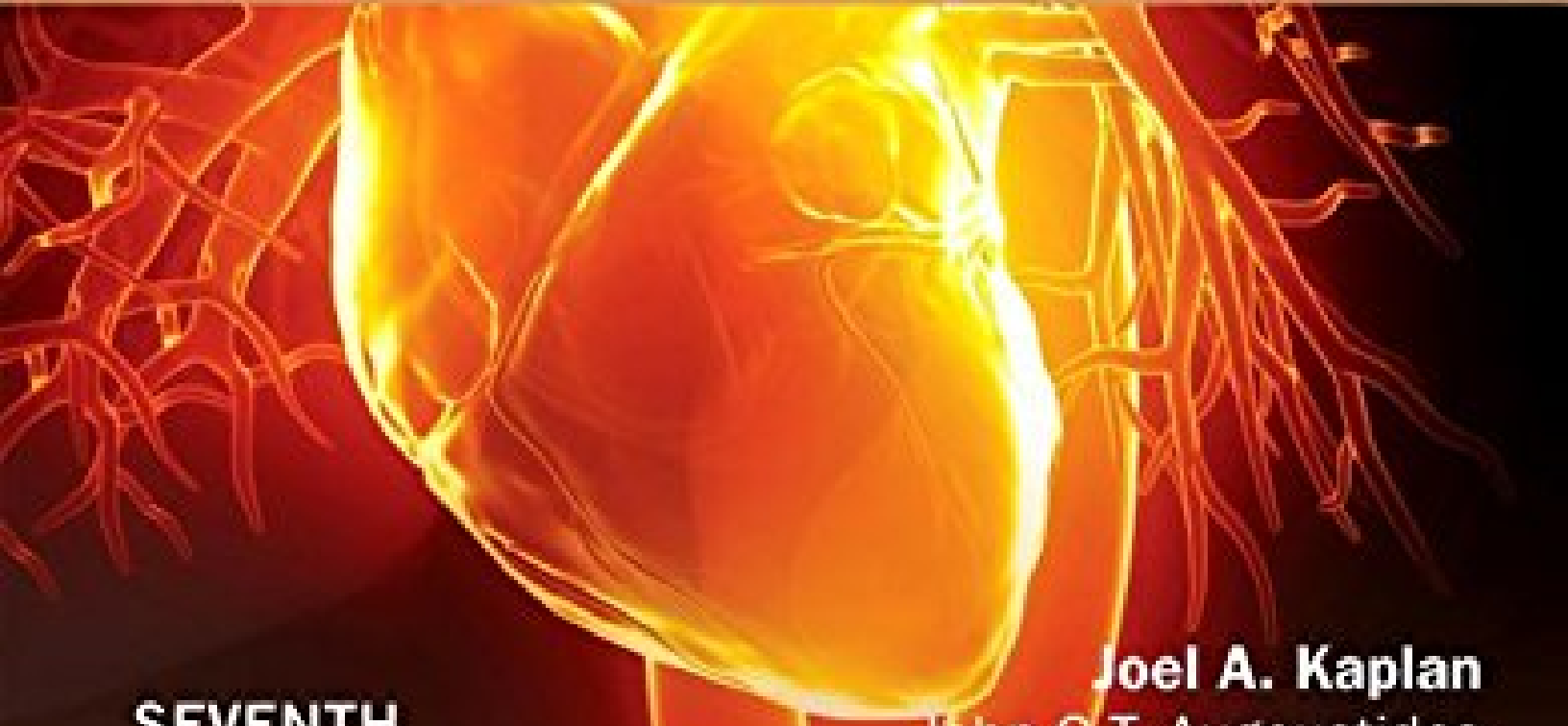
KAPLAN'S CARDIAC ANESTHESIA

FOR CARDIAC AND NONCARDIAC SURGERY



SEVENTH EDITION

Joel A. Kaplan
John G.T. Augoustides
Gerard R. Manecke, Jr.
Timothy Maus
David L. Reich

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Seventh Edition

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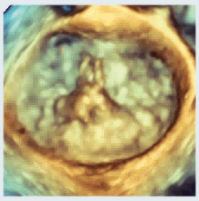

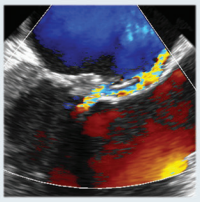

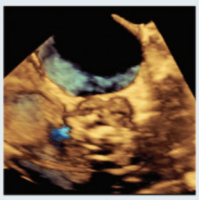

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DEDICATION

To my loving wife, Norma, on the occasion of our fiftieth wedding anniversary

Joel A. Kaplan, MD, CPE, FACC

To our families and loved ones for their support and understanding

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PREFACE

The seventh edition of *Kaplan's Cardiac Anesthesia* has been written to further enhance the perioperative management of the patient with cardiac disease undergoing either cardiac or noncardiac surgery. The first edition was published in 1979 during the early years of modern cardiac surgery when our focus was primarily on anesthetizing cardiac surgical patients. Continued advances in the care of patients with heart disease have expanded the role of cardiac anesthesiologists into preoperative evaluation, advanced cardiac imaging and other monitoring devices, postoperative critical care, and pain management. Today, we are also being asked to care for or help "rescue" the sickest cardiac patients undergoing noncardiac surgery and to participate as a member of the cardiac care team by performing procedures in locations distant from cardiac operating rooms. This edition focuses on the current issues associated with advanced cardiopulmonary assist devices, procedures in hybrid operating rooms, and new anticoagulants and coagulants, as well as being members of the heart team deciding on the best options for our sick patients.

This edition is subtitled "For Cardiac and Noncardiac Surgery" to emphasize the expanded role of all anesthesiologists in the perioperative medical care of high-risk cardiac patients undergoing all types of procedures. Ten chapters have been added as the final section of this book and deal specifically with these patients, the complex care and procedures they require, and the techniques used to reduce the incidence of major adverse cardiac events. This contrasts with only one chapter in the first edition that pointed out that some cardiac patients undergoing noncardiac surgery could be just as sick or sicker than those having cardiac surgery and that their cardiovascular complications could carry a high mortality rate after noncardiac surgery. That chapter, from 1979, stated that "the anesthesiologist should be experienced in modern cardiac anesthesia skills…able to insert the monitoring devices, interpret the data, utilize new pharmacotherapy, and understand the patient's basic pathophysiology." Certainly this is even more important today, with patients presenting for noncardiac surgery and having drug-eluting stents, multiple antiplatelet drugs, ventricular assist devices, multiple drugs for end-stage heart failure, and implanted electrical devices that produce cardiac resynchronization therapy. These advanced levels of cardiac care, no longer rare, are seen routinely in noncardiac surgery performed in operating rooms, outlying areas of the hospital, and even in outpatient surgical settings. In addition, many patients undergoing vascular or thoracic surgery, some cardiac patients with complicated obstetric problems, and many other noncardiac surgical patients may have significant cardiovascular issues requiring that all anesthesiologists have the skills needed (eg, basic cardiac echocardiography) to diagnose and treat these perioperative problems so as to reduce complications.

The content of the seventh edition ranges from the basic sciences to translational medicine and the latest evidence-based clinical care of the sickest and most complex cardiac patients. To maintain its place as the standard reference textbook in the field, this edition has been completely revised, expanded, and updated to reflect the ongoing changes in cardiovascular care, especially the rapid growth and use of new monitoring techniques, minimally invasive cardiac surgical procedures performed with or without cardiopulmonary bypass, advances in postoperative care, and renewed emphasis on patient safety and reductions in postoperative complications. Significant contributions to the text have been made by leading experts in anesthesiology, cardiology, cardiac surgery, and critical care medicine from around the world. The emphasis throughout the book is on using the latest scientific developments to guide proper therapeutic interventions in the perioperative period. In addition, some chapters include expert guidelines published by leading national and international scientific specialty organizations.

Due to the success of the educational aids used in the previous edition, the Key Points of each chapter appear first in the chapter and Teaching Boxes are highlighted with many of the important take-home messages. The content of the book is enhanced by full-color presentations of the text, multicolor echo and Doppler images, cine clips, and supplementary video material on the ExpertConsult website that accompanies the print version of the text. The website also will be used to update the book on a regular basis as new material appears before the next edition.

In preparing this edition, I have been helped enormously by the four associate editors. They have helped recruit new authors, worked with them on timely production and coordination of their chapters, and expanded the educational opportunities with unique contributions. Dr. Augoustides serves as the liason editor between the book and the *Journal of Cardiothoracic and Vascular Anesthesia,* providing updates on the book's website from new material (eg, new oral anticoagulants) appearing in the journal, as well as selected highlights of the previous year in cardiothoracic and vascular anesthesia. Dr. Maus serves as the editor of the *Transesophageal Echocardiography Video Atlas* (ie, 2D and 3D views, Doppler, and hemodynamic videos) and the Pathologic View Library (eg, perivalvular leaks, aortic dissection, and obstructive cardiomyopathy) found on the book's website. This allows the reader of the book to move seamlessly from the text to the echocardiography video supplementary material. Using the ExpertConsult. Inkling platform, the interested reader can further expand his or her reading on advanced echocardiography by moving to the companion textbook entitled *Perioperative Transesophageal Echocardiography* and edited by Dr. Reich. Finally, Dr. Manecke has coordinated the noncardiac surgery section of the book and introduced the new concepts of goal-directed therapy, enhanced recovery from anesthesia, and the perioperative anesthetic/surgical home for cardiac patients. As we transition from volume-based practices by independent experts to value-based care by a team of experts, these new approaches will allow us as cardiac anesthesiologists to position ourselves as participants in the entire episode of care and the success of the entire team.

Kaplan's Cardiac Anesthesia was written by acknowledged experts in each specific area or related specialties. It is the most authoritative and up-to-date collection of material in the field. Each chapter aims to provide the scientific foundation of the subject matter, the clinical basis for practice, and (when available) outcome information. All of the chapters have been coordinated in an effort to maximize their clinical utility. Whenever possible, material has been integrated from anesthesiology, critical care medicine, cardiology, cardiac surgery, physiology, and pharmacology to present a complete clinical picture. Thus this edition should continue to serve as the definitive text for cardiac anesthesia residents, fellows, faculty, practitioners, cardiologists, cardiac surgeons, intensivists, and others interested in the management of the patient with cardiac disease for either cardiac or noncardiac surgery.

This edition should further facilitate the application of the techniques and procedures that have been learned in the cardiac surgical operating rooms to cardiac patients undergoing noncardiac surgery. These patients compose a much larger group than those undergoing cardiac surgery, and they are often sicker and at higher risk because their underlying cardiac disease is not being corrected by the operative procedure. All of our learning and experience dealing with cardiac surgery should be used to improve the outcomes of these noncardiac surgical patients. It is our overall experience and skill demonstrated while caring for the sickest cardiac patients undergoing new and innovative operations that led J. Willis Hurst, MD, one of the world's leading cardiologists, to state in his Foreword to the first edition of this book that "this cardiologist views the modern cardiac anesthesiologist

with awe." If he thought we were good in 1979, he must be amazed to see our care today guided by the latest monitoring techniques in patients who never would have been operated on in the past.

The editors gratefully acknowledge the contributions made by the authors of each of the chapters. They are the dedicated experts who have made the field of cardiac anesthesia what it is today and are the teachers of our young colleagues practicing anesthesiology around the world. This book would not have been possible without their hard work and expertise.

Joel A. Kaplan, MD, CPE, FACC
Editor

A Textbook for All Anesthesiologists

Patients with cardiac disease undergoing noncardiac surgery should be evaluated … and managed … in a similar manner to patients having cardiac surgery. In order to be able to care for these sick patients … the anesthesiologist and his assistants should be experienced in modern cardiac anesthesia skills. The anesthesiologist must be able to insert monitoring devices … interpret the ECG, utilize the new pharmacotherapy, and understand the patients basic pathophysiology.

Joel A. Kaplan and Ronald W. Dunbar
Cardiac Anesthesia, 1979

The first textbook on anesthesia for surgery of the heart was written by Kenneth K. Keown, who provided anesthesia for Charles Bailey (first successful mitral commisurotomy in 1948), and was published in 1956. This single-author text was 109 pages and had 115 references. Although many different textbooks on this subspecialty have appeared since then, the earliest and now longest and most up to date is this one edited by Joel A. Kaplan, which first appeared in 1979. It is justifiably regarded as the definitive standard reference textbook of cardiac anesthesia.

I first became acquainted with Dr. Kaplan during my second decade as a cardiac surgeon at the University of Washington when I heard him speak at a cardiac surgical meeting extolling and reviewing the role cardiac anesthesiologists can play in improving the outcomes of our patients. Shortly thereafter a team consisting of a cardiac anesthesiologist, a perfusionist, and me were sent by our chairmen to see how the experts in the eastern United States practiced cardiac surgery, and one of those sites was Emory University, where Dr. Kaplan practiced. A few years later as an anesthesia resident and then as a cardiac anesthesia fellow, the first edition of his textbook was a major resource for us, as documented by the profuse highlighting and underlining of text in that book (which still sits in my bookshelf), and then guided our initiation of the cardiac anesthesia program at the University of Kentucky more than 30 years ago.

Cardiac Anesthesia has made many contributions to the practice of anesthesia, cardiovascular medicine, and critical care. The first edition emphasized the importance of a firm understanding of cardiovascular and pulmonary physiology and pharmacology and the interaction of drugs on these two systems. *Cardiac Anesthesia* introduced routine monitoring of electrocardiography (ECG) and invasive monitoring of arterial, central venous, and pulmonary artery pressure, venous saturations, and cardiac output. It identified the discrepancy between central venous pressure and left atrial pressure by introducing the direct measurement of left atrial pressure and the subsequent use of monitoring of pulmonary artery occlusion (wedge) pressure. *Cardiac Anesthesia* also introduced the use of ECG to detect perioperative myocardial ischemia (eg, V_5 lead) and the treatment of it with intravenous nitroglycerin.

Cardiac anesthesiologists have revolutionized the evaluation and perioperative management of cardiac patients undergoing cardiac and noncardiac surgery. They were instrumental in the adoption of monitoring of arterial blood gases, the development of the surgical intensive care unit (ICU), and the incorporation of advanced respiratory and hemodynamic monitoring and management (including aggressive pharmacologic therapy with inotropes and others vasoactive drugs) in the ICU and the noncardiac operating room. Cardiac anesthesiologists collaborated with the American Heart Association/American College of Cardiology in developing various guidelines for management of such patients. They called attention to the frequent occurrence and adverse consequences of perioperative myocardial ischemia during noncardiac surgery. They initiated the use of invasive monitoring—first with the pulmonary artery catheter (PAC) and then with transesophageal echocardiography (TEE)—in the perioperative management of cardiac patients and the participation of anesthesiologists in their management in postsurgical ICUs. They introduced narcotic anesthesia (Lowenstein, Stanley) for these patients and the use of newer pharmacologic agents to treat severe heart failure, hypertension, and arrhythmias. Cardiac anesthesiologists were responsible for introducing TEE to cardiology practice in the United States and since that time have collaborated closely in the application of echocardiography to cardiac care, education, and certification.

Perhaps the greatest contribution has been the recognition very early in the history of cardiac surgery of the importance of a team (surgeon, anesthesiologist, perfusionist, nurse) in the successful management of patients. This concept has now been embedded as an important component of enhanced recovery after surgery and the perioperative surgical/anesthesia home. The Society of Cardiovascular Anesthesiologists has been a leader in improving the functioning of these teams and its safety implications, as discussed in the new chapter on patient safety and avoiding errors. All of this has led to the dramatic improvement of the care of patients with cardiac disease, those undergoing not only cardiac surgery but also noncardiac surgery and nonsurgical care, and likely has contributed to the dramatic improvement in outcomes of these sick patients over the last 40 years.

The first edition of this textbook emphasized the importance of a firm knowledge of the physiology, pharmacology, and pathophysiology of cardiac disease and the importance of management guided by detailed hemodynamic monitoring, including the use of the PAC. Subsequent editions reflected advances in cardiac surgery and anesthesia over the last 4 decades. The second edition (1987) introduced echocardiography, cardiac transplantation, circulatory assistance beyond intraaortic balloon pumping, and central nervous system monitoring and devoted more attention to myocardial protection and postoperative care. The third edition (1993) added chapters on noncardiac surgery in patients with cardiac disease and more on echocardiography (including color flow) and devoted more attention to coagulation and bleeding, central nervous system dysfunction, and the importance of outcome studies after cardiac and noncardiac surgery in patients with heart diseases. The fourth edition (1999) included new chapters on systemic inflammatory response and practice management. The fifth edition (2006) provided chapters that discussed the history of cardiac surgery and anesthesia and predictions of future developments, as well as chapters on molecular cardiovascular medicine, minimally invasive cardiac surgery, and surgical approaches to heart failure, including more advanced ventricular assist devices, pain management, strategies to reduce medical errors, and training in cardiac anesthesia. The sixth edition (2011) subtitled, "The Echo Era," emphasized the maturation of echocardiography and integrated its use into various other chapters throughout that edition.

The present edition advances a theme from the first edition of this text, as reflected in the quote at the start of this forward: the application of the principles of cardiac anesthesia to the management of patients

with cardiac disease outside of the cardiac surgery operating room. Many of its chapters provide essential information, but most relevant are those in the last section, which cover the everyday issues of patients with coronary stents, implantable electronic devices, and implantable ventricular assist devices; the benefits of the use of echocardiography outside the cardiac surgery rooms; and what can be done postoperatively to decrease the incidence of major adverse cardiac events and mortality following noncardiac surgery. I believe that this new edition points out that nearly every site where anesthesiologists practice is potentially a "heart room" and therefore, nearly all anesthesiologists must become cardiac anesthesiologists.

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1 Assessment of Cardiac Risk and the Cardiology Consultation[1]

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KEY POINTS

1. Perioperative cardiac morbidity is multifactorial, and understanding the predictive risk factors helps define the risk for individual patients.
2. Assessment of myocardial injury is based on the integration of information from myocardial imaging (eg, echocardiography), electrocardiography (ECG), and serum biomarkers, with significant variability in the diagnosis depending on the criteria selected.
3. Multivariate modeling has been used to develop risk indices that focus on preoperative variables, intraoperative variables, or both.
4. Key predictors of perioperative risk are dependent on the type of cardiac operation and the outcome of interest.
5. New risk models have become available for valvular heart surgery and for combined coronary and valvular cardiac procedures.
6. For patients undergoing noncardiac surgery, the objectives and approach to cardiac evaluation are entirely different. The nature of the noncardiac surgery and the urgency of the surgical procedure are the primary determinants of the extent of cardiac evaluation in these patients.
7. For most patients undergoing noncardiac surgery, preoperative coronary revascularization is not required unless the patient presents with an unstable cardiac condition or has significant myocardial ischemia and the noncardiac surgery is not urgent.

In the early 1980s, coronary artery bypass graft surgery (CABG) was characterized by operative mortality rates in the range of 1% to 2%. Over the ensuing years, urgent and emergent operations and "redo" procedures became common, and greater morbidity and mortality rates were observed. Percutaneous coronary interventions (PCIs) absorbed low-risk patients from the surgery pool, with the net result that the operative mortality rate increased to the range of 5% to 6%. The trend toward PCI has continued, with trials demonstrating the safety of stenting even in left main coronary artery disease (CAD).[1–3] This demographic shift has led governmental health oversight agencies to ask for justification of the observed increase in CABG mortality, often prompting a time-consuming and expensive chart review to identify the differences in patient populations that led to the greater morbidity. Even with this information, however, it has been difficult to *objectively* determine the impact of these new and compelling factors on mortality. The impetus for the development of a risk-adjusted outcome assessment and appropriate risk adjustment scoring system was the need to compare adult cardiac surgery results in different institutions and to benchmark the observed complication rates.[4] With the recent passage of health care reform legislation and interest in controlling health care expenditures, there is increased interest in public reporting of perioperative outcomes with optimal risk adjustment.

The first risk-scoring scheme for cardiac surgery was introduced by Paiement and colleagues at the Montreal Heart Institute in 1983.[5] Since then, many preoperative cardiac surgery risk indices have been developed. The patient characteristics that affected the probability of specific adverse outcomes were identified and weighed, and the resultant risk indices have been used to adjust for case-mix differences among surgeons and centers where performance profiles have been compiled. In addition to comparisons among centers, the preoperative cardiac risk indices have been used to counsel patients and their families in resource planning, to identify high-risk groups for special care or research, to determine cost-effectiveness, to determine effectiveness of interventions, to improve provider practice, and to assess costs related to severity of disease.[6,7]

In contrast, the objectives of cardiac evaluation in a patient undergoing noncardiac surgery are entirely different. In contrast to patients undergoing cardiac surgery, for whom extensive cardiac evaluation is part of the workup, the cardiac status of patients undergoing noncardiac surgery is often unknown (see Chapter 43). In such patients, the benefit of performing cardiac assessment with its inherent time and resource implications needs to be weighed against the impact such information could have on perioperative planning and the potential risks associated with any delay in the noncardiac surgery. The main goal in this setting is to identify a high-risk group of patients who would benefit from either noninvasive or invasive cardiac evaluation and appropriate perioperative medical management or interventional therapy (see Chapters 2 and 3).

Sources of Perioperative Myocardial Injury in Cardiac Surgery

Myocardial injury, manifested as transient cardiac contractile dysfunction ("stunning") or acute myocardial infarction (AMI) or both, is the most frequent complication after cardiac surgery and the most important cause of hospital complications and death. Furthermore, patients who experience a perioperative myocardial infarction (MI) have a poor long-term prognosis; only 51% of such patients remain free from adverse cardiac events after 2 years, compared with 96% of patients without perioperative MI.[8]

It is important to understand the pathogenesis of this morbidity and mortality to clarify the determinants of perioperative risk. This is particularly important with respect to cardiac outcomes because the definition of *cardiac morbidity* represents a continuum rather than

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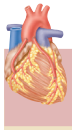


BOX 1.1 DETERMINATIONS OF PERIOPERATIVE MYOCARDIAL INJURY

Disruption of blood flow
Reperfusion of ischemic myocardium
Adverse systemic effects of cardiopulmonary bypass

a discrete event. This understanding can help target the biologically significant risk factors and interventions that may decrease irreversible myocardial necrosis.

Myocardial necrosis is the result of progressive pathologic ischemic changes that start to occur in the myocardium within minutes after interruption of its blood flow (eg, during cardiac surgery) (Box 1.1). The duration of the interruption of blood flow, either partial or complete, determines the extent of myocardial necrosis, and both the duration of the period of aortic cross-clamping (AXC) and the duration of cardiopulmonary bypass (CPB) have consistently been shown to be the main determinants of postoperative outcomes. In a study with an average follow-up of 10 years after complex cardiac surgery, Khuri[9] observed a direct relation between the lowest mean myocardial pH recorded during or after the period of AXC and long-term patient survival. Patients who experienced acidosis (pH <6.5) had decreased survival compared with those who did not. Because myocardial acidosis reflects both myocardial ischemia and poor myocardial protection during CPB, this study demonstrated the relation of the adequacy of intraoperative myocardial protection to long-term outcome (see Chapters 3, 7, 20, and 31).

Reperfusion of Ischemic Myocardium

Surgical interventions requiring interruption of blood flow to the heart must be followed by restoration of perfusion. Numerous experimental studies have provided compelling evidence that reperfusion, although essential for tissue and organ survival, is not without risk because of the potential extension of cell damage as a result of reperfusion itself. Myocardial ischemia of limited duration (<20 min) that is followed by reperfusion leads to functional recovery without evidence of structural injury or biochemical evidence of tissue injury.[10,11] However, reperfusion of cardiac tissue that has been subjected to an extended period of ischemia results in a phenomenon known as *myocardial reperfusion injury.*[12–14] Thus, a paradox exists in that tissue viability can be maintained only if reperfusion is instituted within a reasonable period, but doing so risks extending the injury beyond that caused by the ischemic insult itself. This finding is supported by the observation that ventricular fibrillation was prominent when regionally ischemic canine hearts were subjected to reperfusion.[15] Jennings and associates[16] reported adverse structural and electrophysiologic changes associated with reperfusion of the ischemic canine heart, and Hearse[17] introduced the concept of an oxygen paradox based on cardiac muscle enzyme release and alterations in ultrastructure when isolated hearts were reoxygenated after a period of hypoxic perfusion.

Myocardial reperfusion injury is defined as the death of myocytes, which were alive at the time of reperfusion, as a direct result of one or more events initiated by reperfusion. Myocardial cell damage results from restoration of blood flow to the previously ischemic heart and extends the region of irreversible injury beyond that caused by the ischemic insult alone. The cellular damage that results from reperfusion can be reversible or irreversible, depending on the duration of the ischemic insult. If reperfusion is initiated within 20 minutes after the onset of ischemia, the resulting myocardial injury is reversible and is characterized functionally by depressed myocardial contractility, which eventually recovers completely. Myocardial tissue necrosis is not detectable in the previously ischemic region, although functional impairment of contractility may persist for a variable period, a phenomenon known as *myocardial stunning.* Initiation of reperfusion after longer than 20 minutes, however, results in escalating degrees of irreversible myocardial injury or cellular necrosis. The extent of tissue necrosis that develops during reperfusion is directly related to the duration of the ischemic event. Tissue necrosis originates in the subendocardial region of the ischemic myocardium and extends to the subepicardial region of the area at risk; this is often referred to as the *wavefront phenomenon.* The cell death that occurs during reperfusion can be characterized microscopically by explosive swelling, which includes disruption of the tissue lattice, contraction bands, mitochondrial swelling, and calcium phosphate deposition within mitochondria.[15]

The magnitude of reperfusion injury is directly related to the magnitude of the ischemic injury that precedes it. In its most severe form, it manifests as a "no-reflow" phenomenon. In cardiac surgery, prevention of myocardial injury after release of the AXC, including prevention of no-reflow, is directly dependent on the adequacy of myocardial protection during the period of AXC. The combination of ischemic and reperfusion injury is probably the most frequent and most serious type of injury leading to poor outcomes in cardiac surgery today (see Chapters 2, 3, 7, 13–16, 20, and 31).

Basic science investigations (in mouse, human, and porcine hearts) have implicated acidosis as a primary trigger of apoptosis. Acidosis, reoxygenation, and reperfusion—but not hypoxia (or ischemia) alone—are strong stimuli for programmed cell death, and cardiac apoptosis has been demonstrated to lead to heart failure.[18,19] This suggests that apoptotic changes might be triggered in the course of a cardiac operation, initiating an injurious cascade of adverse clinical events that manifest late in the postoperative course.

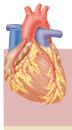
Based on the previous discussion, it is clear that a significant portion of perioperative cardiac morbidity is related primarily to intraoperative factors. However, preoperative risk factors also influence ischemic and reperfusion injury.

Adverse Systemic Effects of Cardiopulmonary Bypass

In addition to the effects of disruption and restoration of myocardial blood flow, cardiac morbidity may result from systemic insults due to CPB circuit-induced contact activation. Inflammation in cardiac surgical patients is produced by complex humoral and cellular interactions, including activation, generation, or expression of thrombin, complement, cytokines, neutrophils, adhesion molecules, mast cells, and multiple inflammatory mediators.[20] Because of the redundancy of the inflammatory cascades, profound amplification occurs to produce multiorgan system dysfunction that can manifest as coagulopathy, respiratory failure, myocardial dysfunction, renal insufficiency, and neurocognitive defects. Coagulation and inflammation also are linked closely through networks of both humoral and cellular components, including tissue factor and proteases of the clotting and fibrinolytic cascades. Vascular endothelial cells mediate inflammation and the cross-talk between coagulation and inflammation. Surgery alone activates specific hemostatic responses, immune mechanisms, and inflammatory responses mediated by the release of various cytokines and chemokines (see Chapters 9, and 31–35). This complex inflammatory reaction can lead to death from nonischemic causes and suggests that preoperative risk factors may not predict morbidity. The ability to risk-adjust populations is critical for the study of interventions that may influence these responses to CPB.

Assessment of Perioperative Myocardial Injury in Cardiac Surgery

The current clinical armamentarium is devoid of a means by which perioperative cardiac injury can be reliably monitored in real time, and this has led to the use of indicators of AMI after the event occurs. There is a lack of consensus regarding how to measure myocardial injury in cardiac surgery because of the continuum of cardiac injury. Electrocardiographic changes, biomarker elevations, and measures



BOX 1.2 ASSESSMENT OF PERIOPERATIVE MYOCARDIAL INJURY

Assessment of cardiac function
- Echocardiography

Nuclear imaging

Electrocardiography
- Q waves
- ST-T wave changes

Serum biomarkers
- Myoglobin
- Creatine kinase
- CK-MB isoenzyme
- Troponin
- Lactate dehydrogenase

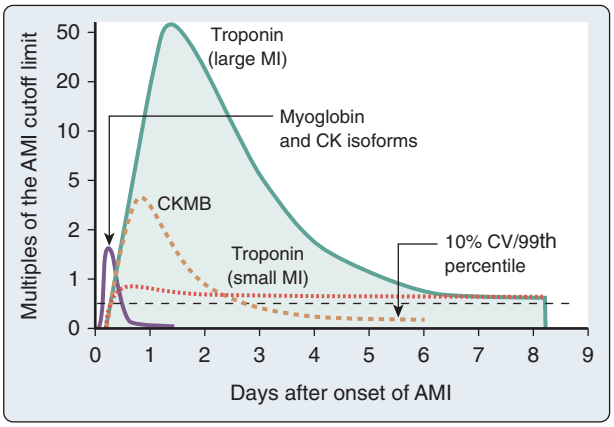


Fig. 1.1 Time course of the appearance of various markers in the blood after acute myocardial infarction *(AMI)*. Shown are the time concentrations/activity curves for myoglobin and creatine kinase *(CK)* isoforms, troponin after large and small infarctions, and CKMB. Note that with cardiac troponin some patients have a second peak in addition. *CKMB,* Creatine kinase, myocardial bound; *CV,* coefficient of variation. *(From Jaffe AS, Babuin L, Apple FS: Biomarkers in acute cardiac disease: the present and the future.* J Am Coll Cardiol. *2006;48[1]:1–11.)*

of cardiac function have all been used (Box 1.2), but all assessment modalities are affected by the direct myocardial trauma of surgery. In 2000, the American College of Cardiology/European Society of Cardiology (ACC/ESC) published a definition of MI that included a characteristic rise and fall in blood concentrations of cardiac troponins or creatine kinase (CK)-MB, or both, in the context of a coronary intervention; other modalities are less sensitive and specific (Fig. 1.1).[21]

The Joint ESC/ACC Foundation (ACCF)/American Heart Association (AHA)/World Heart Federation Task Force published a new Universal Definition of Myocardial Infarction in 2007[22] and revised it in 2012.[23] According to this most recent version of the definition, MI can be diagnosed based on detection of a rise and fall of cardiac biomarkers (preferably troponin) with at least one value above the 99th percentile of the upper reference limit, together with evidence of myocardial ischemia in the form of any of the following: symptoms of ischemia, ECG changes indicative of new ischemia (new ST-T changes or new left bundle branch block), development of pathologic Q waves on ECG, or imaging evidence of new loss of viable myocardium or new regional wall motion abnormality (RWMA). Because CABG itself is associated with cardiac trauma resulting in an increase in the serum levels of cardiac enzymes, an arbitrary cutoff level for elevation of cardiac biomarker values of more than 10 times the 99th percentile of the upper reference limit has been recommended for diagnosing MI during the immediate period after cardiac surgery. However, this threshold is more robust for diagnosing MI after an isolated on-pump CABG; cardiac biomarker release is typically considerably higher after combined valve replacement and CABG and considerably lower after an off-pump CABG.[24]

Assessment of Cardiac Function

Cardiac contractile dysfunction is the most prominent feature of myocardial injury, despite the fact that there are no perfect measures of postoperative cardiac function. The need for inotropic support, low cardiac output (CO) diagnosed with the use of CO measurement technologies, and assessment of abnormal ventricular function by transesophageal echocardiography (TEE) are practical intraoperative options for evaluation of cardiac contractility. Use of inotropic support and CO measurements are not reliable measures, however, because they depend on loading conditions and interpractitioner variability. Failure to wean from CPB, in the absence of systemic factors such as hyperkalemia and acidosis, is the best evidence of intraoperative myocardial injury or cardiac dysfunction, but it also may be multifactorial and therefore is a less robust outcome measure.

Because RWMAs on TEE follow the onset of ischemia within 10 to 15 seconds, echocardiography can be a sensitive and rapid monitor for cardiac ischemia/injury.[25] An irreversible RWMA indicates irreversible myocardial necrosis (see Chapters 12–16). The importance of echocardiographic assessment of cardiac function is further enhanced by its value as a predictor of long-term survival.[26] For patients undergoing CABG, a postoperative decrease in left ventricular (LV) ejection fraction (LVEF) compared with the preoperative baseline predicts decreased long-term survival.[27]

Nevertheless, the use of echocardiography for detecting postoperative LV systolic dysfunction has some challenges. Echocardiographic and Doppler systems have the limitation of being sensitive to alterations in loading conditions, similar need for inotropic support and CO determinations,[28] and the interpretation of TEE images is also operator dependent.[29] Additionally, myocardial stunning (postischemic transient ventricular dysfunction) is a common cause of new postoperative RWMAs, and the resulting wall motion abnormalities and LV systolic dysfunction are often transient. However, the appearance of a new LV RWMA in the postoperative period, whether caused by irreversible AMI or by reversible myocardial stunning, is an indication of some form of inadequate myocardial protection during the intraoperative period and therefore is of interest for assessment of the need for new interventions. At the same time, there are nonischemic causes of RWMAs, such as conduction abnormalities, ventricular pacing, and myocarditis, that confound the use of this measure for assessment of ischemic morbidity.

Electrocardiography Monitoring

The presence of new persistent Q waves of at least 0.03-s duration, broadening of preexisting Q waves, or new QS deflections on the postoperative ECG have been considered evidence of perioperative AMI.[30] However, new Q waves also may be caused by unmasking of an old MI and therefore are not indicative of a new AMI. Crescenzi and colleagues[31] demonstrated that the presence of a new Q wave together with high levels of biomarkers was strongly associated with postoperative cardiac events, whereas the isolated appearance of a new Q wave had no impact on postoperative cardiac outcome. Additionally, new Q waves may actually disappear over time.[32] Signs of non–Q-wave MI, such as ST-T wave changes, are even less reliable signs of AMI after cardiac surgery in the absence of biochemical evidence. ST-segment changes are less specific for perioperative MI because they can also be caused by changes in body position, hypothermia, transient conduction abnormalities, pericarditis, and electrolyte imbalances (see Chapter 12).

Serum Biochemical Markers to Detect Myocardial Injury

Serum biomarkers have become the primary means of assessing the presence and extent of AMI after cardiac surgery. Serum biomarkers that are indicative of myocardial damage include the following (with postinsult peak time given in parentheses): myoglobin (4 h), total CK (16 h), CK-MB isoenzyme (24 h), troponins I and T (24 h), and lactate dehydrogenase (LDH) (76 h). CK-MB isoenzyme has been used most widely, but studies have suggested that troponin I is the most sensitive and most specific marker for depicting myocardial ischemia and infarction.[33–37] Accordingly, cardiac troponin I is the current biomarker of choice for diagnosing myocardial injury.[23]

Numerous studies have demonstrated the value of cardiac biomarkers in predicting short- and long-term outcomes in patients undergoing cardiac surgery. For example, Klatte and coworkers reported on the implications of CK-MB level in 2918 high-risk CABG patients enrolled in a clinical trial of an antiischemic agent.[38] They calculated the postoperative peak CK-MB ratio (ie, the peak CK-MB value divided by the upper limit of normal for the laboratory test) for each patient. The unadjusted 6-month mortality rates were 3.4%, 5.8%, 7.8%, and 20.2% for patients with CK-MB ratios of less than 5, between 5 and 10, between 10 and 20, and greater than 20, respectively.[38] The relation remained statistically significant after adjustment for LVEF, congestive heart failure (CHF), cerebrovascular disease, peripheral vascular disease, cardiac arrhythmias, and the method of cardioplegia delivery.

In the Arterial Revascularization Therapies Study (ARTS), 496 patients with multivessel coronary artery disease undergoing CABG were evaluated by CK-MB testing after surgery and at 30 days and 1 year of follow-up.[39] Patients with increased cardiac enzyme levels after CABG were at increased risk for both death and repeat AMI within the first 30 days. CK-MB increase also was independently related to late adverse outcome. Other studies have similarly documented the prognostic value of cardiac troponin I. Increased cardiac-specific troponin I or T after CABG has been associated with a cardiac cause of death and with major postoperative complications within 2 years after CABG.[40,41]

A few new biomarkers of perioperative cardiac injury or ischemia are under development. Brain natriuretic peptide (BNP) can be detected in the early stages of ischemia and decreases shortly after ischemic insult, allowing better detection of reinjury.[42] BNP concentrations after CABG in patients who experienced cardiac events within 2 years after surgery were significantly greater than those in patients free of cardiac events.[43] Soluble CD40 ligand (sCD40L) is another early biomarker of myocardial ischemia,[44] and CPB causes an increase in the concentration of plasma sCD40L. A corresponding decrease in platelet CD40L suggests that this prothrombotic and proinflammatory protein is derived primarily from platelets and may contribute to the thrombotic and inflammatory complications associated with CPB.[45] Future research will be required to determine how these biomarkers may be used to assess outcome after cardiac surgery.

Variability in Diagnosis of Perioperative Myocardial Infarction

The variability in diagnosis of perioperative AMI was studied by Jain and colleagues,[46] who evaluated data from 566 patients at 20 clinical sites. Twenty-five percent of the patients met Q-wave, CK-MB, or autopsy criteria for AMI. Among them, 19% had increased CK-MB concentrations and ECG changes. Four percent met either Q-wave plus CK-MB or autopsy criteria. Multicenter data collection showed a substantial variation in the incidence of AMI and an overall incidence rate of up to 25%. The determination of perioperative AMI was highly variable depending on the definitions used.

Clinicians are still searching for a gold standard approach to diagnose perioperative AMI. Perioperative myocardial necrosis or injury ranges from mild to severe and can have an ischemic or nonischemic origin in patients undergoing cardiac surgery. Perioperative ECG changes, including Q waves, and new RWMAs on TEE are less reliable than in the nonperioperative arena. As mentioned earlier, troponin I or T is currently the best indicator of myocardial damage after cardiac surgery.

Cardiac Risk Assessment and Cardiac Risk Stratification Models in Patients Undergoing Cardiac Surgery

In defining important risk factors and developing risk indices, each of the studies has used different primary outcomes. Postoperative mortality remains the most definitive outcome that is reflective of patient injury in the perioperative period. Death can be cardiac and noncardiac related, and if cardiac related, it may be ischemic or nonischemic in origin. Postoperative mortality rate is reported as either the in-hospital rate or the 30-day rate. The latter represents a more standardized definition, although it is more difficult to capture because of the difficulty inherent in assessing death rates of discharged patients who may die at home or another facility. Risk-adjusted postoperative mortality models permit assessment of the comparative efficacy of various techniques in preventing myocardial damage, but they do not provide information that is useful in preventing the injury in real time.[47] The postoperative mortality rate also has been used as a comparative measure of quality of cardiac surgical care.[48,49]

Postoperative morbidity includes AMI and reversible events such as CHF and need for inotropic support. The problems of using AMI as an outcome of interest were described earlier. Because resource utilization has become such an important financial consideration for hospitals, the length of stay in the intensive care unit (ICU) increasingly has been used as a factor in the development of risk indices (see Chapters 37 and 38).

Predictors of Perioperative and Postoperative Morbidity and Mortality

Clinical and angiographic predictors of operative mortality were initially defined from the results of the Coronary Artery Surgery Study (CASS).[50,51] A total of 6630 patients underwent isolated CABG between 1975 and 1978. Women had a significantly greater mortality rate than men; mortality increased with advancing age in men, but this was not a significant factor in women. Increasing severity of angina, manifestations of heart failure, and number and extent of coronary artery stenoses all correlated with greater mortality, whereas LVEF was not a predictor. Urgency of surgery was a strong predictor of outcome, and those patients requiring emergency surgery in the presence of a 90% left main coronary artery stenosis sustained a 40% mortality rate.

A risk-scoring scheme for cardiac surgery (CABG and valve) was introduced by Paiement and associates[5] at the Montreal Heart Institute in 1983. Eight risk factors were identified: (1) poor LV function, (2) CHF, (3) unstable angina or recent MI (within 6 wk), (4) age greater than 65 years, (5) severe obesity (body mass index >30 kg/m^2), (6) reoperation, (7) emergency surgery, and (8) other significant or uncontrolled systemic disturbances. The investigators identified three classes of patients: those with none of the listed risk factors (normal), those presenting with one risk factor (increased risk), and those with more than one factor (high risk). In a study of 500 consecutive patients undergoing cardiac surgery, it was found that operative mortality increased with increasing risk score (confirming the scoring system).

zzzOne of the most commonly used scoring systems for CABG was developed by Parsonnet and colleagues[52] (Table 1.1). Fourteen risk factors were identified for in-hospital or 30-day mortality after univariate regression analysis of 3500 consecutive operations. An additive model was constructed and prospectively evaluated in 1332 cardiac procedures. Five categories of risk were identified with increasing mortality rates, complication rates, and length of stay at the Newark Beth Israel Medical Center. The Parsonnet Index frequently is used as a benchmark for comparisons among institutions. However, it was created earlier than the other models and may not be representative

TABLE 1.1 Components of the Additive Model

Risk Factor	*Assigned Weight*
Female sex	1
Morbid obesity (≥1.5 × ideal weight)	3
Diabetes (unspecified type)	3
Hypertension (systolic BP >140 mm Hg)	3
Ejection fraction (%):	
Good >50)	0
Fair (30–49)	2
Poor (<30)	4
Age (y):	
70–74	7
75–79	12
≥80	20
Reoperation	
First	5
Second	10
Preoperative IABP	2
Left ventricular aneurysm	5
Emergency surgery after PTCA or catheterization complications	10
Dialysis dependency (PD or Hemo)	10
Catastrophic states (eg, acute structural defect, cardiogenic shock, acute renal failure)[a]	10–50[b]
Other rare circumstances (eg, paraplegia, pacemaker dependency, congenital HD in adult, severe asthma)[a]	2–10[b]
Valve surgery	
Mitral	5
PA pressure ≥60 mm Hg	8
Aortic	5
Pressure gradient >120 mm Hg	7
CABG at the time of valve surgery	2

[a]On the actual worksheet, these risk factors require justification.
[b]Values were predictive of increased risk for operative mortality in univariate analysis.
BP, Blood pressure; *CABG,* coronary artery bypass graft; *HD,* heart disease; *Hemo,* hemodialysis; *IABP,* intraaortic balloon pump; *PA,* pulmonary artery; *PD,* peritoneal dialysis; *PTCA,* percutaneous transluminal coronary angioplasty.
From Parsonnet V, Dean D, Bernstein A. A method of uniform stratification of risk for evaluating the results of surgery in acquired adult heart disease. *Circulation.* 1989;79:I3, by permission.

TABLE 1.2 Risk Model Results

Variable	*Odds Ratio*
Age (in 10-y increments)	1.640
Female sex	1.157
Race other than white	1.249
Ejection fraction	0.988
Diabetes	1.188
Renal failure	1.533
Serum creatinine (if renal failure is present)	1.080
Dialysis dependence (if renal failure is present)	1.381
Pulmonary hypertension	1.185
Cerebrovascular accident timing	1.198
Chronic obstructive pulmonary disease	1.296
Peripheral vascular disease	1.487
Cerebrovascular disease	1.244
Acute evolving, extending myocardial infarction	1.282
Myocardial infarction timing	1.117
Cardiogenic shock	2.211
Use of diuretics	1.122
Hemodynamic instability	1.747
Triple-vessel disease	1.155
Left main disease >50%	1.119
Preoperative intraaortic balloon pump	1.480
Status	
Urgent or emergent	1.189
Emergent salvage	3.654
First reoperation	2.738
Multiple reoperations	4.282
Arrhythmias	1.099
Body surface area	0.488
Obesity	1.242
New York Heart Association class IV	1.098
Use of steroids	1.214
Congestive heart failure	1.191
PTCA within 6 h of surgery	1.332
Angiographic accident with hemodynamic instability	1.203
Use of digitalis	1.168
Use of intravenous nitrates	1.088

PTCA, Percutaneous transluminal coronary angioplasty.
From Shroyer AL, Plomondon ME, Grover FL, et al: The 1996 coronary artery bypass risk model: the Society of Thoracic Surgeons Adult Cardiac National Database. *Ann Thorac Surg.* 1999;67:1205, by permission of Society of Thoracic Surgeons.

of the current practice of CABG. Since publication of the Parsonnet model, numerous technical advances now in routine use have diminished CABG mortality rates.

Bernstein and Parsonnet[53] simplified the risk-adjusted scoring system in 2000 to provide a handy tool in preoperative discussions with patients and their families and for preoperative risk calculation and stratification. The authors developed a logistic regression model, in which 47 potential risk factors were considered, and a method requiring only simple addition and graphic interpretation was designed for relatively easy approximation of the estimated risk. The final estimates provided by the simplified model correlated well with the observed mortality (Fig. 1.2).

The Society of Thoracic Surgeons (STS) National Adult Cardiac Surgery Database (NCD) represents the most robust source of data for calculating risk-adjusted scoring systems. Established in 1989, the database included 892 participating hospitals in 2008 and has continued to grow. This provider-supported database, one of the largest in the world, allows participants to benchmark their risk-adjusted results against regional and national standards. New patient data are brought into the STS database on a semiannual basis. These new data are analyzed, modeled, and tested using a variety of statistical algorithms.

Since 1990, when more complete data collection was achieved, risk stratification models have been developed for both CABG and valve replacement surgery. Models developed in 1995 and 1996 were shown to have good predictive value[54,55] (Table 1.2 and Fig. 1.3). In 1999, the STS analyzed the database for valve replacement with and without CABG to determine trends in risk stratification. Between 1986 and 1995, 86,580 patients were analyzed. The model evaluated the influence of 51 preoperative variables on operative mortality by univariate and multivariate analyses for the overall population and for each subset. After the significant risk factors were determined by univariate analysis, a standard logistic regression analysis was performed using the training-set population to develop a formal model. The test-set population then was used to determine the validity of the model. The preoperative risk factors associated with greatest operative mortality rates were salvage status, renal failure (dialysis dependent and nondialysis dependent), emergent status, multiple reoperations, and New York Heart Association class IV status. The multivariate logistic regression analysis identified 30 independent preoperative risk factors among the six valvular models that represented isolated valvular surgery or valvular surgery in combination with CABG. The addition of CABG increased the mortality rate significantly for all age groups and for all subset models.[56]

There are currently three general STS risk models: CABG, valve (aortic or mitral), and valve plus CABG. These three models comprise seven specific, precisely defined procedures: the CABG model refers to an isolated CABG; the valve model includes isolated aortic or mitral valve replacement and mitral valve repair; and the valve plus CABG model includes aortic valve replacement with CABG, mitral valve replacement with CABG, and mitral valve repair with CABG. Besides operative mortality, these models were developed for eight additional end points: reoperation, permanent stroke, renal failure, deep sternal wound infection, prolonged (>24 h) ventilation, composite major

CARDIAC SURGERY:
PREOPERATIVE RISK-ESTIMATION WORKSHEET
(not intended for retrospective risk stratification)

Newark Beth Isreal Medical Center
Division of Surgical Research

Patient's Name:
Patient Number:
Date:

INSTRUCTIONS:

Step 1. Fill in the blanks for existing risk factors, using the scores provided. (Note: Scores shown are in arbitrary units, and are not, by themselves, estimates of percent risk.)

Step 2. Add the scores to obtain a total score. (Include common risk factors on this side of the page and less common risk factors on the other side.)

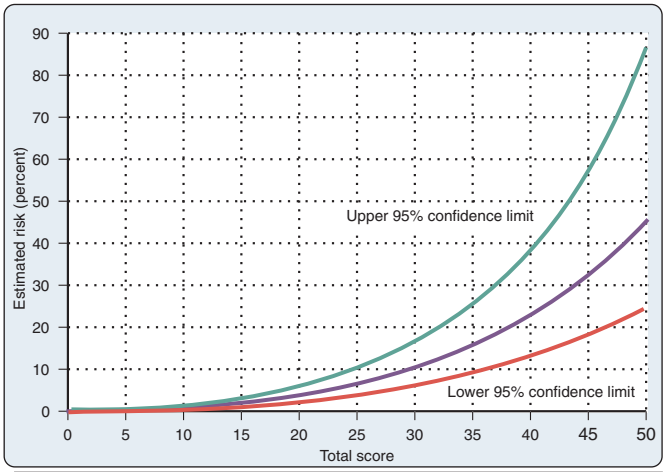
Step 3. See reverse side to interpret the total score.

RISK FACTOR	SCORING (APPROXIMATE SYSTEM 97)		VALUE
Female gender		6	6
Age	70–75 76–79 80+	2.5 7 11	7
Congestive failure		2.5	
COPD, severe		6	
Diabetes		3	
Ejection fraction	30–42% <30%	6.5 8	
Hypertension	Over 140/90, or history of hypertension, or currently taking anti-hypertension medication	3	3
Left-main disease	Left-main stenosis is 50%	2.5	
Morbid obesity	Over 1.5 times ideal weight	1	1
Preoperative IABP	IABP present at time of surgery	4	
Reoperation	First reoperation Second or subsequent reoperation	10 20	
One valve, aortic	Procedure proposed	0	
One valve, mitral	Procedure proposed	4.5	
Valve + ACB	Combination valve procedure and ACB proposed	6	
Special conditions	*(see reverse side)*		
		TOTAL SCORE:	17

(See reverse side for risk estimation.)

RISK VALUES FOR SPECIAL CONDITIONS

Cardiac	
Cardiogenic shock (urinary output <10 cc/hr)	12
Endocarditis, active	5.5
Endocarditis, treated	0
LV aneurysm resected	1.5
One valve, incuspid: procedure proposed	5
Pacemaker dependency	0
Transmural acute MI within 48 hr	4
Ventricular septal defect, acute	12
Ventricular tachycardia, ventricular fibrillation, aborted sudden death	1
Pulmonary	
Asthma	1
Endotracheal tube, preoperative	4
Idiopathic thrombocytopenic purpura	12
Pulmonary hypertension (mean pressure >30)	11
Hepato-renal	
Cirrhosis	12.5
Dialysis dependency	13.5
Renal failure, acute or chronic	3.5
Vascular	
Abdominal aortic aneurysm, asymptomatic	0.5
Cartoid disease (bilateral or 100% unilateral occlusion)	2
Peripheral vascular disease, severe	3.5
Miscellaneous	
Blood products refused	11
Severe neurologic disorder (healed CVA, paraplegia, muscular dystrophy, hemiparesis)	5
PTCA or cathaterization failure	5.5
Substance abuse	4.5



Use the total score to read the estimated preoperative-risk range from this plot, which shows the estimated risk of mortality together with 95% confidence limits.

Fig. 1.2 Preoperative Risk-Estimation Worksheet. *(From Bernstein AD, Parsonnet V. Bedside estimation of risk as an aid for decision-making in cardiac surgery.* Ann Thorac Surg. *2000;69:823, by permission from the Society of Thoracic Surgeons.)*

morbidity or mortality, prolonged length of stay (>14 days), and short length of stay (<6 days and alive).[57–59] These models are updated every few years and are calibrated annually to provide an immediate and accurate tool for regional and national benchmarking, and they have been proposed for public reporting. The calibration of the risk factors is based on the ratio between observed and expected results (O/E ratio), and calibration factors are updated quarterly. The expected mortality (E) is calibrated to obtain a national O/E ratio.

The European System for Cardiac Operative Risk Evaluation (EuroSCORE) is another widely used model for cardiac operative risk evaluation. It was constructed from an analysis of 19,030 patients undergoing a diverse group of cardiac surgical procedures from 128 centers across Europe[60,61] (Tables 1.3 and 1.4). The following risk factors were associated with increased mortality: age, female sex, elevated serum creatinine level, extracardiac arteriopathy, chronic airway disease, severe neurologic dysfunction, previous cardiac surgery, recent MI, reduced LVEF, chronic CHF, pulmonary hypertension, active endocarditis, unstable angina, procedure urgency, critical preoperative condition, ventricular septal rupture, noncoronary surgery, and thoracic aortic surgery. For a given individual, each of these risk factors is assigned a score, and the sum total of these is used to predict surgical risk. In 2003, a more sophisticated, logistic version of EuroSCORE was released to permit more accurate risk assessment in individuals deemed to be at very high risk.[62]

The additive EuroSCORE has been used widely and validated across various centers in Europe and around the world, making it a primary tool for risk stratification in cardiac surgery.[63–74] Although its accuracy has been well established for CABG and isolated valve procedures, its predictive ability in combined CABG and valve procedures has been less well studied. Karthik and associates[65] showed that, in patients undergoing combined procedures, the additive EuroSCORE significantly underpredicted the risk when compared with the observed mortality. The logistic EuroSCORE[62] performed better in this setting.

In 2011, the EuroSCORE was recalibrated to keep up with new evidence. The revised EuroSCORE, known as EuroSCORE II,[75] permits more accurate risk estimation yet preserves the powerful discrimination of the original model. The EuroSCORE II is currently the recommended model for assessment of cardiac surgical risk. It can be accessed online (www.euroscore.org/calc.html) or downloaded as a Smartphone application.

Many other investigators have developed risk assessment models using data representing different populations and different surgical

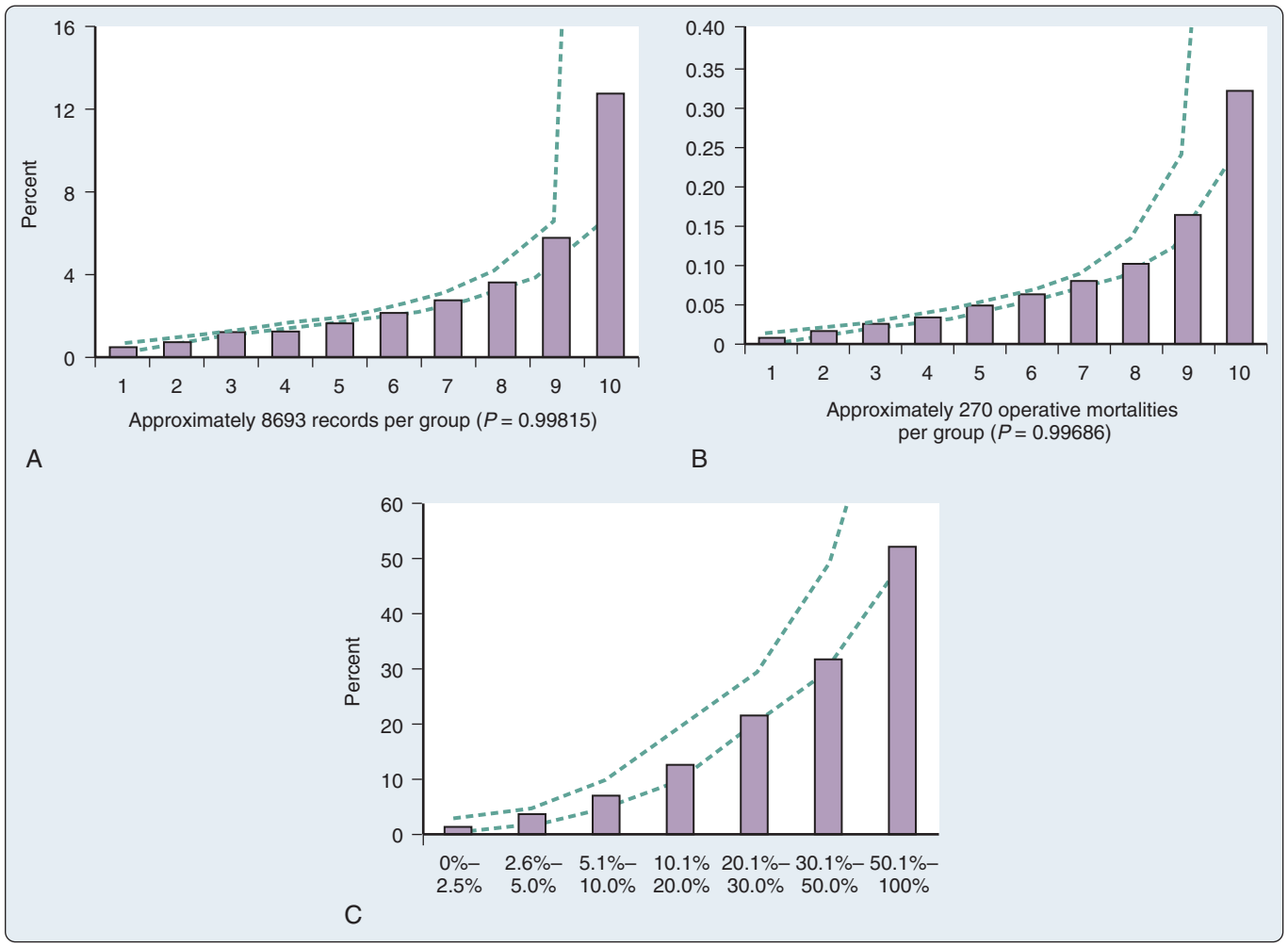


Fig. 1.3 After the predicted risk for each patient in the test set was determined, the patient records were arranged sequentially in order of predicted risk. The population was divided into 10 groups with an equal number of records in each group (A), 10 groups with an equal number of deaths in each group (B), or 7 groups by clinically relevant risk category (C). The predicted mortality rate was compared with the actual mortality for each of the groups. *Dashed lines* represent the range of predicted mortality for a group of patients; *bars* represent actual mortality for that group. *(From Shroyer AL, Plomondon ME, Grover FL, et al. The 1996 coronary artery bypass risk model: the Society of Thoracic Surgeons Adult Cardiac National Database.* Ann Thorac Surg. *1999;67:1205, by permission of the Society of Thoracic Surgeons.)*

practices.[76–84] Hannan and colleagues[82] evaluated predictors of mortality after valve surgery using data from 14,190 patients in New York state. A total of 18 independent risk factors were identified in the six models of differing combinations of valve surgery and CABG. Shock and dialysis-dependent renal failure were among the most significant risk factors in all models. The risk factors and odds ratios are shown in Tables 1.5–1.7. They also studied which risk factors were associated with early readmission (≤30 d) after CABG. Of the 16,325 total patients, 2111 (12.9%) were readmitted within 30 days for reasons related to CABG. Eleven risk factors were found to be independently associated with greater readmission rates: older age, female sex, African American race, greater body surface area, previous AMI within 1 week, and six comorbidities. After controlling for these preoperative patient-level risk factors, two provider characteristics (annual surgeon CABG volume <100 and hospital risk-adjusted mortality rate in the highest decile) and two postoperative factors (discharge to nursing home or rehabilitation/acute care facility and length of stay during index CABG admission ≥5 d) also were related to greater readmission rates.

Dupuis and associates[84] attempted to simplify the approach to evaluating the risk of cardiac surgical procedures in a manner similar to the original American Society of Anesthesiologists (ASA) physical status classification. They developed a score that uses a simple categorization of five classes plus an emergency status (Table 1.8). The Cardiac Anesthesia Risk Evaluation (CARE) score model collected data from 1996 to 1999 and included 3548 patients to predict both in-hospital mortality and a diverse group of major morbidities. It combined clinical judgment and the recognition of three risk factors previously identified by multifactorial risk indices: comorbid conditions categorized as controlled or uncontrolled, the complexity of the surgery, and the urgency of the procedure. The CARE score demonstrated predictive characteristics similar or superior to those of the more complex indices. The development of these several excellent risk models for cardiac valve surgery provides a powerful new tool to improve patient care, select procedures, counsel patients, and compare outcomes (see Chapter 21).

Consistency Among Risk Indices

Many different variables have been found to be associated with the increased risk during cardiac surgery, but only a few have consistently

TABLE 1.3 Risk Factors, Definitions, and Weights (Score)

Risk Factors	*Definition*	*Score*
Patient-Related Factors		
Age	Per 5 y or part thereof over 60 y	1
Sex	Female	1
Chronic pulmonary disease	Long-term use of bronchodilators or steroids for lung disease	1
Extracardiac arteriopathy	One or more of the following: claudication; carotid occlusion or >50% stenosis; previous or planned intervention on the abdominal aorta, limb arteries, or carotids	2
Neurologic dysfunction	Disease severely affecting ambulation or day-to-day functioning	2
Previous cardiac surgery	Requiring opening of the pericardium	3
Serum creatinine	>200 µmol/L before surgery	2
Active endocarditis	Patient still under antibiotic treatment for endocarditis at the time of surgery	3
Critical preoperative state	One or more of the following: ventricular tachycardia or fibrillation or aborted sudden death, preoperative cardiac massage, preoperative ventilation before arrival in the anesthesia room, preoperative inotropic support, intraaortic balloon counterpulsation or preoperative acute renal failure (anuria or oliguria <10 mL/h)	3
Cardiac-Related Factors		
Unstable angina	Rest angina requiring IV nitrates until arrival in the anesthesia room	2
Left ventricular dysfunction	Moderate or LVEF 30–50% Poor or LVEF <30% Recent myocardial infarct (<90 d)	1 3 2
Pulmonary hypertension	Systolic pulmonary artery pressure >60 mm Hg	2
Surgery-Related Factors		
Emergency	Carried out on referral before the beginning of the next working day	2
Other than isolated CABG	Major cardiac procedure other than or in addition to CABG	2
Surgery on thoracic aorta	For disorder of the ascending aorta, arch, or descending aorta	3
Postinfarct septal rupture		4

CABG, Coronary artery bypass graft surgery; *LVEF,* left ventricular ejection fraction.
From Nashef SA, Roques F, Michel P, et al. European system for cardiac operative risk evaluation (EuroSCORE). *Eur J Cardiothorac Surg.* 1999;16:9.

TABLE 1.4 Application of EuroSCORE Scoring System

			95% Confidence Limits for Mortality	
EuroSCORE	*Patients (N)*	*Deaths (N)*	*Observed*	*Expected*
0–2 (low risk)	4529	36 (0.8%)	0.56–1.10	1.27–1.29
3–5 (medium risk)	5977	182 (3.0%)	2.62–3.51	2.90–2.94
≥6 (high risk)	4293	480 (11.2%)	10.25–12.16	10.93–11.54
Total	14,799	698 (4.7%)	4.37–5.06	4.72–4.95

EuroSCORE, European System for Cardiac Operative Risk Evaluation.
From Nashef SA, Roques F, Michel P, et al. European system for cardiac operative risk evaluation (EuroSCORE). *Eur J Cardiothorac Surg.* 1999;16:9, by permission.

BOX 1.3 COMMON VARIABLES ASSOCIATED WITH INCREASED RISK FOR CARDIAC SURGERY

Age
Female sex
Left ventricular function
Body habitus
Reoperation
Type of surgery
Urgency of surgery

BOX 1.4 MEDICAL CONDITIONS ASSOCIATED WITH INCREASED RISK

Renal dysfunction
Diabetes (inconsistent)
Recent acute coronary syndrome

been found to be major risk factors across multiple and very diverse study settings. Age, female sex, LV function, body habitus, reoperation, type of surgery, and urgency of surgery were among the variables consistently present in most of the models (Box 1.3).

Although a variety of investigators have found various comorbid diseases to be significant risk factors, no diseases have been shown to be consistent risk factors, with the possible exception of renal dysfunction and diabetes. These two comorbidities were shown to be important risk factors in a majority of the studies (Box 1.4).

Applicability of Risk Indices to a Given Population

It is critical to appreciate how these indices were created so as to understand how best to apply a given risk index to a specific patient or population. The application of these risk models to a specific population must be done with caution and after careful study. One issue is that the profile of patients undergoing cardiac surgery is constantly changing, and patients who previously would not have been included in the development data set because they were not considered surgical candidates are now undergoing surgery. Therefore, the models require continuous updating and revision. In addition, cardiac surgery itself is changing with the increasing use of off-pump and less invasive procedures, and this may alter the influence of preexisting conditions.

One critical factor in the choice of model for a given practice is to understand the clinical goals used in the original development process. Additionally, despite extensive research and widespread use of risk models in cardiac surgery, there are methodologic problems. The extent of the details in the reports varies greatly. Different conclusions can be reached depending on the risk model used. Processes critical to the development of risk models are shown in Fig. 1.4.

The underlying assumption in the development of any risk index is that specific factors (eg, disease history, physical findings, laboratory data, nature of surgery) cannot be modified with respect to their influence on outcome. For example, the urgency of the planned surgical procedure and the baseline comorbidities cannot be changed. However, the models themselves depend on the appropriate selection

TABLE 1.5 Significant Independent Risk Factors for in-Hospital Mortality: Aortic Valve Surgery With or Without CABG

	Isolated Aortic Valve Replacement (C = 0.809)		Aortic Valvuloplasty or Valve Replacement Plus CABG (C = 0.727)	
Risk Factor	*OR*	*95% CI for OR*	*OR*	*95% CI for OR*
Age ≥55 y	1.06	1.04–1.08	1.04	1.02–1.06
Hemodynamic instability	3.97	1.85–8.51	NS	
Shock	8.68	2.76–27.33	9.09	3.82–21.62
CHF in same admission	2.26	1.54–3.30	NS	
Extensively calcified ascending aorta	1.96	1.22–3.15	1.56	1.16–2.08
Diabetes	2.52	1.67–3.81	NS	
Dialysis-dependent renal failure	5.51	2.58–11.73	3.17	1.70–5.90
Pulmonary artery systolic pressure ≥50 mm Hg	2.35	1.61–3.41	2.28	1.75–2.96
Body surface area	NS		0.28	0.16–0.50
Previous cardiac operation	NS		2.13	1.54–2.96
Renal failure, no dialysis	NS		2.36	1.32–4.21
Aortoiliac disease	NS		1.88	1.26–2.82

C, C statistic; *CABG,* coronary artery bypass graft; *CHF,* congestive heart failure; *CI,* confidence interval; *NS,* not significant; *OR,* odds ratio.
From Hannan EL, Racz MJ, Jones RH, et al. Predictors of mortality for patients undergoing cardiac valve replacements in New York State. *Ann Thorac Surg.* 2000;70:1212, by permission of the Society of Thoracic Surgeons.

TABLE 1.6 Significant Independent Risk Factors for in-Hospital Mortality: Mitral Valve Surgery With or Without CABG

	Isolated Mitral Valve Replacement (C = 0.823)		Mitral Valve Replacement Plus CABG (C = 0.718)	
Risk Factor	*OR*	*95% CI for OR*	*OR*	*95% CI for OR*
Age ≥55 y	1.08	1.06–1.11	1.07	1.05–1.09
Carotid disease	2.98	1.65–5.39	1.81	1.21–2.70
Shock	9.17	4.17–20.16	5.29	3.03–9.22
CHF in same admission	3.03	2.01–4.56	NS	
Dialysis-dependent renal failure	5.07	1.98–12.97	NS	
Endocarditis	4.28	2.49–7.36	NS	
Ejection fraction <30%	NS	1.76	1.23–2.51	
Hemodynamic instability	NS	3.40	2.16–5.36	
Extensively calcified ascending aorta	NA	1.94	1.27–2.96	

C, C statistic; *CABG,* Coronary artery bypass graft surgery; *CHF,* congestive heart failure; *CI,* confidence interval; *NA,* not available; *NS,* not significant; *OR,* odds ratio.
From Hannan EL, Racz MJ, Jones RH, et al. Predictors of mortality for patients undergoing cardiac valve replacements in New York state. *Ann Thorac Surg.* 2000;70:1212, by permission of the Society of Thoracic Surgeons.

TABLE 1.7 Significant Independent Risk Factors for in-Hospital Mortality: Surgery on Multiple Valves With or Without CABG

	Multiple Valvuloplasty or Valve Replacement (C = 0.764)		Multiple Valvuloplasty or Valve Replacement Plus CABG (C = 0.750)	
Risk Factor	*OR*	*95% CI for OR*	*OR*	*95% CI for OR*
Age ≥55 y	1.05	1.03–1.07	1.05	1.10–1.08
Aortoiliac disease	3.55	1.17–10.72	4.63	2.12–10.10
CHF in same admission	2.18	1.44–3.29	NS	
Malignant ventricular arrhythmia	2.62	1.19–5.78	NS	
Extensively calcified ascending aorta	2.13	1.13–4.00	NS	
Diabetes	1.87	1.13–3.10	2.49	1.46–4.24
Renal failure without dialysis	3.55	1.88–6.72	NS	
Dialysis-dependent renal failure	9.37	4.10–21.40	NS	
Female sex	NS	1.95	1.20–3.18	
Hemodynamic instability	NS	3.65	1.50–8.86	
Shock	NS	50.19	6.08–414.44	
Hepatic failure	NS	8.21	1.84–36.66	
Endocarditis	NS	4.70	1.59–13.87	

C, C statistic; *CABG,* Coronary artery bypass graft surgery; *CHF,* congestive heart failure; *CI,* confidence interval; *NA,* not available; *NS,* not significant; *OR,* odds ratio.
From Hannan EL, Racz MJ, Jones RH, et al. Predictors of mortality for patients undergoing cardiac valve replacements in New York state. *Ann Thorac Surg.* 2000;70:1212, by permission of the Society of Thoracic Surgeons.

of baseline variables or risk factors to study, and their prevalence in the population of interest is critical for them to affect outcome. For example, referral patterns to a given institution may result in an absence of certain patient populations, in which case some risk factors may not appear in the model or their influence may be different than in the population on which the model was based. Also, the use of multivariate logistic regression may eliminate biologically important risk factors that are not present in sufficient numbers to achieve statistical significance.

In developing a risk index, it is important to validate the model and to benchmark it against other known means of assessing risks. It is important to determine whether the index predicts morbidity,

TABLE 1.8 Cardiac Anesthesia Risk Evaluation (CARE) Score

1 = Patient with stable cardiac disease and no other medical problem (a noncomplex surgery is undertaken)
2 = Patient with stable cardiac disease and one or more controlled medical problems[a] (a noncomplex surgery is undertaken)
3 = Patient with any uncontrolled medical problem[b] or any patient in whom a complex surgery is undertaken[c]
4 = Patient with any uncontrolled medical problem *and* in whom a complex surgery is undertaken
5 = Patient with chronic or advanced cardiac disease for whom cardiac surgery is undertaken as a last hope to save or improve life
E = Emergency: surgery as soon as diagnosis is made and operating room is available

[a]Examples: controlled hypertension, diabetes mellitus, peripheral vascular disease, chronic obstructive pulmonary disease, controlled systemic diseases, others as judged by clinicians

[b]Examples: unstable angina treated with intravenous heparin or nitroglycerin, preoperative intraaortic balloon pump, heart failure with pulmonary or peripheral edema, uncontrolled hypertension, renal insufficiency (creatinine level >140 µmol/L), debilitating systemic diseases, others as judged by clinicians

[c]Examples: reoperation, combined valve and coronary artery surgery, multiple valve surgery, left ventricular aneurysmectomy, repair of ventricular septal defect after myocardial infarction, coronary artery bypass of diffuse or heavily calcified vessels, others as judged by clinicians

From Dupuis JY, Wang F, Nathan H, et al. The cardiac anesthesia risk evaluation score: a clinically useful predictor of mortality and morbidity after cardiac surgery. *Anesthesiology.* 2001;94:194, by permission.

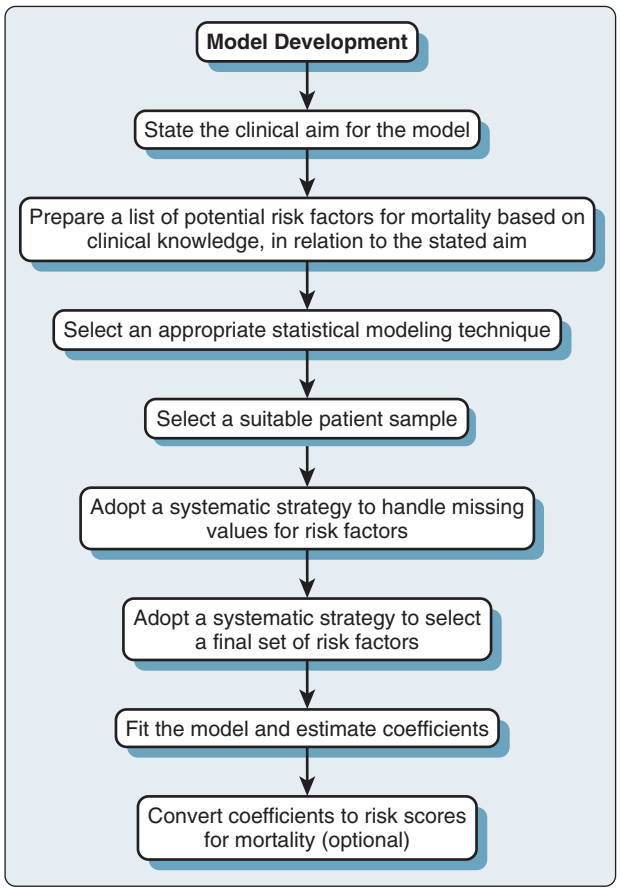


Fig. 1.4 Risk model development. *(From Omar RZ, Ambler G, Royston P, et al. Cardiac surgery risk modeling for mortality: a review of current practice and suggestions for improvement.* Ann Thorac Surg. *2004;77:2232, by permission of the Society of Thoracic Surgeons.)*

mortality, or both. Typically, a model's performance is first evaluated on its goodness of fit to the developmental data (*validation*). Alternatively, the original data can be split and the model can be built on half of the data and validated on the other half. Because this method reduces the total number of patients and outcomes available to create the model, it is best suited to situations in which data on tens of thousands of patients are available. This internal validation does not provide the practitioner with information on the generalizability of the model. External validation on a large, completely independent test data set is the best approach to satisfying this requirement.

Calibration refers to a model's ability to predict mortality accurately. Numerous tests can be applied, the most common being the Hosmer-Lemeshow test. If the *P* value from a Hosmer-Lemeshow test is greater than .05, the current practice of investigators is to claim that the model predicts mortality accurately.

Discrimination is the ability of a model to distinguish patients who die from those who survive. The area under the receiver operating characteristic (ROC) curve is the common method of assessing this facet of the model. In brief, the test is determined by evaluating all possible pairs of patients, determining whether the predicted probability of death should ideally be greater for the patient who died than for the one who survived. The ROC area is the percentage of pairs for which this is true, graphed as the sensitivity versus 1 – the specificity. The current practice in cardiac surgery is to conclude that a model discriminates well if the ROC area is greater than .7. If predictions are used to identify surgical centers or surgeons with unexpectedly high or low rates, achieving a high ROC area alone is not adequate, but good calibration is also critical. A poorly calibrated model may indicate that large numbers of institutions or surgeons have excessively high or low rates of mortality, when in fact the fault lies with the model, not the clinical performance. If predictions are used to stratify patients according to disease severity, to compare treatments, or to decide questions of patient management, both calibration and discrimination are important aspects to consider.

A key problem in the development of cardiac surgery risk stratification models is the evolving practice of surgery. New procedures or variations on older procedures may affect perioperative risk yet not be accounted for in the data used to develop the model. Despite these limitations, the calibrated and validated risk model remains the most objective tool currently available. Clinicians need to understand the specific model they are using, its strengths and weaknesses, to appropriately apply the model in academic research, patient counseling, benchmarking, and management of resources.

Specific Risk Conditions

Renal Dysfunction

Renal dysfunction has been shown to be an important risk factor for surgical mortality in patients undergoing cardiac surgery.[85–87] However, the spectrum of what constitutes renal dysfunction is broad, with some models defining it as increased creatinine levels and others defining it as dialysis dependency.

The Northern New England Cardiovascular Study Group reported a 12.2% in-hospital mortality rate after CABG in patients on chronic dialysis versus a 3.0% mortality rate in patients not on dialysis.[88] However, the incidence of dialysis dependency in the cardiac surgical population is sufficiently low (eg, 0.5% in New York state) that it may not enter into many of the models developed.

Acute kidney injury (AKI) after cardiac surgery carries significant morbidity and mortality. Patients who developed severe renal dysfunction (defined as a glomerular filtration rate [GFR] of <30 mL/min) after CABG had a mortality rate of almost 10%, compared with 1% for patients with normal renal function.[89] Poor outcomes associated with perioperative AKI have led to development of predictive models of AKI to identify patients at risk. One of these models predicts the need for renal replacement therapy (RRT) after cardiac surgery. Wijeysundera and coworkers[90] retrospectively studied a cohort of 20,131 cardiac surgery patients at two hospitals in Ontario, Canada. The multivariate preoperative predictors of RRT were estimated GFR, diabetes mellitus requiring medication, LVEF status (ie, ≤40%), previous cardiac surgery, procedure other than CABG, urgency of surgery (ie, nonelective case), and preoperative use of an intraaortic balloon pump (IABP).

An estimated GFR of 30 mL/min or less was assigned 2 points; a GFR of 31 to 60 mL/min and each of the other components were assigned 1 point each. Among the 53% of patients with low risk scores (≤1 point), the risk for RRT was 0.4%; among the 6% of patients with high-risk scores (≥4 points), it was 10%. Another group developed a robust prediction rule to assist clinicians in identifying patients with normal or near-normal preoperative renal function who are at high risk for development of severe renal insufficiency.[91] In a multivariate model, the preoperative patient characteristics most strongly associated with postoperative severe renal insufficiency included age, sex, white blood cell count greater than 12,000/μL, prior CABG, CHF, peripheral vascular disease, diabetes, hypertension, and preoperative IABP.

A major issue with respect to the development of indices to predict perioperative renal failure is that the pathophysiology of perioperative AKI includes inflammatory, nephrotoxic, and hemodynamic insults. This multifactorial nature of AKI might be one of the reasons that a limited single-strategy approach has not been successful.[92] Contrast agents used for angiography before cardiac surgery represent one of the modifiable nephrotoxic factors. Delaying cardiac surgery beyond 24 hours after the exposure and minimizing the contrast agent load can decrease the incidence of AKI in elective cardiac surgery cases.[93]

Uniformity of AKI definition, achieved by using the RIFLE criteria (risk of renal dysfunction, injury to the kidney, failure of kidney function, loss of kidney function, and end-stage kidney disease), has improved risk stratification models, and use of early biomarkers of AKI should provide additional tools to design clinical trials addressing this important issue.[94,95]

Diabetes

The association between diabetes mellitus and mortality in cardiac surgery has been inconsistent; some studies support the association, but others do not.[96–103] Several randomized trials have evaluated outcome with CABG versus PCI in patients with diabetes. In the Coronary Artery Revascularization in Diabetes (CARDia) trial,[104] a total of 510 patients who had diabetes with multivessel or complex single-vessel CAD from 24 centers were randomized to PCI plus stenting (and routine abciximab) or CABG. After 1 year, the composite rate of death, MI, and stroke was 10.5% in the CABG group and 13.0% in the PCI group (hazard ratio [HR], 1.25; 95% confidence interval [CI], 0.75 to 2.09; $P = .39$); the all-cause mortality rates were 3.2% and 3.2%, respectively; and the rates of death, MI, stroke, or repeat revascularization were 11.3% and 19.3% (HR, 1.77; 95% CI, 1.11 to 2.82; $P = .02$), respectively.

In the Bypass Angioplasty Revascularization Investigation 2 Diabetes (BARI 2D) trial, 2368 patients with both type 2 diabetes and heart disease were randomly assigned to undergo either prompt revascularization along with intensive medical therapy or intensive medical therapy alone, and to undergo either insulin-sensitization or insulin-provision therapy. After a mean follow-up interval of 5.3 years, all-cause mortality, cardiac mortality, MI, and other end points were assessed.[105] The mortality rates did not differ between prompt revascularization versus intensive medical therapy alone or between the two insulin strategies. However, patients in the CABG stratum had significantly fewer MI events with prompt revascularization, and their composite end point of MI plus cardiac death was significantly reduced with insulin-sensitization therapy. The researchers concluded that for patients similar to those in the PCI group, intensive medical therapy alone was an excellent first-line strategy, but for patients with more extensive CAD, similar to those enrolled in the CABG stratum, prompt CABG (in the absence of contraindications), intensive medical therapy, and insulin-sensitization therapy appeared to be a preferred strategy to reduce the incidence of MI.[106]

More recent studies have directly compared CABG with PCI in diabetic patients with multivessel CAD. The Future Revascularization Evaluation in Patients with Diabetes Mellitus: Optimal Management of Multivessel Disease (FREEDOM) trial randomized 1900 diabetic subjects to either PCI or CABG. After 5 years of follow-up, CABG was shown to result in lower rates of death and MI, although the stroke rate was higher in the CABG group.[107] A metaanalysis including eight studies comparing CABG with PCI in diabetic subjects reaffirmed the superiority of CABG in these patients.[108]

Acute Coronary Syndrome

Patients with a recent episode of any non–ST-segment elevation acute coronary syndrome before CABG have greater rates of operative morbidity and mortality than do patients with stable coronary syndromes.[109] However, the American College of Cardiology Foundation, in collaboration with numerous other societies, has published appropriate use criteria for coronary revascularization.[110] Because there are numerous class A recommendations for revascularization, many patients come to the operating room directly after coronary angiography or after attempted stent placement with antiplatelet agents. There is evidence to suggest that it is beneficial to delay CABG for 3 to 7 days in selected stable patients with contraindications to PCI who have experienced ST-elevation myocardial infarction (STEMI) or non–ST-elevation myocardial infarction (NSTEMI).[111] In addition, patients with a hemodynamically significant right ventricular MI should be allowed a period of time for the injured ventricle to recover.[112]

Assessment of Cardiac Risk in Patients Undergoing Noncardiac Surgery

The approach to preoperative cardiac risk assessment and stratification in patients undergoing noncardiac surgery is distinct from that in patients undergoing cardiac surgery. In the latter group, extensive cardiac evaluation is part of the routine preoperative workup, and the patient undergoes corrective therapy for the underlying disease. In contrast, although the presence of a cardiac illness is an added, and often powerful, risk factor for perioperative morbidity and mortality in patients undergoing a noncardiac surgery, the underlying cardiac status in most cases is either inadequately known or not known at all. This necessitates some form of cardiac evaluation in these patients. However, several factors need to be taken into consideration, including overall functional status of the patient, the surgical risk entailed by the proposed noncardiac surgery, the urgency of the noncardiac surgery and the potential risk associated with any delay, the availability of necessary resources for cardiac evaluation, and the impact that the information derived from cardiac evaluation is likely to have on perioperative planning and management (see Chapter 43).

Stepwise Approach to Cardiac Risk Assessment in Patients Undergoing Noncardiac Surgery

Both the ACC/AHA and the ESC recommend a stepwise approach to cardiac risk assessment in patients undergoing a noncardiac surgery (Fig. 1.5).[113,114] Their approaches are based on similar principles and are almost identical.

Step 1: Determine the Urgency of the Noncardiac Surgery

If the noncardiac surgery is emergent (ie, one in which life or limb will be threatened if the surgery is not performed promptly, typically within 6 h), no further cardiac evaluation is warranted in most instances. The clinical risk factors should be assessed, and knowledge about preexisting cardiac illness, if available, should be incorporated to help determine the surgical strategy and optimize perioperative monitoring and management.

Step 2: Identify Any Unstable Cardiac Condition

If the surgery is not emergent, then it should be determined whether the patient has any unstable cardiac condition (Box 1.5). If the patient is diagnosed as having an unstable cardiac condition, a multidisciplinary approach is required, including the patient, the family, and all stakeholders in the patient's caregiver team. A decision needs to be reached about how to stabilize the underlying cardiac condition,

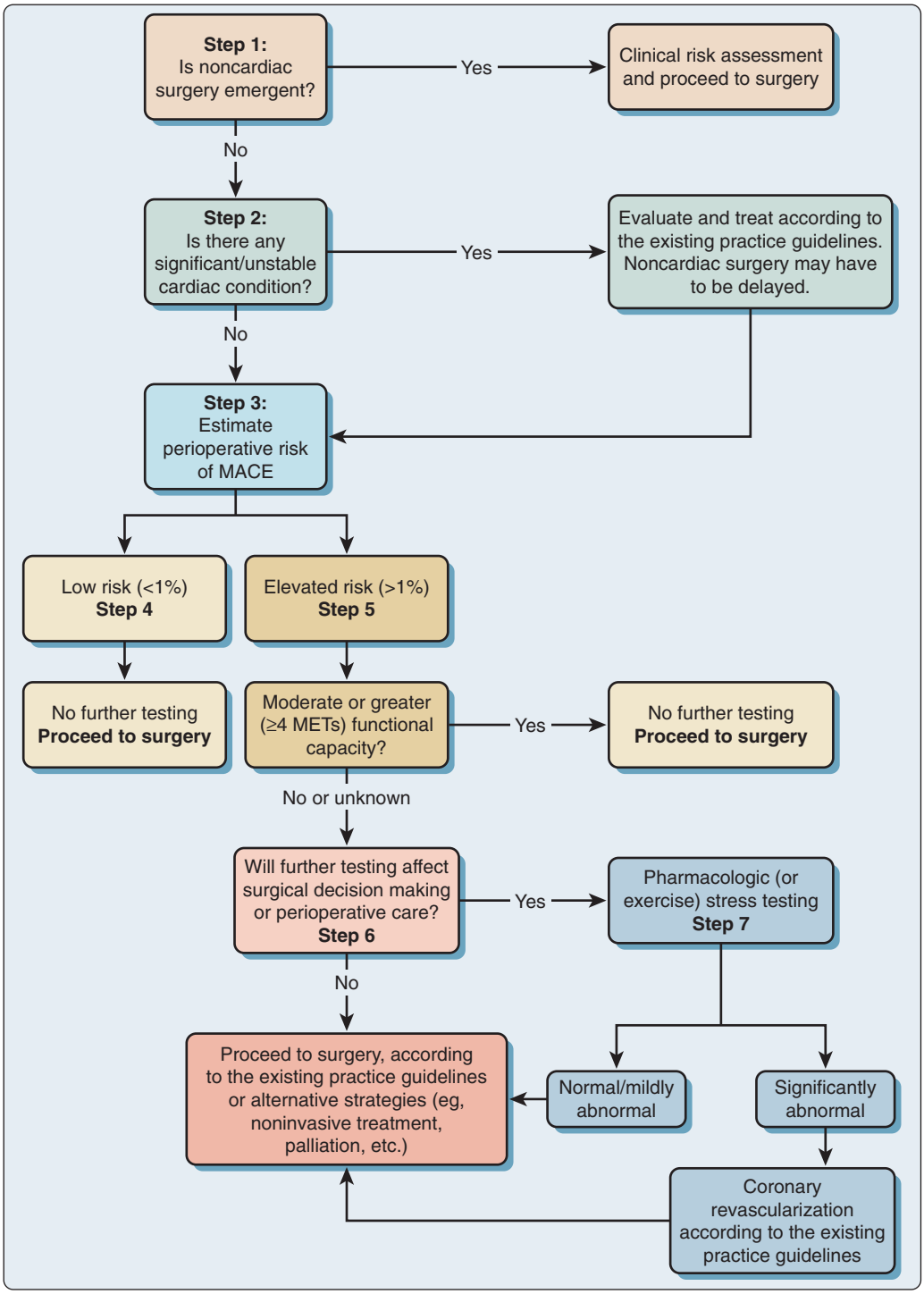
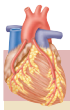


Fig. 1.5 Stepwise approach to perioperative cardiac risk assessment in patients undergoing noncardiac surgery. *MACE*, Major adverse cardiovascular event; *MET*, metabolic equivalent. *(Adapted from Fleisher LA, Fleischmann KE, Auerbach AD, et al. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on practice guidelines.* J Am Coll Cardiol. *2014;64:e77–e137; Kristensen SD, Knuuti J, Saraste A, et al. 2014 ESC/ESA guidelines on non-cardiac surgery: cardiovascular assessment and management. The Joint Task Force on Non-cardiac Surgery: Cardiovascular Assessment and Management of the European Society of Cardiology (ESC) and the European Society of Anaesthesiology (ESA).* Eur Heart J. *2014;35:2383–2431.)*



BOX 1.5 UNSTABLE CARDIAC CONDITIONS

Acute coronary event
Recent myocardial infarction with residual myocardial ischemia
Acute heart failure
Significant cardiac arrhythmias
Symptomatic valvular heart disease

keeping in mind the priority of cardiac versus noncardiac interventions, the risks involved in either approach, and the impact of cardiac treatment (eg, use of antiplatelet or anticoagulant agents) on the subsequent noncardiac surgery. Depending on the outcome of this discussion, patients may either proceed for cardiac intervention if the index surgical procedure can be delayed or proceed directly to the noncardiac surgery with optimal medical therapy if the delay is incompatible.

Step 3: Assess Cardiac Risk of the Noncardiac Surgery

In patients who do not have an unstable heart disease, the next step is to assess the cardiac risk involved in the proposed noncardiac surgery. There are a number of methods to derive this risk estimate, but the most practical approach is to use risk scores or algorithms. The most validated risk indices for this purpose are the Revised Cardiac Risk Index (RCRI)[115] and the American College of Surgeons National Surgical Quality Improvement Program (NSQIP) surgical risk calculator (http://www.surgicalriskcalculator.com)[116] (Table 1.9). The RCRI considers six major risk factors, each of which is assigned a score of 1. Patients who have a score of 0 or 1 are expected to have a low risk of major adverse cardiac events (MACE), whereas those with a score of 2 or higher are likely to have an elevated risk. Although the RCRI is simple to use and has been validated in populations outside the original cohort, it does not provide surgery-specific cardiac risk estimates, and it also tends to underestimate risk in patients undergoing major vascular surgery.[117] The NSQIP surgical risk calculator, which is based on the data derived from more than 1 million surgeries, provides more comprehensive assessment of the surgery-specific risk of a number of clinical outcomes, including MACE and death, and is the preferred approach for this purpose.[116] However, it is more complex to use, requires a Web-based calculator, and has not been validated in populations outside the original cohort.

Based on the anticipated cardiac risk, the surgical interventions can be broadly divided into three groups based on the estimated 30-day rate of cardiac events (ie, cardiac death and MI): low risk (<1%), intermediate risk (1–5%), and high risk (>5%).

Step 4: If the patient Has a Low Cardiac Risk

If the patient is estimated to have a low perioperative risk of an adverse cardiac event, no further cardiac evaluation is required, and the patient may proceed with the planned surgery. However, appropriate guideline-recommended risk-reduction pharmacotherapies should be instituted depending on the clinical condition of the patient.[113,114] Initiation of a beta-blocker regimen may be considered before surgery in patients with known CAD or myocardial ischemia. Treatment should ideally be initiated at least 2 days (and up to 30 days) before surgery, starting with a low dose and gradually titrating the dose to achieve a resting heart rate between 60 and 70 bpm with a systolic blood pressure greater than100 mm Hg. In most patients with atherosclerotic vascular disease, particularly those undergoing vascular surgery, initiation of statin therapy should be strongly considered. In addition, in patients who have heart failure due to LV systolic dysfunction, angiotensin-converting enzyme inhibitors or angiotensin receptor blockers should also be considered before surgery. However, care should be taken to avoid hypotension during the perioperative period. A decision also needs to be reached about discontinuation of antiplatelet and/or anticoagulant agents in those who are already receiving these therapies, and alternative therapies, if any, should be recommended as appropriate.

TABLE 1.9 Risk Factors in Commonly Used Models for Assessment of Cardiac Risk in Patients Undergoing Noncardiac Surgery

Risk Factor	*Revised Cardiac Risk Index*	*American College of Surgeons NSQIP Surgical Risk Calculator*
Ischemic heart disease	Yes	Yes (previous cardiac event)
Cerebrovascular disease	Yes (history of cerebrovascular accident or transient ischemic attack)	No
Type of surgery	Yes (intrathoracic, intraabdominal, or suprainguinal vascular surgery)	Yes (uses Current Procedural Terminology codes)
Renal dysfunction	Yes (creatinine ≥2 mg/dL)	Yes (acute renal failure, need for dialysis)
Diabetes mellitus	Yes (insulin-dependent diabetes mellitus)	Yes
Heart failure	Yes	Yes
Age	No	Yes
Sex	No	Yes
Hypertension	No	Yes
Body mass index	No	Yes
Dyspnea	No	Yes
Smoker	No	Yes
COPD	No	Yes
Functional status	No	Yes
Physical status		Yes (uses ASA Physical Status classification system)
Wound class	No	Yes
Ascites	No	Yes
Systemic sepsis	No	Yes
Ventilator dependency	No	Yes
Disseminated cancer	No	Yes
Steroid use	No	Yes
Emergency surgery	No	Yes

ASA, American Society of Anesthesiologists; *COPD,* chronic obstructive pulmonary disease; *NSQIP,* National Surgical Quality Improvement Program.

Step 5: If the Patient Has an Elevated Cardiac Risk

In patients who are estimated to have an elevated risk of MACE, an assessment of functional capacity is required. Functional capacity is usually expressed in terms of metabolic equivalents (METs) and classified as excellent (>10 METs), good (7–10 METs), moderate (4–6 METs), poor (<4 METs), or unknown.

If the patient has not had a recent exercise test, functional status can usually be estimated from activities of daily living (Fig. 1.6) or, more formally, by using an activity scale such as the Duke Activity Status Index[118] or the Specific Activity Scale.[119] Examples of activities associated with greater than 4 METs include climbing two flights of stairs, walking up a hill, running a short distance, walking on level ground at 4 mph, and performing heavy work around the house. If the patient is able to easily perform 4 METs or more physical activity, he or she can proceed to the planned noncardiac surgery without the need for any further cardiac evaluation.

Step 6: If the Patient Has Poor or Unknown Functional Capacity

In patients with elevated cardiac risk whose functional capacity is poor or unknown, one must determine whether further testing will affect patient decision making (eg, a decision to perform the original surgery or willingness to undergo a cardiac intervention might change depending on the results of the test) or perioperative care. If it will, stress

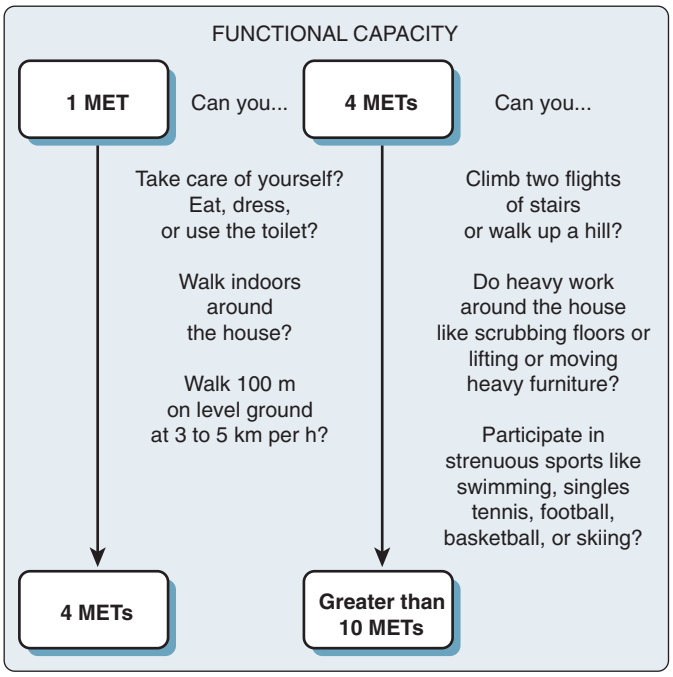


Fig. 1.6 Estimated energy requirements for various activities during daily life. *km per h*, kilometers per hour; *MET*, metabolic equivalent. *(Data from Hlatky MA, Boineau RE, Higginbotham MB, et al. A brief self-administered questionnaire to determine functional capacity (the Duke Activity Status Index).* Am J Cardiol. *1989;64:651–654. and Fletcher GF, Balady GJ, Amsterdam EA, et al. Exercise standards for testing and training: a statement for healthcare professionals from the American Heart Association.* Circulation. *2001;104:1694–1740.)*

testing should be performed, and if the stress test result is normal, the patient can proceed to noncardiac surgery according to the guideline-directed medical therapy (GDMT). However, if the stress test result is abnormal, then depending on the extent of the abnormality, coronary angiography and revascularization should be considered. The patient can then proceed to surgery with GDMT or explore alternative, less invasive or noninvasive therapeutic options for the underlying noncardiac illness.

Although there are no randomized trials on the use of preoperative stress testing, a large number of single-site studies and their metaanalyses have demonstrated its clinical utility in the preoperative evaluation of patients undergoing noncardiac surgery.[120–124] These studies have shown that a normal stress test result has a very high negative predictive value for perioperative cardiac events, whereas the presence of moderate-to-large areas of myocardial ischemia is associated with increased risk of perioperative MI and/or death. In contrast, the presence of RWMAs (unless extensive) on rest imaging has little predictive value.

Most data on the impact of inducible myocardial ischemia on perioperative outcomes are based on pharmacologic stress testing.[120–124] Therefore, it remains the modality of choice in these patients, more so because many are not able to perform adequate exercise. However, in patients who are able to exercise adequately, it seems reasonable that exercise stress testing, when combined with echocardiography or myocardial perfusion scintigraphy, would perform similarly to pharmacologic stress testing.[125–127] Exercise-induced electrocardiographic changes alone are not as predictive and cannot be relied on.[125–128]

There are no randomized trials comparing different imaging modalities (eg, echocardiography vs myocardial perfusion imaging) for predictive accuracy as part of preoperative pharmacologic stress testing. However, a retrospective metaanalysis comparing thallium imaging with stress echocardiography in patients scheduled for elective noncardiac surgery showed that a moderate-to-large defect (present in 14% of the population) detected by either method predicted postoperative cardiac events, with stress echocardiography being slightly superior.[120] Given the lack of adequate evidence, it is recommended that the choice of imaging modality should be guided by the available local expertise in performing pharmacologic stress testing.

Step 7: If Testing Is Unlikely to Affect Decision Making or Care

If a patient with elevated cardiac risk has poor or unknown functional capacity but stress testing is unlikely to affect decision making or care, it is reasonable to proceed to surgery according to the GDMT or to consider alternative, less invasive or noninvasive therapeutic options for the underlying noncardiac illness.

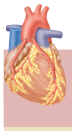
Role of Handheld and Pocket Ultrasound

As outlined earlier, the current guidelines do not recommend routine cardiac imaging in most patients undergoing noncardiac surgery if the surgery is emergent or if the patient is physically active and does not have any overt manifestations of a cardiac illness. These recommendations are based on the assumptions that clinical evaluation is generally sufficient to identify most of the clinically relevant cardiac conditions, that the necessary resources required for cardiac imaging may not be readily available, and that in most instances recognition of a cardiac lesion during preoperative testing would not lead to a cardiac intervention. However, each of these assumptions has an important caveat.

Although a good physical examination remains an integral component of any form of medical assessment and cannot be substituted because of the wealth of information it provides, it has its limitations. It is subjective; it depends heavily on the clinical skills of the examining physician; and even with the most experienced clinicians, it has a diagnostic accuracy that is, at best, suboptimal.[129] There are several cardiac conditions that may be inherently silent (eg, asymptomatic LV systolic dysfunction) and cannot be recognized based on clinical evaluation alone. Further, accurate assessment of lesion severity on clinical examination, even for valvular lesions, remains challenging, especially when the patient has a combination of cardiac lesions. Handheld and pocket ultrasound devices offer an attractive option in these circumstances. Numerous studies have demonstrated that the addition of a screening, bedside echocardiographic examination to clinical assessment significantly increases diagnostic accuracy, reduces unwarranted diagnostic and treatment referrals, facilitates optimal use of health care resources, and is cost-effective[129–133] (see Chapter 46).

The current generation of pocket ultrasound devices offer two-dimensional and color Doppler imaging but lack spectral Doppler. Still, the image quality is usually excellent, which allows a diagnostic accuracy comparable to that achieved with full-scale echocardiography equipment.[131,134–136] At the same time, limited functionalities available on these devices serve to simply their use by physicians. Studies have shown that physicians can quickly be trained in the use of pocket ultrasound devices for rapid screening of patients to recognize major cardiac abnormalities.[137] When required, bedside imaging can also be combined with remote, Web-based interpretation to enhance the diagnostic accuracy of the screening echocardiographic examination and to ensure quality control.[137,138]

Although it is true that a cardiac lesion recognized at the time of preoperative evaluation in patients scheduled to undergo a noncardiac surgery does not necessarily require immediate cardiac intervention, studies have demonstrated that the presence of unrecognized LV systolic dysfunction or valvular heart disease is associated with worse outcomes.[115,137–141] For example, Flu and colleagues studied the value of preoperative echocardiography in 1005 consecutive patients undergoing elective vascular surgery at a single center.[139] LV dysfunction (LVEF <50%) was present in 50% of the patients, 80% of whom were asymptomatic.[58] The 30-day cardiovascular event rate was highest in patients with symptomatic HF (49%), followed by those with asymptomatic systolic LV dysfunction (23%), asymptomatic diastolic LV dysfunction (18%), and normal LV function (10%).



BOX 1.6 HANDHELD OR POCKET ULTRASOUND FOR CARDIAC EVALUATION

Enhances diagnostic accuracy of clinical examination
Can be combined with remote interpretation of acquired images to ensure optimum diagnostic accuracy
Allows recognition of major cardiac lesions
Provides information that may have incremental value in optimizing perioperative outcomes of patients undergoing a noncardiac surgery
Is simple to use and easy to incorporate into the preoperative evaluation
Has been shown to be cost-effective compared with physical examination alone

Bedside echocardiographic examination using a handheld or pocket ultrasound device can have great incremental value in these circumstances (Box 1.6). It permits easy and prompt recognition of significant cardiac lesions, and such information provides the operating team an opportunity to incorporate measures to optimize perioperative outcomes. For example, a less invasive surgical approach can be adopted, regional anesthesia can be used preferentially in certain circumstances, greater caution can be exercised with regard to perioperative fluid and hemodynamic monitoring and management, appropriate cardiac pharmacotherapies can be instituted if circumstances allow; and, importantly, the patient and family can be alerted about the possibility of a cardiac event during the perioperative period. In a recent study of patients undergoing cataract surgery in a community setting, pocket echocardiography allowed recognition of a major cardiac lesion in 14.2% of those patients considered to be free of any major cardiac illness based on clinical examination alone. Of these, roughly one-fourth (3.3% of the entire study population) had cardiac lesions deemed prohibitive for cataract surgery in an unmonitored setting. As a result, these surgeries were rescheduled to be performed later in a hospital under more intensive monitoring.[137] Although this study does not imply that every patient undergoing noncardiac surgery needs to have an echocardiographic examination, it does show that the availability of handheld and pocket ultrasound devices permits easy incorporation of an echocardiographic examination in the preoperative evaluation and may help optimize cardiac outcomes.

Conclusions

Preoperative cardiac risk assessment and stratification of patients undergoing cardiac or noncardiac surgery are pivotal in optimizing perioperative outcomes. In cardiac surgery patients, the main goal of cardiac risk assessment, from the anesthesiologist's perspective, is to provide risk-adjusted mortality rates for preoperative counseling of the patient and family and to identify the group at high risk for a perioperative cardiac event. Based on individual risk factors, perioperative care can be modified to improve the patient's outcome. Various complex or simplified risk-adjusted morbidity and mortality models can serve as tools for facilitating perioperative risk assessment. However, even a well-calibrated model with good discrimination has to be used with caution when applied to individual counseling. First, it is difficult for any model to predict morbidity or mortality, which occur with a low incidence. Second, it has to be clear that the scoring system provides only the probability of death or a major complication and that the individual patient and family members may have difficulty understanding how an adverse outcome could have occurred if the predicted incidence was low in their understanding preoperatively.

In contrast, the underlying cardiac status of noncardiac surgical patients is often unknown or inadequately known. The main goal in this setting is to define an optimal cardiac evaluation strategy considering various patient-related and surgery-related factors and, based on the findings of such evaluation, to provide recommendations for optimal perioperative management to achieve the best possible outcomes. In most such patients, noninvasive cardiac evaluation with optimization of cardiac medications and care to minimize hemodynamic perturbations during the perioperative period are all that is required to ensure cardiac safety. Invasive cardiac evaluation and coronary revascularization usually are not required unless the patient presents with an unstable cardiac condition or has significant myocardial ischemia and the noncardiac surgery is not urgent.

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2 Cardiovascular Imaging

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KEY POINTS

1. Echocardiography is the most widely used modality for cardiac imaging in almost any form of cardiac disease.
2. Echocardiography is noninvasive, safe, readily available, and portable, and it has the ability to provide vast amounts of information about cardiac structure and function. Additionally, echocardiography is the only modality available for imaging during the intraoperative period.
3. A combination of transthoracic and transesophageal imaging permits comprehensive evaluation of most cardiac pathologies. The advent of three-dimensional transesophageal echocardiography has further enhanced the utility of this modality, especially in the evaluation of mitral valve disease.
4. Stress echocardiography is helpful in the assessment of inducible myocardial ischemia, myocardial viability, and certain valve disorders.
5. Myocardial perfusion imaging can be performed using SPECT or PET and is useful in the evaluation of myocardial ischemia and viability.
6. Cardiac computed tomography and cardiac magnetic resonance are increasingly used when there are conflicting results or when further information is required in the preoperative phase of care.
7. Cardiac magnetic resonance is the gold standard for quantitative assessment of ventricular volumes, ejection fraction, and mass. It is also able to evaluate ventricular and valvular function, atherosclerosis, and plaque composition.
8. Computed tomographic aortography is the best modality for the evaluation of aortic aneurysms and dissections. Additionally, computed tomographic coronary angiography offers an alternative to invasive coronary angiography for excluding significant coronary artery disease in patients undergoing noncoronary surgery.

Imaging is fundamental to perioperative evaluation and management of patients undergoing cardiac surgery. Even for those presenting with a noncardiac illness, cardiac imaging is often required to supplement the preoperative evaluation.

For many years, cardiac catheterization and nuclear imaging were the only modalities available for clinical use. The introduction of echocardiography in the early 1970s heralded a revolution in the field of cardiovascular imaging, and echocardiography soon surpassed all other modalities to become the cornerstone of cardiac imaging. Because of its noninvasive nature, safety, easy availability, portability, repeatability, and capacity to provide vast amounts of clinically relevant information, echocardiography has remained the most useful modality for cardiac imaging.

The last several decades have witnessed yet another explosion in imaging techniques with the evolution of cardiac computed tomography (CCT), cardiac magnetic resonance (CMR), and positron emission tomography (PET) as routine clinical evaluation tools. In the field of perioperative evaluation, these alternative imaging modalities have complemented more than supplemented traditional imaging. This chapter provides a brief overview of the roles of these different imaging modalities in the evaluation and management of various cardiac conditions.

Echocardiography

Transthoracic echocardiography (TTE) is required in all patients scheduled for cardiac surgery and is often the basis for surgical decision making itself. In contrast to the nonsurgical setting, in which TTE is usually sufficient to provide most of the information needed to meet the clinical objectives, transesophageal echocardiography (TEE) is also frequently required for patients for whom surgery is being considered. Preoperatively, TEE helps provide information that is critical for surgical planning (eg, valve repair vs valve replacement, coronary artery bypass surgery alone or with concomitant mitral valve repair). During the intraoperative period, TEE is the only modality available for cardiac imaging. In the immediate postoperative period, TEE is often called for because the presence of tissue edema, surgical dressings, and drains and the reduced ability to change the patient's position render transthoracic imaging extremely challenging.

Assessment of Left Ventricular Systolic Function

Left ventricular (LV) systolic function is one of the most important predictors of outcome in all cardiac conditions, and almost all therapeutic decisions in these patients are influenced by the status of LV systolic function. For cardiac anesthesiologists, preoperative knowledge of LV systolic dysfunction is crucial for anticipating and preparing for perioperative complications, whereas subsequent assessments are required for diagnosing and managing the cause of hemodynamic instability. Patients with LV systolic dysfunction who undergo coronary artery bypass graft surgery (CABG) are known to require more inotropic support after cardiopulmonary bypass (CPB).[1,2] Additionally, systolic dysfunction is a reliable prognosticator for surgical mortality.[3–5]

LV ejection fraction (LVEF) is the simplest and the most widely used measure of global LV systolic function. A number of echocardiographic methods are currently available for estimation of LVEF, but the biplane modified Simpson method is the most accurate and is also the method recommended by the American Society of Echocardiography (ASE).[6] In practice, however, LVEF is often estimated semiquantitatively by visual inspection alone, and this technique has been shown to have a reasonably high degree of accuracy when performed by an experienced echocardiographer.[7,8]

The Simpson method considers the LV cavity as a stack of disks of equal height; LV volume is calculated by summing the volumes of all the disks (Fig. 2.1). This is accomplished by manually tracing the LV

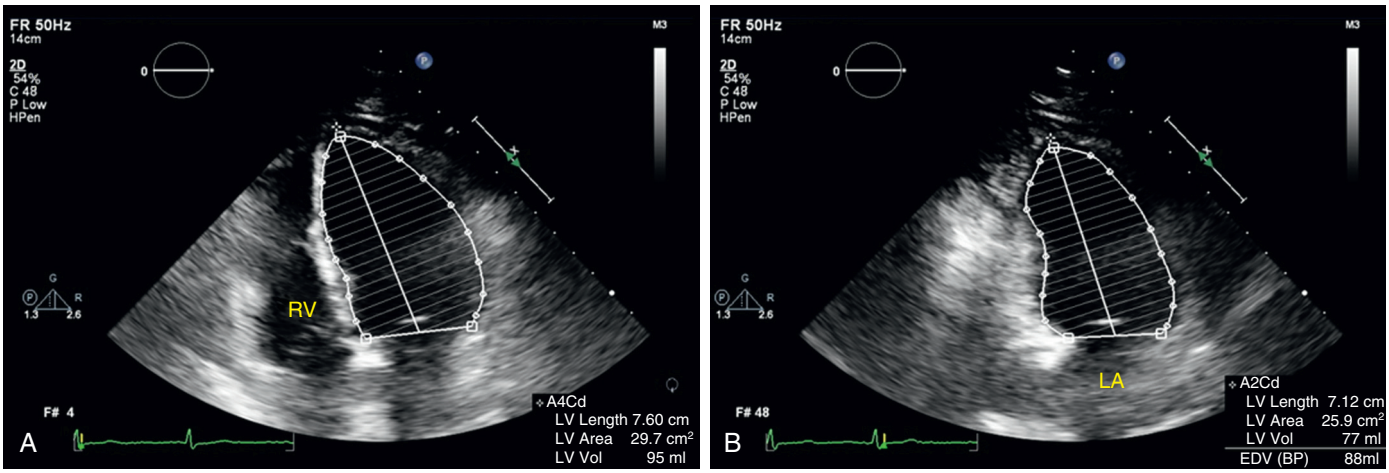

Fig. 2.1 Measurement of left ventricular volumes and ejection fraction using the Simpson summation-of-disks method. A, Apical four-chamber view in end-diastole. B, Same view in end-systole. *LA*, Left atrium; *RV*, right ventricle.

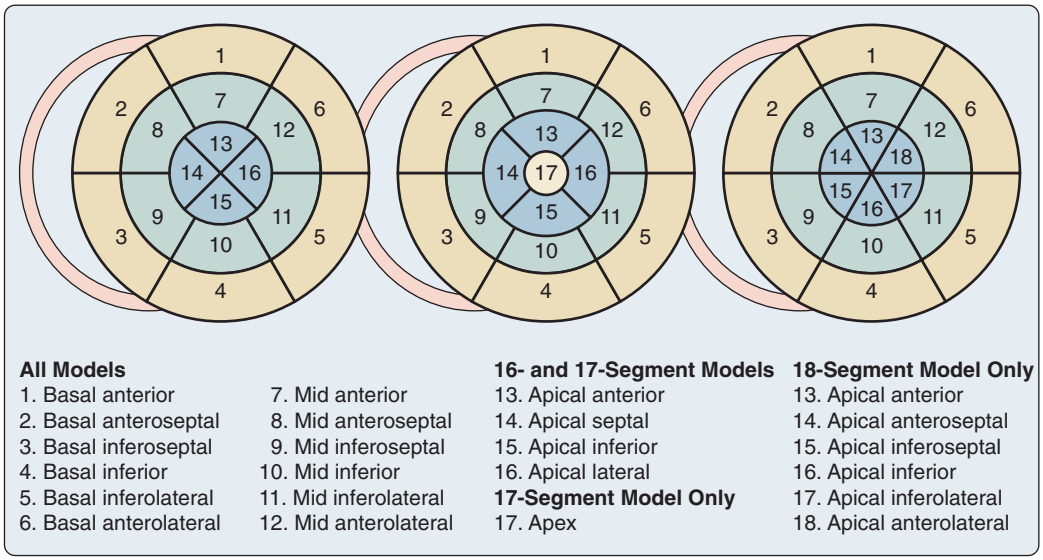

Fig. 2.2 Schematic diagram of the various left ventricular segmentation models: the 16-segment model *(left)*, the 17-segment model *(center)*, and the 18-segment model *(right)*. *(With permission from Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging.* J Am Soc Echocardiogr. *2015;28:1–39 e14.)*

endocardial border in the apical four- and two-chamber views, both at end-diastole and at end-systole. These data are then processed by the internal software of the equipment to provide LV volumes and LVEF. The main advantage of this method is that it remains applicable even when the ventricle is distorted, but its accuracy depends heavily on adequate visualization of the blood-endocardial interface. Currently, harmonic imaging is routinely used to improve endocardial border recognition, and in difficult cases LV cavity contrast can also be used.

Three-dimensional echocardiography (3DE) is the most accurate method for estimation of LVEF by echocardiography.[6] Unlike the two-dimensional (2D) methods, it does not make any assumptions about LV cavity shape and therefore is not influenced by the LV geometry. However, it requires considerable expertise, it has lower temporal resolution, and, as with 2D methods, its accuracy is dependent on image quality. For LVEF estimation by 3DE, a full volume dataset that includes the entire left ventricle is acquired. The image is then analyzed, either online or offline, using quantitation software. The endocardial border is semiautomatically identified in the end-diastolic and the end-systolic frames and adjusted manually to correct for any errors introduced by automatic border recognition. The software then calculates LV volumes and LVEF.

Regional Left Ventricular Systolic Function

In cardiac patients, assessment of regional LV systolic function has considerable clinical relevance. Coronary artery disease (CAD) is the prototype cardiac illness that affects the left ventricle regionally, and the presence of regional LV systolic dysfunction is virtually diagnostic of underlying CAD. The assessment of regional LV systolic function also provides an estimate of the overall extent of myocardial damage, permits recognition of the affected coronary arteries, and facilitates assessment of myocardial viability and inducible myocardial ischemia.

During echocardiography, LV regional function assessment is performed by visual inspection of the extent of *wall thickening* in each myocardial segment. The ventricle is traditionally divided into 16 segments, as recommended by the ASE (Fig. 2.2). A 17-segment model, with the apical cap being the 17th segment, has been proposed for use

when the objective of the examination is to compare the findings with those of nuclear imaging or radiology (CCT or CMR).[6]

Depending on the extent of wall thickening, the wall motion can be classified as normal, hypokinetic, akinetic, or dyskinetic, or it can be formally scored as 1 (normal), 2 (hypokinetic), 3 (akinetic), or 4 (dyskinetic/aneurysmal). Averaging the scores of all the evaluated segments provides a wall motion score index, which can be used as a surrogate for global LVEF. In the assessment of regional wall motion, distinction must be made between actual wall thickening and endocardial motion, which is affected by tethering effects from adjacent myocardial segments and by the translational movement of the heart. The inferior and posterior LV segments are often classified erroneously as hypokinetic by less experienced clinicians who do not appreciate that wall thickening is the gold standard for assessing regional LV function.

Role of Speckle-Tracking Echocardiography

Although LVEF is the most validated measure of LV systolic function, it is operator dependent, load dependent, and relatively insensitive to subtle changes in myocardial contractile function. Additionally, it represents an oversimplification of LV myocardial deformation, which is a complex, multidimensional process. Speckle-tracking echocardiography (STE) offers a potential solution to some of these limitations and serves to complement the information conveyed by assessments of LVEF.[9,10]

STE is a gray-scale–based technique that measures myocardial deformation using frame-by-frame tracking of the acoustic "speckles" present within the gray-scale ultrasound image.[10] The magnitude of the myocardial deformation is expressed as strain, which is the percentage change in length of the myocardial segment. The rate at which this deformation occurs is expressed as the strain rate, which has the unit of sec^{-1}. For clinical purposes, LV myocardial strain is measured in three principal directions: longitudinal, radial, and circumferential. Additionally, LV rotation and twist around the long axis can be measured. The longitudinal strain is derived from the apical four-chamber, two-chamber, and long-axis images; short-axis images are used for estimating radial strain, circumferential strain, rotation, and twist (Fig. 2.3).

Currently, it is feasible to measure global longitudinal strain, which is the average of peak longitudinal strain of all LV myocardial segments, with a high degree of reproducibility that is sufficient for regular clinical use. Accordingly, the ASE recommends measuring global longitudinal strain, along with LVEF, as an estimate of global LV systolic function.[6] The main clinical application of global longitudinal strain is in early recognition of subclinical LV systolic dysfunction in various clinical situations, such as patients receiving potentially cardiotoxic cancer chemotherapies and patients with valvular heart disease and cardiomyopathy. Other components of strain (ie, radial, circumferential) and segmental strain are currently not ready for regular clinical use.

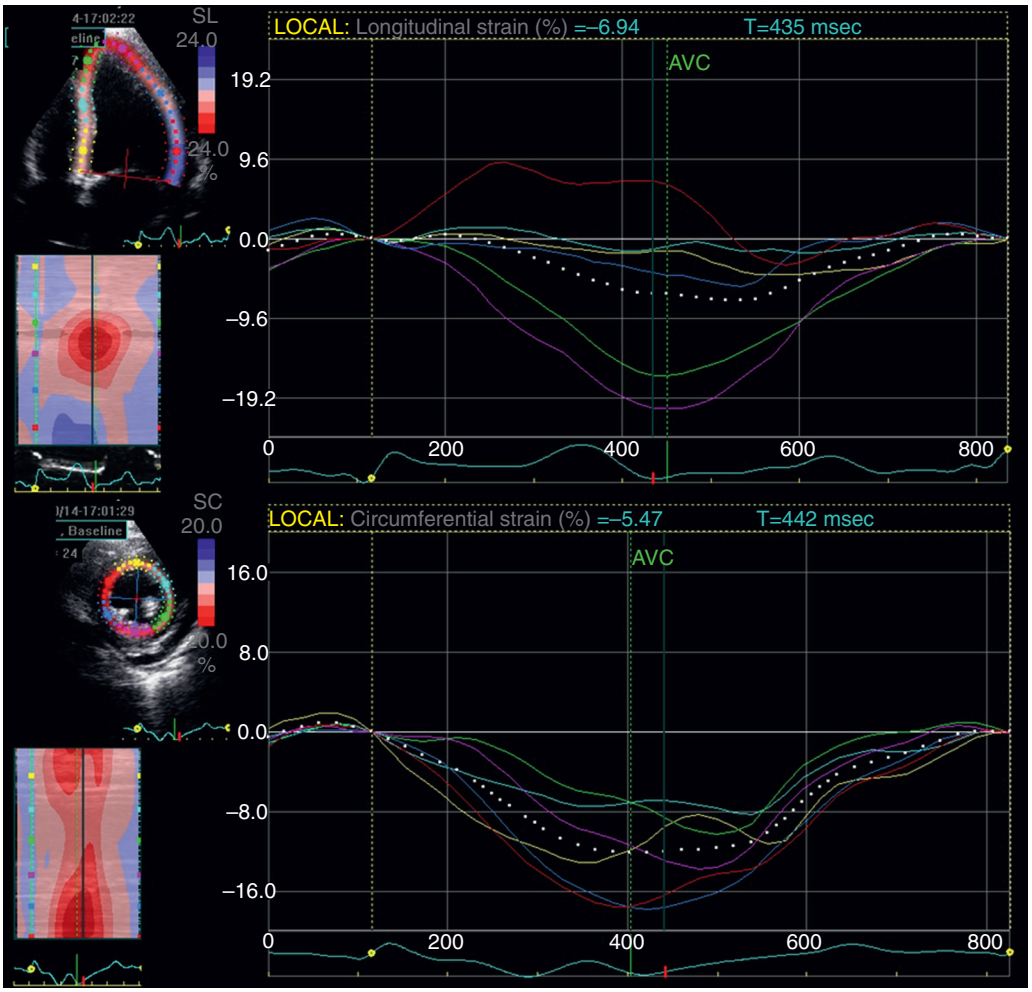

Fig. 2.3 Speckle-tracking echocardiography–based measurement of left ventricular longitudinal strain *(upper panel)* and circumferential strain *(lower panel)*. Colored traces depict segmental strain for corresponding myocardial segments. The dotted white line depicts the average of the six segments visualized in the particular view. *AVC,* Aortic valve closure.

The initial experience with STE has shown great promise; however, because it is a gray-scale–based technique, it is highly dependent on image quality. Additionally, a major limitation of STE at present is intervendor variability, which precludes comparison among measurements obtained on different STE platforms.[11,12] A joint ASE and European Association of Cardiovascular Imaging (EACVI) task force, in collaboration with industry partners, is working toward standardization of all elements involved in STE technology to minimize, and possibly eliminate, this vendor dependence.[12]

Assessment of Left Ventricular Diastolic Function

Abnormalities of LV diastolic function are common in patients undergoing cardiac surgery and have diagnostic and prognostic relevance.[13] Diastolic dysfunction during and after CABG is associated with greater time on CPB and with greater inotropic support up to 12 hours postoperatively.[14] This may be due to deterioration of diastolic dysfunction after CABG, which may persist for several hours.[15–17] Diastolic dysfunction increases the risk of perioperative morbidity and mortality.[18] Assessment of LV diastolic dysfunction permits a more accurate estimation of LV filling pressure (LVFP), information that is crucial to optimal management of critically ill patients. Goal-directed therapy using intravenous fluids, diuretics, vasopressors, and vasodilators requires knowledge of the LVFP.

Echocardiography is currently the best modality for assessing LV diastolic function in clinical practice. Several echocardiographic measures of LV diastolic function are available.[19] The examination usually begins with interrogation of the mitral inflow pattern and measurement of mitral annular velocities. The specific mitral inflow measurements include the mitral inflow early diastolic (E) and late diastolic (A) velocities, the ratio of the two (E/A), and the deceleration time of the E wave (dtE). Mitral annular early diastolic velocity (e′) is measured using tissue Doppler imaging. The ratio of mitral E to e′. (E/e′) provides an accurate and relatively load-independent measure of LVFP. Left atrial (LA) volume and tricuspid regurgitation (TR) jet velocity (a surrogate for pulmonary artery systolic pressure) are other useful measurements. Integrating all this information provides a quick assessment of LV diastolic function in most patients. When required, additional information can be obtained by evaluating pulmonary vein flow patterns, mitral inflow propagation velocity, isovolumic relaxation time, and so on. The ASE has recently published guidelines outlining a stepwise approach to assessment of LV diastolic function and estimation of LVFP or LA pressure in patients with and without LV systolic dysfunction (Fig. 2.4).[19a]

Right Heart Evaluation

Dysfunction of the right side of the heart, in the absence of congenital heart disease, is most often secondary to left heart pathologies, especially MV disease and severe LV systolic dysfunction. Additionally, obstructive airway disease and pulmonary thromboembolism are common in cardiac surgical patients. Primary right heart pathology is encountered less frequently and includes right ventricular (RV) myocardial infarction and organic tricuspid valve disease.

Unlike the left ventricle, the right ventricle has complex anatomy. The RV inflow portion, main body, and outflow tract (RVOT) do not lie in the same anatomic plane, rendering it impossible to image the entire right ventricle from one echocardiographic window. Accordingly, different acoustic windows must be used, including the RV inflow view, the RV outflow view, the RV-focused apical four-chamber view, and the subcostal view. Abnormalities of RV size and shape and global and regional contractility are qualitatively assessed, and various RV and right atrial (RA) dimensions are obtained from these windows[20] (Table 2.1). Additionally, tricuspid valve function, the presence of any intracardiac masses, and so on can be evaluated.

Because of the complex shape of the right ventricle, the RV ejection fraction generally cannot be measured by echocardiography. As a result, RV systolic function is indirectly assessed using alternative parameters, such as M-mode–derived tricuspid annular plane systolic excursion, tricuspid annular systolic velocity on tissue Doppler imaging, RV cavity fractional area change on 2D echocardiography, and myocardial performance index obtained from pulsed-wave Doppler or tissue Doppler imaging[20] (see Table 2.1). Additionally, RV free wall longitudinal strain can be measured with the use of STE.[6] Although it has now become feasible to measure RV ejection fraction by 3DE, the method is cumbersome and not yet ready for regular clinical use.[6]

In contrast, the assessment of RV diastolic function is simpler than that of LV diastolic function. The easy accessibility of the inferior vena cava (IVC) for imaging permits direct estimation of RA pressure, which in the absence of any tricuspid inflow obstruction is the same as RV filling pressure (RVFP).[20] If the IVC is normal in size (≤21 mm) and collapses by more than 50% on deep inspiration, the RA pressure is considered to be normal (ie, ~3 mm Hg; range, 0–5 mm Hg).

TABLE 2.1 Echocardiographic Measures of Right Ventricular Size and Systolic Function

Measurement	*Transthoracic Echocardiographic View*	*Normal Value*
RV Size		
RV basal diameter	RV focused apical four-chamber view	25–41 mm
RV mid-diameter	RV focused apical four-chamber view	19–35 mm
RV longitudinal diameter	RV focused apical four-chamber view	59–83 mm
RV outflow tract proximal	Parasternal long-axis view	21–35 mm
RV outflow tract distal	Parasternal short-axis view	17–27 mm
RV free wall thickness	Subcostal view	1–5 mm
RV Systolic Function		
Fractional area change	RV focused apical four-chamber view	≥35%
Tricuspid annular plane systolic excursion	Apical four-chamber view (providing best alignment of ultrasound beam with annulus motion)	>16 mm
Tricuspid annular peak systolic velocity	Apical four-chamber view (providing best alignment of ultrasound beam with annulus motion)	>10 cm/s
Pulsed Doppler myocardial performance index	A combination of parasternal short-axis view and apical four-chamber view	≤0.43
Tissue Doppler myocardial performance index	Apical four-chamber view (providing best alignment of ultrasound beam with annulus motion)	≤0.54
RV ejection fraction by 3DE	Full volume dataset from apical window	≥45%
RV free wall strain	RV focused apical four-chamber view	–20% or more

3DE, Three-dimensional echocardiography; *RV*, right ventricular.

From Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *J Am Soc Echocardiogr.* 2015;28:1–39 e14; Rudski LG, Lai WW, Afilalo J, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. *J Am Soc Echocardiogr.* 2010;23:685–713; quiz 786–788.

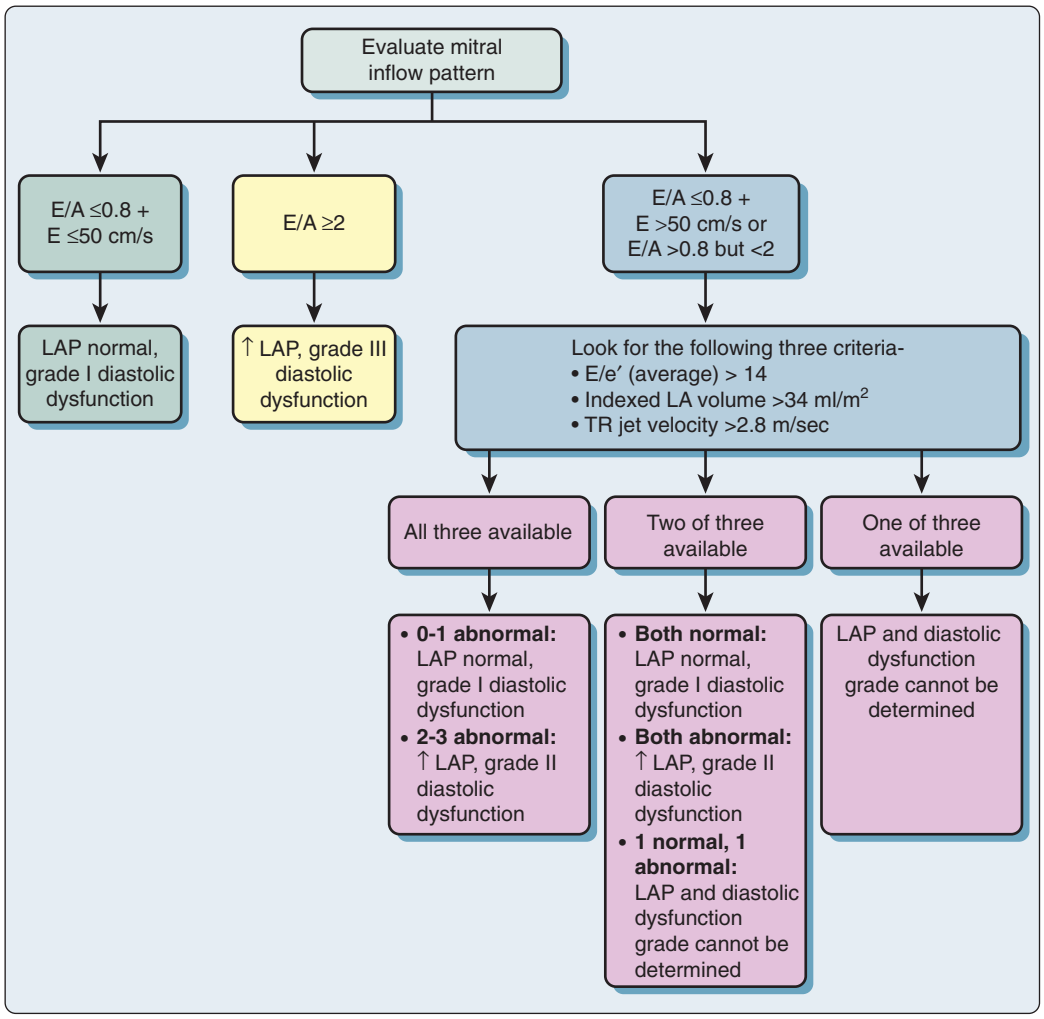

Fig. 2.4 Algorithm for echocardiographic estimation of left atrial pressure and grading left ventricular diastolic function in patients with myocardial disease but with or without left ventricular systolic dysfunction. *A,* Mitral inflow late diastolic velocity; *E,* mitral inflow early diastolic velocity; *e′,* mitral annular early diastolic velocity; *LA,* left atrial; *LAP,* left atrial pressure; *TR,* tricuspid regurgitation. *(Modified from: Nagueh SF, Smiseth OA, Appleton CP, et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging.* J Am Soc Echocardiogr. *2016;29:277–314.)*

However, a dilated IVC (>21 mm) that collapses by less than 50% on inspiration indicates significantly elevated RA pressure (~15 mm Hg; range, 10–20 mm Hg). When IVC size and collapsibility are discrepant with one another, the RA pressure is assumed to be in the intermediate range (~8 mm Hg; range, 5–10 mm Hg). In such cases, additional parameters can be obtained to more accurately define the RA pressure, such as tricuspid inflow E/A and E/e′ ratios, E-wave deceleration time, and hepatic vein flow pattern. Because both IVC size and collapsibility are affected by positive-pressure ventilation, this approach cannot be used for estimating RA pressure in patients who are mechanically ventilated, which is quite common in the perioperative setting. Interrogation of the hepatic vein flow pattern can provide valuable information about RA pressure in this setting.

RV systolic pressure and pulmonary vascular resistance can also be estimated by echocardiography (see later discussion).

Assessment of Valve Lesions

Valvular heart disease is the second most common primary indication, after CAD, for cardiac surgery. Valve lesions also frequently coexist in patients undergoing surgery for other cardiac and noncardiac indications.

Echocardiography is currently the best modality available for evaluation of valvular heart disease. A combination of TTE and TEE permits comprehensive assessment of valve anatomy and function and provides all the relevant information required to determine the need for and type of valve intervention. Additionally, intraoperative TEE is useful in assessing the adequacy of valve surgery and in recognizing any surgery-related complications (eg, LV outflow tract obstruction, paravalvular regurgitation).

Mitral Valve Lesions

Rheumatic heart disease is, by far, the most common cause of mitral stenosis (MS), even in developed nations. Mitral annular calcification, which is common in elderly persons and in those undergoing hemodialysis, can also produce mitral inflow obstruction, although mitral regurgitation (MR) is more frequent.

In patients with rheumatic MS, echocardiography typically reveals mitral leaflet thickening with doming of the anterior mitral leaflet, restriction of posterior mitral leaflet motion, and variable amounts

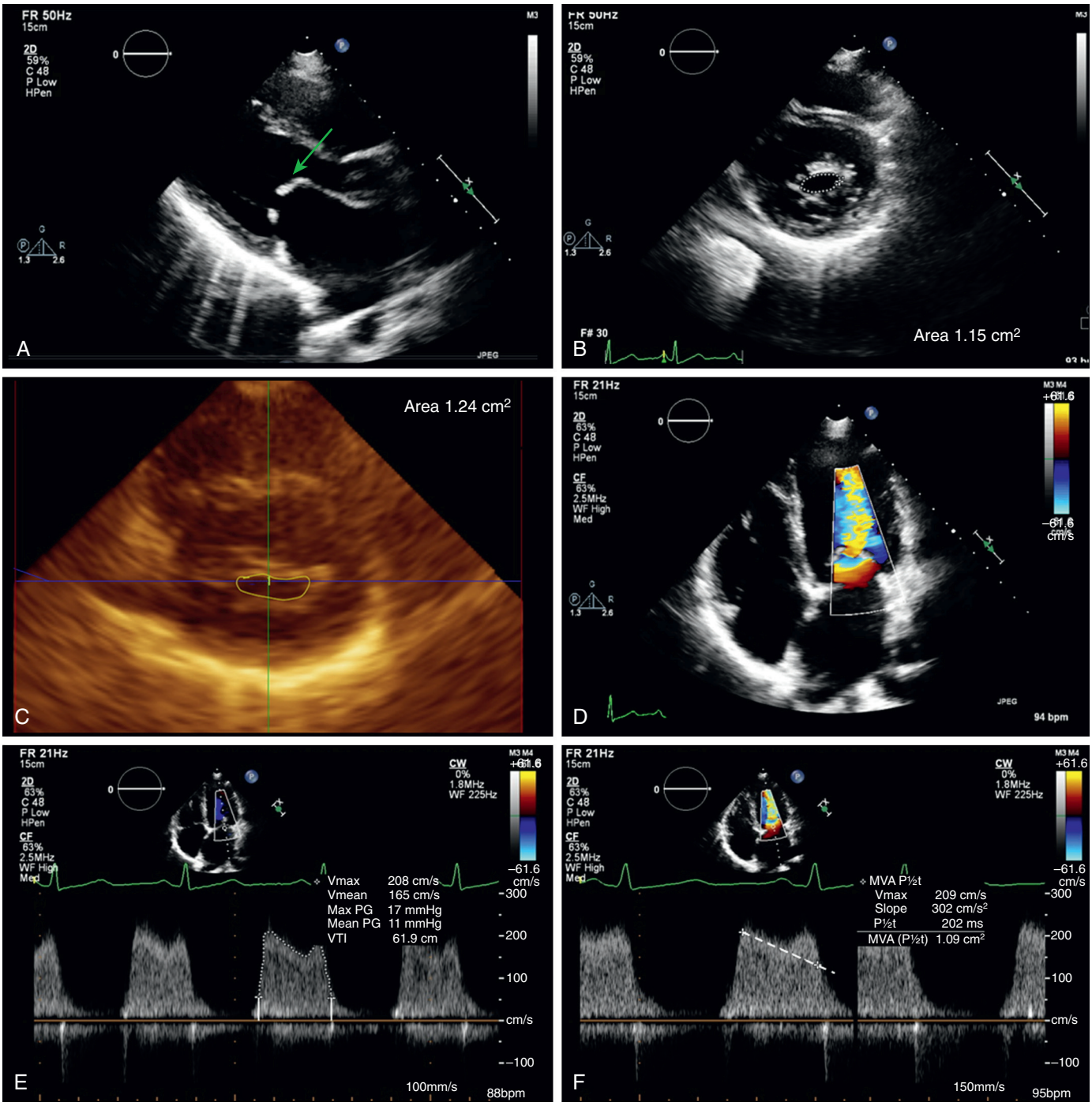

Fig. 2.5 Typical echocardiographic findings in a patient with rheumatic mitral stenosis. A, Parasternal long-axis view demonstrates restricted mitral leaflet opening with doming of anterior mitral leaflet *(arrow)*. Estimation of mitral valve area by planimetry from the parasternal short-axis view using two-dimensional (B) and three-dimensional (C) echocardiography is shown. D, Apical four-chamber view with color Doppler shows turbulent forward blood flow jet through the narrowed mitral orifice. Continuous wave Doppler is shown for estimation of transmitral gradients (E) and mitral valve area (F) by the pressure half-time method.

of leaflet calcification, fusion, and calcification of commissures. Thickening, shortening, and calcification of the subvalvular apparatus are also typically present (Fig. 2.5). These abnormalities can be formally graded with one of the various scoring systems available.[21–25] The severity of MS can be determined by measuring the transmitral gradient or by estimating the MV area, using either planimetry or the pressure half-time method[26] (Table 2.2; see Fig. 2.5). Of these, MV planimetry is the most accurate method (unless the predominant obstruction is at the level of chordae) because it is not influenced by heart rate and hemodynamics. However, its accuracy depends on gray-scale image quality and on ensuring that the measurement is performed exactly at the tips of the MV leaflets. 3DE is the best modality for this purpose because it allows the operator to optimally position the imaging plane for planimetry.[27,28] In addition to assessment of MV

TABLE 2.2 Echocardiographic Criteria for Severity of Mitral Valve Lesions

Parameter	*Mild*	*Moderate*	*Severe*
Mitral Stenosis			
Specific Findings			
Mitral valve area (cm^2)	>1.5	1.0–1.5	<1.0
Supportive Findings			
Mitral valve mean gradient (mm Hg)[a]	<5	5–10	>10
Pulmonary artery systolic pressure (mm Hg)	<30	30–50	>50
Mitral Regurgitation			
Structural Parameters			
Left atrial size	Normal[b]	Normal or dilated	Usually dilated[c]
Left ventricular size	Normal[b]	Normal or dilated	Usually dilated[c]
Mitral valve apparatus	Normal or abnormal	Normal or abnormal	Abnormal
Doppler Parameters			
Color flow jet area (cm^2)[d]	Small, central jet (usually <4 cm^2, or <20% of left atrial area)	Variable	Large central jet (usually >10 cm^2, or >40% of left atrial area) or variable-size wall-hugging jet in left atrium
Mitral inflow (pulsed wave)	A-wave dominance[e]	Variable	E wave dominance (E usually >1.2 m/s)[e]
Jet density (continuous wave)	Incomplete or faint	Dense	Dense
Jet contour (continuous wave)	Parabolic	Usually parabolic	Early peaking triangular
Pulmonary vein flow	Systolic dominance[f]	Systolic blunting[f]	Systolic flow reversal[g]
Quantitative Parameters[h]			
Vena contracta width (cm)	<0.3	0.3–0.69	≥0.7
Regurgitant volume (mL/beat)[i]	<30	30–44 (mild to moderate) 45–59 (moderate to severe)	≥60
Regurgitant fraction (%)	<30	30–39 (mild to moderate) 40–49 (moderate to severe)	≥50
Effective regurgitant orifice area (cm^2)[j]	<0.20	0.20–0.29 (mild to moderate) 0.30–0.39 (moderate to severe)	≥0.40

[a]At heart rates between 60 and 80 beats/minute.
[b]Unless there are other causes of left atrial or left ventricular dilatation.
[c]Acute mitral regurgitation is an exception.
[d]At a Nyquist limit of 50 to 60 cm/s.
[e]Usually in patients older than 50 years of age or in conditions of impaired relaxation, in the absence of mitral stenosis or other causes of elevated left atrial pressure.
[f]Unless other reasons for systolic blunting (eg, atrial fibrillation, elevated left atrial pressure) are present.
[g]Pulmonary venous systolic flow reversal is specific but not sensitive for severe mitral regurgitation.
[h]Quantitative parameters can help subclassify the moderate regurgitation group into mild-to-moderate and moderate-to-severe subgroups, as shown.
[i]For functional mitral regurgitation due to left ventricular systolic dysfunction, ≥30 mL is used as the cutoff point for defining severity.
[j]For functional mitral regurgitation due to left ventricular systolic dysfunction, ≥0.20 cm^2 is used as the cutoff point for defining severity.
From Baumgartner H, Hung J, Bermejo J, et al. Echocardiographic assessment of valve stenosis: EAE/ASE recommendations for clinical practice. *J Am Soc Echocardiogr.* 2009;22:1–23; quiz 101–102; Zoghbi WA, Enriquez-Sarano M, Foster E, et al. Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. *J Am Soc Echocardiogr.* 2003;16:777–802.

morphology and MS severity, echocardiography aids in the evaluation of concomitant valve lesions, if any, and in detection of LA or LA appendage thromboses.

In comparison to MS, MR is a more complex entity with a multitude of etiologies and pathogenic mechanisms. MR can be organic, caused by direct pathologic involvement of one or more components of the MV apparatus; examples include rheumatic heart disease, degenerative diseases, systemic inflammatory diseases, infective endocarditis, ischemic heart disease (chordal or papillary muscle rupture), and congenital malformations (Fig. 2.6). Alternatively, MR can be of functional origin: The MV apparatus itself is normal, but leaflet coaptation during systole is impaired as a result of regional or global dilatation and dysfunction of the LV. Functional MR is common in patients with LV systolic dysfunction undergoing CABG and is an independent predictor of morbidity and mortality in these patients.[29–31]

TEE provides excellent visualization of the MV because of its proximity to the transducer. The exact pathology, the extent of the pathology, mitral annulus size, and papillary muscle and chordal geometry can all be delineated in great detail. Additionally, in patients with functional MR, tenting height, tenting area, and coaptation length can be assessed. When available, 3D TEE provides incremental information regarding MV pathology. One major advantage of 3D TEE is that it provides the "surgeon's view" of the MV, facilitating easy appreciation of the MV pathology preoperatively (Fig. 2.7).

For assessment of MR severity, a number of measures combining 2D and Doppler data are available, as outlined in Table 2.2.[32] Lower thresholds are used for defining the severity of functional MR compared with organic MR.[33]

Aortic Valve Lesions

Senile degeneration, bicuspid aortic valve (AV) disease, and rheumatic heart disease are the most common causes of aortic stenosis (AS). The same conditions can also result in aortic regurgitation (AR). AR can also result from aortic root pathologies, infective endocarditis, systemic inflammatory diseases, and other causes.

The echocardiographic assessment of AV lesions is based on principles similar to those for MV lesions (Table 2.3).[26,32] Compared with the MV, however, adequate visualization of the AV is often difficult, even on TEE. Inability to align the Doppler beam with the direction of blood flow in the conventional TEE views offers further challenges to the assessment of AS severity. Deep transgastric views are required to measure the AS gradient with TEE, but they may not always result in a satisfactory evaluation. An additional challenge in the assessment of AS severity is the frequent occurrence of inappropriately low transaortic gradients despite a significantly reduced AV area.[26,33–35] This discrepancy is common in the setting of LV systolic dysfunction. Dobutamine echocardiography may help in further evaluation of these patients, as described later. However, a paradoxically low gradient may also be seen in the presence of a normal LVEF. The exact pathogenesis underlying this entity, its prognostic significance, and the optimum management strategies remain debatable.[33–35]

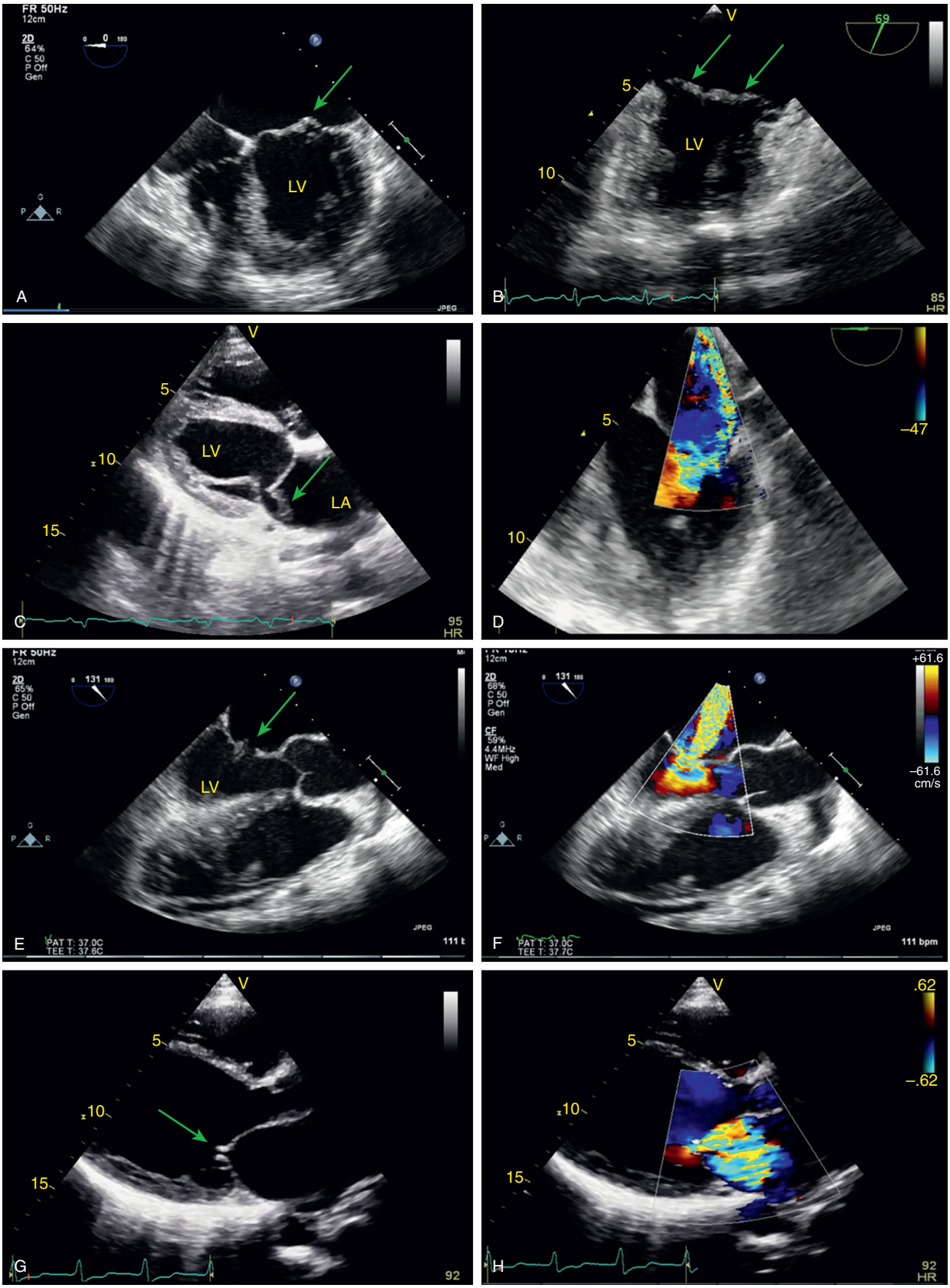

Fig. 2.6 Illustrative examples of mitral regurgitation from various causes. A and B, Bileaflet mitral valve prolapse (*arrows*). C and D, Papillary muscle rupture secondary to acute myocardial infarction. The torn papillary muscle is seen prolapsing into the left atrium (C, *arrow*), resulting in posteriorly directed severe mitral regurgitation (D). E and F, Infective endocarditis with perforation of the anterior mitral leaflet (E, *arrow*) results in severe mitral regurgitation through the perforation (F); G and H, Functional severe mitral regurgitation secondary to left ventricular systolic dysfunction and dilatation. Mitral valve leaflets are markedly tented resulting in noncoaptation (*arrow* in G). *LA*, Left atrium; *LV*, left ventricle.

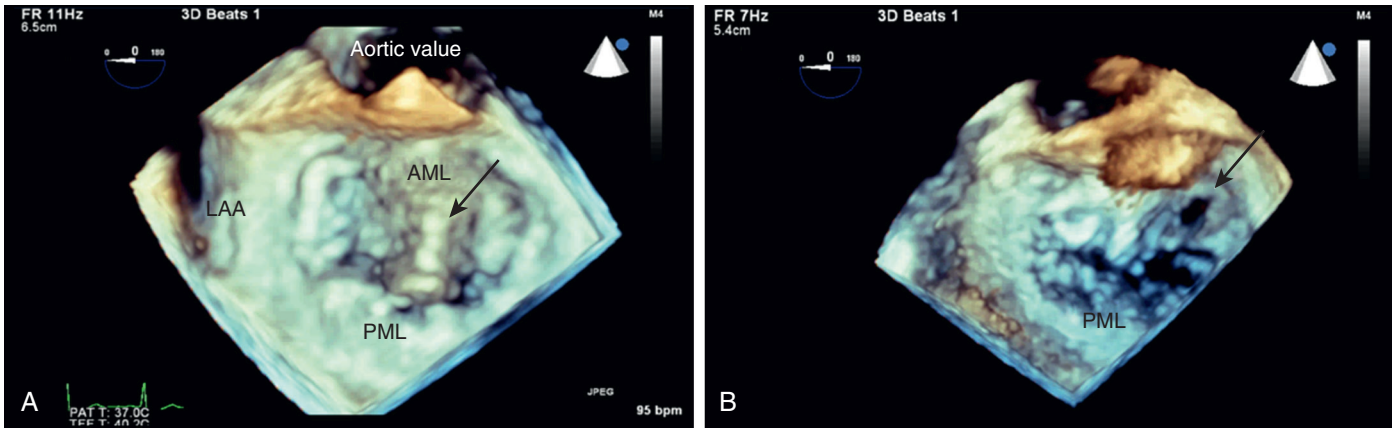

Fig. 2.7 Three-dimensional transesophageal echocardiography shows the mitral valve from the left atrial aspect, with the aortic valve in the 12 o'clock position (the "surgeon's view"). A, Prolapse of the A2 scallop of the anterior mitral leaflet *(arrow)*. B, Perforation in the A3 scallop of the anterior mitral leaflet *(arrow)*. *AML*, Anterior mitral leaflet; *LAA*, left atrial appendage; *PML*, posterior mitral leaflet.

TABLE 2.3 Echocardiographic Criteria for Severity of Aortic Valve Lesions

Parameter	*Mild*	*Moderate*	*Severe*
Aortic Stenosis			
Aortic jet peak velocity (m/s)	2.6–2.9	3.0–4.0	>4.0
Aortic valve mean gradient (mm Hg)[a]	<20	20–40	>40
Aortic valve area (cm^2)	>1.5	1.0–1.5	<1.0
Indexed aortic valve area (cm^2/m^2)	>0.85	0.60–0.85	<0.60
Velocity ratio	>0.50	0.25–0.50	<0.25
Aortic Regurgitation			
Structural Parameters			
Left ventricular size	Normal[b]	Normal or dilated	Usually dilated[c]
Aortic leaflets	Normal or abnormal	Normal or abnormal	Abnormal/ flail, or wide coaptation defect
Doppler Parameters			
Jet width in LVOT (color flow)[d]	Small in central jets	Intermediate	Large in central jets; variable in eccentric jets
Jet density (continuous wave)	Incomplete or faint	Dense	Dense
Jet deceleration rate (continuous wave) (pressure half-time, ms)[c]	Slow, >500	Medium, 500–200	Steep, <200
Diastolic flow reversal in descending aorta (pulsed wave)	Brief, early diastolic reversal	Intermediate	Prominent holodiastolic reversal
Quantitative Parameters[f]			
Vena contracta width (cm)[d]	<0.3	0.3–0.60	≥0.6
Jet width/LVOT width ratio (%)[d]	<25	25–45 (mild to moderate) 46–64 (moderate to severe)	≥65
Jet CSA/LVOT CSA ratio (%)[d]	<5	5–20 (mild to moderate) 21–59 (moderate to severe)	≥60
Regurgitant volume (mL/beat)	<30	30–44 (mild to moderate) 45–59 (moderate to severe)	≥60
Regurgitant fraction (%)	<30	30–39 (mild to moderate) 40–49 (moderate to severe)	≥50
Effective regurgitant orifice area (cm^2)	<0.10	0.10–0.19 (mild to moderate) 0.20–0.29 (moderate to severe)	≥0.30

CSA, Cross-sectional area; *LVOT*, left ventricular outflow tract.
[a]At heart rates between 60 and 80 beats/minute.
[b]Unless there are other causes of left ventricular dilatation
[c]Acute aortic regurgitation is an exception.
[d]At a Nyquist limit of 50 to 60 cm/s.
[e]Pressure half-time is shortened with increasing left ventricular diastolic pressure and vasodilator therapy and may be lengthened with chronic adaptation to severe aortic regurgitation.
[f]Quantitative parameters can help subclassify the moderate regurgitation group into mild-to-moderate and moderate-to-severe subgroups, as shown.
From Baumgartner H, Hung J, Bermejo J, et al. Echocardiographic assessment of valve stenosis: EAE/ASE recommendations for clinical practice. *J Am Soc Echocardiogr.* 2009;22:1–23; quiz 101–102; Zoghbi WA, Enriquez-Sarano M, Foster E, et al. Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. *J Am Soc Echocardiogr.* 2003;16:777–802.

Other Valve Lesions

Functional TR is another common valve lesion encountered in the surgical setting. Any condition resulting in severe pulmonary hypertension can cause TR. In cardiac patients, the most common causes are severe LV systolic dysfunction and significant MV disease. Significant TR in the absence of pulmonary hypertension occurs much less frequently and may be a consequence of previous RV myocardial infarction, atrial septal defect, or direct organic involvement of tricuspid valve leaflets in rheumatic heart disease, carcinoid disease, trauma, infective endocarditis, or the Ebstein anomaly. Organic, significant TR usually requires surgical correction, but the optimum management strategy for functional TR remains controversial. Although correction of the primary

pathology can often lead to partial or complete resolution of TR, it is less likely to regress, and may even progress, if tricuspid annulus dilation is present. In patients undergoing MV surgery, a maximum tricuspid annulus diameter greater than 4 cm (or >21 mm/m^2) on preoperative echocardiography is generally considered to indicate the need for surgical correction of TR at the time of MV surgery.[33,36–37]

Tricuspid valve pathology is best imaged by TTE and TEE, but on occasion the patient may have poor TTE windows, or the TEE study may be insufficient. Morphology of the valve, size of the annulus, TR gradient, status of RV systolic function, and RA pressure are the important data that need to be obtained from echocardiography.

Tricuspid stenosis is very rare and is usually congenital, associated with carcinoid tumor, or rheumatic in origin. Significant pulmonary regurgitation is also rare and typically occurs secondary to pulmonary hypertension or previous repair of congenital pulmonary stenosis. Pulmonary stenosis, however, is not uncommon. It is almost always congenital in origin and usually occurs as an isolated valvular, subvalvular, or supravalvular stenosis. The pulmonic valve is not well visualized on either TTE or TEE, especially in adults, but the transpulmonary gradient can be estimated in most patients.

Apart from native valve disease, patients can present with a wide variety of prosthetic heart valve (PHV)-related complications. Although echocardiography remains the primary modality for the evaluation of PHV function, adequate visualization of mechanical prosthetic valves, especially prosthetic AVs, is difficult because of acoustic shadowing and other artifacts caused by prosthetic materials. Mitral PHVs, however, can be imaged reasonably well, especially with TEE. The mobility of occluders, thickening of the leaflets in cases of tissue valves, valve dehiscence, paraprosthetic leaks, and presence of pannus, thrombus, or vegetation can be diagnosed effectively. Additionally, a reasonable amount of hemodynamic information can be obtained and can be corroborated with the anatomic findings to ascertain the actual mechanisms and severity of PHV malfunction.

Infective endocarditis is another common indication for valve surgery in patients with either native or prosthetic valves. It can be a life-threatening disease, with mortality rates reported to be as high as 40%.[38] The diagnosis is usually based on visualization of vegetations by echocardiography, which is the primary modality for diagnosing infective endocarditis. In severe cases, valve perforations, paravalvular abscesses, and PHV dehiscence can also be seen. With current-generation technologies, the sensitivity of TTE for detection of native valve endocarditis is in the range of 82% to 89%, and the specificity is 70% to 90%.[38–41] For prosthetic valve endocarditis, however, the sensitivity of TTE is rather poor (<50%). TEE is clearly the imaging modality of choice for infective endocarditis. For native valve endocarditis, TEE has a sensitivity of 90% to 100% and a specificity greater than 90%. Even for prosthetic valve endocarditis, TEE is highly accurate, with sensitivity in the range of 80% to 90% and specificity greater than 90%.[38–44]

Hemodynamic Assessment

Although invasive hemodynamic monitoring is routine in patients undergoing cardiac surgery, echocardiography offers an excellent alternative if invasive monitoring is not feasible. Typically, echocardiography is the best option for hemodynamic assessment during the preoperative period, or in the late postoperative period after discharge from the intensive care unit, and also in noncardiac surgery. An added advantage of echocardiography is that it allows hemodynamic data to be directly correlated with cardiac anatomy and function. Vast amounts of hemodynamic information can be obtained from echocardiography, including intracardiac pressures, cardiac output, and vascular resistance.

Intracardiac Pressures

A wide range of intracardiac pressures can be derived using the simplified Bernoulli equation, which states that the pressure gradient (ΔP) driving a blood flow jet within the cardiovascular system is equal to $4v^2$, where v is the peak jet velocity. If the pressure within the upstream or downstream chamber is known, the pressure in the other chamber can be calculated by measuring the peak jet velocity between the two chambers. The most common application of this formula is in estimation of RV systolic pressure or pulmonary artery systolic pressure from a TR jet. The RV systolic pressure is derived by adding mean RA pressure (estimated from IVC measurements as described earlier or from jugular venous observations with head elevation) to the peak TR jet gradient. In the absence of any RVOT obstruction, RV systolic pressure is identical to the pulmonary artery systolic pressure. The same principle can be applied for estimating other intracardiac pressures using other jets (eg, pulmonary artery end-diastolic pressure from a pulmonary regurgitant jet, RV systolic pressure from the jet crossing a ventricular septal defect, LA pressure from the mitral regurgitation jet).

A number of equations are also available for the estimation of peak systolic and mean pulmonary pressure from RVOT acceleration time and from pulmonary regurgitation jet velocity.[45–48] However, these formulas involve certain assumptions, and their accuracy is affected by several hemodynamic factors.

Cardiac Output and Vascular Resistance

During echocardiography, flow through any cardiac structure can easily be estimated by multiplying the cross-sectional area of the structure by the velocity-time integral (VTI) of the blood flow through that structure. This provides stroke volume, which is then multiplied by the heart rate to calculate cardiac output (CO).

In clinical practice, LV outflow tract (LVOT) is the site most commonly used for measurement of CO (Fig. 2.8). LVOT is easy to visualize, it is almost circular in shape, and its size does not change much during the cardiac cycle. Additionally, the blood flow through the LVOT is mostly laminar, which minimizes the chance of inadvertently sampling velocities that are not truly representative of the actual blood flow at that point. The LVOT diameter is measured from the parasternal or TEE long-axis view and the VTI from the apical five-chamber or TEE deep transgastric view (using pulsed-wave Doppler ultrasonography). Precision should be maintained while obtaining these measurements, because any errors will become magnified when the calculations are performed.[49]

There are several circumstances in which the CO measured at the LVOT may not be accurate or representative of true CO, including significant LVOT obstruction and AR. In such cases, the measurements can be performed at the MV or at the RVOT, provided there is no significant MR or intracardiac shunt, respectively.

Once the CO has been determined, systemic vascular resistance (SVR) can be estimated using the principle of Ohm's law, which states that the resistance across any vascular circuit is equal to the pressure gradient across the circuit divided by the flow:

$$\text{SVR} = (\text{Mean aortic pressure} - \text{Mean RA pressure}) / \text{Systemic blood flow}$$

Mean systemic blood pressure is used as a surrogate for mean aortic pressure; all the other values can be obtained by echocardiography, as described earlier. When the pressure gradient is measured in mm Hg and CO in L/min, the derived vascular resistance is described in Wood units, which can be converted to metric units (dynes·s·cm^{-5}) by multiplying by 80.

Although a similar equation can be applied for the estimation of pulmonary vascular resistance (PVR), it is generally not used because of the difficulties in obtaining transpulmonary gradients by echocardiography. An alternate, simpler method has been proposed for measuring the PVR (in Wood units); it relies on the TR jet velocity (in m/s) and the RVOT-VTI (in cm):

$$\text{PVR} = (\text{Peak TR velocity} / \text{RVOT} - \text{VTI}) \times 10 + 0.16$$

Pericardial Diseases

Bleeding into the pericardial space is a common occurrence during the immediate postoperative period after cardiac surgery and an important

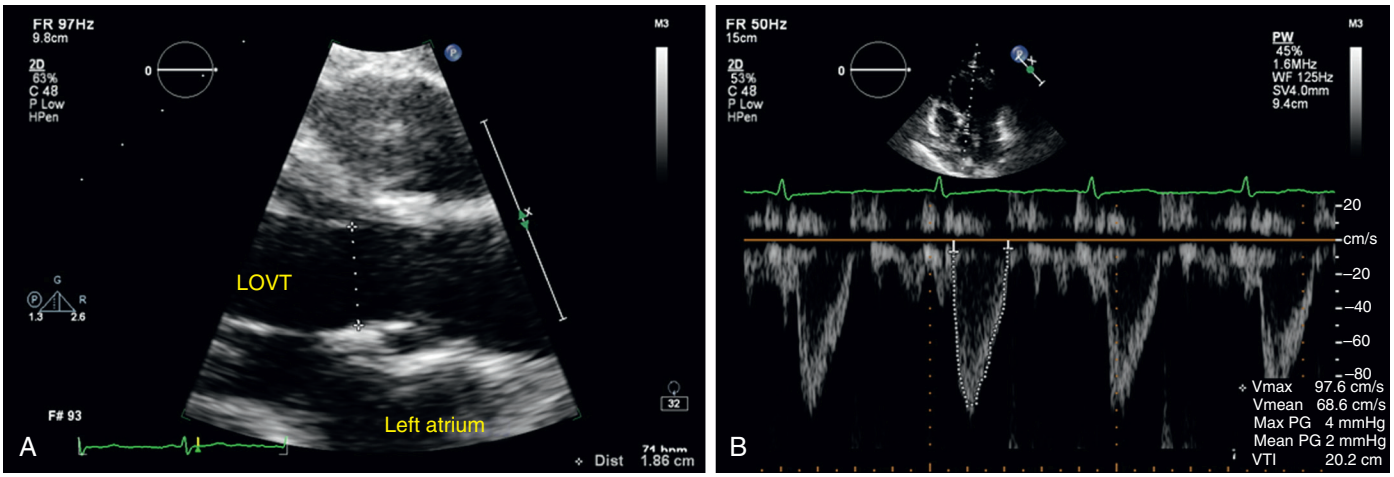

Fig. 2.8 Echocardiographic estimation of cardiac output at the left ventricular outflow tract (LVOT). A, LVOT diameter is measured from the parasternal long-axis view in midsystole. B, Measurement of the LVOT velocity-time integral (VTI) using pulsed-wave Doppler ultrasonography in apical five-chamber view. See text for details.

cause of hemodynamic compromise in this setting. Pericardial effusions can also occur secondary to a number of cardiac and noncardiac conditions; for example, it can occur as an immune reaction after myocardial infarction or cardiac surgery. Echocardiography not only allows detection and quantification of pericardial collections but also permits assessment of the hemodynamic significance of the phenomenon.

The pericardial fluid or blood collection can be circumferential or localized, and it usually appears as an echolucent space outside the heart. When bleeding is the cause, however, the fluid is more echogenic, and even frank clots may be seen. Additionally, fibrinous bands, septa, and pericardial echogenic deposits are commonly visualized in cases of exudative effusions. When a pericardial collection is localized posterior to the LV, it can be difficult to distinguish from a pleural collection. Extension of the collection posterior to the descending thoracic aorta suggests a pleural origin, rather than pericardial.

When a pericardial effusion is visualized, an important clinical question is whether there is any evidence of cardiac tamponade. Evidence of diastolic RA and RV collapse, a restrictive-type mitral inflow pattern with significant respiratory variation in mitral and tricuspid inflow velocities, and a dilated, noncollapsing IVC indicate the presence of cardiac tamponade (Fig. 2.9). The size of the effusion itself is not a reliable indicator because the rapidity of fluid accumulation is more important than the actual quantity of fluid in the causation of cardiac tamponade.

Apart from pericardial collections, constrictive pericarditis is another common pericardial pathology and is itself an indication for cardiac surgery. In patients with suspected chronic constrictive pericarditis, pericardial thickening and calcification may be visualized on echocardiography. More often, however, it is the hemodynamic abnormalities that draw attention to the possibility of constriction physiology. The echocardiographic findings suggestive of constriction include (1) exaggerated ventricular interdependence, as evidenced by septal bounce, deviation of septal position with respiration, and significant respiratory variation in mitral and tricuspid inflow velocities; (2) a restrictive-type mitral inflow pattern with a relatively preserved or exaggerated mitral e′ (known as annulus paradoxus) or a medial mitral e′ that is taller than the lateral e′ (known as annulus reversus); and (3) a dilated, noncollapsing IVC. In uncertain cases, assessment of myocardial strain and torsion abnormalities may provide further clues to help reach an accurate diagnosis.[50–51]

In patients with various pericardial pathologies, echocardiography helps not only in diagnostic evaluation but also in guiding therapeutic interventions (eg, pericardiocentesis, pericardiectomy) and in assessing the adequacy of those procedures.

Diseases of the Aorta

Aortic aneurysm and aortic dissection are commonly encountered cardiac pathologies. Despite advances in endovascular repair, surgery remains the primary treatment modality for most patients. Although CCT is the modality of choice for imaging the aorta, echocardiography is a good modality for initial evaluation, especially in acutely ill or hemodynamically unstable patients.

Ascending aortic aneurysms usually occur because of cystic medial degeneration; they frequently involve the aortic root and cause AR. These aneurysms are more common in patients with hypertension, bicuspid AV, or connective tissue diseases such as Marfan or Ehlers-Danlos syndrome (see Chapter 23).[52] In contrast, descending aortic aneurysms are mostly caused by atherosclerosis and are associated with the same risk factors as for CAD. Among descending aortic aneurysms, abdominal aneurysms are more common than thoracic aortic aneurysms.

Although TTE can provide clear visualization of the aortic root and the proximal segment of the ascending aorta, imaging of the arch and the descending thoracic aorta is usually challenging. In contrast, TEE can image the entire length of the descending thoracic aorta, in addition to aortic root and the proximal segment of ascending aorta, but the aortic arch, especially the proximal portion, is not visualized because of the interposition of the left main bronchus in the path of the ultrasound beam. An aortic aneurysm appears as a localized dilatation of a segment of the aorta to more than 1.5 times its normal size. Echocardiography can be used to measure both the width and the longitudinal extent of the aneurysm, as well as its relationship to major anatomic landmarks. Additionally, mural thrombi are frequently seen, especially in large aneurysms.

Ascending aortic dissection is a true cardiac surgical emergency that needs to be diagnosed urgently and treated surgically. In aortic dissections, there is a tear in the intima that forms a communication with the aortic true lumen. The media is exposed to blood flow, and a false lumen typically forms. The dissection extends in either an anterograde or a retrograde fashion, often involving the branch arteries.[53] Aortic dissections most commonly originate in one of two locations that experience greatest stress: the ascending aorta just above the sinuses of Valsalva (65%) or the descending aorta just distal to the subclavian artery (20%). Other, less common sites include the aortic arch (10%) and the abdominal aorta (5%).

Echocardiography is an excellent modality for the initial assessment of patients with aortic dissection, particularly those with proximal aortic dissection. TTE has a sensitivity of 77% to 80% and a specificity of 93% to 96% for the diagnosis of proximal aortic

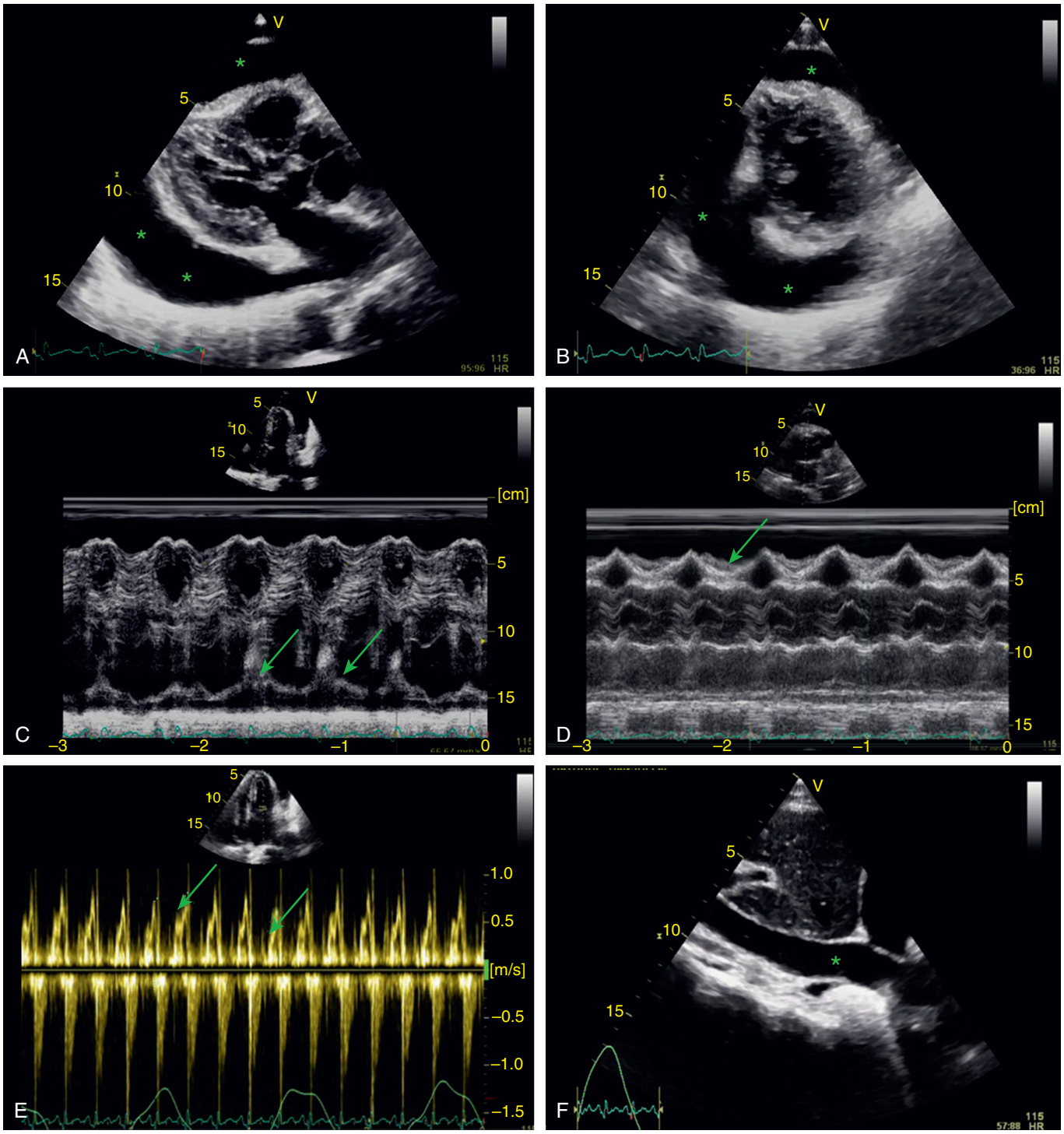

Fig. 2.9 A patient with cardiac tamponade. Parasternal long-axis view (A) and short-axis view (B) show a large circumferential pericardial effusion *(asterisks)*. C, Late diastolic right atrial collapse *(arrows)*. D, Early diastolic right ventricular collapse *(arrow)*. E, Significant respiratory variation in mitral inflow velocities *(arrows)*. F, Dilated inferior vena cava *(asterisk)* suggestive of elevated right heart filling pressure.

dissection, whereas the sensitivity and specificity of TEE in this application are 98% and 95%, respectively.[54,55] The sensitivity of TTE for detecting distal aortic dissection is much lower, for the reasons mentioned earlier, but TEE can diagnose most dissections involving the descending thoracic aorta. TEE can also demonstrate intimal tears in most cases, and it permits recognition of true and false lumina. Additionally, both TTE and TEE are helpful in defining the mechanism of AR if present, in assessing the underlying primary AV pathology if present, and in recognizing pericardial extension of the dissection (Fig. 2.10).

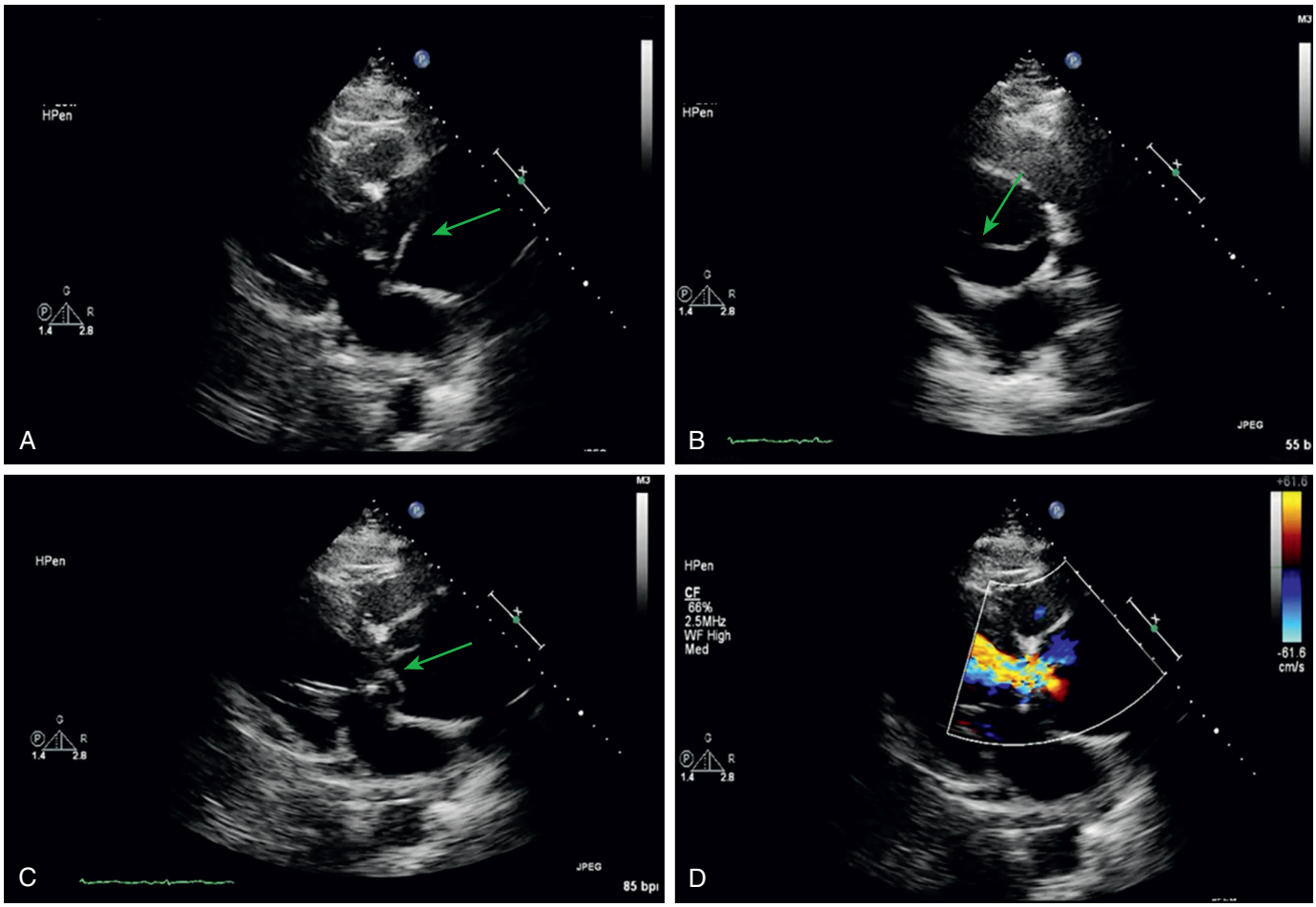

Fig. 2.10 An illustrative example of proximal aortic dissection. Dissection flap *(arrow)* is seen in the ascending aorta in the long-axis view (A) and in the short-axis view (B). The dissection flap is prolapsing though the aortic valve orifice (C, *arrow*), resulting in severe aortic regurgitation (D).

Miscellaneous Disorders

Echocardiography is also useful in providing surgically relevant information about patients undergoing cardiac surgery for various structural lesions such as hypertrophic cardiomyopathy or intracardiac masses. In patients with hypertrophic cardiomyopathy who are undergoing septal myectomy, echocardiography delineates the extent of septal hypertrophy, the site of maximum thickness, the presence of any papillary muscle abnormalities, and the presence, severity, and mechanism of MR, all of which are helpful in planning the surgical procedure.

Stress Echocardiography

Inclusion of a hemodynamic stressor at the time of imaging greatly expands the diagnostic realm of echocardiography. Inducible myocardial ischemia and myocardial viability can be assessed, the patient's symptoms can be corroborated, and the hemodynamic significance of valve lesions can be better assessed if there is ambiguity.

Myocardial Ischemia

Assessment of the presence, extent, and severity of myocardial ischemia is the most common indication for performing stress echocardiography. During stress echocardiography, inducible ischemia is diagnosed by the development of new wall motion abnormalities, which may manifest as delayed thickening, reduced thickening, or no thickening at all.

Patients can be stressed by exercise or by the use of pharmacologic agents.[56] In patients who are physically active, exercise is the preferred modality because it is physiologic, allows symptom correlation, and permits assessment of functional capacity, which itself is a powerful prognostic marker. Exercise is most often done on a treadmill or, less commonly, on a bicycle ergometer.[57] Compared with bicycle ergometry, treadmill exercise typically results in greater workload during the stress test. However, the lower workload achieved during bicycle ergometry is compensated by the ability to image during the exercise itself, so the two modalities have equivalent diagnostic accuracy.[58]

Pharmacologic stress testing is a negative prognosticator in itself because patients who are not able to achieve sufficient physical activity to perform an exercise stress test have a greater incidence of cardiovascular disease and other comorbidities. Pharmacologic stress testing can be performed using either dobutamine, which is a chronotropic/inotropic agent, or a vasodilator such as dipyridamole or adenosine. Atropine is often combined with pharmacologic stress testing to increase the sensitivity of the test.[59–62]

If the patient has an implanted pacemaker and a pacemaker-dependent rhythm, pacing stress echocardiography can be performed using an external programmer to sequentially increase the heart rate. It is usually combined with dobutamine infusion to simultaneously augment contractility.

Regardless of the stress modality, the imaging protocols are almost identical.[56] Representative gray-scale images (typically the parasternal long-axis, midventricular short-axis, apical four-chamber, and apical

two-chamber views) are acquired at baseline and at, or immediately after, peak stress. Bicycle exercise and pharmacologic agents also allow acquisition of low-stress and recovery images. Hemodynamic data (eg, LVFP, TR, MR) are also captured at baseline and at peak stress. The gray-scale images are analyzed for any inducible wall motion abnormalities with the use of a digitized, quad-screen display format to allow side-by-side comparisons.

The accuracy of stress echocardiography for detection of inducible ischemia has been examined in numerous studies. In a large metaanalysis, the average sensitivity and specificity of exercise echocardiography were found to be 83% and 84%, respectively.[63] These values were 80% and 85%, respectively, for dobutamine echocardiography; 71% and 92% for dipyridamole echocardiography; and 68% and 81% for adenosine stress echocardiography.[63] A number of factors that influence the accuracy of stress echocardiography, including the adequacy of stress, delayed imaging after stress, the extent of CAD, the coronary vessel or vessels affected, use of beta-blockers and other antianginal agents, preexisting wall motion abnormalities, previous CABG, the presence of concomitant conduction abnormalities, and the acoustic quality of the gray-scale images. Additionally, the technical expertise of the personnel involved in image acquisition and interpretation greatly influences the diagnostic accuracy of this modality.[56,64,65]

Myocardial Viability

In patients with significant LV systolic dysfunction, the presence of myocardial viability is a good prognostic marker and is associated with greater likelihood of functional recovery after revascularization.[66–70] Accordingly, myocardial viability is often assessed in these patients to determine the need for, and to choose the mode of, revascularization.

Although reduced end-diastolic wall thickness (<6 mm) has been reported to have high negative predictive value for the presence of myocardial viability,[71] the converse is not true. For this reason, dobutamine echocardiography is the primary echocardiographic modality used for the assessment of myocardial viability in clinical practice. The dobutamine infusion is started at a low dose (typically 2.5–5 μg/kg per minute), and the dose is doubled every 3 minutes to a maximum of 40 μg/kg per minute. At low doses (up to 10 μg/kg per minute), dobutamine augments cardiac contractility without any appreciable increase in myocardial oxygen demand. However, when the dose is increased further, there is a progressive increase in heart rate and myocardial oxygen demand. As a result, a dysfunctional but viable segment that is underperfused (ie, a *hibernating* segment) will improve at low doses but worsen again at peak doses. This biphasic response is considered to be the hallmark of myocardial viability and is the most accurate predictor of functional recovery after revascularization.[72] In contrast, when a myocardial segment is dysfunctional but well perfused (i.e., *stunned* myocardium), it will demonstrate a sustained improvement in contractility at both low and peak doses (uniphasic response). Such a segment is likely to improve with time without the need for any revascularization. If there is no improvement in contractility with a low dose, lack of myocardial viability is indicated, and the likelihood of functional recovery is very low. In a metaanalysis comparing various modalities for assessment of myocardial viability, dobutamine echocardiography was reported to have a sensitivity of 84% and a specificity of 81% for predicting functional recovery after revascularization.[73]

An important limitation of stress echocardiography, when used for evaluation of CAD, is the subjectivity of assessment. All interpretation with this modality is based on visual analysis of wall motion. Quantitative techniques such as tissue velocity imaging and strain imaging have been explored to minimize this subjectivity and to improve the diagnostic accuracy of stress echocardiography. The results indicate that the use of tissue velocity and strain as adjuncts to wall motion analysis can indeed enhance the accuracy of stress echocardiography for evaluation of myocardial ischemia and viability.[74–79] Due to several technical limitations, however, the role of these techniques is limited at present.

Valve Lesions

Although valve lesions are typically evaluated at rest only, stress echocardiography may be required in certain situations. For patients who have significant valve disease but are asymptomatic, exercise stress echocardiography can be performed to assess symptoms and to determine overall functional capacity. Development of symptoms at low levels of stress, especially if accompanied by evidence of hemodynamic compromise (eg, fall in blood pressure, significant increase in pulmonary artery systolic pressure), may indicate the need for valve surgery.[33]

A specific indication for stress echocardiography is in the evaluation of low-gradient severe AS in the presence of LV systolic dysfunction.[26] The AS may be truly severe and the gradients spuriously low due to LV systolic dysfunction. Alternatively, the AS may not be severe, with the reduced valve area being a function of inadequate opening force (pseudo-severe AS). Low-dose dobutamine echocardiography (maximum dose, 10–20 μg/kg per minute) can help make this distinction. In the former situation, an increase in LV contractility with dobutamine infusion significantly increases the transaortic gradient without a concomitant increase in valve area. In these cases, LV systolic dysfunction is likely to be secondary to severe AS and can be expected to improve after AV replacement. In contrast, if the AS is not truly severe, dobutamine infusion augments valve opening such that the valve area increases but the gradients do not increase much. In such patients, LV systolic dysfunction is not related to AS, and AV replacement may not be helpful. On occasion, a patient may have fairly advanced LV systolic dysfunction with no contractile reserve, which precludes making the distinction between severe AS and pseudo-severe AS. Patients in this subgroup have high associated surgical mortality rates and poor long-term outcomes, although valve replacement may still improve LV function and outcome in selected individuals.[80]

Stress echocardiography may also have a role in the evaluation of functional MR. An exercise-induced increase in the MR effective regurgitation orifice by greater than 13 mm^2 has been shown to be an independent predictor of mortality and hospital admission for heart failure in patients with chronic ischemic LV systolic dysfunction and functional MR.[81]

Myocardial Nuclear Scintigraphy

Myocardial nuclear scintigraphy is the most widely used modality for assessment of myocardial ischemia and viability, at least in the preoperative setting. There are two main forms of myocardial nuclear imaging, single-photon emission computed tomography (SPECT) and positron emission tomography (PET). Both use the principles of radioactive decay to evaluate the myocardium and its blood supply.

The radionuclides that are used in SPECT are technetium 99m (^{99m}Tc) and thallium 201 (^{201}Tl). ^{99m}Tc is a large radionuclide that emits a single photon or gamma ray per radioactive decay event, with a half-life of 6 hours. The energy of the emitted photon is 140,000 electron volts (140 keV). ^{201}Tl is less commonly used and decays by electron capture. It has a much longer half-life (73 hours), and the energy emitted is between 69 and 83 keV. To obtain images, the gamma rays that are released from the body must be captured and modified by a detector (gamma camera). The standard camera is composed of a collimator, scintillating crystals, and photomultiplier tubes. When a radionuclide emits gamma rays, it does so in all directions. A collimator made of lead that has small, elongated holes is used as a filter to accept only those gamma rays traveling from the target organ toward the camera. Once the selected gamma rays have reached the scintillating crystals, they are converted to visible light and then to electrical signals by the photomultiplier tubes. These signals are processed by a computer to form images. Myocardial regions that are infarcted or ischemic after stress have relatively decreased tracer uptake and therefore decreased signal (counts) in the processed images.

Although PET also uses radioisotopes to produce images, the actual process of image formation is quite distinct from that of SPECT. The most common radioisotopes used for cardiac evaluation by PET

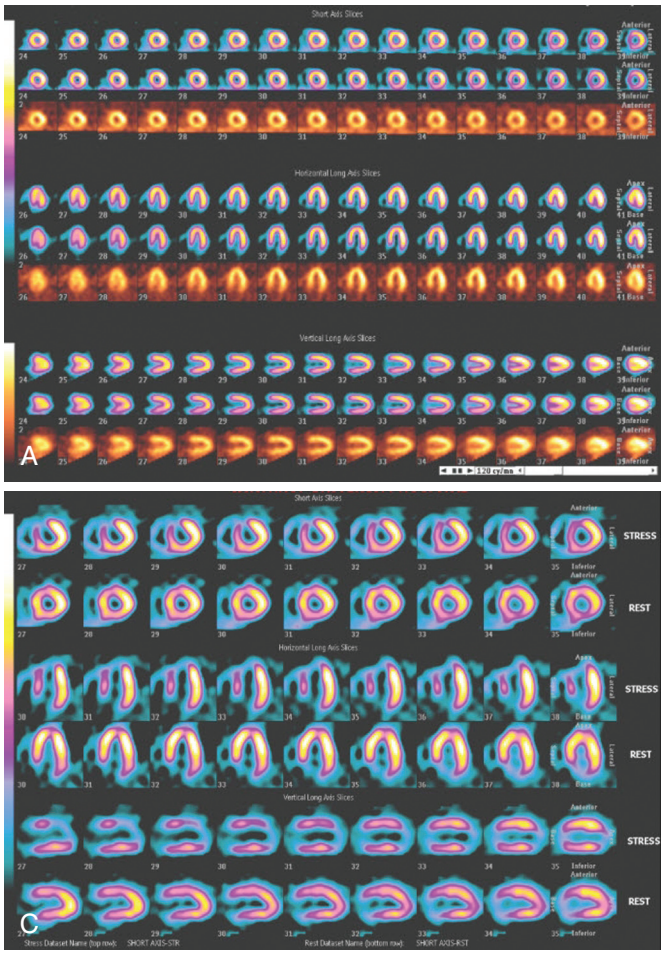

Fig. 2.11 Technetium 99m sestamibi stress myocardial perfusion images demonstrate normal left ventricular size and perfusion (A), apical and anteroapical infarct (B), and moderate-to-severe ischemia involving the apical, septal, anterior, and anteroseptal walls (C).

imaging are rubidium 82, *N*-ammonia 13, and fluorine 18 (^{18}F). ^{18}F is a much smaller radionuclide than ^{99m}Tc. It emits a positron (β^+) antiparticle. This ionized antiparticle travels until it interacts with an electron. The electron and the positron are antiparticles of each other, meaning that they have the same mass but are opposite in charge. When they encounter each other, both particles disintegrate and are converted into energy in the form of two photons, each with the same energy (511 keV), traveling in opposite directions. This phenomenon, known as *pair annihilation*, is used to create the images in PET. PET cameras also differ from SPECT cameras in that they capture only incoming photons that travel in opposite directions and arrive at a circular detector around the body at precisely the same time. PET detectors have much higher sensitivity than SPECT cameras because they do not require a collimator. As in SPECT, PET cameras use scintillating crystals and photomultiplier tubes. PET systems have been combined with CT and magnetic resonance imaging (MRI) systems to simultaneously display PET metabolic images and the corresponding anatomic information.

Detection of myocardial ischemia is the most common indication for performing myocardial perfusion imaging. Either SPECT or PET can be used for this purpose, which is based on assessments of LV myocardial uptake of the radioisotope at rest and after stress. Myocardial uptake is reduced after stress in myocardial regions where significant coronary artery stenosis is present. ^{99m}Tc is the radioisotope most commonly used for this purpose. It must be injected when peak heart rate is reached, and the patient must continue exercising (or the pharmacologic stress must be continued) for at least 1 minute afterward to allow sufficient time for the radioisotope to circulate through the myocardium. The images are displayed in three LV orientations (short-axis, horizontal long-axis, and vertical long-axis) for proper LV wall-segment analysis. The stress images are exhibited directly above the corresponding rest images so that normal myocardium can be differentiated from ischemic myocardium (Fig. 2.11).

PET scanners have inherently less attenuation and higher resolution than those used in SPECT, making PET the more desirable modality.[82] Pharmacologic stressors are usually used in PET myocardial perfusion tests because of the very short half-life of PET radioisotopes. The sensitivity and specificity of SPECT for detection of obstructive CAD are 91% and 72%, respectively; use of PET improves the specificity to 90%.[82] Normal findings on SPECT imply that the probability of experiencing a cardiac event is less than 1% per year, and a normal result on rubidium 82 (^{82}Rb) PET indicates a probability of less than 0.4% per year.

For viability assessment with SPECT imaging, ^{201}Tl is used more frequently, taking advantage of this isotope's long half-life (73 hours). Thallium uptake is dependent on several physiologic factors, including blood flow and sarcolemmal intercellular integrity. The time required for uptake is short in normal myocardium, but 24 hours may be needed in hibernating myocardium that still has metabolic activity. Patients are injected with thallium radioisotope and imaged the same day for baseline studies. After 24 hours, new images are obtained without any further injection. The baseline images and 24-hour images are compared. Defects that are present at baseline but filled in at 24 hours represent viability (Fig. 2.12). Technetium radioisotopes also can be used under different protocols for the evaluation of viable myocardium.

PET imaging is more sensitive than SPECT, and it is considered by many experts to be the gold standard for assessment of viability. PET has the ability to identify the presence of preserved metabolic activity in areas of decreased perfusion when 18-fluorodeoxyglucose (FDG) is used. PET imaging uses both FDG and either rubidium or

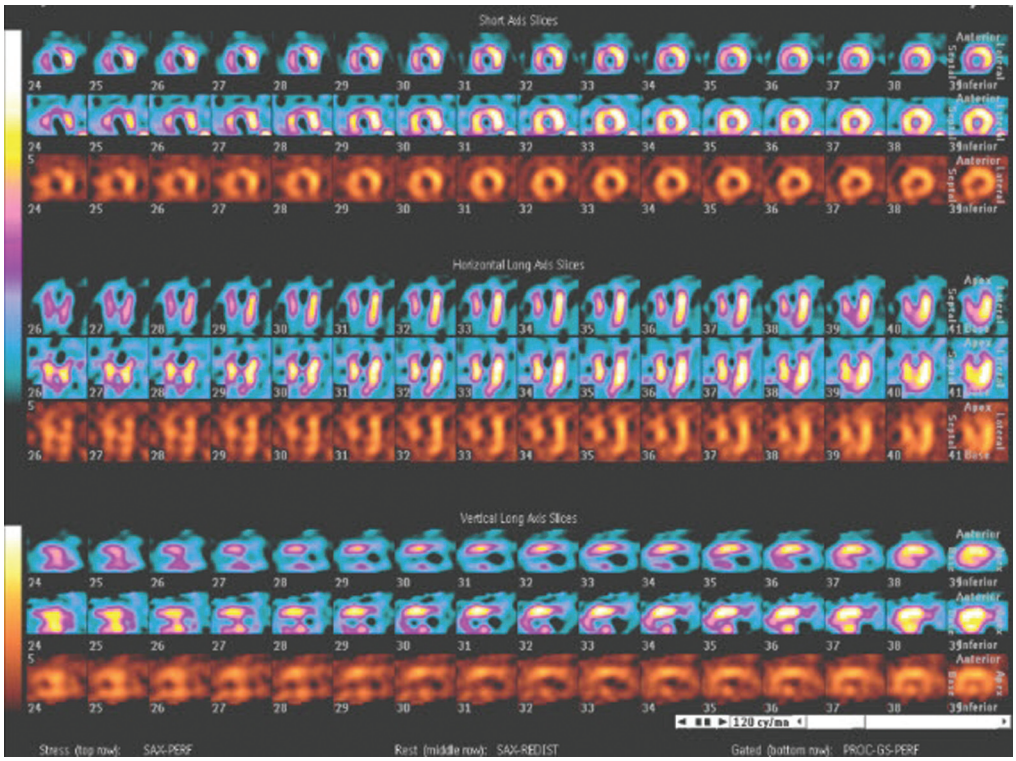

Fig. 2.12 Thallium rest-redistribution scan demonstrates hibernating myocardium involving the apical-basal anteroseptum, midbasal inferior, midbasal inferoseptum, and midbasal inferolateral wall segments. There is infarction of the apex, inferoapical, and apical-lateral wall segments.

ammonia radioisotopes for quantification of energy utilization by the myocardium and for evaluation of patterns of blood flow. Areas with reduced blood flow and reduced FDG uptake are considered to represent scar and infarction. Areas with reduced blood flow and normal FDG uptake are considered viable.[83] A metaanalysis of more than 750 patients demonstrated a sensitivity of 92% and a specificity of 63% for regional functional recovery with this method; the positive and negative predictive values were 74% and 87%, respectively.[84]

Nuclear scintigraphic methods, including both SPECT and PET myocardial perfusion imaging, can also be used to evaluate global and segmental LV systolic function. This is achieved by implementing ECG gating during data acquisition. Most often, eight frames or phases are acquired per cardiac cycle. Gated images can be acquired both at rest and after stress. However, rest images typically involve a lower radiation dose, so the images may be noisy; for this reason, most institutions perform gated imaging on the post-stress images. Accurate LV systolic analysis is limited with this technique in the circumstance of stress-induced ischemia, in which myocardial stunning can transiently reduce the LVEF. Another limitation of ECG-gated SPECT or PET is arrhythmias, specifically frequent premature ventricular contractions or atrial fibrillation. Assessment of LV function also may be inaccurate in patients with extensive myocardial infarction: Because there is absence of isotope in the scar regions, the endocardial border cannot be defined.

An alternative technique is gated blood pool scanning (also known as multiple-gated acquisition, or MUGA). In this technique, the cardiac blood pool is imaged with high resolution during the cardiac cycle. Ventricular function and various temporal parameters can be measured.[85] There is good correlation between echocardiography and MUGA for the evaluation of LVEF, and in fact, MUGA has demonstrated better intraobserver and interobserver reproducibility compared with echocardiography.[86] Nuclear imaging can also be used for assessment of LV diastolic function, but it is much less useful than echocardiography for that purpose. At least 16 phases of the cardiac cycle need to be acquired to evaluate diastolic dysfunction using SPECT. The two main parameters that can be measured by SPECT are LV peak filling rate and time to peak filling rate.

Myocardial infarction causes denervation of the scar, and subsequent interruption of sympathetic nerves induces denervation of adjacent viable myocardium.[87,88] Sympathetic nerves are very sensitive to ischemia and usually become dysfunctional after repeated episodes of ischemia that do not result in irreversible myocyte injury.[89,90] Matsunari and associates[91] demonstrated that the area of denervation is larger than the area of scar and corresponds to the area at risk for ischemia. Additionally, Bulow and coworkers[92] showed that denervation of myocytes can occur in the absence of previous infarction. Myocyte sympathetic innervation can be measured by PET using carbon 11 (^{11}C)-labeled hydroxyephedrine (HED). This is compared with PET resting perfusion images to determine the area of the scar. Areas of normal resting perfusion and reduced HED retention indicate viable myocardium.

SPECT imaging of myocardial uptake of iodine 123 (^{123}I)-labeled metaiodobenzylguanidine (*m*IBG), which is an analog of the sympathetic neurotransmitter norepinephrine, provides an assessment of β-receptor density. Reduced ^{123}I-*m*IBG uptake is associated with adverse outcomes in patients with heart failure and has been proposed as a marker of response to treatment.[93]

Cardiac Computed Tomography

CCT has grown significantly in clinical use since the advent, in the early 2000s, of multidetector CT (MDCT) scanners with submillimeter resolution that allow evaluation of the coronary anatomy. The x-ray tube produces beams that traverse the patient and are received by a detector array on the opposite side of the scanner. The tube and detector arrays are coupled to each other and rotate around the patient at a velocity of 250 to 500 microseconds per rotation. In 1999, the first MDCT scanner used for coronary imaging had four rows of

detectors and a scanning coverage of 2 cm per rotation. Breath-holds on the order of 10 to 20 seconds were required to cover the entire heart. Artifacts produced by the patient's respiration and heart rate variability rendered many studies nondiagnostic for the assessment of coronary stenosis. With today's advanced technology, 256-slice systems are standard, and 320-slice systems with 16 cm of coverage are able to capture the entire heart in one heartbeat and rotation.

CCT uses ionizing radiation for the production of images. Concern about excessive medical radiation exposure has been raised. Although several techniques, such as prospective ECG-gated acquisition, may be implemented to reduce the radiation dose,[94–96] a risk-benefit assessment must be done for the selection of patients who have appropriate indications for CCT.

Coronary angiography is currently one of the most common indications for performing CCT. The patient's heart rate must be lowered to less than 65 beats/minute to achieve adequate results when imaging the coronary arteries. This usually requires the administration of oral or intravenous beta-blockers. After the scan has been completed, images are reconstructed at various points of the cardiac cycle and analyzed on a computer workstation. Cardiac CT angiography (CCTA) has been well studied for the diagnosis of CAD in patients without known ischemic heart disease, demonstrating a sensitivity of 94% and a negative predictive value of 99%.[97] It is being increasingly employed to exclude obstructive CAD before valve surgery in patients with low-to-intermediate pretest probability to avoid invasive testing. In one study of CCTA for this purpose, researchers performed conventional coronary angiography and 64-slice MDCT in 50 patients (mean age, 54 y) who were about to undergo valve replacement for AR. CCTA demonstrated a sensitivity of 100%, a specificity of 95%, and a negative predictive value of 100%. Additionally, it was determined that use of CCTA could have allowed 70% of the patients to avoid invasive catheterization.[98] Two further studies used preoperative 16- and 64-slice CCTA in patients with AS. The mean ages of the patients were 68 and 70 years, respectively. In each study, both the sensitivity and the negative predictive value were 100% for detection of significant stenosis.[99,100] These studies showed that preoperative coronary evaluation with CCTA is safe and accurate. It is important that only patients with no known CAD or those with low-to-intermediate risk be referred for CCTA. In general, patients with degenerative AS are older and have greater risk for CAD. Patients who undergo valve surgery for MR because of MV prolapse are usually younger and are excellent candidates for CCTA.

CT aortography is the imaging modality of choice for evaluation of aortic pathologies such as aortic aneurysms and nonemergency dissections. Imaging protocols used for evaluation of the aorta are similar to those used for CCTA. It is important to have the scan gated to the patient's ECG because the ascending aorta moves significantly during the cardiac cycle. Nongated scans have inherent motion artifacts that can be confused with a dissection. Use of prospective ECG gating also minimizes radiation exposure. Visualization of the aortic root and coronary arteries is crucial because ascending aortic dissections can involve the ostia of the coronary arteries.

Once the images are acquired by specialized CT workstations, the aorta is evaluated and measured. The aorta is lined up in multiple orthogonal views to measure the true short axis throughout the length of the aorta. The excellent spatial and contrast resolution of this modality is useful for the evaluation of dissection. Entry points of dissection, intimal flap location, false lumen, extension into branch arteries, and the abdominal aortic circulation are easily visualized.

Although echocardiography is the gold standard for imaging valvular disease, advanced imaging with CCT or CMR may be required if TTE and TEE are technically difficult or if there are discrepancies in the findings. CMR offers more functional data than CCT, but CCT may be used if further anatomic information about a valve is required. For evaluating prosthetic valves, CCT is usually superior to CMR because of metallic artifact from the valve, which is visualized on CMR (see Chapter 21).

Valvular calcification is common in patients presenting with AS, particularly senile degenerative AS. CCT can accurately quantify AV calcification and can help assess the severity of AS. An AV calcium score of 1100 or greater has 93% sensitivity and an 82% specificity for severe AS.[101] Additionally, contrast-enhanced CCT allows for excellent visualization of the AV morphology and accurately differentiates between bicuspid and tricuspid AVs[102] (Fig. 2.13). Aortic valve planimetry can also be performed. AV areas measured by CCT have been demonstrated to have a strong correlation with valve areas and transvalvular gradients obtained by echocardiography.[103–107]

CCT also can be used for the evaluation of AR. CCT can elucidate the potential mechanism of AR, including inadequate leaflet coaptation during diastole, leaflet prolapse, cusp perforation, and interposition of an intimal flap in cases of type A aortic dissection. Regurgitant orifice areas measured by CCT have an excellent correlation with AR severity parameters, including vena contracta width and the regurgitant jet/LVOT height ratio obtained by TTE.[108,109]

In patients with MS, ECG-gated, contrast-enhanced CCT can permit assessment of MV morphology in a manner analogous to echocardiography.[110,111] Calcium scoring of the MV is also possible but has lower reproducibility than for the AV.[112] The degree of MV calcification correlates significantly with the severity of MS seen on TTE.[113] MV areas obtained by planimetry also correlate significantly with MS data obtained by TTE.[114]

CCT can accurately diagnose MV prolapse by evaluating cine loops of the MV, and the exact scallops that are prolapsing can be identified. Severity of MR can be assessed by planimetry of the regurgitant orifice, which has been shown to correlate with TEE.[115] Additionally, the presence of calcification of the MV annulus and leaflets can help determine whether the valve can be repaired or needs to be replaced.

CCT can also be used to evaluate tricuspid and pulmonary valve (PV) pathologies. For evaluation of the PV, CCT may be superior to echocardiography; adequate visualization of the PV on echocardiography is usually difficult.

CCT is an excellent modality for the evaluation of PHVs. It has the ability to clearly depict the mechanical prosthesis and to detect any abnormality, including valve thrombosis. This is done by using retrospective scanning and acquiring the entire cardiac cycle to play cine loops and visualize the leaflets through systole and diastole. The mechanical valves that are used currently consist of two disks that open symmetrically (Fig. 2.14). In one study, the valve function of the two-disk prosthesis, including opening and closing angles, was evaluated by CCT and compared with findings from fluoroscopy and echocardiography. CCT correlated significantly with both echocardiographic imaging modalities for these valves.[116] The role of CT in the assessment of bioprosthetic valves is similar because the metallic artifacts often preclude adequate assessment by echocardiography.

CCT is excellent for the diagnosis of paravalvular abscesses. The abscesses appear as paravalvular fluid-filled collections on CCT and are imaged by acquiring a delayed scan approximately 1 minute after contrast is administered. The contrast agent is retained within the abscess even after it washes out of the circulation[117] (Fig. 2.15). A study comparing MDCT with intraoperative TEE for the detection of suspected infective endocarditis and abscesses demonstrated excellent correlation. CCT correctly identified 96% of patients with valvular vegetations and 100% of patients with abscess. Additionally, CCT performed better than TEE in the characterization of abscesses.[118]

CCT, with its excellent spatial and temporal resolution, can also facilitate an accurate assessment of LV function.[119–121] CCT uses actual 3D volumes to calculate LV systolic function. Retrospective scanning is used for functional analysis because the entire cardiac cycle (both systole and diastole) must be acquired. The raw data set is then reconstructed in cardiac phase intervals of 10%, from 0% (early systole) to 90% (late diastole), to derive functional information. Advanced computer workstations allow cine images to be reconstructed and displayed in multiple planes. Segmental wall motion analysis can also be performed using the 17-segment model recommended by the American