VOLUME II

Current advances in oral surgery

Edited by

William B. Irby, D.D.S., M.S.

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Preface

The second volume of *Current Advances in Oral Surgery* follows the tradition established by Volume I. The heart of this tradition is to compile and update a variety of subjects applicable to the modern practice of oral and maxillofacial surgery. As the horizons of oral surgery continue to expand at an ever-increasing rate, the need to catalogue pertinent information becomes increasingly acute. The second volume again attempts to help meet this need. Some of the areas selected for emphasis in the second volume include recent developments in research; the management of patients with respiratory, cardiovascular, or neurologic problems; soft tissue management; and a study of facial pain.

The first four chapters of this book, written by physicians, present, I believe, a hallmark in oral surgery literature. Each chapter is written by an extremely well-qualified specialist emphasizing a practical and effective approach to diagnosis and therapy. The subjects are covered in depth and contain a vast amount of information of which the oral surgeon must be knowledgable. The remaining eleven chapters are written by oral surgeons and focus on concepts that concern primarily the oral surgeon. The subjects and authors have been carefully selected, and the chapters reflect the results of extensive review of the literature combined with years of practical experience.

I am certain that this second volume of *Current Advances in Oral Surgery* provides a wealth of information on the modern practice of oral surgery.

I wish to express my sincere appreciation to Mrs. Gail Stout for her assistance in the preparation of this volume. Because of her dedication, skill, and willingness to devote extra time to this endeavor, the task was made much easier.

WILLIAM B. IRBY

Contents

CHAPTER 1

Evaluation and management of patients with heart disease in oral surgery

PETER C. GAZES

Patients with cardiac disease must be evaluated prior to undergoing oral surgery, as many complications can occur during or after operation. One study[5] has shown that 38% of patients over age 35 years who were scheduled for general surgery had a history or physical evidence of heart disease, hypertension, or diabetes, and 45% had abnormal preoperative electrocardiograms (ECGs). The oral surgeon should have sufficient basic understanding of cardiovascular problems so as to be able to recognize and begin treatment of cardiac emergencies until physician consultation can be obtained. In addition, it is important to recognize when consultation should be obtained during the preoperative, operative, or postoperative periods.

CARDIOVASCULAR EXAMINATION

The cardiovascular history is extremely important. Patients with cardiac disease may complain of chest pain, dyspnea, cough, hemoptysis, palpitations, abdominal swelling or discomfort, peripheral edema, or dizziness and syncope. Questioning should be done carefully to exclude functional or noncardiac causes of symptoms. Significant chest pain is frequently substernal; may radiate to the neck, to the jaw, or down both arms (usually the left); occurs frequently with exertion; and is very difficult to describe. It usually is an oppressive, heavy, tight feeling and is not often sharp. Dyspnea during ordinary exertion is often the earliest symptom of heart failure. Paroxysmal nocturnal dyspnea may occur after the patient has been asleep for a few hours and suddenly awakens. Orthopnea refers to the type of dyspnea that occurs when the patient is recumbent. Dyspnea may be a result of lung disease or may be functional. The latter type is described as an inability to breathe well or as sighing respirations. Cough as a result of heart disease is most often noted when the patient is in the recumbent position. Hemoptysis should suggest mitral stenosis or pulmonary embolism. Palpitation is described as a sudden rapid or irregular heartbeat or a skipping, thumping, or pounding sensation. Liver congestion can produce abdominal pain. Peripheral edema may be a result of many diseases other than those of the heart. In fact, it is commonly noted with venostasis. Dizziness or syncope can be a result of cardiac disease as experienced in aortic stenosis or Adams-Stokes attacks (usually a consequence of heart block).

Physical examination of the heart is best done by inspection, palpation, and auscultation. Inspection should include a survey of the body build, skin, neck pulsations, chest

configuration and pulsations, abdomen, and extremities. The following areas should be palpated: sternoclavicular joints; second intercostal space to the right (aortic area) and to the left (pulmonic area) of the sternum; and precordial, apical, and upper abdominal areas. The first heart sound (S_1) is heard best at the apex and the second sound (S_2) at the base of the heart. The second sound may split normally with inspiration. In an individual under age 20 years, a physiologic third heart sound (S_3) may be heard in early diastole at the apex. Heart murmurs may be consequences of valvular defects of rheumatic, syphilitic, or congenital origin or of such other congenital defects as a ventricular septal defect. The most common valvular murmurs result from aortic stenosis or insufficiency and mitral stenosis or insufficiency. The systolic murmur of aortic stenosis is a very harsh ejection type and often sounds like the bark of a bulldog. It can be heard in the aortic area and along the left sternal border and often radiates to the apex. The murmur of aortic insufficiency is diastolic; is very high-pitched, like a pistol shot; and is heard best along the left sternal border. Mitral insufficiency is a high-pitched blowing regurgitant murmur, often heard throughout systole at the apex. Mitral stenosis is best heard at the apex and produces a rumbling, low-pitched diastolic murmur, often preceded by a short snapping sound (opening snap). This murmur is heard best with the bell of the stethoscope when the patient is in the left lateral recumbent position, whereas other murmurs are heard best with the diaphragm.

Murmurs are graded from 1 to 6, depending on the intensity. Innocent murmurs are seldom over grade 3 intensity. Diastolic murmurs, regardless of intensity, are usually abnormal. If the electrocardiogram and chest x-ray film are normal, the murmur may not be hemodynamically significant, but could well be organic.

For many years, emphasis has been placed on murmurs, but now heart sounds and extra sounds are equally important. Splitting of the heart sounds, ejection sounds, clicks, and gallops may give the clue to the diagnosis. Wide splitting of the second sound can occur because of any abnormality that delays the closure of the pulmonic valve, as do right bundle branch block, atrial septal defect, and pulmonic valve stenosis. Ejection sounds occur after the first heart sound and are associated with milder degrees of aortic or pulmonic stenosis and with vibrations in a dilated aortic or pulmonic root. Clicks occur in mid- or late systole and are the result of abnormalities of the chordae tendineae and dysfunction of the papillary muscle and are often associated with prolapse of one or both large, redundant, thickened, voluminous mitral leaflets into the left atrium during systole with production of a late apical systolic murmur (systolic click–murmur syndrome). An S_3 gallop (ventricular gallop) occurring in early diastole after the second heart sound indicates serious myocardial dysfunction. However, in the absence of other findings, it may be physiologic in young persons and is then referred to as a third heart sound and not a gallop. An S_4 gallop occurring before the first sound is abnormal and indicates impaired ventricular filling, but does not carry the significance of an S_3 gallop. Gallops are low-pitched sounds and resemble the cadence of a horse, whereas split sounds and ejection sounds are higher pitched.

Routinely in the preoperative period, a 12-lead ECG should be taken on all patients with a history or physical findings of cardiac disease and even in those without cardiac disease who are age 35 years or older. Often, preoperative tracings, especially in persons more than 65 years of age, will reveal changes even when the patient has no other evidence of heart disease. Nonspecific ST-T changes, bundle branch blocks, and arrhythmias are the most common findings. Although many of these patients may not require specific therapy, this information, especially the knowledge that the changes did not develop during or after surgery, is important in following the postoperative course. Exercise stress ECG (usu-

ally on a treadmill) is performed for diagnosis, prognosis, and evaluation of functional capacity. It is not performed routinely in the preoperative period.

The electromotive force of cardiac activation can be represented by a vector. The sum of all electromotive forces at any instance may be indicated by a single instantaneous vector. If all the instantaneous vectors could be plotted consecutively from a single reference point and their ends connected, a vector loop would be formed. Such loops can be plotted in the horizontal, frontal, and sagittal planes and are referred to as a spatial vectorcardiogram. Vectocardiograms only occasionally may give additional information; therefore these are taken only in certain special instances.

Routinely, four radiographic views of the chest (posteroanterior, right and left anterior oblique, and left lateral) are obtained for cardiac evaluation. Each of these views gives more information about a specific chamber of the heart.

Noninvasive techniques have become very valuable clinical tools. Phonocardiography is the graphic recording of ausculatory events. The carotid pulse, the jugular venous pulse, and the apex cardiogram are often recorded for timing of circulatory events. In addition, certain contour changes in these tracings help in the detection of hemodynamic disturbances. The apex cardiogram is a low-frequency recording of the precordial movement. Echocardiography is the recording of echoes from an ultrasonic beam returning from the cardiac structures when a transducer is positioned in different areas over the precordium. Such abnormalities as mitral stenosis, prolapse of mitral valve leaflets, atrial septal defect, and pericardial effusion can be detected by the echocardiogram.

In many instances, for diagnosis and assessment of acquired or congenital heart lesions, right and left cardiac catheterization studies must be performed. Pressures in the heart chambers and blood vessels can be measured by a catheter passed from the venous or arterial side, and cardiovascular shunts can be determined by measuring oxygen saturation and by indicator dilution curves. In addition, selective angiocardiography can be performed by injecting radiopaque agents directly into the vessels or heart chambers. Cineangiocardiography has become very popular, as has been exemplified by coronary arteriography.

PRINCIPAL CARDIAC CONDITIONS ENCOUNTERED DURING ORAL SURGERY

There are many types of cardiac problems, but only those encountered most frequently during oral surgery, for example, coronary heart disease, hypertensive cardiovascular disease, rheumatic heart disease, congenital heart disease, pulmonary heart disease, and thyroid heart disease, will be discussed. Arrhythmias and cardiac failure, which may develop in any of these conditions, will also be described. Preoperative problems will be emphasized.

Coronary heart disease

The major coronary arteries are the right and the main left, which divides into the left anterior descending and circumflex branches. The anterior descending coronary artery traverses the anterior interventricular groove and supplies the anterior surface of the heart and the septum. The circumflex artery supplies the anterolateral area of the left ventricle and, in 45% of instances, provides the branch to the sinoatrial (S-A) node. In 10% of cases, it supplies the atrioventricular (A-V) node and posterior surface of the left ventricle. The right coronary artery supplies the posterior surface of the left ventricle and septum. It provides branches to the S-A nodal artery in 55% and to the A-V node in about 90% of instances.

The coronary blood flow occurs almost exclusively during diastole. Because of the high oxygen consumption per unit of flow, there is much greater utilization, and the myocardial venous blood is only 25% to 30% saturated with oxygen, whereas peripheral venous blood is 70% saturated

at rest. The myocardium is incapable of anaerobic metabolism and cannot build up an oxygen debt to repay later, as occurs with skeletal muscle.

Coronary or ischemic heart disease, resulting from impairment of blood supply to the myocardium because of partial or complete occlusion of one or more of the coronary arteries, is the leading cause of death in the United States. The cause of the coronary vessel obstruction is not known, although evidence points to several so-called risk factors such as diet, namely, excessive cholesterol and triglyceride intake; hypertension; smoking; inactivity; diabetes; stress; and heredity.

Pathologically, the atheroma has been described as the lesion that begins in the intima, occluding the lumen of the coronary vessels. Foam cells laden with lipids, extracellular cholesterol, and fibrous tissue interlace and become soft, thus constituting the atheroma. The vessel can further be occluded by hemorrhage into this atheromatous plaque or by the development of ulceration with thrombosis and calcification.

Angina and myocardial infarction are the main clinical syndromes that result from coronary heart disease.

Angina pectoris is related to a disproportion between myocardial oxygen needs and oxygen supply. Patients often complain of substernal chest discomfort (heaviness, tightness, pressure) occurring during exertion and excitement and relieved by rest. The discomfort can radiate to the neck; to the jaw; and down both arms, especially the left arm. The physical examination and resting ECG are often normal. An ECG taken during and after exercise may demonstrate abnormalities. Coronary arteriography is reserved primarily for patients who are incapacitated because of chest pain, but in whom a specific diagnosis of angina cannot be made or for patients in whom coronary artery bypass surgery is anticipated. It must be recognized that up to 10% of patients with a history of angina and a positive stress ECG test will have normal coronary arteriograms. Some of these patients have muscle disease (cardiomyopathy) or small arteriole disease that cannot be detected by coronary arteriography.

The treatment of angina is centered on reducing the myocardial oxygen needs. Nitroglycerin is still the best drug for attacks and is taken sublingually. The initial dosage is 0.16 or 0.32 mg and is increased depending on the response. It can be taken repeatedly, but patients are advised to see their physician if, after three doses at 5 min intervals, they have not had relief. Many long-acting nitrates such as isosorbide dinitrate (Isordil) are available for preventing attacks. These drugs increase the heart rate, but lower oxygen needs by reducing blood pressure and shortening the systolic ejection time. Propranolol (Inderal) has recently become widely used, for it reduces myocardial oxygen needs and, subsequently, angina by slowing the heart rate, lowering blood pressure, and reducing the work load of the heart. However, patients have to be observed carefully because it may precipitate heart failure or asthma. Nitrate and propranolol, because of their actions, are often used together.

Angina clinically is divided into stable and unstable varieties. Stable aginia is defined as angina that has not changed its pattern over a 3-month period. In between stable angina and myocardial infarction is a syndrome that has been given many names such as preinfarction angina, impending infarction, and intermediate syndrome. Recently, the term "unstable angina" has been used for this syndrome. Unstable angina presents as crescendo angina (frequent angina occurring with very little exertion and at rest), intermediate angina (prolonged pain at rest, lasting 15 min or longer, without evidence of necrosis), or as Prinzmetal's angina (the same as intermediate angina, but the ECG reveals S-T elevation during attacks).

Patients with stable or unstable angina may be considered for coronary bypass surgery, especially if they are not responding to maximum medical therapy. The revascularization technique widely used at present

is saphenous vein bypass graft from the aorta to the coronary artery distal to the segmental coronary obstruction. In certain instances, the internal mammary artery may be used.

Coronary atherosclerosis can occlude a vessel and produce an acute myocardial infarction. In the United States, approximately 800,000 patients annually sustain an acute myocardial infarction. Such patients will have chest pain similar to angina, but more severe and usually lasting longer than 30 min. Serum enzymes are increased during these acute attacks. Serum glutamic-oxalacetic transaminase (SGOT), lactic dehydrogenase (LDH), alpha hydroxybutyrate dehydrogenase (HBD), and creatine phosphokinase (CPK) are the most commonly used enzymes. The SGOT and CPK levels rise earlier and return to normal in 3 to 5 days, whereas the LDH and HBD levels rise later and return to normal in 7 to 10 days. Changes in other organ systems can affect the enzyme levels, for example, muscle trauma can increase the CPK level, and liver disease or congestion can increase the SGOT level. Enzyme levels can also be used for evaluating the patient's prognosis. If the levels are four to five times normal, there is an increased incidence of arrhythmias, heart failure, and cardiogenic shock. There is over 50% mortality if the levels are six times normal. Specific cardiac isoenzyme measurements (MB fraction of CPK and the ratio of LDH_1 to LDH_2) may eventually replace measuring the total enzyme levels. If serial ECGs are taken, over 90% of patients with infarction will show abnormal changes.

Coronary care units have changed the morbidity and mortality of acute myocardial infarction. Arrhythmias are either prevented or treated promptly. Pump failure (shock and heart failure) still produces a high mortality, but is being more thoroughly investigated since the advent of left ventricular filling pressure measurements. A Swan-Ganz catheter can be inserted in an antecubital vein at the bedside; floated into the right atrium, right ventricle, and pulmonary artery; and then wedged into a distal pulmonary vessel. The pulmonary capillary wedge pressure measurement obtained reflects the left ventricular filling pressure. It is most important, for it indicates left ventricular function, which is most often altered in acute myocardial infarction. The central venous pressure (CVP) reflects right ventricular filling pressure and therefore is of little value in following left ventricular function. Often, the CVP can be normal, yet the pulmonary capillary wedge pressure is elevated to near pulmonary edema level. Such studies have produced new forms of drug therapy such as vasodilators and innovations such as assisted circulation (counterpulsation by use of the intra-aortic balloon).

All patients with coronary artery disease should be advised about the risk factors. In fact, evidence is sufficient at present to encourage preventive measures even in persons who have no evidence of coronary artery disease. Important among the risk factors are poor dietary habits (elevated blood lipids), cigarette smoking, hypertension, sedentary living, genetic predisposition, emotional stress, overweight, diabetes, and hyperuricemia.

In most areas, serum cholesterol, triglycerides, and lipid electrophoresis determinations can be obtained. Fredrickson's system of phenotyping has given a clearer understanding of fat metabolism and its disorders. He has described five types of such disorders. However, types 2 and 4 make up most of the abnormalities. Type 2 is divided into type 2A (hypercholesterolemia) and 2B (hypercholesterolemia and hypertriglyceridemia). Type 4 shows primarily hypertriglyceridemia. Type 2A responds to a low cholesterol, low saturated fat diet with some addition of polyunsaturated fats. In addition to a low cholesterol and low saturated fat diet, patients with type 2B or 4 should have limitations of carbohydrates. If lipid disorders are not corrected in 3 months with diet, then antilipid drugs can be added. Cholestyramine (Cuemid or Questran) is the usual choice for type 2A. Nicotinic acid and clofibrate (Atromid-S) are the drugs most often used for type 2B and 4 abnormalities.

Heavy cigarette smokers have a higher incidence of coronary artery disease and sudden death. Production of carbon monoxide with high levels of carboxyhemoglobin is probably the greatest effect of smoking, since this can lower the partial pressure of oxygen. Hypertension, diabetes, and hyperuricemia should be treated aggressively. Diabetics are more prone to abnormal lipid metabolism and coronary atherosclerosis. In addition to affecting the larger epicardial coronary arteries, diabetes may affect the smaller arterioles.

Exercise is important for both the prevention of coronary artery disease and the rehabilitation of patients with known coronary disease (angina and myocardial infarction). Exercise decreases the myocardial need for oxygen by reducing the heart rate and the peripheral vascular resistance (blood pressure). Patients should have, in addition to a complete examination, a stress test prior to beginning an exercise program. Exercise should be gradually increased, depending on the patient's symptoms and heart rate response. Kinetic exercises such as walking, jogging, swimming, and cycling are recommended rather than isometric or static exercises such as weight lifting.

Coronary heart disease accounts for most of the general operative mortality. Several studies[7, 15] have shown that patients operated on within 6 mo of an infarction have a high reinfarction rate (up to 53%) and that over 50% of patients with a prior myocardial infarction died as a result of recurrent infarction. In view of these facts, elective surgery should be postponed at least 6 months following an infarction unless a life-threatening emergency exists. Patients with a prior history of infarction should be monitored postoperatively by hardwire or telemetry for at least 48 hr either in an intensive care area or in the general care area. In addition, daily ECGs should be taken for at least 3 days, since this may be the only means of detecting a postoperative infarction, especially if the patient is having chest, arm, or shoulder pain; hypotension; arrhythmia; or dyspnea. Pain may be absent or obscured because of sedatives and narcotics, and serum enzyme levels may be altered because of skeletal muscle and liver trauma. Specific cardiac isoenzymes are more helpful. The risk of surgery in patients with angina pectoris often depends on the frequency of the angina. The risk is great in the presence of unstable angina.

Many coronary artery disease patients have diabetes. Mild glycosuria and mildly elevated blood sugar levels lessen the chances of hypoglycemia. Postoperatively, many do better on regular insulin given on a sliding scale according to the sugar levels in the urine and blood.

The dosages of oral anticoagulants should be tapered gradually over several days prior to oral surgery and not abruptly, for a rebound hypercoagulability may occur. In emergency cases, the effect of oral anticoagulants can be neutralized by intravenous vitamin K_1. Surgery is safe when the prothrombin activity is more than 40% or the prothrombin time is less than two times the control level. If the patient is taking heparin, surgery can be performed several hours after the last dose, depending on the clotting or partial thromboplastin time. If necessary, heparin can be neutralized by protamine sulfate given intravenously. Effective hemostasis should be achieved with sutures, gauze dressings, and pressure.

Local anesthesia is generally best, unless the oral surgical procedure is extensive, especially in a tense person; then general anesthesia may be indicated. Vasconstrictors allow for better anesthesia and less absorption of the local anesthetic. A distinct disadvantage associated with the use of vasoconstrictors is the possibility of systemic toxicity following the administration of drugs such as epinephrine. A number of reactions are far too frequently seen in dental and medical practice. These may include anxiety, tachycardia, palpitation, and blood pressure elevation. In extreme situations, these adverse effects may proceed to the development of pulmonary edema and ventricular fibrillation. Caution should be exercised in utilizing epinephrine combined

with a local anesthetic in elderly patients and in patients with diabetes, hyperthyroidism, hypertension, and other cardiovascular diseases. Optimal vasoconstriction can be obtained with a maximum concentration of 1:100,000 epinephrine. One milliliter of this solution contains 0.01 mg of epinephrine. Therefore the use of 4 to 6 ml of anesthetic solution containing 0.04 to 0.06 mg of epinephrine is much less than that used for cardiovascular emergencies, which range from 0.2 to 1.0 mg. Norepinephrine and phenylephrine are also used as vasoconstrictors, but are less effective than epinephrine and require two to three times the concentration. A 2% solution of Lidocaine (Xylocaine) with epinephrine 1:100,000 or a 2% solution of mepivacaine hydrochloride (Carbocaine) with Neo-Cobefrin 1:20,000 is often used today, and both are available in carpules of 1.8 ml. Mepivacaine is very similar in pharmacologic activity to lidocaine, but has a slightly longer duration of action and thus reduces the need for a vasoconstrictor. In many instances, a 3% solution of mepivacaine is used when there is a definite contraindication to the use of a vasoconstrictor.[4] The total dosage of lidocaine or mepivacaine should not be over 400 mg. Each 1.8 ml carpule of these 2% solutions has only 36 mg of the drug. Toxic reactions to both include apprehension, excitement, convulsions, drowsiness, tremor, dizziness, blurred vision, and possible respiratory arrest. Use of an aspiration syringe to avoid intravascular injection is advisable, thereby keeping the incidence of side effects and anesthetic failure at a minimum. Oxygen is the most important initial treatment for toxicity.[9]

Oral surgical procedures in cardiac patients requiring general anesthesia should be done in the hospital. Intravenous narcotics and short-acting barbiturates should not be given to such patients in the office. Morphine can produce hypotension, bradycardia, and respiratory depression. The short-acting barbiturates can produce an abrupt fall in blood pressure accompanied by apnea and can depress myocardial contractility.

Fresh nitroglycerin should be available for anginal attacks occurring in the office if the patient does not have his supply. In addition, oxygen should be available, and the oral surgeon should be familiar with the technique of cardiopulmonary resuscitation.

Hypertensive cardiovascular disease

Hypertension can cause left ventricular enlargement and eventually heart failure, cerebrovascular accidents, or kidney changes. There are conflicting opinions as to whether antihypertensive drugs alter the surgical prognosis and whether or not this therapy should be discontinued preoperatively. Mild (140/90 to 180/110 mm Hg) or moderate (180/110 to 200/120 mm Hg) hypertension is not a problem, and the drugs can be discontinued. However, in severe (200/120 mm Hg and above) hypertension, the blood pressure may rapidly rise to pretreatment levels on withdrawal of potent drug therapy and may produce a vascular catastrophe. In patients with severe hypertension, discontinuing the use of drugs preoperatively may be hazardous.

The most commonly used antihypertensive drugs are rauwolfia compounds, pargyline hydrochloride (Eutonyl), guanethidine sulfate (Ismelin sulfate), methyldopa (Aldomet), hydralazine hydrochloride (Apresoline), the diuretic compounds, and, more recently, propranolol (Inderal) and clonidine hydrochloride (Catapres).

Several years ago, the rauwolfia compounds were more widely used than they are now. Bradycardia and hypotension have occurred during or after administration of an anesthetic in patients taking these drugs. Whereas many physicians do not discontinue the use of rauwolfia therapy before surgery, I believe that it should be discontinued at least 10 days preoperatively, since it is usually given for mild to moderate hypertension. The anesthesiologist can control the side effects of rauwolfia drugs during anesthesia, but the side reactions can continue for several days postoperatively.

When discontinuing rauwolfia therapy is not feasible, the anesthesiologist should be

prepared to use a vasopressor if necessary. Since rauwolfia agents deplete the catecholamines, their hypotensive action is counteracted most effectively by direct-acting vasopressors such as phenylephrine hydrochloride (Neo-Synephrine); methoxamine hydrochloride (Vasoxyl); and, as a last alternative, *l*-norepinephrine (Levophed). Indirect-acting drugs such as ephedrine and metaraminol (Aramine) that depend on the resynthesis of norepinephrine for their action should not be used. Protracted use of vasopressors can cause egress of fluid from the intravascular compartment and produce persistence of the hypotension, which can be corrected by the use of saline or albumin solution. Parenteral administration of 1 to 2 mg of atropine sulfate preoperatively may prevent some of the rauwolfia side effects.

Pargyline, a monoamine oxidase inhibitor, may augment the hypotensive effects of anesthetic agents, surgery, and narcotics, and its administration should be discontinued for at least 10 days before elective surgery.

Both guanethidine and methyldopa are more potent than the rauwolfia compounds, and when these are withdrawn for 48 hr or more, the blood pressure usually rises. Guanethidine and methyldopa are sympathetic depressants that lower blood pressure primarily by decreasing peripheral vascular resistance. These two drugs also potentiate the action of a vasopressor; thus when their administration is continued up to surgery and hypotension develops during the procedure, the therapeutic action of the vasopressor is rapid.

Diuretic agents may deplete body potassium, sodium, chlorides, and total body water and may decrease peripheral vascular resistance. Potassium deficit sensitizes the heart to the toxic effects of digitalis and predisposes to arrhythmias. Body potassium may be depleted in the presence of a normal serum potassium level and may be aggravated by glucose given intravenously. Metabolic alkalosis predisposes to cardiopulmonary arrest. Hypotension can occur during anesthesia when the blood volume is contracted secondary to hyponatremia. Diuretics also reduce the vascular reactivity to catecholamines. Spironolactone (Aldactone) can produce hyperkalemia and cardiac arrest. Before operation, the blood volume should be restored, and any electrolyte imbalance should be corrected.

Propranolol (a beta adrenergic blocking agent) should be withdrawn gradually over several days, because sudden withdrawal may be associated with increasing angina, arrhythmias, or myocardial infarction.[1, 8] Sudden withdrawal of clonidine, which decreases sympathetic outflow from vasomotor centers in the brain, may be associated with a sharp rebound of the blood pressure to pretreatment levels or higher, which would be undesirable before or after anesthesia. Clonidine should be reduced gradually over a period of 2 to 4 days prior to elective surgery.[11]

When the antihypertensive agents have been discontinued or the patient has severe hypertension and has not received drugs, the blood pressure can be controlled by sodium nitroprusside (Nipride) or trimethaphan camphorsulfonate (Arfonad). Sodium nitroprusside acts directly on the smooth muscle of the blood vessels, resulting in peripheral vascular dilatation. A 50 mg vial in 500 ml of 5% dextrose in water constitutes a solution of 100 μg/ml. The range of dosage is usually 0.5 to 8.0 μg/kg/min. Trimethaphan camphorsulfonate is a ganglionic blocking agent and peripheral vasodilator and causes pooling of the blood in the dependent periphery and the splanchnic system; thus in addition to lowering blood pressure, it prevents pulmonary edema. The drug is usually given by intravenous drip (500 mg in 500 ml of glucose in water), beginning with 30 drops/min and then adjusting the amount to individual requirements. Slowing the rate of administration reduces the effect of the drug very quickly.

The phenothiazine tranquilizers such as chlorpromazine (Thorazine), prochlorperazine (Compazine), and other related drugs are cardiotoxic (associated with ECG

changes, myocardial infarction, and sudden death) and can produce hypotension.[12] The same effects can also occur with the antidepressants such as imipramine hydrochloride (Tofranil), amitriptyline hydrochloride (Elavil hydrochloride), and desipramine (Pertofrane). Whenever possible, the use of these drugs should be discontinued long before anesthesia is administered.

Rheumatic heart disease

Rheumatic fever, although its incidence has decreased, is still responsible for most acquired valvular disease. It is related to prior infection with group A beta hemolytic streptococci. The exact mechanism has not been clarified, although a hypersensitivity reaction probably is a factor. A typical history of a preceding streptococcal throat infection occurring 1 to 4 weeks prior to the onset of acute rheumatic fever may be obtained. Carditis is a very important finding in rheumatic fever. The mitral and aortic valves are most frequently involved. After the initial edema, the valves, over a variable time period, may become scarred, thickened, calcified, and retracted with resultant stenosis, insufficiency, or both. When closure of the scarred mitral valve with thicker and shorter chordae tendineae is incomplete, mitral insufficiency will develop. A holosystolic murmur can be noted at the apex. Cardiac enlargement, namely, of the left atrium and ventricle, will occur depending on the degree of regurgitated blood volume. Eventually, atrial fibrillation and heart failure can develop. During the past several years, many nonrheumatic types of mitral insufficiency have been recognized. Rheumatic fever is recognized as the cause only if a definite history can be obtained or if there is also aortic valve involvement. The fibrotic changes of the mitral valve can produce stenosis blocking the inflow of blood into the left ventricle. This mitral stenosis will produce a low-pitched, rumbling diastolic murmur at the apex that is often preceded by a snapping, high-pitched sound (opening snap). Depending on the degree of stenosis, left artial enlargement, pulmonary hypertension, right ventricular enlargement, atrial fibrillation, and eventually heart failure can develop. Mitral valve disease can lead to systemic emboli. An incompetent aortic valve, allowing blood to flow back into the left ventricle during diastole, will produce a high-pitched aortic diastolic murmur heard best in the aortic area and along the left sternal border. There are many nonrheumatic causes of aortic insufficiency. Obstruction caused by aortic stenosis produces a harsh ejection systolic murmur that is heard best in the aortic area, along the left sternal border, and, at times, down to the apex. Left ventricular enlargement can occur with aortic valvular lesions that can lead to heart failure.

Patients with valvular heart disease whose cardiac status is uncompromised (class I) or slightly compromised (class II) usually tolerate oral surgery as well as do noncardiac patients. Patients whose cardiac status is moderately (class III) or severely compromised (class IV), especially those who have aortic or mitral stenosis, often have complications such as pulmonary edema and are more likely to suffer sudden death. Such patients do better having cardiac surgery prior to elective oral surgery. Patients with valvular disease or prosthetic valves who are undergoing oral surgical procedures should receive antibiotic prophylaxis against bacterial endocarditis (Table 1). Rheumatic fever (antistreptococcal) prophylaxis should not be confused with prophylaxis against bacterial endocarditis, which requires extra antibiotic protection.

Bacterial endocarditis often occurs in patients known to have rheumatic heart disease and usually when there are mild degrees of mitral or aortic insufficiency. It rarely occurs in patients with mitral stenosis and in the presence of atrial fibrillation. The portal of entry may give a clue to the causative organism. Viridans streptococcus infections occur frequently after oral surgical procedures. Unexplained fever for more than 7 days after surgery in a patient with a heart murmur should be considered to be a result of bacterial endocarditis until proved other-

Table 1. Prophylaxis against bacterial endocarditis for oral surgical procedures*†

	Before procedure	*After procedure*
Nonallergic patients	600,000 units of procaine penicillin G mixed with 200,000 units of crystalline penicillin G intramuscularly 1 hr before procedure	Same intramuscularly daily for 2 days, or longer if healing is delayed
	500 mg of penicillin V or phenethicillin orally 1 hr before procedure	250 mg every 6 hr orally for 2 days, or longer if healing is delayed
	1.2 million units of penicillin G orally 1 hr before procedure	600,000 units orally every 6 hr for 2 days, or longer if healing is delayed
Patients allergic to penicillin	500 mg of erythromycin orally 1½ hr before procedure	250 mg orally every 6 hr for 2 days, or longer if healing is delayed

*From Prevention of bacterial endocarditis, Dallas, 1972, The Rheumatic Fever Committee and the Committee on Congenital Cardiac Defects of the American Heart Association; copyright 1972 American Heart Association.

†After cardiac surgery, the same precautions should be taken, especially in patients with prosthetic valves. Patients with corrected atrial septal defect or with surgically closed patent ductus arteriosus require no prophylaxis.

wise. Blood cultures should be done to identify the organism. Delay in therapy of bacterial endocarditis may produce severe cardiac damage, heart failure, and emboli.

Congenital heart disease

Common noncyanotic congenital heart lesions with left-to-right shunts are ventricular septal defect, atrial septal defect, and patent ductus arteriosus. Tetralogy of Fallot, tricuspid atresia, and reversed shunts because of pulmonary hypertension are common defects producing right-to-left shunts and cyanosis. Pulmonary and aortic stenosis and coarctation of the aorta are common obstructive defects. Diagnosis of these various defects depends on the history (heart murmur, cyanosis, or both at birth), physical findings, x-ray findings, ECG, echocardiogram, and, often, heart catheterization with angiocardiographic studies.

Patients who have noncyanotic congenital heart lesions without evidence of heart failure can tolerate oral surgery. Cyanotic patients are at greater risk. Problems of postoperative hemorrhage, vascular thrombosis, and profound anoxia are often encountered in the cyanotic patient. Patients with congenital heart disease should receive antibiotic prophylaxis against bacterial endocarditis (same schedule as for valvular heart disease). Patients with mid- or late systolic clicks (because of prolapse of mitral valve) or ejection sounds (because of congenital aortic or pulmonic valve stenosis), with or without associated murmurs, should also received bacterial endocarditis prophylaxis.

Antibiotic prophylaxis for prevention of bacterial endocarditis is not necessary for patients with coronary artery disease, hypertensive vascular disease, pulmonary heart disease, cardiac muscle disease such as that secondary to thyroid disease or for patients with surgically corrected atrial septal defect or surgically closed patent ductus arteriosus.

Pulmonary heart disease

Pulmonary heart disease (cor pulmonale) is a condition in which hypertrophy and dilatation of the right ventricle, with or without failure, results from certain diseases affecting the function and/or structure of the lungs. Chronic airway obstruction (emphysema, bronchitis, or combinations) or extensive fibrotic diseases of the lung eventually, by producing pulmonary hypertension, can cause right ventricular dilatation and hypertrophy and cor pulmonale.

Patients with lung disease should have pulmonary function studies done in addition to a cardiac evaluation. Anesthetic problems and postoperative complications occur more often in patients with pulmonary