Ischemic Heart Disease

(Myocardial Infarction & Angina Pectoris)

Coronary atherosclerotic heart disease is a major health problem in the United States and in other industrialized nations. Atherosclerosis is the thickening of the intimal layer of the arterial wall caused by the accumulation of lipid plaques. The atherosclerotic process results in a narrowed arterial lumen with diminished blood flow and oxygen supply. Atherosclerosis is the most common underlying cause of not only coronary heart disease (angina and myocardial infarction [MI]) but also cerebrovascular disease (stroke) and peripheral arterial disease (intermittent claudication). Symptomatic coronary atherosclerotic heart disease is often referred to as *ischemic heart disease.* Ischemic symptoms are the result of oxygen deprivation caused by reduced blood flow to a portion of the myocardium. Other conditions such as embolism, coronary ostial stenosis, coronary artery spasm, and congenital abnormalities also may cause ischemic heart disease.

GENERAL DESCRIPTION

Incidence and Prevalence

More than 70 million Americans (about 25% of the population) are estimated to have some form of cardiovascular disease, with about 13 million having coronary heart disease. The mortality rate per year from cardiovascular diseases as a group has been declining since 1940. From 1970 to 2000, mortality from coronary heart disease decreased by 50% and from stroke by 60%. In spite of this decline, cardiovascular diseases continue to pose the most serious threat to health in America, accounting for about 40% of all deaths. Coronary heart disease is the leading cause of

death in the United States after age 65, and it is responsible for 1.1 million new or recurrent heart attacks annually, of which 40% are fatal.

Etiology

The cause of coronary atherosclerosis is not known; however, research indicates that the disease is related to a variety of risk factors. These risk factors include male gender, older age, a family history of cardiovascular disease, hyperlipidemia, hypertension, cigarette smoking, physical inactivity, obesity, insulin resistance and diabetes mellitus, mental stress, and depression. Between the ages of 35 and 44 years, the risk is 5 times greater for men than for women. MI and sudden death are rare in premenopausal women; however, after menopause, a rapid reduction occurs in this gender difference. The fact that men are more prone to the clinical manifestations of coronary atherosclerosis is accentuated in nonwhite populations. Studies have confirmed that individuals with parents or siblings affected by coronary atherosclerotic heart disease have a greater risk of developing the disease at a younger age than do those without such a history. This risk may be as high as 5 times greater. Elevation in serum lipid levels is a major risk factor for atherosclerosis. Increased levels of low-density lipoprotein cholesterol pose the greatest risk for coronary atherosclerosis, whereas increased levels of high-density lipoprotein cholesterol have been shown to reduce the risk. Individuals with elevated triglyceride or betalipoprotein levels have an increased risk for the disease. A diet rich in total calories, saturated fats, cholesterol, sugars, and salts also enhances the risk. Increased blood pressure appears to be one of the most significant risk factors for coronary atherosclerotic heart disease. The Framingham Study showed that angina, MI, and nonsudden death were all significantly correlated with elevated blood pressure (140/90 mm Hg or greater). Most epidemiologic studies,

however, recognize the importance of both DBP and SBP in the assessment of cardiovascular risk. Patients with diabetes mellitus have a greater incidence of coronary atherosclerotic heart disease and more extensive lesions. They develop the condition at an earlier age than do persons who do not have diabetes. Although hyperglycemia is associated with microvascular disease, insulin resistance itself promotes atherosclerosis even before it produces frank diabetes, and available data corroborate the role of insulin resistance as an independent risk factor for atherothrombosis. No single risk factor is responsible for the development of coronary atherosclerosis, but many factors act synergistically. Evidence suggests that modification of those risk factors that can be controlled such as cigarette smoking, hypertension, hyperlipidemia, and diabetes may reduce or modify the clinical effects of the disease.

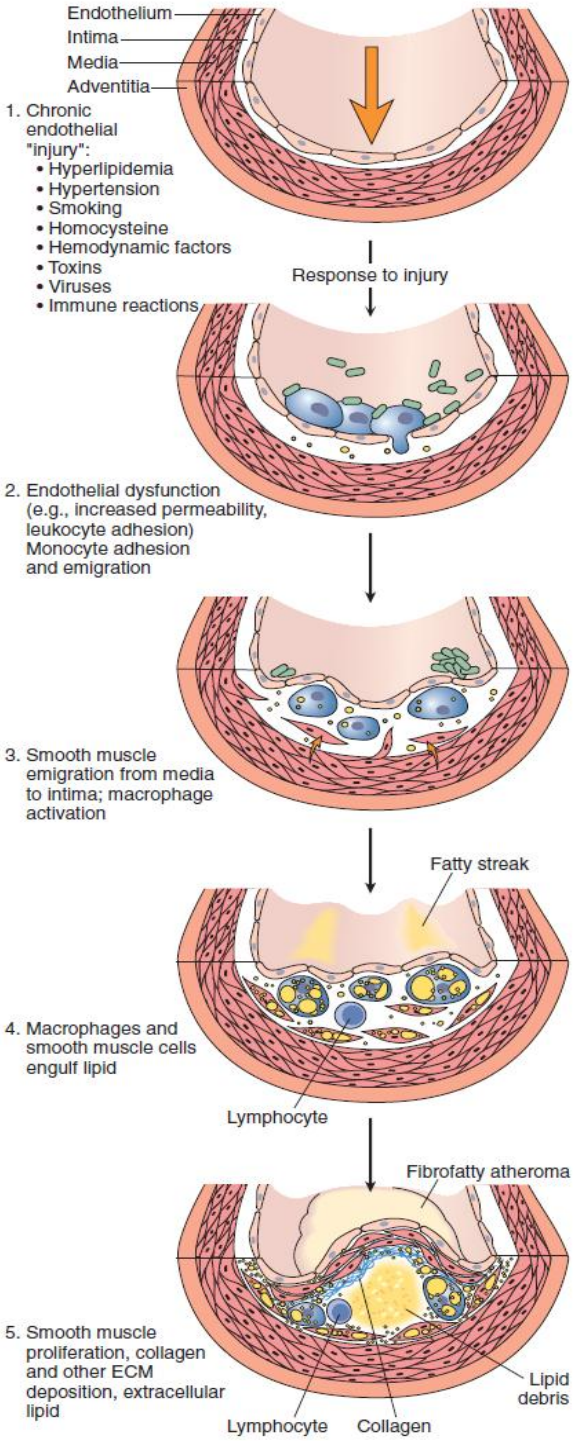

Figure 4-1. Evolution of a plaque within a coronary artery. (Schoen FJ. Blood vessels. In Kumar V, Abbas AK, Fausto N [eds]. Robbins and Cotran Pathologic Basis of Disease, 7th ed. Philadelphia, Saunders, 2005.)

CLINICAL PRESENTATION

Symptoms

Chest pain is the most important symptom of coronary atherosclerotic heart disease. The pain may be brief, as in angina pectoris resulting from temporary ischemia of the myocardium, or it may be prolonged, as in unstable angina or AMI. Ischemic myocardial pain results from an imbalance between the oxygen supply and the oxygen demand of the muscle. Atherosclerotic narrowing of the coronary arteries is an important cause of this imbalance. The exact mechanism or agents involved in producing the cardiac pain are not known. Angina pectoris usually is described as an aching, heavy, squeezing pressure or tightness in the midchest region. The area of discomfort often is described to be approximately the size of the fist and may radiate into the left or right arm to the neck or lower jaw. In rare cases, it may be present in only one of these distant sites, and the patient is free of central chest pain. The pain is brief, lasting 5 to 15 minutes if the provoking stimulus is stopped or for a shorter time if nitroglycerin is used. Angina is defined in terms of its pattern of symptom stability. *Stable angina* is pain that is predictably reproducible and consistent over time. This pain typically is precipitated by physical effort such as walking or climbing stairs but may also occur with eating or stress. Pain is relieved by cessation of the precipitating activity, by rest, or by the use of nitroglycerin. *Unstable angina* is defined as new onset of pain, pain that is increasing in frequency, more intense pain than before, pain that is precipitated by less effort than before, or pain that occurs at rest. This pain is not readily relieved by nitroglycerin. The key feature is the changing character or pattern of the pain. Patients with stable angina have a relatively good prognosis. Patients with unstable angina have a poorer prognosis and

often develop MI within a short time. The term *acute coronary syndrome* describes a continuum of myocardial ischemia that ranges from unstable angina at one end of the spectrum to non–ST segment MI at the other. Differentiation requires diagnostic and laboratory testing. A relatively uncommon form of angina, *Prinzmetal's variant angina,* occurs at rest and is caused by focal spasm of a coronary artery, usually with varying amounts of atherosclerosis.28 Angina also may occur in individuals with normal coronary vessels. Patients with coronary atherosclerosis who develop prolonged pain as a result of myocardial ischemia usually have unstable angina or are having an AMI. This pain usually is more severe and lasts longer than 15 minutes but has the same general character as that described for stable angina. Its location is the same and it may radiate in the same pattern as the brief pain that results from temporary myocardial ischemia. Vasodilators or cessation of activity does not relieve the pain caused by infarction. Neither brief nor prolonged pain resulting from myocardial ischemia is aggravated by deep breathing..

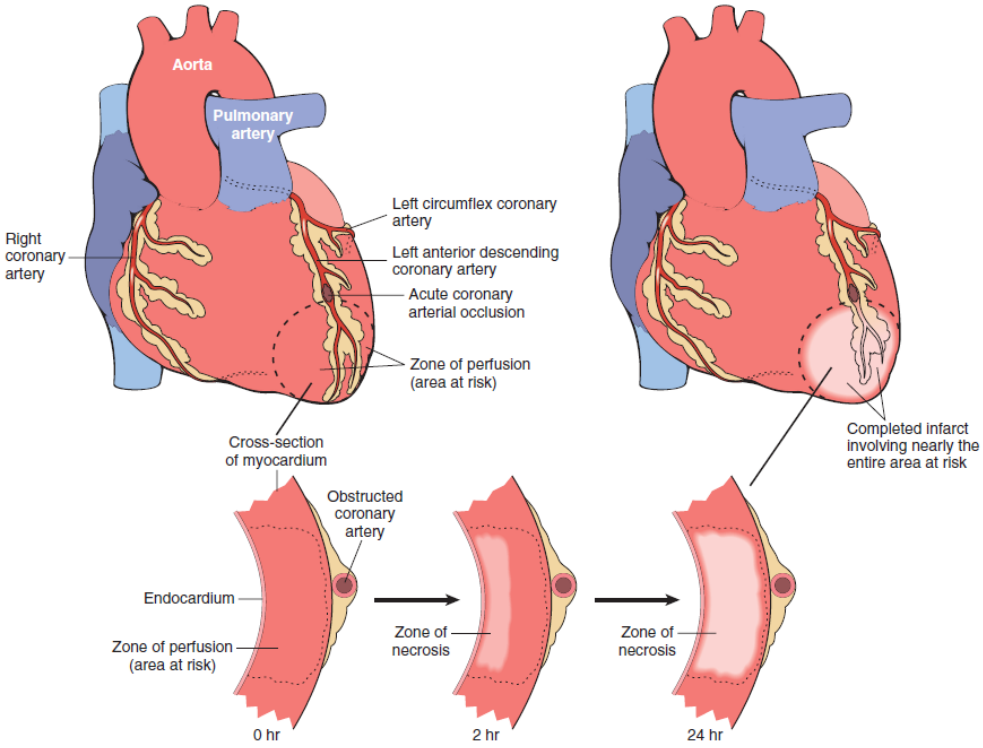

Figure 4-4. Progression of myocardial necrosis after coronary artery occlusion. (From Schoen FJ. The heart. In Kumar V, Abbas AK, Fausto N [eds]. Robbins and Cotran Pathologic Basis of Disease, 7th ed. Philadelphia, Saunders, 2005.)

The most common cause of sudden death is ventricular fibrillation, a form of abnormal electrical activity resulting from interruption of the electrical conduction system. Palpitations of the heart (disagreeable awareness of the heartbeat) may be present in patients with coronary atherosclerotic heart disease with normal or abnormal rhythm. The complaint is not directly related to the seriousness of the underlying cardiac problem. Syncope, a transient loss of consciousness resulting from inadequate cerebral blood flow, also may occur in patients with coronary atherosclerotic heart disease. Symptoms of congestive heart failure experienced as a complication of coronary atherosclerotic heart disease include dyspnea, orthopnea, paroxysmal nocturnal dyspnea, edema,

hemoptysis, fatigue, weakness, and cyanosis. Fatigue and weakness may be present early in the course of the disease before the onset of congestive failure .

Signs

Clinical signs of coronary atherosclerotic heart disease are few, and the patient may appear entirely normal. Most clinical signs relate to other underlying cardiovascular disease or conditions such as congestive failure. Conditions such as corneal arcus and xanthoma of the skin are related to hyperlipidemia and hypercholesterolemia. Blood pressure may become elevated, and abnormalities in the rate and/or rhythm of the pulse may occur. Diminished peripheral pulses in the lower extremities may be seen, along with bruits in the carotid arteries. Panoramic radiographs of the jaws may occasionally demonstrate carotid calcifications in the areas of C3 and C4, consistent with atherosclerotic plaques in the carotid arteries. Retinal changes are common in hypertensive disease and diabetes mellitus. Signs associated with advanced coronary atherosclerotic heart disease usually refl ect the presence of congestive heart failure. Distention of neck veins, peripheral edema, cyanosis, ascites, and enlarged liver may be found.

Laboratory Findings

Blood tests are used in the evaluation of patients with symptoms of angina pectoris to screen for abnormalities that may contribute to or worsen coronary heart disease. These include complete blood count to rule out anemia, thyroid function tests to exclude hyperthyroidism, renal function testing to exclude renal insufficiency, lipid screening for hypercholesterolemia, glucose for diabetes, homocysteine levels, and levels of C-reactive protein. Other diagnostic tests that are specific for coronary heart disease include resting ECG, chest x-ray, exercise stress testing,

ambulatory (Holter) electrocardiography, stress thallium-201 perfusion scintigraphy, exercise echocardiography, ambulatory ventricular function monitoring, and cardiac catheterization and angiography.

MEDICAL MANAGEMENT

Angina Pectoris Medical management of a patient with chronic stable angina involves five aspects:

1. Identification and treatment of associated diseases that can precipitate or worsen angina
2. Reduction in coronary risk factors
3. Application of general and nonpharmacologic methods, with particular attention toward adjustments in lifestyle
4. Pharmacologic management
5. Revascularization by percutaneous catheter–based techniques or by coronary bypass surgery

Management may include general lifestyle measures such as an exercise program; weight control; restriction of salt, cholesterol, and saturated fatty acids; cessation of smoking; and control of exacerbating conditions such as anemia, hypertension, and hyperthyroidism.

Calcium channel blockers are effective in the treatment of chronic stable angina when given alone or in combination with beta blockers and nitrates. These drugs decrease intracellular calcium, resulting in vasodilatation of coronary, peripheral, and pulmonary vasculature, along with decreased myocardial contractility and heart rate. Aspirin used as antiplatelet therapy is another cornerstoneof treatment in patients with angina.

Coronary artery bypass graft (CABG) surgery is an effective means of controlling symptoms in the management of unstable angina; it can improve

the long-term survival rate in certain subsets of patients. It also is effective in controlling symptoms in patients whose pain persists despite medical control. With CABG, a segment of artery or vein is harvested or released from a donor site; it is then grafted to the affected segment of coronary artery, thus bypassing the area of occlusion . Two primary graft donor sites are used: the saphenous vein and the internal mammary artery. Of the two, the internal mammary artery graft is sturdier and much less susceptible to graft atherosclerosis and occlusion than are vein grafts.

Myocardial Infarction

Patients who have experienced an AMI should be hospitalized or should receive emergency treatment as soon as possible. The basic management goal is to minimize the size of the infarction and prevent death from lethal arrhythmias. The size and extent of the infarct are critical in the determination of outcome. Early administration of aspirin is recommended, with 160 to 325 mg being chewed and swallowed to decrease platelet aggregation and limit thrombus formation. The definative treatment of patients with AMI depends on the extent of ischemia as reflected on the ECG by the presence or absence of ST segment elevation. MI *without* ST segment elevation is due to partial blockage of coronary blood fl ow; MI *with* ST segment elevation is due to complete blockage of coronary blood fl ow and more profound ischemia involving a relatively large area of myocardium. This distinction is clinically important because early fi brinolytic therapy improves outcomes in ST segment elevation MI but not in non–ST segment elevation MI..

The management of AMI has undergone significant change over the past several years with the recognition that thrombolytic therapy can result in

significant reduction of morbidity and mortality in ST segment elevation MI. The greatest benefit is realized when patients receive thrombolytic drugs within the first 3 hours after infarction; however, modest benefit is possible even up to 12 hours after the event. The early use of thrombolytic drugs may decrease the extent of necrosis and myocardial damage and dramatically improve outcome and prognosis. Thrombolytic (or fibrinolytic) drugs used in the treatment of AMI include streptokinase, urokinase, alteplase (rt-PA), and reteplase. For most patients with ST segment elevation MI, the preferred method for revascularization is fibrinolysis or percutaneous coronary angioplasty. Percutaneous transluminal coronary angioplasty with stenting is an alternative to thrombolytic therapy that yields better outcomes. In patients with ST segment elevation MI, non–ST segment elevation MI, or unstable angina (acute coronary syndrome), anticoagulation is often applied in the form of unfractionated heparin or low molecular weight heparin; in addition, glycoprotein IIa/IIIb inhibitors (abciximab, eptifi batide, tirofi ban) are administered intravenously for their antiplatelet effects. Antiplatelet drugs are significant in decreasing morbidity and mortality, and aspirin is the drug of choice. Daily doses of 81 to 325 mg are recommended. Clopidogrel and ticlopidine are other antiplatelet drugs that may be used, although the use of ticlopidine has been supplanted by the administration of clopidogrel because of superior outcomes reported with the latter. For pain relief, morphine sulfate is the drug of choice. Sedatives and anxiolytic medications also may be used. Oxygen may be administered by nasal cannula during the acute period to enhance oxygen saturation of the blood and keep the heart workload at a minimum level. A pacemaker may be used with severe myocardial damage and resultant heart failure.

DENTAL MANAGEMENT

Medical Considerations

Risk assessment for the dental management of patients with ischemic heart disease involves three determinants:

1. Severity of the disease
2. Type and magnitude of the dental procedure
3. Stability and reserve of the patient

All must be factored into a dental management plan so that a rational and safe decision can be made, specifically, to determine whether a patient can safely tolerate a planned procedure. The American College of Cardiology and the American Heart Association published risk stratification guidelines for patients with various types of heart disease who are undergoing various noncardiac surgical procedures. These guidelines canprovide significant value in the determination of risk for surgical and nonsurgical dental procedures.

Recent MI (within the past 7 to 30 days) and unstable angina are classified as clinical predictors of major risk for perioperative complications. Stable (mild) angina and past history of MI are identified as clinical predictors of intermediate risk for perioperative complications. The type and magnitude of the planned procedure also must be considered, as well as the perioperative risk conveyed by the diseases themselves. Based on these guidelines, most dental procedures, including minor oral surgery and periodontal surgery, would fall within the low-risk, “superficial procedures” category, with less than 1% risk. Nonsurgical dental procedures are likely to pose even less of a risk than is incurred by surgical procedures. More extensive oral and maxillofacial surgical procedures, and perhaps some of the more extensive periodontal surgical procedures, would fall in the intermediate cardiac risk

category under "head and neck procedures." These procedures carry a risk lower than 5%. Procedures that present the highest risk include emergency major surgery in the elderly, aortic or vascular surgery, and peripheral vascular surgery. These procedures are performed with the patient under general anesthesia and have the potential for significant blood and fluid loss with resultant adverse hemodynamic effects. These risk stratification guidelines may be applied to various dental management scenarios. For example, a patient with unstable angina or recent MI is classified as a major cardiac risk. However, if the planned dental procedure is limited to routine clinical examination with x rays (considered at extremely low risk), and the patient is stable and not anxious, the risk for an adverse occurrence is not significant; thus, alterations needed in the dental management approach would be minimal. Conversely, a patient with stable angina or a past history of MI (intermediate risk) with minimal cardiac reserve, who is scheduled for multiple extractions and implant placement (low to intermediate risk), poses a more significant risk and may require a more complex dental management plan.

Angina Pectoris. A determination should be made about the severity and stability of angina. A patient with stable angina characteristically describes the occurrence of chest pain in a consistent, recurring, and predictable pattern. Pain is precipitated by typical physical activity such as exercising, mowing the lawn, or climbing stairs and subsides within 5 to 15 minutes with rest or the use of nitroglycerin. Pain occurs in a chronic, unchanging pattern over time. These patients pose an intermediate cardiac risk.

A patient with unstable angina conversely may describe the recent onset of chest pain, or progressively worsening chest pain that occurs with physical exertion or at rest. Typically, a pattern of increasing severity, frequency, or

duration of pain occurs. Pain occurring at rest or during sleep is particularly ominous. Patients with unstable angina should be considered a major cardiac risk. Based on the assessment of medical risk, the type of planned dental procedure, and the stability and anxiety level of the patient, general management strategies for patients with stable angina or a past history of MI without ischemic symptoms (intermediate risk) may include the following: short appointments in the morning, comfortable chair position, pretreatment vital signs, availability of nitroglycerin, oral sedation, nitrous oxide/oxygen sedation, excellent local anesthesia, limited amount ofvasoconstrictor, avoidance of epinephrine-impregnated retraction cord, avoidance of anticholinergics, and effective postoperative pain control. For patients who have had balloon angioplasty with placement of a coronary artery stent, or for those who have undergone a CABG procedure, antibiotic prophylaxis is not recommended.

For patients with symptoms of unstable angina or those who have had an MI within the past 30 days (major risk), elective care should be postponed . If treatment becomes necessary, it should be performed as conservatively as possible and directed primarily toward pain relief, infection control, or the control of bleeding. Consultation with the physician is advised. Additional management recommendations may include establishing and maintaining an intravenous line, continuously monitoring the ECG and vital signs, using a pulse oximeter, and administering nitroglycerin prophylactically just before the initiation of treatment. These measures may require the patient be treated in a special patient care facility or hospital dental clinic. The use of vasoconstrictors in local anesthetics poses potential problems for patients with ischemic heart disease because of the possibilities of precipitating cardiac tachycardias, arrhythmias, and increases in blood pressure. Local

anesthetics without vasoconstrictors may be used as needed. If a vasoconstrictor is necessary, patients with intermediate risk and those taking nonselective beta blockers can used with precautions; intravascular injections are avoided. Greater quantities of vasoconstrictor may well be tolerated, but increasing quantities increase the risk of adverse cardiovascular effects. For patients at higher risk, the use of vasoconstrictors should be discussed with the physician. Studies have shown, however, that modest quantities of vasoconstrictors may be used safely even in high-risk patients when accompanied by oxygen, sedation, nitroglycerin, and excellent pain control measures.

Myocardial Infarction. The dentist should determine whether the patient has had an MI or any symptoms of ischemia (e.g., chest pain, shortness of breath, easy fatigue) for the purpose of quantifying risk. Patients who have had an MI have some degree of permanent damage to the heart. The outcome depends on the extent and location of the damage and its effect on the function of the heart. Damage may be minimal, with little effect on the patient's daily activity. Patients who have had an MI within the previous month with residual ischemic symptoms are classifi ed as a *major cardiac risk* and are not candidates for elective dental care. Patients with a past history of an MI longer than 1 month ago who are clinically stable are classified as being an *intermediate cardiac risk* and, in most cases, are at minimal risk for routine dental treatment. However, myocardial damage may be extensive, resulting in cardiac instability and an inability of the heart to function properly (i.e., heart failure). These patients are classified as a *major cardiac risk* and pose a significant risk for the provision of routine dental care. Management recommendations for stable angina are also applicable to patients with an uneventful past history of an MI . For several weeks after an

MI has occurred, risks of cardiac instability, arrhythmias, and reinfarction may be increased. These effects decrease with time, assuming that the electrical conduction system of the heart has not been seriously damaged. In the past, most authorities suggested that the dentist not provide elective dental care for the first 6 months after an MI because of the risks cited earlier. Local anesthesia with vasoconstrictor was used in conjunction with sedation and close monitoring in all cases. Thus, it is apparent that many patients recovering from a recent MI can undergo dental treatment safely if such treatment should become necessary. Delaying elective treatment for at least 4 to 6 weeks after an uncomplicated MI is recommended, prior to the provision of elective care. Again, risk assessment must be undertaken as a precursor to decision making. Consultation with the physician is advisable. Patients with postinfarction disability or heart failure face significant risk and should delay elective care. Patients who take aspirin or another platelet aggregation antagonist can expect some increase in bleeding. This effect generally is not clinically significant, and bleeding may be controlled through local measures. Discontinuation of these agents before dental treatment is provided generally is unnecessary. Laboratory assessment of platelet function is problematic in that no currently available test accurately and predictably reflects the ability of platelets to perform their normal function. Bleeding time can be assessed before invasive procedures Patients who take warfarin for anticoagulation must have a current international normalized ratio (INR) determined before any invasive procedure can be performed. Most dental procedures, including minor surgery, may be performed safely without discontinuation or alteration of the Coumadin dosage, as long as the INR is within the therapeutic range (3.5 or less). Local hemostatic measures generally are adequate to control bleeding and

include the use of hemostatic agents in the sockets, suturing, gauze pressure packs, and tranexamic acid or ε-aminocaproic acid mouth rinses. More extensive surgical procedures associated with anticipated significant blood loss should be discussed with the patient's physician.

Oral Manifestations

No lesions or oral complications are the direct result of coronary atherosclerotic heart disease. Drugs used in the treatment of this disease and its complications, however, may produce oral changes such as dry mouth, taste changes, and stomatitis. Patients who take warfarin or aspirin may have increased bleeding after trauma or surgical procedures. In rare cases, patients with coronary atherosclerotic heart disease with angina may have pain referred to the lower jaw or teeth. The pattern of onset of pain caused by physical activity and its disappearance with rest usually serves as a clue to its cardiac origin.

BOX 4-1

Medical Management of Patients With Stable Angina Pectoris

- Identification and treatment of associated diseases that can precipitate or worsen angina (anemia, obesity, hyperthyroidism)
- Reduction in coronary risk factors (hypertension, smoking, hyperlipidemia)
- Application of general and nonpharmacologic methods (lifestyle modifications)
- Pharmacologic management
  - Nitrates
  - Beta blockers
  - Calcium channel blockers
  - Antiplatelet agents
- Revascularization
  - Percutaneous transluminal coronary angioplasty with stenting
  - Coronary artery bypass grafting

BOX 4-2

Medical Management of Patients With Acute Myocardial Infarction

- Rapid hospitalization and determination of ST segment changes
- Aspirin administration
- Early thrombolytic therapy (for ST segment elevation only)
  - Streptokinase
  - Urokinase
  - Alteplase
  - Reteplase
- Early revascularization
  - Thrombolysis (for ST segment elevation only)
  - Percutaneous transluminal coronary angioplasty with stenting
  - Coronary artery bypass grafting
- Pharmacologic therapy
  - Antiplatelet drugs (glycoprotein IIa/IIIb inhibitor,[61] aspirin, clopidogrel)
  - Nitrates
  - Beta-adrenergic blockers
  - Calcium channel blockers
  - Angiotensin-converting enzyme (ACE) inhibitors
  - Lipid-lowering drugs
  - Anticoagulants (unfractionated heparin, low molecular weight heparin)
  - Morphine
  - Sedatives/hypnotics
- Oxygen

BOX 4-3

Clinical Predictors of Increased Perioperative Cardiovascular Risk (myocardial infarction [MI], heart failure, death)

MAJOR
- Unstable coronary syndromes
  - Acute or recent myocardial infarction (*) with evidence of important ischemic risk seen by clinical symptoms or noninvasive study
  - Unstable or severe angina (Canadian Class III or IV)††
- Decompensated heart failure
- Significant arrhythmias
  - High-grade atrioventricular block
  - Symptomatic ventricular arrhythmias in the presence of underlying heart disease
  - Supraventricular arrhythmias with uncontrolled ventricular rate
- Severe valvular disease

INTERMEDIATE
- Mild angina pectoris (Canadian Class I or II)†
- Previous myocardial infarction by history or pathological Q waves
- Compensated or prior heart failure
- Diabetes mellitus (particularly insulin-dependent)
- Renal insufficiency

MINOR
- Advanced age
- Abnormal ECG electrocardiogram (left ventricular hypertrophy, left bundle-branch block, ST-T abnormalities)
- Rhythm other than sinus (e.g., atrial fibrillation)
- Low functional capacity (e.g., inability to climb one flight of stairs with a bag of groceries)
- History of stroke
- Uncontrolled systemic hypertension (≥180/110 mm Hg)

BOX 4-4

Cardiac Risk* Stratification for Noncardiac Surgical Procedures

HIGH (REPORTED CARDIAC RISK OFTEN GREATER THAN 5%)
- Emergent major operations, particularly in the elderly
- Aortic and other major vascular surgery
- Peripheral vascular surgery
- Anticipated prolonged surgical procedures associated with large fluid shifts and/or blood loss

INTERMEDIATE (REPORTED CARDIAC RISK GENERALLY LESS THAN 5%)
- Carotid endarterectomy
- Head and neck surgery
- Intraperitoneal and intrathoracic surgery
- Orthopaedic surgery
- Prostate surgery

LOW (REPORTED CARDIAC RISK GENERALLY LESS THAN 1%)
- Endoscopic procedures
- Superficial procedures
- Cataract surgery

BOX 4-6

Dental Management Considerations for Patients With Unstable Angina or Recent Myocardial Infarction*

- Avoid elective care
- If treatment is necessary, consult with physician and limit treatment to pain relief, treatment of acute infection, or control of bleeding
- Consider including the following:
  - Prophylactic nitroglycerin
  - Placement of intravenous line
  - Sedation
  - Oxygen
  - Continuous electrocardiographic monitoring
  - Pulse oximeter
  - Frequent monitoring of blood pressure
  - Cautious use of epinephrine in local anesthetic, combined with above measures

*Myocardial infarction within the past 30 days.

BOX 4-5

Dental Management Considerations for Patients With Stable Angina or Past History of Myocardial Infarction*

- Morning appointments
- Short appointments
- Comfortable chair position
- Pretreatment vital signs
- Nitroglycerin readily available
- Stress-reduction measures:
  - Good communication
  - Oral sedation (e.g., triazolam 0.125- to 0.25 mg on the night before and 1 hour before the appointment)
  - Intraoperative N_2O/O_2
  - Excellent local anesthesia
- Limited use of vasoconstrictor (maximum 0.036 mg epinephrine, 0.20 mg levonordefrine); also applicable if patient is taking a nonselective beta-blocker
- Avoidance of epinephrine-impregnated retraction cord
- Antibiotic prophylaxis *not* recommended for patients with coronary artery stents
- Antibiotic prophylaxis *not* recommended for history of coronary artery bypass graft (CABG)
- Avoidance of anticholinergics (e.g., scopolamine, and atropine)
- Adequate postoperative pain control

Suggestive Reading

James W Little, Craig S Miller, Nelson L Rhodus. Dental management of medically compromised patient, 9th edition, Elsevier, 2018