CHAPTER 21

Anesthesia for Patients with Cardiovascular Disease

KEY CONCEPTS

1. Cardiovascular complications account for 25% to 50% of deaths following noncardiac surgery. Perioperative myocardial infarction (MI), pulmonary edema, congestive heart failure, arrhythmias, and thromboembolism are most commonly seen in patients with preexisting cardiovascular disease.
2. Regardless of the level of preoperative blood pressure control, many patients with hypertension display an accentuated hypotensive response to induction of anesthesia, followed by an exaggerated hypertensive response to intubation. Hypertensive patients may display an exaggerated response to both endogenous catecholamines (from intubation or surgical stimulation) and exogenously administered sympathetic agonists.
3. Patients with extensive (three-vessel or left main) coronary artery disease, a history of MI, or ventricular dysfunction are at greatest risk of cardiac complications.
4. Holter monitoring, exercise electrocardiography, myocardial perfusion scans, and echocardiography are important in determining perioperative risk and the need for coronary angiography; however, these tests are indicated only if their outcome would alter patient care.
5. Sudden withdrawal of antianginal medication perioperatively—particularly β-blockers—can precipitate a sudden, rebound increase in ischemic episodes.
6. The overwhelming priority in managing patients with ischemic heart disease is maintaining a favorable myocardial supply–demand relationship. Autonomic-mediated increases in heart rate and blood pressure should be controlled by deep anesthesia or adrenergic blockade, and excessive reductions in coronary perfusion pressure or arterial oxygen content are to be avoided.
7. Intraoperative detection of ischemia depends on recognition of electrocardiographic changes, hemodynamic manifestations, or regional wall motion abnormalities on transesophageal echocardiography. New ST-segment elevations are rare during noncardiac surgery and are indicative of severe ischemia, vasospasm, or infarction.
8. The principal hemodynamic goals in managing mitral stenosis are to maintain a sinus rhythm (if present preoperatively) and to avoid tachycardia, large increases in cardiac output, and both hypovolemia and fluid overload by judicious administration of intravenous fluids.
9. Anesthetic management of mitral regurgitation should be tailored to the severity of regurgitation and to the underlying left ventricular function. Factors that exacerbate the regurgitation, such as slow heart rates and acute increases in afterload, should be avoided. Excessive volume expansion can also worsen the regurgitation by dilating the left ventricle.

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10. Maintenance of normal sinus rhythm, heart rate, vascular resistance and intravascular volume is critical in patients with aortic stenosis. Loss of a normally timed atrial systole often leads to rapid deterioration, particularly when associated with tachycardia. Spinal and epidural anesthesia are relatively contraindicated in patients with severe aortic stenosis.
11. Bradycardia and increase in systemic vascular resistance (SVR) increase the regurgitant volume in patients with aortic regurgitation, whereas tachycardia can contribute to myocardial ischemia. Excessive myocardial depression should also be avoided. The compensatory increase in cardiac preload should be maintained, but excessive fluid replacement can readily result in pulmonary edema.
12. In patients with congenital heart disease, an increase in SVR relative to pulmonary vascular resistance (PVR) favors left-to-right shunting, whereas an increase in PVR relative to SVR favors right-to-left shunting.
13. The presence of shunt flow between the right and left hearts, regardless of the direction of blood flow, mandates the meticulous exclusion of air bubbles or particulate material from intravenous fluids to prevent paradoxical embolism into the cerebral or coronary circulations.
14. The goals of anesthetic management in patients with tetralogy of Fallot should be to maintain intravascular volume and SVR. Increases in PVR, such as might occur from acidosis or excessive airway pressures, should be avoided. The right-to-left shunting tends to slow the uptake of inhalation anesthetics; in contrast, it may accelerate the onset of intravenous agents.
15. The transplanted heart is totally denervated, so direct autonomic influences are absent. Moreover, the absence of reflex increases in heart rate can make patients particularly sensitive to rapid vasodilatation. Indirect vasopressors, such as ephedrine, are less effective than direct-acting agents because of the absence of catecholamine stores in myocardial neurons.

Cardiovascular diseases—particularly hypertensive, ischemic, congenital, and valvular heart disease—are among the medical illnesses most frequently encountered in anesthetic practice and are a major cause of perioperative morbidity and mortality. Management of patients with these diseases continues to challenge the ingenuity and resources of the anesthesiologist. The adrenergic response to surgical stimulation and the circulatory effects of anesthetic agents, endotracheal intubation, positive-pressure ventilation, blood loss, fluid shifts, and alterations in body temperature impose additional burdens on an often already compromised cardiovascular system. Most anesthetic agents cause cardiac depression, vasodilatation, or both. Even anesthetics that have no direct circulatory effects may cause apparent circulatory depression in severely compromised patients who are dependent on the enhanced sympathetic activity characteristic of heart failure or acute blood loss. Decreased sympathetic activity as a consequence of the anesthetized state can lead to acute circulatory collapse.

Good anesthetic management of patients with cardiovascular disease requires a thorough knowledge of normal cardiac physiology, the circulatory effects of the various anesthetic agents, and the pathophysiology and treatment of these diseases. The same principles used in treating cardiovascular diseases in patients not undergoing surgery should be used perioperatively. In most instances, the choice of anesthetic agent is not terribly important; on the other hand, knowing how the agent is used, understanding the underlying pathophysiology, and understanding how the two interact are critical.

Patients with severe cardiovascular illnesses commonly undergo both cardiac and noncardiac surgery. The American College of Cardiology (ACC), in collaboration with the American Heart Association (AHA), have issued numerous guidelines related to the management of patients with heart disease, and many of their recommendations are relevant to patients undergoing anesthesia and invasive procedures. Because guidelines change as new evidence becomes available, anesthesiologists are advised to review the AHA website for current evidence-based indications for the management of heart disease.

Perioperative Cardiovascular Evaluation and Preparation for Noncardiac Surgery

The prevalence of cardiovascular disease increases progressively with advancing age. Moreover, the number of patients over 65 years of age is expected to increase by 25% to 35% over the next two decades. Cardiovascular complications account for 25% to 50% of deaths following noncardiac surgery. [1] Perioperative myocardial infarction (MI), pulmonary edema, systolic and diastolic heart failure, arrhythmias, and thromboembolism are the most common diagnoses in patients with preexisting cardiovascular disease. The incidence of postoperative cardiogenic pulmonary edema is approximately 2% in all patients over 40 years of age, but it is 6% in patients with a history of heart failure and 16% in patients with poorly compensated heart failure. The relatively high prevalence of cardiovascular disorders in surgical patients has given rise to attempts to define *cardiac risk* or the likelihood of intraoperative or postoperative fatal or life-threatening cardiac complications.

In 2007, the ACC/AHA Task Force Report produced revised guidelines for perioperative evaluation. The revised guidelines stated that the patient's medical history is critical in determining the requirements for preoperative cardiac evaluation and that certain conditions (eg, unstable coronary syndromes and decompensated heart failure) warrant cardiology intervention prior to all but emergency procedures (Table 21–1). The history should also review any past procedures, such as cardioverter defibrillator implants, coronary stents, and other interventions.

TABLE 21–1 Active cardiac conditions for which the patient should undergo evaluation and treatment before noncardiac surgery (class I, level of evidence: B).

Condition	Examples
Unstable coronary syndromes	Unstable or severe angina[1] (CCS class III or IV)[2] Recent MI[3]
Decompensated HF (NYHA functional class IV; worsening or new-onset HF)	
Significant arrhythmias	High-grade atrioventricular block Mobitz II atrioventricular block Third-degree atrioventricular heart block Symptomatic ventricular arrhythmias Supraventricular arrhythmias (including atrial fibrillation) with uncontrolled ventricular rate (HR greater than 100 bpm at rest) Symptomatic bradycardia Newly recognized ventricular tachycardia
Severe valvular disease	Severe aortic stenosis (mean pressure gradient greater than 40 mm Hg, aortic valve area less than 1.0 cm^2, or symptomatic) Symptomatic mitral stenosis (progressive dyspnea on exertion, exertional presyncope, or HF)

CCS indicates Canadian Cardiovascular Society; HF, heart failure; HR, heart rate; MI, myocardial infarction; NYHA, New York Heart Association.

[1]According to Campeau L. Letter: Grading of angina pectoris. Circulation. 1976;54:522-523.

[2]May include "stable" angina in patients who are unusually sedentary.

[3]The American College of Cardiology National Database Library defines recent MI as more than 7 days but less than or equal to 1 month (within 30 days).

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TABLE 21–2 Estimated energy requirements for various activities.

	Can you ...		Can you ...
1 MET	Take care of yourself?	4 METs	Climb a flight of stairs or walk up a hill?
↓	Eat, dress, or use the toilet?	↓	Walk on level ground at 4 mph (6.4 kph)?
	Walk indoors around the house?		Run a short distance?
	Walk a block or 2 on level ground at 2 to 3 mph (3.2 to 4.8 kph)?		Do heavy work around the house like scrubbing floors or lifting or moving heavy furniture?
4 METs	Do light work around the house like dusting or washing dishes?		Participate in moderate recreational activities like golf, bowling, dancing, doubles tennis, or throwing a baseball or football?
		Greater than 10 METs	Participate in strenuous sports like swimming, singles tennis, football, basketball, or skiing?

kph indicates kilometers per hour; MET, metabolic equivalent; and mph, miles per hour.

Modified and reproduced, with permission, from Hlatky MA, Boineau RE, Higginbotham MB, et al. A brief self-administered questionnaire to determine functional capacity (the Duke Activity Status Index). Am J Cardiol. 1989;64:651-654.

Additionally, the patient's ability to perform the tasks of daily living should be assessed as a guide to determine functional capacity. A patient with a history of cardiac disease and advanced age, but good exercise tolerance, will likely have a lower perioperative risk than a similar individual with dyspnea after minimal physical activity (Table 21–2).

The patient's history should also seek signs of other disease processes that frequently accompany heart disease. Cardiac patients often present with obstructive pulmonary disease, reduced renal function, and diabetes mellitus.

A physical examination should be performed on all patients, and the heart and lungs should be auscultated. The physical examination is especially useful in patients with certain conditions. For example, if a murmur suggestive of aortic stenosis is detected, additional ultrasound evaluation will likely be warranted, as aortic stenosis substantially increases the risks in patients undergoing noncardiac surgery.

The following conditions are associated with increased risk:

- Ischemic heart disease (history of MI, evidence on electrocardiogram [ECG], chest pain)
- Congestive heart failure (dyspnea, pulmonary edema)
- Cerebral vascular disease (stroke)
- High-risk surgery (vascular, thoracic, abdominal, orthopedic)
- Diabetes mellitus
- Preoperative creatinine >2 mg/dL

Recent ACC/AHA guidelines identify conditions that are a major cardiac risk and warrant intensive management prior to all but emergent surgery. These conditions include: unstable coronary syndromes (recent MI, unstable angina), decompensated heart failure, significant arrhythmias, and severe valvular heart disease. The ACC/AHA guidelines identify an MI within 7 days, or one within 1 month with myocardium at risk for ischemia, as "active" cardiac conditions. On the other hand, evidence of past MI with no myocardium thought at ischemic risk is considered a low risk for perioperative infarction after noncardiac surgery.

The ACC/AHA guidelines suggest a stepwise approach to preoperative cardiac evaluation. Their recommendations are classified as follows:

- Class I: Benefits >> risk
- Class IIa: Benefits >> risk, but scientific evidence incomplete

- Class IIb: Benefits ≥ risk, and scientific evidence incomplete
- Class III: Risks >>benefits

Class I recommendations are as follows:

- Patients who have a need for emergency noncardiac surgery should proceed to the operating room with perioperative surveillance and postoperative risk factor management
- Patients with active cardiac conditions should be evaluated by a cardiologist and treated according to ACC/AHA guidelines
- Patients undergoing low-risk procedures should proceed to surgery
- Patients with poor exercise tolerance (<4 metabolic equivalents [METs]) and no known risk factors should proceed to surgery

Class IIa recommendations are as follows:

- Patients with a functional capacity >4 METs and without symptoms should proceed to surgery
- Patients with a functional capacity <4 METs or those with an unknown functional capacity with three or more clinical risk factors scheduled for vascular surgery should be tested, if management is likely to change based on the results
- Patients with a functional capacity <4 METs or those with an unknown functional capacity with three or more clinical risk factors scheduled for intermediate-risk surgery should proceed to surgery with heart rate control
- Patients with a functional capacity <4 METs or those with an unknown functional capacity with one or two clinical risk factors who are scheduled for vascular or intermediate-risk surgery should proceed to surgery with heart rate control

The ACC/AHA guidelines also note, as class IIb recommendations, that noninvasive testing might be considered *if* patient management changes in patients with poor or unknown functional capacity or in patients undergoing intermediate-risk surgery with three clinical risk factors. Likewise, such testing might be indicated in patients with one or two clinical risk factors scheduled for vascular or intermediate-risk surgery. Table 21–3 classifies surgical procedures according to risk.

TABLE 21–3 Cardiac risk[1] stratification for noncardiac surgical procedures.

Risk Stratification	Procedure Examples
Vascular (reported cardiac risk often more than 5%)	Aortic and other major vascular surgery Peripheral vascular surgery
Intermediate (reported cardiac risk generally 1% to 5%)	Intraperitoneal and intrathoracic surgery Carotid endarterectomy Head and neck surgery Orthopedic surgery Prostate surgery
Low[2] (reported cardiac risk generally less than 1%)	Endoscopic procedures Superficial procedure Cataract surgery Breast surgery Ambulatory surgery

[1]Combined incidence of cardiac death and nonfatal myocardial infarction.

[2]These procedures do not generally require further preoperative cardiac testing.

Fleisher L, Beckman J, Brown K, et al: ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery. Circulation 2007;116:1971-1996.

The ACC/AHA guidelines also provide specific recommendations regarding various conditions likely to be encountered perioperatively.

CORONARY ARTERY DISEASE

The ACC/AHA guidelines note that only the subset of patients with coronary artery disease (CAD) who would benefit from revascularization, irrespective of their need for a nonemergent surgical procedure, would likely benefit from preoperative coronary interventions. Consequently, the indications for the testing of such patients as candidates for a coronary intervention are unrelated to their presenting for surgery and depend only on whether such evaluation would be indicated as part of general medical management.

HYPERTENSION

Patients with hypertension frequently present for elective surgical procedures. Some will have been effectively managed, but unfortunately, many others will not have been. Hypertension is a leading cause of death and disability in most Western societies and the most prevalent preoperative medical abnormality in surgical patients, with an overall prevalence of 20% to 25%. Long-standing uncontrolled hypertension accelerates atherosclerosis and hypertensive organ damage. Hypertension is a major risk factor for cardiac, cerebral, renal, and vascular disease. **Complications of hypertension include MI, congestive heart failure, stroke, renal failure, peripheral occlusive disease, and aortic dissection**. The presence of left ventricular hypertrophy (LVH) in hypertensive patients may be an important predictor of cardiac mortality. However, systolic blood pressures below 180 mm Hg, and diastolic pressures below 110 mm Hg, have not been associated with increased perioperative risks. When patients present with systolic blood pressures greater than 180 mm Hg and diastolic pressures greater than 110 mm Hg, anesthesiologists face the dilemma of delaying surgery to allow optimization of oral antihypertensive therapy, but adding the risk of a surgical delay versus proceeding with surgery and achieving blood pressure control with rapidly acting intravenous agents. Intravenous β-blockers can be useful to treat preoperative hypertension. Of note, patients with preoperative hypertension are more likely than others to develop intraoperative hypotension. This is particularly frequent in patients treated with angiotensin receptor blockers and/or angiotensin-converting enzyme (ACE) inhibitors.

Blood pressure measurements are affected by many variables, including posture, time of day or night, emotional state, recent activity, and drug intake, as well as the equipment and technique used. A diagnosis of hypertension cannot be made by one preoperative reading, but requires confirmation by a history of consistently elevated measurements. Although preoperative anxiety or pain may produce some degree of hypertension in normal patients, patients with a history of hypertension generally exhibit greater preoperative elevations in blood pressure.

TABLE 21–4 Classification of blood pressure (adults).

Category of Blood Pressure	Systolic Pressure (mm Hg)	Diastolic Pressure (mm Hg)
Normal	<130	<85
High normal	130–139	85–89
Hypertension		
Stage 1/mild	140–159	90–99
Stage 2/moderate	160–179	100–109
Stage 3/severe	180–209	110–119
Stage 4/very severe	>210	>120

Epidemiological studies demonstrate a direct and continuous correlation between both diastolic and systolic blood pressures and mortality rates. The definition of systemic hypertension is arbitrary: a consistently elevated diastolic blood pressure greater than 90 mm Hg or a systolic pressure greater than 140 mm Hg. A common classification scheme is listed in Table 21–4. Borderline hypertension is said to exist when the diastolic pressure is 85–89 mm Hg or the systolic pressure is 130–139 mm Hg. Whether patients with borderline hypertension are at some increased risk for cardiovascular complications remains unclear. Accelerated, or severe hypertension (stage 3), is defined as a recent, sustained, and progressive increase in blood pressure, usually with diastolic blood pressures in excess of 110–119 mm Hg. Renal dysfunction is often present in such patients. Malignant hypertension is a true medical emergency characterized by severe hypertension (>210/120 mm Hg) often associated with papilledema and encephalopathy.

Pathophysiology

Hypertension can be either idiopathic (essential), or, less commonly, secondary to other medical conditions such as renal disease, renal artery stenosis, primary hyperaldosteronism, Cushing's disease, acromegaly, pheochromocytoma, pregnancy, or estrogen therapy. Essential hypertension accounts

for 80% to 95% of cases and may be associated with an abnormal baseline elevation of cardiac output, systemic vascular resistance (SVR), or both. An evolving pattern is commonly seen over the course of the disease, where cardiac output returns to (or remains) normal, but SVR becomes abnormally high. The chronic increase in cardiac afterload results in concentric LVH and altered diastolic function. Hypertension also alters cerebral autoregulation, such that normal cerebral blood flow is maintained in the face of high blood pressures; autoregulation limits may be in the range of mean blood pressures of 110–180 mm Hg.

The mechanisms responsible for the changes observed in hypertensive patients seem to involve vascular hypertrophy, hyperinsulinemia, abnormal increases in intracellular calcium, and increased intracellular sodium concentrations in vascular smooth muscle and renal tubular cells. The increased intracellular calcium presumably results in increased arteriolar tone, whereas the increased sodium concentration impairs renal excretion of sodium. Sympathetic nervous system overactivity and enhanced responses to sympathetic agonists are present in some patients. Hypertensive patients sometimes display an exaggerated response to vasopressors and vasodilators. Overactivity of the renin–angiotensin–aldosterone system seems to play an important role in patients with accelerated hypertension.

Long-Term Treatment

Effective drug therapy reduces the progression of hypertension and the incidence of stroke, congestive heart failure, CAD, and renal damage. Effective treatment can also delay and sometimes reverse concomitant pathophysiological changes, such as LVH and altered cerebral autoregulation.

Some patients with mild hypertension require only single-drug therapy, which may consist of a thiazide diuretic, ACE inhibitor, angiotensin-receptor blocker (ARB), β-adrenergic blocker, or calcium channel blocker, although guidelines and outcome studies favor the first three options. Concomitant illnesses should guide drug selection. All patients with a prior MI should receive a β-adrenergic blocker and an ACE inhibitor (or ARB) to improve outcomes, irrespective of the presence of hypertension. In many patients, the "guideline specified" agents will also be more than sufficient to control hypertension.

Patients with moderate to severe hypertension often require two or three drugs for control. The combination of a diuretic with a β-adrenergic blocker and an ACE inhibitor is often effective when single-drug therapy is not. As previously noted, ACE inhibitors (or ARBs) prolong survival in patients with congestive heart failure, left ventricular dysfunction, or a prior MI. Familiarity with the names, mechanisms of action, and side effects of commonly used antihypertensive agents is important for anesthesiologists (**Table 21–5**).

PREOPERATIVE MANAGEMENT

A recurring question in anesthetic practice is the degree of preoperative hypertension that is acceptable for patients scheduled for elective surgery. Except for optimally controlled patients, most hypertensive patients present to the operating room with some degree of hypertension. Although data suggest that even moderate preoperative hypertension (diastolic pressure <90–110 mm Hg) is not clearly statistically associated with *postoperative* complications, other data indicate that the untreated or poorly controlled hypertensive patient is more apt to experience *intraoperative* episodes of myocardial ischemia, arrhythmias, or both hypertension and hypotension. Intraoperative adjustments in anesthetic depth and use of vasoactive drugs should reduce the incidence of postoperative complications referable to poor preoperative control of hypertension.

Although patients should ideally undergo elective surgery only when rendered normotensive, this is not always feasible or necessarily desirable because of altered cerebral autoregulation. Excessive reductions in blood pressure can compromise cerebral perfusion. Moreover, the decision to delay or to proceed with surgery should be individualized, based on the severity of the preoperative blood pressure elevation; the likelihood of coexisting myocardial ischemia, ventricular dysfunction, or cerebrovascular or renal complications; and the surgical procedure (whether major

TABLE 21–5 Oral antihypertensive agents.

Category	Class	Subclass	Agent
Diuretics	Thiazide		Chlorothiazide (Diuril) Chlorthalidone (Thalitone) Hydrochlorothiazide (Microzide) Indapamide (Lozol) Metolazone (Zaroxolyn)
	Potassium sparing		Spironolactone (Aldactone) Triamterene (Dyrenium) Amiloride (Midamor)
	Loop		Bumetanide (Bumex) Ethacrynic acid (Edecrin) Furosemide (Lasix) Torasemide (Demadex)
Sympatholytics	Adrenergic-receptor blockers	B	Acebutolol (Sectral) Atenolol (Tenormin) Betaxolol (Kerlone) Bisoprolol (Zebeta) Carteolol (Cartrol) Metoprolol (Lopressor) Nadolol (Corgard) Penbutolol (Levatol) Pindolol (Visken) Propranolol (Inderal) Timolol (Blocadren)
		A	α_1 Doxazosin (Cardura) Prazosin (Minipress) Terazosin (Hytrin) $\alpha_1+\alpha_2$ Phenoxybenzamine (Dibenzyline)
		α and β	Labetalol (Trandate) Carvedilol (Coreg)
	Central α_2-agonists		Clonidine (Catapres) Guanabenz (Wytensin) Guanfacine (Tenex) Methyldopa (Aldomet)

(*continued*)

surgically induced changes in cardiac preload or afterload are anticipated). With rare exceptions, antihypertensive drug therapy should be continued up to the time of surgery. Some clinicians withhold ACE inhibitors and ARBs on the morning of surgery because of their association with an increased incidence of intraoperative hypotension; however, withholding these agents increases the risk of marked perioperative hypertension and the need for parenteral antihypertensive agents. It also requires the surgical team to remember to restart the medication after surgery. The decision to delay elective surgical procedures in patients with sustained preoperative diastolic blood pressures higher than 110 mm Hg should be made when the perceived benefits of delayed surgery exceed the risks. Unfortunately, there are few appropriate studies to guide the decision-making.

History

The preoperative history should inquire into the severity and duration of the hypertension, the

TABLE 21–5 Oral antihypertensive agents. (continued)

Category	Class	Subclass	Agent
Vasodilators	Calcium channel blockers	Benzothiazepine	
			Diltiazem[1] (Tiazac)
		Phenylalkylamines	
			Verapamil[1] (Calan SR)
		Dihydropyridines	
			Amlodipine (Norvasc)
			Felodipine (Plendil)
			Isradipine[1] (Dynacirc)
			Nicardipine[1] (Cardene)
			Nifedipine[1] (Procardia XL)
			Nisoldipine (Sular)
		ACE inhibitors[2]	Benazepril (Lotensin)
			Captopril (Capoten)
			Enalapril (Vasotec)
			Fosinopril (Monopril)
			Lisinopril (Zestril)
			Moexipril (Univasc)
			Perindopril (Aceon)
			Quinapril (Accupril)
			Ramipril (Altace)
			Trandopril (Mavik)
		Angiotensin-receptor antagonists	Candesartan (Atacand)
			Eprosartan (Tevetan)
			Irbesartan (Avapro)
			Losartan (Cozaar)
			Olmesartan (Benicar)
			Telmisartan (Micardis)
			Valsartan (Diovan)
	Direct vasodilators		Hydralazine (Apresoline)
			Minoxidil

[1]Extended release.

[2]ACE, angiotensin-converting enzyme.

drug therapy currently prescribed, and the presence or absence of hypertensive complications. Symptoms of myocardial ischemia, ventricular failure, impaired cerebral perfusion, or peripheral vascular disease should be elicited, as well as the patient's record of compliance with the drug regimen. The patient should be questioned regarding chest pain, exercise tolerance, shortness of breath (particularly at night), dependent edema, postural lightheadedness, syncope, episodic visual disturbances or episodic neurologic symptoms, and claudication. Adverse effects of current antihypertensive drug therapy (Table 21–6) should also be identified.

Physical Examination & Laboratory Evaluation

Ophthalmoscopy is useful in hypertensive patients. Visible changes in the retinal vasculature usually parallel the severity and progression of arteriosclerosis and hypertensive damage in other organs. An S_4 cardiac gallop is common in patients with LVH. Other physical findings, such as pulmonary rales and an S_3 cardiac gallop, are late findings and indicate congestive heart failure. Blood pressure can be measured in both the supine and standing positions. Orthostatic changes can be due to volume depletion, excessive vasodilatation, or sympatholytic drug

TABLE 21–6 Adverse effects of long-term antihypertensive therapy.

Class	Adverse Effects
Diuretics	
Thiazide	Hypokalemia, hyponatremia, hyperglycemia, hyperuricemia, hypomagnesemia, hyperlipidemia, hypercalcemia
Loop	Hypokalemia, hyperglycemia, hypocalcemia, hypomagnesemia, metabolic alkalosis
Potassium sparing	Hyperkalemia
Sympatholytics	
β-Adrenergic blockers	Bradycardia, conduction blockade, myocardial depression, enhanced bronchial tone, sedation, fatigue, depression
α-Adrenergic blockers	Postural hypertension, tachycardia, fluid retention
Central α_2-agonists	Postural hypotension, sedation, dry mouth, depression, decreased anesthetic requirements, bradycardia, rebound hypertension, positive Coombs test and hemolytic anemia (methyldopa), hepatitis (methyldopa)
Ganglionic blockers	Postural hypotension, diarrhea, fluid retention, depression (reserpine)
Vasodilators	
Calcium channels blockers	Cardiac depression, bradycardia, conduction blockade (verapamil, diltiazem), peripheral edema (nifedipine), tachycardia (nifedipine), enhanced neuromuscular nondepolarizing blockade
ACE inhibitors[1]	Cardiac depression, bradycardia, conduction blockade (verapamil, diltiazem), peripheral edema (nifedipine), tachycardia (nifedipine), enhanced neuromuscular nondepolarizing blockade
Angiotensin-receptor antagonists	Hypotension, renal failure in bilateral renal artery stenosis, hyperkalemia
Direct vasodilators	Reflex tachycardia, fluid retention, headache, systemic lupus erythematosus-like syndrome (hydralazine), pleural or pericardial effusion (minoxidil)

[1]ACE, angiotensin-converting enzyme.

therapy; preoperative fluid administration can prevent severe hypotension after induction of anesthesia in these patients. Although asymptomatic carotid bruits are usually hemodynamically insignificant, they may be reflective of atherosclerotic vascular disease that may affect the coronary circulation. When a bruit is detected, further workup should be guided by the urgency of the scheduled surgery and the likelihood that further investigations, if diagnostic, would result in a change in therapy. Doppler studies of the carotid arteries can be used to define the extent of carotid disease.

The ECG is often normal, but in patients with a long history of hypertension, it may show evidence of ischemia, conduction abnormalities, an old infarction, or LVH or strain. A normal ECG does not exclude CAD or LVH. Similarly, a normal heart size on a chest radiograph does not exclude ventricular hypertrophy. Echocardiography is a sensitive test of LVH and can be used to evaluate ventricular systolic and diastolic functions in patients with symptoms of heart failure. Chest radiographs are rarely useful in an asymptomatic patient, but may show a boot-shaped heart (suggestive of LVH), frank cardiomegaly, or pulmonary vascular congestion.

Renal function is best evaluated by measurement of serum creatinine and blood urea nitrogen levels. Serum electrolyte levels (K) should be determined in patients taking diuretics or digoxin or those with renal impairment. Mild to moderate hypokalemia (3–3.5 mEq/L) is often seen in patients taking diuretics, but does not have adverse outcome effects. Potassium replacement should be undertaken only in patients who are symptomatic

or who are also taking digoxin. Hypomagnesemia is often present and may be a cause of perioperative arrhythmias. Hyperkalemia may be encountered in patients who are taking potassium-sparing diuretics or ACE inhibitors, particularly those with impaired renal function.

Premedication

Premedication reduces preoperative anxiety and is desirable in hypertensive patients. Mild to moderate preoperative hypertension often resolves following administration of an agent such as midazolam.

INTRAOPERATIVE MANAGEMENT

Objectives

The overall anesthetic plan for a hypertensive patient is to maintain an appropriate stable blood pressure range. Patients with borderline hypertension may be treated as normotensive patients. Those with long-standing or poorly controlled hypertension, however, have altered autoregulation of cerebral blood flow; higher than normal mean blood pressures may be required to maintain adequate cerebral blood flow. Because most patients with long-standing hypertension are assumed to have some element of CAD and cardiac hypertrophy, excessive blood pressure elevations are undesirable. Hypertension, particularly in association with tachycardia, can precipitate or exacerbate myocardial ischemia, ventricular dysfunction, or both. Arterial blood pressure should generally be kept within 20% of preoperative levels. If marked hypertension (>180/120 mm Hg) is present preoperatively, arterial blood pressure should be maintained in the high-normal range (150–140/90–80 mm Hg).

Monitoring

Most hypertensive patients do not require special intraoperative monitors. Direct intraarterial pressure monitoring should be reserved for patients with wide swings in blood pressure and those undergoing major surgical procedures associated with rapid or marked changes in cardiac preload or afterload. Electrocardiographic monitoring should focus on detecting signs of ischemia. Urinary output should generally be monitored with an indwelling urinary catheter in patients with a preexisting renal impairment who are undergoing procedures expected to last more than 2 hr. When invasive hemodynamic monitoring is used, reduced ventricular compliance (see Chapter 20) is often apparent in patients with ventricular hypertrophy; these patients may require more intravenous fluid to produce a higher filling pressure to maintain adequate left ventricular end-diastolic volume and cardiac output. Volume administration in patients with decreased ventricular compliance can also result in elevated pulmonary arterial pressures and pulmonary congestion.

Induction

Induction of anesthesia and endotracheal intubation are often associated with hemodynamic instability in hypertensive patients. Regardless of the level of preoperative blood pressure control, many patients with hypertension display an accentuated hypotensive response to induction of anesthesia, followed by an exaggerated hypertensive response to intubation. Many, if not most, antihypertensive agents and general anesthetics are vasodilators, cardiac depressants, or both. In addition, many hypertensive patients present for surgery in a volume-depleted state. Sympatholytic agents attenuate the normal protective circulatory reflexes, reducing sympathetic tone and enhancing vagal activity.

Up to 25% of hypertensive patients may exhibit severe hypertension following endotracheal intubation. Prolonged laryngoscopy should be avoided. Moreover, intubation should generally be performed under deep anesthesia (provided hypotension can be avoided). One of several techniques may be used before intubation to attenuate the hypertensive response:

- Deepening anesthesia with a potent volatile agent
- Administering a bolus of an opioid (fentanyl, 2.5–5 mcg/kg; alfentanil, 15–25 mcg/kg; sufentanil, 0.5–1.0 mcg/kg; or remifentanil, 0.5–1 mcg/kg).

- Administering lidocaine, 1.5 mg/kg intravenously, intratracheally, or topically in the airway
- Achieving β-adrenergic blockade with esmolol, 0.3–1.5 mg/kg; metoprolol 1–5 mg; or labetalol, 5–20 mg.

Choice of Anesthetic Agents

A. Induction Agents

The superiority of any one agent or technique over another has not been established. Propofol, barbiturates, benzodiazepines, and etomidate are equally safe for inducing general anesthesia in most hypertensive patients. Ketamine by itself can precipitate marked hypertension; however, it is almost never used as a single agent. When administered with a small dose of another agent, such as a benzodiazepine or propofol, ketamine's sympathetic stimulating properties can be blunted or eliminated.

B. Maintenance Agents

Anesthesia may be safely continued with volatile agents (alone or with nitrous oxide), a balanced technique (opioid + nitrous oxide + muscle relaxant), or a total intravenous technique. Regardless of the primary maintenance technique, addition of a volatile agent or intravenous vasodilator generally allows convenient intraoperative blood pressure control.

C. Muscle Relaxants

With the possible exception of large bolus doses of pancuronium, any muscle relaxant can be used. Pancuronium-induced vagal blockade and neural release of catecholamines could exacerbate hypertension in poorly controlled patients, but, if given slowly, in small increments, pancuronium is unlikely to cause medically important increases in heart rate or blood pressure. Moreover, pancuronium can be useful in offsetting excessive vagal tone induced by opioids or surgical manipulations. Hypotension following large (intubating) doses of atracurium may be accentuated in hypertensive patients.

D. Vasopressors

Hypertensive patients may display an exaggerated response to both endogenous catecholamines (from intubation or surgical stimulation) and exogenously administered sympathetic agonists. If a vasopressor is necessary to treat excessive hypotension, a small dose of a direct-acting agent, such as phenylephrine (25–50 mcg), may be useful. Patients taking sympatholytics preoperatively may exhibit a decreased response to ephedrine. Vasopressin as a bolus or infusion can also be employed to restore vascular tone in the hypotensive patient.

Intraoperative Hypertension

Intraoperative hypertension not responding to an increase in anesthetic depth (particularly with a volatile agent) can be treated with a variety of parenteral agents (Table 21–7). Readily reversible causes—such as inadequate anesthetic depth, hypoxemia, or hypercapnia—should always be excluded before initiating antihypertensive therapy. Selection of a hypotensive agent depends on the severity, acuteness, and cause of hypertension; the baseline ventricular function; the heart rate; the presence of bronchospastic pulmonary disease; and the anesthetist's familiarity with each of the drug options. β-Adrenergic blockade alone or as a supplement is a good choice for a patient with good ventricular function and an elevated heart rate, but is relatively contraindicated in a patient with bronchospastic disease. Metoprolol, esmolol, or labetolol are readily used intraoperatively. Nicardipine or clevidipine may be preferable to β-blockers for patients with bronchospastic disease. Nitroprusside remains the most rapid and effective agent for the intraoperative treatment of moderate to severe hypertension. Nitroglycerin may be less effective, but is also useful in treating or preventing myocardial ischemia. Fenoldopam, a dopamine agonist, is also a useful hypotensive agent; furthermore, it increases renal blood flow. Hydralazine provides sustained blood pressure control, but also has a delayed onset and can cause reflex tachycardia. The latter is not seen with labetalol because of a combined α- and β-adrenergic blockade.

TABLE 21–7 Parenteral agents for the acute treatment of hypertension.

Agent	Dosage Range	Onset	Duration
Nitroprusside	0.5–10 mcg/kg/min	30–60	1–5 min
Nitroglycerin	0.5–10 mcg/kg/min	1 min	3–5 min
Esmolol	0.5 mg/kg over 1 min; 50–300 mcg/kg/min	1 min	12–20 min
Labetalol	5–20 mg	1–2 min	4–8 hr
Metoprolol	2.5–5 mg	1–5 min	5–8 hr
Hydralazine	5–20 mg	5–20 min	4–8 hr
Clevidipine	1–32 mg/hr	1–3 min	5–15 min
Nicardipine	0.25–0.5 mg 5–15 mg/hr	1–5 min	3–4 hr
Enalaprilat	0.625–1.25 mg	6–15 min	4–6 hr
Fenoldopam	0.1–1.6 mg/kg/min	5 min	5 min

POSTOPERATIVE MANAGEMENT

Postoperative hypertension is common and should be anticipated in patients who have poorly controlled hypertension. Close blood pressure monitoring should be continued in both the recovery room and the early postoperative period. In addition to myocardial ischemia and congestive heart failure, marked sustained elevations in blood pressure can contribute to the formation of wound hematomas and the disruption of vascular suture lines.

Hypertension in the recovery period is often multifactorial and enhanced by respiratory abnormalities, anxiety and pain, volume overload, or bladder distention. Contributing causes should be corrected and parenteral antihypertensive agents given if necessary. Intravenous labetalol is particularly useful in controlling hypertension and tachycardia, whereas vasodilators are useful in controlling blood pressure in the setting of a slow heart rate. When the patient resumes oral intake, preoperative medications should be restarted.

ISCHEMIC HEART DISEASE

Preoperative Considerations

Myocardial ischemia is characterized by a metabolic oxygen demand that exceeds the oxygen supply. Ischemia can therefore result from a marked increase in myocardial metabolic demand, a reduction in myocardial oxygen delivery, or a combination of both. Common causes include coronary arterial vasospasm or thrombosis; severe hypertension or tachycardia (particularly in the presence of ventricular hypertrophy); severe hypotension, hypoxemia, or anemia; and severe aortic stenosis or regurgitation.

By far, the most common cause of myocardial ischemia is atherosclerosis of the coronary arteries. CAD is responsible for about 25% of all deaths in Western societies and is a major cause of perioperative morbidity and mortality. The overall incidence of CAD in surgical patients is estimated to be between 5% and 10%. Major risk factors for CAD include hyperlipidemia, hypertension, diabetes, cigarette smoking, increasing age, male sex, and a positive family history. Other risk factors include

obesity, a history of cerebrovascular or peripheral vascular disease, menopause, use of high-estrogen oral contraceptives (in women who smoke), and a sedentary lifestyle.

CAD may be clinically manifested by symptoms of myocardial necrosis (infarction), ischemia (usually angina), arrhythmias (including sudden death), or ventricular dysfunction (congestive heart failure). When symptoms of congestive heart failure predominate, the term "ischemic cardiomyopathy" is often used.

Unstable Angina

Unstable angina is defined as (1) an abrupt increase in severity, frequency (more than three episodes per day), or duration of anginal attacks (crescendo angina): (2) angina at rest; or (3) new onset of angina (within the past 2 months) with severe or frequent episodes (more than three per day). Unstable angina may occur following MI or be precipitated by noncardiac medical conditions (including severe anemia, fever, infections, thyrotoxicosis, hypoxemia, and emotional distress) in previously stable patients.

Unstable angina, particularly when it is associated with significant ST-segment changes at rest, usually reflects severe underlying coronary disease and frequently precedes MI. Plaque disruption with platelet aggregates or thrombi and vasospasm are frequent pathological correlates. Critical stenosis in one or more major coronary arteries is present in more than 80% of patients with these symptoms. Patients with unstable angina require evaluation and treatment, which may include admission to a coronary care unit and some form of coronary intervention.

Chronic Stable Angina

Anginal chest pains are most often substernal, exertional, radiating to the neck or arm, and relieved by rest or nitroglycerin. Variations are common, including epigastric, back, or neck pain, or transient shortness of breath from ventricular dysfunction (anginal equivalent). Nonexertional ischemia and silent (asymptomatic) ischemia are recognized as fairly common occurrences. Patients with diabetes have an increased incidence of silent ischemia.

Symptoms are generally absent until the atherosclerotic lesions cause 50% to 75% occlusion of the coronary circulation. When a stenotic segment reaches 70% occlusion, maximum compensatory dilatation is usually present distally: blood flow is generally adequate at rest, but becomes inadequate with increased metabolic demand. An extensive collateral blood supply allows some patients to remain relatively asymptomatic in spite of severe disease. Coronary vasospasm is also a cause of transient transmural ischemia in some patients; 90% of vasospastic episodes occur at preexisting stenotic lesions in epicardial vessels and are often precipitated by a variety of factors, including emotional upset and hyperventilation (Prinzmetal's angina). Coronary spasm is most often observed in patients who have angina with varying levels of activity or emotional stress (variable-threshold); it is least common with classic exertional (fixed-threshold) angina.

The overall prognosis of patients with CAD is related to both the number and severity of coronary obstructions, as well as to the extent of ventricular dysfunction.

Treatment of Ischemic Heart Disease

The general approach in treating patients with ischemic heart disease is five-fold:

- Correction of risk factors, with the hope of slowing disease progression.
- Modification of the patient's lifestyle to reduce stress and improve exercise tolerance.
- Correction of complicating medical conditions that can exacerbate ischemia (ie, hypertension, anemia, hypoxemia, hyperthyroidism, fever, infection, or adverse drug effects).
- Pharmacological manipulation of the myocardial oxygen supply–demand relationship.
- Correction of coronary lesions by percutaneous coronary intervention (angioplasty [with or without stenting] or atherectomy) or coronary artery bypass surgery.

The last three approaches are of direct relevance to anesthesiologists. The same principles should be applied in the care of these patients in both the operating room and the intensive care unit.

TABLE 21–8 Comparison of antianginal agents.[1]

Cardiac Parameter	Nitrates	Calcium Channel Blockers			β-Blockers
		Verapamil	*Nifedipine Nicardipine Nimodipine*	*Diltiazem*	
Preload	↓↓	—	—	—	—/↑
Afterload	↓	↓	↓↓	↓	—/↓
Contractility	—	↓↓	—	↓	↓↓↓
SA node automaticity	↑/—	↓↓	↑/—	↓↓	↓↓↓
AV conduction	—	↓↓↓	—	↓↓	↓↓↓
Vasodilatation					
Coronary	↑	↑↑	↑↑↑	↑↑	—/↓
Systemic	↑↑	↑	↑↑	↑	—/↓

[1]SA, sinoatrial; AV, atrioventricular; ↑, increases; —, no change; ↓, decreases.

The most commonly used pharmacological agents are nitrates, β-blockers, and calcium channel blockers. These drugs also have potent circulatory effects, which are compared in Table 21–8. Any of these agents can be used for mild angina. Calcium channel blockers are the drugs of choice for patients with predominantly vasospastic angina. β-Blockers improve the long-term outcome of patients with CAD. Nitrates are good agents for both types of angina.

A. Nitrates

Nitrates relax all vascular smooth muscle, but have a much greater effect on venous than on arterial vessels. Decreasing venous and arteriolar tone and reducing the effective circulating blood volume (cardiac preload) reduce wall tension afterload. These effects tend to reduce myocardial oxygen demand. The prominent venodilatation makes nitrates excellent agents when congestive heart failure is also present.

Perhaps equally important, nitrates dilate the coronary arteries. Even minor degrees of dilatation at stenotic sites may be sufficient to increase blood flow, because flow is directly related to the fourth power of the radius. Nitrate-induced coronary vasodilatation preferentially increases subendocardial blood flow in ischemic areas. This favorable redistribution of coronary blood flow to ischemic areas may be dependent on the presence of collaterals in the coronary circulation.

Nitrates can be used for both the treatment of acute ischemia and prophylaxis against frequent anginal episodes. Unlike β-blockers and calcium channel blockers, nitrates do not have a negative inotropic effect—a desirable feature in the presence of ventricular dysfunction. Intravenous nitroglycerin can also be used for controlled hypotensive anesthesia.

B. Calcium Channel Blockers

The effects and uses of the most commonly used calcium channel blockers are shown in Table 21–9. Calcium channel blockers reduce myocardial oxygen demand by decreasing cardiac afterload and augment oxygen supply by increasing blood flow (coronary vasodilatation). Verapamil and diltiazem also reduce demand by slowing the heart rate.

Nifedipine's potent effects on the systemic blood pressure may precipitate hypotension, reflex tachycardia, or both; its fast-onset preparations (eg, sublingual) have been associated with MI in some patients. Its tendency to decrease afterload generally offsets any negative inotropic effect. The slow-release form of nifedipine is associated with much less reflex tachycardia and is more suitable than other agents for patients with ventricular

TABLE 21–9 Comparison of calcium channel blockers.

				Clinical Use			
Agent	**Route**	**Dosage[1]**	**Half-life**	***Angina***	***Hypertension***	***Cerebral Vasospasm***	***Supraventricular Tachycardia***
Verapamil	PO	40–240 mg	5 hr	+	+		+
	IV	5–15 mg	5 hr	+			+
Nifedipine	PO	30–180 mg	2 hr	+	+		
	SL	10 mg	2 hr	+	+		
Diltiazem	PO	30–60 mg	4 hr	+	+		+
	IV	0.25–0.35 mg/kg	4 hr	+			+
Nicardipine	PO	60–120 mg	2–4 hr	+	+		
	IV	0.25–0.5 mg/kg	2–4 hr	+	+		
Nimodipine	PO	240 mg	2 hr			+	
Bepridil[2]	PO	200–400 mg	24 hr	+	+		
Isradipine	PO	2.5–5.0 mg	8 hr		+		
Felodipine	PO	5–20 mg	9 hr		+		
Amlodipine	PO	2.5–10 mg	30–50 hr	+	+		

[1]Total oral dose per day divided into three doses unless otherwise stated.
[2]Also possesses antiarrhythmic properties.

dysfunction. In contrast, verapamil and diltiazem have greater effects on cardiac contractility and atrioventricular (AV) conduction and therefore should be used cautiously, if at all, in patients with ventricular dysfunction, conduction abnormalities, or bradyarrhythmias. Diltiazem seems to be better tolerated than verapamil in patients with impaired ventricular function. Nicardipine, nimodipine, and clevidipine generally have the same effects as nifedipine; nimodipine is primarily used in preventing cerebral vasospasm following subarachnoid hemorrhage, whereas nicardipine is used as an intravenous arterial vasodilator. Clevidipine is an ultrashort–acting arterial vasodilator.

Calcium channel blockers can have significant interactions with anesthetic agents. All calcium channel blockers potentiate both depolarizing and nondepolarizing neuromuscular blocking agents and the circulatory effects of volatile agents. Both verapamil and diltiazem can potentiate depression of cardiac contractility and conduction in the AV node by volatile anesthetics. Nifedipine and similar agents can potentiate systemic vasodilatation by volatile and intravenous agents.

C. β-Adrenergic Blocking Agents

These drugs decrease myocardial oxygen demand by reducing heart rate and contractility, and, in some cases, afterload (via their antihypertensive effect). Optimal blockade results in a resting heart rate between 50 and 60 beats/min and prevents appreciable increases with exercise (<20 beats/min increase during exercise). Available agents differ in receptor selectivity, intrinsic sympathomimetic (partial agonist) activity, and membrane-stabilizing properties (Table 21–10). Membrane stabilization, often described as a quinidine-like effect, results in antiarrhythmic activity. Agents with

TABLE 21–10 Comparison of β-adrenergic blocking agents.

Agent	β_1-Receptor Selectivity	Half-life	Sympathomimetic	α-Receptor Blockade	Membrane Stabilizing
Acebutolol	+	2–4 hr	+		+
Atenolol	++	5–9 hr			
Betaxlol	++	14–22 hr			
Esmolol	++	9 min			
Metoprolol	++	3–4 hr			±
Bisoprolol	+	9–12 hr			
Oxprenolol		1–2 hr	+		+
Alprenolol		2–3 hr	+		+
Pindolol		3–4 hr	++		±
Penbutolol		5 hr	+		+
Carteolol		6 hr	+		
Labetalol		4–8 hr		+	±
Propranolol		3–6 hr			++
Timolol		3–5 hr			
Sotalol[1]		5–13 hr			
Nadolol		10–24 hr			
Carvedilol		6–8 hr		+	±

[1]Also possesses unique antiarrhythmic properties.

intrinsic sympathomimetic properties are better tolerated by patients with mild to moderate ventricular dysfunction. Certain β-blockers (carvedilol and extended-duration metoprolol) improve survival in patients with chronic heart failure. This has not been shown to be a drug class effect. Blockade of β_2-adrenergic receptors also can mask hypoglycemic symptoms in patients with diabetes, delay metabolic recovery from hypoglycemia, and impair the handling of large potassium loads. Cardioselective (β_1-receptor-specific) agents, although generally better tolerated than nonselective agents in patients with reactive airways, must still be used cautiously in such patients. The selectivity of cardioselective agents tends to be dose dependent. Patients on long-standing β-blocker therapy should have these agents continued perioperatively. Acute β-blocker withdrawal in the perioperative period places patients at a markedly increased risk of cardiac morbidity and mortality.

Documentation of avoidance of β-blocker withdrawal is a frequent tool by which "quality" of anesthesia services can be assessed by regulatory agencies.

D. Other Agents

ACE inhibitors prolong survival in patients with congestive heart failure or left ventricular dysfunction. Chronic aspirin therapy reduces coronary events in patients with CAD and prevents coronary

and ischemic cerebral events in at-risk patients. Antiarrhythmic therapy in patients with complex ventricular ectopy who have significant CAD and left ventricular dysfunction should be guided by an electrophysiological study. Patients with inducible sustained ventricular tachycardia (VT) or ventricular fibrillation are candidates for an automatic internal cardioverter-defibrillator (ICD). Treatment of ventricular ectopy (with the exception of sustained VT) in patients with good ventricular function does not improve survival and may increase mortality. In contrast, ICDs have been shown to improve survival in patients with advanced cardiomyopathy (ejection fraction <30%), even in the absence of demonstrable arrhythmias.

E. Combination Therapy

Moderate to severe angina frequently requires combination therapy with two or all three classes of agents. Patients with ventricular dysfunction may not tolerate the combined negative inotropic effect of a β-blocker and a calcium channel blocker together; an ACE inhibitor is better tolerated and seems to improve survival. Similarly, the additive effect of a β-blocker and a calcium channel blocker on the AV node may precipitate heart block in susceptible patients.

PREOPERATIVE MANAGEMENT

The importance of ischemic heart disease—particularly a history of MI—as a risk factor for perioperative morbidity and mortality was discussed earlier in the chapter. Most studies confirm that perioperative outcome is related to disease severity, ventricular function, and the type of surgery to be undertaken. 3 Patients with extensive (three-vessel or left main) CAD, a recent history of MI, or ventricular dysfunction are at greatest risk of cardiac complications. As mentioned above, current guidelines recommend revascularization when such treatment would be indicated irrespective of the patient's need for surgery.

Chronic stable (mild to moderate) angina does not seem to increase perioperative risk substantially. Similarly, a history of prior coronary artery bypass surgery or coronary angioplasty alone does not seem to substantially increase perioperative risk. In some studies, maintenance of chronic β-receptor blockers in the perioperative period has been shown to reduce perioperative mortality and the incidence of postoperative cardiovascular complications; however, other studies have shown an increase in stroke and death following preoperative introduction of β-blockers to "at risk" patients. Consequently, as with all drugs, the risks and benefits of initiating therapy with β-blockers in at risk patients must be considered. Like β-blockers, statins should be continued perioperatively in patients so routinely treated, as acute perioperative withdrawal of statins is associated with adverse outcomes. ACC/AHA guidelines suggest that β-blockers are useful in patients undergoing vascular surgery with evidence of ischemia on their evaluative workup (class I).

History

The history is of prime importance in patients with ischemic heart disease. Questions should encompass symptoms, current and past treatment, complications, and the results of previous evaluations. This information alone is usually enough to provide some estimate of disease severity and ventricular function.

The most important symptoms to elicit include chest pains, dyspnea, poor exercise tolerance, syncope, or near syncope. The relationship between symptoms and activity level should be established. Activity should be described in terms of everyday tasks, such as walking or climbing stairs. Patients may be relatively asymptomatic despite severe CAD if they have a sedentary lifestyle. Patients with diabetes are particularly prone to silent ischemia. The patient's description of chest pains may suggest a major role for vasospasm (variable-threshold angina). Easy fatigability or shortness of breath suggests impaired ventricular function.

A history of unstable angina or MI should include the time of its occurrence and whether it was complicated by arrhythmias, conduction disturbances, or heart failure. Localization of the areas of ischemia is invaluable in deciding which electrocardiographic leads to monitor intraoperatively. Arrhythmias and conduction abnormalities are

more common in patients with previous infarction and in those with poor ventricular function. This latter group of patients will often have ICDs.

Physical Examination & Routine Laboratory Evaluation

Evaluation of patients with CAD is similar to that of patients with hypertension. Laboratory evaluation in patients who have a history compatible with recent unstable angina and are undergoing emergency procedures should include cardiac enzymes. Serum levels of cardiac-specific troponins, creatine kinase (MB isoenzyme), and lactate dehydrogenase (type 1 isoenzyme) are useful in excluding MI.

The baseline ECG is normal in 25% to 50% of patients with CAD but no prior MI. Electrocardiographic evidence of ischemia often becomes apparent only during chest pain. The most common baseline abnormalities are nonspecific ST-segment and T-wave changes. Prior infarction is often manifested by Q waves or loss of R waves in the leads closest to the infarct. First-degree AV block, bundle-branch block, or hemiblock may be present. Persistent ST-segment elevation following MI may be indicative of a left ventricular aneurysm. A long rate-corrected QT interval ($QT_c > 0.44$ s) may reflect the underlying ischemia, drug toxicity (usually class Ia antiarrhythmic agents, antidepressants, or phenothiazines), electrolyte abnormalities (hypokalemia or hypomagnesemia), autonomic dysfunction, mitral valve prolapse, or, less commonly, a congenital abnormality. Patients with a long QT interval are at risk of developing ventricular arrhythmias—particularly polymorphic VT (torsade de pointes), which can lead to ventricular fibrillation. The long QT interval reflects nonuniform prolongation of ventricular repolarization and predisposes patients to reentry phenomena. In contrast to polymorphic ventricular arrhythmias with a normal QT interval, which respond to conventional antiarrhythmics, polymorphic tachyarrhythmias with a long QT interval generally respond best to pacing or magnesium salts. Patients with congenital prolongation generally respond to β-adrenergic blocking agents. Left stellate ganglion blockade is also effective and suggests that autonomic imbalance plays an important role in this group of patients.

The chest film can be used to exclude cardiomegaly or pulmonary vascular congestion secondary to ventricular dysfunction. Rarely, calcification of the coronaries, aorta, or the aortic valve may be seen on the chest radiograph; such is a more common finding on CT.

Specialized Studies

When used as screening tests for the general population, noninvasive stress tests have a low predictability in asymptomatic patients, but are sufficiently reliable in symptomatic patients with suspect lesions. 4 Holter monitoring, exercise electrocardiography, myocardial perfusion scans, and echocardiography are important in determining perioperative risk and the need for coronary angiography; however, these tests are indicated only if their outcome would alter patient care.

Current ACC/AHA guidelines recommend noninvasive stress testing in patients scheduled for noncardiac surgery with active cardiac conditions (class I). The current guidelines also suggest that there may be benefit of such testing in patients with three or more clinical risk factors and poor functional capacity (class IIa). Likewise, they suggest that noninvasive testing can be of some possible benefit in patients with one or two clinical risk factors undergoing intermediate risk or vascular surgery (class IIb). What they do not recommend is the indiscriminate use of noninvasive cardiac testing in patients with no risk factors undergoing intermediate-risk surgery. Consequently, indications for preoperative cardiac screening tests continue to narrow.

A. Holter Monitoring

Continuous ambulatory electrocardiographic (Holter) monitoring is useful in evaluating arrhythmias, antiarrhythmic drug therapy, and severity and frequency of ischemic episodes. Silent (asymptomatic) ischemic episodes are frequently found in patients with CAD. Frequent ischemic episodes on preoperative Holter monitoring correlate well with intraoperative and postoperative ischemia. Holter monitoring has an excellent negative predictive value for postoperative cardiac complications.

B. Exercise Electrocardiography

The usefulness of this test is limited in patients with baseline ST-segment abnormalities and those who are unable to increase their heart rate (>85% of maximal predicted) because of fatigue, dyspnea, or drug therapy. Overall sensitivity is 65%, and specificity is 90%. The test is most sensitive (85%) in patients with three-vessel or left main CAD. Disease that is limited to the left circumflex artery may also be missed because ischemia in its distribution may not be evident on the standard surface ECG. A normal test does not necessarily exclude CAD, but suggests that severe disease is not likely. The degree of ST-segment depression, its severity and configuration, the time of onset in the test, and the time required for resolution are important findings. A myocardial ischemic response at low levels of exercise is associated with a significantly increased risk of perioperative complications and long-term cardiac events. Other significant findings include changes in blood pressure and the occurrence of arrhythmias. Exercise-induced ventricular ectopy frequently indicates severe CAD associated with ventricular dysfunction. The ischemia presumably leads to electrical instability in myocardial cells. Given that risk seems to be associated with the degree of myocardium potentially ischemic, testing often includes perfusion scans or echocardiographic assessments; however, in ambulatory patients, exercise ECG testing is useful because it estimates functional capacity and detects myocardial ischemia.

C. Myocardial Perfusions Scans and Other Imaging Techniques

Myocardial perfusion imaging using thallium-201 or technetium-99m is used in evaluating patients who cannot exercise (eg, peripheral vascular disease) or who have underlying ECG abnormalities that preclude interpretation during exercise (eg, left bundle-branch block). If the patient cannot exercise, images are obtained before and after injection of an intravenous coronary dilator (eg, dipyridamole or adenosine) to produce a hyperemic response similar to exercise. Myocardial perfusion studies following exercise or injection of dipyridamole or adenosine have a high sensitivity, but only fairly good specificity for CAD. They are best for detecting two- or three-vessel disease. These scans can locate and quantitate areas of ischemia or scarring and differentiate between the two. Perfusion defects that fill in on the redistribution phase represent ischemia, not previous infarction. The negative predictive value of a normal perfusion scan is approximately 99%.

MRI, PET, and CT scans are increasingly being used to define coronary artery anatomy and determine myocardial viability.

D. Echocardiography

This technique provides information about both regional and global ventricular function and may be carried out at rest, following exercise, or with administration of dobutamine. Detectable regional wall motion abnormalities and the derived left ventricular ejection fraction correlate well with angiographic findings. Moreover, dobutamine stress echocardiography seems to be a reliable predictor of adverse cardiac complications in patients who cannot exercise. New or worsening wall motion abnormalities following dobutamine infusion are indicative of significant ischemia. Patients with an ejection fraction of less than 50% tend to have more severe disease and increased perioperative morbidity. Dobutamine stress echocardiography, however, may not be reliable in patients with left bundle-branch block because septal motion may be abnormal, even in the absence of left anterior descending CAD in some patients.

E. Coronary Angiography

Coronary angiography remains the definitive way to evaluate CAD and is associated with a low complication rate (<1%). Nonetheless, coronary angiography should be performed only to determine if the patient may benefit from percutaneous coronary angioplasty or coronary artery bypass grafting prior to noncardiac surgery. The location and severity of occlusions can be defined, and coronary vasospasm may also be observed on angiography. In evaluating fixed stenotic lesions, occlusions greater than 50% to 75% are generally considered significant. The severity of disease is often expressed according to the number of major coronary vessels affected (one-, two-, or three-vessel

disease). Significant stenosis of the left main coronary artery is of great concern because disruption of flow in this vessel will have adverse effects on almost the entire left ventricle.

Ventriculography, measurement of the ejection fraction, and measurement of intracardiac pressures, also provide important information. Indicators of significant ventricular dysfunction include an ejection fraction <50%, a left ventricular end-diastolic pressure >18 mm Hg, a cardiac index <2.2 L/min/m^2, and marked or multiple wall motion abnormalities.

Guidelines suggest that patients with stable angina and significant left main disease, stable angina and three-vessel disease, stable angina and two-vessel disease with an ejection fraction <50%, unstable angina, non-ST segment elevation MI, and acute ST segment elevation MI benefit from revascularization. This recommendation also applies to patients who are scheduled for noncardiac surgery (class I). Conversely, revascularization is not indicated in patients with stable angina (class III). Moreover, elective noncardiac surgery is not recommended within 4–6 weeks following bare metal stent placement or within 12 months of placement of a drug-eluting stent, if the surgery requires that antiplatelet therapy be discontinued.

Premedication

Allaying fear, anxiety, and pain preoperatively are desirable goals in patients with CAD. Satisfactory premedication prevents sympathetic activation, which adversely affects the myocardial oxygen supply–demand balance. Overmedication is equally detrimental and should be avoided because it may result in hypoxemia, respiratory acidosis, and hypotension. A benzodiazepine, alone or in combination with an opioid, is commonly used. (The concomitant administration of oxygen via nasal cannula helps avoid hypoxemia following premedication.) Patients with poor ventricular function and coexistent lung disease should receive reduced doses. Preoperative medications should generally be continued until the time of surgery. They may be given orally (with a small sip of water), intramuscularly, intravenously, sublingually, or transdermally.

5 The sudden withdrawal of antianginal medication perioperatively—particularly β-blockers—can precipitate a sudden, rebound increase in ischemic episodes. In the past, some clinicians prophylactically administered nitrates intravenously or transdermally to patients with CAD in the perioperative period. Although this practice may be theoretically advantageous, there is no evidence of its efficacy in patients not previously on long-term nitrate therapy and without evidence of ongoing ischemia. Transdermal absorption of nitroglycerin may be erratic in the perioperative period.

INTRAOPERATIVE MANAGEMENT

The intraoperative period is regularly associated with factors and events that can adversely affect the myocardial oxygen demand–supply relationship. Activation of the sympathetic system plays a major role. Hypertension and enhanced contractility increase myocardial oxygen demand, whereas tachycardia increases demand and reduces supply. Although myocardial ischemia is commonly associated with tachycardia, it can occur in the absence of any apparent hemodynamic derangement.

Objectives

6 The overwhelming priority in managing patients with ischemic heart disease is maintaining a favorable myocardial supply–demand relationship. Autonomic-mediated increases in heart rate and blood pressure should be controlled by deep anesthesia or adrenergic blockade. Excessive reductions in coronary perfusion pressure or arterial oxygen content are to be avoided. Although exact limits are not defined or predictable, diastolic arterial pressure should generally be maintained at 50 mm Hg or above. Higher diastolic pressures may be preferable in patients with high-grade coronary occlusions. Excessive increases—such as those caused by fluid overload—in left ventricular end-diastolic pressure should be avoided because they increase ventricular wall tension (afterload) and can reduce subendocardial perfusion (see Chapter 20). Transfusion carries its own risks and consequently there is no set transfusion trigger in patients with

CAD; however, anemia can lead to tachycardia, worsening the balance between myocardial oxygen supply and demand.

MONITORING

Intraarterial pressure monitoring is reasonable for all patients with severe CAD and major or multiple cardiac risk factors who are undergoing any but the most minor procedures. Central venous (or rarely pulmonary artery) pressure can be monitored during prolonged or complicated procedures involving large fluid shifts or blood loss. Less invasive methods of cardiac output determination and volume assessment have been previously discussed in this text. Transesophageal echocardiography (TEE) and transthoracic echocardiography (TTE) can provide valuable information, both qualitative and quantitative, on contractility and ventricular chamber size (preload) perioperatively. Intensive care unit staff increasingly use ultrasound to assist in hemodynamic management. Numerous "basic" courses in TEE and TTE are available to assist practitioners in performing "hemodynamic," as opposed to cardiac diagnostic TEE.

7 Intraoperative detection of ischemia depends on recognition of electrocardiographic changes, hemodynamic manifestations, or regional wall motion abnormalities on TEE. Doppler TEE also allows detection of the onset of mitral regurgitation caused by ischemic papillary muscle dysfunction.

A. Electrocardiography

Early ischemic changes are subtle and can often be overlooked. They involve changes in T-wave morphology, including inversion, tenting, or both (**Figure 21–1**). More obvious ischemia may be seen in the form of progressive ST-segment depression. Down-sloping and horizontal ST depressions are of greater specificity for ischemia than is up-sloping depression. New ST-segment elevations are rare during noncardiac surgery and are indicative of severe ischemia, vasospasm, or infarction. However, the increasing number of individuals treated with drug-eluting stents can be problematic perioperatively, especially if surgical concerns necessitate discontinuation of antiplatelet therapy (eg, emergency spine surgery). Such patients are at very increased risk of thrombosis and perioperative MI. Anesthesia staff should never for nonsurgical reasons (eg, desire to perform a spinal anesthetic) discontinue antiplatelet or anti thrombotic agents perioperatively without first discussing the risks and benefits of the proposed anesthetic requiring suspension of antiplatelet therapy with the patient and his or her cardiologist. ACC/AHA offers recommendations on the approach of bringing patients to surgery following percutaneous coronary interventions and the type of interventions suggested when subsequent surgery is expected (**Figures 21–2** and **21–3**). It should be noted that an isolated minor ST elevation in the mid-precordial leads (V_3 and V_4) can be a normal variant in young patients. Ischemia may also present as an unexplained intraoperative atrial or ventricular arrhythmia or the onset of a new conduction abnormality. The sensitivity of the ECG in detecting ischemia is related to the number of leads monitored. Studies suggest that the V_5, V_4, II, V_2, and V_3 leads (in decreasing sensitivity) are most useful. Ideally, at least two leads should be monitored simultaneously. Usually, lead II is monitored for inferior wall ischemia and arrhythmias, and V_5 is monitored for anterior wall ischemia. When only one channel can be monitored, a modified V_5 lead provides the highest sensitivity.

B. Hemodynamic Monitoring

The most common hemodynamic abnormalities observed during ischemic episodes are hypertension and tachycardia. They are almost always a cause (rather than the result) of ischemia. Hypotension is a late and ominous manifestation of progressive ventricular dysfunction. TEE readily will demonstrate a dysfunctional ventricle and ventricular wall motion changes associated with myocardial ischemia. Ischemia is frequently, but not always, associated with an abrupt increase in pulmonary capillary wedge pressure. The sudden appearance of a prominent *v* wave on the wedge waveform is usually indicative of acute mitral regurgitation from ischemic papillary muscle dysfunction or acute left ventricular dilatation.

C. Transesophageal Echocardiography

TEE can be helpful in detecting global and regional cardiac dysfunction, as well as valvular function in selected patients. Moreover, detection of new

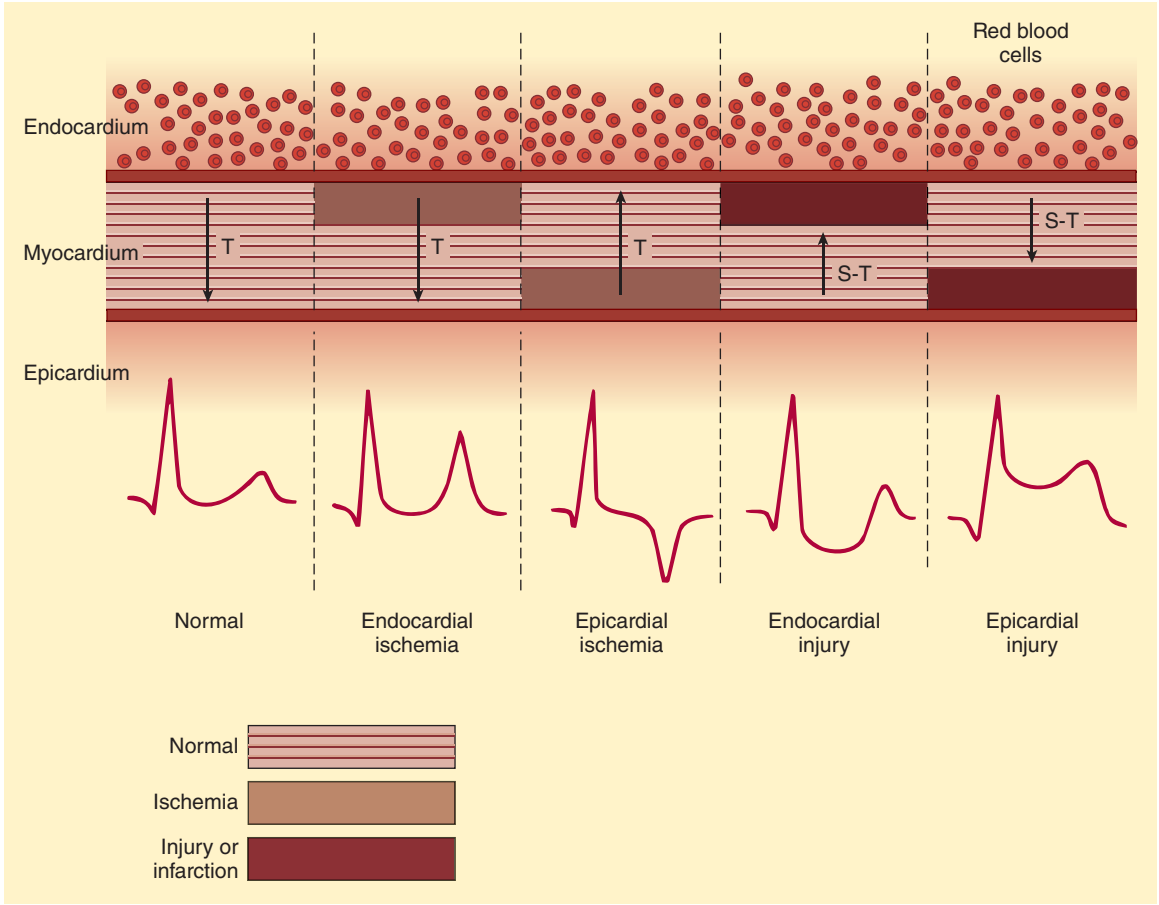

FIGURE 21–1 Electrocardiographic signs of ischemia. Patterns of ischemia and injury. (Information compiled from Schamroth L: *The 12 Lead Electrocardiogram.* Blackwell, 1989.)

regional wall motion abnormalities is a rapid and more sensitive indicator of myocardial ischemia than the ECG. In animal studies in which coronary blood flow is gradually reduced, regional wall motion abnormalities develop before the ECG changes. Although the occurrence of new intraoperative abnormalities correlates with postoperative MIs in some studies, not all such abnormalities are necessarily ischemic. Both regional and global abnormalities can be caused by changes in heart rate, altered conduction, preload, afterload, or drug-induced changes in contractility. Decreased systolic wall thickening may be a more reliable index for ischemia than endocardial wall motion alone.

Arrhythmias, Pacemakers, and Internal Cardioverter-Defibrillator Management

Electrolyte disorders, heart structure defects, inflammation, myocardial ischemia, cardiomyopathies, and conduction abnormalities can all contribute to the development of perioperative arrhythmias and heart block. Consequently, the anesthesia staff must be prepared to manage both chronic and new-onset cardiac rhythm problems.

Supraventricular tachycardias (SVTs) can have hemodynamic consequences secondary to loss of AV synchrony and decreased diastolic filling time.

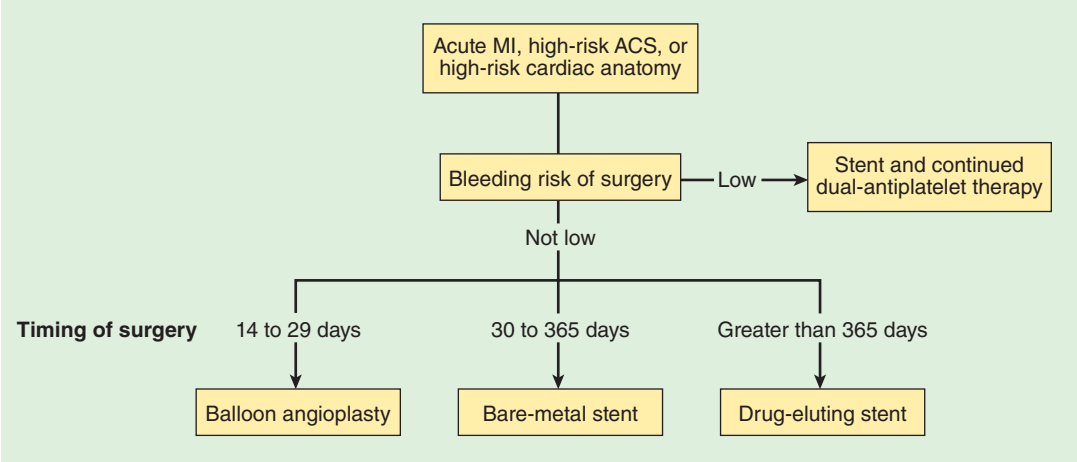

FIGURE 21–2 Proposed treatment for patients requiring percutaneous coronary intervention (PCI) who need subsequent surgery. ACS, acute coronary syndrome; COR, class of recommendation; LOE, level of evidence; MI, myocardial infarction. (Reproduced, with permission, from Fleisher L, Beckman J, Brown K, et al: ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery. Circulation 2007;116: e418.)

Loss of the "P" wave on the ECG with a fast ventricular response is consistent with SVTs. Most SVTs occur secondary to a reentrant mechanism. Reentrant arrhythmias occur when conduction tissues in the heart depolarize or repolarize at varying rates. In this manner, a self-perpetuating loop of repolarization and depolarization can occur in the conduction pathways and/or AV node. SVTs producing hemodynamic collapse are treated perioperatively with synchronized cardioversion. Adenosine can likewise be given to slow AV node conduction and potentially disrupt the reentrant loop. SVTs

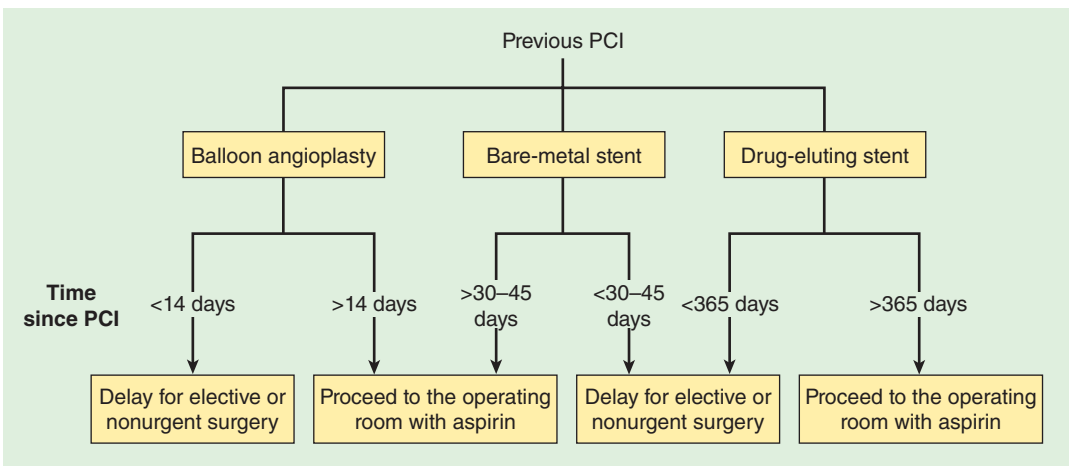

FIGURE 21–3 Proposed approach to the management of patients with previous percutaneous coronary intervention (PCI) who require non-cardiac surgery, based on expert opinion. (Reproduced, with permission, from Fleisher L, Beckman J, Brown K, et al: ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery. Circulation 2007;116: e418.)

in patients without accessory conduction bundles (Wolff–Parkinson–White [WPW] syndrome) are treated with β-blockers and calcium channel blockers. In patients with known WPW, procainamide or amiodarone can be used to treat SVTs. At times, SVTs manifest with a broad QRS complex and seem to be similar to VTs. Such rhythms, when they present, should be treated like VT, until proven otherwise.

Atrial fibrillation (AF) can complicate the perioperative period (**Figure 21–4**) Up to 35% of cardiac surgery patients develop postoperative AF. Moreover, many patients present with AF for anesthesia and noncardiac surgery. The ACC/AHA has issued voluminous guidelines for the outpatient management of AF. The guidelines recommend use of β-blockers or nondihydropyridine calcium antagonists for ventricular rate control in patients without accessory conduction pathways. Amiodarone, procainamide, disopyramide, and ibutilide are suggested for ventricular rate control in patients with accessory pathways. The use of digitalis and nondihyropryidine calcium channel blockers is contraindicated in patients with accessory pathways.

The ACC/AHA guidelines also recommend antithrombotic therapy in patients with long-standing AF. Consequently, many patients with AF will present to the operating room on some form of antithrombotic therapy—often the vitamin K antagonist warfarin. However, ACC/AHA guidelines suggest that aspirin can be an alternative to vitamin K antagonists in low-risk patients or those with contraindications to oral anticoagulation. Likewise, in patients with AF without mechanical prosthetic heart valves, the guidelines suggest that it is acceptable to discontinue anticoagulation for up to 1 week in advance of surgical procedures, without instituting heparin anticoagulation.

When AF develops perioperatively, rate control with β-blockers can often be instituted. Chemical cardioversion can be attempted with amiodarone or procainamide. Of note, if the duration of AF is greater than 48 hours, or unknown, ACC/AHA guidelines recommend anticoagulation for 3 weeks prior to and 4 weeks following either electrical or chemical cardioversion. Alternatively, TEE can be performed to rule out the presence of left atrial or left atrial appendage thrombus.

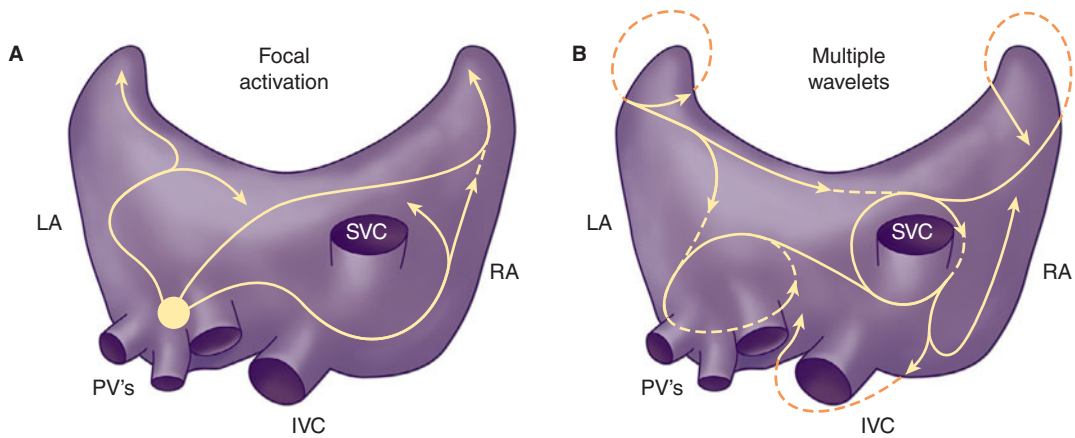

FIGURE 21–4 Posterior view of principal electrophysiological mechanisms of atrial fibrillation. **A**: Focal activation. The initiating focus (indicated by the dot) often lies within the region of the pulmonary veins. The resulting wavelets represent fibrillatory conduction, as in multiple-wavelet reentry. **B**: Multiple-wavelet reentry. Wavelets (indicated by arrows) randomly reenter tissue previously activated by the same or another wavelet. The routes the wavelets travel vary. LA, left atrium; PV, pulmonary vein; ICV, inferior vena cava; SCV, superior vena cava; RA, right atrium. (Reproduced with permission, from Konings KT, Kirchhof CJ, Smeets JR, et al: High-density mapping of electrically induced atrial fibrillation in humans. Circulation 1994 Apr;89(4):1665-1680.)

Should AF develop postoperatively, ventricular rate response can be controlled with AV nodal blocking agents, unless contraindicated. Should AF result in hemodynamic instability, synchronized cardioversion can be attempted. Patients at high risk of AF following cardiac surgery can be treated with prophylactic amiodarone.

AF is most frequently associated with loss of atrial muscle and the development of fibrosis. Fibrosis may contribute to reentrant mechanisms of AF as depolarization/repolarization becomes nonhomogeneous. AF may also develop from a focal source often located in the pulmonary veins. In patients with an accessory bundle, AF can produce rapid ventricular responses and hemodynamic collapse. Drugs that slow conduction across the AV node (eg, digitalis, verapamil, diltiazem) do not slow conduction across the accessory pathway, potentially leading to hemodynamic collapse. The ACC/AHA guidelines likewise recommend caution in the use of β-blockers for AF in patients with preexcitation syndromes.

Ventricular arrhythmias have been the subject of much review by the AHA (**Table 21–11**). Ventricular premature contractions (VPCs) can appear perioperatively secondary to electrolyte abnormalities (hypokalemia, hypomagnesium, hypocalcemia), acidosis, ischemia, embolic phenomenon, mechanical irritation of the heart from central lines, cardiac manipulation, and drug effects. Correction of the underlying source of any arrhythmia should be addressed. Patients can likewise present with VPCs secondary to various cardiomyopathies (dilated, hypertrophic, and arrhythmogenic right ventricular).

The incidence of sudden cardiac death (SCD) is estimated at 1-2/1000 per year. Consequently, some patients will experience an unexpected death in the perioperative period. All anesthesia providers must be prepared to resuscitate and manage patients with ventricular arrhythmias, including VT (nonsustained and sustained) and ventricular fibrillation.

Nonsustained ventricular tachycardia is a short run of ventricular ectopy that lasts <30 sec and spontaneously terminates, whereas sustained VT persists longer than 30 seconds. VT is either monomorphic or polymorphic, depending on the QRS complex. If the QRS complex morphology changes, it is designated as polymorphic VT. Torsades de pointes is a form of VT associated with a prolonged QT interval, producing a sine wave-like VT pattern on the ECG. Ventricular fibrillation requires immediate resuscitative efforts and defibrillation.

Patients presenting with ventricular ectopy and nonsustained runs of VT should undergo investigation prior to surgery. Supraventricular and ventricular arrhythmias constitute active cardiac conditions that warrant evaluation and treatment prior to elective, noncardiac surgery. Exercise testing, echocardiography, and nuclear perfusion studies are all recommended by the ACC/AHA in patients with ventricular arrhythmias as part of their workup and management. Electrophysiologic studies are undertaken to determine the possibility for catheter-mediated ablation of ventricular tachycardias.

Should VT present perioperatively, cardioversion is recommended at any point where hemodynamic compromise occurs. Otherwise, treatment with amiodarone or procainamide can be attempted. At all times, therapy should also be directed at identifying any causative sources of the arrhythmia. β-Blockers are useful in the treatment of VT, especially if ischemia is a suspected causative factor in the development of rhythm. The use of β-blockers following myocardial infarction has reduced the incidence of post-MI ventricular fibrillation.

Torsades de pointes is associated with conditions that lengthen the QT interval. If the arrhythmia develops in association with pauses, pacing can be effective. Likewise, some patients may benefit from isoproterenol infusions, if they develop pause-dependent torsades de pointes. Magnesium sulfate may be useful in patients with long QT syndrome and episodes of torsades.

The development of perioperative ventricular fibrillation (VF) requires defibrillation and the use of resuscitation algorithms. Amiodarone can be used to stabilize the rhythm following successful defibrillation.

Following VF, patients can present to surgery for both ICD placement and other surgical procedures. ICDs are recommended in patients with a history of survived sudden cardiac death (SCD), decreased ventricular function following

TABLE 21–11 Classification of ventricular arrhythmias.

Classification by Clinical Presentation		
Hemodynamically stable	Asymptomatic	The absence of symptoms that could result from an arrhythmia.
	Minimal symptoms, e.g., palpitations	Patient reports palpitations felt in either the chest, throat, or neck as described by the following: • Heartbeat sensations that feel like pounding or racing • An unpleasant awareness of heartbeat • Feeling skipped beats or a pause
Hemodynamically unstable	Presyncope	Patient reports presyncope as described by the following: • Dizziness • Lightheadedness • Feeling faint • "Graying out"
	Syncope	Sudden loss of consciousness with loss of postural tone, not related to anesthesia, with spontaneous recovery as reported by the patient or observer. Patient may experience syncope when supine.
	Sudden cardiac death	Death from an unexpected circulatory arrest, usually due to a cardiac arrhythmia occurring within an hour of the onset of symptoms.
	Sudden cardiac arrest	Death from an unexpected circulatory arrest, usually due to a cardiac arrhythmia occurring within an hour of the onset of symptoms, in whom medical intervention (e.g., defibrillation) reverses the event.
Classification by Electrocardiography		
Nonsustained VT		Three or more beats in duration, terminating spontaneously in less than 30 s. VT is a cardiac arrhythmia of three or more consecutive complexes in duration emanating from the ventricles at a rate of greater than 100 bpm (cycle length less than 600 ms)
	Monomorphic	Nonsustained VT with a single QRS morphology.
	Polymorphic	Nonsustained VT with a changing QRS morphology at cycle length between 600 and 180 ms.
Sustained VT		VT greater than 30 s in duration and/or requiring termination due to hemodynamic compromise in less than 30 s.
	Monomorphic	Sustained VT with a stable single QRS morphology.
	Polymorphic	Sustained VT with a changing or multiform QRS morphology at cycle length between 600 and 180 ms.
Bundle-branch reentrant tachycardia		VT due to reentry involving the His-Purkinje system, usually with LBBB morphology; this usually occurs in the setting of cardiomyopathy.
Bidirectional VT		VT with a beat-to-beat alternans in the QRS frontal plane axis, often associated with digitalis toxicity.
Torsades de pointes		Characterized by VT associated with a long QT or QTc, and electrocardiographically characterized by twisting of the peaks of the QRS complexes around the isoelectric line during the arrhythmia: • "Typical," initiated following "short-long-short" coupling intervals. • Short coupled variant initiated by normal-short coupling.

(*continued*)

TABLE 21–11 Classification of ventricular arrhythmias. (continued)

Classification by Electrocardiography	
Ventricular flutter	A regular (cycle length variability 30 ms or less) ventricular arrhythmia approximately 300 bpm (cycle length—200 ms) with a monomorphic appearance; no isoelectric interval between successive QRS complexes.
Ventricular fibrillation	Rapid, usually more than 300 bpm/200 ms (cycle length 180 ms or less), grossly irregular ventricular rhythm with marked variability in QRS cycle length, morphology, and amplitude.
Classification by Disease Entity	
Chronic coronary heart disease Heart failure Congenital heart disease Neurological disorders Structurally normal hearts Sudden infant death syndrome Cardiomyopathies: Dilated cardiomyopathy Hypertrophic cardiomyopathy Arrhythmogenic right ventricular cardiomyopathy	

LBBB, left bundle-branch block; VT, ventricular tachycardia.

Reproduced, with permission, from Zipes DP, Camm AJ, Borggrefe M, et al: ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death—executive summary. Circulation 2006;114:1088.

MI, and left ventricular ejection fractions <35%. Additionally, ICDs are used to treat potential sudden cardiac death in patients with dilated, hypertrophic, arrhythmogenic right ventricular, and genetic cardiomyopathies.

ICDs usually have a biventricular pacing function that improves the effectiveness of left ventricular contraction. Patients with heart failure frequently have a widened QRS complex >120 msec. In such patients, ventricular systole is less efficient, as the lateral and septal left ventricular walls do not effectively contract because of the conduction delay. Cardiac resynchronizaton therapy (CRT) has been shown to improve functional status in patients with heart failure (Table 21–12).

Anesthetic management for the placement of ICDs and other electrophysiologic procedures (eg, catheter ablation) depends on the patient's underlying conditions. Many patients present with systolic and diastolic heart failure, and, as such, are dependent on sympathetic tone to maintain blood pressure. Many patients tolerate ICD placement using deep sedation rather than general anesthesia. However, catheter-based electrophysiologic studies can be quite time consuming, and patients can develop atelectasis and airway obstruction. Should the patient's blood pressure suddenly decline during electrophysiololgic studies, development of pericardial tamponade should be ruled out. Emergent drainage of tamponade may be necessary.

TABLE 21–12 Functional benefits of CRT.

↑ 6-Minute walking distance
↑ Health-related quality-of-life score
↑ Peak oxygen consumption
↓ Hospitalizations for decompensated heart failure
↓ NYHA functional classification

↑ indicates increased; ↓, decreased.

Reproduced, with permission, from Strickberger SA, Conti J, Daoud EG, et al: Patient selection for cardiac resynchronization therapy: from the Council on Clinical Cardiology Subcommittee on Electrocardiogrpahy and Arrhythmias and the Quality of Care and Outcomes Research Interdisciplinary Working Group, in Collaboration with the Heart Rhythm Society. Circulation 2005;111:2146.

Many patients present to surgery with ICDs in place. Published guidelines of the American Society of Anesthesiologists can provide assistance in the management of such patients.

Management is a three-step process, as follows:

- *Preoperative*. Identify the type of device and determine if it is used for antibradycardia functions. Consult with the patient's cardiologist preoperatively as to the device's function and use history.
- *Intraoperative*. Determine what electromagnetic interference is likely to present intraoperatively and advise the use of bipolar electrocautery where possible. Assure the availability of temporary pacing and defibrillation equipment and apply pads as necessary. Patients who are pacer dependent can be programmed to an asynchronous mode to mitigate electrical interference. Magnet application to ICDs may disable the antitachycardia function, but not convert to an asynchronous pacemaker. Consultation with the patient's cardiologist and interrogation of the device is advised.
- *Postoperative*. The device must be interrogated to ensure that therapeutic functions have been restored. Patients should be continuously monitored until the antitachycardia functions of the device are restored and its function has been confirmed.

ICDs are particularly problematic intraoperatively when electrocautery is used because the device may (1) interpret cautery as ventricular fibrillation; (2) inhibit pacemaker function due to cautery artifact; (3) increase the pacing rate due to activation of a rate-responsive sensor; or (4) temporarily or permanently reset to a backup or reset mode. Use of bipolar cautery, placement of the grounding pad far from the ICD device, and limiting use of the cautery to only short bursts help to reduce the likelihood of problems, but will not eliminate them.

ICD devices should have the defibrillator function programmed off immediately before surgery and reprogrammed back on immediately afterward. External defibrillation pads should be applied and attached to a defibrillator machine intraoperatively. Careful monitoring of the arterial pulse with pulse oximetry or an arterial waveform is necessary to ensure that the pacemaker is not inhibited and that there is arterial perfusion during episodes of ECG artifact from surgical cautery. The manufacturer should be contacted to determine the best method for managing the device (eg, reprogramming or applying a magnet) prior to surgery. A large number of ICD models are in use; however, most suspend their antitachycardia function in response to a magnet.

HEART FAILURE

An increasing number of patients present for surgery with either systolic and/or diastolic heart failure. Congestive heart failure affects more than 5 million Americans. Heart failure may be secondary to ischemia, valvular heart disease, infectious agents, and many types of cardiomyopathy. Most patients seek medical attention secondary to heart failure because of complaints of dyspnea and fatigue. Heart failure develops over time, as symptoms worsen (**Figure 21–5**). Patients generally undergo echocardiography to diagnose structural heart defects, to detect signs of cardiac "remodeling", to determine the left ventricular ejection fraction, and to assess the heart's diastolic function. Laboratory evaluations of concentration of brain natriuretic peptide (BNP) are likewise obtained to distinguish heart failure from other causes of dyspnea. BNP is released from the heart, and its elevation is associated with impaired ventricular function.

In response to ventricular failure, the body attempts to compensate for LV systolic function through the sympathetic and renin–angiotensin–aldosterone system. Consequently, patients experience salt retention, volume expansion, sympathetic stimulation, and vasoconstriction. The heart dilates to maintain the stroke volume in spite of decreased contractility. Over time, compensatory mechanisms fail and contribute to the symptoms associated with heart failure (eg, edema, tachycardia, decreased tissue perfusion). Patients with systolic heart failure are likely to present to surgery having been previously treated with diuretics, ACE inhibitors, angiotensin receptor blockers, and

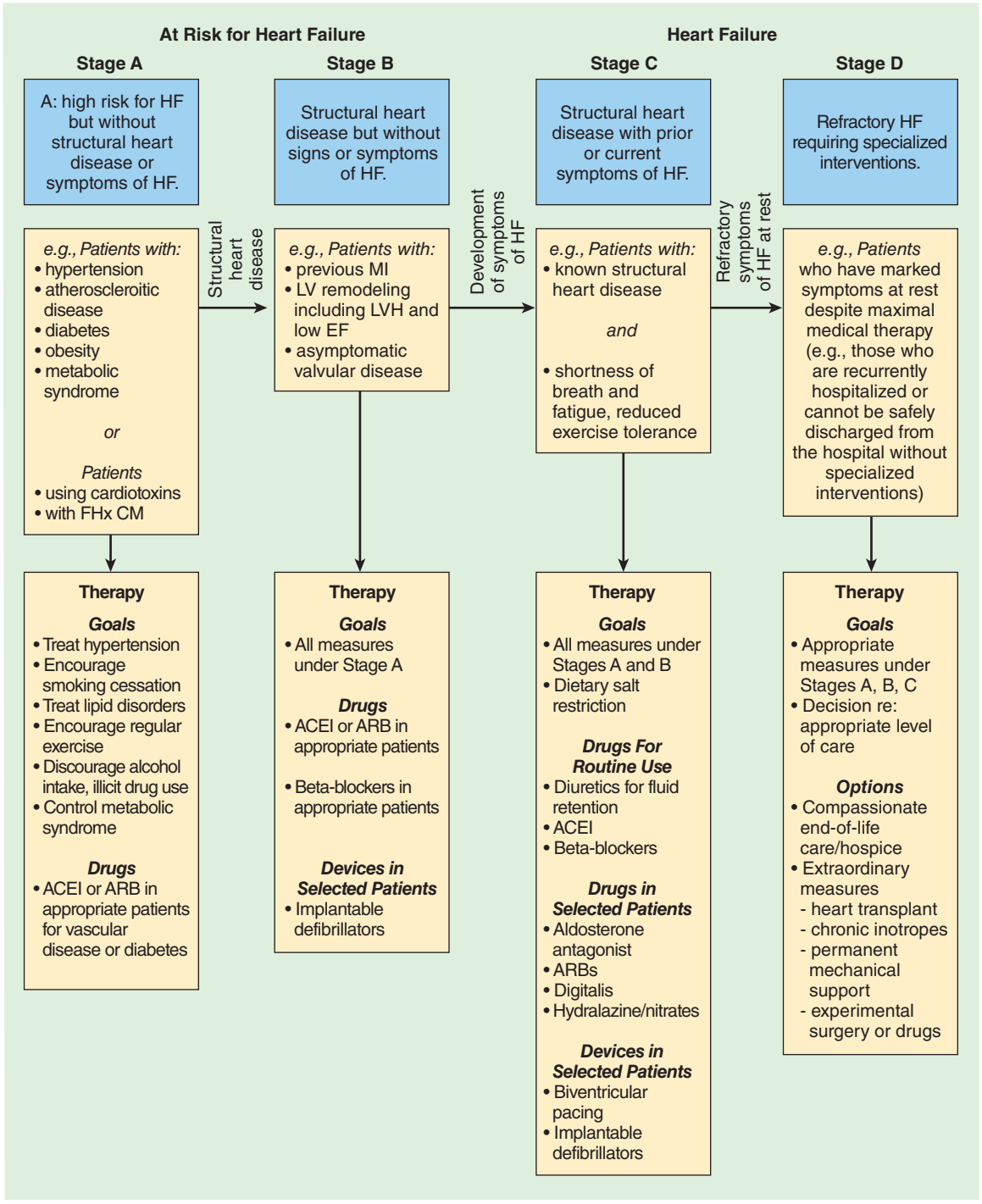

FIGURE 21–5 Stages in the development of heart failure/recommended therapy by stage. ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; EF, ejection fraction; FHx CM, family history of cardiomyopathy; HF, heart failure; LVH, left ventricular hypertrophy; MI, myocardial infarction. (Reproduced, with permission, from Jessup M, Abraham W, Casey D, et al: 2009 Focused update: ACCF/AHA guidelines for the diagnosis and management of heart failure in adults: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation 2009;119:1977.)

possibly aldosterone antagonists. Electrolytes must be measured, as heart failure therapies frequently lead to changes in serum potassium concentration. Angiotensin receptor blocker or ACE inhibitor use may contribute to periinduction hypotension in the patient with heart failure. ACE inhibitors are rarely associated with angioedema requiring emergent airway management.

Diastolic ventricular dysfunction produces symptoms of congestion and heart failure. Myocardial relaxation is a dynamic, not passive, process. The heart with preserved diastolic function accommodates volume during diastole, with minimal increases in left ventricular end-diastolic pressure. Conversely, the heart with diastolic dysfunction relaxes poorly and produces increased left ventricular end-diastolic pressure. The left ventricular end-diastolic pressure is transmitted to the left atrium and pulmonary vasculature resulting in symptoms of congestion.

Anesthetic management of the patient with heart failure requires careful assessment and optimization of intravascular fluid volume—especially if positive inotropic agents, vasoconstrictors, or vasodilators are used. In particular, patients with diastolic dysfunction may tolerate increases in volume poorly, leading to pulmonary congestion.

HYPERTROPHIC CARDIOMYOPATHY

Hypertrophic cardiomyopathy (HCM) is an autosomal dominant trait that affects 1 in 500 adults. Many patients are unaware of the condition, and some will present with SCD as an initial manifestation. Symptoms include dyspnea, exercise intolerance, palpitations, and chest pain. Clinically, HCM is detected by the murmur of dynamic left ventricular outflow tract (LVOT) obstruction in late systole. Symptomatic patients frequently have a thickened intraventricular septum of 20 to 30 mm. Mutations in the genes that code for the cardiac sarcomeres and their supporting proteins are implicated. The myocardium of the intraventricular septum is abnormal, and many patients can develop diastolic dysfunction and SCD without pronounced dynamic obstructive gradients. During systole, the anterior leaflet of the mitral valve abuts the intraventricular septum (Figure 21–6), producing obstruction and a late systolic murmur.

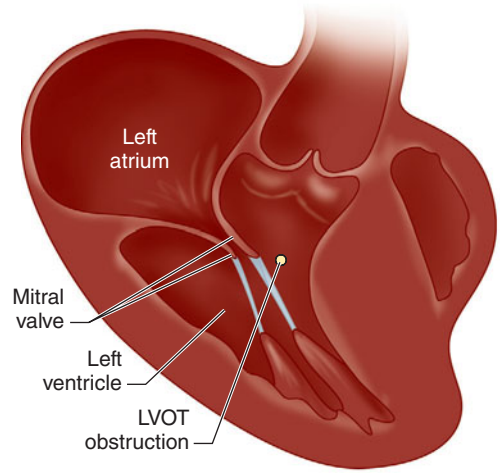

FIGURE 21–6 The midesophageal long axis view is shown. As a consequence of the hypertrophied interventricular septum, flow patterns within the heart are altered so that the anterior leaflet of the mitral valve is drawn during ventricular systole into the left ventricular outflow tract (LOVT), producing obstruction. This is known as systolic anterior motion of the mitral valve (SAM). (Reproduced, with permission, from Wasnick J, Hillel Z, Kramer D, et al: *Cardiac Anesthesia & Transesophageal Echocardiography,* McGraw-Hill, 2011.)

Perioperative management is aimed at minimizing the degree of LVOT obstruction. This is accomplished by maintaining adequate intravascular volume, avoiding vasodilatation, and reducing myocardial contractility through the use of β-blockers.

Valvular Heart Disease

1. General Evaluation of Patients

Regardless of the lesion or its cause, preoperative evaluation should be primarily concerned with determining the identity and severity of the lesion and its hemodynamic significance, residual ventricular function, and the presence of any secondary effects on pulmonary, renal, and hepatic function.

Concomitant CAD should not be overlooked, particularly in older patients and those with known risk factors (see above). Myocardial ischemia may also occur in the absence of significant coronary occlusion in patients with severe aortic stenosis or regurgitation.

History

The preanesthesia history should focus on symptoms related to decreased ventricular function. Symptoms and signs should be correlated with laboratory data. Questions should evaluate exercise tolerance, fatigability, and pedal edema and shortness of breath in general (dyspnea), when lying flat (orthopnea), or at night (paroxysmal nocturnal dyspnea). The New York Heart Association functional classification of heart disease (Table 21–13) is useful for grading the severity of heart failure symptoms and estimating prognosis. Patients should also be questioned about chest pains and neurological symptoms. Some valvular lesions are associated with thromboembolic phenomena. Prior procedures, such as valvotomy or valve replacement and their effects, should also be well documented.

A review of medications should evaluate efficacy and exclude serious side effects. Commonly used agents include diuretics, vasodilators, ACE inhibitors, β-blockers, antiarrhythmics, and anticoagulants. Preoperative vasodilator therapy may be used to decrease preload, afterload, or both. Excessive vasodilatation worsens exercise tolerance and is often first manifested as postural (orthostatic) hypotension.

TABLE 21–13 Modified New York Association functional classification of heart disease.

Class	Description
I	Asymptomatic except during severe exertion
II	Symptomatic with moderate activity
III	Symptomatic with minimal activity
IV	Symptomatic at rest

Physical Examination

The most important signs to identify on physical examination are those of congestive heart failure. Left-sided (S_3 gallop or pulmonary rales) and right-sided (jugular venous distention, hepatojugular reflux, hepatosplenomegaly, or pedal edema) signs may be present. Auscultatory findings may confirm the valvular dysfunction (Figure 21–7), but echocardiographic studies are more reliable. Neurological deficits, usually secondary to embolic phenomena, should be documented.

Laboratory Evaluation

In addition to the laboratory studies discussed for patients with hypertension and CAD, liver function tests may be useful in assessing hepatic dysfunction caused by passive hepatic congestion in patients with severe or chronic right-sided failure. Arterial blood gases can be measured in patients with significant pulmonary symptoms. Reversal of warfarin or heparin should be documented with a prothrombin time and international normalized ratio (INR) or partial thromboplastin time, respectively, prior to surgery.

Electrocardiographic findings are generally nonspecific. The chest radiograph is useful to assess cardiac size and pulmonary vascular congestion.

Special Studies

Echocardiography, imaging studies, and cardiac catheterization provide important diagnostic and prognostic information about valvular lesions, but should only be obtained if the results will change therapy or outcomes. More than one valvular lesion is often found. In many instances, noninvasive studies obviate the need for cardiac catheterization, unless there are concerns about CAD. Information from these studies is best reviewed with a cardiologist. The following questions must be answered:

- Which valvular abnormality is most important hemodynamically?
- What is the severity of an identified lesion?
- What degree of ventricular impairment is present?
- What is the hemodynamic significance of other identified abnormalities?
- Is there any evidence of CAD?

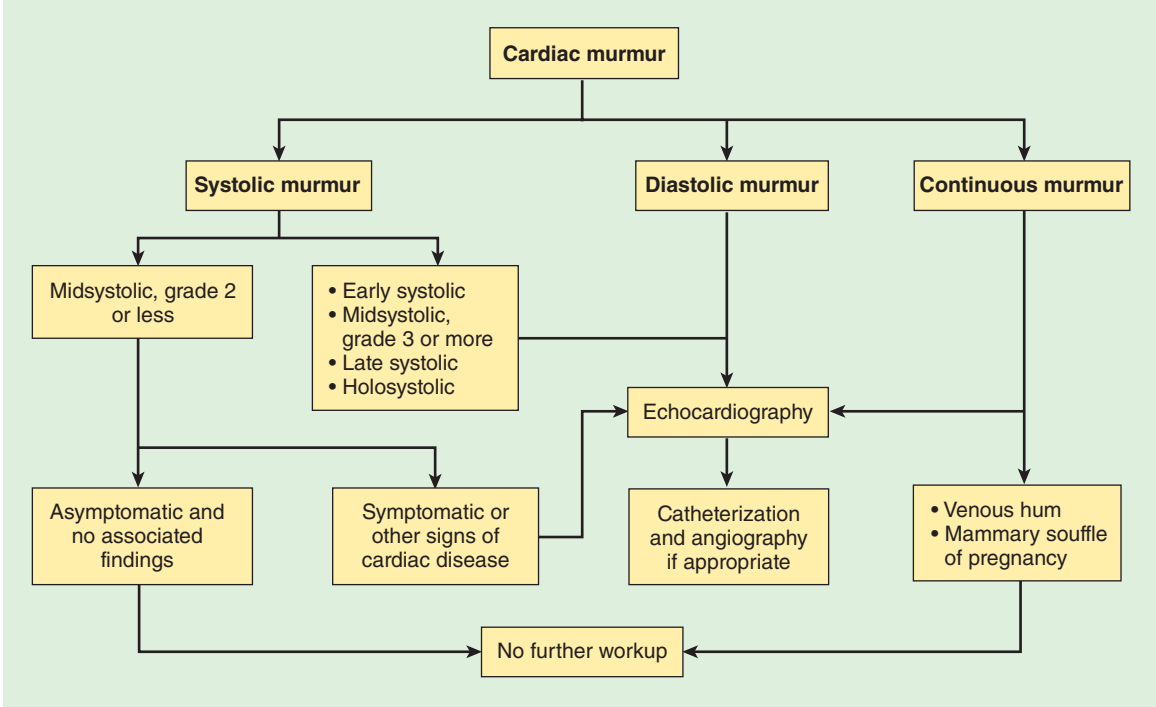

FIGURE 21–7 Strategy for evaluating heart murmurs. (Reproduced, with permission, from Bonow RO, Carabello BA, Chatterjee K, et al: 2008 focused update incorporated in the ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (writing committee to revise the 1998 guidelines for the management of patients with valvular heart disease): endorsed by the Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. Circulation 2008;118:e523.)

The ACC/AHA have prepared detailed guidelines to assist in the management of the patient with valvular heart disease. Although the evaluation of the patient with a heart murmur generally rests with the cardiologist, anesthesia providers will on occasion discover the presence of a previously undetected murmur on preanesthetic examination. In particular, anesthetists are concerned that undiagnosed, critical aortic stenosis might be present, which could potentially lead to hemodynamic collapse with either regional or general anesthesia. In the past, most valvular heart diseases were a consequence of rheumatic heart disease; however, with an aging surgical population, increasing numbers of patients have degenerative valve problems. More than one in eight patients older than age 75 years may manifest at least one form of moderate to severe valvular heart disease. A study conducted in the Netherlands reported that the prevalence of aortic stenosis was 2.4% in patients older than age 60 years who were scheduled for elective surgery. Underdiagnosed valvular disease is particularly prevalent in elderly females.

According to the ACC/AHA guidelines, auscultation of the heart is the most widely used method to detect valvular heart disease. Murmurs occur as a consequence of the accelerated blood flow through narrowed openings in stenotic and regurgitant lesions. Although systolic murmurs may be related to increased blood flow velocity, the ACC/AHA guidelines note that all diastolic and continuous murmurs reflect pathology. Other than murmurs that are thought to be innocent, such as mid-systolic flow murmurs (grade 2 or softer), the ACC/AHA guidelines recommend echocardiographic evaluation.

When new murmurs are detected in a preoperative evaluation, consultation with the patient's personal physician is helpful to determine the need for echocardiographic evaluation. In many centers, immediate echocardiographic evaluation can be performed in the preoperative area.

2. Specific Valvular Disorders

MITRAL STENOSIS

Preoperative Considerations

Mitral stenosis almost always occurs as a delayed complication of rheumatic fever. However, mitral stenosis can also occur in dialysis-dependent patients. Two-thirds of patients with mitral stenosis are female. The stenotic process is estimated to begin after a minimum of 2 years following rheumatic heart disease and results from progressive fusion and calcification of the valve leaflets. Symptoms generally develop after 20–30 years, when the mitral valve orifice is reduced from its normal 4–6 cm^2 opening to less than 1.5 cm^2. Less than 50% of patients have isolated mitral stenosis; the remaining patients also have mitral regurgitation, and up to 25% of patients also have rheumatic involvement of the aortic valve (stenosis or regurgitation).

Pathophysiology

The rheumatic process causes the valve leaflets to thicken, calcify, and become funnel shaped; annular calcification may also be present. The mitral commissures fuse, the chordae tendinae fuse and shorten, and the valve cusps become rigid; as a result, the valve leaflets typically display bowing or doming during diastole on echocardiography.

Significant restriction of blood flow through the mitral valve results in a transvalvular pressure gradient that depends on cardiac output, heart rate (diastolic time), and cardiac rhythm. Increases in either cardiac output or heart rate (decreased diastolic time) necessitate higher flows across the valve and result in higher transvalvular pressure gradients. The left atrium is often markedly dilated, promoting SVTs, particularly AF. Blood flow stasis in the atrium promotes the formation of thrombi, usually in the left atrial appendage. Loss of normal atrial systole with AF (which is usually responsible for 20% to 30% of ventricular filling) necessitates even higher diastolic flow across the valve to maintain the same cardiac output and increases the transvalvular gradient.

Acute elevations in left atrial pressure are rapidly transmitted back to the pulmonary capillaries. If mean pulmonary capillary pressure acutely and significantly rises transudation of capillary fluid may result in pulmonary edema. Chronic elevations in pulmonary capillary pressure are partially compensated by increases in pulmonary lymph flow, but eventually result in pulmonary vascular changes, leading to irreversible increases in pulmonary vascular resistance (PVR) and pulmonary hypertension. Reduced lung compliance and a secondary increase in the work of breathing contribute to chronic dyspnea. Right ventricular failure is frequently precipitated by acute or chronic elevations in right ventricular afterload. Marked dilatation of the right ventricle can result in tricuspid or pulmonary valve regurgitation.

Embolic events are common in patients with mitral stenosis and AF. Dislodgment of clots from the left atrium results in systemic emboli, most commonly to the cerebral circulation. Patients also have an increased incidence of pulmonary emboli, pulmonary infarction, hemoptysis, and recurrent bronchitis. Hemoptysis most commonly results from rupture of pulmonary–bronchial venous communications. Chest pain occurs in 10% to 15% of patients with mitral stenosis, even in the absence of CAD; its etiology often remains unexplained, but may be emboli in the coronary circulation or acute right ventricular pressure overload. Patients may develop hoarseness as a result of compression of the left recurrent laryngeal nerve by the enlarged left atrium.

Left ventricular function is normal in the majority of patients with pure mitral stenosis (**Figure 21–8**), but impaired left ventricular function may be encountered in up to 25% of patients and presumably represents residual damage from rheumatic myocarditis or coexistent hypertensive or ischemic heart disease.

The left ventricle is chronically underloaded in the patient with mitral stenosis. At the same time, the left atrium, right ventricle, and right atrium are frequently dilated and dysfunctional. Vasodilatation that occurs following both neuraxial and general

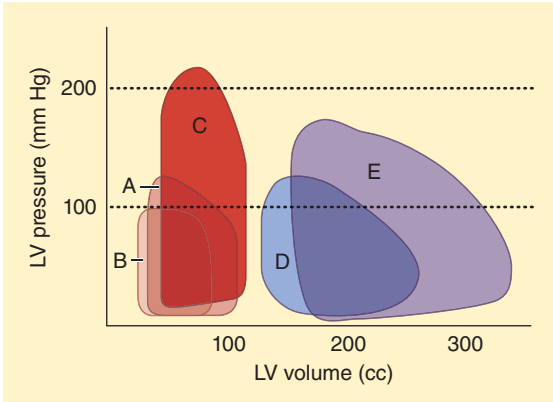

FIGURE 21–8 Pressure–volume loops in patients with valvular heart disease. A, normal; B, mitral stenosis; C, aortic stenosis; D, mitral regurgitation (chronic); E, aortic regurgitation (chronic). LV, left ventricular. (Reproduced, with permission, from Jackson JM, Thomas SJ, Lowenstein E: Anesthetic management of patients with valvular heart disease. Semin Anesth 1982;1:239.)

anesthesia can lead to peripheral venous blood pooling and inadequate volume delivery to the left ventricle. This can precipitate hemodynamic collapse.

Calculating Mitral Valve Area & Transvalvular Gradient

Two-dimensional and Doppler echocardiography can be used to estimate both the pressure drop across a stenotic valve and the valve area. Based on the assumption that the velocity of blood flow is much greater distal than proximal to an obstruction, the Bernoulli equation can be simplified:

$$\Delta P = 4V^2$$

where ΔP is the pressure gradient (mm Hg) and V is blood flow velocity (m/s) distal to the obstruction. Valve orifice can be estimated from the time it takes for the initial peak pressure gradient to fall to one-half of its original value, the pressure half-time ($T_{1/2}$). This relationship is approximated by

$$A = \frac{220}{T_{1/2}}$$

where A is valve orifice (cm^2) and $T_{1/2}$ is the time from peak flow velocity (V_{max}) to $V_{max}/1.4$. This relationship is based on the observation that $T_{1/2}$ remains relatively constant for a given orifice over a wide range of flows. A pressure half-time of 220 msec corresponds to a mitral valve area of 1 cm^2.

Mitral valve areas less than 1 cm^2 are typically associated with transvalvular gradients of 20 mm Hg at rest and dyspnea with minimal exertion; a mitral valve area less than 1 cm^2 is often referred to as critical mitral stenosis. Patients with valve areas between 1.5 and 2.0 cm^2 are generally asymptomatic or have only mild symptoms with exertion. When the mitral valve area is between 1 and 1.5 cm^2, most patients are symptomatic with mild to moderate exertion. Although cardiac output may be normal at rest, it fails to increase appropriately during exertion because of decreased left ventricular preload.

Treatment

The time from onset of symptoms to incapacitation averages 5–10 years. At that stage, most patients die within 2–5 years. Surgical correction is therefore usually undertaken once significant symptoms develop. Percutaneous transseptal balloon valvuloplasty may be used in selected young or pregnant patients, as well as older patients who are poor surgical candidates. Medical management is primarily supportive and includes limitation of physical activity, sodium restriction, and diuretics. Small doses of a β-adrenergic blocking drug may also be useful in controlling heart rate in patients with mild to moderate symptoms. Patients with a history of emboli and those at high risk (age older than 40 years; a large atrium with chronic atrial fibrillation) are usually anticoagulated.

Anesthetic Management

A. Objectives

8 The principal hemodynamic goals are to maintain a sinus rhythm (if present preoperatively) and to avoid tachycardia, large increases in cardiac output, and both hypovolemia and fluid overload by judicious administration of intravenous fluids.

B. Monitoring

Invasive hemodynamic monitoring is often used for major surgical procedures, particularly those associated with large fluid shifts. TEE can also be used to

help guide perioperative management. Overzealous fluid replacement readily precipitates pulmonary edema in patients with severe disease. Pulmonary capillary wedge pressure measurements in the presence of mitral stenosis reflect the transvalvular gradient and not necessarily left ventricular end-diastolic pressure. Prominent *a* waves and a decreased *y* descent are typically present on the pulmonary capillary wedge pressure waveform in patients who are in sinus rhythm. A prominent *cv* wave on the central venous pressure waveform is usually indicative of secondary tricuspid regurgitation. The ECG typically shows a notched P wave in patients who are in sinus rhythm.

C. Choice of Agents

Patients may be very sensitive to the vasodilating effects of spinal and epidural anesthesia. Epidural anesthesia may be easier to manage than spinal anesthesia because of the more gradual onset of sympathetic blockade. There is no "ideal" general anesthetic, and agents should be employed to achieve the desired effects of permitting sufficient diastolic time to adequately load the left ventricle. Vasopressors are often needed to maintain vascular tone following anesthetic induction.

Intraoperative tachycardia may be controlled by deepening anesthesia with an opioid (excluding meperidine) or β-blocker (esmolol or metoprolol). In the presence of atrial fibrillation, ventricular rate should be controlled. **Marked hemodynamic deterioration from sudden SVT necessitates cardioversion.** Phenylephrine is preferred over ephedrine as a vasopressor because the former lacks β-adrenergic agonist activity. Vasopressin can also be employed to restore vascular tone should hypotension develop secondary to anesthetic induction.

MITRAL REGURGITATION

Preoperative Considerations

Mitral regurgitation can develop acutely or insidiously as a result of a large number of disorders. Chronic mitral regurgitation is usually the result of rheumatic fever (often with concomitant mitral stenosis); congenital or developmental abnormalities of the valve apparatus; or dilatation, destruction, or calcification of the mitral annulus. Acute mitral regurgitation is usually due to myocardial ischemia or infarction (papillary muscle dysfunction or rupture of a chorda tendinea), infective endocarditis, or chest trauma.

Pathophysiology

The principal derangement is a reduction in forward stroke volume due to backward flow of blood into the left atrium during systole. The left ventricle compensates by dilating and increasing end-diastolic volume (Figure 21–8). Regurgitation thorugh the mitral valve initially maintains a normal end systolic volume in spite of an increased end diastolic volume. However, as the disease progresses the end systolic volume increases. By increasing end-diastolic volume, the volume-overloaded left ventricle can maintain a normal cardiac output despite blood being ejected retrograde into the atrium. With time, patients with chronic mitral regurgitation eventually develop eccentric left ventricular hypertrophy and progressive impairment in contractility. In patients with severe mitral regurgitation, the regurgitant volume may exceed the forward stroke volume. In time, wall stress increases, resulting in an increased demand for myocardial oxygen supply.

The regurgitant volume passing through the mitral valve is dependent on the size of the mitral valve orifice (which can vary with ventricular cavity size), the heart rate (systolic time), and the left ventricular–left atrial pressure gradient during systole. The last factor is affected by the relative resistances of the two outflow paths from the left ventricle, namely, SVR and left atrial compliance. Thus, a decrease in SVR or an increase in mean left atrial pressure will reduce the regurgitant volume. Atrial compliance also determines the predominant clinical manifestations. Patients with normal or reduced atrial compliance (acute mitral regurgitation) have primarily pulmonary vascular congestion and edema. Patients with increased atrial compliance (long-standing mitral regurgitation resulting in a large dilated left atrium) primarily show signs of a reduced cardiac output. Most patients are between the two extremes and exhibit symptoms of both pulmonary congestion and low cardiac output. Patients with a regurgitant fraction of less than 30% of the total stroke volume generally have mild symptoms. Regurgitant fractions of 30% to 60% generally cause

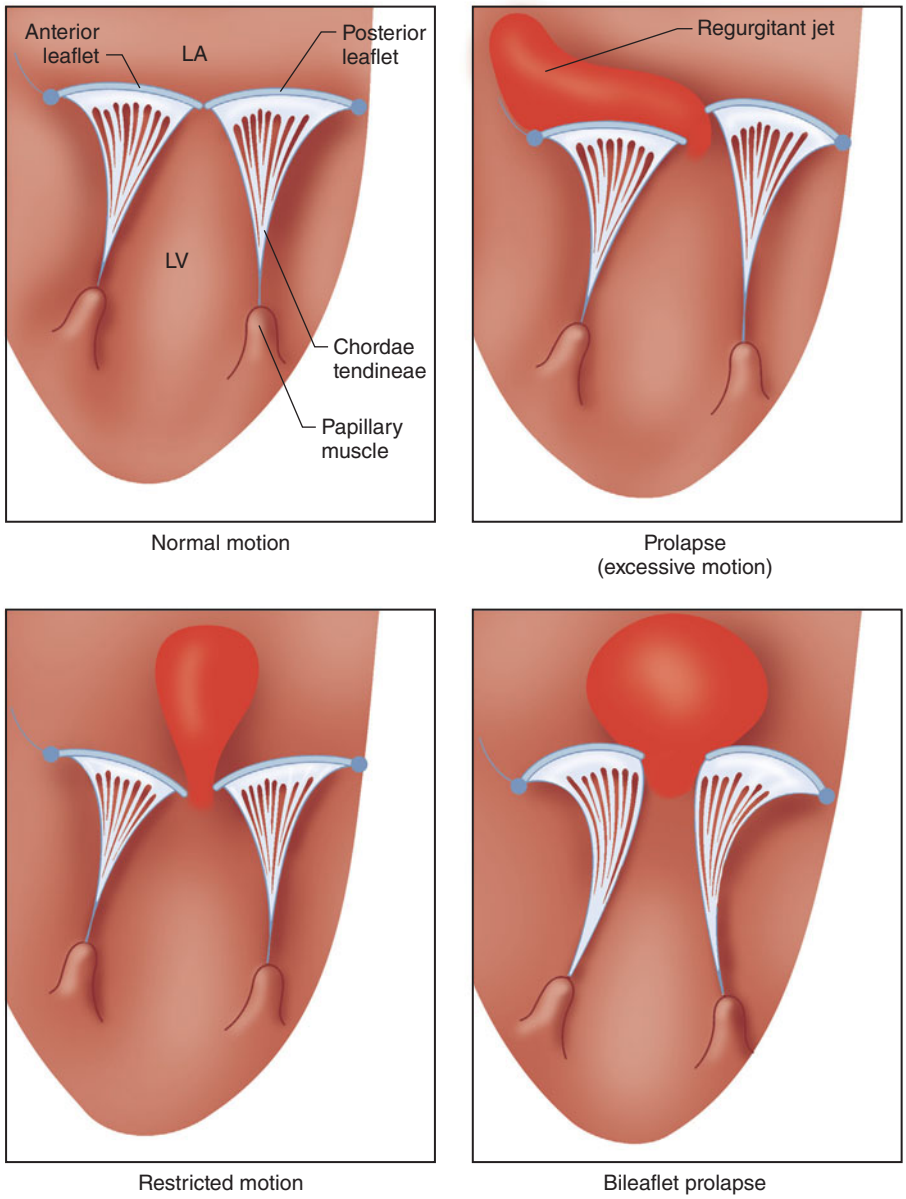

FIGURE 21–9 Classification of mitral valve leaflet motion (as seen from transesophageal echocardiography). Note that with prolapse, the free edge of the leaflet(s) extends beyond the plane of the mitral annulus producing an eccentric jet. With restricted motion, the leaflets fail to coapt, resulting in a central jet.

moderate symptoms, whereas fractions greater than 60% are associated with severe disease.

Echocardiography, particularly TEE, is useful in delineating the underlying pathophysiology of mitral regurgitation and guiding treatment. Mitral valve leaflet motion is often described as normal, prolapsing, or restrictive (Figure 21–9). Excessive motion or prolapse is defined by systolic movement of a leaflet beyond the plane of the mitral valve and into the left atrium (see the section below on mitral valve prolapse).

Calculating Regurgitant Fraction

To calculate regurgitant fraction (RF), forward stroke volume (SV) and the regurgitant stroke volume (RSV) must be measured. Although they can both be estimated by catheterization data, pulsed Doppler echocardiography provides reasonably acute calculations. Stroke volume is measured at the left ventricular outflow tract (LVOT) and at the mitral valve (MV), where

$$\text{Stroke volume} = \text{cross-sectional area } (A) \times (\text{TVI})$$

and cross-sectional area (A) can be approximated as,

$$A = 0.785 \times (\text{diameter})^2$$

The time–velocity integral (TVI) is the integral of the velocity versus the time signal obtained with pulsed Doppler. The TVI reflects the distance the blood has traveled during a heart beat. By knowing the area through which the blood travels and the distance traveled, it is possible to estimate the stroke volume. This is the case because the area is expressed in centimeters squared, and the distance is expressed in centimeters. The product of these measures is cubic centimeters or milliliters—hence, the stroke volume for each heartbeat.

Thus, the volume of blood that enters through the mitral valve must be the same as that passing through the left ventricular outflow track. Any difference between the two represents the amount of the volume that initially entered the left ventricle, but that did not pass the LVOT. This is the volume that regurgitated into the left atrium.

$$RSV_{\text{mitral regurgitation}} = (A_{MV} \times VTI_{MV}) - (A_{LVOT} \times TVI_{LVOT}),$$

and

$$RF = RSV/SV$$

An RSV greater than 65 mL usually correlates with severe mitral regurgitation.

Treatment

Afterload reduction is beneficial in most patients and may even be lifesaving in patients with acute mitral regurgitation. Reduction of SVR increases forward SV and decreases the regurgitant volume. Surgical treatment is usually reserved for patients with moderate to severe symptoms. Valvuloplasty or valve repair are performed whenever possible to avoid the problems associated with valve replacement (eg, thromboembolism, hemorrhage, and prosthetic failure). Catheter-mediated valve repairs are continually being refined, potentially reducing the need for "open" surgery. Anesthesiologists skilled in advanced perioperative echocardiography assist in correctly identifying the leaflet(s) to be repaired and determining the repair's success. Three-dimensional echocardiography is increasingly employed to assist in the assessment of the mitral valve (see Figure 5-29).

Anesthetic Management

A. Objectives

9 Anesthetic management should be tailored to the severity of mitral regurgitation as well as the underlying left ventricular function. Factors that exacerbate the regurgitation, such as slow heart rates and acute increases in afterload, should be avoided. Bradycardia can increase the regurgitant volume by increasing left ventricular end-diastolic volume and acutely dilating the mitral annulus. The heart rate should ideally be kept between 80 and 100 beats/min. Acute increases in left ventricular afterload, such as with endotracheal intubation and surgical stimulation under "light" anesthesia, should be treated rapidly but without excessive myocardial depression. Excessive volume expansion can also worsen the regurgitation by dilating the left ventricle.

B. Monitoring

Monitors are based on the severity of ventricular dysfunction, as well as the procedure. Mitral regurgitation may be recognized on the pulmonary artery wedge waveform as a large v wave and a rapid y descent (**Figure 21–10**). The height of the v wave is inversely related to atrial and pulmonary vascular compliance, but is directly proportional to pulmonary blood flow and the regurgitant volume; thus, the v wave may not be prominent in patients with chronic mitral regurgitation, except during acute deterioration. Very large v waves are often apparent on the pulmonary artery pressure waveform, even without wedging the catheter. Color-flow Doppler TEE can be invaluable in quantitating the severity of

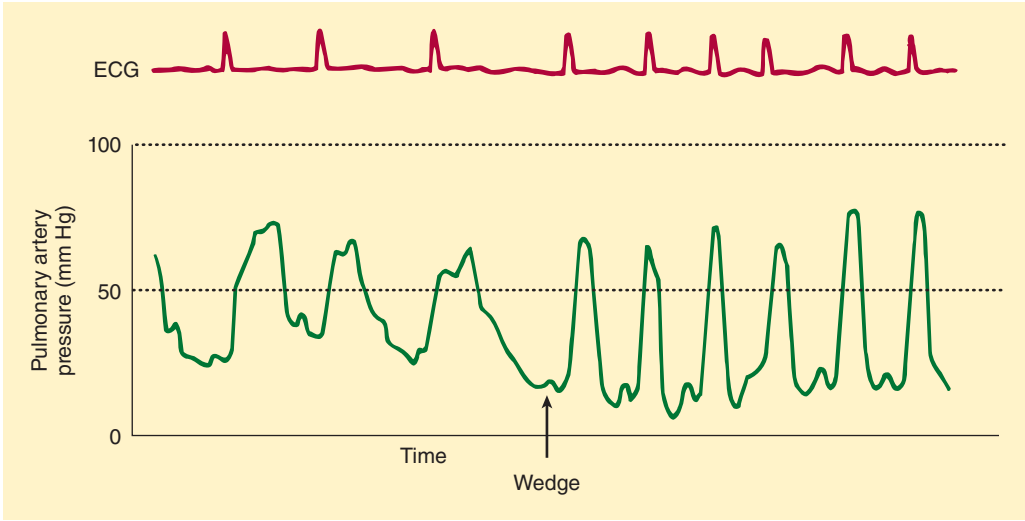

FIGURE 21–10 The pulmonary capillary wedge waveform in mitral regurgitation, demonstrating a large *v* wave.

the regurgitation and guiding therapeutic interventions in patients with severe mitral regurgitation. By definition, blood flow reverses in the pulmonary veins during systole with severe mitral regurgitation.

C. Choice of Agents

Patients with relatively well-preserved ventricular function tend to do well with most anesthetic techniques. Spinal and epidural anesthesia are well tolerated, provided bradycardia is avoided. Patients with moderate to severe ventricular impairment may be sensitive to depression from high concentrations of volatile agents. An opioid-based anesthetic may be more suitable for those patients—again, provided bradycardia is avoided.

MITRAL VALVE PROLAPSE

Preoperative Considerations

Mitral valve prolapse is classically characterized by a mid-systolic click, with or without a late apical systolic murmur on auscultation. It is a relatively common abnormality that is present in up to 1% to 2.5% of the general population. The diagnosis is based on auscultatory findings and is confirmed by echocardiography, which shows systolic prolapse of mitral valve leaflets into the left atrium. Patients with the murmur often have some element of mitral regurgitation. The posterior mitral leaflet is more commonly affected than the anterior leaflet. The mitral annulus may also be dilated. Pathologically, most patients have redundancy or some myxomatous degeneration of the valve leaflets. Most cases of mitral valve prolapse are sporadic or familial, affecting otherwise normal persons. A high incidence of mitral valve prolapse is found in patients with connective tissue disorders (particularly Marfan syndrome).

The overwhelming majority of patients with mitral valve prolapse are asymptomatic, but in a small percentage of patients, the myxomatous degeneration is progressive. Manifestations, when they occur, can include chest pains, arrhythmias, embolic events, florid mitral regurgitation, infective endocarditis, and, rarely, sudden death. The diagnosis can be made preoperatively by auscultation of the characteristic click, but must be confirmed by echocardiography. The prolapse is accentuated by maneuvers that decrease ventricular volume (preload). Both atrial and ventricular arrhythmias are common. Although bradyarrhythmias have been reported, paroxysmal supraventricular tachycardia is the most commonly encountered sustained arrhythmia. An increased incidence of abnormal AV bypass tracts is reported in patients with mitral valve prolapse.

Most patients have a normal life span. About 15% develop progressive mitral regurgitation. A smaller percentage develops embolic phenomena or infective endocarditis. Patients with both a click and a systolic murmur seem to be at greater risk of developing complications. Anticoagulation or antiplatelet agents may be used for patients with a history of emboli, whereas β-adrenergic blocking drugs are commonly used for arrhythmias.

Anesthetic Management

The management of these patients is based on their clinical course. Most patients are asymptomatic and do not require special care. Ventricular arrhythmias may occur intraoperatively, particularly following sympathetic stimulation, and will generally respond to lidocaine or β-adrenergic blocking agents. Mitral regurgitation caused by prolapse is generally exacerbated by decreases in ventricular size. Hypovolemia and factors that increase ventricular emptying or decrease afterload should be avoided. Vasopressors with pure α-adrenergic agonist activity (such as phenylephrine) may be preferable to those that are primarily β-adrenergic agonists (ephedrine).

AORTIC STENOSIS

Preoperative Considerations

Valvular aortic stenosis is the most common cause of obstruction to left ventricular outflow. Left ventricular outflow obstruction is less commonly due to hypertrophic cardiomyopathy, discrete congenital subvalvular stenosis, or, rarely, supravalvular stenosis. Valvular aortic stenosis is nearly always congenital, rheumatic, or degenerative. Abnormalities in the number of cusps (most commonly a bicuspid valve) or their architecture produce turbulence that traumatizes the valve and eventually leads to stenosis. Rheumatic aortic stenosis is rarely isolated; it is more commonly associated with aortic regurgitation or mitral valve disease. In the most common degenerative form, calcific aortic stenosis, wear and tear results in the buildup of calcium deposits on normal cusps, preventing them from opening completely (Figure 21–11).

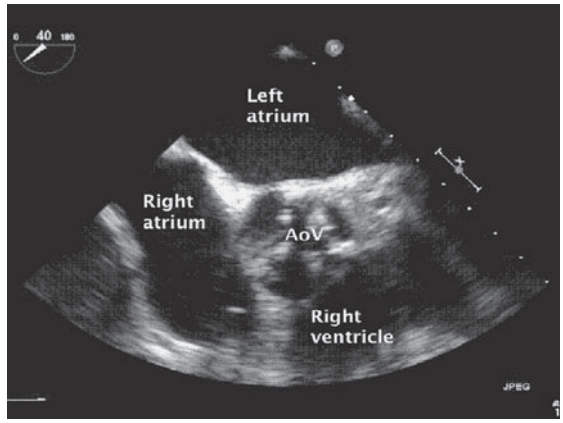

FIGURE 21–11 A stenotic aortic valve is clearly seen in this midesophageal short axis aortic valve view. Calcification of the aortic valve is usually associated with senile degeneration. However, congenitally abnormal (bicuspid) and rheumatic presentations also occur. (Reproduced, with permission, from Wasnick J, Hillel Z, Kramer D, et al: *Cardiac Anesthesia & Transesophageal Echocardiography,* McGraw-Hill, 2011.)

Pathophysiology

Left ventricular outflow obstruction caused by valvular aortic stenosis is almost always gradual, allowing the ventricle, at least initially, to compensate and maintain SV. Concentric left ventricular hypertrophy enables the ventricle to maintain SV by generating the needed transvalvular pressure gradient and to reduce ventricular wall stress.

Critical aortic stenosis is said to exist when the aortic valve orifice is reduced to 0.5–0.7 cm^2 (normal is 2.5–3.5 cm^2). With this degree of stenosis, patients generally have a transvalvular gradient of approximately 50 mm Hg at rest (with a normal cardiac output) and are unable to increase cardiac output in response to exertion. Moreover, further increases in the transvalvular gradient do not significantly increase SV. With long-standing aortic stenosis, myocardial contractility progressively deteriorates and compromises left ventricular function.

Classically, patients with advanced aortic stenosis have the triad of dyspnea on exertion, angina, and orthostatic or exertional syncope. A prominent feature of aortic stenosis is a decrease in left ventricular compliance as a result of hypertrophy.

Diastolic dysfunction is the result of an increase in ventricular muscle mass, fibrosis, or myocardial ischemia. In contrast to left ventricular end-diastolic volume, which remains normal until very late in the disease, left ventricular end-diastolic pressure is elevated early in the disease. The decreased diastolic pressure gradient between the left atrium and left ventricle impairs ventricular filling, which becomes quite dependent on a normal atrial contraction. Loss of atrial systole can precipitate congestive heart failure or hypotension in patients with aortic stenosis. Cardiac output may be normal in symptomatic patients at rest, but characteristically, it does not appropriately increase with exertion. Patients may experience angina even in the absence of CAD. Myocardial oxygen demand increases because of ventricular hypertrophy, whereas myocardial oxygen supply decreases as a result of the marked compression of intramyocardial coronary vessels caused by high intracavitary systolic pressures (up to 300 mm Hg). Exertional syncope or near-syncope is thought to be due to an inability to tolerate the vasodilatation in muscle tissue during exertion. Arrhythmias leading to severe hypoperfusion may also account for syncope and sudden death in some patients.

CALCULATING AORTIC VALVE AREA & TRANSVALVULAR GRADIENT

As with mitral stenosis, the pressure gradient across the aortic valve can be determined noninvasively using continuous wave Doppler echocardiography:

$$\Delta P = 4V^2$$

where ΔP is the peak pressure gradient (mm Hg) and V is peak blood flow velocity (m/s) distal to the obstruction. Peak velocities greater than 4.5 m/sec are usually indicative of severe stenosis. Moreover, if the area proximal to the stenosis (LVOT) can be measured, the continuity equation can then be applied to estimate valve area. Either TVIs or maximum velocities can be used:

$$A_2 = \frac{A_1 V_1}{V_2}$$

where A_2 is valve area, A_1 is the cross-sectional area of the LVOT, V_1 is maximum blood flow velocity in LVOT, and V_2 is maximum flow velocity through the aortic valve.

Treatment

Once symptoms develop, most patients, without surgical treatment, will die within 2–5 years. Percutaneous balloon valvuloplasty is generally used in younger patients with congenital aortic stenosis; it can also be used in elderly patients with calcific aortic stenosis who are poor candidates for aortic valve replacement. Its efficacy for the latter group is short-lived, however, and restenosis usually occurs within 6–12 months. Catheter-delivered aortic valves are increasingly being perfected and deployed in the treatment of aortic valve disease. Surgical replacement of the stenotic aortic valve remains the mainstay of therapy.

Anesthetic Management

A. Objectives

10 Maintenance of normal sinus rhythm, heart rate, vascular resistance, and intravascular volume is critical in patients with aortic stenosis. Loss of a normally timed atrial systole often leads to rapid deterioration, particularly when associated with tachycardia. The combination of the two (AF with rapid ventricular response) seriously impairs ventricular filling and necessitates immediate cardioversion. The reduced ventricular compliance also makes the patient very sensitive to abrupt changes in intravascular volume. Many patients behave as though they have a fixed SV in spite of adequate hydration; under these conditions, cardiac output becomes very rate dependent. Extreme bradycardia (<50 beats/min) is therefore poorly tolerated. Heart rates between 60 and 90 beats/min are optimal in most patients.

B. Monitoring

Close monitoring of the ECG and blood pressure is crucial. Monitoring for ischemia is complicated by baseline ST-segment and T-wave abnormalities. Intraarterial pressure monitoring is desirable in patients with severe aortic stenosis, as

many of these patients do not tolerate even brief episodes of hypotension. Pulmonary artery catheterization data should be interpreted carefully; a higher than normal pulmonary capillary wedge pressure is often required to maintain adequate left ventricular end-diastolic volume and cardiac output. Prominent *a* waves are often visible on the pulmonary artery wedge pressure waveform. Vasodilators should generally be used cautiously because patients are often very sensitive to these agents. TEE can be useful in these patients for monitoring ischemia, ventricular preload, contractility, valvular function, and the effects of therapeutic interventions.

C. Choice of Agents

Patients with mild to moderate aortic stenosis (generally asymptomatic) may tolerate spinal or epidural anesthesia. These techniques should be employed very cautiously, however, because hypotension readily occurs as a result of reductions in preload, afterload, or both. Epidural anesthesia may be preferable to single-shot spinal anesthesia in many situations because of its slower onset of hypotension, which allows more timely correction. Continuous spinal catheters can similarly be used to gradually increase the level of regional anesthesia and limit the possibility of blood pressure collapse. Spinal and epidural anesthesia are relatively contraindicated in patients with severe aortic stenosis.

In the patient with severe aortic stenosis the choice of general anesthetic agents is less important than managing their hemodynamic effects. Most general anesthetics can produce both vasodilation and hypotension, which require treatment post induction. If a volatile agent is used, the concentration should be controlled to avoid excessive vasodilatation, myocardial depression, or loss of normal atrial systole. Significant tachycardia and severe hypertension, which can precipitate ischemia, should be treated immediately by increasing anesthetic depth or administration of a β-adrenergic blocking agent. Most patients with aortic stenosis tolerate moderate hypertension and are sensitive to vasodilators. Moreover, because of an already precarious myocardial oxygen demand–supply balance, they tolerate even mild degrees of hypotension poorly. Hypotension should generally be promptly treated with escalating doses (25–100 mcg) of phenylephrine. Intraoperative supraventricular tachycardias with hemodynamic compromise should be treated with immediate synchronized cardioversion. Frequent ventricular ectopy (which often reflects ischemia) is usually poorly tolerated hemodynamically and should be treated. Amiodarone is generally effective for both supraventricular and ventricular arrhythmias.

AORTIC REGURGITATION

Preoperative Considerations

Aortic regurgitation usually develops slowly and is progressive (chronic), but it can also develop quickly (acute). Chronic aortic regurgitation may be caused by abnormalities of the aortic valve, the aortic root, or both. Abnormalities in the valve are usually congenital (bicuspid valve) or due to rheumatic fever. Diseases affecting the ascending aorta cause regurgitation by dilating the aortic annulus; they include syphilis, annuloaortic ectasia, cystic medial necrosis (with or without Marfan syndrome), ankylosing spondylitis, rheumatoid and psoriatic arthritis, and a variety of other connective tissue disorders. Acute aortic insufficiency most commonly follows infective endocarditis, trauma, or aortic dissection.

Pathophysiology

Regardless of the cause, aortic regurgitation produces volume overload of the left ventricle. The effective forward SV is reduced because of backward (regurgitant) flow of blood into the left ventricle during diastole. Systemic arterial diastolic pressure and SVR are typically low. The decrease in cardiac afterload helps facilitate ventricular ejection. Total SV is the sum of the effective stroke volume and the regurgitant volume. The regurgitant volume depends on the heart rate (diastolic time) and the diastolic pressure gradient across the aortic valve (diastolic aortic pressure minus left ventricular end-diastolic pressure). Slow heart rates increase regurgitation because of the associated disproportionate increase in diastolic time, whereas increases in diastolic arterial pressure favor regurgitant volume by increasing the pressure gradient for backward flow.

With chronic aortic regurgitation, the left ventricle progressively dilates and undergoes eccentric hypertrophy. Patients with severe aortic regurgitation have the largest end-diastolic volumes of any heart disease. The resulting increase in end-diastolic volume maintains an effective SV. Any increase in the regurgitant volume is compensated by an increase in end-diastolic volume. Left ventricular end-diastolic pressure is usually normal or only slightly elevated, because ventricular compliance initially increases. Eventually, as ventricular function deteriorates, the ejection fraction declines, and impaired ventricular emptying is manifested as gradual increases in left ventricular end-diastolic pressure and end-systolic volume.

Sudden incompetence of the aortic valve does not allow compensatory dilatation or hypertrophy of the left ventricle. Effective SV rapidly declines because the normal-sized ventricle is unable to accommodate a sudden large regurgitant volume. The sudden rise in left ventricular end-diastolic pressure is transmitted back to the pulmonary circulation and causes acute pulmonary venous congestion.

Acute aortic regurgitation typically presents as the sudden onset of pulmonary edema and hypotension, whereas chronic regurgitation usually presents insidiously as congestive heart failure. Symptoms are generally minimal (in the chronic form) when the regurgitant volume remains under 40% of SV, but become severe when it exceeds 60%. Angina can occur even in the absence of coronary disease. The myocardial oxygen demand is increased from muscle hypertrophy and dilatation, whereas the myocardial blood supply is reduced by low diastolic pressures in the aorta as a result of the regurgitation.

Calculating Regurgitant Fraction & Other Measurements of Severity

As with mitral regurgitation, RSV and RF for aortic regurgitation can be estimated by pulsed Doppler echocardiography. Stroke volume is measured at the left ventricular outflow tract (LVOT) and at the mitral valve (MV). The stroke volume ejected at the LVOT includes both the stroke volume that entered the left ventricle through the mitral valve and the volume of blood that entered the left ventricle through the leaky aortic valve.

Thus,

$$\mathrm{RSV}_{\text{aortic regurgitation}} = (A_{\mathrm{LOVT}} \times \mathrm{TVI}_{\mathrm{LVOT}}) - (A_{\mathrm{MV}} \times \mathrm{TVI}_{\mathrm{MV}})$$

and

$$\mathrm{RF} = \mathrm{RSV}/\mathrm{SV}$$

Pressure half-time ($T_{1/2}$, see the section on mitral stenosis above) of the regurgitant jet is another useful echocardiographic parameter for clinically assessing the severity of aortic regurgitation. The shorter the half-time, the more severe the regurgitation; severe regurgitation rapidly raises left ventricular diastolic pressure and results in more rapid pressure equilibration. Unfortunately, $T_{1/2}$ is affected not only by the regurgitant orifice area, but also by aortic and ventricular pressure. An aortic regurgitation jet with a $T_{1/2}$ less than 240 msec is associated with severe regurgitation.

Treatment

Most patients with chronic aortic regurgitation remain asymptomatic for 10–20 years. Once significant symptoms develop, the expected survival time is about 5 years without valve replacement. Diuretics and afterload reduction, particularly with ACE inhibitors, generally benefit patients with advanced chronic aortic regurgitation. The decrease in arterial blood pressure reduces the diastolic gradient for regurgitation. Patients with chronic aortic regurgitation should receive valve replacement before irreversible ventricular dysfunction occurs.

Patients with acute aortic regurgitation typically require intravenous inotropic and vasodilator therapy. Early intervention is indicated in patients with acute aortic regurgitation: medical management alone is associated with a high mortality rate.

Anesthetic Management

A. Objectives

The heart rate should be maintained toward the upper limits of normal (80–100 beats/min). 11 Bradycardia and increases in SVR increase the regurgitant volume in patients with aortic regurgitation, whereas tachycardia can contribute to myocardial ischemia. Excessive myocardial depression should also be avoided. The compensatory

increase in cardiac preload should be maintained, but overzealous fluid replacement can readily result in pulmonary edema.

B. Monitoring

Invasive hemodynamic monitoring should be employed in patients with acute aortic regurgitation and in those with severe chronic regurgitation. Premature closure of the mitral valve often occurs during acute aortic regurgitation and may cause pulmonary capillary wedge pressure to give a falsely high estimate of left ventricular end-diastolic pressure. The appearance of a large *v* wave suggests mitral regurgitation secondary to dilatation of the left ventricle. The arterial pressure wave in patients with aortic regurgitation characteristically has a very wide pulse pressure. *Pulsus bisferiens* may also be present in patients with moderate to severe aortic insufficiency and is thought to result from the rapid ejection of a large SV. Color-flow Doppler TEE can be invaluable in quantitating the severity of the regurgitation and guiding therapeutic interventions. By definition, some reversal of blood flow is present in the aorta during all of diastole (holodiastolic) with severe aortic regurgitation; moreover, the more distal the detection of holodiastolic flow reversal is in the aorta, the more severe the regurgitation.

C. Choice of Agents

Most aortic insufficiency patients tolerate spinal and epidural anesthesia well, provided intravascular volume is maintained. When general anesthesia is required, inhalational agents may be ideal because of the associated vasodilatation. Phenylephrine (25–50 mcg) can be used to treat hypotension secondary to anesthetic-induced vasodilatation. Large doses of phenylephrine increase SVR (and arterial diastolic pressure) and may exacerbate the regurgitation.

TRICUSPID REGURGITATION

Preoperative Considerations

Up to 70% to 90% of patients have trace to mild tricuspid regurgitation on echocardiography; the regurgitant volume in these cases is almost always insignificant. Clinically significant tricuspid regurgitation, however, is most commonly due to dilatation of the right ventricle from pulmonary hypertension that is associated with chronic left ventricular failure. Tricuspid regurgitation can also follow infective endocarditis (usually in injecting drug abusers), rheumatic fever, carcinoid syndrome, or chest trauma or may be due to Ebstein's anomaly (downward displacement of the valve because of abnormal attachment of the valve leaflets).

Pathophysiology

Chronic left ventricular failure often leads to sustained increases in pulmonary vascular pressures. The chronic increase in afterload causes progressive dilatation of the thin-walled right ventricle, and excessive dilatation of the tricuspid annulus eventually results in regurgitation. An increase in end-diastolic volume allows the right ventricle to compensate for the regurgitant volume and maintain an effective forward flow. Because the right atrium and the vena cava are compliant and can usually accommodate the volume overload, mean right atrial and central venous pressures are generally only slightly elevated. Acute or marked elevations in pulmonary artery pressures increase the regurgitant volume and are reflected by an increase in central venous pressure. Moreover, sudden marked increases in right ventricular afterload sharply reduce the effective right ventricular output, reduce left ventricular preload, and can precipitate systemic hypotension.

Chronic venous hypertension leads to passive congestion of the liver and progressive hepatic dysfunction. Severe right ventricular failure with underloading of the left heart may also produce right-to-left shunting through a patent foramen ovale, which can result in marked hypoxemia.

The normal right ventricle does not extend to the apex of the heart when visualized using echocardiography. As the right heart dilates, it acquires a more spherical shape, the right ventricle extends to the apex of the heart, and the interventricular septum is flattened. These changes can impair left heart function.

Calculating Pulmonary Artery Pressure

With severe tricuspid regurgitation, the normal systolic inflow into the right atrium is reversed, and the reversal of flow is also observed in the hepatic veins.

Systolic pulmonary artery pressure (PAS) can be estimated from the peak velocity of the regurgitant jet:

$$\Delta P = 4 \times V^2$$

where ΔP is the systolic pressure gradient (mm Hg) between the right ventricle and right atrium, and V is peak blood flow velocity (m/s) of the regurgitant jet. If the central venous pressure (CVP) is known or assumed, then

$$PAS = CVP + \Delta P$$

Treatment

Tricuspid regurgitation is generally well tolerated by most patients. Because the underlying disorder is generally more important than the tricuspid regurgitation itself, treatment is aimed at the underlying disease process. With moderate to severe regurgitation, tricuspid annuloplasty may be performed in conjunction with replacement of another valve. Recent studies suggest that correction of significant tricuspid regurgitation is beneficial when patients are brought to surgery for replacement of another valve.

Anesthetic Management

A. Objectives

Hemodynamic goals should be directed primarily toward the underlying disorder. Hypovolemia and factors that increase right ventricular afterload, such as hypoxia and acidosis, should be avoided to maintain effective right ventricular SV and left ventricular preload. Positive end-expiratory pressure and high mean airway pressures may also be undesirable during mechanical ventilation because they reduce venous return and increase right ventricular afterload.

B. Monitoring

In these patients, invasive monitoring may be useful. Pulmonary artery catheterization is not always possible; rarely a large regurgitant flow may make passage of a pulmonary artery catheter across the tricuspid valve difficult. Increasing CVP implies worsening right ventricular dysfunction. The *x* descent is absent, and a prominent *cv* wave is usually present on the CVP waveform. Thermodilution cardiac output measurements are falsely elevated because of the tricuspid regurgitation. Color-flow Doppler TEE is useful in evaluating the severity of the regurgitation and other associated abnormalities.

C. Choice of Agents

The selection of anesthetic agents should be based on the underlying disorder. Most patients tolerate spinal and epidural anesthesia well. Coagulopathy secondary to hepatic dysfunction should be excluded prior to any regional technique. During general anesthesia, nitrous oxide may exacerbate pulmonary hypertension and should be administered cautiously, if at all.

ENDOCARDITIS PROPHYLAXIS

The ACC/AHA guidelines regarding prophylactic antibiotic regimens in patients with prosthetic heart valves and other structural heart abnormalities have dramatically changed in recent years, decreasing the number of indications for antibiotic administration. The risk of antibiotic administration is often considered greater than the potential for developing perioperative endocarditis. At present, the ACC/AHA guidelines suggest the use of endocarditis prophylaxis in the highest risk patients undergoing dental procedures involving gingival manipulation or perforation of the oral mucosa (class IIa); see Tables 21–14 and 21–15. Such conditions include:

- Patients with prosthetic cardiac valves or prosthetic heart materials
- Patients with a past history of endocarditis

TABLE 21–14 Endocarditis prophylaxis for dental procedures (UPDATED)[1].

Reasonable	Not Recommended
Endocarditis prophylaxis is reasonable for patients with the highest risk of adverse outcomes who undergo dental procedures that involve manipulation of either gingival tissue or the periapical region of teeth or perforation of the oral mucosa.	Endocarditis prophylaxis is not recommended for: • Routine anesthetic injections through noninfected tissue • Dental radiographs • Placement or removal of prosthodontic or orthodontic appliances • Adjustment of orthodontic appliances • Placement of orthodontic brackets • Shedding of deciduous teeth • Bleeding from trauma to the lips or oral mucosa

[1]Corresponds to the 2008 focused update incorporated into the ACC/AHA 2006 guidelines for the management of patients with valvular heart disease.

Reproduced, with permission, from Dajani AS, Taubert KA, Wilson W, et al: Prevention of bacterial endocarditis: recommendations by the American Heart Association. Circulation. 1997;96:358.

TABLE 21–15 Regimens for a dental procedure (UPDATED).

Situation	Agent	Regimen: Single Dose 30 to 60 min Before Procedure	
		Adults	Children
Oral	Amoxicillin	2 g	50 mg/kg
Unable to take oral medication	Ampicillin	2 g IM or IV	50 mg/kg IM or IV
	OR		
	Cefazolin or ceftriaxone	1 g IM or IV	50 mg/kg IM or IV
Allergic to penicillins or ampicillin—oral	Cephalexin[1,2]	2 g	50 mg/kg
	OR		
	Clindamycin	600 mg	20 mg/kg
	OR		
	Azithromycin or clarithromycin	500 mg	15 mg/kg
Allergic to penicillins or ampicillin and unable to take oral medication	Cefazolin or ceftriaxone‡	1 g IM or IV	50 mg/kg IM or IV
	OR		
	Clindamycin	600 mg IM or IV	20 mg/kg IM or IV

[1]Or use other first- or second-generation oral cephalosporin in equivalent adult or pediatric dosage.

[2]Cephalosporins should not be used in an individual with a history of anaphylaxis, angioedema, or urticaria with penicillins or ampicillin.

IM indicates intramuscular; and IV, intravenous.

Reproduced, with permission, from Nishimura RA, Carabello BA, Faxon DP, et al: ACC/AHA 2008 guideline update on valvular heart disease: focused update on infective endocarditis prophylaxis. J Am Coll Cardiol 2008;52:676.

- Patients with congenital heart disease that is either partially repaired or unrepaired
- Patients with congenital heart disease with residual defects following repair
- Patients with congenital heart disease within 6 months of a complete repair, whether catheter-based or surgical
- Cardiac transplant patients with structurally abnormal valves

Class III recommendations indicate that prophylaxis is not necessary for nondental procedures, including TEE and esophagogastroduodenoscopy, except in the presence of an active infection.

Endocarditis is believed to occur in areas of cardiac endothelial damage, where in cases of bacteremia, bacteria can be deposited and multiply. Areas of increased myocardial blood flow velocity lead to damaged endothelium, providing a template for bacterial adherence and growth. Indeed, the latest ACC/AHA guidelines do not suggest prophylaxis for genitourinary or gastrointestinal procedures; however, the AHA does note that it is reasonable to administer antibiotics to prevent wound infection. Moreover, they note that although prophylaxis is not suggested for respiratory tract procedures, it is a reasonable strategy in high-risk patients in whom an incision has been made in the respiratory tract (eg, in tonsillectomy).

In spite of these much reduced indications, the ACC/AHA notes that many patients and physicians expect the administration of endocarditis

prophylaxis in patients with valvular heart disease, aortic coarctation, and hypertrophic cardiomyopathy. As always, the risk of antibiotic administration must be considered in offering prophylaxis to patients outside of the ACC/AHA high-risk category. Guidelines are ever changing, and although not considered to be "standard of care," they are increasingly present in medical practice; furthermore, deviation from guidelines often requires explanation as being outside of "evidenced-based" practice. Review of ACC/AHA guidelines, which are now available online, are recommended when high-risk patients are encountered.

ANTICOAGULATION

Patients with mechanical prosthetic heart valves require anticoagulation, which is currently accomplished with warfarin. Aspirin is also indicated in this population, as well as in patients with bioprosthetic valves, to prevent thrombus formation. Warfarin is sometimes also used initially for mitral bioprosthetic valves (Table 21–16).

Patients with prosthetic valves often present for noncardiac surgery that will require temporary discontinuation of anticoagulation. The ACC/AHA guidelines indicate that patients at low risk of thrombosis, such as those with bileaflet mechanical valves

TABLE 21–16 Recommendations for antithrombotic therapy in patients with prosthetic heart valves.

	Aspirin (75–100 mg)	Warfarin (INR 2.0–3.0)	Warfarin (INR 2.5–3.5)	No Warfarin
Mechanical prosthetic valves				
AVR-low risk				
Less than 3 months	Class I	Class I	Class IIa	
Greater than 3 months	Class I	Class I		
AVR–high risk	Class I		Class I	
MVR	Class I		Class I	
Biological prosthetic valves				
AVR–low risk				
Less than 3 months	Class I	Class IIa		Class IIb
Greater than 3 months	Class I			Class IIa
AVR–high risk	Class I	Class I		
MVR–low risk				
Less than 3 months	Class I	Class IIa		
Greater than 3 months	Class I			Class IIa
MVR-high risk	Class I	Class I		

Depending on patients' clinical status, antithrombotic therapy must be individualized (see special situations in text). In patients receiving warfarin, aspirin is recommended in virtually all situations. Risk factors: atrial fibrillation, left ventricular dysfunction, previous thromboembolism, and hypercoagulable condition. International normalized ratio (INR) should be maintained between 2.5 and 3.5 for aortic disc valves and Starr-Edwards valves.

AVR indicates aortic valve replacement; and MVR, mitral valve replacement.

Data from McAnuty JH, Rahimtoola SH. Antithrombotic therapy in valvular heart disease. In: Schlant R, Alexarder RW. editors. *Hurst's The Heart.* New York, McGraw-Hill, 1998:1867-1874.

in the aortic position with no additional problems (eg, no AF or no hypercoaguable state) can discontinue warfarin 48–72 hours preoperatively so that the INR falls below 1.5. In patients at greater risk of thrombosis, warfarin should be discontinued and heparin, either unfractionated or low molecular weight, started when the INR falls below 2.0. Heparin can be discontinued 4–6 hours prior to surgery and then restarted as soon as surgical bleeding permits, until the patient can be restarted on warfarin therapy. Fresh frozen plasma may be given, if needed, in an emergency situation to interrupt warfarin therapy. Vitamin K should not be administered, as it could potentially lead to a hypercoaguable state. Anesthesia staff should always consult with the patient's surgeon and the physician responsible for prescribing the anticoagulation before adjusting anticoagulation or antiplatelet regimens perioperatively.

Congenital Heart Disease

Preoperative Considerations

Congenital heart disease encompasses a seemingly endless list of abnormalities that may be detected in infancy, early childhood, or, less commonly, adulthood. The incidence of congenital heart disease in all live births approaches 1%. The natural history of some defects is such that patients often survive to adulthood (Table 21–17). Moreover, the number of surviving adults with congenital heart disease is steadily increasing, possibly as a result of advances in surgical and medical treatment. An increasing number of patients with congenital heart disease may therefore be encountered during noncardiac surgery and obstetric deliveries. Knowledge of the anatomy of the original heart structure defect and of any corrective repairs is essential prior to anesthetizing the patient with congental heart disease (CHD).

TABLE 21–17 Common congenital heart defects in which patients typically survive to adulthood without treatment.

Bicuspid aortic valve
Coarctation of the aorta
Pulmonic valve stenosis
Ostium secundum atrial septal defect
Ventricular septal defect
Patent ductus arteriosus

TABLE 21–18 Classification of congenital heart disease.

Lesions causing outflow obstruction
Left ventricle
Coarctation of the aorta
Aortic stenosis
Right ventricle
Pulmonic valve stenosis
Lesions causing left-to-right shunting
Ventricular septal defect
Patent ductus arteriosus
Atrial septal defect
Endocardial cushion defect
Partial anomalous pulmonary venous return
Lesions causing right-to-left shunting
With decreased pulmonary blood flow
Tetralogy of Fallot
Pulmonary atresia
Tricuspid atresia
With increased pulmonary blood flow
Transposition of the great vessels
Truncus arteriosus
Single ventricle
Double-outlet right ventricle
Total anomalous pulmonary venous return
Hypoplastic left heart

The complex nature and varying pathophysiology of congenital heart defects make classification difficult. A commonly used scheme is presented in Table 21–18. Most patients present with cyanosis, congestive heart failure, or an asymptomatic abnormality. Cyanosis is typically the result of an abnormal intracardiac communication that allows unoxygenated blood to reach the systemic arterial circulation (right-to-left shunting). Congestive heart failure is most prominent with defects that either obstruct left ventricular outflow or markedly increase pulmonary blood flow. The latter is usually due to an abnormal intracardiac communication that returns oxygenated blood to the right heart (left-to-right shunting). Whereas right-to-left shunts generally decrease pulmonary blood flow, some complex lesions increase pulmonary blood flow—even in the presence of right-to-left shunting. In many cases, more than one lesion is present.

In fact, survival (prior to surgical correction) with some anomalies (eg, transposition, total anomalous venous return, pulmonary atresia) depends on the simultaneous presence of another shunting lesion (eg, patent ductus arteriosus, patent foramen ovale, ventricular septal defect). Chronic hypoxemia in patients with cyanotic heart disease typically results in erythrocytosis. This increase in red cell mass, which is due to enhanced erythropoietin secretion from the kidneys, serves to restore tissue oxygen concentration to normal. Unfortunately, blood viscosity can also rise to the point at which it may interfere with oxygen delivery. When tissue oxygenation is restored to normal, the hematocrit is stable (usually <65%), and symptoms of the hyperviscosity syndrome are absent, the patient is said to have compensated erythrocytosis. Patients with uncompensated erythrocytosis do not establish this equilibrium; they have symptoms of hyperviscosity and may be at risk of thrombotic complications, particularly stroke. The last is aggravated by dehydration. Children younger than age 4 years seem to be at greatest risk of stroke. Phlebotomy is generally not recommended if symptoms of hyperviscosity are absent and the hematocrit is <65%.

Coagulation abnormalities are common in patients with cyanotic heart disease. Platelet counts tend to be low-normal, and many patients have subtle or overt defects in the coagulation cascade. Phlebotomy may improve hemostasis in some patients. Hyperuricemia often occurs because of increased urate reabsorption secondary to renal hypoperfusion. Gouty arthritis is uncommon, but the hyperuricemia can result in progressive renal impairment.

Preoperative Doppler echocardiography is invaluable in helping to define the anatomy of the defect(s) and to confirm or exclude the existence of other lesions or complications, their physiological significance, and the effects of any therapeutic interventions.

Anesthetic Management

This population of patients includes four groups: those who have undergone corrective cardiac surgery and require no further operations, those who have had only palliative surgery, those who have not yet undergone any cardiac surgery, and those whose conditions are inoperable and may be awaiting cardiac transplantation. Although the management of the first group of patients may be the same as that of normal patients (except for consideration of prophylactic antibiotic therapy), the care of others requires familiarity with the complex pathophysiology of these defects. Even patients who have had corrective surgery may be prone to the development of perioperative problems (Tables 21–19 and 21–20). Some surgical procedures eliminate the risk of endocarditis, whereas others increase the risk through the use of prosthetic valves or conduits or the creation of new shunts.

For the purpose of anesthetic management, congenital heart defects may be divided into obstructive lesions, predominantly left-to-right shunts, or predominantly right-to-left shunts. In reality, shunts can also be bidirectional and may reverse under certain conditions.

TABLE 21–19 Common problems in survivors of surgery for congenital heart defects.

Arrhythmias
Hypoxemia
Pulmonary hypertension
Existing shunts
Paradoxical embolism
Bacterial endocarditis

TABLE 21–20 Congenital cardiac lesions and perioperative risk for noncardiac surgery.

High risk
Pulmonary hypertension, primary or secondary
Cyanotic congenital heart disease
New York Heart Association class III or IV
Severe systemic ventricular dysfunction (ejection fraction less than 35%)
Severe left-sided heart obstructive lesions
Moderate risk
Prosthetic valve or conduit
Intracardiac shunt
Moderate left-sided heart obstruction
Moderate systemic ventricular dysfunction

Warnes C, Williams R, Bashore T, et al: ACC/AHA 2008 guidelines for the management of adults with congenital heart disease. Circulation 2008;118:2395.

1. Obstructive Lesions

Pulmonic Stenosis

Pulmonary valve stenosis obstructs right ventricular outflow and causes concentric right ventricular hypertrophy. Severe obstruction presents in the neonatal period, whereas lesser degrees of obstruction may go undetected until adulthood. The valve is usually deformed and is either bicuspid or tricuspid. Valve leaflets are often partially fused and display systolic doming on echocardiography. The right ventricle undergoes hypertrophy, and poststenotic dilatation of the pulmonary artery is often present. Symptoms are those of right ventricular heart failure. Symptomatic patients readily develop fatigue, dyspnea, and peripheral cyanosis with exertion as a result of the limited pulmonary blood flow and increased oxygen extraction by tissues. With severe stenosis, the pulmonic valve gradient exceeds 60–80 mm Hg, depending on the age of the patient. Right-to-left shunting may also occur in the presence of a patent foramen ovale or atrial septal defect. Cardiac output is very dependent on an elevated heart rate, but excessive increases in the latter can compromise ventricular filling. Percutaneous balloon valvuloplasty is generally considered the initial treatment of choice in most patients with symptomatic pulmonic stenosis. Anesthetic management for patients undergoing surgery should maintain a normal or slightly high heart rate, augment preload, and avoid factors that increase PVR.

2. Predominantly Left-to-Right (Simple) Shunts

Simple shunts are isolated abnormal communications between the right and left sides of the heart. Because pressures are normally higher on the left side of the heart, blood usually flows across from left to right, and blood flow through the right heart and the lungs increases. Depending on the size and location of the communication, the right ventricle may also be subjected to the higher left-sided pressures, resulting in both pressure and volume overload. Right ventricular afterload is normally 5% that of the left ventricle, so even small left-to-right pressure gradients can produce large increases in pulmonary blood flow. The ratio of pulmonary (Qp) to systemic (Qs) blood flow is useful to determine the directionality of the shunt.

A ratio greater than 1 usually indicates a left-to-right shunt, whereas a ratio less than 1 indicates a right-to-left shunt. A ratio of 1 indicates either no shunting or a bidirectional shunt of opposing magnitudes.

Large increases in pulmonary blood flow produce pulmonary vascular congestion and increase extravascular lung water. The latter interferes with gas exchange, decreases lung compliance, and increases the work of breathing. Left atrial distention also compresses the left bronchus, whereas distention of pulmonary vessels compresses smaller bronchi.

Over the course of several years, chronic increases in pulmonary blood flow produce vascular changes that irreversibly increase PVR. Elevation of right ventricular afterload produces hypertrophy and progressively raises right-sided cardiac pressures. With advanced disease, the pressures within the right heart can exceed those within the left heart. Under these conditions, the intracardiac shunt reverses and becomes a right-to-left shunt (Eisenmenger syndrome).

When a communication is small, shunt flow depends primarily on the size of the communication (restrictive shunt). When the communication is large (nonrestrictive shunt), shunt flow depends on the relative balance between PVR and SVR. **12** An increase in SVR relative to PVR favors left-to-right shunting, whereas an increase in PVR relative to SVR favors right-to-left shunting. Common chamber lesions (eg, single atrium, single ventricle, truncus arteriosus) represent the extreme form of nonrestrictive shunts; shunt flow with these lesions is bidirectional and totally dependent on relative changes in the ventricular afterload.

13 The presence of shunt flow between the right and left hearts, regardless of the direction of blood flow, mandates the meticulous exclusion of air bubbles and particulate material from intravenous fluids to prevent paradoxical embolism into the cerebral or coronary circulations.

Atrial Septal Defects

Ostium secundum atrial septal defects (ASDs) are the most common type and usually occur as isolated

lesions in the area of the fossa ovalis. The defect is sometimes associated with partial anomalous pulmonary venous return, most commonly of the right upper pulmonary vein. A secundum ASD may result in single or multiple (fenestrated) openings between the atria. The less common sinus venosus and ostium primum ASDs are typically associated with other cardiac abnormalities. Sinus venosus defects are located in the upper interatrial septum close to the superior vena cava; one or more of the right pulmonary veins often abnormally drains into the superior vena cava. In contrast, ostium primum ASDs are located in the lower interatrial septum and overlie the mitral and tricuspid valves; most patients also have a cleft in the anterior leaflet of the mitral, valve and some have an abnormal septal leaflet in the tricuspid valve.

Most children with ASDs are minimally symptomatic; some have recurrent pulmonary infections. Congestive heart failure and pulmonary hypertension are more commonly encountered in adults with ASDs. Patients with ostium primum defects often have large shunts and may also develop significant mitral regurgitation. In the absence of heart failure, anesthetic responses to inhalation and intravenous agents are generally not significantly altered in patients with ASDs. **Large increases in SVR should be avoided because they may worsen the left-to-right shunting**.

Ventricular Septal Defects

Ventricular septal defect (VSD) is a common congenital heart defect, accounting for up to 25% to 35% of congenital heart disease. The defect is most frequently found in the membranous part of the interventricular septum (membranous or infracristal VSD) in a posterior position and anterior to the septal leaflet of the tricuspid valve. Muscular VSDs are the next most frequent type and are located in the mid or apical portion of the interventricular septum, where there may be a single defect or multiple openings (resembling Swiss cheese). Defects in the subpulmonary (supracristal) septum are often associated with aortic regurgitation because the right coronary cusp can prolapse into the VSD. Septal defects at the ventricular inlet are usually similar in development and location to AV septal defects (see the following section).

The resulting functional abnormality of a VSD is dependent on the size of the defect, PVR, and the presence or absence of other abnormalities. Small VSDs, particularly of the muscular type, often close during childhood. Restrictive defects are associated with only small left-to-right shunts (pulmonary–systemic blood flow ratios less than 1.75:1). Large defects produce large left-to-right shunts (shunts larger than 2:1) that vary directly with SVR and indirectly with PVR. Recurrent pulmonary infections and congestive heart failure are common with pulmonary–systemic flow ratios of 3–5:1. Patients with small VSDs are treated medically and followed by electrocardiography (for signs of right ventricular hypertrophy) and echocardiography. Surgical closure is usually undertaken in patients with large VSDs before pulmonary vascular disease and Eisenmenger physiology develop. As with atrial defects, in the absence of heart failure, anesthetic responses to inhalation and intravenous agents are generally not significantly altered. Similarly, increases in SVR worsen the left-to-right shunting. **When right-to-left shunting is present, abrupt increases in PVR or decreases in SVR are poorly tolerated.**

Atrioventricular Septal Defects

Endocardial cushion (AV canal) defects produce contiguous atrial and ventricular septal defects, often with very abnormal AV valves. This is a common lesion in patients with Down syndrome. The defect can produce large shunts both at the atrial and ventricular levels. Mitral and tricuspid regurgitation exacerbate the volume overload on the ventricles. Initially, shunting is predominately left to right; however, with increasing pulmonary hypertension, Eisenmenger syndrome with obvious cyanosis develops.

Patent Ductus Arteriosus

Persistence of the communication between the main pulmonary artery and the aorta can produce restrictive or nonrestrictive left-to-right shunts. This abnormality is commonly responsible for the cardiopulmonary deterioration of premature infants and occasionally presents later in life when it can be

corrected thoracoscopically. Anesthetic goals should be similar to atrial and ventricular septal defects.

Partial Anomalous Venous Return

This defect is present when one or more pulmonary veins drains into the right side of the heart; the anomalous veins are usually from the right lung. Possible anomalous entry sites include the right atrium, the superior or inferior vena cava, and the coronary sinus. The resulting abnormality produces a variable amount of left-to-right shunting. The clinical course and prognosis are usually excellent and similar to that of a secundum ASD. A very large coronary sinus on TEE suggests anomalous drainage into the coronary sinus, which may complicate the management of cardioplegia during cardiac surgery. Total anomalous venous return is corrected immediately after birth.

3. Predominantly Right-to-Left (Complex) Shunts

Lesions within this group (some also called **mixing lesions**) often produce both ventricular outflow obstruction and shunting. The obstruction favors shunt flow toward the unobstructed side. When the obstruction is relatively mild, the amount of shunting is affected by the ratio of SVR to PVR, but increasing degrees of obstruction fix the direction and magnitude of the shunt. Atresia of any one of the cardiac valves represents the extreme form of obstruction. Shunting occurs proximal to the atretic valve and is completely fixed; survival depends on another distal shunt (usually a patent ductus arteriosus [PDA], patent foramen ovale, ASD, or VSD), where blood flows in the opposite direction. This group of defects may also be divided according to whether they increase or decrease pulmonary blood flow.

Tetralogy of Fallot

This anomaly classically includes right ventricular outflow obstruction, right ventricular hypertrophy, and a VSD with an overriding aorta. Right ventricular obstruction in most patients is due to infundibular stenosis, which is due to hypertrophy of the subpulmonic muscle (crista ventricularis). At least 20% to 25% of patients also have pulmonic stenosis, and a small percentage of patients has some element of supravalvular obstruction. The pulmonic valve is often bicuspid, or, less commonly, atretic. Infundibular obstruction may be increased by sympathetic tone and is therefore dynamic; this obstruction is likely responsible for the hypercyanotic spells observed in very young patients. **The combination of right ventricular outflow obstruction and a VSD results in ejection of unoxygenated right ventricular blood, as well as oxygenated left ventricular blood into the aorta**. The right-to-left shunting across the VSD has both fixed and variable components. The fixed component is determined by the severity of the right ventricular obstruction, whereas the variable component depends on SVR and PVR.

Neonates with severe right ventricular obstruction may deteriorate quickly, as pulmonary blood flow decreases when a PDA starts to close. Intravenous prostaglandin E_1 (0.05–0.2 mcg/kg/min) is used to prevent ductal closure in such instances. Surgical palliation with a left-to-right systemic shunt or complete correction is then usually undertaken. For the former, a modified Blalock–Taussig (systemic–pulmonary artery) shunt is most often used to increase pulmonary blood flow. In this procedure, a synthetic graft is anastomosed between a subclavian artery and an ipsilateral pulmonary artery. Complete correction involves closure of the VSD, removal of obstructing infundibular muscle, and pulmonic valvulotomy or valvuloplasty, when necessary.

14 The goals of anesthetic management in patients with tetralogy of Fallot should be to maintain intravascular volume and SVR. Increases in PVR, such as might occur from acidosis or excessive airway pressures, should be avoided. **Ketamine (intramuscular or intravenous) is a commonly used induction agent because it maintains or increases SVR and therefore does not aggravate the right-to-left shunting**. Patients with milder degrees of shunting generally tolerate inhalation induction. The right-to-left shunting tends to slow the uptake of inhalation anesthetics; in contrast, it may accelerate the onset of intravenous agents. Oxygenation often improves following induction of anesthesia. Muscle relaxants that release histamine should be

avoided. Hypercyanotic spells may be treated with intravenous fluid and phenylephrine (5 mcg/kg). Beta blockers (eg, propranolol) may also be effective in relieving infundibular spasm. Sodium bicarbonate to correct the resulting metabolic acidosis, may also be helpful when the hypoxemia is severe and prolonged.

Tricuspid Atresia

With tricuspid atresia, blood can flow out of the right atrium only via a patent foramen ovale (or an ASD). Moreover, a PDA (or VSD) is necessary for blood to flow from the left ventricle into the pulmonary circulation. Cyanosis is usually evident at birth, and its severity depends on the amount of pulmonary blood flow that is achieved. Early survival is dependent on prostaglandin E_1 infusion, with or without a percutaneous Rashkind balloon atrial septostomy. Severe cyanosis requires a modified Blalock–Taussig shunt early in life. The preferred surgical management is a modified Fontan procedure, in which the venous drainage is directed to the pulmonary circulation. In some centers, a superior vena cava to the main pulmonary artery (bidirectional Glenn) shunt may be employed before or instead of a Fontan procedure. With both procedures, blood from the systemic veins flows to the left atrium without the assistance of the right ventricle. Success of the procedure depends on a high systemic venous pressure and maintaining both low PVR and a low left atrial pressure. Heart transplantation may be necessary for a failed Fontan procedure.

Transposition of the Great Arteries

In patients with transposition of the great arteries, pulmonary and systemic venous return flows normally back to the right and left atrium, respectively, but the aorta arises from the right ventricle, and the pulmonary artery arises from the left ventricle. Thus, deoxygenated blood returns back into the systemic circulation, and oxygenated blood returns back to the lungs. Survival is possible only through mixing of oxygenated and deoxygenated blood across the foramen ovale and a PDA. The presence of a VSD increases mixing and reduces the level of hypoxemia. Prostaglandin E_1 infusion is usually necessary. Rashkind septostomy may be necessary if surgical correction is delayed. Corrective surgical treatment involves an arterial switch procedure in which the aorta is divided and reanastomosed to the left ventricle, and the pulmonary artery is divided and reanastomosed to the right ventricle. The coronary arteries must also be reimplanted into the old pulmonary artery root. A VSD, if present, is closed. Less commonly, an atrial switch (Senning) procedure may be carried out if an arterial switch is not possible. In this latter procedure, an intraatrial baffle is created from the atrial wall, and blood from the pulmonary veins flows across an ASD to the right ventricle, from which it is ejected into the systemic circulation.

Transposition of the great vessels may occur with a VSD and pulmonic stenosis. This combination of defects mimics tetralogy of Fallot; however, the obstruction affects the left ventricle, not the right ventricle. Corrective surgery involves patch closure of the VSD, directing left ventricular outflow into the aorta, ligation of the proximal pulmonary artery, and connecting the right ventricular outflow to the pulmonary artery with a valved conduit (Rastelli procedure).

Truncus Arteriosus

With a truncus arteriosus defect, a single arterial trunk supplies the pulmonary and systemic circulation. The truncus always overrides a VSD, allowing both ventricles to eject into it. As PVR gradually decreases after birth, pulmonary blood flow increases greatly, resulting in heart failure. If left untreated, PVR increases, and cyanosis develops again, along with Eisenmenger physiology. Surgical correction closes the VSD, separates the pulmonary artery from the truncus, and connects the right ventricle to the pulmonary artery with a conduit (Rastelli repair).

Hypoplastic Left Heart Syndrome

This syndrome describes a group of defects characterized by aortic valve atresia and marked underdevelopment of the left ventricle. The right ventricle is the main pumping chamber for both systemic and pulmonary circulations. It ejects normally into the pulmonary artery, and all (or nearly all) blood flow entering the aorta is usually derived

from a PDA. Surgical treatment includes both the Norwood repair and a hybrid approach to palliation. In the Norwood repair, a new aorta is created from the hypoplastic aorta and the main pulmonary artery. Pulmonary blood flow is delivered via a Blalock–Taussig shunt. The right ventricle becomes the heart's systemic pumping ventricle. A hybrid approach has also been advocated for the treatment of hypoplastic left heart syndrome. In this approach, the pulmonary arteries are banded to reduce pulmonary blood flow, and the PDA is stented to provide for systemic blood flow.

The Patient with a Transplanted Heart

Preoperative Considerations

The number of patients with cardiac transplants is increasing because of both the increasing frequency of transplantation and improved post-transplant survival rates. These patients may present to the operating room early in the postoperative period for mediastinal exploration or retransplantation, or they may appear later for incision and drainage of infections, orthopedic surgery, or unrelated procedures.

15 The transplanted heart is totally denervated, so direct autonomic influences are absent. Cardiac impulse formation and conduction are normal, but the absence of vagal influences causes a relatively high resting heart rate (100–120 beats/min). Although sympathetic fibers are similarly interrupted, the response to circulating catecholamines is normal or even enhanced because of denervation sensitivity (increased receptor density). Cardiac output tends to be low-normal and increases relatively slowly in response to exercise because the response is dependent on an increase in circulating catecholamines. Because the Starling relationship between end-diastolic volume and cardiac output is normal, the transplanted heart is also often said to be preload dependent. Coronary autoregulation is preserved.

Preoperative evaluation should focus on evaluating the functional status of the transplanted organ and detecting complications of immunosuppression. Rejection may be heralded by arrhythmias (in the first 6 months) or decreased exercise tolerance from a progressive deterioration of myocardial performance. Periodic echocardiographic evaluations are commonly used to monitor for rejection, but the most reliable technique is endomyocardial biopsy. Accelerated atherosclerosis in the graft is a very common and serious problem that limits the life of the transplant. Moreover, myocardial ischemia and infarction are almost always silent because of the denervation. Because of this, patients must undergo periodic evaluations, including angiography, for assessment of coronary atherosclerosis.

Immunosuppressive therapy usually includes cyclosporine, azathioprine, and prednisone. Important side effects include nephrotoxicity, bone marrow suppression, hepatotoxicity, opportunistic infections, and osteoporosis. Hypertension and fluid retention are common and typically require treatment with a diuretic and an ACE inhibitor. Stress doses of corticosteroids are needed when patients undergo major procedures.

Anesthetic Management

Almost all anesthetic techniques, including regional anesthesia, have been used successfully for transplanted patients. The preload-dependent function of the graft makes maintenance of a normal or high cardiac preload desirable. Moreover, the absence of reflex increases in heart rate can make patients particularly sensitive to rapid vasodilatation. Indirect vasopressors, such as ephedrine, are less effective than direct-acting agents because of the absence of catecholamine stores in myocardial neurons. Isoproterenol or epinephrine infusions should be readily available to increase the heart rate if necessary.

Careful electrocardiographic monitoring for ischemia is necessary. The ECG usually demonstrates two sets of P waves, one representing the recipient's own sinoatrial node (SA) (which is left intact), and the other representing the donor's SA node. The recipient's SA node may still be affected by autonomic influences, but it does not affect cardiac function. Direct arterial pressure monitoring should be used for major operations; strict asepsis should be observed during placement.

In a recently transplanted patient, the right ventricle of the transplanted heart may not be able to overcome the resistance of the pulmonary vasculature. Right ventricular failure can occur perioperatively, requiring the use of inhaled nitric oxide, inotropes, and, at times, right ventricular assist devices.

CASE DISCUSSION

Hip Fracture in an Elderly Woman who Fell

A 71-year-old woman presents for open reduction and internal fixation of a left hip fracture. She gives a history of two episodes of lightheadedness several days prior to her fall today. When questioned about her fall, she can only recall standing in her bathroom while brushing her teeth and then awakening on the floor with hip pain. The preoperative ECG shows a sinus rhythm with a P–R interval of 220 msec and a right bundle-branch block (RBBB) pattern.

Why should the anesthesiologist be concerned about a history of syncope?

A history of syncope in elderly patients should always raise the possibility of arrhythmias and underlying organic heart disease. Although arrhythmias can occur in the absence of organic heart disease, the two are commonly related. Cardiac syncope usually results from an abrupt arrhythmia that suddenly compromises cardiac output and impairs cerebral perfusion. Lightheadedness and presyncope, may reflect lesser degrees of cerebral impairment. Both bradyarrhythmias and tachyarrhythmias (see Chapter 20) can produce syncope. **Table 21–21** lists other cardiac and noncardiac causes of syncope.

How do bradyarrhythmias commonly arise?

Bradyarrhythmias may arise from either SA node dysfunction or abnormal AV conduction of the cardiac impulse. A delay or block of the impulse can occur anywhere between the SA node and the distal His-Purkinje system). Reversible abnormalities may be due to abnormal vagal tone, electrolyte abnormalities, drug toxicity, hypothermia, or myocardial ischemia. Irreversible abnormalities, which initially may be only intermittent before they become permanent, reflect either isolated conduction system abnormalities or underlying heart disease (most commonly hypertensive, coronary artery, or valvular heart disease).

TABLE 21–21 Causes of syncope.

Cardiac
Arrhythmias
Tachyarrhythmias (usually >180 beats/min)
Bradyarrhythmias (usually <40 beats/min)
Impairment of left ventricular ejection
Aortic stenosis
Hypertrophic cardiomyopathy
Massive myocardial infarction
Atrial myxoma
Impairment of right ventricular output
Tetralogy of Fallot
Primary pulmonary hypertension
Pulmonary embolism
Pulmonic valve stenosis
Biventricular impairment
Cardiac tamponade
Massive myocardial infarction
Noncardiac
Accentuated reflexes
Vasodepressor reflex (ie, vasovagal syncope)
Carotid sinus hypersensitivity
Neuralgias
Postural hypotension
Hypovolemia
Sympathectomy
Autonomic dysfunction
Sustained Valsalva maneuver
Cerebrovascular disease
Seizures
Metabolic
Hypoxia
Marked hypocapnia
Hypoglycemia

What is the pathophysiology of sinus node dysfunction?

Patients with sinus node dysfunction may have a normal baseline 12-lead ECG but abrupt pauses in SA node activity (sinus arrest) or intermittent block of conduction of the SA impulse to the surrounding tissue (exit block). Symptoms are usually present when pauses are prolonged

(>3 s) or the effective ventricular rate is less than 40 beats/min. Patients may experience intermittent dizziness, syncope, confusion, fatigue, or shortness of breath. Symptomatic SA node dysfunction, or sick sinus syndrome, is often unmasked by β-adrenergic blocking agents, calcium channel blockers, digoxin, or quinidine. The term tachycardia–bradycardia syndrome is often used when patients experience paroxysmal tachyarrhythmias (usually atrial flutter or fibrillation) followed by sinus pauses or bradycardia. The latter, bradycardia, probably represents failure of the SA node to recover normal automaticity following suppression by the tachyarrhythmia. The diagnosis must be based on electrocardiographic recordings made during symptoms (Holter monitoring) or after provocative tests (carotid baroreceptor stimulation or rapid atrial pacing).

How are AV conduction abnormalities manifested on the surface 12-lead ECG?

AV conduction abnormalities are usually manifested by abnormal ventricular depolarization (bundle-branch block), prolongation of the P–R interval (first-degree AV block), failure of some atrial impulses to depolarize the ventricles (second-degree AV block), or AV dissociation (third-degree AV block; also called complete heart block).

What determines the significance of these conduction abnormalities?

The significance of a conduction system abnormality depends on its location, its likelihood for progression to complete heart block, and the likelihood that a more distal pacemaker site will be able to maintain a stable and adequate escape rhythm (>40 beats/min). The His bundle is normally the lowest area in the conduction system that can maintain a stable rhythm (usually 40–60 beats/min). When conduction fails anywhere above it, a normal His bundle can take over the pacemaker function of the heart and maintain a normal QRS complex, unless a distal intraventricular conduction defect is present. When the escape rhythm arises farther down the His-Purkinje system, the rhythm is usually slower (<40 beats/min) and is often unstable; it results in a wide QRS complex.

What is the significance of isolated bundle-branch block with a normal P–R interval?

A conduction delay or block in the right bundle-branch results in a typical RBBB QRS pattern on the surface ECG (M-shape or rSR′ in V_1) and may represent a congenital abnormality or underlying organic heart disease. In contrast, a delay or block in the main left bundle-branch results in a left bundle-branch block (LBBB) QRS pattern (wide R with a delayed upstroke in V_5) and nearly always represents underlying heart disease. The term hemiblock is often used if only one of the two fascicles of the left bundle-branch is blocked (left anterior or left posterior hemiblock). When the P–R interval is normal—and in the absence of an acute MI—a conduction block in either the left or right bundle rarely leads to complete heart block.

Can the site of an AV block always be determined from a 12-lead ECG?

No. A first-degree AV block (P–R interval >200 ms) can reflect abnormal conduction anywhere between the atria and the distal His-Purkinje system. Mobitz type I second-degree AV block, which is characterized by progressive lengthening of the P–R interval before a P wave is not conducted (a QRS does not follow the P wave), is usually due to a block in the AV node itself, and can be caused by digitalis toxicity or myocardical ischemia; progression to a third-degree AV block is uncommon.

In patients with Mobitz type II second-degree AV block, atrial impulses are periodically not conducted into the ventricle without progressive prolongation of the P–R interval. The conduction block is nearly always in or below the His bundle and frequently progresses to complete (third-degree) AV block, particularly following an acute anteroseptal MI. The QRS is typically wide.

In patients with a third-degree AV block, the atrial rate and ventricular depolarization rates are independent (AV dissociation) because atrial impulses completely fail to reach the ventricles. If the site of the block is in the AV node, a stable His

bundle rhythm will result in a normal QRS complex, and the ventricular rate will often increase following administration of atropine. If the block involves the His bundle, the origin of the ventricular rhythm is more distal, resulting in wide QRS complexes. A wide QRS complex does not necessarily exclude a normal His bundle, as it may represent a more distal block in one of the bundle branches.

Can AV dissociation occur in the absence of AV block?

Yes. AV dissociation may occur during anesthesia with volatile agents in the absence of AV block and results from sinus bradycardia or an accelerated AV junctional rhythm. During isorhythmic dissociation, the atria and ventricles beat independently at nearly the same rate. The P wave often just precedes or follows the QRS complex, and their relationship is generally maintained. In contrast, interference AV dissociation results from a junctional rhythm that is faster than the sinus rate—such that sinus impulses always find the AV node refractory.

How do bifascicular and trifascicular blocks present?

A bifascicular block exists when two of the three major His bundle-branches (right, left anterior, or left posterior) are partially or completely blocked. If one fascicle is completely blocked and the others are only partially blocked, a bundle-branch block pattern will be associated with either first-degree or second-degree AV block. If all three are affected, a trifascicular block is said to exist. A delay or partial block in all three fascicles results in either a prolonged P–R interval (first-degree AV block) or alternating LBBB and RBBB. Complete block in all three fascicles results in third-degree AV block.

What is the significance of the electrocardiographic findings in this patient?

The electrocardiographic findings (first-degree AV block plus RBBB) suggest a bifascicular block. Extensive disease of the conduction system is likely. Moreover, the patient's syncopal and near-syncopal episodes suggest that she may be at risk of life-threatening bradyarrhythmias (third-degree AV block). Intracardiac electrocardiographic recordings would be necessary to confirm the site of the conduction delay.

What is appropriate management for this patient?

Cardiological evaluation is required because of the symptomatic bifascicular block. One of two approaches can be recommended, depending on the urgency of the surgery. If the surgery is truly emergent, a temporary transvenous pacing catheter or a transcutaneous pacemaker is indicated prior to induction of general or regional anesthesia. If the surgery can be postponed 24–48 hr (as in this case), continuous electrocardiographic monitoring, serial 12-lead ECGs, and measurements of cardiac isoenzymes are required to exclude myocardial ischemia or infarction and to try to record findings during symptoms.

What are general perioperative indications for temporary pacing?

Suggested indications include the following: any documented symptomatic bradyarrhythmia; second-degree (type II) AV block, or third-degree AV block and refractory supraventricular tachyarrhythmias.

The first three indications generally require ventricular pacing, whereas the fourth requires atrial pacing electrodes and a programmable rapid atrial pulse generator.

How can temporary cardiac pacing be established?

Pacing can be established by transvenous, transcutaneous, epicardial, or transesophageal electrodes. The most reliable method is generally via a transvenous pacing electrode in the form of a pacing wire or a balloon-tipped pacing catheter. A pacing wire should always be positioned fluoroscopically, but a flow-directed pacing catheter can also be placed in the right ventricle under pressure monitoring. A pacing wire must be used when blood flow has ceased. If the patient has a rhythm, intracardiac electrocardiographic recording showing ST-segment elevation when the

electrode comes in contact with the right ventricular endocardium confirms placement of either type of electrode. Specially designed pulmonary artery catheters have an extra port for passage of a right ventricular pacing wire. These catheters are particularly useful in patients with LBBB, who can theoretically develop complete heart block during catheter placement. Transcutaneous ventricular pacing is also possible via large stimulating adhesive pads placed on the chest and should be used whenever transvenous pacing is not readily available. Epicardial electrodes are usually used during cardiac surgery. Pacing the left atrium via an esophageal electrode is a simple, relatively noninvasive technique, but it is useful only for symptomatic sinus bradycardias and for terminating some supraventricular tachyarrhythmias.

Once positioned, the pacing electrodes are attached to an electrical pulse generator that periodically delivers an impulse at a set rate and magnitude. Most pacemaker generators can also sense the heart's spontaneous (usually ventricular) electrical activity: when activity is detected, the generator suppresses its next impulse. By altering the generator's sensing threshold, the pacemaker generator can function in a fixed (asynchronous) mode or in a demand mode (by increasing sensitivity). The lowest current through the electrode that can depolarize the myocardium is called the threshold current (usually <2 mA for transvenous electrodes).

What is AV sequential pacing?

Ventricular pacing often reduces cardiac output because the atrial contribution to ventricular filling is lost. When the AV conducting system is diseased, atrial contraction can still be maintained by sequential stimulation by separate atrial and ventricular electrodes. The P–R interval can be varied by adjusting the delay between the atrial and ventricular impulses (usually set at 150–200 ms).

How are pacemakers classified?

Pacemakers are categorized by a five-letter code, according to the chambers paced, chambers sensed, response to sensing, programmability, and arrhythmia function (**Table 21–22**). The two most commonly used pacing modes are VVI and DDD (the last two letters are frequently omitted).

If a pacemaker is placed in this patient, how can its function be evaluated?

If the patient's underlying rhythm is slower than the rate of a demand pacemaker, pacing spikes should be seen on the ECG. The spike rate should be identical to the programmed (permanent pacemaker—usually 72/min) or set (temporary) pacemaker rate; a slower rate may indicate a low battery. Every pacing spike should be followed by a QRS complex (100% capture). Moreover, every impulse should be followed by a palpable arterial pulse. If the patient has a temporary pacemaker, the escape rhythm can be established by temporarily slowing the pacing rate or decreasing the current output.

When the patient's heart rate is faster than the set pacemaker rate, pacing spikes should not be observed if the generator is sensing properly. In this instance, ventricular capture cannot be evaluated

TABLE 21–22 Classification of pacemakers.

Chamber-Paced	Chamber-Sensed	Response to Sensing	Programmability	Antitacharrhythmia Function
O = none	O = none	O = none	O = none	O = none
A = atrium	A = atrium	T = triggered	P = simple	P = pacing
V = ventricle	V = ventricle	I = inhibited	M = multi-programmable	S = shock
D = dual (atrium and ventricle)	D = dual (atrium and ventricle)	D = dual (triggered and inhibited)	C = communicating R = rate modulation	D = dual (pacing and shock)

unless the pacemaker rate increases or the spontaneous heart rate decreases. Fortunately, when the battery is low, sensing is generally affected before pacing output decreases. A chest radiograph is useful in excluding fracture or displacement of pacing leads. If pacemaker malfunction is suspected, cardiological consultation is essential.

What intraoperative conditions may cause the pacemaker to malfunction?

Electrical interference from surgical electrocautery units can be interpreted as myocardial electrical activity and can suppress the pacemaker generator. Problems with electrocautery may be minimized by limiting its use to short bursts, limiting its power output, placing its grounding plate as far from the pacemaker generator as possible, and using bipolar cautery. Moreover, continuous monitoring of an arterial pulse wave (pressure, plethysmogram, or oximetry signal) is mandatory to ensure continuous perfusion during electrocautery.

Both hypokalemia and hyperkalemia can alter the pacing electrodes' threshold for depolarizing the myocardium and can result in failure of the pacing impulse to depolarize the ventricle. Myocardial ischemia, infarction, or scarring can also increase the electrodes' threshold and cause failure of ventricular capture.

What are appropriate measures if a pacemaker fails intraoperatively?

If a temporary pacemaker fails intraoperatively, the inspired oxygen concentration should be increased to 100%. All connections and the generator battery should be checked. Most units have a battery-level indicator and a light that flashes with every impulse. The generator should be set into the asynchronous mode, and the ventricular output should be set on maximum. Failure of a temporary transvenous electrode to capture the ventricle is usually due to displacement of the electrode away from the ventricular endocardium; careful slow advancement of the catheter or wire while pacing often results in capture. Pharmacological management (atropine, isoproterenol, or epinephrine) may be useful until the problem is resolved. If an adequate arterial blood pressure cannot be maintained with adrenergic agonists, cardiopulmonary resuscitation should be instituted until another pacing electrode is placed or a new generator box is obtained. Transcutaneous pacing can be employed.

If a permanent pacemaker malfunctions (as with electrocautery), it should generally be converted to an asynchronous mode. Some units will automatically reprogram themselves to the asynchronous mode if malfunction is detected. Other pacemaker units must be reprogrammed by placing either an external magnet, or, preferably, a programming device over the generator. The effect of an external magnet on some pacemakers—particularly during electrocautery—may be unpredictable and should generally be determined prior to surgery.

Which anesthetic agents are appropriate for patients with pacemakers?

All anesthetic agents have been safely used in patients who already have pacemakers. Even volatile agents seem to have no effect on pacing electrode thresholds. Local anesthesia with moderate to deep intravenous sedation is usually used for placement of permanent pacemakers.

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