The diseases discussed in this chapter include developmental and congenital malformations of the heart, atherosclerotic thromboembolic vascular occlusive disease, acquired cardiac inflammatory and infectious diseases (rheumatic heart disease and endocarditis in particular), and congestive heart failure. These common conditions have significant impact on dental professionals and their patients. Dental implications include disease detection by cognizance of salient signs and symptoms, management of emergencies, treatment-plan alterations and prophylaxis owing to the patient’s medically compromised status, and treatment considerations in the patient who is under pharmacologic management for cardiovascular disease.

The clinical tools used to ascertain the presence or absence of cardiovascular disease include the health history, vital sign assessment, electrocardiography, specific serum chemistries, echocardiography and other imaging techniques, and exercise tolerance testing. The findings that one encounters using these tests are detailed in the section on clinical features.

Pathophysiology

The heart is a four-chambered muscle that is lined by endothelium. This organ is often considered in the context of a double pump: a right heart and a left heart. The two pumps each have an upper chamber, the atrium, and a lower chamber, the ventricle. These two chambers are interfaced by a valve. The right atrium receives blood through large vessels draining the liver and systemic circulation, whereas the left atrium receives oxygenated blood from the lungs. Blood is pumped from the atria through the valves into the right and left ventricles. The two ventricles contract in unison, sending blood out into the circulation while the valves close, preventing regurgitation back into the atria. When the ventricles are at rest and the atria are pumping blood into them, the term diastole is used. When the atria are at rest and the ventricles are contracting, the term systole is used. The right ventricle pumps its blood through the pulmonic valve and pulmonary artery into the lungs and the left ventricle delivers blood to the systemic circulation through the aortic valve and into the aorta. It is the systolic contraction of the left ventricle that accounts for the higher recorded level of the blood pressure, whereas the lower level of pressure recorded is the resting pressure during diastole. If peripheral vessels are constricted, there is an increase in peripheral resistance to the outflow of blood through the circulation and the diastolic pressure is elevated (hypertension). In general, normal blood pressure is considered to be less than 130 systolic and less than 85 diastolic. When the diastolic pressure exceeds 90 mm, the patient is considered to be borderline hypertensive.

The contractions of the atria and ventricles are a biologic miracle. The heart has an innate capacity for muscular contraction that emanates from a focus in the right atrium known as the sinoatrial (SA) node. The impulse involves the movement of ions across the cardiac sarcolemmic membrane and specialized conduction fibers, an event termed depolarization. The impulse travels through the cell membranes switching from negativity to positivity. These migrating positive charges are detectable with an electrical recording instrument, the electrocardiograph. Positively charged electrodes are placed on the skin, and when a positive depolarization wave migrates toward the electrode, there is an upward (positive) deflection of the recording devise. When the positivity moves away from the positive electrode, there is a downward (negative) deflection of the record.

The electrocardiogram (EKG) is a graphic tracing of the heart’s electrical activity over time (Figure 3–1). The recording paper strip moves at a constant rate and is calibrated longitudinally and vertically. The longitudi-
nal divisions allow one to calculate the heart rate, and the vertical dimensions are a measure of charge intensity. If an electrode is placed on the chest, directly over the anterior lower aspect of the left ventricle (so-called lead 5), a standard EKG pattern can be recorded. In this lead, the first wave one observes is the P wave. This is the passage of the depolarization impulse from the SA node through the atria. When the depolarization impulse reaches the atrial-ventricular (AV) node there is a slight pause followed by a small negative deflection, the Q wave. Immediately there is an abrupt positive spike with a downward deflection to the baseline, the R wave, then continuation of the negative component with an upward deflection back to baseline, the S wave. The QRS complex represents the electrical activity of the conduction of fibers of the ventricles. The impulse travels from the AV node down the interventricular septum through the right and left main bundle branches, then up through the ventricles via Purkinje fibers and on into the myocardial cells to induce contraction There is then a lull in electrical activity followed by a slight positive then negative deflection, the T wave, that represents repolarization of the ventricles. The interposed quiet period is the S-T interval. This, of course, is the normal pattern of polarization and depolarization of the heart. The atrial repolarization wave is masked by the ventricular QRS complex.

There are numerous other leads that the cardiologist can use to assess electrical activity in the heart. There are six limb leads and six chest leads. These leads allow one to pinpoint foci of cardiac pathology and also to assess the axis of the cardiac electrical conduction system. The electrocardiogram can be analyzed to assess rate, rhythm, conduction blocks, infarction, electrolyte imbalances, drug toxicity, and other organ-system pathology, particularly pulmonary dis-

Ammendement in the analysis of coronary ischemia and infarction, comparison of the various chest leads is essential to pinpoint the region of the left ventricle in which the vascular occlusive change has had its effects. An overview of EKG interpretation is given in the section on clinical features.

The opening and closing of valves during the cardiac cycle can be detected by placing a stethoscope on the chest, directly over the heart. Auscultation of specific areas on the chest discloses a major sound during systole, when the mitral and tricuspid valves snap shut, and during diastole when the aortic and pulmonic valves close (Figure 3–2). The mitral-tricuspid sound is designated S1; the aortic-pulmonic sound is termed S2. The aortic valve may close slightly ahead of the pulmonic valve, and when auscultated at the region of the base during inspiration, S2 is split into two closely sequenced sounds. The S2 split on inspiration is a normal finding. Some patients also manifest S3 or S4 sounds that are interposed between the two major “lub-dub” (S1-S2) sounds. During diastole ventricular filling is initially passive and then muscular contraction in the atria results in rapid ejection. During the passive phase, a vibration in the ventricles occurs and is perceived as faint extra sound, sounding like “Ken-tuck-y”; this is S3. During the muscular ejection phase another sound may be detected, sounding like “Tenn-e-see”; this is S4. The S3 and S4 sounds are often undetected and when present in a normal subject, they are usually quite faint. If they are pronounced, they are termed a gallop and cardiac pathology is likely to be present.

A murmur is an abnormal heart sound or a deviation from normal sounds, including physiologic splits of S2. Murmurs occur in patients with septal defects, patent ductus, valvular stenosis or insufficiency, and mitral valve prolapse with regurgitation. Murmurs may also occur in high cardiac output states, such as anemia, thyrotoxicosis, and pregnancy. Valvular diseases or deformities in which murmurs are detected are subdivided into two major groups: those that cause a nar-
narrowing of the valve aperature between the heart chambers at the exit of the great vessels and those that result in failure of valve closure. Narrowed valves are said to be stenotic, whereas valvular insufficiency is the terminology used to denote defects in closure. In many cases, valvular stenosis and insufficiency may coexist.

Before the discussion of the pathophysiology of various cardiac disease states, an overview of cardiac failure is presented (Figure 3–3). Heart failure simply means there is inadequacy of the heart as a pump and tissues can no longer be perfused. The heart fails as a consequence of (1) myocardial damage, as in myocardial infarction; (2) increased peripheral resistance; (3) compression of the heart, a process that occurs when the pericardium is filled with fluid or blood; (4) malfunction of the heart valves; and (5) pathologic shunts. Because the heart is a closed vascular system, what affects the left heart will in fact have implications for the right heart and vice versa. Nevertheless, it is convenient to consider the heart as two sided. Right-sided heart failure occurs when there are right valvular defects, pathologic shunts, pulmonary disease, or pericardial compression. When the right heart fails, blood returning from the venous hepatic and systemic circulations becomes congested, leading to distention and edema from increased venous hydrostatic pressure. When the left heart fails, congestion into the lungs occurs leading to pulmonary edema. Eventually, regardless of the source, congestive heart failure progresses to involve both systemic and pulmonary vasculature.

### Table 3–1  Cardiac Diseases That May Predispose to Endocarditis

<table>
<thead>
<tr>
<th>Cardiac Diseases That May Predispose to Endocarditis</th>
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<tbody>
<tr>
<td>Congenital heart defects</td>
</tr>
<tr>
<td>Atrial septal communication</td>
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<tr>
<td>Ventricular septal communication</td>
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<tr>
<td>Patent ductus arteriosus</td>
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<tr>
<td>Aortic stenosis</td>
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<td>Mitral stenosis</td>
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<tr>
<td>Pulmonic stenosis</td>
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<tr>
<td>Bicuspid valve stenosis</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
</tr>
<tr>
<td>Eisenmenger complex</td>
</tr>
<tr>
<td>Miscellaneous other combined deformities</td>
</tr>
<tr>
<td>Acquired heart defects</td>
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<tr>
<td>Rheumatic valve disease</td>
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<tr>
<td>Fenfluramine-dexfenfluramine valvulopathy</td>
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<tr>
<td>Prosthetic valves</td>
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Congenital deformities of the heart occur during the process of growth and development and are, therefore, evident as congenital malformations. Table 3–1 lists the more common congenital heart defects. Atrial and ventricular septal defects reflect an embryologic failure of septal closure. Patent ductus arteriosus is a shunt extending from the pulmonary artery to the aorta, an embryologic structure that normally closes to become the ligamentum arteriosum. Congenital valvular stenosis (narrowing of the valve orifice) involves primarily the aortic, mitral, and pulmonic valves. All of these defects, if severe enough, can culminate in congestive failure and may predispose to infection of the internal aspect of the heart (endocarditis). There are numerous combination defects or cardiac syndromes, the two more common of which are the tetralogy of Fallot and the Eisenmenger complex. In the tetralogy, there is a ventricular septal defect, a dextroposed aorta (such that the opening of the aorta directly overlies the septal defect), pulmonic valve stenosis, and a secondary right ventricular hypertrophy. Because unoxgenated blood can pass across the septal defect and enter the aorta, cyanosis is a classic feature. In the Eisenmenger complex, the defects mirror tetralogy, yet there is no pulmonic valve stenosis.

Rheumatic fever is a streptococcal systemic infection in which immunopathologic sequelae evolve. Following immune clearance of infectious organisms, streptococcal antigens remain in the circulation as immune complexes. These complexes filter into various tissues where they fix complement, and initiate inflammatory lesions that are host-tissue destructive. The three target tissues and organs affected by immune complex post-streptococcal events are the heart, kidneys, and synovial membranes. Rheumatic heart disease comprises sterile fibrotic lesions of the heart valves (mitral and aortic being the favored sites) and inflammatory lesions along conduction fiber pathways in the myocardium. Valvular stenosis and insufficiency are...
detected as cardiac murmurs, and myocardial lesions result in arrhythmias. Immune complex lesions in the glomerulus lead to glomerulonephritis, and synovial inflammatory lesions result in the formation of a pannus in rheumatoid arthritis. The cardiac lesions of rheumatic heart disease can cause congestive heart failure, and damaged valves can be colonized by pathogenic bacteria, a disease known as endocarditis (see Chapter 17).

Another form of acquired valvular deformation is attributed to a combination of two drugs that are used in diet control. Fenfluramine and dexfenfluramine, commonly known as fen/fen, are associated with cardiac valvulopathy but with variable differences in risk assessment. These valvular lesions are often associated with murmurs.

One of the most common diseases to affect mankind is atherosclerotic thromboembolic vascular occlusive disease (Figure 3–4). Atherosclerosis evolves as a consequence of endothelial damage followed by accumulation of lipids within the intima of arteries and veins. The primary risk factors include genetic predisposition; hyperlipidemia, particularly with very low density (VLDL) and low density lipoproteins (LDL); hypertension; sedentary life-style; male gender, and increasing age. Vascular adhesion molecules, such as selectins, cell adhesion molecules (CAMs), and integrins, are upregulated and facilitate the emigration of leukocytes into the atheroma. These complicated lesions may then develop tunneling fissures with erosion of the endothelial lining, resulting in activation of the intrinsic pathway of coagulation. In this manner, thrombi form within vessel lumens, leading to ischemia or infarction; thrombi may be dislodged to become emboli. Arterial emboli become lodged in lumens, whereas venous emboli break free, travel in the enlarging

![Figure 3-4](image-url) Consequences of atherosclerosis with thrombosis.
veins to enter the right heart, into the pulmonary artery, and become lodged in the lung arteries (pulmonary embolism). The consequences of arterial vascular occlusion include ischemia and infarction, coronary heart disease, cerebrovascular accident, and aneurysm.

Coronary occlusion leads to ischemic heart disease that is often detectable on EKG. Complete vascular occlusion is responsible for coronary infarction or heart attack. Occlusion can result in small infarcts that may be inconsequential, whereas large vessel occlusion can result in massive necrosis and death. Sudden infarcts lead to myocardial necrosis with seepage of cardiac enzymes into the circulating blood. Elevated lactic dehydrogenase and creatinine phosphokinase enzymes are detected, and the EKG reveals specific conduction defects. Epidemiologic studies have shown a correlation between dental and periodontal infections and coronary heart disease. Even so, no causal relation can be documented.

**Clinical features**

The clinical manifestations of cardiovascular diseases are signs and symptoms of cardiac failure, arrhythmia, hypertension (see Chapter 4), angina, myocardial infarction, stroke, and risk of endocarditis. As has been mentioned previously, the signs of heart failure include ankle edema, jugular vein distension, and hepatomegaly if there are right heart lesions and dyspnea in left heart lesions. Syncope and fatigue are also signs and symptoms of cardiovascular disease. In ischemic heart disease, rheumatic heart disease, and selected drug toxicities, conduction disturbances occur and are detectable on the EKG (Figure 3–5). When the SA node fires rapidly, over 70 times per minute, the condition is referred to as sinus tachycardia. Reading cardiac rate on an EKG strip is facilitated by the bold black vertical lines. If a QRS complex occurs at the first major subdivision, the rate is 300 per minute. If the depolarization occurs every two major divisions, the rate is 150; at three subdivisions, the rate is 100; at four it is 75, and at five subdivisions the rate is 60. Bradycardia is assessed by noting how many depolarizations occur during a 15-second segment and multiplying by 4.

When there are no coordinated depolarization waves originating in the SA node and multiple ectopic foci fire, the P waves disappear and the EKG pattern becomes highly erratic. Although irregularly spaced QRS complexes are evident as some of these ectopic foci trigger the ventricular node, such a pattern is termed atrial fibrillation, and in essence, there is no coordination of atrial contractions. Ventricular fibrillation is a dire medical emergency; it is characterized by a jagged EKG pattern with no identifiable normal depolarization waves. In ventricular fibrillation and flutter, pumping action is no longer extant. In sinus arrhythmia, extra beats are identifiable on the EKG, and such ectopic beats are evident as early P waves followed by normal QRS complexes. Wandering pacemakers represent depolarizations emanating in the atria, outside the SA node, and under these conditions, the P waves are heterogeneous in shape.

Premature ventricular contractions (PCVs) are considered pathologic if there are more than five per minute (Figure 3–6). They are characterized by QRS intervals that are widened. Sinus arrest is characterized by a lengthy interval between the end of the P wave and the origin of the QRS complex. In AV arrest, there is a repeatable delay from P to QRS. Conduction system blocks are easily visualized on selected chest leads.

**Figure 3–5** Abnormal EKG patterns. Top Panel, sinus tachycardia is portrayed by normal-appearing P waves followed by normal QRS and T waves occurring with a periodicity exceeding 70 beats per minute. Middle panel, atrial fibrillation is characterized by the firing of multiple pacemakers in the atria. No normal P waves are evident, rather there is a staccato pattern of P waves with occasional QRS, indicating that some of these ectopic pacemakers transmit to the AV node. Ventricular fibrillation evolves when multiple ectopic sites fire within the ventricles, yielding a sporadic irregular EKG pattern. During ventricular fibrillation, the heart has no effective pumping action. Lower panel, sinus tachycardia is represented by periods of nonactivity, indicating that the SA node is failing to fire in sequence, a sign of coronary artery disease. Wandering pacemaker signifies variable ectopic foci in the atria, replacing the normal SA node, and is characterized by P wave pattern heterogeneity.
Recall that the depolarization wave travels down the right and left main bundles and then back up through the ventricular walls and out into the musculature via Purkinje fibers. These impulses are portrayed by the QRS complex. Main bundle branch blocks are differentiated with chest leads V1, V2, V5, and V6, since the V1 and V2 leads overlie the right heart and the V5 and V6 leads overlie the septum and left ventricle. The peak of the S wave is bifurcated when depolarization is blocked and then resumes through the conduction fiber pathways. In right main bundle branch blocks, the bimodal peak in V1 and V2 is markedly accentuated, whereas in left bundle branch block, a dip is witnessed at the vertical peak of the R wave in leads V5 and V6.

Myocardial injury, ischemia, and infarction all show characteristic EKG patterns. Recall that ischemia and infarction are mainly left ventricular events. Myocardial injury shows an elevated repolarization T wave, infarction may manifest with an accentuated Q wave downward deflection, and ischemia often reveals itself with an inverted T wave. There are many other characteristic patterns indicative of cardiac pathology. Cardiac hypertrophy, depolarization axis perturbations, certain drug toxicities, and various other disorders can be detected on the basis of EKG patterns.

Angina pectoris is chest pain that radiates into the left jaw or down the left arm as a consequence of coronary artery spasticity. Angina is a prelude to infarction and requires further workup that includes cardiac enzymes, EKG, and sonography. If these studies suggest coronary occlusion, cardiac catheterization is performed, to detect arterial narrowing or blockage by a thrombus or embolus. The consequences of coronary artery ischemia range from mild arrhythmia to cardiac arrest and death. Infarction invariably occurs in the left ventricular and ventricular septum, and its precise localization can be determined by assessment of the various chest leads on the EKG. Small infarcts may have no clinical significance, whereas larger foci may lead to papillary muscle collapse with resulting valvular insufficiency. Conduction pathway blocks occur in infarcted areas leading to significant arrhythmias. Coronary artery balloon angioplasty and bypass surgery are required when significant vascular occlusion is detected.

Cardiac disease diagnosis employs auscultation. Heart sounds are detected through the stethoscope, and in a normal heart, the sounds that are heard are those of valve closure. Valvular stenoses and insufficiencies create abnormal heart sounds, termed murmurs, that are referred to as splits and clicks. As mentioned previously, a split of S2 during inspiration is normal. When there is a delay between the two split sounds, cardiopathology is present, such as a bundle branch block. Many heart sounds are complex and subtle, requiring an experienced ear for adequate detection, classification, and interpretation. Chest radiographs disclose heart size, cardiomegaly being a sign of hypertrophy and compensation during congestive failure. Serum chemistries are particularly useful in the diagnosis of myocardial infarction. Lactic dehydrogenase and creatinine phosphokinase isoenzymes can be assessed when a patient reports chest pain or pressure and also in the emergency room when a patient is admitted unconscious.

Medical management involves surgical techniques and or medications to reduce morbidity and mortality. Drugs can be used as single agents or in combination. For coronary artery disease, balloon angioplasty and bypass surgery are the primary interventions. Open heart surgery is performed to repair congenital defects.
and place artificial valves. Heart transplant is reserved for instances of severe infarction leading to cardiac failure. Preventive interventions include low-fat, low-sodium diet, regular exercise, and smoking cessation. It follows then that dental care of the cardiac patient may require special precaution and understanding. These are discussed below and in Chapter 11, where drug interactions are discussed.

**Dental management**

The risk of endocarditis evolving as a consequence of bacteremia from oral microorganisms is a major concern in dental practice. Any patient with a history of organic murmur is at risk. Invasive dental procedures, such as exodontias and oral, periodontal, or endodontic surgery, are considered to increase the risk of endocarditis in patients with valvular disease, septal defects, patent ductus, and mitral valve prolapse with concomitant regurgitation. It matters not whether valve defects are congenital or acquired; in both instances turbulent flow occurs within the heart chambers, and in areas of flow voids, bacterial colonization can ensue. The protocols for prevention of endocarditis by antibiotic prophylaxis are detailed in Chapter 17. As discussed previously, the lesions at risk usually manifest as a cardiac murmur. When a patient indicates that he or she has been diagnosed with a murmur, it is recommended that the physician be contacted, to ascertain whether it is “organic” or “functional”; the former indicates cardiac damage, the latter is of no clinical consequence. Mitral valve prolapse is a common malady among females. If no regurgitation is detected, antibiotic coverage is unnecessary; if present, the valve is susceptible to bacterial colonization, and prophylaxis is required for invasive dental procedures. Recall from earlier discussion that the fen-fen diet regimen was found to be associated with or perhaps even to cause valvular lesions in which a murmur can be detected. If a patient indicates a history of taking this regimen, it is recommended that he or she consult their physician to determine if any valvular pathosis was incurred and whether antibiotic prophylaxis may be indicated for invasive dental procedures.

Some patients may indicate having had a detectable murmur as a child and that as an adult, it is no longer detectable. It would be prudent to pursue this in more detail with the patient’s physician by telephone, electronic mail, or written consultation. When a patient indicates having been diagnosed with a murmur, yet is not aware of the necessity for antibiotic prophylaxis during invasive dental procedures, the prophylactic recommendations should be followed if a dental emergency is extant and there is no time to confirm the status of the murmur.

Coronary atherosclerosis is the commonest cause of cardiovascular disability and death in the industrialized world. The average age for males is 50 to 65 years and for females, 60 to 70 years. Atherosclerotic vascular occlusive disease is a common medical illness that requires attention when dental procedures are being performed. Coronary ischemia, myocardial infarction, and stroke are all complications of thromboembolic disease. Any of these complications may occur during the undertaking of dental procedures, and all of them are more likely to evolve during periods of anxiety and stress. These same concerns apply to hypertensive patients, since they are at increased risk for thromboembolic events. Among patients with a history of angina, myocardial infarction, or stroke, efforts must be made to reduce stress levels by achieving sound and profound anesthesia for dental and soft-tissue surgical procedures. In consultation with the patient’s physician, the dentist may elect to prescribe an anxiolytic medication prior to dental procedures. Any of the diazepam group drugs can be used, such as 1.0 mg of lorazepam or 10 mg of benzodiazepam 1 hour prior to an office visit. When prescribing anxiolytics, it is important to realize that sedation and drowsiness occur, thereby precluding the driving of an automobile. Patients should be advised to arrange for transportation following completion of the dental procedure.

If during a dental procedure a patient complains of angina, the procedure should be stopped and a coronary vasodilator must be administered. A differential diagnosis is critically important, to ensure that the patient’s symptoms are indeed indicative of angina. Recall that angina may arise as substernal pain, a severe pressure sensation, and may be confused by the patient as indigestion. When the pain radiates over the left arm or into the left jaw, coronary ischemia is the primary clinical diagnosis. Many patients with chronic angina wear nitroglycerin patches, and the dentist should be aware of this practice, emphasizing to the patient that the patch should be worn the day of their appointment. For those patients who take nitroglycerin sublingually, it is prudent to have them bring their medication with them when they are receiving dental care, and keeping the tablets within easy reach is important. During severe angina, cardiac arrest may evolve. If the patient slips into unconsciousness, 911 should be dialed, and the dental team should assess breathing and pulse. If a carotid pulse is detectable yet no respirations are perceived, assisted breathing by mouth-to-mouth or a bag device should be initiated. When neither pulse nor respirations are present, cardiopulmonary resuscitation must commence immediately. Automated external cardiac defibrillators are being placed in many public places and will eventually find their way into the dental office as a significant addition to the office emergency.
kit. In the event of sudden coronary arrest in a subject with no detectable pulse, defibrillation should be attempted. These automated devices are applied to the chest wall and activated. If repeated attempts fail, CPR should be performed until the mobile emergency care unit arrives. The American Heart Association’s CPR Guidelines should be followed.

If the patient experiences signs of cerebrovascular accident while in the dental operatory, he or she should be reclined and oxygen should be administered. Impending stroke may be signaled by transient ischemic attacks manifested by sudden vision changes. In other cases, no warning signs appear and a patient loses consciousness. Unfortunately, there are no emergency interventions that will prevent cerebrovascular occlusion or rupture of aneurysms. Certain strokes are the consequence of emboli derived from carotid artery aneurysms. Calcified atheromatous plaques of the carotid artery can sometimes be seen on panoramic radiographs; the plaques appear as focal opacities or extend longitudinally up the course of the artery and appear just posterior and inferior to the mandible on the film (Figure 3–7). For patients who have not been diagnosed with carotid vascular occlusion or aneurysm, this finding on routine dental films should be noted and reported to the patient’s physician. Vascular surgery and replacement of the diseased vessel can be a life-saving event. Administration of fibrinolytic enzymes within minutes of a stroke may remove thrombi.

Another important consideration for the patient with a history of stroke, coronary artery disease, or thrombophlebitis is the fact that such individuals may be medicated to reduce the risk of thrombosis. Anticoagulant regimens may be of no clinical consequence when the patient is taking a single preventive daily dose of aspirin; alternatively, some high-risk patients are placed on Coumadin and have significantly depressed coagulative capability. The effects of Coumadin are monitored by the prothrombin time and the international normalized ratio (INR). In general, invasive surgical procedures should be avoided or undertaken with great caution in patients with a prothrombin time that is 50% of normal (or double the control time) or an INR exceeding 2.5 (see Chapter 7). When these markers of coagulation status are indicative of a potential for severe bleeding and the patient requires immediate dental intervention, the procedure should be performed in a hospital dental setting in consultation with an internist or hematologist. Expertise in coagulopathies is required in the event that factor supplementation may be needed. In nonemergency situations, the patient’s physician should be consulted about the possibility of withdrawing Coumadin 3 to 4 days prior to the dental surgical procedure, to allow the INR to decrease. Importantly, the dentist should never alter the patient’s anticoagulant regimen without consultation. If the coagulability is restored to normal levels, there is the possibility that the patient could experience a thromboembolic event.

Patients who are experiencing dental pain should be treated with analgesics that will not interfere with coagulation or platelet adhesion. In this context, aspirin, ibuprofen, ketorolac, and other nonsteroidal anti-inflammatory drugs (NSAIDs) with platelet adhesion inhibitory effects should be avoided. Codeine, oxycodone, and hydrocodone without aspirin can be used in pain management.

Other complications occurring in the patient with atherosclerosis involve medications that are prescribed to reduce edema in congestive failure, lower blood pressure, and treat arrhythmias. Diuretics and antihyperten-
sives may render the patient prone to the development of orthostatic hypotension. Standing suddenly after residing for some time in a reclined dental chair may bring about such an episode. Calcium channel blockers reduce vascular tone, and these medications often induce gingival hyperplasia (see Chapter 23).

Many mild arrhythmias are managed medically, whereas more severe arrhythmic disease may be treated by placement of a pacemaker. Most pacemakers are shielded from external electrical and microwave radiation; nevertheless, ultrasonic devices, cleaners, and microwave ovens should not be operated within the immediate vicinity of a patient with a pacemaker until the patient’s physician is contacted and reassurance is given that the specific device brand is safe under such circumstances. Specific management considerations for the hypertensive patient are detailed in Chapter 4.

### Suggested reading


