
Informational Paper

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.

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INTRODUCTION

Cardiovascular diseases affect approximately 43 million people in the United States today. They afflict more Americans than any other category of serious disease.¹⁻² More than half the population over age 75 are affected and it is reasonable to assume that the incidence of heart disease will continue to increase among the elderly. The increased incidence of periodontal diseases in older individuals indicates that the periodontist must be prepared to provide periodontal therapeutic support for increasing numbers of cardiovascular patients. Successful and safe patient management is predicated on obtaining a thorough medical history; conducting a physical examination to identify any physical signs and symptoms of cardiac dysfunction; evaluating vital signs when appropriate, including blood pressure, pulse rate, and respiratory function; and obtaining medical consultation when indicated.³⁻⁷ Adherence to these management procedures, however, will not necessarily prevent cardiovascular complications.

CONGESTIVE HEART FAILURE

Congestive heart failure (left-side heart failure) is defined as the inability of the heart to supply sufficient oxygenated blood to meet the metabolic needs of the body. Heart failure is associated with pulmonary congestion and venous hypertension. Patients with congestive heart failure manifest variable levels of compensation that must be assessed before considering dental treatment. The presence

of increasing dyspnea with minimal exertion, dyspnea at rest, or nocturnal angina indicates poor compensation. Elective dental treatment for these patients ideally should be delayed until the condition has been stabilized with medical treatment. Emergency dental care should consist of conservative therapy, analgesics, and antibiotics. Well compensated patients may be considered for dental care without mandatory medical consultation. Appointments are typically short, and the dental chair should be maintained in a partially reclining or erect position, 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 position allows peripheral blood to return to the central circulation and overwhelm the decompensated myocardium, resulting in orthopnea.⁵⁻⁹

Digitalis has been used for many years to increase contraction of cardiac muscle and enhance cardiac output in patients with heart failure. The dental clinician should be alert for evidence of toxicity in any patient receiving this drug. Common toxicity symptoms may include nausea and vomiting, yellow-green vision, anorexia, diarrhea, fatigue, headache, dizziness, or delirium, but the most dangerous manifestation is altered cardiac rhythm.¹⁰ Diuretics, vasodilators, angiotensin converting enzyme inhibitors, and calcium channel blocking agents may also be used in treatment of congestive heart failure (Table 1). Each has potential side effects which must be monitored in dental practice. For example, in a recent study, 43.6% of patients receiving the calcium channel blocking agent nifedipine were reported to experience some gingival overgrowth.¹¹

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Table 1. Medications Commonly Used in Congestive Heart Failure

Inotropic Agents
digitalis (Lanoxin, Crystodigin)
beta adrenergic blockers
non-selective
propranolol (Inderal)
labetalol (Normodyne)
nadolol (Corgard)
timolol (Blocadren)
cardioselective
atenolol (Tenormin)
acebutolol (Sectral)
betaxolol (Kerlone)
pindolol (Visken)
Diuretics
thiazides
chlorothiazide (Diuril)
hydrochlorothiazide (Esidrix, Maxzide, Oretic, others)
chlorthalidone (Hygroton, others)
metolazone (Microx, Diulo)
trichlormethiazide (Naqua, Metahydron)
loop diuretics
furosemide (Lasix, Myrosemide)
ethacrynic acid (Edecnn)
bumetanide (Bumex)
Vasodilators
nitrates
nitroglycenn (Deponit, Nitro-bid)
isorbide dinitrate (Iso-Bid, Isordil)
erythryl tetranitrate (Cardilate)
pentacrythnyl tetranitrate (Dutrate, Pritrate, others)
hydalazine hydrochlondel (apresoline)
Angiotensin Converting Enzyme Inhibitors
captopril (Capoten)
enalapril (Vasotec)
lisinopril (Primivil, Zestril)
Calcium Channel Blockers
substituted dihydropyridines
amlodipine (Norvasc)
nifedipine (Adalat, Procardia)
nicardipine (Cardene)
nimodipine (Nimotop)
isradipine (Dynacirc)
nisoldipine
nitrendipine (Baypress)
felodipine (Plendil)
Phenylalkylamine derivative
verapamil (Calan, Isoptin)
Benzothiazine derivative
diltiazem (Cardizen)
Bepridil hydrochloride (Vascor)

CARDIAC ARRHYTHMIAS

Cardiac arrhythmias may be caused by a variety of reversible abnormal physiologic events such as hypoxia and electrolyte or acid-based abnormalities. Other causes include myocardial ischemia, bradycardia, increased sympathetic activity, and congestive heart failure. Antiarrhythmic drugs (Table 2) may induce potentially adverse side effects, including xerostomia, gingival enlargement,

Table 2. Common Cardiac Antiarrhythmic Drugs

digitalis (Lanoxin, Crystodigin, others)
quinidine (carioguin, Quenalon, others)
procainamide (Pronestyl, others)
disopyramide (Norpace)
lidocaine (Xylocaine, LidoPen)
mexiletine (Mexitil)
tocainide (Tonocard)
phenytoin (Dilantin)
propranolol (Inderol)
amiodarone (Cordarone)
verapamil (Calan)
diltiazem (Cardizen)

and blood dyscrasias, any of which may contribute to development and severity of periodontal disease.¹¹⁻¹³

Some arrhythmias are treated by pacemakers, most of which are implanted in the upper chest wall and inserted into the heart transvenously.¹⁴ This route of insertion creates a low risk for development of infective endocarditis in pacemaker patients, but the American Heart Association (AHA) does not recommend prophylactic antibiotic coverage for dental procedures in these patients.¹⁵⁻¹⁶ Pacemakers may be disrupted by external electrical fields such as those generated by powerful magnets including resonance imaging.^{9,14,17} The demand pacemaker is the most common type and the most sensitive to external electromagnetic forces.^{18,19} Pacemaker disruption was a greater problem with older models which were unipolar and less well insulated. Newer pacemakers are bipolar and well insulated so that the small amount of electromagnetic radiation generated by dental equipment poses little threat to function.²⁰ Information regarding the type of pacemaker in place can usually be obtained from the patient's physician.

Recurrent supraventricular and ventricular tachyarrhythmias are increasingly being managed by implantation of automatic cardioverter defibrillators. Defibrillation devices are usually placed in the subcutaneous paraumbilical area of the abdomen. Patch electrodes are attached to the epicardium, but electrodes of newer devices can sometimes be placed transvenously.¹⁴ 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.¹⁵ However, certain precautions should be considered for dental procedures. The device may activate without significant warning, potentially causing the patient to perform sudden movements that may result in injury to the patient or the clinician. Placement of a mouth prop may minimize this risk. Some patients with implanted defibrillators experience loss of consciousness when the device is activated. This is probably less likely to occur with newer devices that initially emit low level bursts followed by stronger electrical shocks if cardioversion does not occur immediately. Epinephrine is contraindicated in

all refractory arrhythmias²¹ and it 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. The patient with ischemic heart disease may experience angina pectoris, myocardial infarction or other changes. Coronary artery disease is more prevalent in the elderly, but can occur at any age.^{22,23}

Angina Pectoris

Anginal pain is always caused by a discrepancy between myocardial oxygen demands and the ability of the coronary arteries to deliver it. In most instances this occurs due to narrowing of a major coronary artery. Spasm of the coronary arteries may produce a variant angina.²⁴

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 that results from a predictable amount of exertion and that responds to rest or nitroglycerin.²⁵ Patients with stable angina are usually under medical care, which commonly includes combinations of beta-adrenergic blocking agents, nitrates, and calcium channel blocking agents.

Patients with stable angina typically receive dental care in short, minimally stressful morning appointments. Profound local anesthesia is recommended to prevent large amounts of endogenous epinephrine from being released in response to pain.¹⁷ The smallest quantity of local anesthetic necessary with the lowest concentration of epinephrine (not to exceed 1:100,000) should be used. As a general rule, the dosage of epinephrine should not exceed 0.04 to 0.08 mg/appointment for a patient with stable angina. This converts to 2 to 4 carpules of local anesthetic with 1:100,000 epinephrine (0.02 mg/carpule of 1:100,000 epinephrine).²⁶⁻³¹ Conscious sedation (e.g., tranquilizers and nitrous oxide) may be indicated. Should angina occur during dental treatment, the procedure should be terminated and the patient placed in a semi-supine position. Oxygen (100%) should be administered and 0.32 or 0.4 mg nitroglycerin (preferably the patient's own drug) placed sublingually. Nitroglycerin should be repeated if necessary, but the minimal dose necessary to abort the angina should be used. Vital signs should be monitored and further emergency measures taken if necessary.⁹ Pain that persists after 3 doses of nitroglycerin

given every 5 minutes; lasts more than 15 to 20 minutes; or is associated with diaphoresis (sweating), 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 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.³²

Unstable angina represents a clinical syndrome which 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 after minimal exertion or it may become more resistant to relief by nitrates. Patients with diagnosed unstable angina should only receive emergency or minimal dental care and only 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.^{21,25}

Variant angina (Prinzmetal's angina) is believed to be precipitated by coronary artery spasm with or without coronary artery compromise.²⁵ Arrhythmias are common during painful episodes and the pain is usually quickly relieved by administration of nitrates.^{24,33} Coronary artery spasm has been reported in association with cocaine abuse. The presence of variant angina, especially in the absence of vascular lesions, should lead to a thorough investigation to rule out the possibility of drug abuse. Vasoconstrictors should be used with caution in this condition.

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.³⁵ 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.³⁶

Patients who have sustained a myocardial infarction within the last 6 months are at increased risk of an additional infarction. Consequently, only minimal treatment for acute dental problems is advised within 6 months of an infarction, and generally only after consultation with the patient's physician.³⁷ Elective dental care can usually be provided after 6 months have elapsed following 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 angina pectoris.³⁸ These principles include morning appointments, profound local anesthesia, oral or inhalation sedation if needed, and close monitoring of the patient's vital signs.^{17,32}

HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic cardiomyopathy is often an autosomal dominant genetically derived condition that features unexplained myocardial hypertrophy. The muscle enlargement may restrict movement of the septal leaflets of the mitral valve leading to valvular insufficiency and regurgitation. Patients with this disorder who have mitral regurgitation are susceptible to infective endocarditis. These patients are also at risk for myocardial ischemia and arrhythmias, including ventricular fibrillation. Sudden death induced by exercise is a constant risk. It is important to note that nitroglycerin and related drugs are contraindicated in patients with hypertrophic cardiomyopathy. Nitroglycerin should not be administered to these patients if they develop angina pectoris in the dental office. Epinephrine should be administered with extreme caution.³⁹

VALVULAR HEART DISEASE

Valvular heart disease is relatively common in individuals of all ages and 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.⁴⁰⁻⁴² The conditions are associated with valvular stenosis and regurgitation. Rheumatic fever can induce fibrotic scarring of valvular tissue that may gradually progress in adult life. Kawasaki's disease is an acute febrile disease complex of unknown etiology. It features conjunctival congestion; dryness of lips; skin and the oral cavity; cervical lymphadenopathy; and cardiovascular changes, including coronary thromboarteritis, mitral valve insufficiency, and myocardial ischemia.⁴³ Congenital heart anomalies may induce cardiac blood turbulence. Therefore, patients with congenital defects should be considered at risk for infective endocarditis.⁴⁴

Mitral valve prolapse (floppy valve syndrome) is characterized by idiopathic loss of the fibrous and elastic tissue of mitral valve leaflets or the chordae tendineae. It is highly prevalent in heritable connective tissue disorders, particularly Ehlers-Danlos syndrome and Marfan's syndrome.⁴⁴ Mitral valve prolapse is quite common in the general population, especially in young women and in individuals suffering from psychiatric disorders (e.g., panic disorder), severe depression, anorexia nervosa, or Down's syndrome.⁴⁵⁻⁴⁷ It is important for the physician to determine whether or not regurgitation is present in patients with mitral valve prolapse since the condition requires prophylactic antibiotic coverage for dental procedures only if regurgitation is present.⁴⁰

Table 3. Cardiac Conditions Requiring Antibiotic Prophylaxis

- Prosthetic cardiac valves, including bioprosthetic and homograft valves
- Previous infective endocarditis, even in the absence of heart disease
- Most congenital cardiac malformations
- Rheumatic and other acquired valvular dysfunction even after valvular surgery
- Hypertrophic cardiomyopathy
- Mitral valve prolapse with valvular regurgitation

Table 4. Cardiac Conditions Not Requiring Antibiotic Prophylaxis

- Isolated secundum atrial septal defect
- Surgical repair without residue beyond 6 months of secundum atrial septal defect, ventricular defect, or patent ductus arteriosus
- 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

Ischemic heart disease may result in calcific degeneration, rupture, or scarring of perivalvular muscle tissue leading to mitral valve regurgitation.⁴⁸ The presence of any of these or related conditions would cause the patient to describe a previous history of heart murmur. Therefore, any such history requires medical consultation and a thorough understanding of the patient's condition and its possible ramifications.⁴⁹⁻⁵⁰

The dental patient with valvular heart disease faces 3 basic risks: heart failure, hemodynamically significant arrhythmia, and infective endocarditis. Most often, the dentist is required to manage patients at risk of infective endocarditis. Untreated periodontal disease or other oral infections place these patients at increased risk of bacteremias. Dental procedures involving manipulation of soft tissue resulting in bleeding can produce a transient bacteremia. There is some evidence that use of oral irrigators or air abrasive polishing devices may create a low risk of bacteremia if improperly used.⁵¹⁻⁵⁵ Blood-borne microorganisms may lodge on damaged and abnormal heart valves, in the endocardium, or in the endothelium near congenital anatomic defects, resulting in infective endocarditis or endarteritis. It is not possible to predict which patient will develop this infection or which particular procedure will be responsible.^{15,56}

Certain cardiac conditions are more often associated with endocarditis than others. In 1990, the AHA established prophylactic antibiotic guidelines for patients with the cardiac conditions identified in Tables 3 and 4. Although these recommendations should be followed, there is no conclusive evidence that prophylactic antibiotics prior to dental treatment will prevent infective endocarditis. Consequently, some authorities have suggested that the risks of adverse side effects from administration of prophylactic antibiotics may outweigh the demonstrated benefits.

In patients at risk, antibiotic prophylaxis is recommended with all dental procedures likely to induce gingival or mucosal bleeding, including professional cleaning.¹⁵ If a series of dental procedures is required, it may be prudent to observe an interval of 7 to 14 days between procedures to minimize the risk of the emergence of resistant strains of organisms.⁵⁷⁻⁵⁹

The AHA recommendations for specific prophylactic regimens for dental procedures are widely published and will not be repeated in this text. For most adults, administration of 3 grams of amoxicillin one hour before the dental procedure is recommended, followed by 1.5 grams 6 hours after the initial dose. Erythromycin or clindamycin are recommended as alternatives in patients who are allergic to penicillin. Alternative intramuscular or intravascular antibiotic regimens are described for patients who cannot take oral medications. The basic recommendations are considered adequate for patients who are at high risk from infective endocarditis, including those with cardiac valve prostheses.¹⁵ Rinsing or irrigation with an antimicrobial agent containing chlorhexidine can be used prior to manipulation of dental tissues. This may be especially important in high risk patients and in those with poor oral hygiene. However, there is no conclusive evidence to confirm that rinsing reduces the risk of bacteremias.⁶⁰⁻⁶³ Individuals who take an oral penicillin for secondary prevention of rheumatic fever or for other purposes may harbor oral microorganisms that are relatively resistant to penicillin, amoxicillin, or ampicillin. In such cases, the dentist should select erythromycin, clindamycin, or another of the alternative regimens for endocarditis prophylaxis. Individual judgments may have to be made for patients who do not fit established guidelines set forth by the AHA. Tetracyclines are not recommended for prophylactic cardiovascular antibiotic coverage.¹⁵ Some authorities have suggested that patients with periodontal diseases associated with tetracycline-sensitive organisms can best be treated by administration of tetracyclines for 2 to 3 weeks followed by a 1 week delay, followed by tissue manipulative therapy under coverage of AHA recommended prophylactic regimens.⁶⁴ Medical consultation should be obtained as indicated for patients who require multiple, prolonged, or unusual regimens of prophylactic antibiotic coverage. Antibiotic prophylaxis may reduce the risk of infective endocarditis but does not preclude its occurrence.^{2,15,32,66} The clinician must remain alert for persistent fever or other symptoms associated with endocarditis. A dental examination must be performed on all patients who are to have a valve replacement and potential sources of bacteremia eliminated, if possible, prior to the valve surgery.

ANTICOAGULATED PATIENTS

Patients with prosthetic valves, thromboembolic phenomena, or other flow disturbances are often therapeutically anticoagulated. Coumarin preparations are the agents currently used for outpatient anticoagulation. They exert their effect through the competitive inhibition of vitamin K with subsequent depletion of those coagulation factors dependent on that substance for their synthesis (II, VII, IX, and X). Coumarin has a delayed onset and a prolonged effect. Its effectiveness is monitored via the prothrombin time (PT). The most common therapeutic dose of anti coagulant is one that results in the patient's prothrombin time being 1-1/2 to 2 times that of normal clotting time.²³ This is considered within the range of safety to perform dental procedures associated with bleeding. On occasion, however, prothrombin time is maintained between 2 to 2-1/2 times control.⁶⁶ In that event some adjustment may be necessary for periodontal surgical procedures. Prothrombin time standards tend to vary between laboratories and can lead to significant errors in anticoagulant therapy.⁶⁷ Recently an international reference thromboplastin has been developed for use in obtaining a standardized universal PT in all laboratories.⁶⁸ This "corrected" PT is called the International Normalized Ratio (INR). INR is approximately 1.0 for patients with normal PT and the recommended level of anticoagulant modification for most outpatients is 2.0 to 3.5 INR although higher levels may be required for some patients with severe coagulation problems.^{69,70} It has been suggested that an INR of 3.0 to 4.0 or lower is safe for performance of dental procedures likely to induce bleeding provided appropriate local hemostatic measures are taken. These may include the use of foamed gelatin, synthetic collagen, direct pressure, primary closure, and tranexamic acid rinses.⁷⁰⁻⁷⁵ Tetracyclines are contraindicated in patients on anticoagulant drugs since tetracyclines interfere with prothrombin formation.⁷⁶

When contemplating procedures likely to cause bleeding, it is appropriate to communicate with the physician and request any dosage adjustments necessary to keep the prothrombin time within the safety level. Adjustments may need to be initiated 2 to 3 days prior to the procedure. To insure hemorrhage control the appropriate prothrombin time should be verified on the morning of the dental appointment. After dental treatment the patient should be advised to resume taking the anticoagulant as soon as possible (usually day of procedure).⁷⁷ When local hemostatic measures fail in the anticoagulated patient, pharmacologic manipulation becomes necessary.³⁷ If the anticoagulated patient can tolerate a wait of several hours or more, vitamin K administration will reverse the effect or coumarin. More urgent situations may require a transfusion of fresh-frozen plasma.

Aspirin is often used as an antithrombotic agent because of its inhibition of platelet aggregation. Most car

diologists prescribe very small daily dosages (80 to 325 mg). At this dose level the medication will not significantly alter bleeding time.²³ On occasion, however, patients on higher aspirin levels are at slight risk for prolonged postoperative hemorrhage following periodontal therapy. For these individuals, the medication should be discontinued for 1 to 2 weeks prior to the scheduled procedure with the concurrence of the cardiologist.

HYPERTENSION

High blood pressure is the major risk factor for cardiovascular disease and a major cause of renal failure and stroke. It affects 15% to 20% of adults in the United States.⁷⁸ A blood pressure reading of >160/95 mm Hg is generally considered to represent hypertension for an adult.⁷⁸⁻⁸⁰ A reading between 140/90 mm Hg and 160/95 mm Hg is borderline hypertension.¹⁷ Some authorities, however, define hypertension at varying blood pressure levels according to the patient's age, sex, and race.^{81,82} The prevalence of hypertensive heart disease increases sharply with age.⁷⁷⁻⁷⁸

Dental health care workers can have an important role in detection and management of hypertensive patients. With routine blood pressure monitoring, consisting of several readings taken at different visits, undiagnosed hypertensive patients may be identified and informed of their elevated blood pressure readings. These patients should be advised to seek medical consultation. Previously identified hypertensive patients should have their blood pressure taken at each visit.⁸¹ Elective dental treatment for the uncontrolled hypertensive patient should be deferred until control is achieved. Emergency dental treatment for an uncontrolled hypertensive patient should be as conservative as possible. There are no contraindications, however, to providing dental care for the well-controlled hypertensive patient. Some studies have indicated that absorption of sodium bicarbonate during oral hygiene procedures or following use of an air abrasive cleansing device may alter serum electrolyte balance. Any such effects appear minimal and transient, however, and there is no strong evidence to suggest that use of these substances are contraindicated in patients with cardiovascular disease.⁸³⁻⁸⁶

Drugs used in treatment of hypertension include, diuretics, antiadrenergics, vasodilators, and angiotensive converting enzyme inhibitors (Table 4). Complications and side effects of these drugs include hypokalemia with associated arrhythmias, postural hypotension, mental confusion, depression, drowsiness, and xerostomia.⁸⁷ Some non-steroidal anti-inflammatory drugs (indomethacin, ibuprofen, and naproxen) can reduce the efficacy of some antihypertensive agents.⁸⁸ 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.⁸⁹⁻⁹¹ Most authorities recommend small amounts of epinephrine; 0.04 to 0.08 mg/appointment (approximately 2 to 4 carpules containing 1:100,000) as compared to a maximum of 0.2 mg in a healthy 70 kilogram adult male (10 carpules). Profound local anesthesia is indicated to minimize release of endogenous epinephrine in response to pain.^{92,93} Adequate aspiration is critical to prevent intravascular injection.^{17,94} The use of vasopressors to control local bleeding and gingival retraction cord containing vasopressors is contraindicated. Psychosedation techniques and oral and inhalation sedation; e.g., tranquilizers and nitrous oxide may be useful in treating this group of patients. Caution must be exercised, however, in the use of nitrous oxide for outpatient conscious sedation since hypoxia may produce startling and calamitous increases in arterial blood pressure. General anesthesia is not recommended on an outpatient basis in patients with significant hypertensive disease because of the risk of secondary hypertension. Medical consultation and care in a hospital setting may be indicated.²³

HEART TRANSPLANTATION

Protocols have not yet been established for dental management of recipients of organ transplants. Application of common treatment principles, however, should facilitate the provision of safe and effective periodontal therapy, including participation of the dentist where indicated in the treatment planning for patients about to undergo elective heart transplantation. Active and potential sources of infection should be eliminated and necessary dental care should be accomplished whenever possible before the transplant. Patients who receive a heart transplant on an emergency basis and who have existing dental infection should be given antibiotics before and after the transplant until dental treatment is rendered. Following heart transplantation, recipients may be maintained on immunosuppressive drugs for life to blunt host rejection of the graft. These drugs may include cyclosporine, corticosteroids, antilymphocyte globulin (ALG), azathioprine, or combinations thereof.³⁹

Immunosuppressive agents may mask early manifestations of oral infection, or feature atypical presentations of common lesions such as recurrent oral herpes simplex. Oral mucosal lesions suggestive of herpes simplex, candidiasis, or other fungal infections should be evaluated by cytological examination, culture and/or biopsy when indicated. The practitioner should remain alert for signs or symptoms of unusual systemic fungal infections such as cryptococcus and mucormycosis. These infections may lead to severe disseminated disease in immunosuppressed patients and must be detected early so appropriate antimicrobial therapy can be instituted.

Prophylactic antibiotics are recommended for all dental procedures likely to cause a bacteremia in transplant patients taking immunosuppressive drugs, and physician

consultation is appropriate. In addition, chlorhexidine application to the surgical site may reduce the incidence of bacteremia.¹⁵ In patients taking cyclospoline, the potential for gingival enlargement exists.^{11,20} For patients taking adrenal suppressive doses of corticosteroids, supplementation of steroids may be necessary prior to stressful dental procedures.

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