Periodontal Management of Patients With Cardiovascular Diseases*

Periodontists are often called upon to provide periodontal therapy for patients with a variety of cardiovascular diseases. Safe and effective periodontal treatment requires a general understanding of the underlying cardiovascular diseases, their medical management, and necessary modifications to dental/periodontal therapy that may be required. In this informational paper more common cardiovascular disorders will be discussed and dental management considerations briefly described. This paper is intended for the use of periodontists and members of the dental profession. *J Periodontol 2002;73:954-968.*

In recent years tremendous progress has been made regarding the prevention, diagnosis, and treatment of cardiovascular diseases. The importance of proper diet, weight control, exercise, reduced alcohol and tobacco consumption, and life-style changes has been emphasized both in prevention and treatment of these diseases.[1-12] A wide variety of new drugs have been developed and multidrug therapy is commonly used.[1] Use of newer diagnostic devices such as transesophageal or transthoracic echocardiology are increasing and additional devices are currently being tested.[6,7] Because of these medical advances survival of individuals with cardiovascular diseases (CVD) has markedly increased, yet CVD continues to be the most serious and common health problem in the United States.[1,8] Recent evidence suggests that the presence of severe generalized periodontitis may predispose individuals to coronary artery disease.[9,13-17] The above factors coupled with the increased numbers of dentate elderly who develop periodontal disease indicates that periodontists must be prepared and will be expected to provide periodontal therapeutic support for increasing numbers of individuals with CVD. Successful and safe patient management is predicated on obtaining a thorough medical history and physical examination. The examination should include identification of any physical signs and symptoms of cardiac dysfunction and evaluation of vital signs when appropriate, including blood pressure, pulse rate and respiratory function. Medical consultation should be sought when indicated.[18-20]

CONGESTIVE HEART FAILURE

Congestive heart failure (CHF) is characterized by the inability of the heart to supply sufficient oxygenated blood to meet the metabolic needs of body tissues. Coronary artery disease is the most common cause of CHF although hypertension, valvar (or valvular) heart disease, cardiomyopathy, or diabetes mellitus may also be causal or contributing factors.[21] Ventricular arrythmia and sudden death are common in patients with CHF and intractable CHF may be best treated by heart transplantation.[1] Heart failure is associated with pulmonary congestion and venous hypertension. Patients with CHF manifest variable levels of functional compensation that must be assessed before considering dental treatment.[21,22] The presence of increasing dyspnea with minimal exertion, dyspnea at rest, or nocturnal angina indicates poor functional compensation.[23,24] Elective dental treatment for patients with poor compensation should be delayed until the condition has been stabilized with medical treatment. Emergency dental care for the unstable patient should be conservative, principally consisting of the use of analgesics and antibiotics. Medical consultation is indicated prior to treatment. In contrast, well-compensated patients may sometimes be considered for dental care without mandatory medical consultation. Appointments should be short, and the dental chair kept in a partially reclining or erect position. Appropriate sedatives should be considered for the anxious patient, and supplemental oxygen should be readily available. Patients should not be placed in a supine position, since this may allow peripheral blood to return to the central circulation and overwhelm the decompensated myocar-dium, resulting in orthopnea.[22,23]

Medical treatment for CHF has become more effective and often results in increased survival and quality of life. Monodrug therapy or combined drug regimens are used[24,25] (Tables 1 through 3). Each drug has potential side effects which must be monitored in dental practice. For example, digitalis toxicity is relatively common and the dental clinician should be alert for evidence of toxicity in any patient receiving

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Table 1.

Medications Commonly Used in Congestive Heart Failure

Inotropic agents
- digoxin (Lanoxin, Crystodigin)
- beta adrenergic blockers
  - Non-selective
    - carvedilol
    - propranolol (Inderal)
    - labetalol (Normodyne)
    - nadolol (Corgard)
    - timolol (Blocadren)
  - Cardioselective
    - atenolol (Tenormin)
    - acebutolol (Sectral)
    - betaxolol (Kerlone)
    - bisoprolol (Zebeta)
    - metoprolol (Lopressor)
    - pindolol (Visken)

Diuretics
- thiazides
  - bendoflumethiazide
  - chlorothiazide (Diuril)
  - chlorthalidone (Hygronton)
  - hydrochlorothiazide (Exidrix, Mictrin, Oretic)
  - methyclothiazide
  - metolazone (Neptazane, Microx, Diulo)
  - polythiazide (Renese)
  - quinethazone
  - trichlormethiazide (Naqua, Metahydron)
- loop diuretics
  - furosemide (Lasix, Myrosemide)
  - ethacrynic acid (Edecrin)
  - butetanide (Bumex)
  - torsemide (Demadex)

Vasodilators

nitrates
- nitroglycerin (Deponit, Nitro-bid)
- isoorbide dinitrate (Iso-Bid, Isordil)
- erythityl tetranitrate (Cardilate)
- pentacrythrityl tetranitrate (Duotrate, Peritrate, others)

hydralazine hydrochloride (apresoline)

Angiotensin-converting enzyme inhibitors See Table 3.

Angiotensin-converting enzyme receptor blockers See Table 3.

Calcium channel blockers

See Table 3. These drugs are in increasing use for congestive heart failure.

this drug. Symptoms may include anorexia, diarrhea, fatigue, headache, dizziness, or delirium, but the most dangerous manifestation is altered cardiac rhythm.[26] Angiotensin enzyme inhibitors may induce a cough reflex which could interfere with periodontal therapy[1] while the use of calcium channel blocking agents may result in unwanted gingival overgrowth.[27,28]

CARDIAC ARRHYTHMIAS

Cardiac arrhythmias may be caused by a variety of reversible abnormal physiologic events such as hypoxia and electrolyte or acid-base abnormalities. Cardiovascular causes include myocardial ischemia, bradycardia, hypertensive heart disease, valvar heart disease, increased sympathetic activity, and CHF. Sudden cardiac arrest (SCA) is a constant threat among refractory dysrhythmic individuals.[12,29] Recent evidence suggests that SCA is poorly responsive to drugs or cardiopulmonary resuscitation measures while early use of automated external cardiac defibrillation devices may markedly increase the likelihood of victim survival.[10,30,31] Drug therapy is often prescribed for chronic arrhythmias (Table 2). However, these drugs may induce adverse side effects, including xerostomia, gingival enlargement, and blood dyscrasias, any of which may contribute to development and severity of periodontal disease.[27,32,33]

Concerns have been expressed regarding the safety of using local anesthetics which contain vasoconstrictors such as epinephrine or levonordefrin in arrhythmic individuals. These agents may increase blood pressure or lead to unanticipated atrial or ventricular arrythmias to include fibrillation or even asystole.[12] In addition, vasoconstrictors may adversely interact with digoxin, non-selective B adrenergic blocking drugs, antidepressants or cocaine.[33] Most studies indicate, however, that the judicious use of local anesthetics containing vasoconstrictors is desirable in obtaining profound anesthesia for arrhthymic individuals but the quantity of vasoconstrictor should be controlled.[33-39] There appears to be no advantage or disadvantage to using levonordefrin as a substitute for epinephrine.[37,38] Intraosseous or intraligamental injections with anesthetic agents containing these drugs should usually be avoided to prevent excessive systemic absorption of the vasoconstrictor.[12,37] With careful adherance to established safety principles, local anesthetics with vasoconstrictors can be administered to patients with arrythmia, partially controlled hypertension, or other forms of cardiovascu-

Table 2.

Common Cardiac Antiarrythmics

amiodarone (Cordarone)
disopyramide (Norpace)
flecainide acetate (Tambocor)
lidocaine (Xylocaine, LidoPen)
mexiletine (Mexitil)
moricizine (Ethmozine)
phenytoin (Dilantin)
procainamide (Pronestyl, others)
propafenone (Rythmol)
propranolol (Inderol)
quinidine (Cardioguin, Quenalon, others)
sotalol (Betapace)
tocainide (Tonocard)
others

lar disease although limiting the total epinephrine to 0.04 to 0.054 mg per appointment is often recommended.[2,38-40] This translates into two-three carpules of lidocaine with 1:100,00 epinephrine (0.02 mg per carpule), as compared to a maximum of 0.2mg in a healthy adult male (11 carpules). Others have disputed this dosage restriction although most dental local anesthetic studies conducted in the past 15 years have used a quantity of local anesthetic near or below the recommended levels.[35,39,41-45] There is little doubt that most patients can safely tolerate epinephrine but patient response can be widely variable and careful monitoring is indicated.[35,39] Several controlled studies have confirmed significant changes in heart function when local anesthetics with vasoconstrictors are used for patients with cardiovascular disease[35,36,42,43,45] while others have not.[39,44,46] This appears to relate to variations in individual response to the agents although higher dosages are more likely to induce cardiac dysfunction.[36,44,47,48] Elevation in blood pressure has been described in older patients prior to initiation of treatment or in the midst of treatment rather than during administration of local anesthetics.[39] This suggests that endogenous epinephrine precipitates this rise in blood pressure and that stress reduction procedures are indicated. In one case report, however, a patient experienced markedly elevated blood pressure despite the administration of preoperative IV sedation.[47]

Some arrhythmias and even refractory vasovagal syncope[49] are best managed by implantation of cardiac pacemakers, most of which are placed in the upper chest wall and inserted into the heart by the transvenous route.[50] This creates a low risk for infective endocarditis, but the American Heart Association (AHA) does not recommend prophylactic antibiotic coverage for dental procedures in these patients.[51] Pacemakers may be disrupted by external electrical fields such as those generated by airport security devices, powerful magnets (including magnetic resonance imaging), and even cellular telephones.[23,52-54] Pacemaker dysfunction was a greater problem, however, with older models which were unipolar and poorly insulated. In the past, concerns were expressed over the potential for electrical dental devices to disrupt pacemaker function.[55,56] These concerns were largely resolved with the advent of bipolar titanium insulated cardio-pacer devices[54] and dual chamber pacemakers significantly reduce the incidence of life threatening arrhythmias in individuals at risk.[1,50] Some dental electrical devices capable of generating electromagnetic radiation may continue, however, to pose a low-grade threat to dental patients. In an evaluation of the effect of 14 electrical dental devices on cardio-pacemakers it was determined that three of these devices (electrosurgical units, ultrasonic instrument baths, and magnetorestrictive ultrasonic scalers) were capable of disrupting pacemaker function if the devices were placed in close approximation to the pacemaker. Neither sonic scalers nor electric toothbrushes adversely affected pacemaker function.[54]

Recurrent supraventricular and ventricular tachyarrhythmias are increasingly being managed by implantation of automatic cardioverter defibrillators often in combination with single or dual chamber pacemakers. Defibrillation devices were originally placed in the subcutaneous paraumbilical area of the abdomen. Patch electrodes were attached to the epicardium but electrodes of newer automatic implanted cardioversion devices or combined pacemaker/cardioversion devices are most often implanted in the chest wall and inserted into the heart transvenously.[50] According to the AHA, patients with implanted defibrillators are not at increased risk for infective endocarditis and prophylactic antibiotic coverage is not necessary unless other risk factors are present.[51] Certain precautions are recommended, however, for den-

tal procedures. The defibrillator may activate without significant warning, potentially causing the patient to flinch, bite down, or perform other sudden movements that may result in injury to the patient or the clinician. Some patients with implanted defibrillators experience loss of consciousness when the device is activated. This is less likely to occur with newer devices that initially emit low level electrical bursts followed by stronger shocks if cardioversion does not occur immediately. Epinephrine or other vasoconstrictors are contraindicated in all intractable arrhythmias[50,57] and should be used with caution (reduced dose with careful monitoring) in patients with pacemakers and implanted defibrillators.

CORONARY ARTERY DISEASE

Atherosclerotic changes in the coronary arteries produce ischemic heart disease which is the leading cause of sudden death in the United States.[9,21] The patient with ischemic heart disease may also experience atrial fibrillation,[12] angina pectoris, myocardial infarction, or other changes. Coronary artery disease (CAD) is more prevalent in the elderly, but can occur at any age.[58-60] Atherosclerotic CAD may represent a response to injury to the vascular wall by mechanical, biochemical, immunochemical, viral or bacterial insult including chlamydial infection or possibly, severe generalized periodontitis.[9,13,15,16]

Angina Pectoris

Anginal pain is always caused by a discrepancy between myocardial oxygen demands and the ability of the coronary arteries to deliver this substrate. In most instances this occurs due to narrowing of a major coronary artery. Spasm of the coronary arteries may produce a variant angina.[60]

Angina is characterized by pain, pressure, or heaviness in the retrosternal area that may radiate across the chest, into the left shoulder, down either arm, possibly between the shoulder blades, and occasionally to the side of the neck, mandible, and face. Pain duration is measured in minutes and is constricting, crushing or burning in nature. Any situation, physical or psychological, that may increase the demands on the myocardium beyond the capacity of the coronary circulation may initiate such pain.

"Stable angina" refers to chest pain which results from a predictable amount of exertion and which responds to rest or nitroglycerin.[59,60] Patients with stable angina are usually under medical care, which commonly includes combinations of beta-adrenergic blocking agents, nitrates, and calcium channel blocking agents.[61]

Patients with stable angina may receive dental care in short, minimally stressful appointments. Traditionally, morning appointments have been recommended. However, recent evidence indicates that endogenous epinephrine levels peak during morning hours and the majority of sudden cardiac arrests occur between the hours of 8 a.m. to 11 a.m. Consequently, late morning or early afternoon appointments have been recommended although scheduling is properly at the discretion of the practitioner.[12,61] Profound local anesthesia is necessary to prevent large amounts of endogenous epinephrine from being released in response to pain as described above.[39,53] If angina occurs during dental treatment, the procedure should be terminated and the patient placed in a semi-supine position; 100% oxygen should be administered; and 0.32 or 0.4 mg nitroglycerin (preferably the patient's own drug if it does not exceed its expiration date) placed sublingually. Nitroglycerin should be repeated if necessary but the minimal dose required for patient comfort should be used. Vital signs should be monitored and further emergency measures taken if necessary.[23] Pain that persists after 3 doses of nitroglycerin given every 5 minutes; that lasts more than 15 to 20 minutes; or that is associated with diaphoresis, nausea, vomiting, syncope, or hypertension may be suggestive of a myocardial infarction. While arrangements are made for immediate transportation to a hospital, vital signs must be closely monitored. The patient should continue oxygen, and chew 160 to 325 mg of aspirin. In addition, 5 to 10 mg of morphine sulfate may be given intravenously for pain and anxiety. Should cardiopulmonary arrest occur while aid is still forthcoming, resuscitative measures must be undertaken to include application of automatic external cardio-defibrillation if available.[10,30,31]

"Unstable angina" represents a clinical syndrome that is intermediate between stable angina and myocardial infarction. It features a significant change in the patient's previous anginal pattern. The patient may experience a progressive increase in frequency or severity of pain. The angina may occur at rest, or after minimal exertion, It may become more resistant to relief by nitrates. Patients with unstable angina should receive only emergency or minimal dental care after consultation with a physician. Administration of vasoconstrictors is contraindicated and the hospital may be the most appropriate environment for dental treatment of these patients.[57,60]

"Variant angina" (Prinzmetal's angina) may be precipitated by coronary artery spasm with or without

coronary artery compromise.[60] Arrhythmias are common during painful episodes although the pain is usually quickly relieved by administration of nitrates.[20,29,60] Coronary artery spasm has been reported in association with cocaine abuse. The presence of variant angina, especially in the absence of vascular lesions, should be reported to the patient's physician to rule out the possibility of drug abuse. Vasoconstrictors should be used with extreme caution in this condition.[60,62,63]

Myocardial Infarction

Myocardial infarction occurs when the narrowed atherosclerotic coronary arteries become acutely occluded by thrombus formation leading ultimately to necrosis of the portion of the heart muscle supplied by that artery. Affected patients generally report crushing substernal pain frequently with radiation to the neck, jaw, or left arm.[64] The pain may be accompanied by shortness of breath, anxiety, nausea, and diaphoresis. The highest risk of death following acute myocardial infarction occurs during the first 12 hours when the risk of ventricular fibrillation is greatest.[59,65]

Patients who have sustained a myocardial infarction are at increased risk of an additional infarction for 6 months thereafter. Consequently, current guidelines indicate that only minimal treatment for acute dental problems is advised within 6 months of an infarction after consultation with the patient's physician.[65] Elective dental care can usually be provided 6 months after a myocardial infarction. Consultation with the physician is recommended, and if no problems are noted, the dentist may proceed with treatment employing those principles used when caring for the patient with stable angina pectoris.[66] These principles include late morning appointments, profound local anesthesia, oral or inhalation sedation if needed, and close monitoring of the patient's vital signs.[53,67] Most individuals with a history of CAD are taking maintenance medication or those medications are available for use as indicated. The dental practitioner should ascertain what medications the patient is taking and should seek to avoid use of any drug known to produce an adverse interaction with such medication. Additionally, the practitioner should remain alert for signs or symptoms of adverse drug reactions or multi-drug interactions in this patient group.[68,69]

HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic cardiomyopathy is an autosomal dominant, genetically derived condition.[70] Heart muscle enlargement may restrict movement of the septal leaflets of the mitral valve leading to valvar insufficiency and regurgitation. Patients with this disorder are therefore susceptible to infective endocarditis and antibiotic prophylaxis should be considered.[70-72] Such patients are also at risk for myocardial ischemia and arrhythmias, including ventricular fibrillation. Exercise-induced sudden death is a constant risk. Epinephrine should be used with caution in these patients and nitroglycerin or similar drugs are contraindicated.[70] If angina pectoris, myocardial infarction, or fibrillation occurs in the dental office the clinician should administer oxygen and be prepared to perform cardiopulmonary resuscitation and to activate the medical emergency response system.

VALVAR HEART DISEASE

Valvar heart disease is relatively common in individuals of all ages. It results from diverse pathological processes such as rheumatic fever, congenital heart defects, ischemic heart disease, mitral valve prolapse, Kawasaki's disease (mucocutaneous lymph node syndrome), and systemic lupus erythematosus.[51,72-74] These conditions are associated with valvar stenosis and regurgitation. In recent years a significant decline in the incidence of rheumatic fever has occurred in developed countries although the incidence of infective endocarditis (IE) remains unchanged.[75] Rheumatic fever is most often initiated by Streptococcal sepsis. It can induce fibrotic scarring of valvar tissue that may be gradually progressive in adult life. Kawasaki's disease is an acute febrile disease complex of unknown etiology.[76,77] It features conjunctival congestion; dryness of lips; skin and the oral cavity; cervical lymphadenopathy; and cardiovascular changes, including coronary thromboarteritis, aneurysms, mitral valve insufficiency, and myocardial ischemia.[77] Congenital heart anomalies may induce cardiac blood turbulence and permanent valve damage even after surgical repair. Therefore, patients with congenital defects should be considered at risk for IE.[78]

Heart transplantation or ischemic heart disease may lead to valvar calcification, rupture, or scarring and predispose elderly patients to IE.[78] A previous incident of IE at any age may result in valvar damage and predispose to recurrence of IE.[75,78,79]

Mitral valve prolapse (floppy valve syndrome) occurs in response to idiopathic loss of the fibrous and elastic tissue of mitral valve leaflets or the chordae tendineae. It is highly prevalent in Down syndrome or in heritable connective tissue disorders, particularly

Ehlers-Danlos syndrome and Marfan syndrome.[80] It is also quite common in the general population, especially in young women and in individuals suffering from psychiatric disorders (e.g. panic disorder), severe depression or anorexia nervosa.[80-82] According to the AHA the degree of risk for IE in association with mitral valve prolapse may have been overstated[83] and prophylactic antibiotic coverage for dental procedures is required only if regurgitation is present.[51]

The use of fenfluramine-phenteramine, a combination of weight control drugs, has been associated with an increased incidence of valvar thickening with regurgitation, which may place individuals at risk of IE. The degree of risk associated with this drug is unknown, but a 1998 study suggests that the incidence is low (4.3%) and remission of valvar lesions may occur after discontinuing the drugs.[84]

Systemic lupus erythematosus (SLE) is sometimes associated with vegetative valvar or perivalvar lesions which increase the potential for subsequent IE, although occurrence is relatively rare. Some authors, however, recommend prophylactic antibiotic coverage for SLE patients when dental procedures are performed.[73-75]

A patient history suggesting the presence of a heart murmur requires medical consultation and a thorough understanding of the patient's condition and its possible ramifications.[85] Echocardiographic examination is extremely accurate in identifying valvar damage and some evidence suggests that magnetic resonance may be of benefit in establishing the degree of risk for the patient.[7,75]

The individual with valvar heart disease faces 3 basic risks: heart failure, hemodynamically significant arrhythmia, and IE. Of these, the dentist is most frequently required to manage patients at risk of IE. Patients who have received valvar prostheses are at special risk for occurrence of IE.[78,86,87] Dental procedures that involve manipulation of soft tissue and bleeding can produce transient bacteremias.[51] These procedures include periodontal probing, administration of intraligamental analgesia, and use of oral irrigators or air abrasive polishing devices.[75,88-95] However, transient odontogenic bacteremias also occur in association with chewing and toothbrushing, bringing into question the additional benefit gained when prophylactic antibiotic coverage is administered for dental procedures.[75,79]

Bloodborne microorganisms may lodge on damaged and abnormal heart valves, in the endocardium or in the endothelium near congenital anatomic

Table 3.

Drugs for Hypertension

Angiotensin-converting enzyme (ACE) inhibitors
- benazepril (Lotensin)
- captopril (Capoten)
- enalapril (Vasotec)
- fosinopril (Monopril)
- lisinopril (Prinivil, Zestril)
- moexipril (Univasc)
- quinapril (Accupril)
- ramipril (Altace)
- trandolapril (Mavik)

Angiotensin receptor antagonist
- candesartan cilexetil (Atacand)
- irbesartan (Avapro)
- losartan (Cozaar)
- telmisartan (Micardis)
- valsartan (Diovan)

Beta-adrenergic blocking agents (See Table 1)

Diuretics (See Table 1)

Calcium-channel blocking agents
- Substituted dihydropyridines
  - amlodipine (Norvasc)
  - felodipine (Plendil)
  - isradipine (Dynacirc)
  - nicardipine (Cardene)
  - nifedipine (Adalat, Procardia)
  - nisoldipine (Sulcar)
- Phenylakylamine derivative
  - verapamil (Calan, Isoptin, Verelan, Covera-HS)
- Benzothiazine derivative
  - diltiazem (Cardizem, Dilacor, Diltia, Timate, Tiazac)

Alpha-adrenergic blocking drugs
- prazosin (Minipress)
- terazosin (Hytrin)
- doxazosin (Cardura)

Central alpha-adrenergic agonists
- clonidine (Catapres)
- guanabenz (Wytensin)
- guanfacine (Ternex)
- methyldopa (Aldomet)

Direct vasodilators
- hydralazine (Apresoline)
- minoxidil (Loniten)

Peripheral adrenergic neuron antagonists
- guanethidine (Ismelin)
- guanadrel (Hyloral)
- reserpine

defects, resulting in IE or endarteritis. It is not possible to predict which patient will develop this infection or which particular procedure will be responsible.[51,96] This has caused many experts to consider dental treatment involving manipulation of soft tissues a risk factor for IE. In 1997, the AHA revised its recommendations regarding dental management of patients at risk for infectious endocarditis. Although not all authorities agree, the AHA continues to recommend prophylactic antibiotic coverage in the presence of certain cardiac anomalies and during specific dental treatment procedures.[51] These recommendations are principally directed toward prevention of endocarditis induced by oral *Streptococcus viridans*. The degree of risk generated by the presence of specific valvar disease is identified in Tables 4 and 5 while specific dental procedures likely to induce significant bacteremias are listed in Tables 6 and 7.

Risk for IE increases in individuals with poor periodontal health or other oral infections.[51,97-99] Rinsing with antimicrobial agents containing chlorhexidine gluconate or povidone-iodine is recommended prior to manipulation of dental tissues. To date there is no conclusive evidence, however, confirming that reduction of the oral bioload reduces the risk of bacteremias or IE.[95-103] Frequent home use of antiseptic rinses is not recommended due to the potential for developing resistant microorganisms.[51,104-108]

In patients at risk, antibiotic prophylaxis is recommended for dental procedures likely to induce significant bleeding of hard or soft oral tissues. This includes most surgical or non-surgical periodontal therapy with the possible exception of judicious dental polishing that does not induce bleeding (prophylaxis). If a series of dental procedures is required, an interval of 9 to14 days between procedures may minimize the risk of the emergence of resistant strains of organisms.[51,75] If unanticipated bleeding occurs during low risk dental procedures, antibiotics administered within 2 hours of the event may have some benefit although there is no evidence of prophylac-

Table 4.

Cardiac Conditions Requiring Prophylaxis for Dental Treatment

High risk
Prosthetic cardiac valves, including bioprosthetic and homograft valves.
Previous infective endocarditis, even in the absence of heart disease.
Complex congenital cardiac malformations.
Surgically constructed systemic/pulmonary shunts.
Moderate risk
Rheumatic and other acquired valvular dysfunction even after valvular surgery.
Hypertrophic cardiomyopathy.
Mitral valve prolapse with valvular regurgitation.
Non-complex congenital cardiac malformations.

Modified from: Dajani AS, Taubert KA, Wilson W, et al. Prevention of bacterial endocarditis. Recommendations by the American Heart Association. *Circulation* 1997;96:358-366.

Table 5.

Cardiac Conditions Not Requiring Prophylaxis for Dental Treatment

Isolated secundum atrial septal defect.
Surgical repair of secundum atrial septal defects, ventricular septal defects, or patent ductus arteriosis after 6 months and without residua.
Previous coronary artery bypass graft surgery.
Mitral valve prolapse without valvular regurgitation.
Physiologic, functional, or innocent heart murmurs.
Previous rheumatic fever without valvular dysfunction.
Previous Kawasaki disease without valvular dysfunction.
Cardiac pacemakers and implanted defibrillators.
Transplants.

Modified from: Dajani AS, Taubert KA, Wilson W, et al. Prevention of bacterial endocarditis. Recommendations by the American Heart Association. *Circulation* 1997;96:358-366.

Table 6.

Dental Procedures Creating Risk of Significant Bacteremia

Dental extractions.
Implant placement and tooth reimplantation.
Periodontal treatment procedures likely to cause bleeding.
Endodontic surgery or instrumentation beyond the root apex.
Intraligamentary injections.
Subgingival placement of antibiotic fibers or strips.

Modified from: Dajani AS, Taubert KA, Wilson W, et al. Prevention of bacterial endocarditis. Recommendations by the American Heart Association. *Circulation* 1997;96:358-366.

Table 7.

Dental Procedures Creating Low Risk of Bacteremias

Restorative procedures with or without placement of retraction cord.
Local anesthetic injections.
Placement of rubber dams.
Suture removal.
Placement or adjustment of orthodontic or removable prosthodontic appliances.
Oral impression.
Topical fluoride treatments.
Oral radiographs.
Shedding of primary teeth.

Modified from: Dajani AS, Taubert KA, Wilson W, et al. Prevention of bacterial endocarditis. Recommendations by the American Heart Association. *Circulation* 1997;96:358-366.

tic benefit if administered four or more hours after the incident.[51]

The AHA recommendations for specific prophylactic antibiotic regimens for dental procedures are widely published and will only be briefly described (Table 8). These measures are considered adequate for patients who are at high risk from IE including those with cardiac valve prostheses.[51]

Individuals who take penicillin frequently may harbor oral microorganisms that are relatively resistant to penicillin, amoxicillin, or ampicillin. In this event, clindamycin or another of the alternative regimens is recommended for endocarditis prophylaxis. Cephalosporins should be used with caution in these individuals due to the potential for microbial cross-resistance between cephalosporin and penicillin derivatives.

Professional judgment is required in managing patients who do not fit the established AHA guidelines. Tetracyclines are not recommended for prophylactic antibiotic coverage.[51] However, patients with periodontal infections induced by tetracycline-sensitive organisms may be best managed by pretreatment with tetracyclines for 2 to 3 weeks, followed by a week delay and periodontal therapy performed using AHA recommended prophylactic regimens.[109] Medical consultation should be obtained for patients who require multiple, prolonged, or unusual regimens of prophylactic antibiotic coverage. A variety of antibiotic or antimicrobial agents have recently been suggested for local delivery in treatment of periodontitis.[110] The AHA recommends systemic prophylaxis when these agents are inserted in high risk patients, due to the potential for traumatic injury and bleeding during these procedures.[51] Antibiotic prophylaxis only minimizes the risk of IE and the clinician must remain alert for symptoms associated with the condition. These may include: persistent fever, night sweats, myalgia, arthralgia, malaise, anorexia, and fatigue.[51,75,87,97]

Individuals with prosthetic heart valves experience high morbidity and mortality if IE occurs.[75,78] Under ideal circumstances a dental/periodontal examination should be performed on all patients scheduled for valve replacement open heart surgery. When possible all potential oral foci of infection should be eliminated or minimized prior to any heart surgery, including transplantation.[51,75] Routine periodontal therapy is not appropriate within 6 months of valve placement and periodontal health is an extremely important goal for the lifetime of the patient. Prophylactic antibiotic coverage should be provided during performance of most periodontal treatment procedures.[51,75]

ANTICOAGULATED PATIENTS

Anticoagulant therapy is frequently administered for patients with prosthetic valves, thromboembolic phenomena, or other flow disturbances.[75,111] This therapy may be used for a few months following placement of porcine artificial heart valves but recipients of mechanical heart prostheses may use anticoagulants for life. Warfarin sodium preparations are the agents used most often for outpatient anticoagulation. Warfarin inhibits vitamin K utilization and depletes coagulation factors II, VII, IX, and X.[112] The drug has a delayed onset and a prolonged effect. Serum level is monitored via the corrected prothrombin time, called the International Normalized Ratio (INR). Normal prothromin time has an INR value of approximately 1.0 while therapeutic doses of anticoagulant usually sustain the INR between 2.0 to 3.5.[97] Home PT/INR monitoring devices have proven to be accurate in sustaining target INR levels.[113] On occasion, INR levels of 4.0 to 4.5 may be required to prevent intravascular clotting.[12] Most evidence indicates that dental surgical procedures such as extractions or limited periodontal surgery can be performed without modifying INR levels except in extreme circumstances. Prolonged postoperative bleeding rarely occurs within an INR range of 1.0 to 3.0 although higher INR levels may be

Table 8.

Synopsis of AHA Recommendations for Adults at Risk of IE

Situation	Agent	Regimen*
Standard general prophylaxis	amoxicillin	Adults: 2.0 g; Children: 50 mg/kg orally 1 hour before procedure
Unable to take oral medications	ampicillin	Adults: 2.0 g intramuscularly (IM) or intravenously (IV); Children: 50 mg/kg IM or IV within 30 minutes before Procedure
Allergic to penicillin	clindamycin	Adults: 600 mg; Children: 20 mg/kg orally 1 hour before procedure
	or cephalexin or cefadroxil	Adults: 2.0 g; Children: 50 mg/kg orally 1 hour before procedure
	or azithromycin or clarithromycin	Adults: 500 mg; Children: 15 mg/kg orally 1 hour before procedure
Allergic to penicillin and unable to take oral medications	clindamycin	Adults: 600 mg; Children: 20 mg/kg IV within 30 minutes before procedure
	or cefazolin	Adults: 1.0 g; Children: 25 mg/kg IM or IV within 30 minutes before procedure

* Modified from: Dajani AD, Taubert KA, Wilson W, et al. Prevention of bacterial endocarditis: Recommendations by the American Heart Association. *JADA* 1997;128:1142-1151.

associated with mild to moderate localized hemorrhage.[97,99,114] Patients receiving systemic anticoagulants can usually be managed using local hemostatic measures. These include atraumatic surgical technique; adequate wound closure; application of postsurgical pressure; and use of topical clotting agents such as foamed gelatin, oxidized regenerative cellulose, thrombin, or synthetic collagen.[115-117] There is little evidence to indicate that one agent is preferable to another, although one recent paper indicated more rapid wound healing when oxidized regenerative cellulose was used.[118] Oral rinsing with tranexamic acid has been reported to further promote post-surgical hemostasis although this drug is costly and its use is rarely necessary.[115-117] Tetracyclines, erythromycin, clarithromycin, and metronidazole are contraindicated in patients on anticoagulant drugs since the antibiotics may increase prothrombin time.[119]

When contemplating procedures likely to cause bleeding, it is appropriate to communicate with the physician to determine if any anticoagulant dosage adjustments are necessary. On rare occasions it may be necessary to reduce the warfarin dosage prior to a surgical procedure. Checking the INR level on the day of the procedure may therefore be prudent.[120,121] In event of prolonged, uncontrolled post-surgical hemorrhage, blood transfusion or infusions of packed platelets or fresh frozen plasma may be necessary.[97,120,121]

Aspirin may be used alone or in combination with warfarin or ticlopidine to prevent coronary artery stent thrombois. Hemorrhagic complications are reportedly common if ticlopidine is used.[122] Aspirin is often recommended in very low doses (80 to 325 mg daily) as a maintenance antithrombotic agent because of its inhibition of platelet aggregation. This dose level will not significantly alter bleeding time. Patients using higher aspirin dosages or those routinely taking non-steroidal anti-inflammatory drugs are at some risk for prolonged postoperative hemorrhage following periodontal therapy. For these individuals, the medication may be discontinued for 1 to 2 weeks prior to the scheduled procedure to allow normal or near-normal platelet aggregation.[97,99]

HYPERTENSION

High blood pressure is the primary risk factor for cardiovascular disease and stroke as well as a major cause of end stage renal disease.[2] It affects 15% to 20% of adults in the United States.[123] In 1997 the Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of Hypertension released new guidelines defining the condition.[123] According to this report, isolated ele-

Table 9.

Classification of Adult Blood Pressure (modified from reference 2)

Optimal	<120 systolic/<80 diastolic
Normal	<130 systolic/<85 diastolic
High normal	130-139 systolic/85-89 diastolic
Hypertension	
Stage 1	140-159 systolic/90-99 diastolic
Stage 2	160-179 systolic/100-109 diastolic
Stage 3	180 or > systolic/110 or > diastolic

vations of either systolic or diastolic blood pressure are of concern in patient management and in prevention of unwanted sequelae. An individual is considered to have hypertension if blood pressure reaches 140 mm/Hg systolic or 90 mm/Hg diastolic. Diagnosis is based on average values obtained from at least 2 readings obtained on separate visits after an initial baseline measurement. Hypertension was stratified into stages (Table 9). Individuals with blood pressure readings within normal range but who are using anti-hypertension drugs should also be considered to have hypertension and be carefully monitored. Therapeutic decisions are based on the presence or absence of risk factors and the level of hypertension. Lifestyle changes and/or drug therapy are recommended for individuals with high normal blood pressure if they are afflicted with target organ disease, other cardiovascular disorders, or diabetes mellitus.[2,123,124] Individuals with Stage 3 hypertension should only receive elective dental procedures until the blood pressure is controlled. Stress reduction protocols should be used with any individual with high normal blood pressure or hypertension.[2] Therapeutic goals may vary according to the patient's age, race and medical status.[2,125,126]

An alarming number of individuals with known hypertension are not compliant with recommended medical therapy while many hypertensives remain undiagnosed.[2] For these reasons, dental health care workers can have an important role in detection and management of hypertensive patients. With routine blood pressure monitoring, undiagnosed hypertensive patients may be identified, informed of their elevated blood pressure readings, and advised to seek medical consultation. Previously identified hypertensive patients should have their blood pressure taken at each visit.[127,128] Emergency dental treatment should be as conservative as possible for the uncontrolled or untreated hypertensive individual. There are no contraindications, however, to providing dental care for the well-controlled patient.[128]

In some instances, illicit drugs (cocaine, amphetamines) or prescribed drugs (immunosuppressives, erythropoietin, mineralocorticoids, anabolic steroids) may elevate blood pressure readings. Patients using these drugs should be identified when possible and managed with care in the dental office.[2] These substances are usually contraindicated in patients with cardiovascular disease.[129]

A variety of drugs are used in treatment of hypertension and multidrug therapy is common[129] (Table 3). Complications and side effects of these drugs include hypokalemia with associated arrhythmias, postural hypertension, mental confusion, depression, drowsiness, paroxysimal coughing, and xerostomia.[2,68,128,129] Some non-steroidal anti-inflammatory drugs (indomethacin, ibuprofen and naproxen) can reduce the efficacy of antihypertensive agents.[97,129] Under most circumstances the use of epinephrine in combination with local anesthetics is not contraindicated in the hypertensive patient unless the systolic pressure is over 200 mm/Hg and/or the diastolic is over 115 mm/Hg.[2,128] As previously described, use of vasoconstrictors in local anesthetics should be carefully monitored to assure patient safety. However, profound anesthesia is indicated to minimize release of endogenous epinephrine in response to pain.[129,130] Adequate aspiration is critical to prevent intravascular injection.[53,130] Vasopressors are contraindicated for use in achieving gingival retraction or to control local bleeding.[99] Psychosedation techniques and oral and inhalation sedation; e.g., tranquilizers and nitrous oxide may be useful in treating this group of patients. General anesthesia is not recommended on an outpatient basis in individuals with significant hypertensive disease and care in a hospital setting may be indicated.[130]

VASCULAR STENTS

Vascular stents are increasingly being used to maintain patent vessels in many parts of the cardiovascular system. Although the risk of postoperative stent infection is rare, it has been reported. Nevertheless, antibiotic prophylaxis for dental treatment is generally not considered necessary for successfully engrafted cardiovascular stents. However it may be prudent to provide antibiotic coverage for emergent dental treatment during the first 4 to 6 weeks postoperatively.[131,132] Stent recipients may require long-term anticoagulant medication and appropriate action

should be taken to manage these individuals during periodontal treatment.[133]

HEART TRANSPLANTATION

Heart transplantation has become a major component in management of cardiovascular diseases. It may be indicated for patients with congestive heart failure, ischemic heart disease, hypertrophic cardiomyopathy, severe valvar defects, or intractable ventricular tachyarrhythmias.[134,135] Patients scheduled for organ transplants are carefully selected and the dentist should be an important participant in treatment planning both before and after elective transplantation. Most organ transplant centers are limited in acquisition of donor organs. Consequently, elective procedures are generally projected only for those individuals not expected to survive more than 2 years without transplantation. As a result, periodontal intervention is a realistic possibility for these patients although the underlying cardiovascular condition may limit choices of periodontal therapy. Pre-transplant dental therapy should be directed toward elimination of active or potential oral sources of infection with awareness that the patient may receive immunosuppressant therapy for the remainder of his/her life to prevent organ rejection. Patients who require emergency heart transplantation, yet have concomitant oral infections, may require antibiotics during and after transplantation until the necessary dental treatment can be rendered.[97]

Complications are common following heart transplantation.[135,136] These may include acute or chronic graft rejection, heart failure, infection or sudden death. Bacterial, viral, and protozoal infections are common due to long-term administration of anti-inflammatory and immunosuppressive drugs. Some evidence indicates a correlation between the presence of severe, generalized periodontitis and risk of myocardial infarction.[137-140] Therefore periodontal health may be extremely important for heart transplant recipients.

In successful organ transplantation, maximal heart function may approach 70% of normal.[135] The transplanted heart usually remains denervated although an active vaso-vagal reflex has been reported suggesting occasional reestablishment of neural function or an alternative non-neural mechanism for this reflex.[6,141] Due to the absence of innervation, angina is rare and patients may experience "silent" myocardial infarction or sudden death. The prudent practitioner should maintain close interaction with the patient's cardiologist and be fully aware of any known post-transplant complications.

Immunosuppressive drugs may mask early manifestations of oral infection, leading to locally severe or disseminated disease.[97] These drugs include cyclosporin, corticosteriods, antilymphocyte globulin, azathioprine or others. Cyclosporin-induced gingival overgrowth and increased susceptibility to skin and oral squamous cell carcinoma have been reported.[27,140-142] Impaired bone marrow function may lead to thrombocytopenia, anemia or neutropenia all of which could affect the oral cavity and patient response to periodontal treatment.[99]

No firm dental management protocols have been described for recipients of solid organ transplants. However, prophylactic antibiotic therapy is probably indicated if periodontal therapy is required within the first 6 months following heart transplantation. Prophylactic antibiotics may continue to be required for individuals who do not achieve maximal restoration of cardiac function or who experience acute or chronic organ rejection and ongoing immunosuppressant therapy. Prophylactic antibiotic therapy may be consistent with the guidelines established by the AHA.[51] However, more stringent antibiotic usage may be requested by the cardiologist.[99]

SUMMARY

Patients with a wide variety of cardiovascular diseases are frequently encountered in periodontal practice. Periodontal health and absence of other oral foci of infection are essential and on some occasions prophylactic antibiotic coverage is required. Safe and effective periodontal management of such patients requires close medical and dental coordination, an understanding of the potential hazards during dental treatment, knowledge of drugs used in treatment of cardiovascular diseases, and the potential adverse effects of drugs commonly used in periodontal practice.

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