To my wonderful wife of 30 years. Thank you for all your support and understanding.—F.A.H.

To my wife and best friend—an indispensable partner in the school of life.—D.E.M.

To my father, James L. Gravlee, who gave me the self-confidence to believe I could accomplish whatever I “set my mind” to.—G.P.G.
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Preface

When the original two editors (Drs. Hensley and Martin) began *A Practical Approach to Cardiac Anesthesia* back in 1988, they had no idea how popular and useful this text would be to the perioperative teams who care for cardiothoracic patients around the world. Originally, the concept was to create an institutional teaching manual at Penn State for our residents, but it mushroomed into something much more.

Almost 20 years later, we strive to enhance and refine the content to continue the book's relevance and value as a handy and portable reference both in and out of the cardiothoracic operating room. The addition of Dr. Gravlee as contributing editor on the third and fourth editions has certainly enhanced the value of this reference (F.A.H., D.E.M.).

Our main structure of the book remains intact from the last edition. There are four main sections. Part I deals with general perioperative management of the adult patient undergoing cardiac surgery. Part II presents a detailed explanation of specific cardiac disorders. Part III deals with mechanical circulatory support and organ preservation. Part IV addresses aspects of thoracic surgery and pain management.

Since the time between editions three and four was far shorter than that between editions two and three (4 years versus 8 years), our emphasis has been on significant chapter updates. We have also added an important chapter on ventricular restoration procedures, which are currently performed at a limited number of centers. The future relevance of these procedures in generalized practice will be defined by evidence-based studies that are under way. We deleted the chapter on cardiothoracic emergencies and incorporated the most relevant issues into other existing chapters. Additionally in Part I, we combined the previous separate chapters on weaning from bypass and the postbypass period.

We hope this edition will continue to meet the needs of the perioperative team in the challenging and exciting field of cardiothoracic surgery.

F.A.H.
D.E.M.
G.P.G.
Anesthetic Management for Cardiac Surgery
The Cardiac Surgical Patient

Donald E. Martin and Charles E. Chambers

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CARDIOVASCULAR DISEASE IS OUR SOCIETY'S number one health problem, affecting 70.1 million Americans, or 34% of our population. More than 17 million Americans have diseases affecting the heart itself. According to the most recent CDC data, of the 34 million Americans hospitalized in 2003, 4.4 million (13%) had heart disease. Furthermore, of the 44 million surgical procedures performed in the United States in 2003, approximately 6.8 million procedures involved the cardiovascular system, 467,000 were coronary artery bypass procedures, and 93,000 were valvular procedures. Large series have estimated the mortality rates in adults to be approximately 1.8% for coronary artery surgery and 1.9% for aortic valve surgery. In 2005, cardiovascular disease cost the United States over $254 billion, of which $142 billion was spent to treat coronary artery disease (CAD) alone. The prime goals of preoperative evaluation and therapy for cardiac
surgical procedures, therefore, are to quantify and reduce the patient’s risk during surgery and the postoperative period.

The factors that are important in determining perioperative morbidity and anesthetic management must be assessed carefully for each patient.

I. Patient presentation

A. Age and sex

1. Age. It is estimated that the number of people over age 65 in the United States will increase 25% to 35% over the next 30 years. In patients undergoing cardiac revascularization, the Collaborative Study in Coronary Artery Surgery (CASS) [1] found operative mortality in patients older than 70 to be approximately 7.9%. Others have confirmed their findings more recently, concluding that patients between 80 and 90 years of age had higher mortality rates, as well as a higher incidence of respiratory failure, blood usage, and a longer ICU stay, but not a longer hospital stay. Age has been found to be a significant risk factor in cardiac surgery, strongly correlating with morbidity and 30-day mortality. However, more than one third of patients with cardiovascular disease are currently over 65 years of age, and more than half of all cardiac surgical procedures are performed on patients in this age group. Further, both medical therapy such as the control of lipids and blood pressure, and procedures such as percutaneous coronary intervention (PCI) with drug-eluting stents, may further increase the age at which patients present for cardiac surgical procedures. Therefore, special care and therapeutic measures are required in elderly patients undergoing cardiac surgical procedures.

2. Sex. When patients of all ages are considered, operative mortality during coronary artery surgery is more than twice as high among women than among men.

B. Functional status. For patients undergoing most general and thoracic surgical procedures, perhaps the simplest and single most useful risk index is the patient’s functional status, or exercise tolerance. In major noncardiac surgery, Girish and colleagues found the inability to climb two flights of stairs showed a positive predictive value of 82% for postoperative pulmonary or cardiac complications [2]. Exercise tolerance is commonly measured in metabolic equivalents, or METs. One MET is the energy consumed by the body at rest. The energy requirements, in METs, of common daily activities are shown in Table 1.1. The ability to perform activities of daily living, such as climbing a flight of stairs, corresponds to an exercise capacity of approximately 4 METs. This is an easily measured and sensitive index of cardiovascular risk, which takes into account a wide range of specific cardiac and noncardiac factors.

C. Surgical problems and procedures. The complexity of the surgical procedure itself may be the most important predictor of perioperative morbidity for many patients. Most, but not all, cardiac surgical procedures include the risks associated with cardiopulmonary bypass. Recent studies, however, failed to demonstrate any reduction

<table>
<thead>
<tr>
<th>Table 1.1 Energy requirements for common activities</th>
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<tbody>
<tr>
<td>1 MET</td>
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<tr>
<td>↓</td>
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<tr>
<td>4 METS</td>
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</tbody>
</table>
in 30-day mortality, cognitive impairment at 12 months, or major morbidity in off-pump as compared to conventional coronary artery bypass. Any procedure requiring more than 40 minutes on cardiopulmonary bypass is associated with greater morbidity, which increases with increased bypass duration. The coronary artery bypass procedure itself presents the risks of graft occlusion and intracoronary air. Valvular and other intracardiac procedures present the risk of systemic and coronary air emboli. Procedures requiring ventriculotomy imply damage to ventricular muscle. Procedures on multiple heart valves, or on both the aortic valve and coronary arteries, carry a statistical morbidity much higher than that for procedures involving only a single valve or coronary artery bypass grafting (CABG) alone.

II. Clinical assessment and medical management of cardiac disease

A. Myocardial ischemia. In patients with known CAD, the most important risk factors that need to be assessed preoperatively are: (i) the amount of myocardium at risk, (ii) the ischemic threshold, or the heart rate at which ischemia occurs, (iii) the patient’s ventricular function or ejection fraction (EF), (iv) the stability of symptoms, because recent acceleration of angina may reflect a ruptured coronary plaque, and (v) current medical therapy as well as the potential for improvement perioperatively.

1. Stable coronary syndrome (stable angina pectoris). Angina pectoris, chest pain from myocardial ischemia, is most commonly related to exertion, but may occur after eating, or with emotion. It occurs whenever the energy demands of the myocardium exceed the supply. Chronic stable angina most often results from obstruction to coronary artery blood flow by a fixed atherosclerotic coronary lesion in at least one of the large epicardial arteries. In the absence of such a lesion, however, the myocardium may be rendered ischemic by coronary artery spasm, vasculitis, trauma, or hypertrophy of the ventricular muscle, as occurs in aortic valve disease.

Neither the location, duration, or severity of angina, nor the presence of diabetes or peripheral vascular disease (PVD), indicate the extent of myocardium at risk, or the anatomic location of the coronary artery lesions. Therefore, the clinician must depend on diagnostic studies, such as myocardial perfusion imaging (MPI), stress echocardiography, and cardiac catheterization to assist in establishing risk.

In patients with chronic stable angina, a reproducible amount of exercise, with its associated increases in heart rate and blood pressure, predictably precipitates angina. This angina threshold is an important guide to perioperative hemodynamic management.

The level of exercise producing symptoms, as described classically by the New York Heart Association and Canadian Cardiovascular Society classifications, predicts the risk of both an ischemic event and operative mortality. During coronary revascularization procedures, operative mortality for patients with class IV symptoms is almost double that for patients with class I angina [1].

Angina occurring at rest implies a subtotal obstruction by atherosclerotic plaque representing in most instances a ruptured plaque, coronary artery spasm, or spasm around a partially obstructing lesion. In patients with valvular heart disease, particularly aortic stenosis, angina at rest frequently implies coexisting CAD.

Stable angina often responds to medical therapy as well as to PCI. Patients are referred for surgery when refractory to medical therapy and not candidates for PCI because of such things as unprotected left main disease or diabetes and multivessel disease.

a. Principles of the medical management of stable angina [3]

1. Aspirin with clopidogrel used for aspirin allergic patients or in combination therapy for acute coronary syndromes
2. \(\beta\)-Blockade as initial therapy when not contraindicated
3. Calcium antagonists or long-acting nitrates as second-line therapy, or as first-line therapy when \(\beta\) blockade is contraindicated
4. Blood pressure control with ACE inhibitors in patients with diabetes or heart failure

5. Reducing risk by lowering cholesterol and blood pressure, stopping smoking, preventing or treating diabetes, and improving diet and exercise

2. Acute coronary syndrome (unstable angina pectoris). Sometimes called crescendo angina, preinfarction angina, or unstable coronary syndrome, this symptom complex usually presents as:
   a. Rest angina, usually prolonged >20 minutes, within the first week of onset
   b. New onset angina markedly limiting activity within 2 weeks of onset
   c. Increasing angina, which is more frequent, of longer duration, or occurs with less exercise.

   These symptoms often indicate rapid growth, rupture, or embolus of an existing plaque. Patients in this category have a higher incidence of myocardial infarction and sudden death, increased incidence of left main occlusion, and an operative mortality 3.5 times the average of that for all myocardial revascularization procedures [1]. The clinical factors important in determining the risk of myocardial infarction or death in patients with unstable angina are shown in Table 1.2.

   a. Medical management for acute coronary syndrome. The medical management of unstable angina or of a non-ST segment elevation myocardial infarction has two parts: (i) anti-ischemic therapy and (ii) antiplatelet and anticoagulant therapy. Medical anti-ischemic therapy depends largely on the presence or absence of ongoing ischemia and includes an aggressive approach to risk factor modification (Table 1.3 and Table 1.4).

---

**Table 1.2** Risk factors for death or myocardial infarction (MI) in patients with unstable angina

<table>
<thead>
<tr>
<th></th>
<th>High risk</th>
<th>Intermediate risk</th>
<th>Low risk</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Angina</strong></td>
<td>Accelerated angina within 48 hours</td>
<td>Past (&gt;20 min) rest pain, now resolved, with risk factors</td>
<td>Increased angina</td>
</tr>
<tr>
<td></td>
<td>Prolonged rest angina (&gt;20 min)</td>
<td>Rest angina relieved with NTG</td>
<td>Lower angina threshold</td>
</tr>
<tr>
<td></td>
<td>Angina at rest with &gt;1 mm ST changes</td>
<td>Angina with dynamic T-wave changes</td>
<td>New onset class III or IV angina within 2 weeks</td>
</tr>
<tr>
<td><strong>Associated Symptoms</strong></td>
<td>Pulmonary edema</td>
<td>Prior MI, vascular disease, CABG, prior Aspirin use</td>
<td>New onset angina 2 weeks to 2 months</td>
</tr>
<tr>
<td></td>
<td>New or worsening MR murmur</td>
<td>Age &gt; 70</td>
<td>None</td>
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<td>S3 or rales</td>
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<tr>
<td></td>
<td>Hypotension</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Age &gt; 75</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>EKG</strong></td>
<td>New bundle branch block</td>
<td>Pathologic Q-waves or resting ST depression in multiple leads (anterior, inferior, lateral)</td>
<td>Unchanged EKG</td>
</tr>
<tr>
<td></td>
<td>Sustained ventricular tachycardia</td>
<td></td>
<td></td>
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<tr>
<td><strong>Markers</strong></td>
<td>Elevated Troponin-T or Troponin-L &gt;0.1 ng/mL</td>
<td>Slightly elevated troponin-T &gt;0.01 but &lt;0.1 ng/mL</td>
<td>Normal</td>
</tr>
</tbody>
</table>

3. **Myocardial ischemia without angina** may be manifest by fatigue, rapid onset of pulmonary edema, cardiac arrhythmias, syncope, or an “anginal equivalent,” most often characterized as indigestion or jaw pain. Silent ischemia is more common in the elderly and diabetic patients, with 15% to 35% of all myocardial infarctions occurring as silent events, documented only on routine electrocardiogram (EGC). Whether related to coexisting disease or delayed therapy, silent ischemia has been associated with an unfavorable prognosis. Mangano and coworkers [4] found that perioperative myocardial ischemia is common, occurring before noncoronary surgery in 20%, during surgery in 25%, and postoperatively in 40% of patients with demonstrated, or risk factors for, CAD. **Perioperative ischemia was silent in greater than 75% of the patients studied.** More than half of perioperative infarctions associated with all types of surgery are believed to be “silent.”

4. **Prior myocardial infarction. Interval between prior infarction and surgery.** Patients who have suffered a preoperative myocardial infarction any
time greater than 1 month preoperatively no longer appear to benefit from the delay of noncardiac surgery. A history of complications, such as heart failure or dysrhythmias, in the early postinfarction period may nevertheless help to further predict perioperative problems [5].

In patients undergoing coronary revascularization procedures, the risk of death after perioperative infarction is lower, perhaps because the surgical procedure itself alters the course of the disease. Among patients reviewed in 1989 having isolated CABG surgery within 30 days of an acute myocardial infarction, the inhospital death rate was 5.7%. Mortality was highest (11.0%) for those undergoing cardiac surgery in the first 3 days after infarction, decreased to 5.9% from 4 to 7 days after infarction, and was only 2.4% in those patients operated on between 8 and 30 days following infarction. These data led to the recommendation that elective CABG surgery be delayed until at least 1 week after myocardial infarction [6].

Perioperative infarction occurs most often in the first 2 to 3 postoperative days and has a very high (50% to 70%) mortality, much higher than that for myocardial infarctions not occurring in the setting of surgery, perhaps because perioperative infarctions are commonly “silent” and therefore untreated.

Perioperative myocardial infarction in patients with prior coronary bypass. In patients who have undergone coronary artery bypass surgery and have returned to the operating room for noncardiac procedures, the risk of perioperative infarction (0% to 1.2%) and perioperative cardiac death (0.5% to 0.9%) is low and is similar to that in patients with no prior infarction. In contrast, higher mortality appears to be associated with noncardiac surgery performed at the same time as CABG surgery.

Location and extent of infarction. An anterior infarction is more likely to be associated with left ventricular (LV) failure, whereas an inferior infarction is likely to be associated with bradycardia and heart block.

B. Congestive heart failure

1. Systolic vs. diastolic heart failure. Systolic ventricular failure impairs the ventricular ability to contract, or empty and expel blood normally. Manifestations of systolic dysfunction usually relate to reduced cardiac output, such as reduced exercise tolerance, weakness, fatigue, and in some cases, low resting cardiac output or metabolic acidosis.

Diastolic heart failure, in contrast, reduces the ventricular ability to relax, and fill normally, and is associated with increased filling pressures and systemic or pulmonary edema. Disorders that impede ventricular outflow, such as severe aortic stenosis, lead to systolic failure, whereas those limiting ventricular inflow, like myocardial fibrosis or hypertrophy or constrictive pericarditis, lead to diastolic dysfunction. Acute heart failure is usually manifest as systolic dysfunction, whereas diastolic dysfunction, as a cause of heart failure, has a more insidious presentation. The heart often responds to injury by remodeling, structural changes to minimize wall stress. This remodeling usually takes the form of concentric hypertrophy, with increased wall thickness, in response to pressure loads and eccentric hypertrophy, or increased chamber size, in response to volume loads.

In many patients, especially those with advanced disease, both types of dysfunction coexist.

2. Clinical assessment and medical management of heart failure. Ventricular dysfunction can occur almost immediately in association with an ischemic event and may be permanent following myocardial infarction. Ventricular dysfunction and heart failure have been classified into three stages, A through C, based on cardiac structural changes and symptoms of heart failure. Patients with Stage A disease have a high risk of developing heart failure, those in Stage B manifest
cardiac remodeling without symptoms, and those in Stage C have current or past heart failure symptoms. Management depends on the stage of the disease. ACE inhibitors and angiotensin II receptor blockers are usually used as first-line therapy, with the addition of β blockers and diuretics for Class B and C patients [7].

3. **Perioperative morbidity.** Evidence of congestive heart failure or ventricular dysfunction preoperatively is associated with an increased operative mortality. Recent series show a two- to threefold greater risk of postoperative morbidity or mortality in CAGB patients with preoperative congestive heart failure, and a 30-fold greater risk in patients with preoperative cardiogenic shock [8,9].

In patients undergoing aortic valve replacement (AVR) for critical aortic stenosis and depressed EF, a cardiothoracic ratio of ≥0.6 is possibly the most important predictor of operative mortality, increasing the risk in some series more than 10-fold.

C. **Dysrhythmias**

1. **Incidence.** Cardiac dysrhythmias are common in patients presenting for cardiac surgery, particularly in the elderly. In the perioperative period, abnormal rhythms occur in more than 75% of patients. However, those dysrhythmias are life-threatening in less than 1%.

2. **Supraventricular tachycardia (SVT).** Supraventricular tachycardias (SVTs) appear most often in the preoperative history as palpitations or near-syncope. Atrial fibrillation and flutter, the most common SVTs, increase in frequency with age and in association with organic heart disease. **Preoperative patients with SVT who are hemodynamically stable are usually managed with vagal maneuvers, adenosine, verapamil, or diltiazem.**

3. **Ventricular tachycardia (VT).** Ventricular dysrhythmias may lead directly to ventricular fibrillation, especially if they occur in the setting of acute or recent infarction. However, asymptomatic ventricular arrhythmias, even nonsustained VT, have not been associated with complications following noncardiac surgery. Patients with wide complex tachycardia of unknown origin, which may be ventricular, are usually managed with DC cardioversion or amiodarone. If they have good ventricular function, procainamide and sotolol may also be used.

4. **Bradyarrhythmias.**Anesthetics frequently affect sinus node automaticity but rarely cause complete heart block. Asymptomatic patients with ECG-documented atrioventricular conduction disease (PR prolongation or single or bifascicular bundle branch block) rarely require temporary pacing perioperatively. However, symptomatic patients, or patients with Mobitz II or complete heart block, require preoperative evaluation for permanent pacing. Patients with a recent myocardial infarction or with both first-degree atrioventricular (AV) block and bundle branch block may need temporary transvenous or transcatheter pacing perioperatively.

**Patients with left bundle branch block in whom a Swan-Ganz catheter is being placed may need availability of a transcatheter pacemaker because of the risk of inducing right bundle branch block, and thus complete heart block, during passage of the pulmonary artery catheter.** Patients with a left bundle branch block and right CAD may be at particular risk during the passage of a Swan-Ganz catheter.

Patients with an indwelling cardiac pacemaker or implantable cardioverter defibrillator need to have their device identified and evaluated preoperatively. Special precautions need to be considered, as outlined in Chapter 15, to prevent untoward effects of electromagnetic interference in the operating room.

D. **Multifactorial risk indices.** Multifactorial risk indices, which combine and assign relative importance to many clinical parameters, are perhaps more useful than any single factor in determination of a patient's cardiovascular risk, or risk of overall morbidity. Since the 1970s, many of these indices have been proposed [5,10-13] for patients undergoing noncardiac surgery as well as for cardiac surgery, particularly CAGB [8,9,14].
Table 1.5 Multifactorial indices of perioperative cardiovascular risk for noncardiac surgical procedures: summary of significant risk factors in recent multivariate analyses

<table>
<thead>
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</tr>
</thead>
<tbody>
<tr>
<td>Advanced ≥ 70 yr</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Angina</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Acute coronary syndrome</td>
<td>XX</td>
<td>X</td>
<td>X</td>
<td>XX</td>
<td>XX</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td>XX</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Heart failure</td>
<td></td>
<td>XX</td>
<td>X</td>
<td>XX</td>
<td>XX</td>
</tr>
<tr>
<td>Exercise tolerance</td>
<td></td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Valvular aortic stenosis</td>
<td>X</td>
<td>XX</td>
<td>XX</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Supraventricular dysrhythmias</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Ventricular dysrhythmias</td>
<td></td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
<td></td>
<td></td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Pulmonary disease</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Renal disease</td>
<td></td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Liver disease</td>
<td></td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Stroke</td>
<td></td>
<td>XX</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td></td>
<td>X</td>
<td></td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>Surgical site</td>
<td></td>
<td>X</td>
<td></td>
<td>XX</td>
<td>XX</td>
</tr>
<tr>
<td>Emergency surgery</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>β-blocker therapy</td>
<td></td>
<td></td>
<td></td>
<td>X</td>
<td>X</td>
</tr>
</tbody>
</table>

X, risk factor; XX, major risk factor

The clinical risk factors for noncardiac surgery, and the relative importance assigned to them by five of the major multifactorial indices, are shown in Table 1.5. Most of these indices contain some factors which assess the severity of ischemic heart disease, congestive heart failure, valvular heart disease, general medical status, and type of surgery. An index described by Boersma and colleagues in JAMA in 2001 includes for the first time the preoperative use of beta blockade as an independent factor reducing risk. Gilbert and colleagues compared the ability of four indices to predict cardiovascular morbidity in 2,035 patients and found no significant difference [15].

In 1997, the American College of Cardiologists and American Heart Association (ACC/AHA) first joined forces to craft a simple three-tiered scheme, as revised in 2002, which emphasizes the importance of the patient’s exercise tolerance and surgical procedure to perioperative risk. This is now probably the most widely used of all indices. It is shown schematically in Figure 1.1 [5,12].

The preoperative clinical factors which affect hospital survival following CABG surgery have been studied, retrospectively and prospectively, by several authors from 1995 to 1999 [8,9,14,16,17]. The most important risk factors in these studies are...
Figure 1.1 Preoperative assessment of cardiac disease based on clinical risk factors, patient exercise tolerance, and the nature of the surgical procedure. "High surgical risk" procedures include aortic and other vascular procedures and prolonged procedures with anticipated large fluid shifts and/or blood loss. "Intermediate surgical risk" procedures include carotid endarterectomy, as well as major head and neck, intrathoracic, intraperitoneal, orthopedic, and prostate surgery. "Low surgical risk" procedures include superficial, extremity, and endoscopic procedures. (Reproduced from Eagle KA, Berger PB, Callens H, et al. ACC/AHA Guideline update on perioperative cardiovascular evaluation for noncardiac surgery - Executive Summary. Circulation 2002;105:1260, Figure 1, with permission.)

compared in Table 1.6. In 2001, Dupuis and colleagues developed and validated the cardiac risk evaluation (CARE) score, which incorporated similar factors but viewed them more intuitively in a manner similar to American Society of Anesthesiologists (ASA) physical status (Table 1.7). In 2004, Ouattara et al. [18] compared the CARE score to two other multifactorial indices, the Tu score and Euroscore. Their analysis found no difference among these scores in predicting mortality and morbidity following
Table 1.6 Multifactorial indices of cardiovascular risk for cardiac surgical procedures: summary of significant risk factors in recent multifactorial indices

<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>2-3</td>
<td>XX</td>
<td>3</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td><strong>Cardiac factors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous cardiac operation</td>
<td>2</td>
<td>XX</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Urgency of surgery</td>
<td>1-4</td>
<td>XX</td>
<td>5</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>Complexity of surgery</td>
<td>2-3</td>
<td>XX</td>
<td>6</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>(single or multiple valve, valve/CABG)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Catheter-induced coronary closure</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severity of angina</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of previous MIs</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td></td>
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<tr>
<td>Congestive heart failure</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>LV ejection fraction</td>
<td>1-3</td>
<td></td>
<td></td>
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<tr>
<td>Ventricular arrhythmias</td>
<td></td>
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<tr>
<td>Systolic PAP &gt; 60 mm Hg</td>
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<td></td>
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<tr>
<td>Endocarditis</td>
<td></td>
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<tr>
<td><strong>Systemic factors</strong></td>
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<td></td>
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<tr>
<td>Cerebrovascular disease</td>
<td>X</td>
<td></td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>PVD</td>
<td>X</td>
<td></td>
<td>2</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>COPD</td>
<td>X</td>
<td></td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Insulin-dependent diabetes mellitus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Renal insufficiency</td>
<td></td>
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<tr>
<td>Anemia</td>
<td></td>
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<tr>
<td>Albumin &lt; 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low body mass index</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

*d* Jones, et al.: X, risk factor; XX, major (core) risk factor.  
*Magovern, et al., Higgins, et al.*: clinical risk score (1=lowest risk to 7=highest risk).  
*c* Higgins, et al.: only preoperative factors included.  
*d* Tu, et al.: points for each risk factor (maximum points 16).  
*e* Nashef, et al.: (Euroscore) points for each factor (maximum points 28-30).

cardiac surgery. Therefore, the relative simplicity of the CARE score, like that of the ACC/AHA risk index for noncardiac surgery, may have some advantage.

### III. Noninvasive cardiac diagnostic studies

**A. Electrocardiogram.** ECG abnormalities are common, occurring in 2% to 45% of patients undergoing noncardiac surgery, depending on their age and cardiovascular history. Some authors have found similar rates of ECG abnormalities in patients undergoing CABS surgery and those undergoing noncardiac surgery, thus calling into question the predictive value of the ECG, particularly for patients with known coronary disease. On the other hand, several authors have shown a close relationship between the presence of ischemic changes, Q waves, LV hypertrophy, or left bundle branch block detected by preoperative resting ECG before and during surgery and postoperative cardiac morbidity after noncardiac surgery [4]. The ACC/AHA guidelines recommend a resting 12-lead ECG particularly in patients with a recent episode of chest pain or its equivalent or in diabetic patients, because of their increased risk...
Table 1.7 Cardiac anesthesia risk evaluation (CARE)

<table>
<thead>
<tr>
<th>Risk score</th>
<th>Cardiac disease</th>
<th>Systemic medical disease</th>
<th>Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Stable</td>
<td>None</td>
<td>Noncomplex</td>
</tr>
<tr>
<td>2</td>
<td>Stable</td>
<td>Controlled</td>
<td>Noncomplex</td>
</tr>
<tr>
<td>3</td>
<td>Unstable cardiac disease</td>
<td>(and/or) Uncontrolled medical problem</td>
<td>(or) Complex surgery*</td>
</tr>
<tr>
<td>4</td>
<td>Unstable cardiac disease</td>
<td>(and/or) Uncontrolled medical problem</td>
<td>(and) Complex surgery*</td>
</tr>
<tr>
<td>5</td>
<td>Chronic or advanced cardiac disease—surgery undertaken as last hope</td>
<td></td>
<td></td>
</tr>
<tr>
<td>E</td>
<td>Emergency: surgery undertaken as soon as diagnosis made and facilities available</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Complex surgery: examples include reoperation, multiple valve or combined valve/CABG surgery, LV aneurysmectomy, CABG of diffuse or calcified disease.


of silent ischemia [12]. Preoperative electrocardiography should ideally be performed within 24 to 48 hours before the procedure.

B. Chest roentgenogram. Routine preoperative chest films have not been found to be cost effective for most noncardiac or nonthoracic procedures. However, specific information derived from these studies in cardiac and thoracic patients justifies their routine preoperative use.

C. Echocardiography. Transthoracic echocardiography is a noninvasive test that provides specific preoperative assessment of several types of cardiac abnormalities. First, two dimensional (2D) and Doppler echocardiography together provide quantitative assessment of the severity of valvular stenosis or insufficiency, and of pulmonary hypertension. Second, assessment of regional wall motion provides a more sensitive and specific assessment of the existence and extent of myocardial infarction than a surface EKG. Third, 2D echocardiography provides a quantitative assessment of global ventricular function, or EF. Fourth, echocardiography can detect even small pericardial effusions. Fifth, echocardiographs can detect anatomic cardiac abnormalities, from atrial septal defects (ASD) and ventricular septal defects (VSD) to aneurysms and mural thrombi.

Perioperative trans-thoracic echocardiography provides independent information to predict postoperative cardiac events in noncardiac surgical patients at increased clinical cardiac risk. Preoperative systolic dysfunction has been associated with postoperative myocardial infarction, pulmonary edema, and “major cardiac events,” such as ventricular fibrillation, cardiac arrest, or complete heart block. LV hypertrophy, mitral regurgitation, and increased aortic valve gradient on preoperative echo also appear to predict postoperative “major cardiac events.”

D. Preoperative testing for myocardial ischemia

1. Exercise tolerance testing. The exercise tolerance test (ETT) is often used as a simple and inexpensive initial test to evaluate chest pain of unknown etiology. It is also used preoperatively to determine functional capacity and identify significant ischemia or dysrhythmias for prognostic stratification preoperatively. ETT is rarely useful as a screening test in asymptomatic patients. To better address the prognostic value of the ETT, the Duke risk score was developed [19]. This risk score equals the exercise time in minutes, minus five times the extent of the ST segment depression in millimeters, minus four times the level of angina with exercise (0-no angina, 1-typical angina, 2-typical angina requiring stopping
the test). The score typically ranges from $-25$ to $+15$. These values correspond to low risk (with a score of $\geq +5$), moderate risk (with scores ranging from $-10$ to $+4$), and high-risk (with a score of $<-11$) categories.

a. **Limitations of ETT**
   1. Inability to exercise because of systemic disease, particularly PVD
   2. Abnormal resting ECG precluding ST segment analysis (left bundle branch block, LV hypertrophy, digoxin therapy)
   3. β-Blocker therapy that prevents the patient from achieving 85% of his or her maximum permissible heart rate

2. **Stress echocardiography.** Stress echocardiography can use exercise stress or pharmacologic stress, with dobutamine, to increase myocardial work. Abnormally contracting myocardial segments seen on stress echocardiography are classified as either *ischemic*, if their reduced contraction pattern is in response to stress, or *infarcted*, if their contractility remains consistently depressed before, during, and after stress.

   Twelve recent studies evaluated in the 2002 ACC/AHA Guidelines for Perioperative Cardiovascular Evaluation showed that 7% to 25% of vascular patients who had a positive preoperative **Dobutamine Stress Echocardiogram (DSE)** subsequently suffered a post operative MI or death. The negative predictive value was much higher—93% to 100% [5]. Wall motion abnormalities at low workloads were especially important predictors. DSE has indications similar to pharmacologic perfusion imaging with comparable sensitivity, but possibly increased specificity. Preoperative dipyridamole thallium perfusion imaging, radionuclide ventriculography, ambulatory EKG, and DSE are all able to predict adverse outcomes in with the relative risk for DSE likely to be slightly, but not significantly, greater than the other studies.

   For patients with poor acoustic windows due to body habitus or severe lung disease, myocardial contrast agents are now available to improve imaging. Still, for some patients, a difficult echocardiography window or global poor ventricular function may preclude its use. Further, this test cannot be used for those patients in whom a recent myocardial infarction, an intracranial or abdominal aneurysm, or other vascular malformation would make tachycardia or hypertension risky.

3. **Radionuclide imaging.** Radionuclide stress imaging is used to assess the perfusion and the viability of areas of myocardium. This technique cannot provide an anatomic diagnosis of a cardiac lesion. It is a more sensitive and specific test than ETT and can provide an assessment of global LV function as well. **Myocardial perfusion imaging** is a nuclear technique employing intravenous radioisotopes, either thallium-201 or the cardiac specific technetium-99 perfusion agents, sestamibi (Cardiolite), or tetrofosmin (Myoview), as an indicator of the presence or absence of CAD.

   Exercise stress or pharmacologic stress is necessary to increase coronary blood flow for the test. Pharmacologic vasodilators are preferable but contraindicated in patients with severe bronchospastic lung disease, in which case dobutamine may be used. The available pharmacologic vasodilators, adenosine and dipyridamole (Persantine), are used to produce maximal coronary vasodilation of approximately four to five times resting values. Vessels with fixed coronary stenoses will not dilate, allowing fewer isotopes to reach the myocardium. Myocardium perfused by these vessels will show up as a “defect” on stress scans when compared to surrounding myocardium supplied by nonobstructed coronaries. When compared to the images acquired at rest, any defects still present—*fixed or persistent defects*—are suggestive of nonviable or infarcted myocardium. Defects present on stress and not at rest, termed *reversible defects*, suggest viable myocardium at risk for ischemia when stressed.
A perfusion scan may be performed three different ways. When thallium is chosen, only a single injection is required because the isotope redistributes; however, a repeat image should be taken 4 hours after the stress images are taken. With the technetium agents, particularly best for larger patients due to the higher energy (KeV), separate rest and stress injections are required because no redistribution is seen. Finally, dual isotopes studies utilizing thallium for the initial rest image and technetium for the stress image allows for the fastest patient through put.

The technique used to acquire these images is single photon-emission computed tomography (SPECT). In the studies of noncardiac surgical patients reviewed by the ACC/AHA Task Force on Perioperative Cardiovascular Evaluation, thallium perfusion scanning identified 4% to 20% of patients suffering postoperative myocardial infarction or cardiac death. The negative predictive value of a normal scan is much better, at approximately 99%. The sensitivity and specificity of nuclear perfusion imaging is similar for pharmacologic and stress-based techniques [12].

The predictive value of the test can be improved by using it in high-risk subgroups. Selective use of pharmacologic perfusion imaging in patients who have at least one of two clinical risk factors for ischemic disease (age >70 years and congestive heart failure) can maximize the usefulness of this procedure in predicting cardiac outcome in patients undergoing noncardiac surgery of all types.

Contraindications to pharmacologic stress with adenosine or dipyridamole are:
- Unstable angina or myocardial infarction within 48 hours
- Severe primary bronchospasm
- Methylxanthine ingestion within 24 hours
- Allergy to dipyridamole or aminophylline
- For adenosine only, first-degree heart block (PR interval >0.28 seconds) and recent oral dipyridamole ingestion (<24 hours ago)

Pharmacologic vasodilators should be used in patients who cannot exercise, or have a medical condition, such as a cerebral aneurysm, which would contraindicate exercise. Pharmacologic stress testing with vasodilators, such as adenosine or dipyridamole, is also preferable to exercise or dobutamine in patients with left bundle branch block, because of spurious septal changes with exercise or catecholamines, which lead to false positive tests [12].

4. PET scan. Positron emission tomography (PET) scanning techniques use different radioisotopes than SPECT imaging. These isotopes have more rapid energy decay, with a shorter half life, and can assess both regional myocardial blood flow and myocardial metabolism on a real-time basis. The higher cost of this technique, which also includes a cyclotron to produce the isotopes, often precludes its widespread use.

5. Magnetic resonance imaging (MRI). MRI has been used for some time to provide both high resolution and three-dimensional imaging of cardiac structures. It is now becoming important in perfusion imaging, atherosclerosis imaging, and coronary artery imaging. With the development of dedicated cardiovascular MRI scanning, molecular imaging techniques and biochemical markers are providing the capacity for MRI diagnosis of cardiac function. Changes in molecular composition of the myocardium can change its magnetic moment and MRI signal, allowing MRI to detect lipid accumulation, edema, fibrosis, rate of phosphate turnover, and intracellular pH in ischemic areas. Finally, MRI imaging can be gated to the cardiac cycle, allowing rapid and accurate assessment of myocardial function. Gated images are used to detect regional myocardial abnormalities that may be caused by ischemia, infarction, stunning, hibernation, and postinfarct remodeling. MRI is the diagnostic technique of choice for arrhythmogenic RV dysplasia and can differentiate myocardial infiltration and diastolic dysfunction associated with sarcoidosis, hemochromatosis, amyloidosis, and endomyocardial
fibrosis. Contrast enhanced MRI has a higher sensitivity and specificity than either CT scan or TEE in aortic dissection. Dobutamine stress MRI is an accurate and rapid test for myocardial ischemia, which may eventually replace dobutamine echocardiography.

MRI can be used to diagnose CAD involving the native major epicardial arteries with an accuracy of approximately 87% and is even better for assessing saphenous vein and internal mammary artery graft patency. However, it is still not commonly used clinically for this purpose.

6. **Computed tomography.** Since its introduction into clinical practice in 1973, computed tomography (CT) has undergone significant advances. CT for calcium scoring has been utilized clinically to estimate cardiac risk but is not effective for defining atherosclerotic disease. With the development of a higher resolution scan, in conjunction with contrast injection, coronary imaging is now possible. As these imaging techniques advance in the cardiology arena, they can be used for imaging the pericardium, cardiac chambers, and great vessels. However, imaging protocols require aggressive beta blockade to achieve heart rates of 60 bpm or less in order to decrease image blurring and improve resolution.

Lack of extensive clinical expertise in this field as well as the limited prognostic data as to the significance of CT findings preclude recommending this technique as a substitute for diagnostic cardiac catheterization at this time.

IV. **Cardiac catheterization**

A. **Overview.** Cardiac catheterization still is considered the gold standard for diagnosis of cardiac pathology before most open heart operations and for definition of lesions of the coronary vessels. More than 95% of all patients undergoing open heart operations have had catheterization prior to the procedure. The remaining 5% are assessed only by noninvasive techniques, such as echocardiography and Doppler flow studies. They have pathologic findings, such as an ASD or VSD, which are adequately defined by noninvasive means.

As an invasive procedure, serious complications occur in approximately in 0.1% of patients and include stroke, heart attack, and death. Significant access site complications occur approximately in 0.5%. Numerous and more extensive publications on the cardiac catheterization laboratory and indications for left and right heart catheterization are available [20].

Most formal catheterization reports contain:
- A brief summary of indications for the catheterization procedure
- A description of the catheterization procedure itself
- Hemodynamic data, including chamber pressures, arterial and venous saturations, and cardiac output
- Descriptive information on coronary anatomy, ventricular function, and valvular regurgitation
- Calculation of derived parameters including valve areas, EF, and pulmonary and systemic vascular resistances

If only coronary anatomy is to be delineated, often only a systemic-arterial or left-sided catheterization will be performed. However, if any degree of LV dysfunction, valvular abnormality, pulmonary disease, or impaired right ventricular (RV) function exists clinically, a right-sided (Swan-Ganz) catheterization will also be performed. A range of normal hemodynamic values obtained from right- and left-sided catheterization is included in Table 1.8.

Interpretation of catheterization data emphasizes the following areas.

B. **Assessment of coronary anatomy**

1. **Procedure.** Radiopaque dye is injected through a catheter placed at the coronary ostia to delineate the anatomy of both the right and left coronary arteries. Multiple views are important to define branch lesions best, decrease artifacts at points of tortuosity or vessel overlap, and determine more clearly the degree of stenosis,
Table 1.8 Normal hemodynamic values obtained at cardiac catheterization

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Measurement</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peripheral arterial or aortic</td>
<td>Systolic/diastolic</td>
<td>≤140/90 mm Hg</td>
</tr>
<tr>
<td>pressure</td>
<td>Mean</td>
<td>≤105 mm Hg</td>
</tr>
<tr>
<td>Right atrial pressure</td>
<td>Mean</td>
<td>≤6 mm Hg</td>
</tr>
<tr>
<td>Right ventricular pressure</td>
<td>Systolic/end-diastolic</td>
<td>≤30/6 mm Hg</td>
</tr>
<tr>
<td>Pulmonary artery pressure</td>
<td>Systolic/diastolic</td>
<td>≤30/15 mm Hg</td>
</tr>
<tr>
<td></td>
<td>Mean</td>
<td>≤22 mm Hg</td>
</tr>
<tr>
<td>Pulmonary artery wedge pressure</td>
<td>Mean</td>
<td>≤12 mm Hg</td>
</tr>
<tr>
<td>Left ventricular pressure</td>
<td>Systolic/end-diastolic</td>
<td>≤140/12 mm Hg</td>
</tr>
<tr>
<td>Cardiac index</td>
<td></td>
<td>2.5–4.2 L/min⁻¹.m⁻²</td>
</tr>
<tr>
<td>End-diastolic volume index</td>
<td></td>
<td>≤100 mL/min⁻²</td>
</tr>
<tr>
<td>Arteriovenous O₂ content difference</td>
<td></td>
<td>≤5.0 mL/dL</td>
</tr>
<tr>
<td>Pulmonary vascular resistance</td>
<td></td>
<td>20–130 dyne-sec-cm⁻⁵</td>
</tr>
<tr>
<td></td>
<td>or</td>
<td>0.25–1.6 Woods units</td>
</tr>
<tr>
<td>Systemic vascular resistance</td>
<td></td>
<td>700–1,600 dyne-sec-cm⁻⁵</td>
</tr>
<tr>
<td></td>
<td>or</td>
<td>9–20 Woods units</td>
</tr>
</tbody>
</table>

particularly in eccentric lesions. Two common projections of the coronary arteries are the right anterior oblique (RAO) and the left anterior oblique (LAO) views (Fig. 1.2).

2. **Interpretation.** The degree of vessel stenosis generally is assessed by the percent reduction in diameter of the vessel, which in turn correlates with the reduction in cross-sectional area of the vessel at the point of narrowing. Lesions that reduce vessel diameter by greater than 50%, reducing the cross-sectional area by greater than 75%, are considered significant. Lesions are also characterized as either focal...
or segmental. There is a great deal of interobserver variability in interpretation of the degree of stenosis in the range of 40% to 70%.

C. **Assessment of left ventricular function.** Both global and regional measures of ventricular function can be obtained from catheterization data.

1. **Global ventricular measurements**
   a. **Left ventricular end-diastolic pressure (LVEDP).** An elevated value above 15 mm Hg usually indicates some degree of ventricular dysfunction. LVEDP is an index that may reflect either systolic or diastolic dysfunction and is acutely affected by preload and afterload. Without examining other indices of function, an isolated measurement of elevated LVEDP simply indicates that something is abnormal. Associated with a normal LV contractile pattern and cardiac output, an elevated LVEDP measurement may indicate a decrease in left ventricular compliance.

   b. **Left ventricular EF**
      (1) **Calculation.** EF is defined as the volume of blood ejected (stroke volume) per beat divided by the volume in the left ventricle (LV) before ejection [end-diastolic volume (EDV)]. The stroke volume (SV) is equal to the EDV minus the end-systolic volume (ESV). The equation for EF determination is therefore:
      \[
      EF = \frac{EDV - ESV}{EDV} = \frac{SV}{EDV}
      \]

      (2) **Mitral regurgitation.** An EF of greater than 50% is normal in the presence of normal valvular function. However, in the presence of significant mitral regurgitation, an EF of 50% to 55% suggests moderate LV dysfunction, because part of the volume is ejected backward into a low-resistance pathway (i.e., into the left atrium).

   c. **Diastolic volume index.** The end-diastolic volume indexed to the patient's body surface area is another global measure of ventricular performance. It can, however, be elevated in patients with regurgitant or volume overload lesions with preserved LV function (similar to the LVEDP). A normal index is considered less than 100 mL/m².

2. **Regional assessment of ventricular function.** LV contraction observed during ventriculography provides a qualitative assessment of overall ventricular function but is not as specific as the calculated EF. Qualitative regional differences in contraction may be evident. For examination, the heart is divided into segments. The anterior, posterior, apical, basal, inferior (diaphragmatic), and septal regions of the LV are examined (Figs. 1.3 and 1.4). Motion of each one of these particular regions is defined as normal, hypokinetic (decreased inward motion), akinetic (no motion), or dyskinetic (outward paradoxical motion) in relation to the other normally contracting segments.

   Regional wall motion abnormalities are usually secondary to prior infarction or acute ischemia. However, very infrequently myocarditis as well as rare infiltrative processes by myocardial tumors may lead to regional wall motion abnormalities.

D. **Assessment of valvular function.** This section will be limited to a brief discussion of the methods utilized to study lesions of the aortic and mitral valves. The specific hemodynamic patterns of acute and chronic valvular disease will be discussed in Chapter 11.

1. **Regurgitant lesions**
   a. **Qualitative assessment.** A relative scale of 1+ to 4+ (4+ being the most severe) is used to quantify the severity of valvular incompetence during the injection of dye. Visual inspection is utilized to determine the intensity
and rapidity of washout of dye from the LV after aortic root injection (aortic regurgitation) or from the left atrium after ventricular injection (mitral regurgitation).

**b. Calculation of regurgitant fraction.** The percentage of regurgitant blood flow can be calculated by the following equation:

\[
\text{Regurgitant fraction} = \frac{\text{SV angiographic} - \text{SV forward}}{\text{SV angiographic}}
\]

where forward stroke volume refers to the volume calculated from the thermodilution method divided by simultaneous heart rate. Significant heart rate variations will render this calculation erroneous. If both aortic and mitral regurgitation occur in combination, the total amount of regurgitation can be detected only qualitatively.

**c. Pathologic V waves.** In patients with mitral regurgitation, the pulmonary capillary wedge trace may manifest giant V waves. Normal or physiologic V
waves are seen in the left atrium at the end of systole and are secondary to filling from the pulmonary veins against a closed mitral valve. With valvular incompetence, the regurgitant wave into the left atrium is superimposed on a physiologic V wave, producing a giant V wave (Fig. 1.5).

2. Stenotic lesions. The severity of valvular stenosis can be determined only by knowing the size of the pressure drop across the stenotic valve and the amount of flow across the stenosis during either systolic ejection or diastolic filling. One cannot uniformly assess the severity of stenosis solely by examining the pressure gradient (either peak to peak or mean) across the valve.

Gorlin and Gorlin [20a] described an equation for determining valve area based on these two factors in the American Heart Journal in 1951. A simplified version of this equation is:

\[
\text{Valve area} = \frac{\text{cardiac output (Liters/min)}}{\sqrt{\text{mean pressure gradient}}}
\]

If the mean pressure gradient and cardiac output are given on the catheterization report, a quick estimate of either aortic or mitral valve area can be made.

When examining combined regurgitant and stenotic lesions of the same valve, the total or angiographic cardiac output must be used in the calculation; otherwise, the severity of stenosis will be overestimated. Values for normal and abnormal valve areas are discussed in Chapter 11.

Remember that catheterization data represent only one point in time, and medical management may have changed the hemodynamic pattern and catheterization results at the time of cardiac operation.

E. Percutaneous Coronary Intervention (PCI). In 1977, Andreas Gruentzig brought therapeutic options to the invasive cardiology practice with the first percutaneous transluminal coronary angioplasty (PTCA). Multiple technologies have
advanced since the initial balloon dilation. Current technology has evolved to include niche devices including rotational coronary atherectomy, various thrombectomy techniques, distal protection devices for saphenous vein graphs, and coronary stents [20].

Intracoronary stents provide local stabilization for PCI-induced coronary dissection and have significantly reduced the need for emergent coronary bypass surgery to <1%. Post-PCI re-stenosis, occurring in one third of balloon dilations, is a recurrent blockage resulting from a local vascular response to injury. Stents alone and in conjunction with polymer-based medications, drug-eluting stents (DES), have reduced re-stenosis rates to <10%.

With a stent as a foreign body, endothelialization is required to prevent thrombosis. During this period of endothelialization, approximately 1 month for nonmedicated stents and at least 6 months for DES, both clopidogrel (Plavix) and aspirin are required therapy and must be continued. Without aggressive antiplatelet therapy, stent thrombosis and closure is common with significant patient morbidity and mortality.

V. Systemic disease

A. Atherosclerotic vascular disease

1. Carotid disease. Approximately 20% of patients presenting for CABG surgery can be expected to have some coexisting carotid artery disease. Therefore, symptoms of transient ischemic attack or visual disturbance should be sought in all preoperative cardiac patients. The presence of these symptoms or of an asymptomatic carotid bruit should warrant at least noninvasive Doppler carotid flow studies before cardiac surgery. Patients with symptoms and a greater than 80% carotid stenosis, associated with stable cardiac disease, may benefit from carotid endarterectomy before elective cardiac surgery. There is no clear answer, however, for those patients who have both severe carotid stenosis and unstable CAD. CABG does present a 9% to 17% risk of stroke to patients with known carotid disease. Therefore, the options would usually be carotid endarterectomy before CABG, or both during the same anesthetic. However, recent series have found worse outcomes for the combined procedure, so endarterectomy, perhaps, under local anesthesia, followed by CABG at a later date, may be the preferred approach. Even in the absence of symptoms, it is wise to assume that every patient older than 70 with CAD also has some cerebrovascular disease, and intraoperative blood pressure management should be planned accordingly.

2. Renovascular disease. Severe recent-onset or recent exacerbation of previously controlled hypertension, abdominal bruits, or renal insufficiency should prompt a thorough investigation of the renal vasculature, including renin measurement. The use of angiotensin-converting enzyme inhibitors preoperatively in patients with renal artery stenosis can induce renal insufficiency.

3. PVD. Preoperative assessment of pulse strength is necessary to form a baseline for postoperative evaluation, to determine the most appropriate sites for arterial cannulation, and to locate the best peripheral insertion site for an intra-aortic balloon pump or arterial cardiopulmonary bypass cannula should the need arise.

B. Hypertension. The contribution of hypertension to perioperative morbidity and the implications for anesthetic management depend on (1) blood pressure level, both with stress and at rest, (2) the etiology of hypertension, (3) pre-existing complications of hypertension, and (4) physiologic changes due to drug therapy.

1. Blood pressure level. In contrast to the usual emphasis on the resting, unstimulated blood pressure in determining chronic medical management, the patient's blood pressure under stress, as in the preoperative clinic or holding area, may be a better predictor of their perioperative morbidity. Intraoperative cardiac morbidity in the form of dysrhythmias and ischemic ECG changes has been observed more frequently in hypertensive patients with awake diastolic blood pressures of greater than 180/110 mm Hg and
Section I. Anesthetic Management for Cardiac Surgery

Table 1.9 High risk factors for secondary hypertension

<table>
<thead>
<tr>
<th>Factor</th>
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<tbody>
<tr>
<td>Blood pressure &gt;180 systolic or &gt;110 diastolic</td>
</tr>
<tr>
<td>Blood pressure not controlled by two or more agents</td>
</tr>
<tr>
<td>Increase in previously well controlled blood pressure</td>
</tr>
<tr>
<td>Sudden onset, labile, or paroxysmal hypertension</td>
</tr>
<tr>
<td>Hypertension with onset before age 25 or after age 50</td>
</tr>
</tbody>
</table>

This morbidity can be reduced by preoperative treatment. Blood pressure between 140–180/90–110 has not been found to be an independent predictor of increased perioperative cardiac risk, but may be a marker for chronic cardiovascular disease [12]. Recent data summarized in the JNC VII report in 2003 indicate that cardiovascular risk begins to increase at blood pressures above 115/75 and doubles with each increment of 20/10. Patients with blood pressures of 120–139/80–89 are considered prehypertensive and require drug therapy if they have associated diabetes or renal disease. Patients with blood pressures of 140–159/90–99 are considered hypertensive and all require chronic drug therapy. Those with blood pressures greater than 160/100, classified as stage 2 hypertensive, usually require combination drug therapy. Further, for patients older than 50 years, systolic blood pressure >140 is a much more important cardiovascular risk factor than diastolic blood pressure [21].

2. Etiology. It is important preoperatively to exclude the 5% to 15% of patients with treatable causes of secondary hypertension, especially patients shown in Table 1.9. Common causes of secondary hypertension are usually renal, endocrine, or drug related, which account for an additional 5% to 10% of hypertensive patients. Other rare disorders are found in less than 1% of patients (Table 1.10). A laboratory investigation of secondary hypertension, when indicated, should include urinalysis, creatinine, glucose, electrolytes, calcium, EKG, and chest films. More extensive testing is usually not indicated unless blood pressure cannot be controlled.

Table 1.10 Causes of hypertension

<table>
<thead>
<tr>
<th>Medical cause (Incidence)</th>
<th>Drug-induced hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Essential hypertension (85%–95%)</td>
<td>Amphetamines</td>
</tr>
<tr>
<td>Renal (2%–6%)</td>
<td>Caffeine</td>
</tr>
<tr>
<td>Renal parenchymal disease</td>
<td>Cocaine</td>
</tr>
<tr>
<td>Renovascular disease</td>
<td>Chlorpromazine</td>
</tr>
<tr>
<td>Endocrine (1%–2%)</td>
<td>Cyclosporine</td>
</tr>
<tr>
<td>Pheochromocytoma</td>
<td>Erythropoietin</td>
</tr>
<tr>
<td>Cushing's disease</td>
<td>Ethanol</td>
</tr>
<tr>
<td>Thyroid or parathyroid disease</td>
<td>Licorice</td>
</tr>
<tr>
<td>Hyperaldosteronism</td>
<td>MAO inhibitors</td>
</tr>
<tr>
<td>Aortic coarctation (2%–5%)</td>
<td>Nicotine</td>
</tr>
<tr>
<td>Sleep apnea (1%)</td>
<td>NSAIDs</td>
</tr>
<tr>
<td>Rare causes of secondary hypertension</td>
<td>Oral contraceptives</td>
</tr>
<tr>
<td>Renin-producing tumors</td>
<td>Steroids</td>
</tr>
<tr>
<td>Adrenogenital syndrome</td>
<td>Sympathomimetics</td>
</tr>
<tr>
<td>Acromegaly</td>
<td>Nasal decongestants</td>
</tr>
<tr>
<td>Hypercalcemia</td>
<td>Weight loss regimens</td>
</tr>
<tr>
<td>Familial dysautonomia</td>
<td></td>
</tr>
<tr>
<td>Porphyria, neuropathies</td>
<td></td>
</tr>
</tbody>
</table>

Adapted from Chobanian AV, Bakris GL, Black HR, et al. The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure. JAMA 2003;289:2560–2572, with permission.