surgery can be performed. If excessive bleeding should occur, it can usually be controlled by using local measures such as pressure packs, Gelfoam/thrombin (Pharmacia, New York, NY), Oxycel (Parke-Davis, New York, NY), Surgicel (Johnson and Johnson, New Brunswick, NJ), microfibrillar collagen, or tranexamic acid.⁹

8. Digitalis intoxication: Medical consultation to determine the digitalis dosage prescribed by the physician, as well as the potential for toxicity or adverse drug reactions, is recommended. Therapeutic doses of digitalis range from 0.5 to 2.0 ng/mL. Levels greater than 2.5 ng/mL may result in digitalis toxicity. Patients with arrhythmias medicated with digitalis may be susceptible to digitalis toxicity if they are elderly or have hypothyroidism, renal dysfunction, dehydration, hypokalemia, hypomagnesemia, or hypocalcemia. Patients with electrolyte imbalances are more susceptible to digitalis toxicity because of the heightened sensitivity of the heart to these changes accompanying certain arrhythmias. The dentist should avoid using erythromycin because it can increase the absorption of digitalis by altering the intestinal flora and it can lead to toxicity.46

Patients should be assessed in light of the signs and symptoms of digitalis toxicity, which are found in 3 systems: gastrointestinal (eg, anorexia, excessive salivation, nausea, vomiting, and diarrhea), neurologic (eg, headache, visual disturbances, fatigue, and drowsiness), and cardiovascular (eg, AV block, excessive slowing of the heart, ventricular extrasystoles, and other arrhythmias).^{29,46}

9. Endocarditis prevention: A few cases of endocarditis involving the cardiac leads of pacemakers and defibrillators have been reported. However, the risk of such infection resulting from dental bacteremias is remote. The AHA does not recommend antibiotic prophylaxis to prevent endocarditis for patients with implanted pacemakers or defibrillators, or both, when invasive dental procedures are performed.³⁷

REFERENCES

- 1. Little JW, Simmons MS, Kunik RL, Rhodus NL, Merry JW. Evaluation of an EKG system for the dental office. Gen Dent 1990;38:278-81.
- Little JW, Simmons MS, Rhodus NL, Merry JW, Kunik RL. Dental patient reaction to electrocardiogram screening. Oral Surg Oral Med Oral Pathol 1990;70:433-9.
- Gallagher JJ. Cardiac arrhythmias. In: Wyngaardin JB, Smith LH, editors. Cecil textbook of medicine. 20th ed. Philadelphia: W. B. Saunders; 1996.
- Furberg CD, Psaty BM, Manolio TA, Gardin JM, Smith VE, Rautaharju PM. Prevalence of atrial fibrillation in elderly subjects (the Cardiovascular Health Study). Am J Cardiol 1994;74:236-41.
- Ansari N, Manis T, Feinfeld DA. Symptomatic atrial arrhythmias in hemodialysis patients. Ren Fail 2001;23:71-6.
- 6. Reinelt P, Karth GD, Geppert A, Heinz G. Incidence and type of

cardiac arrhythmias in critically ill patients: a single center experience in a medical-cardiological ICU. Intensive Care Med 2001;27:1466-73.

- Fishberger S. Management of ventricular arrhythmias in adults with congenital heart disease. Curr Cardiol Rep 2002;4:76-80.
- Luck JC, Engel TR. Cardiac arrhythmias. In: Rose LF, Kaye D, editors. Internal medicine for dentistry. 2nd ed. St Louis: Mosby; 1990.
- Little JW, Falace DA, Miller CS, Rhodus NL. Bleeding disorders. In: Little JW, Falace DA, Miller CS, editors. Dental management of the medically compromised patient. 6th ed. St. Louis: Mosby; 2002. p. 362-95.
- Ghuran AV, Camm AJ. Ischaemic heart disease presenting as arrhythmias. Br Med Bull 2001;59:193-210.
- Myerberg RJ, Kessler KM, Castellanos A. Recognition, Clinical assessment and management of arrhythmias and conduction disturbances. In: Alexander RE, Schlant RC, Fuster V, editors. Hurst's the heart, arteries, and veins. 9th ed. New York: McGraw-Hill; 1998. p. 873-942.
- Little JW, Falace DA, Miller CS, Rhodus NL. Cardiac arrhythmias. In: Little JW, Falace DA, Miller CS, editors. Dental management of the medically compromised patient. 6th ed. St Louis: Mosby, Inc.; 2002. p. 94-114.
- Bialy D, Lehmann DN, Steinman RT, et al. Hospitalization for arrhythmias in the United States: Importance of atrial fibrillation. J Am Coll Cardiol 1992;19:716-4.
- Feinberg WM, Blackshear JL, Laupacis A, Kronmal R, Hart RG. Prevalence, age distribution, and gender of patients with atrial fibrillation. Analysis and implications. Arch Intern Med 1995;155: 469-73.
- Maron BJ. Sudden Death in Hypertrophic Cardiomyopathy. 14th ed: McGraw-Hill, Harrison's online update; 2002.
- Hennersdorf MG, Strauer BE. Arterial hypertension and cardiac arrhythmias. J Hypertens 2001;19:167-77.
- Mitrani RD, Myeburg RJ, Castellanos A. Cardiac pacemakers. In: Hurst JW, editor. The heart, arteries, and veins. 9th ed. New York: McGraw-Hill; 1998. p. 1023-59.
- O'Callaghan PA, Ruskin JN. The implantable cardioverter. In: Hurst JW, editor. The heart, arteries, and veins. 9th ed. New York: McGraw-Hill; 1998. p. 1007-23.
- Stevenson WG, Ellison KE, Sweeney MO, Epstein LM, Maisel WH. Management of arrhythmias in heart failure. Cardiol Rev 2002;10:8-14.
- Braunwald E. Update to Chapter 230. The tachyarrhythmias predictors of arrhythmic death. 14th ed. Harrison's online; 2001.
- Campbell JH, Huizinga PJ, Das SK, Rodriguez JP, Gobetti JP. Incidence and significance of cardiac arrhythmia in geriatric oral surgery patients. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;82:42-6.
- Akhtar M. Techniques of electrophysiologic testing. In: Alexander RE, Schlant RC, Fuster V, editors. Hurst's the heart, arteries, and veins. 9th ed. New York: McGraw-Hill; 1998. p. 955-67.
- Wellens HJ. Future of device therapy for arrhythmias. J Cardiovasc Electrophysiol 2002;13:122-4.
- Members of the Sicilian Gambit. New approaches to antiarrhythmic therapy, Part I: emerging therapeutic applications of the cell biology of cardiac arrhythmias. Circulation 2001;104:2865-73.
- 25. New use for pacemakers and defibrillators. Harv Heart Lett 2002;13:3-4.
- Woosley RL. Antiarrhythmic drugs. In: Alexander RW, Schlant RC, Fuster V, O'Rourke RA, Roberts R, Sonnenblick EH, editors. Hurst's the heart, arteries, and veins. 9th ed. New York: McGraw-Hill; 1998. p. 969-94.
- 27. Roden DM. Pharmacogenetics and drug-induced arrhythmias. Cardiovasc Res 2001;50:224-31.
- Lown B, DeSilva RA. External cardioversion and defibrillation. In: Alexander RE, Schlant RC, Fuster V, editors. Hurst's the heart, arteries, and veins. 9th ed. New York: McGraw-Hill; 1998. p. 1003-7.
- 29. Tasota FJ, Tate J. Assessing digoxin levels. Nursing 2000;30:24-6.
- Stewart KJ. Cardiac pacemakers and physical activity. J Am Med Assoc 1999;283:1267-9.

- Wood MA, Ellenbogen KA. Cardiology patient pages. Cardiac pacemakers from the patient's perspective. Circulation 2002;105: 2136-8.
- Dubernet J, Chamorro G, Gonzalez J, Fajuri A, Jalil J, Casanegra P, et al. [A 36 years experience with implantable pacemakers. A historical analysis]. Rev Med Chil 2002;130:132-42.
- American Heart Association. Pacemakers (patient education brochure). 1999.
- 34. Defibrillator/monitor/pacemakers. Health Devices 2002;31:45-64.
- 35. Hauer RN, Aliot E, Block M, Capucci A, Luderitz B, Santini M, et al. Indications for implantable cardioverter defibrillator (ICD) therapy. Study Group on Guidelines on ICDs of the Working Group on Arrhythmias and the Working Group on Cardiac Pacing of the European Society of Cardiology. Eur Heart J 2001;22:1074-81.
- Kusumoto FM, Goldschlager N. Device therapy for cardiac arrhythmias. JAMA 2002;287:1848-52.
- Dajani AS, Taubert KA, Wilson W, Bolger AF, Bayer A, Ferrieri P, et al. Prevention of bacterial endocarditis: recommendations by the American Heart Association. JAMA 1997;277:1794-801.
- Alexander RE. The automated external cardiac defibrillator: lifesaving device for medical emergencies. J Am Dent Assoc 1999; 130:837-45. Erratum in: J Am Dent Assoc 1999;130:1162.
- 39. Eagle KA, Berger PB, Calkins H, Chaitman BR, Ewy GA, Fleischmann KE, et al. ACC/AHA guideline update for perioperative cardiovascular evaluation for noncardiac surgery—executive summary a report of the American College of Cardiology/ American Heart Association Task Force on Practice Guidelines (Committee to Update the 1996 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery). Circulation 2002;105:1257-67.
- Simmons MS, Little JW, Rhodus NL, Verrusio AC, Kunik RL, Merry JW. Screening dentists for risk factors associated with cardiovascular disease. Gen Dent 1994;42:440-5.
- Little JW, Falace DA, Miller CS, Rhodus NL. Hypertension. In: Little JW, Falace DA, Miller CS, editors. Dental management of the medically compromised patient. 6th ed. St Louis: Mosby; 2002. p. 64-79.
- 42. Little JW, Falace DA, Miller CS, Rhodus NL. Infective endocarditis. In: Little JW, Falace DA, Miller CS, editors. Dental management of the medically compromised patient. 6th ed. St Louis: Mosby; 2002. p. 21-52.
- 43. Little JW, Falace DA, Miller CS, Rhodus NL. Cardiac conditions associated with endocarditis. In: Little JW, Falace DA, Miller CS, editors. Dental management of the medically compromised patient. 6th ed. St Louis: Mosby; 2002. p. 52-64.
- 44. Little JW, Falace DA, Miller CS, Rhodus NL. Ischemic heart disease. In: Little JW, Falace DA, Miller CS, editors. Dental management of the medically compromised patient. 6th ed. St Louis: Mosby; 2002. p. 79-94.
- Little JW, Falace DA, Miller CS, Rhodus NL. Congestive heart failure. In: Little JW, Falace DA, Miller CS, editors. Dental management of the medically compromised patient. 6th ed. St Louis: Mosby; 2002. p. 114-25.
- 46. Dowd FJ. Cardiac glycosides and other drugs used in heart failure. In: Yagiela JA, Neidle EA, Dowd FJ, editors. Pharmacology and therapeutics for dentistry. 4th ed. St Louis: Mosby Inc.; 1998. p. 349-61.
- Miller CS, Leonelli FM, Latham E. Selective interference with pacemaker activity by electrical dental devices. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1998;85:33-6.

Reprint requests:

Nelson L. Rhodus, DMD, MPH

School of Dentistry

7-536 Moos HST

- 515 Delaware St, SE
- University of Minnesota
- Minneapolis, MN 55455
- rhodu001@maroon.tc.umn.edu