Contemporary Surgical Clerkships
Series Editor: Adam E. M. Eltorai

Jordan P. Bloom
Thoralf M. Sundt *Editors*

Cardiac Surgery Clerkship

A Guide for Senior Medical Students

Springer

Contemporary Surgical Clerkships

Series Editor
Adam E. M. Eltorai
Marlborough, MA, USA

This series of specialty-specific books will serve as high-yield, quick-reference reviews specifically for the numerous third- and fourth-year medical students rotating on surgical clerkships. Edited by experts in the field, each book includes concise review content from a senior resident or fellow and an established academic physician. Students can read the text from cover to cover to gain a general foundation of knowledge that can be built upon when they begin their rotation, or they can use specific chapters to review a subspecialty before starting a new rotation or seeing a patient with a subspecialty attending.

These books will be the ideal, on-the-spot references for medical students and practitioners seeking fast facts on diagnosis and management. Their bullet-pointed format, including user-friendly figures, tables and algorithms, make them the perfect quick-reference. Their content breadth covers the most commonly encountered problems in practice, focusing on the fundamental principles of diagnosis and management. Carry them in your white coat for convenient access to the answers you need, when you need them.

Jordan P. Bloom • Thoralf M. Sundt
Editors

Cardiac Surgery Clerkship

A Guide for Senior Medical Students

Springer

Editors
Jordan P. Bloom
Cardiac Surgery
Massachusetts General Hospital
Boston, MA, USA

Thoralf M. Sundt
Cardiac Surgery
Mass General Hospital
Boston, MA, USA

ISSN 2730-941X ISSN 2730-9428 (electronic)
Contemporary Surgical Clerkships
ISBN 978-3-031-41300-1 ISBN 978-3-031-41301-8 (eBook)
https://doi.org/10.1007/978-3-031-41301-8

This Springer imprint is published by the registered company Springer Nature Switzerland AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

Paper in this product is recyclable.

To our teachers, who contributed so much to the successes of today, and to the students, who will define the successes of tomorrow.

Editors Addenda

Chapter 1—Cardiac Surgery History

Surgeons are often teased about their fondness for history, but you will likely have a receptive audience to comments or questions that reflect your awareness of the historical roots of any surgical specialty. Perhaps the most remarkable fact about cardiac surgery is the incredible tempo of advancement, particularly in the last 50 years. After perhaps a slow start at the turn of the last Century, the introduction of mechanical cardiopulmonary support transformed the field making it a hotbed for innovation and life-saving therapy. Drs. Miller and Cameron have provided you with a succinct summary of important individuals and the milestones they achieved. It is worth taking a moment to review where the field has been as you consider where it is going. And if nothing else you may be able to inspire personal reflections or even some inside stories from the cardiac surgery staff.

Chapter 2—Surgical Anatomy of the Heart

Surgeons are of course applied anatomists. Cardiac anatomy is complex and compact; you will struggle to understand the procedures being performed if you do not have a basic understanding of that anatomy and you will be expected to have foundational knowledge of the relevant anatomy when you enter the operating room. And as is so often the case, the more you know, the more you will learn. Surgeons love to demonstrate advanced anatomy to students that demonstrate initiative and have educated themselves on the basics. Conversely, ignorance of that foundational knowledge is unlikely to play well. Seeing and touching the beating heart is an experience every medical student should have.

Chapter 3—Preoperative Evaluation and Risk Assessment

Preoperative evaluation of cardiac surgical patients and risk assessment has evolved from a simple "foot of the bed test" as to a patient's general fitness, to a sophisticated, data-driven science as summarized by Drs. Calle and Shahian. The evolution of a specialty-specific clinical database by the Society of Thoracic Surgeons has enabled the development of a sophisticated tool to calculate

patient-specific risks for common procedures to a remarkably high degree of accuracy. This risk prediction tool is the envy of many other specialties in medicine and should be of interest to any physician as a model both of what can be accomplished and what is required to get there. Whatever your specific clinical interest, consider whether or not similar tools exist and whether or not you could play a role in their development. There is a great deal to be explored as well in comparing risk models developed using administrative versus clinical databases, the strengths and weaknesses, pitfalls and promise.

Chapter 4—Echocardiography

Perhaps more than any other surgical specialty, cardiac surgery is characterized by remarkable overlap and codependence if you will on our medical and anesthesia colleagues. Nowhere is this more apparent than in the application of echocardiographic technology in preoperative diagnosis, intraoperative assessment, and postoperative care. Drs. Secor and Yucel have laid out a succinct summary of the echocardiographic evaluation of the cardiac surgical patient. It is increasingly critical in our specialty that all members of the team understand this imaging modality. This will certainly be true and all medical subspecialties as advanced imaging plays a role in patient care. We can see this as ultrasound devices become smaller and more portable, with application by practitioners everywhere from endocrine surgeons doing outpatient thyroid aspirations to emergency room FAST scans. For all of us a basic knowledge and understanding of this technology is critical to the modern physician.

Chapter 5—Cardiac CT and MRI

As patient's have become increasingly more complex and more comorbid, we have become more dependent on axial imaging to plan and execute cardiac surgical operations. Moreover, as these axial imaging techniques have become more advanced they often obviate the need for invasive procedures such as heart catheterization. Drs. Parakh, Baliyan, and Hedgire have written a very useful summary of the different imaging types including their indications and limitations. A fundamental understanding of these imaging modalities is important regardless of your ultimate specialty as you inevitably will be ordering these tests on your patients.

Chapter 6—Coronary Angiography

Coronary artery bypass grafting is the most commonly performed heart operation in the modern era and coronary angiography remains the cornerstone for planning the operation. While technology certainly has added useful adjuncts to the interpretation of these studies, there is nothing they can replace sitting down and looking at the imaging. Drs. O'Kelly and Patel have written a very useful summary of basic coronary angiography as well as newer physiologic assessment tools, endovascular imaging modalities, and right heart catheterization. Given the prevalence of ischemic heart disease in the world, the information in this chapter is pertinent to anybody practicing medicine.

Chapter 7—Cardiac Anesthesia

Cardiac anesthesiologists have evolved as a recognized subspecialty of anesthesia over the years and now play a critical role on the modern heart team. Performing complex operations on comorbid patients would not be possible without their expertise intraoperatively as well as postoperatively. This chapter, written by Drs. Dalia and Convissar, represent a nice summary of physiology, pharmacology, and monitoring in patients undergoing heart operations from the perspective of the anesthesiologist (behind the ether screen). Their insights into the physiology of patients with corrected—and uncorrected—cardiovascular disease is pertinent to anybody interested in critical care.

Chapter 8—Transfusion and Blood Management

Management of blood product transfusions in both the intra- and post-operative setting is critically important to a successful and high-quality cardiac surgical program. Blood products are unquestionably life-saving in some circumstances. Their profligate use, however, can actually be harmful with risks extending beyond transfusion-related infectious conditions to long-term impacts on immune reactivity and, potentially, cancer risk. The literature concerning appropriate transfusion practice is rich and in many instances conflicting, and institutional biases and culture may be strong. Drs. He and Berical have written a comprehensive summary of the salient points with respect to blood product management including a discussion of anticoagulants and platelet inhibitors. Moreover, there is a discussion on thromboelastography which is rapidly gaining popularity as a tool for targeted blood product usage.

Chapter 9—Cardiopulmonary Bypass

Development of cardiopulmonary bypass has been the most important technological advancement facilitating heart surgery. Many inventors explored mechanical circulatory support devices, but it was Dr. John Gibbon and his wife Mary developed the heart-lung machine that served as the predecessor to the pumps we use today. He was the first to use the pump when in 1953 he successfully repaired a large atrial septal defect. In the years since there have been major advances in the technology making it more biocompatible and safer.. Those who operate the machine, cardiac perfusionists, are critically important members of the heart team throughout all phases of care. Dr. Marso and Mr. Shann have summarized the key considerations with respect to the heart-lung machine and its utility.

Chapter 10—Myocardial Protection

As discussed in the last chapter, cardiopulmonary bypass has enabled us to “take the heart off-line” and do its work to support the body while we address pathology; however, sophisticated repair of complex cardiac pathology often require us to stop the heart and open chambers. Virtually all surgeons today use the cardioplegia solutions discussed in this chapter by Drs. Potz and del Nido to minimize myocardial oxygen consumption to protect cardiomyocytes while blood flow to the heart itself is interrupted. This chapter not only provides an excellent summary of cardioplegia but also reminds us the importance of cellular biology and translational research to the practice of cardiac surgery.

Chapter 11—Management of CAD

Given the prevalence of cardiovascular disease, there are few subjects more important to all disciplines than knowledge of coronary artery disease. Moreover, it seems you cannot pick up a medical journal without finding something related to either goal-directed medical therapy, interventional or surgical management of coronary disease. To date, for most patients, a coronary bypass still remains the most effective treatment strategy to mitigate symptoms and prolong survival. This operation is truly the backbone of cardiac surgery and is in no threat of being replaced by percutaneous interventions anytime in the near future. Given the likelihood that you will encounter patients either in need of or having undergone coronary artery bypass grafting, this is a fundamental chapter for any healthcare professional.

Chapter 12—Mechanical Complications of MI

Mechanical complications of myocardial infarction remain highly morbid and represent surgical emergencies. . Thanks to the advances in rapid percutaneous intervention for coronary artery disease we see fewer and fewer of these complications Advances in temporary mechanical circulatory support to improve end-organ perfusion and provide resuscitation prior to high-risk intervention represent the most recent innovations in this space. Still surgical intervention is the cornerstone of therapy.

Chapter 13—Aortic Valve Repair and Replacement

Aortic valve disease is increasing in prevalence as the population ages, making intervention whether transcatheter or via open surgery an increasing fraction of the procedures performed by cardiac surgeons, and equally an increasingly frequent comorbidity for patients in any physician's practice. While surgical aortic valve replacement has become increasingly safe, with predicted operative risk as low as 0.5% in many patients, prostheses are imperfect and their implantation substitutes prosthetic valve disease for native valve disease. Accordingly, interest is great in durable approaches to valve repair. Repair of mitral regurgitation has become the standard of care whenever possible and attention is now turning to repair of the aortic valve. In the modern era, it has become increasingly important for surgeons to understand how to repair the aortic valve and/or perform operations that result in very low gradients to avoid patient prosthesis mismatch and subsequent associated mortality. This is of particular importance in young patients and/or patients with small aortic annuli.

Chapter 14—Mitral Valve Repair and Replacement

There has been a tremendous evolution in the management of mitral valve regurgitation. With the demonstration of superiority both in perioperative risk and long-term outcome of repair over replacement for degenerative disease, mitral repair has become the gold standard. Mitral stenosis, secondary either to calcific disease or rheumatic disease, is more vexing and accordingly stenosis continues to be managed with replacement. Drs. Leya and Melnitchouk have written a comprehensive chapter that succinctly summarizes the key considerations in mitral valve anatomy, physiology, and therapies. Given its complexity, mitral valve surgery can be

humbling and has a demonstrable volume-outcome relationship. To this extent it remains as much an art as science.

Chapter 15—Tricuspid Valve Repair and Replacement

The tricuspid valve has been often referred to as the "forgotten valve." In years past surgeons paid little attention to it, focused on the aortic and mitral while cardiologists were unclear as to the associated symptoms and long-term consequences of disease .Isolated tricuspid valve disease is fairly rare and something we see from endocarditis or congenital abnormalities while much more commonly we encounter secondary tricuspid valve disease associated with other pathologies such as coronary artery disease or other valvular disease. Knowledge about who to operate on and when to intervene on the tricuspid valve whether in isolation or as a concomitant intervention continues to be challenging. Drs. Griffeth and Dearani have laid out an excellent summary of the key considerations for addressing the tricuspid valve.

Chapter 16—Pulmonary Valve Repair and Replacement

The pulmonary valve is the last thing on most adult cardiac surgeon's minds. It is not supported by the cardiac fibrous skeleton and is quite difficult to effectively image. Comfort with the pulmonary valve is typically reserved for congenital heart surgeons, as many diseases necessitating intervention are associated with congenital abnormalities. The Ross procedure (pulmonary autograft) uses the pulmonary valve as a replacement conduit for a diseased aortic valve. Given a recent resurgence in this procedure, more adult cardiac surgeons are seeking experience with the pulmonary valve.

Chapter 17—Transcatheter Therapies for Structural Heart Disease

Transcatheter therapies for structural heart disease likely represent the single greatest advancement in cardiac intervention since the advent of coronary stents. The rate of technical advancement as well as dispersal of utilization has been staggering. The modern cardiac surgeon must be well versed in the indications as well as technical execution of transcatheter therapies.

Chapter 18—Management of Endocarditis

While percutaneous approaches may lead to alternative interventions in stenotic or regurgitant valve disease, it is likely that infectious problems will remain in the domain of surgeons. Infective endocarditis continues to be a challenge with heterogeneous presentations that can be easily missed for extended periods of time. It is one condition for which the history and physical exam remain pivotal. Any patient with a fever of unknown origin or persistent malaise may have endocarditis and thus should undergo diagnostic evaluation. Surgical management, when indicated, can be challenging since the intraoperative anatomic findings are not completely predictable and "game time" decisions must often be made. It is surely a realm in which experience matters. . A particularly complex topic is the management of patients with endocarditis among people who inject drugs. Their care should be done in an organized multidisciplinary fashion to give patients the best chance of a durable outcome.

Chapter 19—Aneurysmal Disease of the Ascending Aorta, Root, and Arch

Aneurysms in the aorta are sometimes referred to as "silent killers" and as such all medical professionals should be aware of their appropriate care. Acute aortic syndromes, names as such in contrast to acute coronary syndromes, are those entities that present as emergencies and most often require immediate intervention to save a life. Prophylactic operations on the segments of the aorta are typically designed to prevent progression to an acute aortic syndrome. As described in the chapter, aortic root aneurysms may cause aortic insufficiency and when appropriate, can be replaced with valve-sparing approaches.

Chapter 20—Descending Thoracic and Thoracoabdominal Aortic Aneurysms

This segment of the aorta represents a region of overlap in interest and expertise among cardiac and vascular surgeons. As such the modern cardiac surgeon needs to have an understanding of minimally invasive and endovascular options to treat a variety of pathologies. The management of descending thoracic and thoracoabdominal aortic aneurysm disease exemplifies the importance of this understanding and is a perfect opportunity for collaboration with vascular surgical colleagues. Taking care of these patients requires a broad multidisciplinary team well versed in the pre-, intra-, and post-operative management of the physiology and expected issues after surgical intervention.

Chapter 21—Acute Aortic Syndromes

Acute aortic syndromes are one of the most common causes of sudden death and must not be missed when patients present with appropriate symptoms. Irrespective of medical specialty, particularly in the setting of the emergency room, providers need to be able to recognize patients at risk of these syndromes to ensure appropriate diagnostics and expedited triage. While the specific management of these syndromes has some nuance, it is fair to assume that any patient with acute aortic pathology proximal to the left subclavian is a surgical emergency. Permissive hypotension and expedited transfer to a center capable of treating these patients have the potential to save lives.

Chapter 22—Aortic Trauma

Similar to the previous chapter on acute aortic syndromes, aortic trauma is something that while fairly uncommon, cannot be missed. Patients who have injury patterns that increase the probability of aortic injury must undergo appropriate imaging studies. Management of these injuries has undergone evolution in recent years, and guideline-based care should be offered to these patients. This may involve the transfer of care to an institution with both the endovascular and open expertise necessary to appropriately care for patients with aortic injury.

Chapter 23—Acute and Chronic Pulmonary Embolism

Chest pain and shortness of breath are among the most common reasons for presentation to the emergency department. The differential diagnosis for either always includes pulmonary embolism and thus it is imperative that all doctors have some

basic understanding of the common presentation, diagnostics, and management. This chapter includes the key issues related to both the acute and chronic pulmonary embolic disease. A patient with circulatory collapse from an acute PE should be considered for some sort of interventional therapy. While the mainstay of this has historically been surgery, newer catheter-based therapeutics have largely obviated the need for open surgical intervention in most patients. Patients with CTEPH are more complex and usually need multidisciplinary expertise to determine who will most benefit from intervention.

Chapter 24—Surgery for Atrial Fibrillation

Afib is an incredibly common diagnosis and no matter what type of medicine you practice, it will benefit you to have some knowledge about the management of these patients. Issues include anticoagulation, rate control, rhythm control, and intervention to cure. Anticoagulation for AF is largely based on stroke risk and the necessity for this is based on the CHAD scoring system. Pharmacologic management remains the most common approach, but intervention either catheter-based or surgically should always be kept in mind as an option. Surgery for AF has largely been pioneered by a single surgeon, Dr. James Cox, who has dedicated his life to treating this disease. Large high-quality studies continue to reaffirm the importance of surgical ablation and surgical occlusion of the left atrial appendage in patients undergoing cardiac surgery.

Chapter 25—Pericardial Disease

Pericardial disease is uncommon, but it is important as it is so often treatable or even curable. Pericardial diseases encompass a variety of pathologic diagnoses ranging from a single episode of acute inflammation to recurrent relapsing pericarditis to chronic constriction leading to heart failure. The most complex of these is constriction associated with prior mediastinal radiation in which a distinction must be made between constrictive physiology and restrictive cardiomyopathy. . The nuances to differentiating these processes are challenging and can remain so even with the best diagnostics. A basic understanding of these diseases is critical to cardiologists and cardiac surgeons and when indicated patients can have an amazing improvement in quality of life after intervention.

Chapter 26—Cardiac Neoplasms

Neoplasms can form anywhere in the body, and the heart is no exception. While somewhat rare, these are most commonly metastases from other primary tumors. The management principles are similar to that of other oncologic processes in that resection is largely based on curability or symptom management. One important consideration is the potential for a cardioembolic complication which is based on the location of the tumor. For example, tumors on the left side of the heart are more likely to require intervention than tumors on the right side of the heart. Finally, since many cardiac tumors are not accessible for biopsy, surgery may be indicated for diagnostic purposes, treatment planning, and prognosis.

Chapter 27—Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy tends to get the most attention when young athletes experience sudden cardiac death but is in fact not an uncommon condition with diagnosis usually in the fifth decade of life. When obstructive and symptomatic, surgery is the cornerstone of care. Septal myectomy is essentially curative although performed in only a small number of centers in large numbers. The role of a sonographer who is also well-versed in the disease process cannot be understated.

Chapter 28—Temporary Mechanical Circulatory Support

Temporary mechanical circulatory support has revolutionized the care of patients both pre- and post-cardiac surgery. These devices have made it possible to perform lifesaving procedures with the assistance of circulatory support which has significantly expanded the eligibility of high-risk patients. In addition, these devices have made it possible to provide temporary support in patients who need to recover from end-organ failure or other infectious insults. Finally, these devices can provide an invaluable bridge to decision or bridge to transplant in eligible patients. The bottom line is that MCS often gives the gift of time which is invaluable when taking care of sick patients. As devices are simplified for insertion and operation, one can expect their use to expand beyond her current footprint in cardiac surgical units. Will they someday be as common in ICUs as hemofiltration or dialysis machines? Time will tell.

Chapter 29—ECMO

While MCS devices were discussed in the prior chapter, ECMO has been one of the most significant advances in the care of sick patients in the past decade or so. The technology which is increasingly biocompatible in the duration of ECMO support seems unlimited. Some of the challenges with ECMO are now determining eligibility and mitigating some of the hematologic problems while on the circuit. Irrespective of your discipline, you are likely to care for patients in need of ECMO during your training and thus some knowledge on this is important. Finally, the principle of E-CPR, which is using ECMO to resuscitate a pulseless patient, is becoming more commonly requested when patients arrive with circulatory collapse in the emergency department.

Chapter 30—Durable Mechanical Circulatory Support

Heart failure is a common diagnosis in the modern era. With ongoing limitations in organ availability and donor suitability, there has been significant progress in durable support devices. When these devices are placed, they are either done so as a bridge or as destination therapy. Patients can live very normal lives with high quality, thanks to the technological advancements and improvements in surgical implantation techniques.

Chapter 31—Heart Transplantation

Heart transplantation is the best treatment option for eligible patients with end-stage heart failure and can restore life to patients of all ages. The most exciting advances today are the inclusion of donation after circulatory death (DCD) organs

for consideration and xenotransplantation. Heart transplantation can also be used to treat certain congenital abnormalities and is likely somewhat underutilized in this population.

Chapter 32—Lung Transplantation

Few disease processes are more disabling than end-stage lung disease which renders patients essentially air hungry at all times. In the right patient, a single or double lung transplant can make a huge difference in survival and quality of life. Unfortunately, longevity is still quite limited after lung transplant demonstrating there is still much to do to advance the medical management post-transplant. Similar to heart transplantation, DCD donors are increasingly being used and therapeutic options to rehabilitate donor organs ex vivo have shown some very promising results.

Chapter 33—ACHD

Adult patients with congenital heart disease represent one of the fastest growing patient populations in need of cardiac surgical care. More adults are currently living with congenital heart disease than there are children being born with defects. These patients are often complex and may have undergone many prior procedures. Alternatively, there are a number of diagnoses covered in the chapter which may not present until adulthood. A dedicated multidisciplinary team is a necessary prerequisite for any center hoping to take care of this patient population. Moreover, there is an ongoing discussion about who should care for these patients and in what type of hospital. This has fueled the expansion of many centers to include dedicated ACHD teams.

Chapter 34—Teamwork in the Cardiac Surgical Operating Room

The cardiac surgery operating theater is a complex environment with many moving parts. Teamwork is absolutely critical to ensuring the highest quality of care and minimizing error. Understanding the different roles and their times of high cognitive load in the operating room is fundamental to working well together. Effective leaders use different leadership styles depending on the situation. In general, we recommend that the cardiac surgeon makes it very clear to the team members that everyone's voice is valued and ensures personal safety to encourage speaking up "if you see something say something."

Chapter 35—Principles of Postoperative Care

One of the most important factors in ensuring the highest quality of care delivery in cardiac surgery is the postoperative phase. The physiology can be challenging given there is typically mixed-shock physiology. Having a high-quality multidisciplinary heart team that includes intensivists, nurses, trainees, and advanced practice providers well-versed in the common issues in the postoperative setting is critical. Recognizing patients who are slipping off of the expected pathway is of paramount importance to avoid failure to rescue scenarios. Getting a complex patient through a big operation is just the beginning and often the most straightforward part of the hospitalization.

Contents

Chapter 1
History of Cardiac Surgery

Cynthia L. Miller and Duke E. Cameron

Early Heart Surgery (Rehn, Cutler, Gross, Blalock): [1–4]

In 1896, British surgeon Sir Stephen Paget predicted that "surgery of the heart has probably reached the limits set by Nature to all surgery: no new method, and no new discovery, can overcome the natural difficulties that attend a wound of the heart." Nevertheless, that same year the first successful operation on the human heart was performed, marking the birth of cardiac surgery. Subsequent accomplishments in congenital heart surgery helped pave the way for the development of adult cardiac surgery (Fig. 1.1).

Ludwig Rehn: First Successful Surgery on the Heart

- In 1896, Ludwig Rehn of Frankfurt, Germany repaired a stab wound to the right ventricle, becoming the first to successfully operate on the human heart.

C. L. Miller
Department of Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA
e-mail: Cmiller19@mgh.harvard.edu

D. E. Cameron (✉)
Division of Cardiac Surgery, The Johns Hopkins Hospital, Baltimore, MD, USA
e-mail: Dcameron@jhmi.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_1

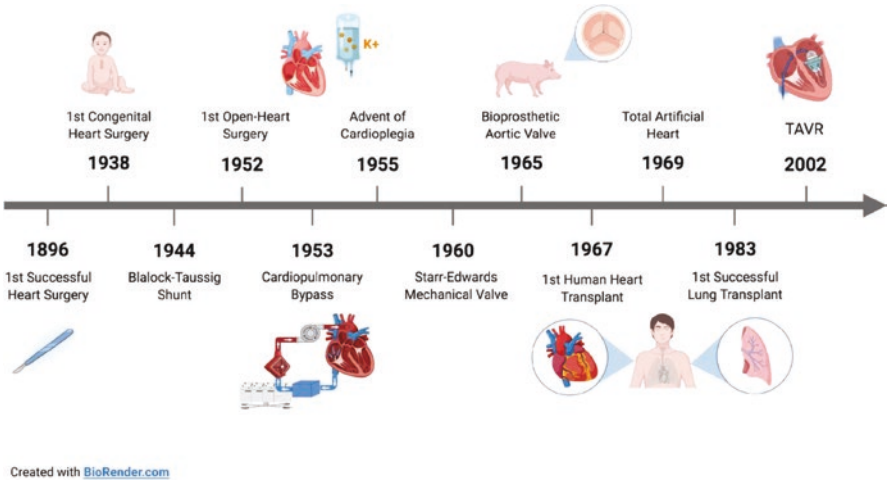


Fig. 1.1 Timeline of milestones in cardiac surgery

Elliott Cutler: The Origin of Heart Valve Surgery

- Early attempts to open stenotic heart valves involved passage of the finger or an instrument to dilate the valve, with high mortality.
- Cutler achieved the first successful surgical repair of mitral stenosis in 1923, when he performed a mitral commissurotomy using a tenotomy knife on a 12-year-old patient with rheumatic mitral stenosis.

Robert Gross: Dawn of Congenital Cardiac Surgery

- In 1938 at Boston Children's Hospital, Gross performed a patent ductus arteriosus (PDA) ligation on a 7-year-old girl, the first successful congenital heart surgical procedure.

Alfred Blalock: A Landmark Operation in Congenital Heart Surgery

- At Johns Hopkins Hospital in 1944, Blalock created an artificial shunt between the subclavian and pulmonary arteries to increase blood flow to the lungs in a 15-month-old with Tetralogy of Fallot.
- The procedure (known as the "Blalock-Taussig Shunt") was conceived by pediatric cardiologist Helen Taussig who observed that infants with pulmonary ste-

nosis developed profound cyanosis after ductus closure and suggested that creating an artificial ductus would improve cyanosis.
- By transforming "blue babies" into healthy, pink children, Blalock demonstrated that heart surgery could save lives and improve quality of life.

The Birth of Open-Heart Surgery: Surface Cooling (Bigelow, Lewis, Swan), Cardiopulmonary Bypass (Gibbon, Kirklin), and Cross-Circulation (Lillehei) [1–3, 5]

Rapid evolution of cardiac surgery occurred in the late 1950s–1960s with the introduction of open-heart surgery. Before then, the majority of cardiac operations were performed on the closed heart; however, for cardiac surgery to progress, surgeons needed a method by which to safely perform intracardiac operations. For this to occur, blood flow to the heart would need to be interrupted without causing irreversible damage to the brain or other vital organs. The emergence of cardiopulmonary bypass enabled surgeons to perform cardiac interventions under direct vision in a motionless, bloodless field.

Wilfred Bigelow: Development of Surface Cooling for Open-Heart Surgery

- In 1950, Bigelow and his research team at the University of Toronto performed experiments in dogs demonstrating that systemic hypothermia achieved by surface cooling to 20–30 °C enabled the heart to be stopped for 6–8 min without cerebral complications. Although 50% of the dogs were successfully revived after the procedure, there was a high mortality upon rewarming due primarily to ventricular fibrillation.

F. John Lewis: First Successful Open-Heart Surgery

- Using the technique of surface cooling, Lewis performed the first successful open-heart surgery in 1952 at the University of Minnesota: atrial septal defect (ASD) closure in a 5-year-old girl. The operation was performed under direct vision through an atrial incision using systemic hypothermia to 28 °C and a 5.5-min period of inflow occlusion.

Henry Swan: Open-Heart Surgery in a Series of Patients

- Swan reported a series of 100 patients who underwent open-heart surgery using hypothermia from surface cooling with a low mortality rate in 1955.
- Use of hypothermia alone to enable open-heart surgery was soon phased out due to limited operating time, associated complications (ventricular fibrillation, air embolization), and emergence of cardiopulmonary bypass.

John Gibbon: The Heart-Lung Machine for Cardiopulmonary Bypass

- In 1953, Gibbon closed a large ASD in an 18-year-old girl with heart failure using the heart-lung machine he developed over a 20-year period in laboratories at Massachusetts General Hospital, University of Pennsylvania, and Thomas Jefferson University. The patient survived and became the first person to undergo successful open-heart surgery with cardiopulmonary bypass.
- After several intraoperative deaths, Gibbon declared a 1-year moratorium on the heart-lung machine and eventually abandoned it altogether. However, his work propagated the ongoing development of cardiopulmonary bypass by others and represented one of the greatest advancements in the history of cardiac surgery.

John Kirklin: Advancement of the Heart-Lung Machine

- Kirklin and colleagues at the Mayo Clinic made modifications to the Gibbon heart-lung machine, and in 1955 performed the first successful series of open-heart operations using cardiopulmonary bypass. After successfully closing a ventricular septal defect (VSD) in a 5-year-old patient using cardiopulmonary bypass, Kirklin operated on eight children with various types of VSDs with good results.
- By the 1960s, cardiopulmonary bypass had become the standard of care for open-heart surgery.

Clarence Walton Lillehei: Cross-Circulation

- While Gibbon was working on the heart-lung machine, Lillehei and colleagues at the University of Minnesota were investigating “cross-circulation” as a technique for maintaining the circulation during open-heart surgery (Fig. 1.2).
- In 1954, Lillehei closed a VSD under direct vision in a 15-month-old boy using cross-circulation. An adult served as the heart-lung machine to support the child’s circulation.

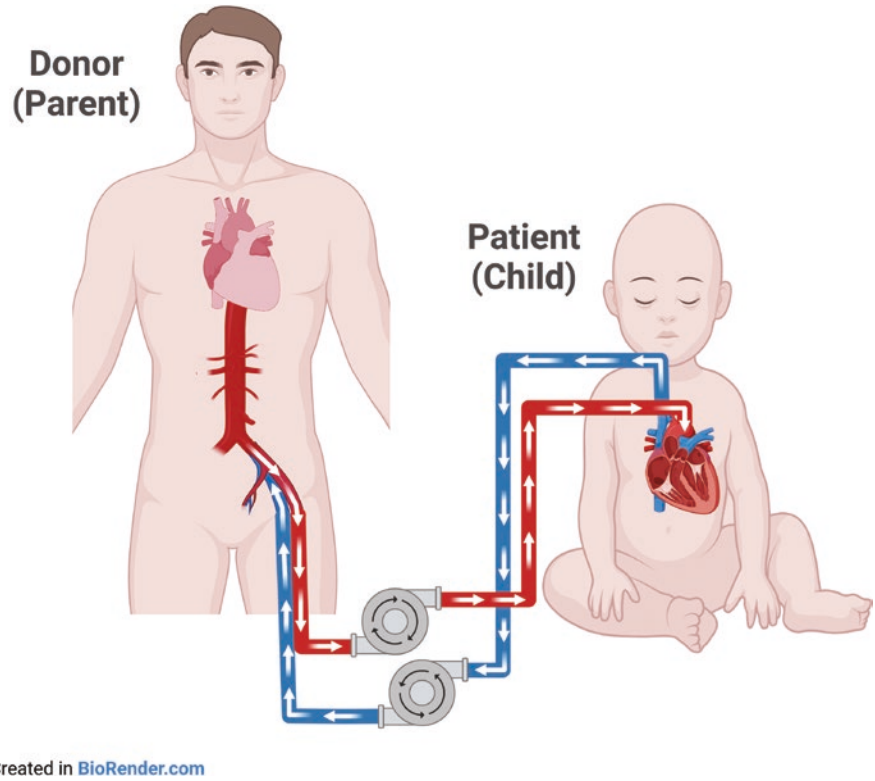


Fig. 1.2 Cross-circulation technique for open-heart surgery, developed by Walton Lillehei in the 1950s

- Within a year, Lillehei published a report of 32 children who underwent repair of VSDs, Tetralogy of Fallot, and atrioventricularis communis ("AV Canal") defects using cross-circulation.
- Although it was soon phased out in favor of cardiopulmonary bypass, cross-circulation was an important catalyst in the development of open-heart surgery.

Myocardial Protection (Melrose, Gay and Ebert, Bretschneider, Buckberg, Daggett, del Nido) [1–3, 6]

Prior to the development of cardioplegia, aortic cross-clamping was routinely used to induce cardiac arrest for performance of intracardiac operations. However, reperfusion was associated with severe myocardial damage ("stone heart") and postoperative heart failure due to lack of myocardial protection. The development of myocardial protective strategies, specifically cardioplegia, was critical for the progression of open-heart surgery. To date, the optimal cardioplegia remains controversial, with ongoing developments in its composition and delivery.

Dennis Melrose: Advent of Cardioplegia

- In 1955, Melrose and colleagues described use of a potassium-based blood cardioplegic solution to induce rapid chemical cardiac arrest before global myocardial ischemia.
- However, the Melrose solution proved toxic to the myocardium and potassium-based cardioplegia was abandoned for over a decade in favor of other techniques (topical hypothermia, intermittent aortic occlusion, direct coronary artery perfusion).

William Gay and Paul Ebert: Reemergence of Cardioplegic Solutions

- In the 1970s, Gay and Ebert renewed interest in potassium-based cardioplegia by demonstrating that use of an osmotically balanced solution with lower potassium concentration (as compared to the Melrose solution) was safe.

Hans-Jürgen Bretschneider: Histidine-Tryptophan-Ketoglutarate (HTK) Cardioplegia

- In the early 1970s, German physiologist Bretschneider developed an intracellular-like cardioplegia (low potassium, low sodium) with histidine, tryptophan, and ketoglutarate as its principal constituents ("HTK solution"). These additives serve as protectants to buffer metabolic acidosis, improve ATP production during reperfusion, and stabilize the cellular membrane.

Gerald Buckberg: Blood Cardioplegia

- In 1978 at UCLA, Buckberg introduced the concept of blood cardioplegia; until that time, the majority of cardioplegic solutions were crystalloid-based. In the late 1980s, Buckberg described use of combined antegrade and retrograde cardioplegia, which continues in use today.

Willard Daggett: Optimization of Cardioplegia

- Throughout the 1970s–1980s, Daggett performed extensive research to further refine the chemical composition of cardioplegic solutions and ultimately demonstrated the superiority of cold oxygenated dilute blood cardioplegia.

Pedro del Nido: Cardioplegia for Congenital Heart Surgery

- In 1995 at Boston Children's Hospital, del Nido introduced a unique blood and crystalloid mixed cardioplegic formula for use in congenital heart surgery.
- The "del Nido Solution" is a single-dose, four parts crystalloid to one part whole blood solution that has been associated with a longer duration of safe arrest.
- It has been widely adopted for use in congenital heart surgery and has increasing application in adult cardiac surgery.

Valve Repair and Replacement (Starr, Ross, Barratt-Boyes, McGoon, Carpentier, Bentall, Yacoub and David): [1–3, 7]

Open-heart surgery made it possible to perform complex valve repair and enabled valve replacement. As such, the 1960s marked an era of extensive advancement in the treatment of valvular disease, including the development of mechanical valves, homografts (from a human patient), and xenografts (from a different species such as a pig).

Albert Starr: First Mechanical Heart Valve

- In 1960, Albert Starr and engineer Lowell Edwards introduced the first mechanical heart valve to be successfully implanted in a human.
- The Starr-Edwards caged-ball mechanical valve was subsequently used for decades for aortic and mitral valve replacement and revolutionized the treatment of valvular disease.

Donald Ross: Pioneer of Homograft Valve Replacement

- In 1961, Ross performed the first successful human cadaveric homograft replacement of the aortic valve.
- He subsequently described a pulmonary autograft procedure for aortic valve replacement (Ross Procedure) in 1967. This involved transferring the patient's pulmonary valve to the aortic position and then placing a homograft valve in the pulmonary position.
- The Ross procedure has become known for durability of the pulmonary autograft, growth potential, and lack of need for anticoagulation, making it especially appealing for infants with congenital aortic stenosis.

Brian Barratt-Boyes: Development of Homograft Valves

- Along with Ross, Barratt-Boyes was largely responsible for the development of aortic homografts for aortic valve replacement in the early 1960s.

Dwight McGoon: Proficiency in Valve Surgery

- In 1960 at the Mayo clinic, McGoon first described surgical repair for mitral valve regurgitation in the setting of ruptured chordae.
- Later in 1965, he reported a series of 100 consecutive aortic valve replacements with no in-hospital mortality, a remarkable achievement in the period prior to cardioplegia.

Alain Carpentier: Xenograft Heart Valves

- Carpentier and his team in Paris performed the first successful xenograft valve replacement in 1965, in which a porcine valve was used to replace the diseased aortic valve in a human patient.
- He later developed several methods to reduce harmful immune responses against the xenograft valve and mounted it onto a metal frame in the creation of a "bio-prosthetic valve."

Hugh Bentall: Aortic Root Replacement

- In 1968, Bentall described a method for complete replacement of the ascending aorta and aortic valve for treatment of aneurysms. This involved use of a composite graft containing a prosthetic aortic valve and prosthetic graft replacement of the ascending aorta with reimplantation of the coronary arteries.

Sir Magdi Yacoub and Tirone David: Valve-Sparing Aortic Root Replacement

- In 1979, Yacoub pioneered a valve-sparing procedure for aortic root replacement in patients with normal aortic valve cusps and an aneurysmal or dissected aortic root. This involved sewing a vascular graft onto the coronary sinuses and became known as the "remodeling" technique given that the native aortic valve was remodeled into the vascular graft.

- David and colleagues at the University of Toronto in 1989 described an alternative method for valve-sparing root replacement, referred to as the "reimplantation" technique. By reimplanting the native aortic valve directly into a vascular graft of fixed circumference, there was a reduced risk of developing aortic insufficiency and less bleeding as compared to the remodeling technique.

Transplantation (Carrel, Shumway, Barnard, DeBakey and Cooley, Cooper): [1–3, 8]

The first experimental studies in cardiac transplantation were performed at the beginning of the twentieth century, however, more than six decades would pass before the first successful human heart transplant in 1967. The excitement of this accomplishment was soon overshadowed by disappointing patient survival. The arrival of cyclosporine for immunosuppression in the 1980s revitalized the field, and today heart transplantation represents the definitive treatment for end-stage heart disease. Similarly, the advent of modern immunosuppression and advanced surgical techniques have enabled lung transplantation to become a reality.

Alexis Carrel: Experimental Heart Transplantation

- Carrel and Guthrie first reported attempts at experimental heart transplantation in 1905. Using a heterotopic approach, they transplanted the heart of a puppy into the neck of an adult dog and observed ventricular contractility within an hour of the operation.
- Earlier in 1902, Carrel described methods for vascular anastomoses, which we continue to use today.

Norman Shumway: The Father of Heart Transplantation

- In 1960 at Stanford University, Shumway and his colleague Robert Lower established the technique of orthotopic heart transplantation.
- He and his team subsequently addressed many challenges in heart transplantation, including cardiac preservation, immunosuppression, detection of rejection, and the effect of denervation.
- After performing the first human heart transplant in the US in 1968, Shumway continued the Stanford heart transplant program during the 1970s when nearly all other programs had ceased due to poor outcomes.

Christiaan Barnard: First Human-to-Human Heart Transplant

- On December 3, 1967 in Cape Town, South Africa, Barnard stunned the world by performing the first human-to-human heart transplant.
- Although his first patient died after 18 days, Barnard's second heart transplant recipient lived for 19 months and became the first long-term survivor. This provided hope for the future of clinical heart transplantation as a routine therapy for end-stage heart disease.

Michael DeBakey and Denton Cooley: Ventricular Assist Device and the Artificial Heart

- In 1963, DeBakey performed the first successful implantation of a left ventricular assist device (LVAD) in a patient with severe heart failure due to complications after aortic valve replacement.
- Several years later in 1969 and using a device designed by DeBakey, Cooley performed the first total artificial heart implant in a patient with severe heart failure awaiting transplantation. This demonstrated the feasibility of using a mechanical support device as a bridge to transplantation.

Joel Cooper: First Successful Lung Transplant

- Cooper and colleagues at the University of Toronto performed the first successful lung transplant in 1983, after numerous failed attempts in the preceding two decades by other groups. This success was enabled by innovations to prevent bronchial anastomotic dehiscence, a dreaded complication that previously hindered progress in the field of lung transplantation.
- Later, Cooper directed the first successful double-lung transplants in 1986 and 1987.

Minimally Invasive Heart Surgery (Chitwood, Cribier): [9, 10]

The field of minimally invasive cardiac surgery originated in the 1990s and has since undergone significant transformation and increasing popularity. Two of the most notable advances include the development of transcatheter approaches for valve replacement and the emergence of robotic heart surgery.

W. Randolph Chitwood: Video-Assisted and Robotic Mitral Valve Surgery

- In the late 1990s and early 2000s, Chitwood advanced the field of minimally invasive cardiac surgery by performing video-assisted and later robotic mitral valve surgery.

Alain Cribier: Percutaneous Valve Surgery

- Cribier pioneered the field of percutaneous valve surgery, performing the first transcatheter aortic valve replacement (TAVR) in 2002.
- Since then, TAVR has become standard of care for treatment of aortic stenosis in high-risk patients unsuitable for surgery, with increasing application in lower-risk populations.

References

1. Kouchoukos NT, Scharff JR. The history of cardiac surgery [internet]. In: Baumgartner WA, Jacobs JP, Darling GE, editors. Adult and pediatric cardiac surgery. STS cardiothoracic surgery E-book. Chicago: Society of Thoracic Surgeons; 2020. [cited 2022 April 22]. ebook.sts.org.
2. Stephenson LW, Baciewicz JFA. History of cardiac surgery. In: Cohn LH, Adams DH, editors. Cardiac surgery in the adult. 5th ed. New York: McGraw-Hill Education; 2017.
3. Bouchard T, Subichin M, Firstenberg M. Fifty years of cardiac surgery: innovation, evolution, and revolution in cardiovascular therapies. Int J Acad Med. 2019;5(3):156–64. https://doi.org/10.4103/ijam.Ijam_49_18.
4. Paget S. The surgery of the chest. John Wright & Co: Bristol; 1896. p. 121.
5. Stoney WS. Evolution of cardiopulmonary bypass. Circulation. 2009;119(21):2844–53. https://doi.org/10.1161/CIRCULATIONAHA.108.830174.
6. Daggett WM Jr, Randolph JD, Jacobs M, O'Keefe DD, Geffin GA, Swinski LA, et al. The superiority of cold oxygenated dilute blood cardioplegia. Ann Thorac Surg. 1987;43(4):397–402.
7. Maddalo S, Beller J, DeAnda A. A Bentall is not a Bentall is not a Bentall: the evolution of aortic root surgery. Aorta (Stamford). 2014;2(5):169–78. https://doi.org/10.12945/j.aorta.2014.14-021.
8. Venuta F, Van Raemdonck D. History of lung transplantation. J Thorac Dis. 2017;9(12):5458–71. https://doi.org/10.21037/jtd.2017.11.84.
9. Chitwood WR Jr. Robotic mitral valve surgery: overview, methodology, results, and perspective. Ann Cardiothorac Surg. 2016;5(6):544–55.
10. Andersen HR. How transcatheter aortic valve implantation (TAVI) was born: the struggle for a new invention. Front Cardiovasc Med. 2021;8:722693. https://doi.org/10.3389/fcvm.2021.722693.

Chapter 2
Surgical Anatomy of the Heart

Dane C. Paneitz and Gus J. Vlahakes

Introduction

- A comprehensive understanding of the surgical anatomy of the heart is essential to be a precise heart surgeon. This chapter serves as a foundation from which to build such an understanding.

Surface Anatomy

- The heart is positioned obliquely within the mediastinum with 1/3 of its mass located behind the sternum and the remaining 2/3 to the left of the sternal border. The right ventricle is the anterior-most chamber (susceptible to injury during reoperative sternotomies), and the left atrium is the most posterior chamber (anterior to the esophagus on transesophageal echocardiography, Fig. 2.1). In a patient who has not had prior heart surgery via sternotomy, the thymus gland lies in the upper third of the anterior mediastinum, and it protects the aorta during routine sternotomy. However, in reoperative situations, the thymus is often not present in the midline, as its lobes have retracted laterally from prior surgery. In this situation, the ascending aorta may also be vulnerable to injury during sternal entry in a reoperative case.
- The heart lies within the pericardium which consists of an outer fibrous layer and an inner serous layer. The serous layer is further divided into the parietal (attached to pericardium) and visceral (attached directly to the heart) layers, and between

D. C. Paneitz · G. J. Vlahakes (✉)
Division of Cardiac Surgery, Department of Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA
e-mail: dpaneitz@mgh.harvard.edu; gvlahakes@mgh.harvard.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_2

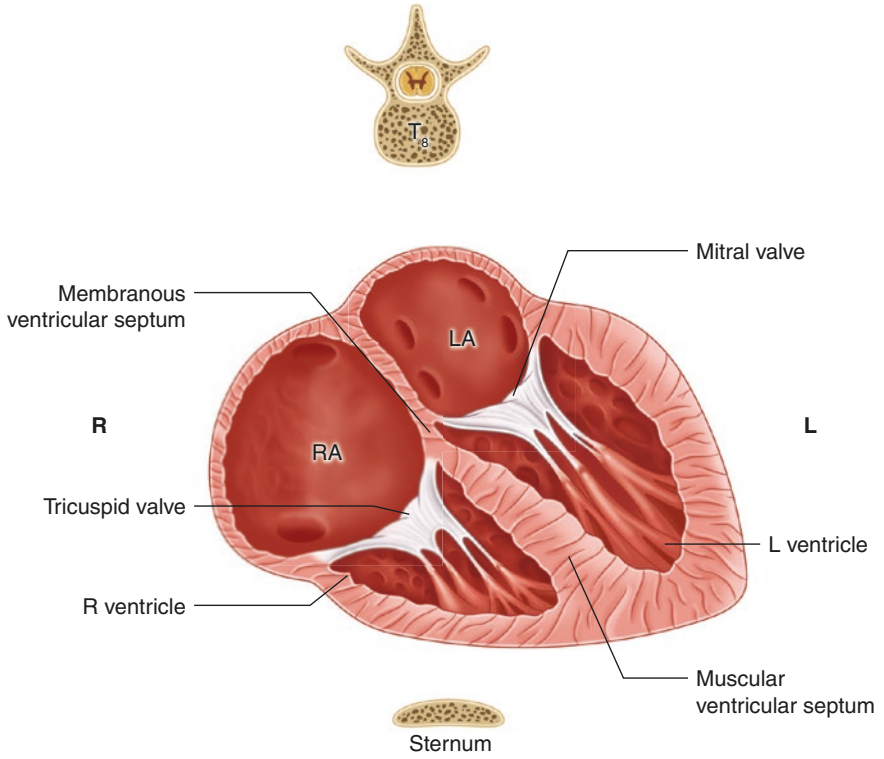


Fig. 2.1 The right ventricle is the anterior-most chamber located behind the sternum, and the left atrium is the posterior-most chamber

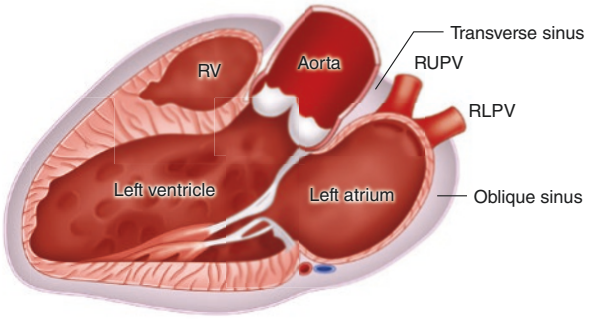


Fig. 2.2 The transverse sinus is located between the arterial structures (aorta and pulmonary artery) anteriorly and the venous structures (SVC and pulmonary veins) posteriorly. *RUPV* right upper pulmonary vein, *RLPV* right lower pulmonary vein

these two layers is a small amount of serous fluid which serves as a lubricant. Pericardial tissue can be harvested and used to fashion an autologous patch or a pledget.

- As a result of cardiac embryological development, two important pericardial sinuses, the transverse and oblique sinuses, are formed (Fig. 2.2). The **transverse**

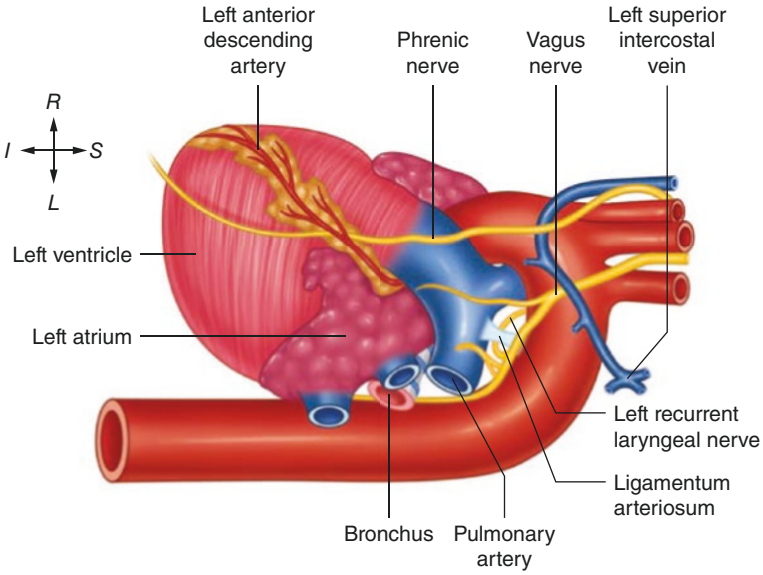


Fig. 2.3 Highlights the courses of the phrenic nerve anterior to the pulmonary hilum and the vagus nerve posterior to the hilum, as well as the left recurrent laryngeal nerve

sinus is posterior to the aorta and main pulmonary artery and anterior to the **superior vena cava** (SVC), while the **oblique sinus** is a blind-ending sac between the posterior surface of the left atrium and the anterior surface of the posterior pericardium.

- Figure 2.3 depicts important nerves to identify during dissection. The **phrenic nerves** course along the lateral aspects of the pericardium bilaterally after passing anteriorly to the pulmonary hila. The **vagus nerves** run posterior to the pulmonary hila after giving off the right and left **recurrent laryngeal branches**, which loop around the right subclavian artery and aortic arch at the **ligamentum arteriosum**, respectively.
- There are several important external grooves (sulci) to recognize. The **interatrial groove**, also known as Waterston's groove or Sondergaard's groove, is located posteriorly and is an important landmark for accessing the left atrium, as is routinely done for operations on the mitral valve. The **anterior interventricular groove** courses along the anterior surface of the heart between the right and left ventricles carrying the left anterior descending artery and great cardiac vein. The **posterior interventricular groove** travels along the posterior surface of the heart and contains the posterior descending artery and middle cardiac vein. The **atrioventricular groove** has a right and left component. The right atrioventricular groove carries the right coronary artery and small cardiac vein. The left atrioventricular groove contains the left circumflex artery and the coronary sinus.

Coronary Vasculature

- Figures 2.4 and 2.5 depict the anterior and posterior courses of the coronary arteries, respectively. The left and right coronary arteries arise from the left and right **sinuses of Valsalva** within the aortic root, respectively. The coronary arteries receive the majority of their perfusion during diastole, which is one of the reasons an intra-aortic balloon pump, inflating during diastole and raising diastolic pressure, can be beneficial by improving coronary artery filling during diastole.

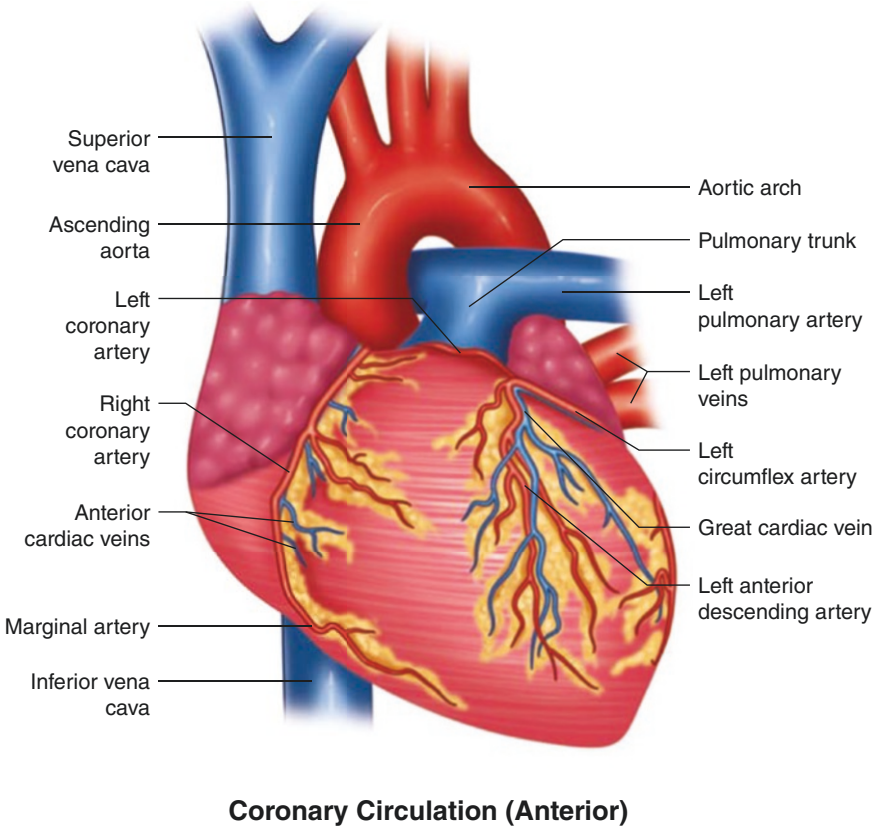


Fig. 2.4 The courses of the right and left coronary arteries and their associated venous drainage on the anterior surface of the heart are depicted here

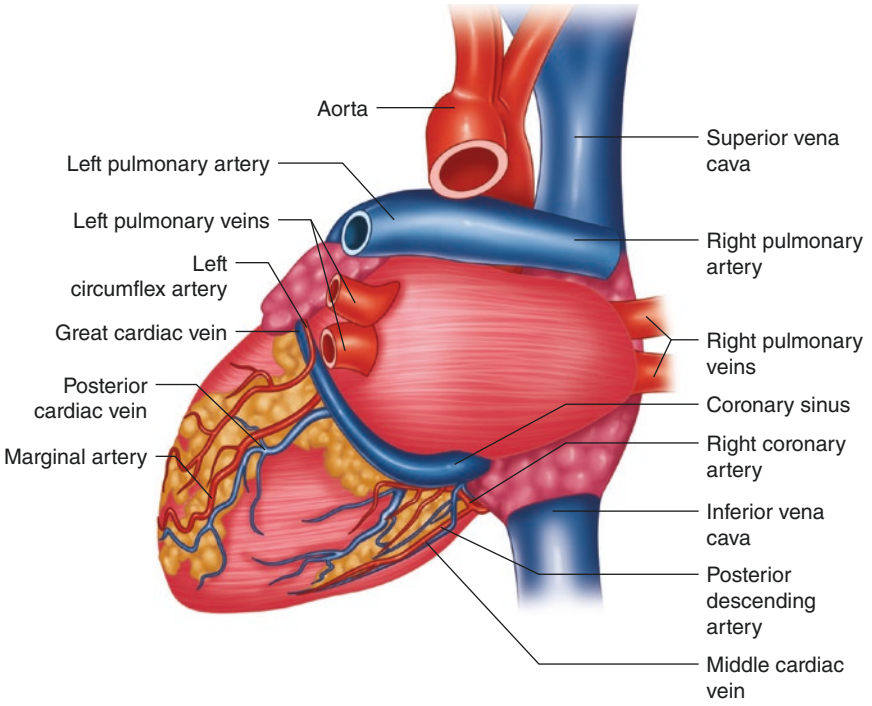


Fig. 2.5 The courses of the right and left coronary arteries and their associated venous drainage on the posterior surface of the heart are depicted here

Left Coronary Artery

- The left coronary artery (LCA) begins as the **left main coronary artery (LM)** before bifurcating into the **left anterior descending artery (LAD)** and the **left circumflex artery (LCX).** The **ramus intermedius** is a common variant coronary artery that typically branches from the LM between the bifurcation of the LAD and LCX. The LAD gives off septal perforators and diagonal branches which supply the interventricular septum and the anterolateral left ventricle, respectively. The LCX has **obtuse marginal (OM) branches** which supply the lateral and posterior left atrium and left ventricle. In approximately 10% of people, the left circumflex supplies the posterior descending artery (PDA) which is termed a "left dominant system."
- The left coronary system supplies the left atrium, left ventricle, anterior two-thirds of the interventricular septum, and anterolateral papillary muscle (PM). It supplies the sinoatrial node in approximately 40% of people.

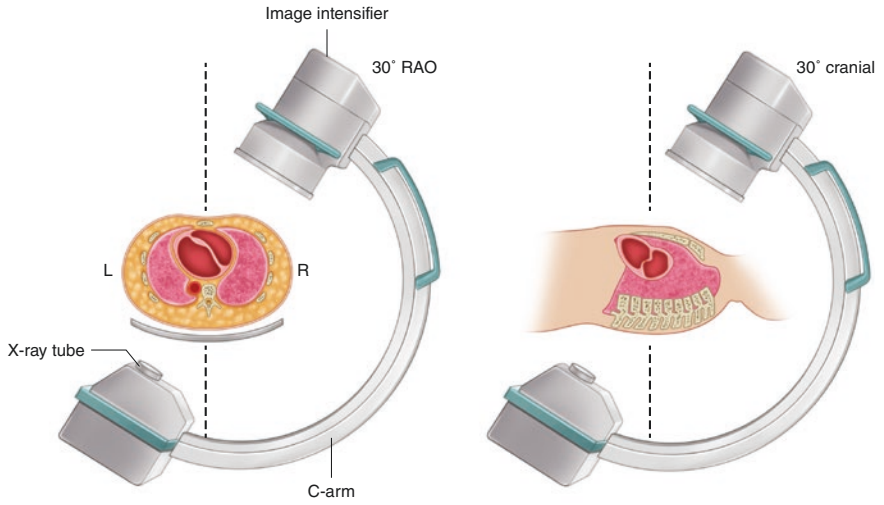


Fig. 2.6 The different views of coronary angiograms are produced by adjusting the position of the X-ray source and image intensifier in relation to the patient. The position of the image intensifier in relation to the patient is used to name the different views

- Coronary angiography via cardiac catheterization is commonly performed during the workup of coronary artery disease and localizes diseased segments of the coronary arteries. Figure 2.6 shows the relationship of the patient to the image intensifier which is changed to obtain the different views. The details of cardiac catheterization are covered in a following chapter, but briefly, the optimal views of the left main are right anterior oblique (RAO)/cranial, anteroposterior (AP) cranial, left anterior oblique (LAO)/cranial, and AP straight. The LM bifurcation views include AP caudal, spider, and RAO cranial. Proximal LAD views are LAO caudal, LAO cranial, RAO cranial, and AP caudal. Mid/distal LAD views are AP cranial and RAO cranial. The proximal LCX is viewed in spider and AP caudal while the distal LCX is viewed in AP cranial and RAO cranial [1].

Right Coronary Artery

- The right coronary artery (RCA) has three main segments: proximal, mid, and distal. The middle segment gives off acute marginal arteries, and the distal segment contributes the PDA in approximately 80% of people.
- The right coronary system supplies the right atrium, right ventricle, sinoatrial and atrioventricular nodes (most commonly), inferior interventricular septum, inferior left ventricle, and posteromedial papillary muscle.

- The optimal coronary angiography views for the RCA include LAO caudal for proximal RCA, RAO for mid RCA and AP cranial, RAO cranial, and LAO cranial for distal RCA.

Coronary Veins

- The coronary venous system courses with the coronary arteries as described earlier and terminates as the **coronary sinus** which empties into the right atrium.
- When extensive coronary artery disease, particularly in the LAD territory, makes it difficult to deliver cardioplegia myocardial protection to that territory, the coronary sinus is commonly cannulated through the right atrial wall to provide "retrograde cardioplegia" which is delivered to the myocardium via the coronary venous system, hence the term "retrograde." When present, a persistent left SVC commonly drains into the coronary sinus and must be controlled for retrograde cardioplegia to be effectively delivered to the myocardium. In addition, depending on what type of operation is being performed, a persistent left SVC may need to be separately cannulated to avoid flooding the surgical field when the right atrium is opened.
- **Thebesian veins** drain a small portion of the venous return directly into the heart chambers.

The Fibrous Skeleton

- The cardiac valves are supported by a fibrous skeleton (Fig. 2.7) that also prevents direct ventricular activation by depolarizing atrial muscle; the cardiac conduction system serves this role in a controlled fashion.
- The **central fibrous body** of the fibrous skeleton of the heart lies at the convergence of the mitral, aortic, and tricuspid valves and is made up of the right fibrous trigone, which is a thickened portion of the fibrous skeleton, and the membranous septum, which separates the right-sided chambers from the left ventricular outflow tract (LVOTO).
- The **right fibrous trigone** separates the aortic valve and anterior mitral valve leaflet and is associated with the conduction Bundle of His.
- The **left fibrous trigone** is located between the aortic valve and the lateral aspect of the anterior mitral valve leaflet and is associated with the LCX artery.
- The **aortomitral curtain** is the fibrous continuity between the right and left fibrous trigone that provides support for the valves and provides separation between the aortic and mitral valves.

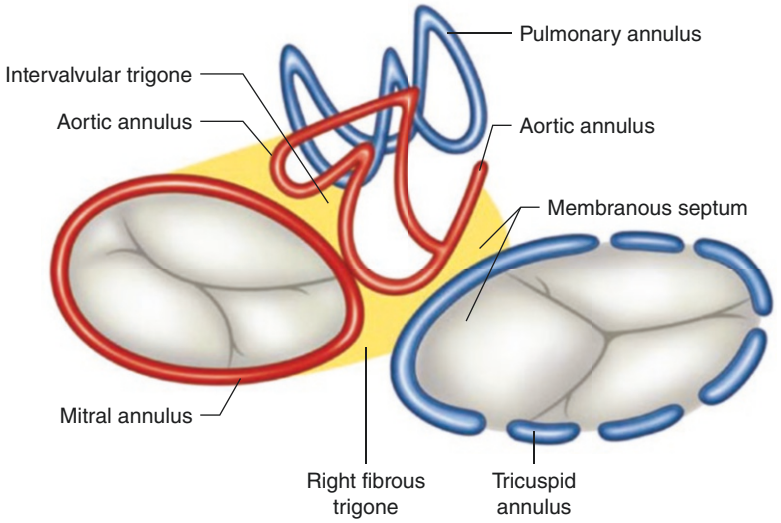


Fig. 2.7 The fibrous skeleton of the heart is a critical structure to understand. The right fibrous trigone is at the junction of the tricuspid, mitral, and aortic valves and the left fibrous trigone is at the lateral junction of the mitral and aortic valves

Tricuspid Valve

- The tricuspid valve is located between the right atrium and right ventricle and consists of three leaflets: the septal, anterior, and posterior leaflets (Fig. 2.8).
- The septal leaflet attaches to the septal wall (hence, its name), and the anterior-septal commissure (the junction of the anterior and septal leaflets at the annulus) is associated with the right fibrous trigone. The Bundle of His can be damaged in this area.
- The septal leaflet is one of the boundaries of the **Triangle of Koch** which also includes the coronary sinus and the **Tendon of Todaro**. The **atrioventricular node** is located at the apex of this triangle. This is a very important surgical anatomic landmark.
- There are three right ventricular papillary muscles: the septal, anterior, and posterior papillary muscles. These muscles support the tricuspid valve leaflets via chordae tendineae which attach the leaflets to the papillary muscles to prevent prolapse during ventricular systole.

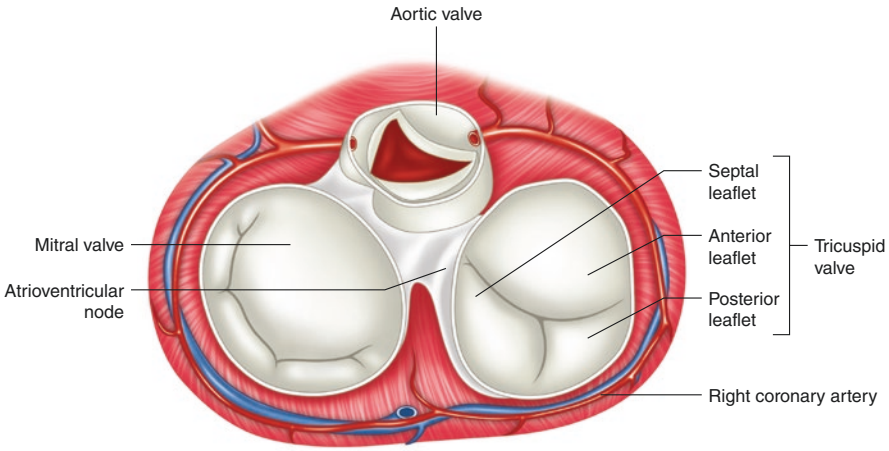


Fig. 2.8 Highlights the relationships of the coronary arteries with the tricuspid and mitral valve

Mitral Valve

- The mitral valve is located between the left atrium and left ventricle and has two leaflets: the anterior and posterior leaflets.
- The anterior leaflet is attached to the aortomitral curtain which separates it from the aortic valve, and care should be taken when passing mitral valve sutures at this location to avoid injury the aortic valve.
- During mitral valve surgery, the medial aspect of the anterior leaflet is adjacent to the right trigone which is associated with the Bundle of His and is at risk for injury. The lateral aspect of the anterior leaflet annulus is associated with the left fibrous trigone and the LCX is at risk of injury in this location, particularly in patients with left-dominant coronary artery anatomy where the circumflex artery can be closely related to the posterior mitral annulus. The coronary sinus is at risk for injury when placing mitral valve sutures in the posterior leaflet annulus.
- There are two papillary muscles in the left ventricle that support the mitral valve: the **anterolateral papillary muscle** and **posteromedial papillary muscle**. The anterolateral PM receives a dual blood supply from the LAD and LCX which makes it less susceptible to injury/rupture than the posteromedial PM which only receives blood supply from the PDA.

Pulmonic Valve

- The pulmonic valve, located between right ventricle and pulmonary artery, has three semilunar cusps: the anterior, right, and left cusps.

- The **supraventricular crest**, a muscular fold, separates the pulmonary and tricuspid valves and is part of the subpulmonic infundibulum which supports the pulmonic valve.
- The RCA passes posterior to the pulmonary trunk and can be injured during pulmonic valve replacement.

Aortic Valve

- The aortic valve separates the left ventricle from the aorta and has three semilunar cusps: the right coronary cusp (RCC), left coronary cusp (LCC), and noncoronary cusp (NCC).
- It is located within the aortic root which begins at the basilar attachments of the aortic cusps and extends to the sinotubular junction. Sinotubular junction is exactly what it seems: the junction between the aortic sinuses and the "tube" of the ascending aorta.
- The aortic valve is centrally positioned between the tricuspid, pulmonary, and mitral valves.
- The structures at risk for injury during an aortic valve replacement include the coronary arteries, the aortomitral curtain, and anterior mitral valve leaflet.

Aorta

- The ascending aorta extends from the sinotubular junction to the origin of the innominate artery.
- The aortic arch begins at the origin of the innominate artery and ends after the aortic isthmus, which is the segment of aorta between the left subclavian artery and the ligamentum arteriosum.
- The aortic arch is divided into anatomic zones (Fig. 2.9):
  - Zone 0 starts at the ascending aorta and ends just distal to innominate artery origin.
  - Zone 1 starts after the innominate artery origin and ends just distal to the left common carotid artery origin.
  - Zone 2 starts after the left common carotid origin and ends just distal to the left subclavian artery origin.
  - Zone 3 starts after the left subclavian artery origin and ends at the mid descending thoracic aorta.
  - Zone 4 involves the mid descending thoracic aorta.
- The normal arch anatomy is a left-sided aortic arch that first gives off the innominate artery, followed by the left common carotid artery, followed by the left subclavian artery.

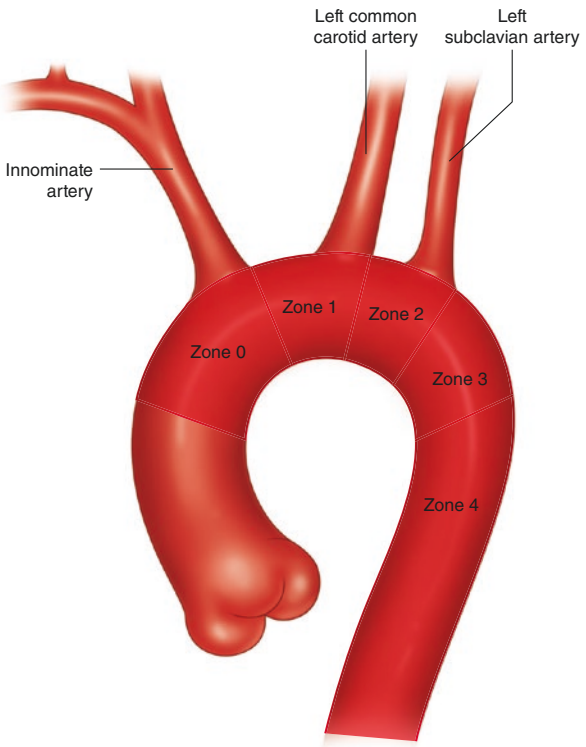


Fig. 2.9 Zones of the thoracic aorta

- Aortic arch variants:
  - Bovine arch: The innominate artery and left common carotid artery share a common origin.
  - Aberrant right subclavian artery (*arteria lusoria*): Instead of the innominate artery giving rise to the right subclavian artery, the right subclavian artery originates from the aortic arch distal to the left subclavian and courses posteriorly, typically behind the esophagus, to supply the right arm and can cause esophageal compression leading to dysphagia. A common associated anomaly is a right non-recurrent laryngeal nerve.
  - Right aortic arch: The aortic arch courses over the right bronchus instead of the left bronchus. The two most common types include the mirror image branching pattern and the aberrant left subclavian artery. From proximal to distal, the mirror imaging pattern gives off the left innominate artery, the right common carotid artery and the right subclavian artery. The aberrant left subclavian variant involves the left subclavian artery originating distal to the right subclavian artery and coursing posteriorly to supply like left arm. It can also cause esophageal compression.
  - Vascular ring: A congenital anomaly where the aorta and its vessels form a ring around the esophagus and/or trachea.

Conduction System

- Figure 2.10 shows the course of the conduction system.
- Sinoatrial (SA) node
  - Located at the posterolateral junction of the SVC and right atrium.
  - Blood supply: sinoatrial nodal artery most commonly from the RCA but can also originate from the LCx artery.
  - Propagates electrical impulse to the atrioventricular node.
- Atrioventricular (AV) node
  - Located at the apex of the Triangle of Koch (see above).
  - Blood supply: atrioventricular nodal branch most commonly from the RCA but can also originate from the LCx artery.
  - Propagates electrical impulse to the Bundle of His.
- Bundle of His
  - Travels from the AV node to the interventricular septum where it gives off the left and right bundle branches. The left bundle branch divides into the left anterior and left posterior fascicular branches. Along with the right bundle branch, these branches lead to the Purkinje fibers which propagate the electrical impulse to the ventricles, leading to contraction.

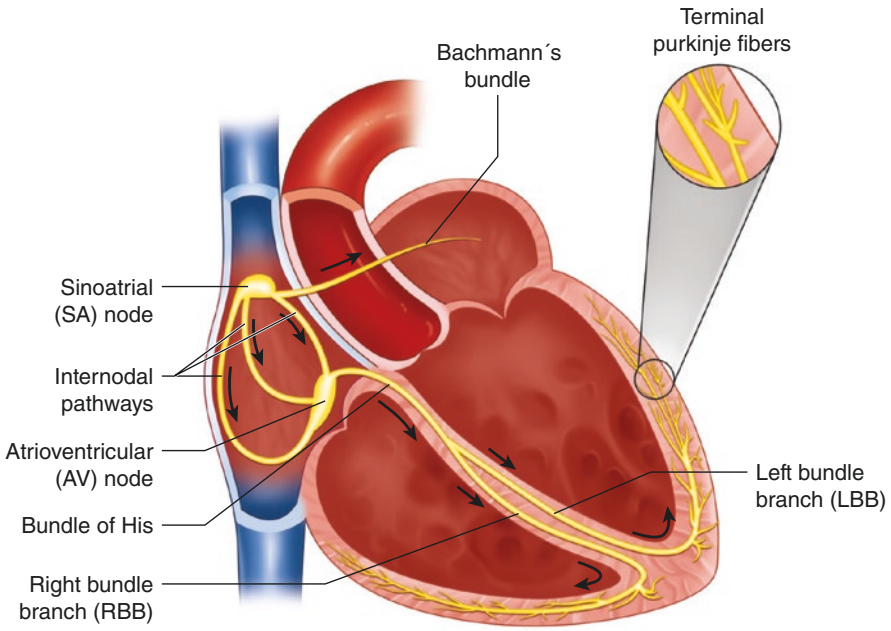


Fig. 2.10 Highlights the conduction system

- Bachmann's Bundle
  - A muscular bundle that serves as the main electrical conductor between the right atrium and left atrium.

Another great resource for cardiac anatomy is the Society of Thoracic Surgeons E-Book [2].

References

1. Rigatelli G, Gianese F, Zuin M. Modern atlas of invasive coronary angiography views: a practical approach for fellows and young interventionalists. Int J Cardiovasc Imaging [Internet]. 2021 [cited 2022 Feb 11]. https://link.springer.com/10.1007/s10554-021-02489-5.
2. Cardiac anatomy | adult and pediatric cardiac [Internet]. [cited 2022 Mar 2]. https://ebook.sts.org/sts/view/Cardiac-and-Congenital/1864009/all/Cardiac_Anatomy?refer=true.

Chapter 3
Preoperative Evaluation and Risk Assessment

Elizabeth A. Calle and David M. Shahian

Abbreviations

ACE	Angiotensin converting enzyme
ACSD	Adult Cardiac Surgery Database
AKI	Acute kidney injury
ARB	Angiotensin receptor blocker
AVR	Aortic valve replacement
BID	Twice a day
BMP	Basic metabolic panel
CAB	Coronary artery bypass
CABG	Coronary artery bypass graft
CAD-RADS	Coronary artery disease reporting and data system
CBC	Complete blood count
CMP	Complete metabolic panel
COPD	Chronic obstructive pulmonary disease
CPAP	Continuous positive airway pressure
CPB	Cardiopulmonary bypass
CPET	Cardiopulmonary exercise testing
CT	Computed tomography

E. A. Calle
Department of Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: ecalle@mgh.harvard.edu

D. M. Shahian (✉)
Division of Cardiac Surgery and Department of Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: dshahian@mgh.harvard.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_3

CTA	Computed tomography angiography
CTP	Child-Turcotte-Pugh (classification)
CXR	Chest X-ray
DES	Drug-eluting stent
DOAC	Direct oral anticoagulant
DSE	Dobutamine stress echocardiogram
ECG	Echocardiogram
HD	Hemodialysis
HIT	Heparin-induced thrombocytopenia
IABP	Intra-aortic balloon pump
INR	International normalized ratio (normalized value for PT—prothrombin time)
IV	Intravenous
LFTs	Liver function tests
LIMA	Left internal mammary artery
MELD	Model for end-stage liver disease
MR	Mitral regurgitation
MRSA	Methicillin resistant staph aureus
MSSA	Methicillin sensitive staph aureus
MV	Mitral valve
MVR	Mitral valve replacement
OR	Operating room
PET	Positron emission tomography
PFTs	Pulmonary function tests
PTT	Partial thromboplastin time
RIMA	Right internal mammary artery
SC	Subcutaneous
SPECT	Single-photon emission computed tomography
STS	The Society of Thoracic Surgeons
TSH	Thyroid stimulating hormone
TURP	Transurethral resection of prostate
VAD	Ventricular assist device

Introduction

Common operations in adult cardiac surgery include coronary artery bypass graft surgery (CABG), generally performed for symptomatic blockages of the coronary arteries; valve repair or replacement for stenosis or regurgitation; and surgery of the thoracic aorta for aneurysm or dissection. Heart and heart-lung transplants also fall within the purview of cardiac surgery but are beyond the scope of this chapter.

Preoperative evaluation includes:

1. Classification of the severity of disease—e.g., identification of which coronary vessels are blocked, and the location and extent of these obstructions; quantification of the degree of stenosis or regurgitation of a diseased valve.

2. Assessment of the overall medical fitness of the patient for surgery, including their risk of postoperative complications and mortality.

The former considerations help to guide the technical conduct of the operation, while the latter are the basis of shared decision-making with the patient—which specific operation is best for them, how the benefit to risk ratio may be optimized, and whether the patient is willing and able to accept the expected risk of undergoing heart surgery.

In this section, we outline the basic preoperative evaluation of all cardiac surgery patients including management of commonly used medications. We also describe several additional tests that may be useful in specific scenarios. Finally, we review some of the most frequently used risk calculators and their use to counsel patients and to tailor perioperative care.

Basic Preoperative Patient Evaluation

The following sections are designed as a practical aide for the bedside clinician. More extensive discussion of patient assessment and clinical care can be found in Dr. Bojar's Manual of Perioperative Care in Adult Cardiac Surgery [1]; detailed descriptions of the pathophysiology of heart disease can be found in Dr. Lily's textbook [2].

History

- A thorough history can help determine whether additional testing is required to stratify a patient's risk; to identify possible risk mitigation strategies; and to individualize patient-specific perioperative care needs.
- Risk mitigation of comorbidities may require consultation or management by other specialties, such as endocrinology for diabetic patients, hematology for patients with bleeding disorders, or nephrology for patients with kidney disease (discussed further below).
- Urgent or emergent surgery may impose practical limits on preoperative testing or supplemental consultation.

  – **General:**

    Level of routine daily activity, an approximation of cardiovascular fitness:

    - Any difficulty in walking up a flight of stairs or along a city block
    - Issues with housework or other activities of daily living

    History of falls, instability, incontinence, memory or cognition issues, impaired vision, unexplained weight loss
    Symptoms that suggest possible comorbidities (coexisting patient conditions that increase risk), especially if there is significant impairment of other organs

Substance use such as alcohol; tobacco (any form); marijuana; vaping; use of opiates, other controlled substances, or recreational drugs; misuse of other prescription medications
All prescription or non-prescription agents, including biologics (also see specific sub-sections below)
All vitamins and supplements, many of which have the potential to impact perioperative care

– **Neurological:**

Symptoms: Neurological deficits; headache; dizziness; peripheral neuropathy, including distribution and severity
History: Prior transient ischemic attacks (TIAs) or stroke, including symptoms at the time and any residual deficits. History of intracranial aneurysm or arterio-venous malformation. History of glaucoma (baseline increased intra-ocular pressure may further increase during cardiac surgery and can lead to the rare complication of ischemic optic neuropathy)
Medications: Aspirin (ASA) or other antiplatelet agents; anticoagulants (warfarin or direct-acting oral anticoagulants (DOACs)); glaucoma drops

– **Cardiac:**

Patients with coronary artery disease most commonly report inability to tolerate exercise, which may or may not be accompanied by chest or arm pain, "tightness," or pressure. In severe instances, this pain may occur with minimal exertion or even at rest. Some patients may not have exertional chest pressure but rather may experience acute exertional shortness of breath related to cardiac ischemia.
Patients with clinically significant valvular disease may report shortness of breath with activity, cough, or difficulty lying down when heart failure has developed. They may also report dizziness, light-headedness, or frank syncope, especially with advanced aortic stenosis.
Assessment of symptom severity, duration, and patterns of onset and resolution help to determine disease severity and urgency of intervention. Unstable angina, for example, requires more immediate treatment than stable angina.

- *Symptoms:* Chest, neck, or arm pressure or pain with exertion or at rest, including frequency or severity. Exertional dyspnea.
- *History:* Hypertension, prior myocardial infarction, hyperlipidemia. Prior CABG or other cardiothoracic surgery (obtain operative note and discharge summary if possible).
- *Medications:* ASA, statin or other anti-lipid drugs, beta-blockers, anti-arrhythmics, antihypertensives, specifically including ACE inhibitors or ARBs.

– **Pulmonary:**

Symptoms: Wheezing or shortness of breath (exertional or at rest); cough, including onset and chronicity; use of CPAP.

History: Asthma, COPD; any prior hospitalization for respiratory difficulty, including admission to the ICU or prior intubation; difficult airway issues. History of chest or chest wall surgery or trauma, spontaneous pneumothorax, pleural effusion, empyema, chest tube placement, or radiation to the chest (e.g., for lymphoma, lung cancer, breast cancer) [3], all of which might impact internal mammary artery harvest difficulty or usability.
Medications: Inhalers, oral bronchodilators, steroids. Home oxygen.

– **Gastrointestinal:**

Signs and symptoms: Recent or active GI bleeding; severe reflux (e.g., "heartburn"); ascites; jaundice.
History: History of liver disease including any hepatitis, cirrhosis, or varices. History, source, and severity of GI bleeding (hospitalization, ICU admission, need for transfusion). Bowel obstruction (in the event patient has slow postoperative return of bowel function). Chronic malnutrition or inflammatory bowel disease (e.g., Crohn's or ulcerative colitis). History of cholecystitis or asymptomatic cholelithiasis (postoperative cholecystitis can arise in the setting of known gallbladder disease or as acalculous cholecystitis related to cardiopulmonary bypass, "low-flow" ischemia, or critical illness).
Medications: Agents for inflammatory bowel diseases such as Crohn's or ulcerative colitis, including steroids and biological agents.

– **Urological:**

Symptoms: Active symptoms of a urinary tract infection (UTI)
History: Frequent UTIs, hematuria, renal insufficiency or failure; prior or active dialysis; prostate obstruction (e.g. difficulty voiding) or surgery, including TURP or prostatectomy—may indicate need for urological assistance for Foley placement.
Medications: Finasteride, doxazosin, or other alpha blockers, as these may have affect blood pressure.

– **Hematological:**

Symptoms: Easy bleeding or bruising.
History: History of bleeding that was difficult to stop; spontaneous epistaxis, heavy menstrual bleeding; spontaneous blood clots in the extremities or brain. Family history of bleeding or blood clotting issues. Any prior exposure to anticoagulants, especially. History of heparin-induced thrombocytopenia (HIT). History of genetic polymorphisms that might affect anticoagulant or antiplatelet drug metabolism or effectiveness (e.g., CYP2C19).
Medications: Antiplatelet or anticoagulant agents, other agents with bleeding potential such as fish oil supplements.

– **Endocrine:**

History: Diabetes, hyper or hypo-thyroidism, adrenal insufficiency.
Medications: Metformin, insulin, GLP-1 agonists, SGLT2 inhibitors, other hypoglycemics. Levothyroxine or medications for hyperthyroidism. Steroids.

– **Extremities:**

History: Surgery or trauma. Past or current fistulas for dialysis. Prior history of non-healing wounds or ulcers.
Upper or lower extremity surgery or trauma (availability of saphenous vein and radial artery conduits).
Symptoms: Lower extremity edema; exertional discomfort of buttocks or lower extremities; pain at rest (e.g., at night), numbness, or tingling.

Physical Exam

- Together with the history and the results of preoperative testing, a carefully conducted physical exam can help guide pre- and postoperative management, suggest the need for additional testing, and optimize outcomes.
- **Vital Signs:** BP (both arms if left or right internal mammary artery—LIMA or RIMA—planned), pulse rate and rhythm, pulse oximetry (O_2 saturation on room air).
- **General:** Overall appearance, alertness, muscle mass, habitus including obesity or cachexia.
- **Neurological**: The neurologic exam is important not only to detect issues that might need preoperative intervention, but also to serve as a detailed baseline against which postoperative findings can be compared if there is concern for a possible stroke or other neurologic injury.

  Neuro exam should include gentle palpation of bilateral carotid pulses (avoid rubbing or excessive pressure that could lead to bradycardia or carotid embolization); auscultation for bruits; cranial nerve evaluation, including eye movement, pupillary symmetry, size, and reactivity, and gross visual acuity. Bilateral upper and lower extremity strength, sensation, and symmetry.
- **Cardiovascular:** Heart murmur; arrhythmia, including tachycardia, bradycardia, or irregularly irregular pattern. Pulse exam, including bilateral radial, femoral, and dorsalis pedis (DP) or posterior tibial (PT) pulses. Allen test or ultrasound evaluation of palmar circulation if possibility of radial artery graft for CABG**.**
- **Pulmonary**: Quality of breath sounds—coarse, wheezing, diminished, absent, symmetric.
- **Abdomen:** Palpable masses, including pulsatile masses concerning for aneurysm. Tenderness. Bowel sounds. Scars from previous surgery (if so, attempt to document precise procedure performed, when, and why).

- **Extremities:** Lower extremity edema, lack of hair or other indications of peripheral vascular disease, such as changes in skin color (characteristic dependent rubor or "bronze" skin coloring), or non-healing wounds; rashes; incisions; diminished sensation. Varicose veins.
- **Skin**: Rashes, pustules, cellulitis, ecchymoses, or suspicious lesions, especially in potential incision areas (e.g., sternotomy, thoracotomy, radial artery, saphenous veins, groin.
- **Dental:** Quality of dentition; concern for infection or carries, gum disease.

Laboratory and Imaging Studies

Labs

- **Hematologic**
  - CBC and differential

    Leukocytosis (occult infection); anemia; thrombocytopenia (possible increased risk of excessive bleeding). Severe or unexplained anemia or significant thrombocytopenia may warrant hematology consultation.

  - PT/INR; PTT to evaluate coagulation.
  - Special coagulation studies ordered at the discretion of hematology if patient or family history of bleeding or clotting issues.
- **Metabolic**
  - Complete metabolic profile (CMP); this is a single test that includes both a basic metabolic panel (BMP) and liver function tests (LFTs).

    Creatinine, electrolytes to evaluate renal function.
    Abnormal electrolytes or uremia in the setting of renal failure may indicate need for preoperative dialysis.
    LFTs evaluate hepatic function and/or hepatitis, potential bleeding, or clotting issues.
- **Endocrine**
  - HgbA1c to evaluate for diabetes; if Hb > 8%, consider preoperative endocrine consult.
- **Lipids**
  - Lipid panel for inpatients.
- **Cardiac**
  - Troponin levels for inpatients.

- **Renal**
  - Estimation of function based on CrCl, calculated from BMP.
  - If history of renal disease, consider a preoperative renal consult.
  - If already on dialysis, arrange for preoperative dialysis with renal team.
- **Hepatic**
  - Patients with preoperative liver disease who are undergoing either CABG or valve surgery have an increased rate of mortality (up to five-fold increase), as well as an increased risk for infectious complications, renal or respiratory dysfunction, and bleeding [4].
  - Preoperative albumin and INR/PT may suggest deficits in synthetic liver function.
  - If history or laboratory values suggest cirrhosis or liver dysfunction, follow-up imaging is recommended.
  - Cirrhosis can be evaluated with right upper quadrant ultrasound or CT scan.
  - Child-Turcotte-Pugh score or model for end-stage liver disease (MELD) score can provide guidance regarding the risk of cardiac surgery. Liver disease is included as a factor in the 2018. STS risk calculator, but not in EuroScore.
  - MELD is calculated directly from objective laboratory data; Child-Turcotte-Pugh score includes subjective assessments of degree of ascites and presence or absence of encephalopathy, in addition to laboratory values.
  - Risk of mortality and complications both increase with increasing Child-Turcotte-Pugh class. A systematic literature review of cases up to 2014, which included 19 papers and 638 cirrhotic patients, reported 30 day and 1-year mortality rates by Child-Pugh score [5]:

    Class A—9% (30 day); 27.2% (1 year)
    Class B—37.7% (30 day); 66.2% (1 year)
    Class C—52% (30 day); 78.9% (1 year)

  - In general, patients in Class A may be operated on with proper management [5, 6], whereas Class B and C disease may be considered a relative contraindication [7] who should only be approached with high caution [5].
  - In many cases, the use of cardiopulmonary bypass in patients with liver disease may worsen outcomes for cirrhotic patients [4, 6], whereas off-pump cardiac surgery in cirrhotic patients did not increase mortality unless liver dysfunction was severe, where severity of disease was inferred by collateral data from ICD-9 codes [8].

Infectious Disease/Microbiology

- MSSA PCR swab.
- MRSA swab: if positive, treat with mupirocin BID for 10 doses (5 days).
- COVID-19 PCR test performed in accordance with local or hospital guidelines.

- Routine urinalysis, but culture and sensitivity only in patients who are symptomatic or actively demonstrating signs of potential UTI (fever, hematuria, dysuria). If UTI found, treat according to patient history and/or local antibiotic resistance patterns.

Chest X-Ray (CXR)

- All patients should have a preoperative CXR.
  - Current admission for inpatients.
  - Within 3 months for outpatients—includes outside hospital-provided films that can be viewed in the electronic medical record.
- Identify occult pneumonia, document full lung expansion bilaterally; evidence of interstitial lung disease or fibrosis. Check for pleural effusions or other abnormal findings.
  - If pneumonia identified, surgery would be delayed unless emergent; initiate antibiotics.
  - If incidental new or previously undiagnosed pulmonary nodules are identified, must follow up with a pre-op CT chest. *Failure to follow up incidentally discovered lung nodules, which may be malignant, is a common and preventable failure mode in healthcare.*

Electrocardiogram (ECG)

- All patients, during current admission or within 30 days for outpatients.
- Identify signs of myocardial infarct (e.g., Q waves, poor R wave progression, or ST elevation), ischemia (e.g., ST depression), or arrhythmia, including tachycardia or bradycardia.
- Conduction abnormalities, such as right or left bundle branch block, QT prolongation (which may predispose to lethal arrhythmias).
- New changes, concerning findings, or extremes of heart rate may require further evaluation or interventions, including cardiology or electrophysiology consultation.

Chest CT

- All redo sternotomy patients; otherwise at discretion of surgeon.
- When performed for aortic aneurysmal disease, should be done with EKG gating to minimize motion artifact in the aortic root and ascending aorta.

- If prior sternotomy, assess proximity of cardiac structures to posterior sternum and the risk of inadvertent injury during sternal re-entry.
- Assess for severe ascending aortic calcification that could complicate cannulation for bypass and lead to consideration of alternative arterial cannulation approaches or off-pump procedures.
- Identify aortic aneurysmal disease, which could also complicate cannulation or require concomitant repair.

Computed Tomography Angiography (CTA) of the Coronary Arteries

- May be used to assess coronary arteries for planned TAVR or ascending aortic cases.
- The severity of coronary artery disease is communicated as a CAD-RADS score (coronary artery disease reporting and data system); the scale is zero to five, with five signifying the most severe disease.
- RADS 1–2 correlates to 1–49% stenosis and usually requires no further testing. If RADS ≥3, a patient should undergo cardiac catheterization and angiography [9]; note that the imaging findings, reported score, and associated recommended action should always be placed in clinical context.
- Coronary artery stenosis; calcification of valves, aortic root, and ascending aorta [1]. The quality of the aorta is important when planning cannulation for bypass.

Echocardiography

- Typically will already have been performed prior to CT surgery consultation; if not, ordered at surgeon discretion.
- Evaluation of LV and RV systolic and diastolic function, ejection fraction, regional wall motion abnormalities, chamber size, intracardiac shunts, valve stenosis and/or regurgitation, pericardial effusion or restriction, intracardiac thrombi, ascending aortic pathology (e.g., dilatation, aneurysm, protruding mural deposits that may increase the risk of stroke) [1].

Stress Testing

- Generally, patients who have been referred to cardiac surgery have already had physiologic or anatomic studies including echocardiograms and cardiac catheterization with coronary angiograms.

- Stress tests are most often ordered by outpatient providers, such as a primary care provider (PCP), or in the context of presentation to the Emergency Department. A stress test is the best noninvasive test available to providers in these settings.
- Assesses the ability of the heart to respond to increased oxygen demand; inability of the heart to increase perfusion with increased demand may be indicative of coronary occlusion.
- Testing may use exercise (treadmill) or pharmacologically induced stress (dobutamine, regadenoson) to increase cardiac demand.
- Cardiac assessment with radionucleotide myocardial perfusion imaging during stress (known as a nuclear stress test) is more sensitive and specific than ECG-based assessment since it shows blood flow and visualizes the entirety of the heart muscle.
- Limitations exist to both the sensitivity and specificity of exercise tolerance ECG tests. Both false positives and false negatives can arise due to confounding comorbidities as well as the pretest probability of coronary artery disease [1].

Cardiac Catheterization with Coronary Angiography

- Diagnostic test of choice for most cardiac disease [1]. Additional details on the procedures and its application may be found in Bojar [1] and Lilly [2].
- Access usually obtained via the radial artery or femoral artery.
- If radial artery graft planned for CABG, notify cardiology team to avoid accessing or manipulating the artery intended for grafting, if possible.
- Usually performed with iodinated IV contrast; if patient has a contrast allergy, can be performed with carbon dioxide, which is radiopaque.
- Patients are anticoagulated for the procedure, typically with heparin (unless known history of heparin-induced thrombocytopenia).
- The heart is evaluated from multiple views to identify right or left coronary dominance, evaluate coronary artery stenoses, determine and quantify valvular stenosis or regurgitation, and estimate ventricular function.
- Stenosis >50% is considered clinically significant, especially for the left main coronary artery. Maximum blood flow is limited by a lesion that causes >70% occlusion; perfusion at rest is impaired if the stenosis is >90% [1, 2]. Fractional Flow Reserve, instantaneous wave-free ratio (iFR), or coronary intravascular ultrasound (IVUS) imaging are also sometimes employed to better define the extent and physiologic significance of stenoses.
- For CABG, assess quality of target vessels beyond major stenoses.
- Typically not performed in type A aortic dissection (surgical urgency and potential technical issues related to the dissected true lumen) and in cases of endocarditis (to avoid disruption of vegetations and possible septic emboli) [1].

Viability Studies

- Designed to assess the composition, perfusion, and metabolism of areas of the heart that may have been affected by ischemia or infarct.
- Goal is to distinguish between areas of the myocardium that have necrosed and are permanently non-viable vs. those that are "hibernating." The latter are persistently dysfunctional due to chronically limited blood flow but are not dead and may regain function after successful revascularization [2]. "Stunned" myocardium is characterized by an extended period of myocardial dysfunction despite restoration of blood flow [2].
- Imaging modalities include single-photon emission CT (SPECT-CT) with technetium 99 or thallium; SPECT or positron emission tomography (PET) with fluorodeoxyglucose uptake; dobutamine stress echo (DSE); or delayed enhancement magnetic resonance imaging (MRI). MRI may be most useful for detection of hibernating myocardium, as late gadolinium enhancement may have a sensitivity of up to 95% and dobutamine stress MR may have a specificity of up to 91% [10].
- Theoretically, these techniques help to predict which patients are most likely to benefit from revascularization, though the degree of benefit or extent of recovery often remain unknown until the patient recovers postoperatively.
- Viability studies are not routinely used in current practice but may be helpful in a carefully selected subset of patients with advanced age, significant comorbidities, or other factors where the amount of potentially recoverable myocardium may be an important factor in surgical decision-making [10, 11].

Cardiopulmonary Exercise Testing (CPET)

- Typically completed during cardiology evaluation and prior to cardiac surgery consultation, and includes measurement of VO_2 (oxygen uptake), respiratory quotient (respiratory exchange ratio), and anaerobic threshold.
- Most useful to discriminate between cardiac and non-cardiac pathology when the patient's history and other testing (e.g. echocardiogram or angiogram) are inconclusive regarding the etiology of the patient's symptoms [12]. Important findings include whether the patient fatigues prior to reaching the ventilatory anaerobic threshold (VAT, the point at which oxygen supply cannot meet the oxygen demand of exercising muscle) [12].
- VAT can be measured invasively (serum lactic acid levels) or non-invasively (based on plots of oxygen and carbon dioxide consumption).
- These tests should be used with caution and closely monitored in patients who have low exercise tolerance and who may acutely worsen in the setting of exercise.

Pulmonary Function Tests (PFTs)

- Not routinely used, ordered at surgeon discretion.
- In addition to the standard risks of general anesthesia (e.g., positive pressure ventilation, decreased respiratory drive, and atelectasis), pulmonary risk is increased in cardiac surgery secondary to cardiopulmonary bypass (CPB), since neither the heart nor the lungs are perfused during bypass.
- Most often prescribed for patients on home oxygen; using multiple inhalers; or with history of asthma, COPD, or smoking (cigarettes, vaping, marijuana).
- Results can provide prognostic information with respect to the need for perioperative bronchodilators and additional chest physiotherapy, expected length of intubation, and possible need for tracheostomy.
- Also provides an additional data point for use in risk calculators (discussed below).

Carotid Artery Studies

- Preoperative evaluation of carotid artery patency aims to detect significant carotid artery stenosis, which may identify (1) patients at increased risk of perioperative stroke, and (2) patients who might benefit from carotid artery intervention (endarterectomy or stent) either prior to or concomitant with open heart surgery.
- Significant stenosis is correlated with increased risk of stroke [13]. However, whether carotid stenosis is an *indicator* of overall cardiovascular disease burden that is only *correlated* with increased stroke risk, or whether carotid stenosis is a *causative* factor for stroke after cardiac surgery remains unclear [13, 14].
- Perioperative stroke may result from carotid emboli, acute occlusion, or from cardiac or ascending aortic emboli [15].
- Incidence of stroke after cardiac surgery is directly associated with the type of operation; CABG has the lowest stroke rate, and combined valve + CABG has the highest stroke rate [16], perhaps related to the degree to which the aorta is manipulated during these procedures.
- Uncertain benefit of performing CEA at the time of CABG, compared to CABG alone with respect to stroke reduction [17], though this is complicated by the fact that CEA carries an independent risk of perioperative stroke [18].
- As neither randomized controlled trials nor meta-analyses have shown definitive benefit of *routine* screening in a *general population* of *asymptomatic* patients preparing for surgery, use of carotid screening is variable [19].
- However, multiple societies and cumulative consensus guides do recommend screening even asymptomatic patients if there are additional significant risk factors for stroke or severe cardiovascular disease, many of which often apply to the populations undergoing cardiac surgery, particularly those undergoing surgery for ischemic heart disease [14, 19].

- Predictors of stroke include age, diabetes, known cerebrovascular disease, presence of atrial fibrillation, and emergent procedure [16].
- Consistent with these stroke risk predictors, various sources suggest that carotid ultrasound screening should be considered for:
  - Age > 70 years [15] or >65 years [19]
  - History of TIA or stroke [15, 19]
  - Carotid bruit on examination [15, 19]
  - Left main coronary disease [15, 19]
  - History of smoking [19]
  - Known peripheral arterial disease [19]
- If a carotid duplex ultrasound is performed, it should be done in a vascular-accredited radiology lab [19].
- Carotid duplex scanning may sometimes be followed with MRI if additional anatomical information is needed, such as evaluation of the Circle of Willis.
- Current MGH practice:
  - Patients with known carotid or cerebrovascular disease, history of stroke or TIA, active symptoms, or bruit on exam. Notify the surgeon if stenosis >50% is seen in either right or left internal carotid arteries. Vascular surgery should only be consulted if requested by surgeon.

Vein Mapping: Vein mapping is used to identify possible graft options and is particularly important if the patient has a history of CABG or prior surgery or trauma to the lower extremities. Additional indications for vein mapping include varicose veins, history of vein stripping, comorbidities that have the potential to adversely affect the lower extremity veins (e.g., diabetes, hypertension), or small or large body habitus.

Risk Models and Categories

Cardiac surgery risk models estimate the patient-specific risk of death and serious complications for specific procedures (e.g., CABG, or AVR). They are used for patient counseling and shared decision-making, performance evaluation of a surgeon or hospital, and research.

The Society of Thoracic Surgeons (STS) Risk Models and Short-Term Risk Calculator [20]

- This is the most common and most widely used portfolio of risk models and is based upon data compiled by the STS Adult Cardiac Surgery Database (ACSD). The most recent risk model revisions (2018) were developed based on data from July 2011 to June 2014 and validated on July 2014–December 2016 data.

- Depending on the specific procedure, the calculator includes data from dozens to nearly 100 (for CABG mortality) predictor variables (available at https://www.sts.org/sites/default/files/Risk%20Model%20Variables%20-%202017%204.20.2%2006292020.pdf).
- Some variables that can have a significant impact on risk (e.g., 5-m walk test) are excluded due to excessive missing data in the benchmark population, and some rare but important comorbidities are difficult to model accurately.
- This risk calculator simultaneously reports nine individual risk scores to predict: risk of mortality; renal failure; permanent stroke; prolonged ventilation; deep sternal wound infection; reoperation; morbidity or mortality; short length of stay (<6 days); long length of stay (>14 days).
- Each category of the nine endpoints is specific to a particular procedure except for sternal wound infection, the risk of which is based on combined data for all procedures.
- The operations for which a score is provided include isolated CABG; isolated AVR; isolated MVR; AVG + CABG; MVR + CABG; MV repair; MV repair + CABG, multiple valve procedures ± CABG. (CABG, coronary artery bypass; AVR, aortic valve replacement; MVR, mitral valve replacement; MV, mitral valve).
- To learn more about the risk model: **https://www.sts.org/resources/risk-calculator.**
- To use the calculator: http://riskcalc.sts.org.

Other risk calculators used less commonly in the US include **EuroScore II** (euroscore.org/calc), as well as risk calculators developed by the **Northern New England Cardiovascular Disease Study Group,** and the **New York Cardiac Surgery Reporting system.**

Additional Preoperative Considerations

Dental Evaluation:

- Required for non-emergent valve surgery, repair of aneurysms, or any other procedures in which prosthetic valves, grafts, or other foreign materials will be implanted.
- Rationale: prevent bacteremia and seeding of prosthetic implants when brushing or flossing diseased teeth or gums postop.
- Outpatient: see own dentist, obtain "clearance" letter specifying no infection or acute concern for possibility of dissemination of infection.
- Inpatient: Panorex + urgent dental consult for preoperative evaluation.
  - If urgent operation and dental concern, requires antibiotic prophylaxis and postoperative continuation.
  - If not emergent and any teeth require removal, the oral and maxillofacial surgery team (OMFS) will be consulted by the primary team and the patient's surgery will be delayed at least 48 h from the time of extraction.
- Chlorhexidine is occasionally prescribed for preoperative oral cleansing.

Nutrition: Consider preoperative nutrition labs (e.g., albumin, pre-albumin, LFTs) if there is any indication of malnutrition, diminished appetite, weight loss, or low BMI. If appropriate, a nutrition consult, especially for inpatients, may be helpful to optimize the patient for surgery and subsequent postoperative recovery.

Smoking Cessation: Smoking cessation is encouraged in all patients, especially those with demonstrated cardiovascular disease. This includes the use of cigarettes, marijuana, vaping, and cigars. The sooner a patient can cease smoking, the better, but at least 4 weeks prior to anesthesia and surgery is preferrable. The use of smoking cessation medication can be used to facilitate this goal [1].

Penicillin Allergy Testing: At our center, the general recommendation is that all patients with a penicillin allergy listed in the chart who have not undergone formal testing should do so prior to surgery. This allows the physician and team to determine whether the patient has a true penicillin allergy or not (often they do not), which may significantly influence the antibiotic choice for preoperative prophylaxis or for use in the case of postoperative infections. Valve patients, especially those who undergo valve replacement (vs repair), who will require lifelong antibiotics before dental work, may especially benefit from expanded access to standard antibiotics such as amoxicillin if testing shows that they do not have a true penicillin allergy.

Consultations

The following is a list of recommendations used on the Cardiac Surgery service at Massachusetts General Hospital. For inpatients, consider the following consulting services if patient meets criteria listed. Discuss with surgeon before obtaining any consult:

- **Cardiology:** if MI or acute coronary syndrome during current admission.
- **Heart Failure**: if EF < 30% and/or new signs of CHF.
- **Pulmonology**: if history of severe COPD, on home oxygen, or complicated pulmonary history.
- **Vascular Surgery**: if new findings of carotid bruit or peripheral vascular disease.
- **Renal**: if baseline creatinine >2.0.
- **Neurology**: if residual neurologic deficit from previous neurologic event or new neurologic findings.
- **Addiction Services**: if patient has current or prior opioid use or substance use history.
- **Hematology**: if patient has history of heparin-induced thrombocytopenia (HIT) or new thrombocytopenia.
- **Endocrinology**: if patient has HgB A1c > 8.0% or newly diagnosed diabetes.

MGH Perioperative Medication Management

Patients should be advised regarding home medication use in advance of surgery as well in advance of the operation, as some medications may need to be held for several days prior to undergoing a cardiac operation. Details regarding types of medication, recommended timing for last dose, and brief explanation of physiological or biological rationale are provided below (Table 3.1). Additional detail regarding the preoperative management of commonly used medications may be found in Bojar [1]. Local practice regarding these medications may differ and may need to be modified based on specific clinical context. Additionally, these recommendations are not meant to supercede information provided by the FDA, manufacturers, or professional societies, which may change as new information becomes available.

N.B. We recommend the excellent 2018 review from the American College of Surgeons: "American College of Surgeons' Guidelines for the Perioperative Management of Antithrombotic Medication" for more detailed information regarding indications and pharmacokinetics of anticoagulant and antiplatelet agents, as well as further information regarding perioperative decision-making. Note that this document is *not* specific to cardiac surgery.

Table 3.1 Management of home medications in the immediate preoperative period

Medication class	Perioperative management	Rationale
Anticoagulants		
Warfarin (Coumadin) Oral	Stop 5 days prior to surgery *Determine whether a bridge to heparin is needed or not, based on the reason for anticoagulation and risk associated with time off anticoagulation*	• Inhibits vitamin K-dependent coagulation factors; need time for synthesis of new factors
Warfarin bridging	Hold 5 days prior to surgery Additional suggestions for use of heparin bridge, based on patient pathology: *(MGH practice, for outpatients)* • Afib only -> No bridge[a] • AFib + MR -> No bridge • AFib + MS -> Admit 3 days prior to OR for IV heparin & discuss with surgeon • Mechanical MVR -> Admit 2 days prior to OR for IV heparin & discuss with surgeon • Mechanical AVR -> Discuss with surgeon	• Replace warfarin with an anticoagulant with shorter half-life (heparin), to avoid risks that would be incurred by complete cessation.
Heparin IV	Stop on call to OR for non-VAD patients. Pre-op VAD patients stop 4 h before OR. Heparin remains running if IABP in place pre-op	• Heparin has a relatively short half-life. Stopping 4–6 h prior to surgery will restore adequate clotting ability to proceed with surgery

(continued)

Table 3.1 (continued)

Medication class	Perioperative management	Rationale
Bivalirudin (Angiomax) IV	Stop 4 h before if CrCl < 30; if normal stop 2 h before	• Direct thrombin inhibitor, cleared renally
Antiplatelet agents		
ASA (Oral)	Continue through day of surgery	• Aspirin is an irreversible inhibitor of platelet aggregation; new platelets must be generated from the bone marrow to be functional, requiring 7–10 days
Clopidogrel (Plavix) Oral	Hold 5 days (unless DES in last 6 months)	• Irreversible inhibitor of platelet aggregation; as above, need to wait for regeneration of functional platelets • DES have delayed endothelialization compared to BMS; exposure of blood to the stent material can increase risk of clot formation
Prasugrel (Effient) Oral	Stop 7 days prior to surgery	• Irreversible inhibitor of platelet aggregation; same as above
Eptifibatide (Integrilin) IV	Stop 3 h before OR	• Reversible glycoprotein IIb/IIIa inhibitor; short half-life
Cangrelor (Kengreal)	Stop 1–2 h prior to the OR	• Very short-acting, reversible platelet inhibitor
Direct Oral anticoagulants (DOACs)	• No definitive evidence for the use of heparin bridging [21] • At MGH, determination of whether a bridge to heparin is needed or not is based on reason for anticoagulation and risk associated with time off anticoagulation • Time to hold prior to OR is dependent on CrCl; for normal renal function, ~48h is usually sufficient [21] • Hold times will be extended for patients with impaired renal function	
Apixaban (Eliquis)	Stop 5 days prior to OR *(MGH practice)*	• Direct Xa inhibitor, renally cleared; half-life is 12 h, so need time to clear sufficiently
Rivaroxaban (Xarelto)	Stop 5 days prior to OR *(MGH practice)*	• Direct Xa inhibitor, renally cleared; half-life is 5–13 h, so need time to clear sufficiently

Table 3.1 (continued)

Medication class	Perioperative management	Rationale
ACE inhibitors, ARBs	Stop 2 days prior to surgery	• Decrease risk for intra-op and postop vasoplegia and kidney injury
Steroids	Continue to the day of surgery; if >10 mg/day and taking for at least 1 week, will need stress dose steroids intra-op + postoperative taper	• Patient may have adrenal suppression secondary to high-dose and/or chronic steroid usage and be unable to mount an endogenous, physiologic stress response
Diabetes medications		
Insulin	Continue normal regimen with the evening meal the night before surgery Hold the morning of surgery (Note—may additionally depend on whether first or second case)	• Avoid hypoglycemia
Metformin	Hold 2 days prior to OR	• Prevent hypoglycemia and avoid increased risk of metabolic acidosis
GLP-1 inhibitors	Hold at least 24 h prior to surgery	• Prevent hypoglycemia
SGLT2 inhibitors (Canagliflozin, dapagliflozin, empagliflozin)	Stop 7 days prior to surgery	• Avoid potential risk of ketoacidosis
DVT prophylaxis	Heparin 5000 units SC Q8 h or Lovenox 40 mg SC Q24 h should be ordered on all preoperative patients at the time of consultation unless they are on any form of IV anticoagulation or there is a contraindication. Last dose of SC heparin should be given evening before surgery. Lovenox, last dose given morning prior to surgery	• Mitigate thrombotic risk in patients with cardiovascular pathology
Anti-arrhythmics	Continue through day of surgery Amiodarone—consider TSH, PFTs if planning to continue for extended period postoperatively	• Maintain normal rhythm for hemodynamic stability; minimize cardiac metabolic stress • Amiodarone can adversely affect the thyroid and lungs, so a baseline assessment will help with monitoring if drug is used long term
NSAIDs	Stop 7 days prior to surgery	• Reversible inhibitors of platelet aggregation; recovery of platelet function in hours to days (based on agent half-life)

(continued)

Table 3.1 (continued)

Medication class	Perioperative management	Rationale
Beta-blockers	Continue regular dose day of surgery Consider changing regimen from long-acting home agent to fractionated, short-acting agents—e.g., convert long-acting metoprolol succinate (Toprol-XL) to metoprolol tartrate (Lopressor)	• Abrupt withdrawal (unless necessary for hypotension or bradycardia) may lead to rebound tachycardia, hypertension, or ischemia • Change to short acting allows tighter control and more flexibility if blood pressure has greater fluctuation in immediate peri-op and postoperative period
Vitamins and supplements, including fish oil, omega-3 fatty acids, ginger, garlic, ginkgo	Discontinue all at least 7 days prior to surgery	• Some may interfere with platelet function [1] • Avoid interactions with perioperative medications such as anxiolytics, sedatives, anesthetics, analgesics, and postoperative medications, including anticoagulants
Glaucoma drops	Check with anesthesiologist Generally best to administer day of surgery	• Prevent excessive elevation of intra-ocular pressure during prolonged cardiopulmonary bypass and potential ischemic neuropathy

ASA acetylsalicylic acid (aspirin), *ACE* angiotensin converting enzyme, *Afib* atrial fibrillation, *AVR* aortic valve replacement, *DES* drug-eluting stent, *DOAC* direct oral anticoagulant, *GLP-1* glucagon-like peptide-1, *IABP* intra-aortic balloon pump, *IV* intravenous, *MR* mitral regurgitation, *MVR* mitral valve replacement, *OR* operating room, *PFTs* pulmonary function tests, *SC* subcutaneous, *TSH* thyroid function studies, *VAD* ventricular assist device

[a] Bridge: replacing home anticoagulant with IV heparin since heparin has a shorter half-life than all of the oral anticoagulants. This allows the patient to remain on therapeutic anticoagulation as close to surgery as possible, to minimize the risk of thrombosis

Acknowledgments Thank you to Alysia Monaco, N.P., Chis Stager, P.A., and all the staff and team members of Massachusetts General Hospital Cardiac Surgery, for discussion and collation of local practice guidelines.

References

1. Bojar RM. Manual of perioperative care in adult cardiac surgery. 6th ed. Hoboken: Wiley; 2021.
2. Lilly LS, editor. Pathophysiology of heart disease: an introduction to cardiovascular medicine. 7th ed. Alphen aan den Rijn: Wolters Kluwer; 2021.
3. Ruff CT, O'Gara PT. Preoperative evaluation for cardiac surgery | Cardiac surgery in the adult, 5th ed. | AccessSurgery | McGraw Hill Medical. https://accesssurgery.mhmedical.com/content.aspx?bookid=2157&sectionid=164288767. Accessed 31 May 2022.

4. Araujo L, Dombrovskiy V, Kamran W, Lemaire A, Chiricolo A, Lee LY, Lemaire A. The effect of preoperative liver dysfunction on cardiac surgery outcomes. J Cardiothorac Surg. 2017;12:73. https://doi.org/10.1186/s13019-017-0636-y.
5. Jacob KA, Hjortnaes J, Kranenburg G, de Heer F, Kluin J. Mortality after cardiac surgery in patients with liver cirrhosis classified by the Child-Pugh score. Interact Cardiovasc Thorac Surg. 2015;20:520–30.
6. Modi A, Vohra HA, Barlow CW. Do patients with liver cirrhosis undergoing cardiac surgery have acceptable outcomes? Interact Cardiovasc Thorac Surg. 2010;11:630–4.
7. Morimoto N, Okada K, Okita Y. The model for end-stage liver disease (MELD) predicts early and late outcomes of cardiovascular operations in patients with liver cirrhosis. Ann Thorac Surg. 2013;96:1672–8.
8. Gopaldas RR, Chu D, Cornwell LD, Dao TK, Lemaire SA, Coselli JS, Bakaeen FG. Cirrhosis as a moderator of outcomes in coronary artery bypass grafting and off-pump coronary artery bypass operations: a 12-year population-based study. Ann Thorac Surg. 2013;96:1310–5.
9. Cury RC, Abbara S, Achenbach S, et al. Coronary artery disease—reporting and data system (CAD-RADS): an expert consensus document of SCCT, ACR and NASCI: endorsed by the ACC. JACC Cardiovasc Imaging. 2016;9:1099–113.
10. Ipek EG, Blumenthal R, Sharma G. Non-invasive assessment of myocardial viability. In: Latest in cardiology; 2020. https://www.acc.org/latest-in-cardiology/articles/2020/08/14/07/44/non-invasive-assessment-of-myocardial-viability.
11. Garcia MJ, Kwong RY, Scherrer-Crosbie M, Taub CC, Blankstein R, Lima J, Bonow RO, Eshtehardi P, Bois JP. State of the art: imaging for myocardial viability: a scientific statement from the American Heart Association. Circ Cardiovasc Imaging. 2020;13:e000053. https://doi.org/10.1161/HCI.0000000000000053.
12. Albouaini K, Egred M, Alahmar A, Wright DJ. Cardiopulmonary exercise testing and its application. Postgrad Med J. 2007;83:675–82.
13. Li Y, Walicki D, Mathiesen C, Jenny D, Li Q, Isayev Y, Reed Iii JF, Castaldo JE. Strokes after cardiac surgery and relationship to carotid stenosis. Arch Neurol. 2009;66(9):1091–6.
14. Naylor AR, Ricco JB, de Borst GJ, et al. Editor's choice—management of atherosclerotic carotid and vertebral artery disease: 2017 clinical practice guidelines of the European Society for Vascular Surgery (ESVS). Eur J Vasc Endovasc Surg. 2018;55:3–81.
15. Santarpino G, Nicolini F, de Feo M, et al. Prognostic impact of asymptomatic carotid artery stenosis in patients undergoing coronary artery bypass grafting. Eur J Vasc Endovasc Surg. 2018;56:741–8.
16. Sultan I, Bianco V, Kilic A, et al. Predictors and outcomes of ischemic stroke after cardiac surgery. Ann Thorac Surg. 2020;110:448–56.
17. Klarin D, Patel VI, Zhang S, et al. Concomitant carotid endarterectomy and cardiac surgery does not decrease postoperative stroke rates. J Vasc Surg. 2020;72:589–596.e3.
18. Giri J, Nathan A. How should we address carotid artery stenosis around the time of open-heart surgery? JACC Cardiovasc Interv. 2017;10(3):299–301.
19. AbuRahma AF, Avgerinos ED, Chang RW, et al. Society for Vascular Surgery clinical practice guidelines for management of extracranial cerebrovascular disease. J Vasc Surg. 2022;75:4S–22S.
20. Shahian DM, Jacobs JP, Badhwar V, et al. The Society of Thoracic Surgeons 2018 adult cardiac surgery risk models: part 1—background, design considerations, and model development. Ann Thorac Surg. 2018;105:1411–8.
21. Hornor MA, Duane TM, Ehlers AP, Jensen EH, Brown PS, Pohl D, da Costa PM, Ko CY, Laronga C. American College of Surgeons' guidelines for the perioperative management of antithrombotic medication. J Am Coll Surg. 2018;227:521–536.e1.

Chapter 4
Echocardiography

Jordan Secor and Evin Yucel

Transthoracic Echocardiography (TTE)

- Non-invasive, performed with handheld probe on chest surface
- Allows evaluation of the structure and function of the atria, ventricles, valves, great vessels, and pericardium
- Ultrasound (US) probe emits US wave and detects US waves as they are reflected through patient's tissue, air, and fluid within body back to the probe
- US machine software creates images and measurements using the detected US waves based on the known frequency and intensity of the energy emitted by the probe
- Doppler echocardiography displays blood flow in cardiac chambers and vessels based on the change in frequency imparted to a sound wave by the movement of erythrocytes and is used to quantify hemodynamic effects of valve lesions and intracardiac shunts
- Pulsed Doppler and continuous wave Doppler can be utilized to measure pressure gradient change in between two cardiac chambers. Color Doppler permits the assessment of the presence and direction of blood flow
- Performed at the bedside without sedation
- Anesthesiologists, intensive care physicians, surgeons, and cardiologists perform TTE at bedside and in operating room (OR)
- Provides real-time information; much faster to perform, more accessible, less expensive, and lower risk than CT, MRI, or conventional angiography
- Limited by the knowledge and experience of the providers capturing and interpreting the images

J. Secor · E. Yucel (✉)
Massachusetts General Hospital, Boston, MA, USA
e-mail: jsecor@mgb.org; eyucel@mgh.harvard.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_4

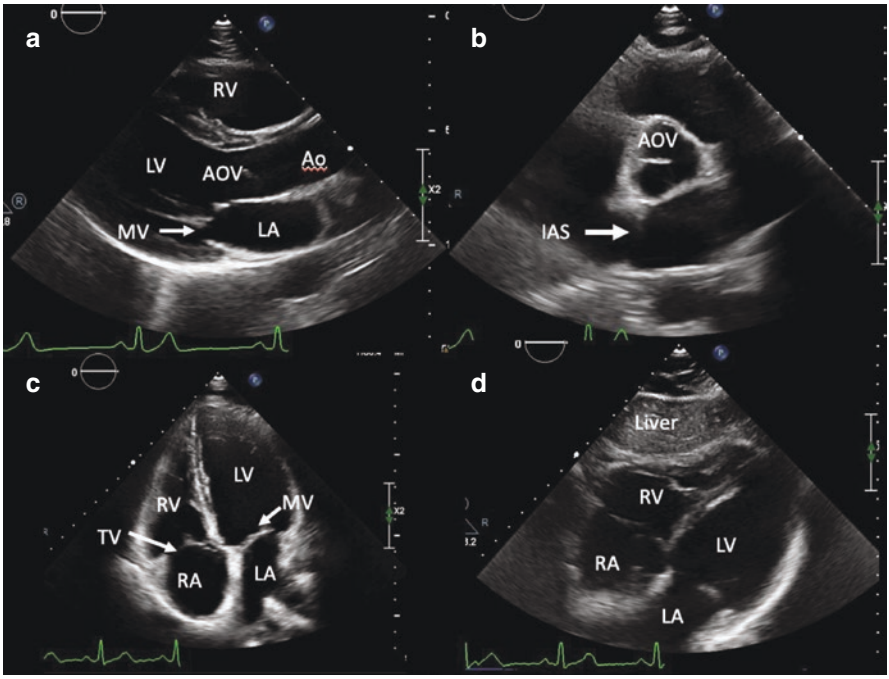


Fig. 4.1 Transthoracic echocardiography views. (**a**) Parasternal long axis view showing left ventricle (LV), left atria (LA), mitral valve (MV), aortic valve (AOV), aorta (Ao) and right ventricle (RV). (**b**) Parasternal short axis view showing aortic valve (AOV) and interatrial septum (IAS). (**c**) Apical 4 chamber view showing both ventricles (RV and LV), both atria (LA and RA) along with tricuspid valve (TV) and mitral valve (MV). (**d**) Subcostal view showing all chambers of the heart and the inferior aspect of the right ventricle with liver in view

- "Formal" TTE studies performed by ultrasound technicians are interpreted by cardiologists
- Standard TTE windows are parasternal, apical, subcostal, and suprasternal (Fig. 4.1)
- Cardiac surgeons must be familiar with the applications, interpretation, and limitations of TTE

Transesophageal Echocardiography (TEE)

- Ultrasound probe is placed into the esophagus to visualize structures in more detail and requires sedation
- Similar to TTE, TEE relies on the emission and detection of US waves

- Requires specialized training, typically only cardiac anesthesiologists and cardiologists perform TEE
- Carries roughly ~1 in 10,000 risk of esophageal perforation
- Significantly better than TTE at visualizing posterior structures (left atrium, left ventricle, mitral valve, descending aorta) and prosthetic valves
- Performed in the OR before, during, and after many cardiac surgery operations
- Can be used to make decisions regarding the adequacy of an operation and evaluating possible complications. Examples include evaluating for aortic dissection after cannulation of aorta for cardiopulmonary bypass, determining the extent of air in the pulmonary veins and left ventricle prior to separating from cardiopulmonary bypass, and determining presence or absence of paravalvular leak after valve replacement.
- Acoustic windows include esophageal, deep esophageal, and transgastric (Fig. 4.2)

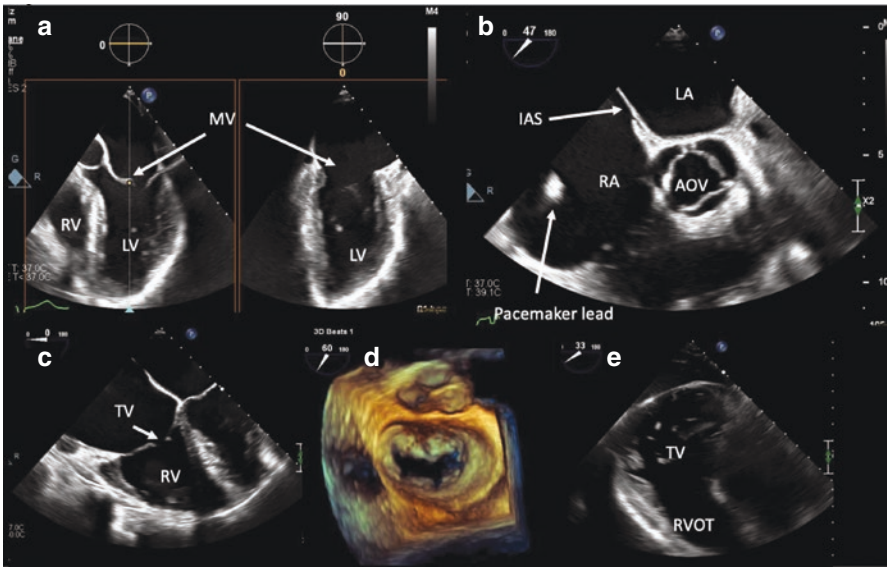


Fig. 4.2 Transesophageal echocardiography views. (**a**) Mid-esophageal 4-chamber view of the left ventricle (LV), right ventricle (RV) and mitral valve (MV). There is a x-plane through the middle of the heart which shows an orthogonal view of the LV. (**b**) Mid-esophageal short axis view of the aortic valve (AOV) with interatrial septum (IAS) in view. In this patient, there is pacemaker lead in the right atria (RA). (**c**) Deep esophageal view showing the right ventricle (RV) and tricuspid valve (TV). (**d**) 3-dimensional view of the mitral valve. (**e**) Transgastric view focusing on the right heart with right ventricular outflow tract (RVOT and tricuspid valve (TV)

Echocardiography for Common Cardiac Pathologies

Valvular Aortic Stenosis (AS)

- Calcification and/or fibrosis of the aortic valve which leads to obstruction of blood flow from the left ventricle into the aorta.
- Typically, the result of one of two pathologies:
  - Calcification of a normal trileaflet or bicuspid valve (most common cause)
  - Rhematic aortic aortic stenosis
- Severity of AS is categorized based on the criteria in Table 4.1
- Patients with low left ventricular ejection fraction (LVEF) may not have sufficient flow to generate high velocity across the aortic valve
- Maximum aortic jet velocity or mean gradient may underestimate severity of AS in patients low LVEF
- Dimensionless index (DI) can be utilized to aid in diagnosis of severity of AS in low LVEF.
- DI = velocity time index (VTI) of the left ventricular outflow tract/aortic VTI

Aortic Regurgitation (AR)

- Incompetency of the aortic valve results in blood traveling backwards from the aorta into the left ventricle during diastole
- Typically results from degeneration of the aortic valve leaflets or dilation of the aortic root
- Myxomatous disease (progressive leaflet thinning and redundancy) can cause AR
- Endocarditis (infection of the valve) can lead to AR by causing leaflet degeneration and/or preventing normal leaflet coaptation
- Aneurysmal dilation of the aortic root may lead to insufficient aortic valve coaptation and regurgitation

Table 4.1 Aortic stenosis severity classification

	Mild	Moderate	Severe
Vmax (m/s)	2.6–2.9	3.0–4.0	≥4.0
Mean gradient (mmHg)	<20	20–40	≥40
AVA (cm^2)	>1.5	1.0–1.5	<1.0
AVAi (cm^2/BSA)	>0.85	0.6–0.85	<0.6
Dimensionless index	>0.5	0.25–0.5	<0.25

Vmax maximum velocity by Doppler, *AVA* aortic valve area, *AVAi* indexed AVA; should be only used for patients who has extreme small body habitus, *BSA* body surface area (m^2)
Adapted from American Society of Echocardiography guidelines on valve assessment [1, 2]

Table 4.2 Aortic regurgitation severity classification

	Mild	Moderate	Severe
Regurgitant jet to LVOT ratio (%)	<25	25–64	≥65
Vena contracta[a] width (cm)	<0.3	0.3–0.6	≥0.6
Regurgitant volume (mL/beat)	<30	30–59	≥60
Regurgitant fraction (%)	<30	30–49	≥50
Regurgitant orifice (cm^2)	<0.1	0.1–0.29	≥0.3

LVOT left ventricular outflow tract
Adapted from American Society of Echocardiography guidelines on valve assessment [3]
[a] Width of the regurgitant jet at the orifice by color Doppler

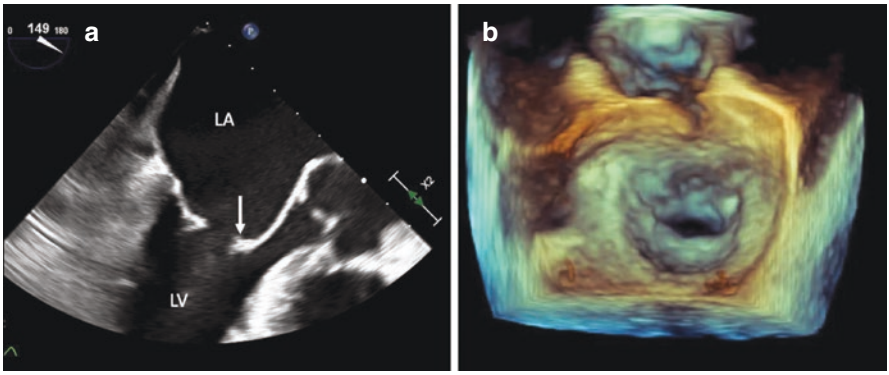


Fig. 4.3 Transesophageal view of a patient with rheumatic mitral stenosis. (**a**) Zoom image of mitral valve from a mid-esophageal view on transesophageal echocardiogram (TEE) of a patient with rheumatic heart disease. There is doming of the anterior mitral valve leaflet (white arrow) with almost immobile posterior leaflet leading to left ventricular (LV) inflow obstruction. (**b**) 3-D TEE image of mitral valve from the same patient showing fusion of the medial and lateral commissures

- AR can be concomitant with AS when calcified aortic valve leaflets neither open nor close normally
- Severity of AR is characterized by the criteria listed in Table 4.2.

Mitral Stenosis (MS)

- MS is restriction of blood flow across the mitral valve that limits flow from the left atrium into the left ventricle
- Rheumatic heart disease (antibody-mediated inflammation of valve secondary to streptococcus infection) is the most common cause of MS in the developing world
- Commissural fusion, thickening of the mitral valve leaflets and chords, doming of the anterior leaflet and immobile posterior leaflet are hallmark echocardiographic findings of rheumatic mitral valve disease (Fig. 4.3)

- Severe mitral annular calcification (MAC) is a less common cause, which occurs primarily in elderly patients
- Severity of rheumatic MS is characterized by the criteria listed in Table 4.3.

Mitral Regurgitation (MR)

- MR is a failure of the mitral valve leaflets to close completely which leads to backward blood flow from left ventricle into the left atria during systole
- MR is characterized as primary (intrinsic abnormality of the valve leaflet tissue) or secondary (abnormality of the mitral valve annulus or the left ventricle)
- Most common cause of primary MR is mitral valve prolapse followed by endocarditis and rheumatic heart disease
- Secondary MR can be caused by coronary artery disease, dilated cardiomyopathy, or long-standing atrial fibrillation
- Papillary muscle rupture leading to acute severe MR is caused by a myocardial infarction and is a surgical emergency (Fig. 4.4)
- Severity of MR is characterized by the criteria in Table 4.4.

Table 4.3 Rheumatic mitral stenosis severity classification

	Mild	Moderate	Severe
Valve area (cm^2)	>1.5	1.0–1.5	<1.0[a]
Mean gradient (mmHg)	<5	5–10	>10
Pulmonary artery mean pressure (mmHg)	<30	30–50	>50

Adapted from American Society of Echocardiography guidelines on valve assessment [1]
[a] Severe rheumatic mitral stenosis is defined as a mitral valve area ≤1.5 cm^2 based on the most recent ACC/AHA guidelines on management of valvular heart disease [4]

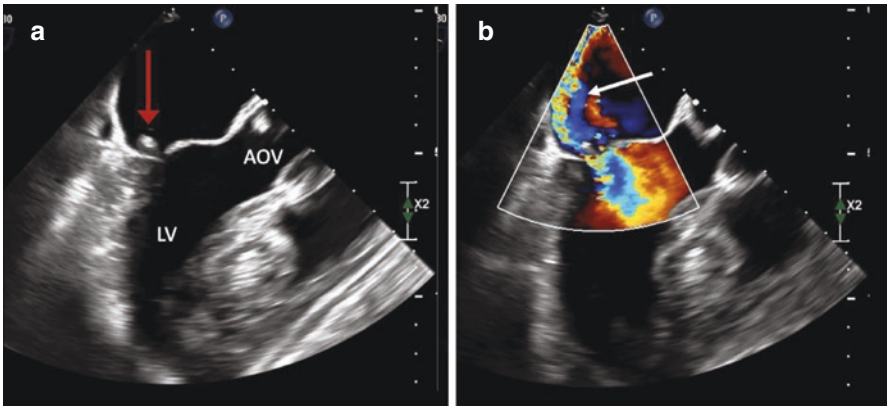


Fig. 4.4 Mitral regurgitation due to papillary muscle rupture. (**a**) Mid-esophageal long axis view on TEE showing mitral valve in a patient who has papillary muscle rupture. Red arrow shows the papillary muscle that prolapses into the left atria during systole. (**b**) Same view with color Doppler showing severe eccentric mitral regurgitation (white arrow) during systole. *LV* left ventricle, *AOV* aortic valve

Table 4.4 Mitral regurgitation severity classification

	Mild	Moderate	Severe
Vena contracta[a] width (cm)	<0.3	0.3–0.69	≥0.7
Regurgitant volume (mL/beat)	<30	30–59	≥60
Regurgitant fraction (%)	<30	30–49	≥50
Regurgitant orifice (cm^2)	<0.2	0.2–0.39	≥0.4

Adapted from American Society of Echocardiography guidelines on valve assessment [3]
[a] Width of the regurgitant jet at the orifice by color Doppler

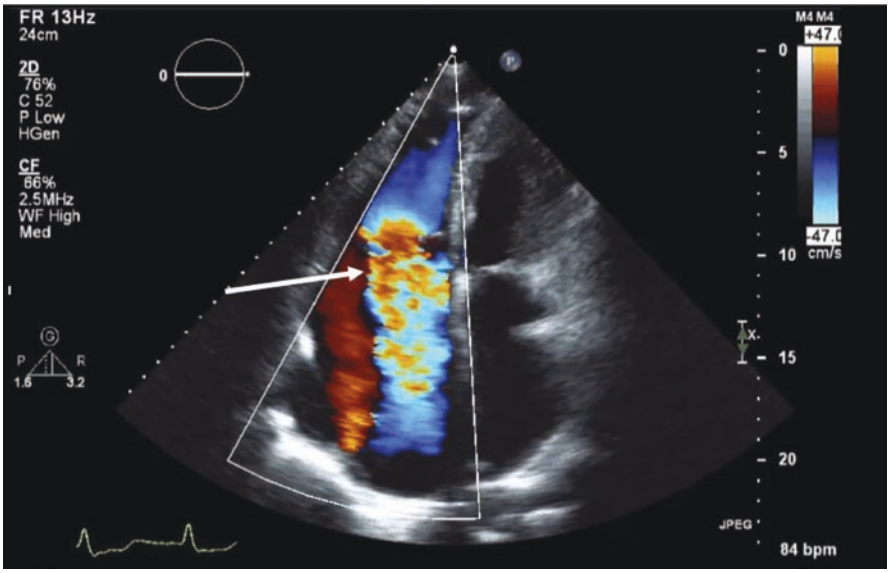


Fig. 4.5 Tricuspid regurgitation. Apical 4 chamber image on TTE with color Doppler flow which shows severe tricuspid regurgitation. White arrow shows the regurgitant jet

Tricuspid Regurgitation (TR)

- TR is a failure of the tricuspid valve leaflets to close completely which leads to backward blood flow from right ventricle into the right atria during systole (Fig. 4.5)
- Like MR, TR has primary and secondary causes
- Etiologies of primary TR include prolapse, endocarditis, carcinoid disease, injury to the leaflets from implantable cardiac devices (i.e., pacemaker leads), trauma, or injury to the leaflets during right heart biopsies.
- Secondary TR is more common and can be caused by
  - Left sided heart failure
  - Aortic, mitral, and/or pulmonic valvulopathies
  - Ventricular septal defect

Table 4.5 Tricuspid regurgitation severity classification

	Mild	Moderate	Severe
Vena contracta[a] width (cm)	<0.3	0.3–0.69	≥0.7
Regurgitant volume (mL/beat)	<30	30–59	≥60
Regurgitant fraction (%)	<30	30–49	≥50
Regurgitant orifice (cm^2)	<0.2	0.2–0.39	≥0.4

RA right atrium
Adapted from American Society of Echocardiography guidelines on valve assessment [3]
[a] Width of the regurgitant jet at the orifice by color Doppler

  - Pulmonary hypertension

- Severity of TR is characterized by the criteria listed in Table 4.5.

Left Ventricular Function

- LVEF is the most used metric to quantify the function of the left ventricle
- LVEF = stroke volume (SV)/left ventricle end-diastolic volume (LVEDV)
- SV = left ventricle end-systolic volume (LVESV) − LVEDV
- Normal LVEF is 54–75%.
- In OR, TEE is frequently used to assess ventricular function when weaning from cardiopulmonary bypass
- Temperature, cardioplegia, electrolytes, air within heart or coronaries, conduction abnormalities, and volume status all affect ventricular function after cardiac surgery
- Remember: echocardiography captures structure and function at one moment in time
- The heart is a dynamic organ that changes quickly in response to medications, exercise, environment, conduction abnormalities, and surgical intervention
- Real-time echocardiography is an excellent way to monitor cardiac function before, during, and after cardiac surgery

Tamponade and Constriction

- The pericardial space is normally a potential space that contains only trace amount of clear, colorless pericardial fluid
- A pericardial effusion is a pathologic accumulation of fluid in the pericardial space
- Pericardial effusion can result from accumulation of blood (hemopericardium), response to malignancy, or excessive pericardial fluid production from autoimmune, infectious, and inflammatory conditions

- The pericardium is a relatively inelastic membrane
- As little as 200–300 cc of pericardial fluid can exhibit as significant mass effect on the heart if rapid accumulation occurs
- Cardiac tamponade is a physiologic state in which pericardial fluid under pressure affects cardiac function
- The right atrium is typically the lowest pressure chamber and, as a result, is most susceptible to impaired filling during cardiac tamponade
- Cardiac tamponade can be treated with percutaneous drainage and/or surgical intervention depending on the context
- Following cardiac surgery, cardiac tamponade from mediastinal bleeding should be suspected in all patients with worsening hemodynamics
- Bedside TTE is a fast and effective method for evaluating cardiac tamponade but in many circumstances TEE may be required due to technically difficult TTE imaging windows in patients who are post-op.
- Treatment of cardiac tamponade following cardiac surgery often requires emergent sternotomy and mediastinal exploration to identify the source of bleeding
- Pericarditis is inflammation of the pericardium that can happen after surgery (post-pericardiotomy syndrome) or from non-surgical conditions like autoimmune, infections, and idiopathic disorders
- Chronic and/or recurrent episodes of pericarditis can result in constrictive pericarditis
- Constrictive pericarditis is pathologic thickening and fibrosis of the pericardium that limits filling of the atria and ventricles
- Medically refractory constrictive pericarditis is treated with pericardiectomy

Diastolic Function

- Heart failure (HF) is categorized into heart failure with reduced ejection fraction (HFrEF) and heart failure with preserved ejection fraction (HFpEF)
- HFpEF results from diastolic dysfunction of the left ventricle
- Diastolic dysfunction is the result of impaired relaxation and/or increased resistance to filling
- Old age, obesity, diabetes, and coronary artery disease can contribute to HFpEF
- Diastolic heart failure can be appreciated via echocardiography as left ventricular hypertrophy (LVH, increased LV thickness), a restrictive mitral flow pattern, increased left atrial pressure, and increased right-sided pressures

Adjuncts for Echocardiography

Stress Echocardiography

- Stress echocardiography is mostly used to evaluate suspected or known ischemic heart disease. It can also be used to evaluate valvular disease during physiological or pharmacological stress
- A baseline TTE exam is performed prior to the stress test
- Stress is induced by having the patient exercise on a treadmill or recumbent bike, or by administering dobutamine
- Changes in ventricular function, wall motion, and valvular function during stress are compared to baseline looking for inducible changes
- Further studies (conventional angiography, nuclear medicine viability, cardiac MRI), interventions, or surveillance can be determined based on the results

Contrast Echocardiography

- Hand-agitated saline contrast can be injected in peripheral intravenous line to evaluate for presence of right to left intracardiac or intrapulmonary shunting.
- Ultrasound-enhancing agents have been used to improve left ventricular cavity opacification during TTE and in some instances, evaluating the presence of a left atrial appendage thrombus on TEE

References

1. Baumgartner H, Hung J, Bermejo J, Chambers JB, Evangelista A, Griffin BP, Iung B, Otto CM, Pellikka PA, Quiñones M. Echocardiographic assessment of valve stenosis: EAE/ASE recommendations for clinical practice. J Am Soc Echocardiogr. 2009;22:1–23.
2. Echocardiographic assessment of aortic valve stenosis: a focused update. https://www.asecho.org/document/12069#page=1. Accessed 26 June 2022.
3. Zoghbi WA, Adams D, Bonow RO, Enriquez-Sarano M, Foster E, Grayburn PA, Hahn RT, Han Y, Hung J, Lang RM, Little SH, Shah DJ, Shernan S, Thavendiranathan P, Thomas JD, Weissman NJ. Recommendations for noninvasive evaluation of native valvular regurgitation. J Am Soc Echocardiogr. 2017;30:303–71.
4. Writing Committee Members, Otto CM, Nishimura RA, Bonow RO, Carabello BA, Erwin JP, Gentile F, Jneid H, Krieger EV, Mack M, McLeod C, O'Gara PT, Rigolin VH, Sundt TM, Thompson A, Toly C. ACC/AHA guideline for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Joint Committee on clinical practice guidelines. J Am Coll Cardiol. 2020;2021(77):450–500.

Chapter 5
Cardiac CT and MRI

Anushri Parakh, Vinit Baliyan, and Sandeep Hedgire

Introduction

Cardiac CT and MRI are important non-invasive tools for evaluation of heart conditions. Both these techniques use ECG-based gating/triggering to generate images that provide anatomic and functional information.

CT uses ionizing radiation to generate volumetric data that can be reconstructed and viewed in multiple imaging planes. Images are displayed on a gray scale where brightness of a structure is proportional to its ability to attenuate X-ray photons (higher attenuation structures such as calcification appear bright, and lucent structures such as air or fat appear dark). Iodinated contrast (which attenuates X-rays appears dense/bright on images) is used to generate contrast between anatomic structures. Cardiac CT requires high flow rate of contrast administration to generate diagnostic quality images. CT imaging is very fast (imaging time of few seconds for heart) and provides excellent spatial resolution, offers multiplanar imaging capabilities and generation of 3D volume rendered images.

Cardiac MR generates cross-sectional images without the use of ionizing radiation. The image signal is derived from an interplay between high-strength magnetic field, radiofrequency pulses, magnetic field gradients, and protons inside the human body. Different combinations of radiofrequency pulses, magnetic field gradients, and timing of image acquisition provide different types of image contrast. These specific combinations are known as imaging sequences. Images are again displayed on a gray scale where signal intensity may represent the concentration of protons, tissue contents (water/fat/blood), tissue vascularity, or blood flow velocity/direction depending on the specific type of sequence. CMR also has ability to generate images

A. Parakh · V. Baliyan · S. Hedgire (✉)
Department of Cardiovascular Imaging, Massachusetts General Hospital, Boston, MA, USA
e-mail: APARAKH1@mgh.harvard.edu; VBALIYAN@mgh.harvard.edu; Hedgire.Sandeep@mgh.harvard.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_5

with very high temporal resolution and superior soft tissue characterization. Due to such capabilities, CMR is a very useful tool for characterization of function, flow, and tissue composition.

Cardiac CT

- Computed Tomography (CT) is performed routinely to delineate cardiac anatomy and diagnose pathologies non-invasively.
- It can be acquired with or without intravenous iodinated contrast media (IVCM), depending on the specific clinical concern.

Indications

- A non-contrast scan is also known as "calcium score scan" because it is mostly commonly performed to assess the burden of calcified coronary arterial atherosclerosis [1].
- Besides coronary arteries, calcium scoring of the aortic valve can also be performed. This is particularly indicated when the severity of aortic stenosis is inconclusive on echocardiogram (for example, low-flow low-gradient aortic stenosis) [2].
- Majority of the rest of the indications require IVCM. The most common indication is to assess the coronary arteries. Coronary CT angiogram (CTA) is typically indicated to evaluate anomalous coronary origin or course, and coronary artery disease (CAD) in patients (1) who are symptomatic and have low-intermediate probability for CAD, (2) with new heart failure or stress cardiomyopathy, (3) at an intermediate-risk patients for pre-procedural (surgery or transcatheter procedures) assessment, (4) with equivocal results on stress imaging, (5) who have undergone prior revascularization procedures to assess anatomy and patency of stents and bypass grafts, (6) with chronic total occlusion (CTO) to provide anatomic roadmap for guiding procedure. Additionally, it can be used in evaluation of coronary artery dissection and aneurysms.
- Cardiac chamber assessment including anatomic delineation in congenital anomalies and assessment of benign and malignancy masses (baseline and post-therapeutic imaging).
- Assessment of native and prosthetic valves. Native valvular evaluation includes anatomic valvular morphologic assessment, evaluation for feasibility of transcatheter procedures and valvular mass lesions. Prosthetic valvular evaluation includes assessment of post-surgical complications such as paravalvular leak, pseudoaneurysms, and pannus formation. Lesions that affect both native and prosthetic valves include vegetations, and thrombi.

- CT can also be used to assess wall motion abnormality and ventricular function. This provides assessment of left ventricular aneurysms, pseudoaneurysms, crypts, and diverticulum.
- Multi-phase cardiac or coronary CT can also be done in conjunction with imaging of the thoracic and abdominal aorta. This is discussed in sect. IV.

Patient Selection

- Prior history of anaphylactic reaction to IVCM is considered an absolute contraindication, patients with less severe allergic reactions may undergo a CT after premedication with corticosteroids and diphenhydramine.
- Unless contraindicated, coronary CT is ideally performed after vasodilatation with nitroglycerine. Some institutions also premedicate with beta-blockers to control (regularize) the heart rate for improving image quality.
- Good ECG gating, wide bore intravenous access (preferably 18G for adults; to support high injection rates), and patients' ability to perform short but steady breath-holds during image acquisition are key for getting diagnostic quality imaging. Inability to breath-hold or follow instructions can have a dramatic impact on image quality.

CT Protocol

- Non-contrast and contrast-enhanced images are obtained by syncing the image acquisition with the electrocardiogram (ECG). Syncing can be done by prospective triggering or retrospective gating.
- Prospectively triggered (step-and-shoot technique) scans acquire images during a predetermined phase of the cardiac cycle. This type of imaging can either be performed over the shortest possible time window at the limits of temporal resolution (for example, at 35% or 75% of the R-R interval) or can be acquired over a wider time window (padding) which allows the generation of data at few different time points/phases during R-R interval (for example, 30–80%). The CT tube is turned off during the rest of the cardiac cycle. In contrast, the tube current is on during the entire R-R cycle in retrospective gating (spiral/ helical technique); therefore, it comes at an expense of a higher radiation dose.
- In general, prospectively triggered scans have a lower radiation dose. To reduce radiation dose in retrospective gating and prospective scan with padding, the tube current can be modulated during certain phases of the cycle. These techniques provide short bursts of full radiation exposure that coincide with key time points in the cardiac cycle that provide best assessment of structure of interest

(mid diastole or end systole coronary imaging). Lower baseline radiation is used during rest of the acquisition window.

- Retrospective gating provides a more comprehensive dynamic assessment (for example, valvular function, volumetric measurements) but this technique works best with a steady heart rate. Alternatively, a prospective scan with wide padding can be used for functional assessment in patients with arrhythmia, which employs arrhythmia rejection techniques.
- Most indications require an arterial phase with the maximum contrast opacification of the left-heart chambers and aorta. This requires good (ideally 18 gauge) intravenous access. Delayed images (typically 1 min) can also be obtained to assess myocardium, thrombi, and masses.

Imaging Findings

Non-contrast/Calcium Score Scan

- Agatston scores are a semi-automatic quantification of the calcification burden (Fig. 5.1) and is a weighted sum of calcification densities in a defined voxel. Coronary arterial calcium score can be categorized into very low risk (score 0), mildly increased risk (score 1–99), moderately increased risk (score 100–299), and moderate-severely increased risk (score ≥300).
- An aortic valve calcium score of >1300 in females and >2000 in males is considered severe.

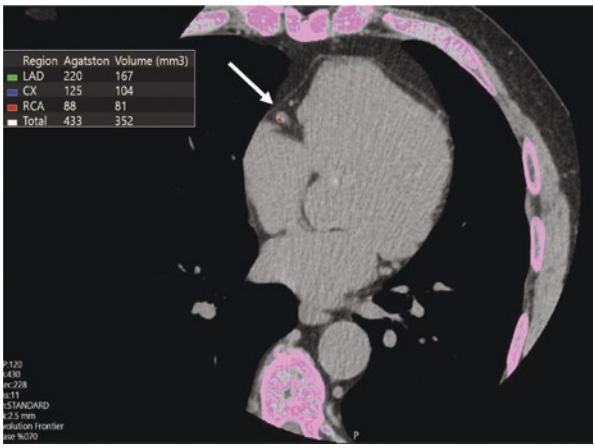


Fig. 5.1 Non-contrast cardiac CT depicting the burden of calcified coronary plaque with the Agatston scoring method. Arrow denotes calcified plaque in the right coronary artery

Contrast-Enhanced Scan

Coronary Arteries

Anomalies

- When anomalies are suspected clinically or when they are not imaged on echocardiogram/catheter angiogram, coronary CTA is excellent in imaging anomalies in origin and course. It is necessary when surgery is indicated.
- Anomalous origin: Normally, the right coronary cusp and left coronary cusp give rise to right (RCA) and left main (LM) coronary arteries, respectively. Some examples of anomalies in origin include pulmonary artery origin of the left main or right coronary arteries, anomalous origin of the RCA from the left coronary cusp, origin of left circumflex (LCx) or left anterior descending (LAD) from the right coronary cusp or RCA, separate origins of the LAD and LCx, or single coronary artery. CTA helps determine the coronary ostium, delineate the presence of intramural or inter-arterial course, provide a dynamic assessment with visualization of luminal area (in the presence of compression), and ascertain the presence of slit-like lumen.
- Anomalous course or termination: Myocardial bridging (example of anomalous course) and coronary artery fistula (which can be congenital or acquired; example of anomalous termination) can also be assessed by CTA. CTA provides accurate depiction of the length and depth of bridging. In the case of fistulae, it depicts the number and sites of fistulae which can be coronary to a cardiac chamber (coronary-cameral) or coronary to systemic or pulmonary circulation.

Coronary Artery Disease (CAD)

- CAD is the most common indication for coronary CTA. The degree of coronary artery stenosis is graded based on a standardized reporting system called CAD-RADS (Coronary Artery Disease Reporting and Data System) [3]. It includes seven categories (numerical) with some modifiers (alphabetical) (Table 5.1 and Fig. 5.2). At the time of writing this chapter, a new CAD-RADS 2.0 system is underway which will take into account the amount of plaque, lesion-specific ischemia, CT perfusion, and exemptions to CAD-RADS.
- Vulnerable plaque is characterized by the presence of at least two high risk features (spotty calcifications, positive remodeling, low-attenuation, and napkin ring sign). These may not be obstructive plaques but are called vulnerable due to a higher risk of future rupture and/or thrombosis.
- Coronary CT fractional flow reserve (CT FFR) is a novel technique which is applied after coronary CTA images have been obtained. CT FFR provides non-invasive assessment of the pressure and flow across the coronary arterial system

Table 5.1 Detailed description of the CAD-RADS system to grade coronary artery stenosis

CAD-RADS	Highest overall grade of stenosis on CTA	Interpretation	Further workup
0	0	CAD absent	None
1	<25%	Minimal narrowing (non-obstructive CAD)	None
2	25–49%	Mild narrowing (Non-obstructive CAD)	None
3	50–69%	Moderated narrowing (potentially obstructive CAD)	Consider functional imaging
4	A: Single vessel 70–99% B: Three vessel 70–99% or LM > 50%	Severe narrowing (obstructive CAD)	Consider catheter angiogram and/or functional assessment
5	100%	Total occlusion	Consider catheter angiogram and/or viability assessment
N	–	Presence of non-evaluable segments	Additional imaging may be needed
V	–	Presence of vulnerable plaque	–
S	–	Presence of stent	–
G	–	Presence of bypass graft	–

CAD coronary artery disease, *LM* left main coronary artery

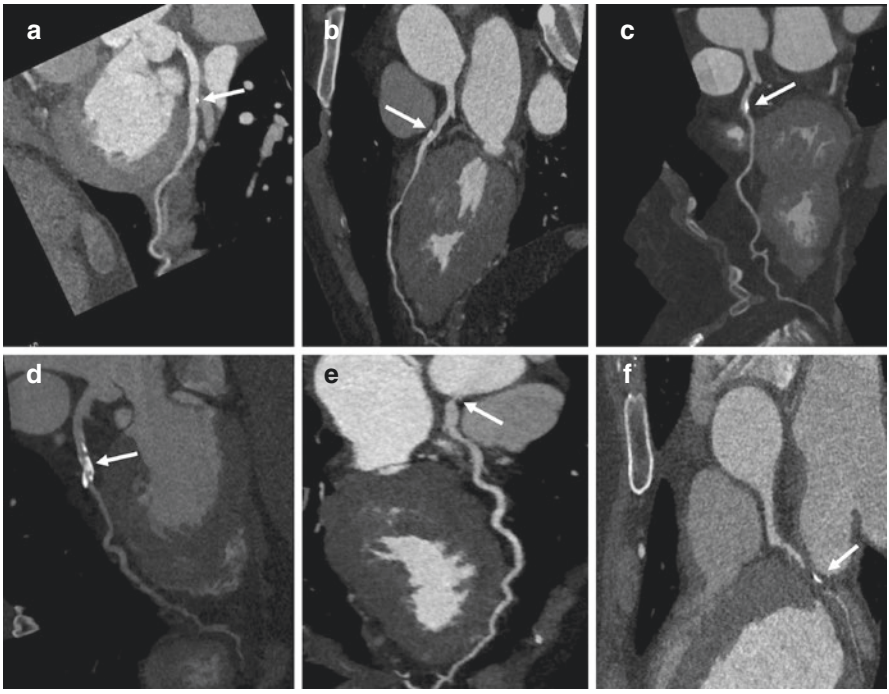


Fig. 5.2 Arterial phase contrast-enhanced CT angiogram images in curved planar reformat demonstrate coronary artery disease in varying degree of stenoses (arrows): (**a**) minimal (<25% =; CAD-RADS 1), (**b**) mild (25–49%; CAD-RADS 2), (**c**) moderate (50–69%; CAD-RADS 3), (**d**) severe (70–99%; CAD-RADS 4a), (**e**) severe left main (>50%; CAD-RADS 4b), (**f**) occlusion (100%; CAD-RADS 5)

via computational flow dynamics to assess the hemodynamic significance of a stenosis (or simply lesion-specific ischemia). At the time of writing this chapter, there is only one company that has been approved for this analysis by the United States Food and Drug Administration and thus most of the data is based on this vendor-specific method (HeartFlow, Redwood City, California, US). CT FFR > 0.80 is considered not hemodynamically significant, while CT FFR ≤ 0.75 is considered hemodynamically significant. CT FFR value between 0.76 and 0.80 is borderline. Since it is early on in its inception, CT FFR has a variable use, but a majority of its use is in patients with potentially obstructive CAD (CAD-RADS 3; 50–69% lesions) with a vascular caliber over 1.8 mm (Fig. 5.3). This can help reduce false positive rate and streamline the use of catheter angiograms. More work is still needed in understanding and ability of CT FFR in patients with serial stenoses, stents, grafts, and CTAs not performed with nitroglycerine.

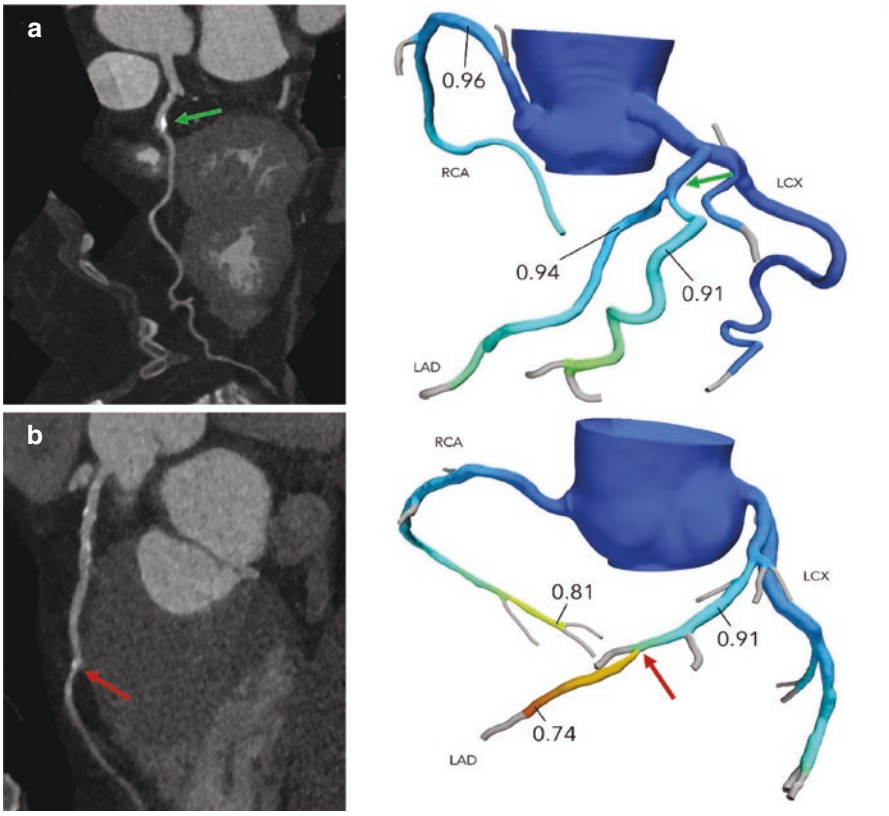


Fig. 5.3 Coronary CT fractional flow reserve (CT FFR) in two patients (a, and b) with moderate luminal narrowing. In patient (**a**), the CT FFR distal to the area of concern (green arrow) is >0.8 indicating lack of hemodynamic significance. In patient (**b**), the CT FFR distal to the area of concern (red arrow) is <0.8 indicating hemodynamic significance

Coronary Artery Dissections

- Invasive catheter angiogram for coronary artery dissections has a potential risk of propagating the dissection flap. CTA can be used to identify and follow-up dissection flaps and delineates the vascular segments involved. Imaging findings include multiple lumina with contrast opacification of a patent segment (type 1), diffuse long segment smooth stenosis (type 2; Fig. 5.4), or focal short segment stenosis (type 3).
- In the setting of both CAD and dissection, CT can also be used to assess the timeline of a myocardial infarct based on characteristic myocardial features. Acute-subacute myocardial infarct can be seen as a perfusion abnormality (on arterial and delayed images) with normal wall thickness(Fig. 5.4). Whereas in chronic myocardial infarct, wall thinning (Fig. 5.5), fatty metaplasia, and or myocardial calcifications are seen. When the entire cardiac cycle images are available, regional wall motion abnormalities can also be seen in both these settings, in addition to other complications such as ventricular aneurysm or pseudoaneurysm.

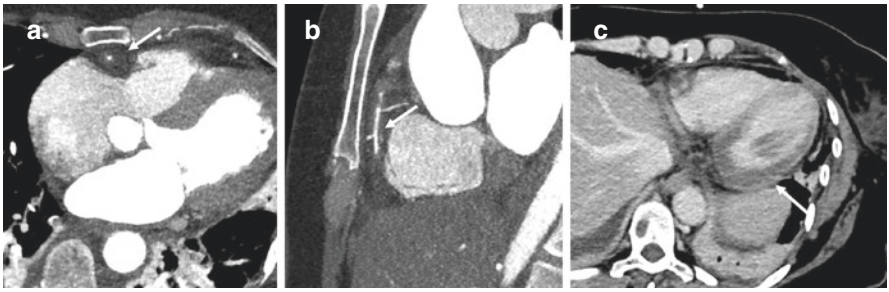


Fig. 5.4 Arterial phase contrast-enhanced CT angiogram images (**a**, **b**) demonstrate perivascular soft tissue thickening and smooth narrowing along the right coronary artery (arrows) indicating coronary artery dissection. Delayed images (**c**) show hypoenhancement of the inferior walls (arrow) consistent with acute infarct

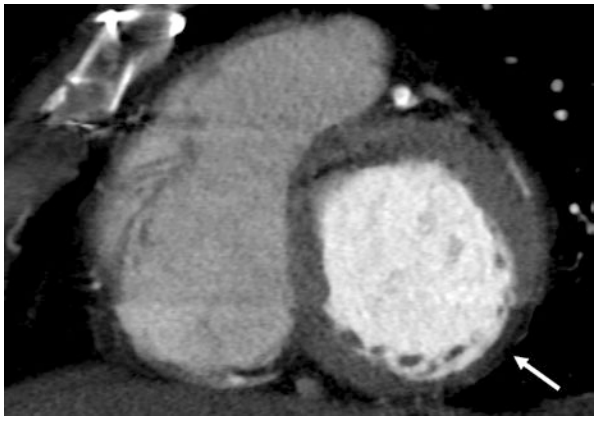


Fig. 5.5 Myocardial thinning involving the basal inferior and inferolateral walls consistent with chronic infarct

Coronary Artery Aneurysms

- Dilatation of coronary arteries up to one and a half times its normal caliber is defined as an aneurysm. Most commonly, native coronary artery aneurysms can be seen in the setting of atherosclerosis, Kawasaki disease, or IgG4-related disease and can be seen in the bypass graft as a sequela of degeneration. CTA can be used to identify and monitor (1) aneurysmal sac size, (2) thrombosis, and (3) complications such as rupture.

Valvular Disease

Pre-transcatheter Procedure Assessment

- CT, generally performed as a retrospectively gated acquisition (or prospective with padding), provides several measurements that aid in device sizing and planning the procedure (particularly indicated for valvular stenosis). It can also predict appropriate fluoroscopic angles for orienting the valves during the procedure (TAVR).
- Transcatheter aortic valve repair (TAVR): CT can help with the determination of valvular morphology (tricuspid, bicuspid, or unicuspid) (Fig. 5.6). In equivocal cases, especially in low-flow low-gradient aortic stenosis, it can estimate the degree of stenosis by providing aortic valve calcium score and planimetry. Dimensions of the aortic root, annulus, and sinotubular junction guide the sizing of the prosthetic valve. Height of sinotubular junction and length of right and left coronary valve leaflets (Fig. 5.7) also help assess the type of prosthetic valve [4]. In the case of a valve-in-valve TAVR assessment, coronary artery ostial distances to the simulated valve are also necessary.
- Transcatheter mitral valve repair (TMVR): CT visualizes the mitral valve, degree of annular calcifications, subvalvular apparatus, annular size, basal septal thickness, and length of the anterior mitral valve leaflet. It also estimates the area of the left ventricular outflow tract (LVOT) and predicts neo-LVOT after simulating prosthetic valves and the degree of narrowing to help select an appropriate size and configuration of the prosthetic valve.

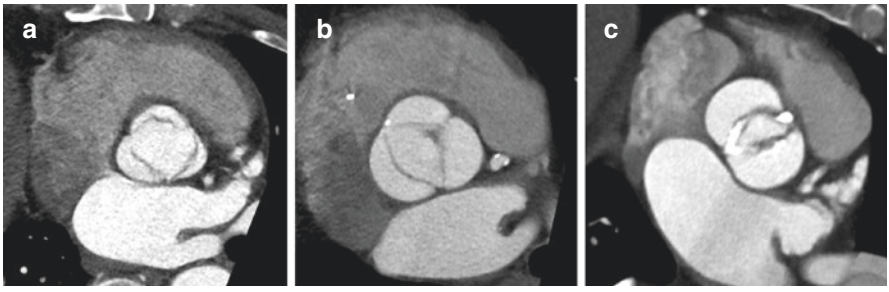


Fig. 5.6 Aortic valve morphology on cardiac CT. (**a**) Tricuspid, (**b**) bicuspid (Sievers type 1), and (**c**) bicuspid (Sievers type 0)

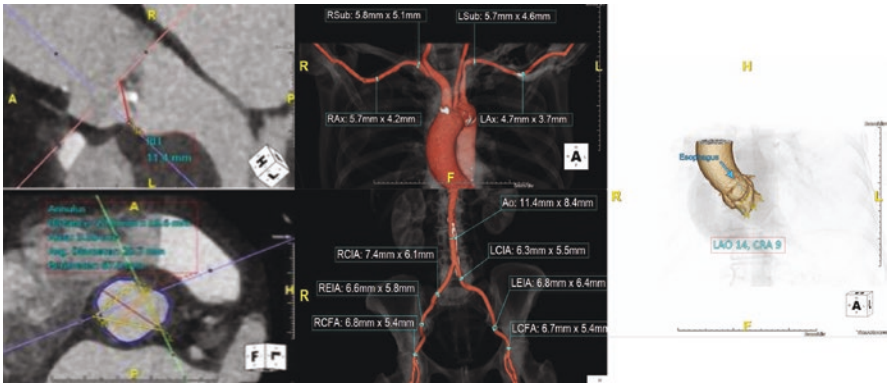


Fig. 5.7 Pre-TAVR assessment by cardiac CT demonstrating measurements of the aortic annulus, leaflet lengths, gantry angles, and minimal aortoiliac & subclavian and axillary arterial dimensions for procedural planning

- Transcatheter tricuspid (TTVR) and pulmonic valve (TPVR) repair: Besides providing spatial information of structures adjacent to the tricuspid (especially RCA) and pulmonic (especially LM and LAD) valves, CT provides systolic and diastolic measurements of the tricuspid annulus and right ventricular outflow tract for device sizing for TTVR and TPVR, respectively. Additional measurements include pulmonary artery diameter and length to bifurcation to gauge the landing zone.

Infective Endocarditis

- Cardiac CT has a complementary role to echocardiogram in the assessment of valvular endocarditis, especially in the detection of paravalvular abscesses and pseudoaneurysms.
- Echocardiography (especially TEE) is more sensitive for vegetations. Vegetations on native and prosthetic valves are low-intermediate density lesions along the valve leaflets or endocardium. The role of CT in small vegetations (less than 10 mm) is thought to be inferior to an echocardiogram, however, majority of the studies were on older CT scanners with lower temporal and spatial resolution. Modern scanners have submillimeter resolution and can detect smaller lesions as well.
- CT is also useful in the assessment of local and distal complications.
- Local complications seen on CT include leaflet perforation, valvular regurgitation, flailed leaflet components, paravalvular abscess, pseudoaneurysms, fistula, and valvular dehiscence. An abscess can be seen as an ill-defined soft tissue thickening (Fig. 5.8) and/or peripherally enhancing collection with a central low-

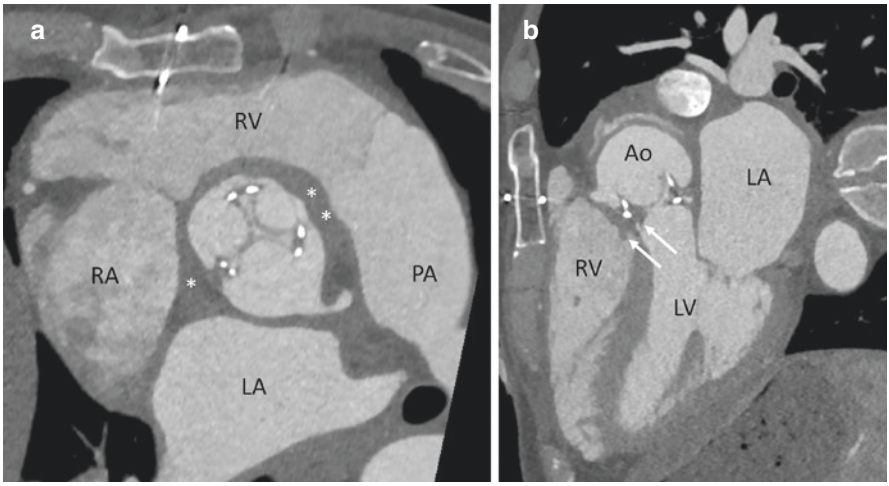


Fig. 5.8 Infective endocarditis in the setting of a bioprosthetic aortic valve on cardiac CT in short-axis (**a**) and three chamber (**b**) views. (**a**) Periaortic soft tissue thickening (asterisk) and (**b**) soft tissue density (arrow) are seen associated with the valvular ring projecting into the right ventricular outflow tract. Note: *RA* right atrium, *LA* left atrium, *RV* right ventricle, *PA* pulmonary artery, *LV* left ventricle, *Ao* aorta

density. A pseudoaneurysm is a blood (and thus contrast-filled) structure that communicates with adjacent cardiac structures (most commonly aorta and cardiac chambers). Fistula is a sequela of an abscess or pseudoaneurysm that appears as contrast-filled tract connecting adjacent cavities. Valvular dehiscence is caused by the destruction of the valvular ring around a prosthetic valve resulting in a gap between the annulus and prosthetic valve ring and/or rocking motions of varying degrees.

- Distal complications include embolization (common in large vegetations) to pulmonary artery (in right-sided vegetations) and the aorta or its branches (in left-sided vegetations) are visualized as intraluminal filling defects resulting in vascular occlusion of varying degrees. End-organ sequela such as pulmonary, renal, splenic, and bowel infarcts can also be seen on CT.

Valvular Mass

- Most common valvular masses include thrombus, and tumors (such as fibroelastoma) which are seen as filling defects along the valvular surface, and diagnosis is aided by clinical context.
- CT can help narrow differential diagnosis based on mass characteristics, determine mass mobility, and define its relationship to adjacent structures.

Cardiac Mass

- Cardiac CT performed in the setting of mass lesion helps differentiate thrombus from a tumor based on CT attenuation. It typically requires non-contrast, arterial, and delayed phases to assess enhancement characteristics.
- Most common cardiac mass is a thrombus (Fig. 5.9) followed by metastasis and then a primary tumor. Of the primary tumors, the most common mass (in adults) is myxoma (Fig. 5.10). The differential diagnosis is also narrowed based on location.
- CT provides the following details: size, number, location, attachment, morphology (presence of calcifications, necrosis, enhancement), margin assessment, and presence of invasion [5]. It can also be coupled with nuclear imaging.

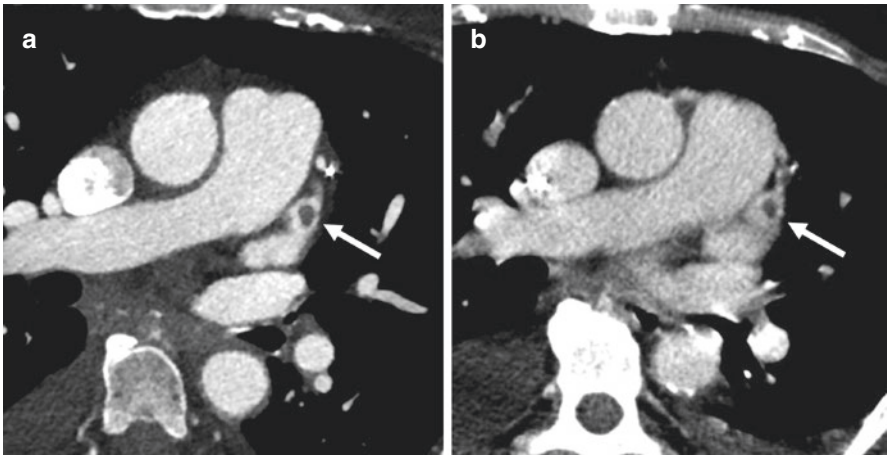


Fig. 5.9 Thrombus in the left atrial appendage seen on arterial (**a**) and delayed (**b**) phase CT images as a non-enhancing filling defect

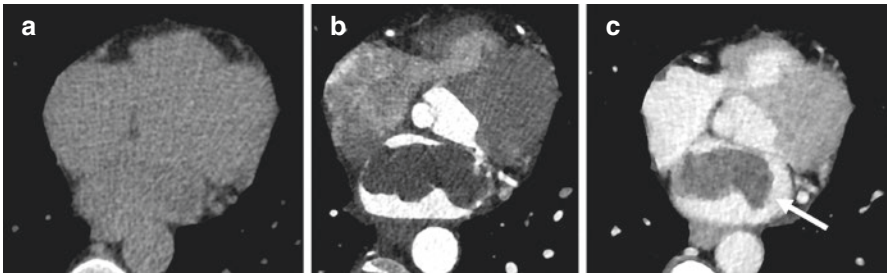


Fig. 5.10 Large left atrial myxoma seen on (**a**) non-contrast, (**b**) arterial, and (**c**) delayed phase contrast-enhanced CT images as an enhancing mass associated with the interatrial septum

Cardiac MR

Indications

Flow and Function Evaluation

- Precise quantification of left and right ventricular volume, function (ejection fraction), wall thickness.
- Valvular evaluation with quantification of degree of stenosis or regurgitation volume & fraction.
- Congenital heart disease and shunt quantification (flow through the systemic and pulmonary circulation).
- Evaluation of constrictive physiology.

Tissue Characterization

- Assessment of myocardial perfusion without and with stress imaging, and myocardial viability.
- Evaluation of myocarditis, pericarditis, infiltrative disease (e.g., hemochromatosis, sarcoidosis, and amyloidosis) and cardiomyopathy (e.g., dilated, non-ischemic, hypertrophic, and arrhythmogenic).
- Characterization of cardiac mass (neoplastic and non-neoplastic masses such as thrombus, myxoma, lipoma, lymphoma, sarcoma, and metastases).

Patient Selection

- Inability to breath-hold, lie flat and arrythmias significantly impact scan acquisition and image quality.
- Careful assessment of the compatibility of implantable devices (e.g., implantable cardiac defibrillators, pacemaker) is required to prevent device heating and migration. In select cases, devices that are not compatible or conditional may still undergo MRI if the benefits outweigh risk and patient understands the risks and provides consent.
- Indications involving tissue characterization require administration of gadolinium-based contrast agent. However, quantification of function and flow can be performed without contrast. Contraindications for the use of IVCM include severely impaired renal function (with an eGFR <30 mL/min/1.73 m^2).
- Anxious and claustrophobic patients may need sedation.

Protocol

- Typical sequences, in addition to the planning (localizer) sequences include ECG-gated cine images (movies) performed in 2-, 3-, and 4- chamber views, and short-axis views to provide volumetric and systolic functional assessment. For select indications, cine images can also be obtained in additional views. For example, right ventricular inflow-outflow plane provides better detail of the right ventricular wall to assess for focal aneurysms and dyskinesia. These images have very high inherent contrast between myocardium and blood pool without use of GBCAs [6].
- Dark blood T2- weighted images provide assessment of focal myocardial edema.
- Late gadolinium enhancement (LGE) images are obtained after contrast administration which are very sensitive for detection of regional myocardial scar. These images use a special type of imaging sequence (inversion recovery) which makes the reference (relatively normal) myocardium dark and boosts the conspicuity of scar tissue (which appears bright). It is the most important sequence for characterizing myocardial tissue by cardiac MRI that allows for narrowing differential diagnosis of cardiomyopathies. Presence of LGE indicates expansion of extracellular space which can result from myocardial inflammation, infiltration, and/or scarring.
- Myocardial perfusion imaging and post-contrast T1-weighted imaging are helpful for assessing tissue vascularity.
- Congenital heart disease and shunt quantification (flow through the systemic and pulmonary circulation) require imaging of the entire chest to assess co-existing anomalies. Shunt quantification involves phase contrast imaging through the aortic and pulmonic valves (Qp:Qs) to quantify flow through these valves and assess presence of shunt. A Qp:Qs of 1 is normal.
- Other non-contrast sequences performed based on the clinical indication include: (1) T2* images to assess & quantify myocardial iron deposition, (2) T1 mapping to quantify diffuse fibrosis, and (3) T2 mapping to assess diffuse myocardial edema. Pre- and post-contrast T1 mapping, in conjunction with hematocrit, can be used to compute extracellular volume.

Imaging Findings

(a) The most common indication for CMR is for tissue characterization in cardiomyopathy. The pattern and location of LGE are most critical for differential diagnosis of cardiomyopathy. Subendocardial LGE is typical for an ischemic pattern and depending on severity can involve the myocardium up to varying thickness (up to full thickness or transmural) (Fig. 5.11). Non-ischemic patterns

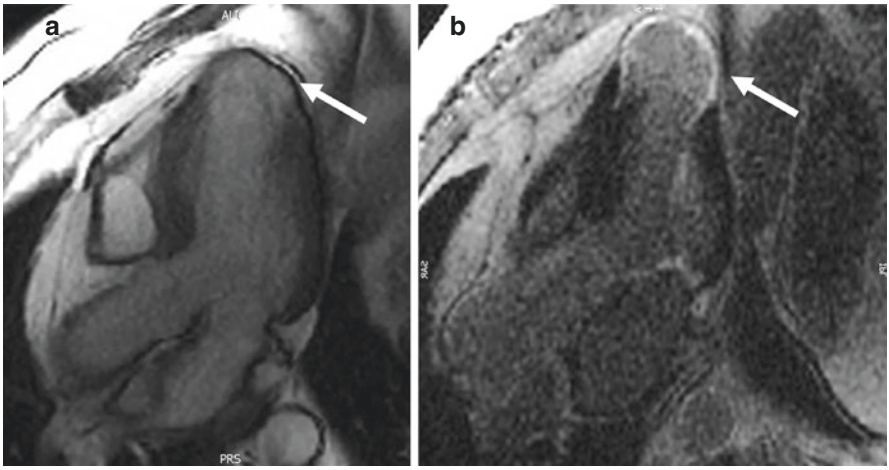


Fig. 5.11 Cardiac MR images in coronary artery disease. (**a**) Cine image shows myocardial thinning at the left atrial apex with corresponding transmural scar on the (**b**) late gadolinium enhancement images suggestive of non-viable myocardium

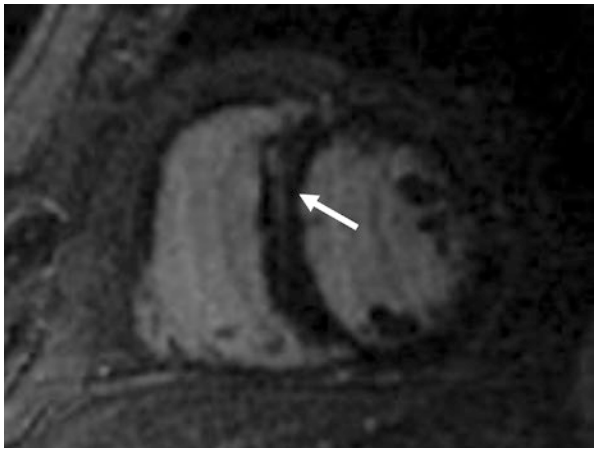


Fig. 5.12 Cardiac MR late gadolinium enhancement image showing linear mid myocardial late gadolinium enhancement suggestive of a dilated cardiomyopathy

of LGE include mid-wall LGE which is seen in idiopathic dilated cardiomyopathy (Fig. 5.12), hypertrophic cardiomyopathy (Fig. 5.13), sarcoidosis, myocarditis, Anderson-Fabry disease and Chagas disease, or subepicardial LGE which is seen in sarcoidosis (Fig. 5.14) and myocarditis. Subendocardial LGE which is diffuse and circumferential (not confirming to a vascular territory) is seen in cardiac amyloidosis (Fig. 5.15) or eosinophilic myocarditis. T2-weighted imaging can detect myocardial edema (myocarditis, pericarditis, and sarcoidosis) and thus is also useful in narrowing differential diagnosis.

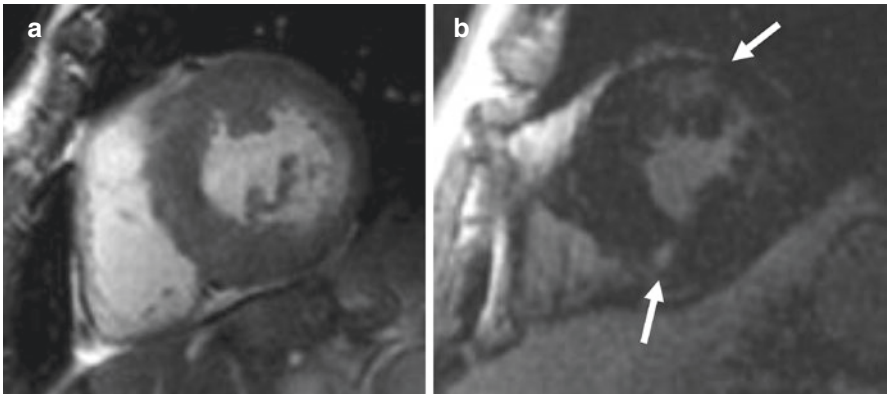


Fig. 5.13 Cardiac MR images in hypertrophic cardiomyopathy. (**a**) Cine image shows asymmetric left ventricular wall hypertrophy measuring up to 25 mm on end-diastole with patchy mic-wall late gadolinium enhancement (**b**) consistent with fibrosis

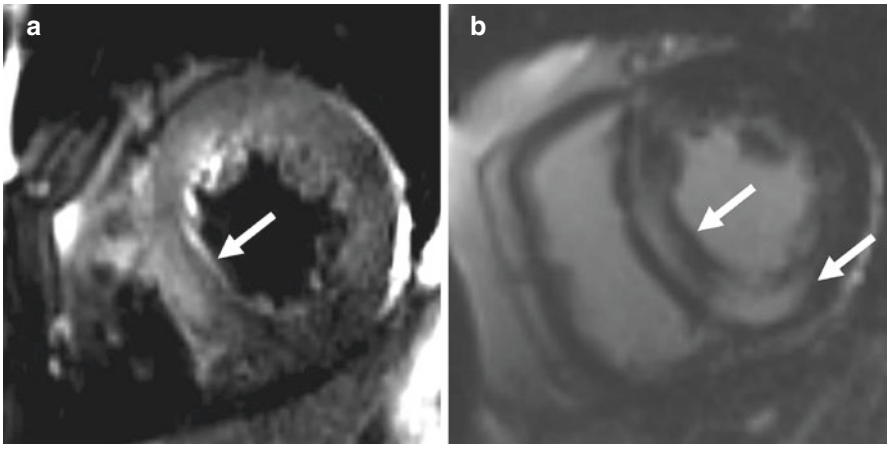


Fig. 5.14 Cardiac MR images in cardiac sarcoidosis. (**a**) T2-weighted images show hyperintense areas in the septum and inferior walls suggestive of edema with co-localizing nodular mid-wall late gadolinium enhancement (**b**)

(b) In hypertrophic cardiomyopathy, MR helps in prognostication by providing precise ventricular wall thickness measurement, presence/quantification of LGE and assessing for apical aneurysms.
(c) T1, T2, and inversion characteristics of masses help narrow the differential diagnosis of cardiac masses (Fig. 5.16). In general, thrombus appears as hypointense filling defect within a chamber that shows no enhancement. Benign cardiac tumors have well circumscribed margins, often pedunculated and are

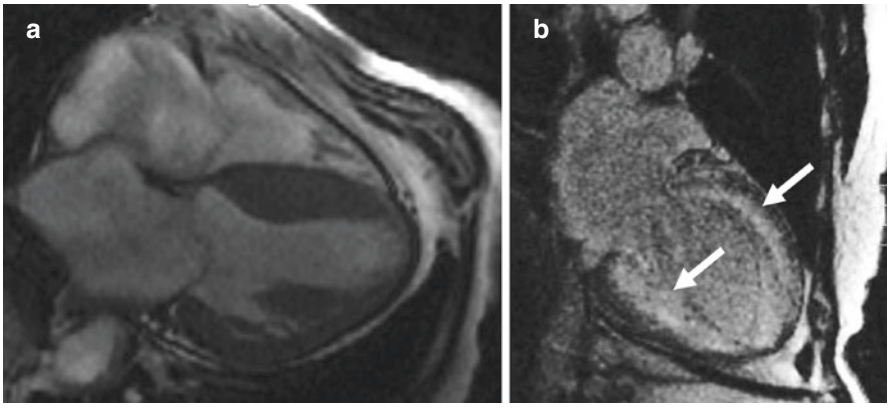


Fig. 5.15 Cardiac MR images in cardiac amyloidosis. (**a**) Cine image shows concentric left ventricular hypertrophy with diffuse subendocardial late gadolinium enhancement (**b**) not conforming to a vascular territory

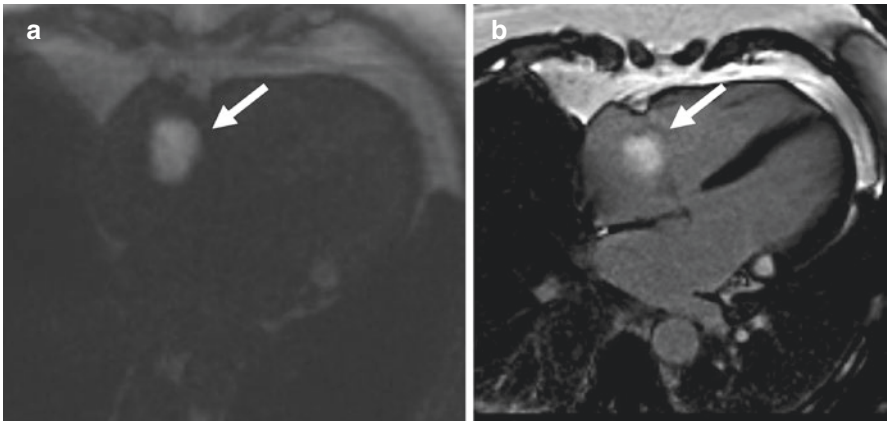


Fig. 5.16 Cardiac MR images in a patient with melanoma. (**a**) Pre-contrast T1-weighted image shows a hyperintense lesion in the right atrium with enhancement on (**b**) late gadolinium enhancement image consistent with a melanoma

usually homogenous. Lymphomas are "soft" tumors that don't invade or deform adjacent structures and show homogeneous enhancement. While primary cardiac malignancies such as angiosarcomas are heterogenous (due to necrosis) and infiltrate/deform adjacent structures.

(d) Calculation of extracellular volume helps prognosticate patients with cardiac amyloidosis.

(e) The extent of a shunt is determined by the size of the defect and pressure gradient. Smaller shunts have a Qp:Qs <1.5, whereas larger shunts have a ratio > 2.

Aorta

- Both CTA and MR can be used for aortic imaging in a majority of conditions.
- Higher spatial resolution and more accessibility make CTA the preferred imaging modality in urgent settings. In the setting of young patients, or patients in whom multiple surveillance imaging may be necessary, MR may be preferred to reduce cumulative radiation dose. MR can be contraindicated in patients with incompatible device(s).

Indications

- Aortic aneurysms, particularly in the setting of connective tissue disorders and bicuspid aortic valve.
- Suspected acute aortic syndrome (AAS) including penetrating atherosclerotic ulcer (PAU), intramural hematoma (IMH), and dissection. Suspected aortic trauma.
- Congenital anomalies including aberrant anatomy and aortic coarctation.
- Thoracic aortic assessment without or with contrast is performed for preprocedural assessment prior to re-do sternotomy, and cardiopulmonary bypass.
- Thoracic and abdominal aortic imaging is indicated as part of procedure planning prior to thoracic endovascular aortic repair (TEVAR), TAVR, left ventricular assist device (LVAD) placement, and mini-mitral valve repair.
- Surveillance imaging post aortic surgeries, to assess for complications.

Imaging Findings

Aneurysm

- CT and MR can be used for obtaining angiographic images for aortic sizing. Aortic diameters must be measured in a plane where the aorta is aligned in its true short-axis (called as double-oblique multiplanar reformat). Although there are absolute cut-off values that are as dilatation or aneurysm, these do not account of differences based on gender and body surface area/body mass index. Indexed values are typically suggested.
- 5.0–5.5 cm is the surgical threshold for ascending aortic aneurysm repair. However, this threshold is lower (4.5 cm) in patients with connective tissue disorders such as Marfan's (Fig. 5.17) and Ehlers Danlos syndrome.

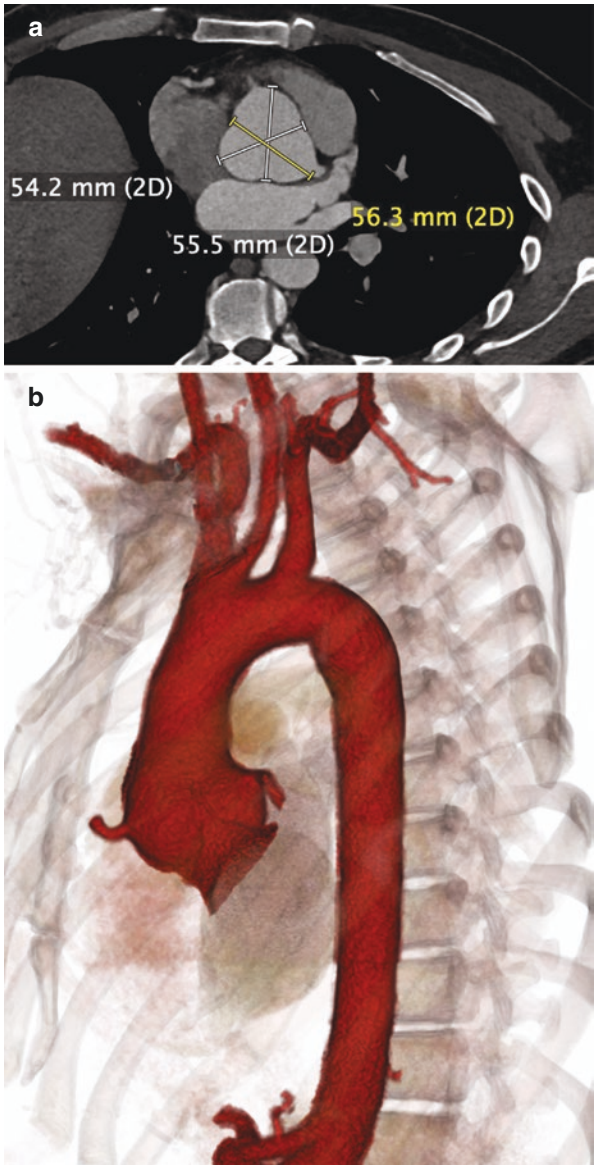


Fig. 5.17 CT angiogram in a patient with Marfan syndrome in double-oblique axial plane (**a**) shows aneurysmal aortic root at the level of the sinuses of Valsalva. (**b**) 3D volume rendered image displays the aneurysm and rest of the thoracic aorta

- In patients with known predisposition for aortic aneurysm (family history or presence of connective tissue disorder, or history of bicuspid aortic valve), serial follow-up is necessary to evaluate whether the dimensions have reached surgical threshold to prevent future risk of rupture.

Acute Aortic Syndrome

- CTA is preferred in acute aortic syndrome (AAS) due to a faster imaging technique in this subset of patients who are likely to be unstable. At least, non-contrast and arterial phases are necessary for this evaluation. Additional delayed images can help assess for aortic rupture and thrombosis.
- AAS encompasses three disease processes: intramural hematoma (IMH), aortic dissection, and penetrating atherosclerotic ulcer (PAU). Traumatic aortic injury is other acute condition which can present with intimal tear, aortic transection, rupture, and pseudoaneurysm.

  1. IMH is classically thought to be a result of the rupture of vasa vasorum leading to bleeding within the vessel wall (media). However, some believe that IMH is a result of a single intimal tear without a reentry leading and represents a thrombosed false lumen. Regardless of the pathophysiology, IMH is seen as smooth crescentic thickening of aortic wall which is hyperdense on non-contrast images (Fig. 5.18) in the acute setting and reflects clotted blood. Assessment of extent and aortic caliber is important with serial imaging to identify progression.
  2. Dissection resulting from splitting of the wall layers and separation of the aortic lumen into a true and false lumen by an intimal flap(Fig. 5.19). The true lumen is continuous with the non-dissected aorta. Imaging helps ascertain the extent of the dissection flap and determines whether it is a Stanford type A

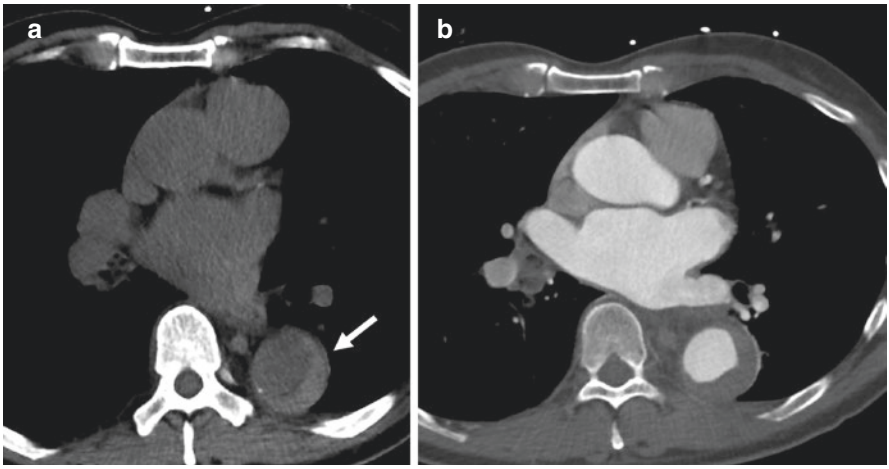


Fig. 5.18 CT thoracic angiogram in a patient with acute aortic syndrome. (**a**) Non-contrast and (**b**) arterial phase images show hyperdense crescentic thickening in the descending thoracic aorta consistent with an intramural hematoma

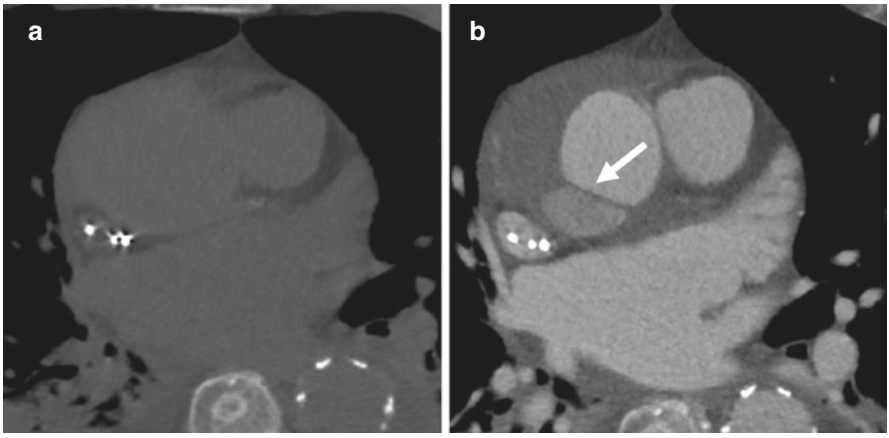


Fig. 5.19 CT thoracic angiogram in a patient with acute aortic syndrome. (**a**) Non-contrast and (**b**) arterial phase images show a flap in the ascending aorta consistent with a type A dissection

(involvement of the ascending aorta) or Stanford type B (involvement of the aorta distal to the origin of the left subclavian artery) dissection to guide surgical or medical management [7]. CTA and MR also demonstrate coronary and branch vessel involvement, and sequela of end-organ involvement such as renal, splenic, or bowel ischemia and infarction. It can assess for patency and caliber of the true and false lumina, and complications such as associated aneurysm, pericardial or pleural effusion, and rupture. Post-operative imaging after repair of dissection is used to assess stability of extent and complications such as pseudoaneurysm.

3. PAU is atherosclerotic plaque that extends across a variable depth through the internal elastic lamina to the media and can further spread to form a IMH or pseudoaneurysm. On imaging, it is seen as an area of atherosclerotic plaque that burrows into the aortic wall and extends beyond the intima.
4. Intimal tear is a focal discontinuity or partial thickness tear in the internal elastic lamina
5. Traumatic aortic injury occurs from deceleration injury. The root and aortic isthmus are common sites of injury.
6. Aortic rupture (Fig. 5.20) is seen as a contained leak/pseudoaneurysm or active extravasation of contrast. It is typically associated with adjacent hematoma in the mediastinum, pleural, or retroperitoneal spaces, which appears as a hyperdense collection on CT.
7. Aortic pseudoaneurysm, seen on CT and MR as a contrast-filled outpouching, that doesn't contain all three wall layers and can occur secondary to atherosclerosis, trauma, infection, or post-surgery.

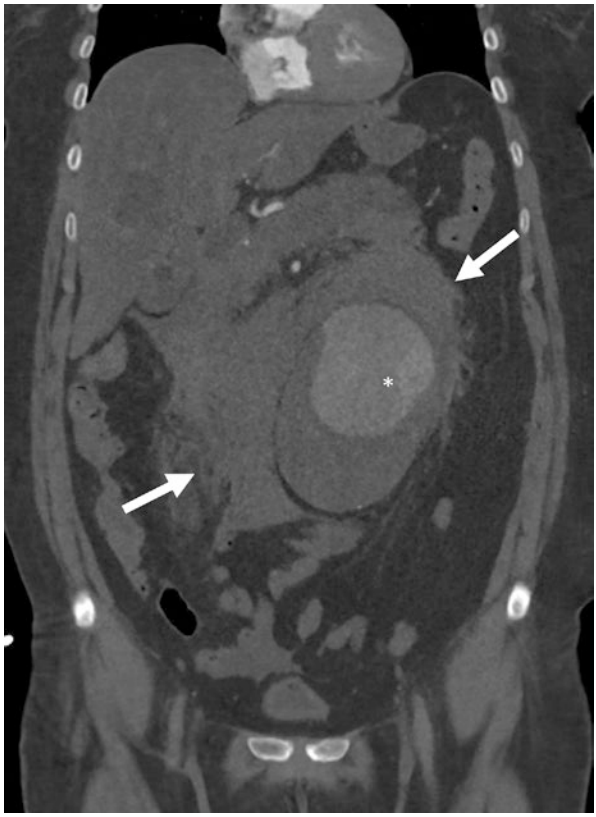


Fig. 5.20 Coronal plane CT angiogram in a patient with abdominal pain shows an abdominal aortic aneurysm (asterisk) with adjacent hyperdense hematoma (arrow) suggestive of an infrarenal abdominal aortic rupture

Coarctation

- Characterized by aortic narrowing in the juxta ductal region. Imaging can depict the caliber of the aorta proximal, at, and distal to the site of narrowing. It also demonstrates whether the coarctation is hemodynamically significant by illustrating presence of collaterals. Typical collateral pathway includes the internal mammary, intercostal, thyrocervical, and thoracoacromial arteries. Post-surgical imaging helps assessment of graft patency and presence of residual collaterals.

Pre-procedural CT

- Prior to redo-sternotomy, CT is performed to assess the proximity of the innominate vein, great vessels, and major coronary arterial branches to the posterior sternal edge to prevent complications prior to sternal entry. This provides an

estimate of possible mediastinal adhesions, and consideration of femoral or axillary cannulation to decompress these structures prior to incision.
- High risk of peri-operative stroke in patients undergoing cardiopulmonary bypass exists. One of the important risk factor for its prediction is the degree of ascending aortic calcifications which can be assessed on non-contrast CT. In the presence of significant calcifications, axillary cannulation with antegrade cerebral perfusion may be preferred.
- With IVCM, CTA of the aorta, particularly done prior to TAVR and mini-mitral valve repairs, provide information regarding the patency as well as minimal luminal diameters of the aorta, and iliofemoral system. This aids to evaluate whether large vascular sheaths used in device delivery and/access can be feasible. Vessel tortuosity, degree of calcifications, presence of abrupt kinks, and dissections are also important to note.
- CTA is done prior to TEVAR to assess anatomy, pathology, and quantitative analysis (length and diameter of the pathology, the distance between the pathology and aortic branches, diameters at targeted landing zones). Post-procedural CTA is done to assess patency of stent-graft lumen, apposition of stent-graft against the aortic wall, presence of endoleak, size of excluded aneurysmal sac size, and signs of stent-graft migration.

Double Rule Out

- Majority of the clinical indications in this chapter necessitate maximal contrast opacification of the left cardiac chambers and aorta for best depiction. By altering contrast timing and delivery, scans can also be optimized to achieve concomitant right-sided opacification and rule out both pulmonary emboli and aortic pathologies concurrently. However, this must be indicated at the outset for optimal scan acquisition and cannot be done retroactively.

Volume Rendered Reconstruction

- Done for both cardiac and aortic imaging after the scan has been acquired. Although can be done with both CT and MR, CT-derived rendered images have a better resolution.
- Volume rendered or cinematic rendering provide visually appealing images useful for pre-procedural planning and display complex anatomy. It can also be used for effective patient communication.
- Thin-slice imaging dataset can also be for 3-D printing anatomic or pathologic models for surgical planning and patient education.

Conclusion

CT and MR are very useful non-invasive imaging tools for evaluation of a wide range of cardiac and aortic diseases. Understanding the strengths and limitations of these modalities is key to maximize their diagnostic utility. CT is fast and widely available which makes it a valuable tool in emergency settings. It has a very high spatial resolution which is helpful for assessing small structures such as coronaries and getting accurate sizing of valve devices. MR is time intensive and has limited availability, so it is more suited for non-emergent settings. It is primarily used for tissue characterization, and quantification of flow and function in valvular and congenital heart disease.

References

1. Hecht HS, Cronin P, Blaha MJ, Budoff MJ, Kazerooni EA, Narula J, Yankelevitz D, Abbara S. 2016 SCCT/STR guidelines for coronary artery calcium scoring of noncontrast noncardiac chest CT scans: a report of the Society of Cardiovascular Computed Tomography and Society of Thoracic Radiology. J Cardiovasc Comput Tomogr. 2017;11(1):74–84.
2. Pawade T, Sheth T, Guzzetti E, Dweck MR, Clavel MA. Why and how to measure aortic valve calcification in patients with aortic stenosis. JACC Cardiovasc Imaging. 2019;12(9):1835–48.
3. Cury RC, Leipsic J, Abbara S, Achenbach S, Berman D, Bittencourt M, Budoff M, Chinnaiyan K, Choi AD, Ghoshhajra B, Jacobs J. CAD-RADS™ 2.0–2022 coronary artery disease-reporting and data system: an expert consensus document of the Society of Cardiovascular Computed Tomography (SCCT), the American College of Cardiology (ACC), the American College of Radiology (ACR), and the North America Society of Cardiovascular Imaging (NASCI). Cardiovasc Imaging. 2022;15(11):1974–2001.
4. Blanke P, Weir-McCall JR, Achenbach S, Delgado V, Hausleiter J, Jilaihawi H, Marwan M, Nørgaard BL, Piazza N, Schoenhagen P, Leipsic JA. Computed tomography imaging in the context of transcatheter aortic valve implantation (TAVI)/transcatheter aortic valve replacement (TAVR) an expert consensus document of the Society of Cardiovascular Computed Tomography. JACC Cardiovasc Imaging. 2019;12(1):1–24.
5. Kassop D, Donovan MS, Cheezum MK, Nguyen BT, Gambill NB, Blankstein R, Villines TC. Cardiac masses on cardiac CT: a review. Curr Cardiovasc Imaging Rep. 2014;7(8):1–3.
6. Kramer CM, Barkhausen J, Bucciarelli-Ducci C, Flamm SD, Kim RJ, Nagel E. Standardized cardiovascular magnetic resonance imaging (CMR) protocols: 2020 update. J Cardiovasc Magn Reson. 2020;22(1):1–8.
7. Lempel JK, Frazier AA, Jeudy J, Kligerman SJ, Schultz R, Ninalowo HA, Gozansky EK, Griffith B, White CS. Aortic arch dissection: a controversy of classification. Radiology. 2014;271(3):848–55.

Chapter 6
Coronary Angiography

Anna C. O'Kelly and Nilay K. Patel

Indications

Coronary angiography is the gold standard for invasive ischemic evaluation. Angiography typically occurs in the cardiac catheterization laboratory ("cath lab") and is performed through percutaneous arterial access. A catheter is advanced over a guidewire from the access site to the coronary artery through which contrast media is directly injected into the vessel. Contrast media can then be visualized with simultaneous use of a real-time two-dimensional X-ray, known as fluoroscopy. The most common indication for coronary angiography is for detection of coronary artery disease or atherosclerosis, which appears as a narrowing in the vessel. If obstructive coronary artery disease is detected, percutaneous coronary intervention (PCI) may be warranted to restore blood flow to the myocardium.

In some cases, coronary angiography should be pursued emergently, such as in patients with ST segment elevation myocardial infarction (STEMI) and in patients with non-ST segment elevation acute coronary syndromes (NSTE-ACS) with hemodynamic or electrical instability or ongoing angina (Class I recommendation) [1]. Importantly, decisions regarding whether to pursue coronary angiography should be guided by clinical need and should not be biased by patient factors such as sex or race [2]. Unfortunately, there are data showing that there can be bias in making these decisions—there are more delays in definitive revascularization for Black patients and for women than for White patients and men, respectively [3–5]. Focused efforts must be undertaken to reduce these biases and the disparities in care to which they can contribute.

A. C. O'Kelly · N. K. Patel (✉)
Cardiology Division, Department of Medicine, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA
e-mail: npatel28@mgh.harvard.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_6

As with any invasive procedure, there are notable risks with coronary angiography. The most common risks include bleeding or vascular compromise at the access site. Though rare, cardiovascular complications including stroke, myocardial infarction, and death are also possible and must be discussed with patients beforehand. The contrast used to visualize the coronary arteries can be nephrotoxic, and in rare cases may necessitate dialysis, especially in patients with advanced chronic kidney disease. Overall however, coronary angiography is a common and safe procedure performed in cardiac catheterization labs across the world.

Access

Coronary angiography is most commonly performed by accessing the radial artery or the common femoral artery. Alternate access sites, including the brachial artery or superficial femoral artery, may also be used. In general, transradial arterial access is associated with fewer vascular complications and less bleeding than transfemoral access [6]. As such, radial artery access is the preferred access site in most patients [6].

Coronary angiography can be successfully performed from the left radial artery (LRA) or right radial artery (RRA). The LRA is the preferred access site in those with a history of coronary artery bypass grafting (CABG) that utilized the left internal mammary artery (LIMA) as a bypass graft. As the LIMA originates from the left subclavian artery, graft angiography cannot be readily performed from the RRA. Radial artery occlusion and dissection are both known complications of transradial access and can preclude that artery from future use, e.g., as an arterial bypass conduit during CABG or as a site for hemodialysis A-V fistula creation [7].

Because of the fewer complications associated with radial overall femoral arterial access, the indications for femoral access are dwindling. Femoral access should be pursued if there are barriers to radial artery use, including the desire to preserve the radial artery for future use or anatomical constraints such as small caliber or a history of severe radial spasm.

Standard Projections and Catheterization Laboratory Setup

The equipment used in the cardiac catheterization laboratory is standardized (Fig. 6.1). There is a "C-arm" beside the table, with the X-ray source located beneath the table and the imaging intensifier above the table. The patient is positioned supine on the table. The C-arm is then manipulated by the cardiologist performing the procedure to obtain the projections, or specific views of the heart, that they desire.

Fig. 6.1 Standard setup for the cardiac catheterization laboratory

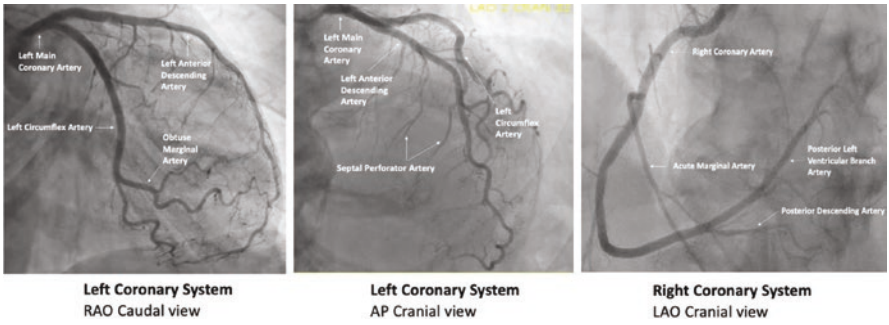


Fig. 6.2 Common angiographic projections of the left and right coronary systems

The left and the right coronary arteries are engaged sequentially. Standardized angiographic views allow for optimal visualization of different segments of the coronary arteries. Angulation of the C-arm can be accomplished in two planes. Cranial-caudal angulation rotates the C-arm toward the head or foot of the patient, respectively. Left and right anterior oblique angulation rotates the C-arm toward the left and right of the patient, respectively. Common angiographic projections can be seen in Fig. 6.2.

Physiologic Assessment

Although interventional cardiologists visually estimate the severity of coronary stenoses, angiographic estimates can sometimes be unreliable, especially when a lesion is borderline (40–69% stenosis). A desire to better define borderline lesion severity has prompted a growing field of invasive hemodynamic indices to better define if a visualized stenosis results in flow limitation to the myocardium it supplies. Fractional flow reserve (FFR), instantaneous wave-free ratio (iFR), and diastolic hyperemia-free ratio (DFR) are three of the numerous physiologic indices to help better quantify the hemodynamic significance of coronary stenoses and guide decisions about PCI.

- Fractional Flow Reserve (FFR): Gold standard physiologic index for assessing coronary severity. Measured by placing a pressure-sensing guidewire distal to the stenotic lesion of interest and measuring pressure across the lesion during a hyperemic state (achieved by adenosine administration). A *low* FFR value suggests poor coronary blood flow and thus an obstructive lesion. An FFR threshold value <0.75–0.8 is consistent with a flow-limiting obstructive lesion.
- Instantaneous wave-free ratio (iFR): Using the same pressure-sensing wire as in FFR, iFR assesses pressure differences across the lesion in question during a non-hyperemic state. The benefit of iFR is that adenosine does not need to be administered, which can be a cause for patient discomfort. Additionally, studies suggest no difference in clinical outcomes if the decision to pursue PCI is performed on the basis of iFR or FFR [8]. An iFR threshold value of ≤0.89 is consistent with a flow-limiting obstructive lesion.
- Diastolic hyperemia-free radio (DFR): A newer technology that provides a non-hyperemic index based on the average ratio of the mean distal coronary pressure (Pd) and the mean aortic pressure (Pa) (Pd/Pa) during a specific portion of diastole. A culprit lesion is considered obstructive if the DFR is ≤0.89.

Additional Imaging Modalities

Intravascular ultrasound (IVUS) is emerging as an important imaging adjunct to traditional coronary angiography. An ultrasound transducer is passed over a guidewire directly into the coronary artery to allow for high-fidelity visualization of the vessel. Whereas coronary angiography can provide a silhouette of the coronary lumen, IVUS can provide additional insight into characteristics of the vascular intima, media, and adventitia, as well as lesion size and composition. Furthermore, IVUS has an important role in determining whether an indeterminate coronary lesion is obstructive when visual angiographic estimates are borderline. Numerous studies have shown that IVUS is a more accurate modality for determining plaque size and thus appropriateness for PCI [9, 10]. Moreover, IVUS can be used to guide PCI by determining the caliber and length of stents utilized. Studies suggest

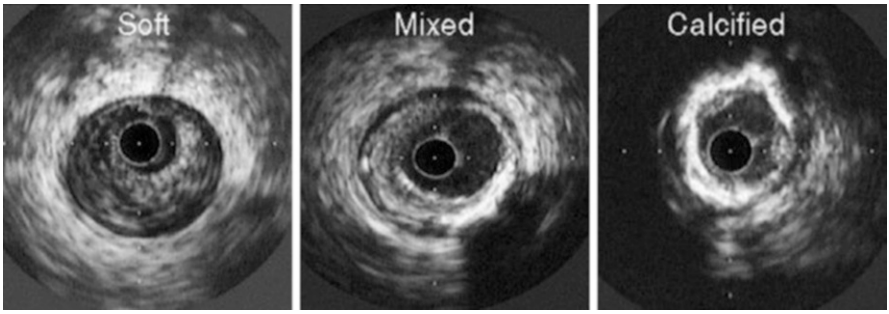


Fig. 6.3 Coronary plaque appearance by IVUS. (Adapted from "Intravascular ultrasound: novel pathophysiological insights and current clinical applications," by S.E. Nissen and P. Yock, 2001, *Circulation, 103* (4) [12]

improved clinical outcomes when IVUS is used routinely to guide complex PCI [11]. As seen in Fig. 6.3, the various components of a coronary plaque appear differently on IVUS. Lipid-rich plaques appear hypoechoic, whereas calcium-rich plaques are relatively hyperechoic.

Basics of Percutaneous Coronary Intervention

Percutaneous coronary intervention (PCI) is an invasive, non-surgical procedure that involves the use of wires and catheters to place a coronary stent ("stent") at a location of coronary obstruction, restoring blood flow to the downstream myocardium. Original bare metal stents (BMS) were constructed as metal mesh tubes. While effective at restoring blood flow, in-stent restenosis was a common cause of failure of these stents in long-term follow-up. Contemporary stents therefore are drug coated. Drug-eluting stents (DES) have three components: (1) a metallic stent platform akin to bare metal stents, (2) a polymer carrier vehicle, and (3) an antiproliferative drug to reduce the risk of in-stent restenosis.

Deciding Between Percutaneous Coronary Intervention Versus Surgical Revascularization

In many instances, it is clear when to pursue PCI over surgical revascularization. In patients who present with STEMI with <12 h symptoms, and in those with stable single-vessel coronary disease with stenoses amenable to percutaneous intervention but without significant left main lesions, PCI is a Class 1 indication [1]. In patients who present with STEMI and mechanical complications (e.g.: ventricular septal defect), in patients with stable coronary disease with multi-vessel stenoses with

LVEF <35%, and in patients with stable coronary disease with obstructive left main stenosis, surgical revascularization is a class 1 indication [1].

When the decision between percutaneous and surgical revascularization is not clear, however, a Heart Team meeting—a multi-disciplinary meeting between interventional cardiologists, cardiac surgeons, and often non-invasive cardiologists—is recommended [1]. The Heart Team discussion will take numerous patient factors into consideration, including coronary anatomy (vessel tortuosity, degree of calcification) and lesion characteristics (long lesions, lesions located at bifurcations), LV systolic function, concomitant valve disease, patient comorbidities, and social support and likelihood of medication adherence, as well as procedural considerations.

PCI Versus Percutaneous Transluminal Coronary Angioplasty (PTCA)

Percutaneous transluminal coronary angioplasty (sometimes referred to as "plain old balloon angioplasty" or "POBA") is another option for intervening percutaneously to address coronary lesions. In lieu of conventional stent placement, a balloon catheter is inflated at the site of a coronary stenosis. This approach is an inherently less durable solution than PCI, and so there are only a very limited number of circumstances in which PTCA is preferred over stent placement. There are a growing number of alternatives to conventional PTCA, including drug-eluting balloons (DEB), which may prove to be more effective [13]. The current availability of DEB is limited mostly to clinical trials and is not yet in common practice.

Stent Choice and DAPT Duration

The majority of stents used now are drug-eluting stents (DES), which have largely replaced the use of bare mental stents (BMS). Historically, the primary advantage of BMS was the shorter associated period in which dual-antiplatelet therapy was warranted, typically with aspirin and a P2Y12 inhibitor such as clopidogrel or ticagrelor. As contemporary DES have evolved, studies suggest that shortened courses of DAPT are feasible. Therefore, the indications for BMS are dwindling. BMS can be considered in patients who are unlikely to tolerate DAPT for more than one month, or in those who have non-cardiac surgery within 4–6 weeks after their PCI. Even in these patients, however, the utility of BMS may become obsolete as the US Food and Drug Administration (FDA) has now approved <30 days DAPT in specific types of DES [14].

Though recommended DAPT duration may evolve in the coming years, current recommendations depend on both the indication for DAPT and patient-specific factors. In general, for patients with stable coronary disease who undergo DES

placement, DAPT should be continued for 6 months (Class 1 recommendation) [15]. For those without high bleeding risk, DAPT can be considered for 12 months [15]. These patients should remain on daily aspirin therapy indefinitely. For patients who present with an acute coronary syndrome and receive a DES, they should remain on DAPT for 12 months though longer can be considered at the discretion of their provider [15]. Thereafter, they should remain on lifelong daily aspirin therapy.

Right Heart Catheterization

Indication

Right heart catheterization (RHC) is a procedure to facilitate invasive hemodynamic monitoring. It is performed using a Swan-Ganz catheter (Fig. 6.4).

Central venous access is obtained (most commonly via the internal jugular vein or femoral vein as summarized below), and the catheter then follows the body's natural venous drainage into the heart. There is a balloon at the tip that can be inflated to help with advancement of the catheter. It is then slowly sequentially advanced through the right atrium, right ventricle, pulmonary artery, and finally

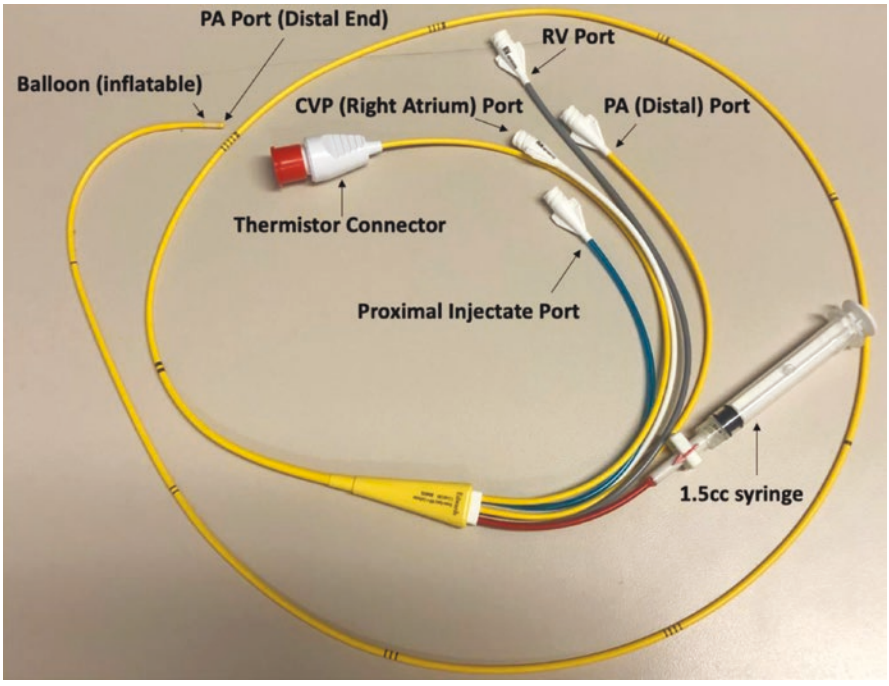


Fig. 6.4 Swan-Ganz Catheter

ends in the pulmonary capillary bed. By gaining central venous access, a RHC can provide direct assessment of the heart's filling pressures and cardiac output. This catheter can be left in situ to provide real-time feedback to help guide titration of vasoactive medications and to help guide volume management. RHC is also the gold standard for diagnosing pulmonary hypertension.

Access

When performing a RHC, right internal jugular (RIJ) access is often preferred unless there are contraindications (including neck trauma, potential future need for hemodialysis catheter placement, etc.). Alternatively, left internal jugular or femoral vein access can be used. It is a generally safe procedure, with a significant adverse event rate of approximately 1% [16]. It is frequently performed even if patients are on therapeutic anticoagulation, though this can vary by center. The most common complications are local access site hematomas, arrhythmia, pneumothorax, and vagal reaction [16, 17].

Data

Normal values for RHC parameters are summarized in Table 6.1, recognizing there is a range of what is considered "normal." It can be helpful to remember these values by "the rule of fives" as these values are all multiples of five.

As illustrated in Fig. 6.5, the waveforms seen from the Swan-Ganz catheter are different in each chamber of the heart. These unique waveforms provide orientation to the operator regarding which chamber the catheter is in and can facilitate bedside placement of a Swan-Ganz catheter without the use of fluoroscopy. The waveforms in each chamber differ both in their morphology and their absolute pressure value. Understanding both of these elements can help troubleshoot if the catheter is no longer functioning properly.

Table 6.1 Normal filling pressures during right heart catheterization

Chamber	Normal filling pressure
Right atrium (RA)	5 mmHg
Right ventricle (RV)	20/5 mmHg (systolic/diastolic)
Pulmonary artery (PA)	20/10/15 mmHg (systolic/diastolic/mean)
Pulmonary capillary wedge pressure (PCWP)	10 mmHg

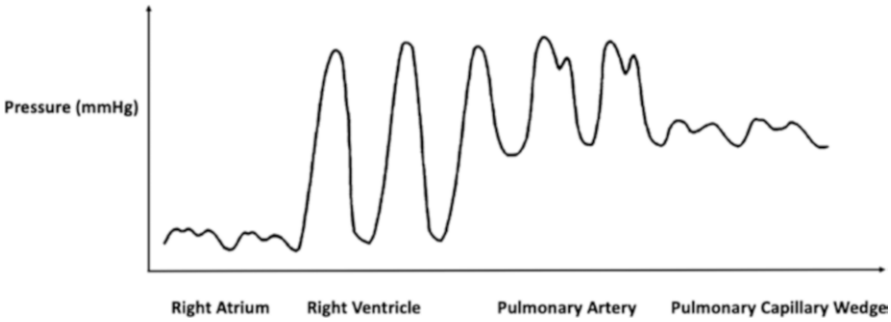


Fig. 6.5 Distinct waveforms by position during a right heart catheterization. (Adapted from Kaplan's Essentials of Cardiac Anesthesia (p. 182), by J. Kaplan, B. Cronin, T. Maus. Copyright 2018 Elsevier Inc. [18] Permission not yet granted)

References

1. Lawton JS, Tamis-Holland JE, Bangalore S, Bates ER, Beckie TM, Bischoff JM, et al. 2021 ACC/AHA/SCAI guideline for coronary artery revascularization: executive summary: a report of the American College of Cardiology/American Heart Association Joint Committee on clinical practice guidelines. Circulation. 2022;145(3):e4–e17.
2. Hameed AB, Lawton ES, McCain CL, Morton CH, Mitchell C, Main EK, et al. Pregnancy-related cardiovascular deaths in California: beyond peripartum cardiomyopathy. Am J Obstet Gynecol. 2015;213(3):379.e1–379.e10.
3. Hinohara TT, Al-Khalidi HR, Fordyce CB, Gu X, Sherwood MW, Roettig ML, et al. Impact of regional systems of care on disparities in care among female and black patients presenting with ST-segment-elevation myocardial infarction. J Am Heart Assoc. 2017;6(10):e007122.
4. Zeitouni M, Al-Khalidi HR, Roettig ML, Bolles MM, Doerfler SM, Fordyce CB, et al. Catheterization laboratory activation time in patients transferred with ST-segment-elevation myocardial infarction: insights from the mission: lifeline STEMI Accelerator-2 project. Circ Cardiovasc Qual Outcomes. 2020;13(7):e006204.
5. Lawesson SS, Alfredsson J, Fredrikson M, Swahn E. Time trends in STEMI—improved treatment and outcome but still a gender gap: a prospective observational cohort study from the SWEDEHEART register. BMJ Open. 2012;2(2):e000726.
6. Mason PJ, Shah B, Tamis-Holland JE, Bittl JA, Cohen MG, Safirstein J, et al. An update on radial artery access and best practices for transradial coronary angiography and intervention in acute coronary syndrome: a scientific statement from the American Heart Association. Circ Cardiovasc Interv. 2018;11(9):e000035.
7. Rashid M, Kwok CS, Pancholy S, Chugh S, Kedev SA, Bernat I, et al. Radial artery occlusion after transradial interventions: a systematic review and meta-analysis. J Am Heart Assoc. 2016;5(1):e002686.
8. Gotberg M, Christiansen EH, Gudmundsdottir IJ, Sandhall L, Danielewicz M, Jakobsen L, et al. Instantaneous wave-free ratio versus fractional flow reserve to guide PCI. N Engl J Med. 2017;376(19):1813–23.
9. Mintz GS, Popma JJ, Pichard AD, Kent KM, Satler LF, Chuang YC, et al. Limitations of angiography in the assessment of plaque distribution in coronary artery disease: a systematic study of target lesion eccentricity in 1446 lesions. Circulation. 1996;93(5):924–31.
10. Topol EJ, Nissen SE. Our preoccupation with coronary luminology. The dissociation between clinical and angiographic findings in ischemic heart disease. Circulation. 1995;92(8):2333–42.

11. Gao XF, Ge Z, Kong XQ, Kan J, Han L, Lu S, et al. 3-year outcomes of the ULTIMATE trial comparing intravascular ultrasound versus angiography-guided drug-eluting stent implantation. JACC Cardiovasc Interv. 2021;14(3):247–57.
12. Nissen SE, Yock P. Intravascular ultrasound: novel pathophysiological insights and current clinical applications. Circulation. 2001;103(4):604–16.
13. Lee JM, Park J, Kang J, Jeon KH, Jung JH, Lee SE, et al. Comparison among drug-eluting balloon, drug-eluting stent, and plain balloon angioplasty for the treatment of in-stent restenosis: a network meta-analysis of 11 randomized, controlled trials. JACC Cardiovasc Interv. 2015;8(3):382–94.
14. Abbott. Abbott's XIENCE stent receives FDA approval for shortest blood thinner course for high bleeding risk patients. 2021. https://abbott.mediaroom.com/2021-06-30-Abbotts-XIENCE-Stent-Receives-FDA-Approval-for-Shortest-Blood-Thinner-Course-for-High-Bleeding-Risk-Patients.
15. Levine GN, Bates ER, Bittl JA, Brindis RG, Fihn SD, Fleisher LA, et al. 2016 ACC/AHA guideline focused update on duration of dual antiplatelet therapy in patients with coronary artery disease: a report of the American College of Cardiology/American Heart Association task force on clinical practice guidelines. J Am Coll Cardiol. 2016;68(10):1082–115.
16. Hoeper MM, Lee SH, Voswinckel R, Palazzini M, Jais X, Marinelli A, et al. Complications of right heart catheterization procedures in patients with pulmonary hypertension in experienced centers. J Am Coll Cardiol. 2006;48(12):2546–52.
17. Callan P, Clark AL. Right heart catheterisation: indications and interpretation. Heart. 2016;102(2):147–57.
18. Reich D, Mittnacht AJ, London MJ, Kaplan AJ. Monitoring of the heart and vascular system. In: Kaplan JA, Cronin B, Maus T, editor. Kaplan's essentials of cardiac anesthesia for cardiac surgery. 2nd ed. Elsevier; 2017.

Chapter 7
Cardiac Anesthesia

David Convissar and Adam A. Dalia

Hemodynamic and Physiologic Monitoring

Required Standard American Society of Anesthesiologists (ASA) Monitoring

- *Pulse Oximeter*: Most commonly placed on the finger, used to monitor a patient's oxygen saturation and heart rate [1]
- *Electrocardiogram*: Often a 5 lead ECG, that is used to monitor the patient's cardiac rhythm throughout the operation [1]
- *Blood Pressure Monitor*: In cardiac surgery, continuous invasive arterial monitoring is recommended with newer methods of noninvasive continuous monitoring becoming available. At minimum, a noninvasive blood pressure cuff is recommended to evaluate the patient's blood pressure intraoperatively [1]
- *Temperature Probe*: Placed in the oropharynx, nasopharynx, bladder, and/or pulmonary artery, this monitor allows the anesthesiologist to trend a patient's temperature throughout the procedure [1]
- Carbon Dioxide (CO_2) Monitor: In line with the ventilator circuit, the CO_2 monitor allows for real-time monitoring of patient's CO_2 level [1]

D. Convissar · A. A. Dalia (✉)
Division of Critical Care and Cardiac Anesthesia, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA
e-mail: dconvissar@mgh.harvard.edu; aadalia@mgh.harvard.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_7

Advanced Hemodynamic Monitoring

- *Arterial Line*: Oftentimes placed preoperatively in the radial artery (sometimes in the brachial and others in the femoral) used for real-time continuous blood pressure monitoring [1]
- *Processed EEG:* This device placed on the forehead aids in evaluating the depth of anesthesia of the patient [1]
- *Swan-Ganz Catheter (PA Catheter):* Also known as a pulmonary artery catheter (discussed below), this catheter is inserted via an introducer from the internal jugular or subclavian veins, traversing the right atrium, right ventricle and terminating in the pulmonary artery in order to measure pressures throughout the various chambers of the heart [1]
- *Cerebral Oximeter*: Most often used during surgeries requiring circulatory arrest, cerebral oximeters measure the oxygen saturation of the frontal lobe of the brain and help to guide length of circulatory arrest permitted during surgery [1]
- *Transesophageal Echocardiography (TEE)*: Placed in the esophagus after induction of general anesthesia, the transesophageal echo allows the anesthesiologist to perform a number of critical cardiac evaluations including but not limited to ventricular function, valvular function, cardiac anatomy, aortic integrity, device positioning, and more [1]

Pharmacology of Induction and Maintenance of General Anesthesia

- The physiologic goal of induction of general anesthesia is to maintain hemodynamic stability despite the patient's pathological cardiac disease. This is one of the most critical moments of anesthesia and one of the most common times hemodynamic instability occurs [2].
- These goals of hemodynamic maintenance are usually specific to the specific pathology of that patient (see section "Lesion Specific Anesthetic Considerations")
- This is accomplished through the use of a variety of pharmacologic interventions including but not limited to:
  - Benzodiazepines such as midazolam are used to help with patient anxiety and antegrade amnesia in the preoperative period, which can be crucial in anxious patients with certain cardiac pathology such as coronary artery disease or aortic dissections.
  - Narcotics such as fentanyl can be used to help blunt the sympathetic response to laryngoscopy, endotracheal intubation, echo probe placement, and central line placement, keeping blood pressure and heart rate low, as well as decreasing the amount of hypnotic required for patients go to sleep.
  - Hypnotics such as propofol and etomidate are commonly used to induce anesthesia prior to intubation, with etomidate often being reserved for those with

severely compromised cardiac function due to the myocardial depressant effects of propofol.
- Paralytics such as rocuronium are used both on induction to maintain paralysis for intubation as well as infusions intraoperatively to maintain lack of movement, especially of the diaphragm, during the operation.
- Vasoactive agents play a critical role in cardiac anesthesia in maintaining adequate coronary and peripheral perfusion. These come in the forms of vasopressors such as phenylephrine, norepinephrine and vasopressin, inopressors such as epinephrine, inodilators such as milrinone and dobutamine, and vasodilators such as nitroprusside and nitroglycerine [2].

Lesion Specific Anesthetic Considerations

- Depending on the cardiac pathology present, different physiologic goals are preferred in order to maintain hemodynamic stability
  - Incompetent lesions such as aortic regurgitation and mitral regurgitation are characterized by the backflow of blood across an incompetent valve into the previous chamber.

    This results in a volume overload state resulting in dilation of the chamber into which blood is flowing back into.
    Anesthetic strategies for incompetent lesions include maintaining a normal to increased heart rate, as bradycardia results in increased regurgitant time. Decreasing afterload helps to drive blood flow forward, rather than retrograde across the regurgitant valve.
  - Stenotic lesions such as aortic stenosis and mitral stenosis are characterized by tightening of the valve due to factors such as leaflet sclerosis, bicuspid leaflets, restriction of leaflet motion, and more.

    This results in a pressure overload state, making the chamber pre-lesion to work harder to push blood across the valve, increasing the muscle mass of the chamber, decreasing the chamber's volume.
    Anesthetic strategies for stenotic lesions include increasing afterload to help increase coronary perfusion during diastole, keeping the heart rate slow to decrease myocardial oxygen demand of a thickened myocardium and maintaining preload to ensure enough volume to eject.
  - Coronary lesions are characterized by blockages along the coronary arterial tree from either the left or right coronary artery, resulting in myocardial ischemia.

    The most important thing in patients with severe coronary disease is to maintain a low myocardial oxygen demand. We do this by ensuring a low heart rate, adequate preload to decrease myocardial wall tension, and decreased afterload to decrease inotropy of the heart.

- Aortic lesions such as dissections and aneurysms are characterized by ballooning or tears within the aorta.

  Management of these lesions are characterized by impulse control, decreasing both blood pressure and heart rate so as to decrease shearing forces across the lesion and reducing further dilatation or dissection.

Use of Vasoactive Medications

- The use of vasoactive medications is critical in the management of patients undergoing cardiothoracic surgery each of which have unique receptors and properties utilized to optimize a patient's hemodynamic status in the perioperative setting [3].
  - *Phenylephrine*: A pure alpha 1 agonist, this drug acts primarily on the vasculature of arteries promoting vasoconstriction, increases in blood pressure and reflex bradycardia. Because of these properties, phenylephrine is the pressor of choice in patients with stenotic valvular lesions.
  - *Norepinephrine*: A mixed alpha 1, beta 1 agonist, norepinephrine is a very commonly used centrally delivered drug in cardiothoracic surgery. These properties promote increases in blood pressure via constriction of peripheral arteries as well as a minor amount of beta 1 agonism on the heart, increasing both rate and contractility. This makes it a good first line drug for coming off bypass.
  - *Vasopressin*: A D1 and D2 receptor agonist and analogue of antidiuretic hormone (ADH), vasopressin is a potent vasoconstrictor that increases blood pressure, relatively sparing the pulmonary artery. This is a good drug for helping with blood pressure on bypass when phenylephrine is not sufficient due to its lack of cardiac stimulation.
  - *Epinephrine*: An endogenous catecholamine that acts on alpha 1 and 2 and beta 1 and 2 receptors, acting as a potent inotropic agent while increasing blood pressure. This drug is especially helpful in patients with decreased ejection fractions coming off bypass, those with long bypass runs and more.
  - *Nitroglycerine*: Converted to nitric oxide in vivo, nitroglycerine promotes vasodilation through smooth muscle relaxation. Nitroglycerine preferentially vasodilates the venous system over the arterial system and is used to improve coronary perfusion as well as decrease blood pressure during instances of hypertension.
  - *Milrinone*: Commonly referred to as an inodilator, milrinone is a powerful inotropic agent during systolic failure. As a phosphodiesterase inhibitor, this may also result in a significant drop in systemic as well as pulmonary arterial pressures.
  - *Dobutamine*: As a beta 1 agonist, dobutamine works directly on the heart to improve inotropy and chronotropy. As a result, there is a sympathetic withdrawal of vascular tones that can result in vasodilation and decrease in blood pressure.

Pulmonary Artery Catheter

- The pulmonary artery catheter, also known as the Swan-Ganz catheter, is a thin catheter that is floated with the assistance of a balloon from a central vein (most commonly the right internal jugular) through right atrium, right ventricle and seated in the pulmonary artery [4].
- The catheter has multiple ports along its length, each of which allow from transducing pressures in various chambers as well as the administration of medications.
- The catheter is floated into position by watching the changing waveform and pressures as it is advanced through the heart [4].
  - *Central Venous Pressure*: Measured at the most proximal port of the catheter, this is the pressure in the right atrium and vena cava, normally between 2 and 10 mmHg
  - *Right Ventricular Pressure*: This is a transient pressure observed while floating the swan and is characterized by a systolic pressure between 20 and 25 mmHg and a lower diastolic pressure, around 2–10 mmHg.
  - *Pulmonary Artery Pressure*: When the tip of the swan have been advanced beyond the pulmonic valve, the systolic pressure should be the same as the RV systolic pressure, about 20–25 mmHg, with a sharp rise in diastolic pressures to 10–15 mmHg. This is called the diastolic step up and can help to determine when you have entered the PA.
  - *Pulmonary Artery Occlusion Pressure*: Commonly known as the "wedge" pressure, this roughly reflects the pressure in the left atrium of the heart. By inflating the balloon at the tip of the Swan slowly, you can occlude a portion of the pulmonary artery allowing the pressure sensor to become continuous with the left atrium, resulting in a pressure of about 4–12 mmHg

Cardiopulmonary Bypass

- Cardiopulmonary bypass (CPB) is an integral part of cardiothoracic surgery. Emptying, and often times stopping the heart, provides the surgical team with a largely bloodless and motionless operating field [5].
- *Initiation of CPB*:
  - In order to facilitate arterial cannulation, the anesthesiologist will often times work to decrease the systolic blood pressure to about 100 mmHg to help avoid aortic dissection.
  - Prior to initiation, heparin is given to prevent clotting of the bypass circuit, with activated clotting times (ACT) acceptable for initiation varying by institution.
  - In order to allow for retrograde autologous priming (RAPing) of the bypass circuit, the anesthesiologist will sometimes support the blood pressure to allow for adequate drainage from the patient if needed [5].

- *Liberation from CPB:*
  - Liberation from CPB is the process by which the heart is filled with blood and allowed to eject for itself, withdrawing support from the pump circuit.
  - There are a number of physiologic changes that accompany cardiopulmonary bypass including, but not limited to cardiac swelling, lactic acidosis, vasoplegia, etc. that the anesthesiologist will work to mitigate in order to promote independent cardiac function.

    The use of vasopressors such as norepinephrine and vasopressin is commonplace in order to promote vascular tone that is often the result of inflammatory mediators released due to exposure to the CPB circuit.
    Inotropic support in the form of milrinone, dobutamine, and/or epinephrine becomes critical with hearts that had pre-existing ventricular dysfunction or prolonged bypass runs in order to help the stiff swollen heart to produce an adequate ejection fraction.
    In some cases, pulmonary vasodilators may be required in order to promote vasodilation of the pulmonary vasculature and help decrease the afterload of the right ventricle.
    Blood products such as red blood cells, plasma, and platelets are sometimes required at this time, especially in the setting of long bypass runs where clotting factors may be destroyed, consumed, or diluted in order to facilitate adequate surgical field hemostasis for closing.

  - The role of echo is critical at this time in the surgery as it allows the anesthesiologist to ensure that the operation is successful and there are no, or acceptable, residual lesions [5].
  - In some cases, the decision may be made to return to CPB temporarily in order to optimize the patient's condition both in vascular tone and inotropic function if initial liberation fails.

    In rare cases, patients are unable to liberated from CPB support immediately after surgery for a variety of reasons, at which time the initiation of mechanical circulatory support may be warranted.

References

1. Mittnacht AJC, Reich DL, Sander M, Kaplan J. Monitoring of the heart and vascular system. In: Kaplan's essentials of cardiac anesthesia for cardiac surgery. 2nd ed. Elsevier; 2018. p. 203–319.
2. Mittnacht AJC, London MJ, Puskas JD, Kaplan J. Anesthesia for cardiac surgical procedures. In: Kaplan's essentials of cardiac anesthesia for cardiac surgery. 2nd ed. Elsevier; 2018. p. 322–389.
3. Royster RL, Groban L, Grosshans DW, Jones-Haywood MM, Slaughter TF. Cardiovascular pharmacology. In: Kaplan's cardiac anesthesia: the echo era. 6th ed. Elsevier; 2011. p. 235–295.
4. Mitter N, Grogan K, Nyhan D, Berkowitz DE. Cardiovascular pharmacology. In: Kaplan's cardiac anesthesia: the echo era. 6th ed. Elsevier; 2011. p. 193–234.
5. Bojar RM. Cardiopulmonary bypass. In: Manual of perioperative care in adult cardiac surgery. 5th ed. Hoboken: Blackwell Publishing Ltd; 2011. p. 229–54.

Chapter 8
Transfusion Medicine and Blood Management During Cardiac Surgery

Derek He and Kinza Berical

Introduction

Blood transfusions and the management of blood products are necessary and common life-saving interventions during the care of cardiac surgery patients undergoing cardiopulmonary bypass (CPB). These interventions include preoperative optimization, intraoperative blood conservation, and perioperative transfusion of different blood products prompted by specific triggers and patient needs.

Preoperative Risk Assessment

- Emphasis should be placed on identifying patients who have a higher likelihood of requiring blood transfusions perioperatively. Risk factors include advanced age, pre-existing anemia with a hemoglobin of less than 8 g/dL, chronic illnesses or comorbidities, and abnormal coagulation profiles on preoperative labs [1].
- Typically, the more severe a patient's anemia is preoperatively, the more severe their complications are with surgery. Failure to correct preoperative anemia is associated with increased transfusion rates, longer intensive care unit (ICU) and hospital lengths of stay, increased rate of acute kidney injury (AKI), and overall higher long-term mortality [1, 2].
- Special care should be placed on preoperative discussions on acceptable interventions with patients, such as Jehovah's Witnesses, who may refuse blood products for religious, cultural, or personal reasons [3].

D. He (✉) · K. Berical
Massachusetts General Hospital and Harvard Medical School, Boston, MA, USA
e-mail: dhe2@mgh.harvard.edu; kberical@partners.org

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_8

Preoperative Treatment of Anemia

Pharmacologic Interventions

- Treatment of anemia prior to cardiac surgery is a strongly recommended intervention and should be done by addressing the potential causes of anemia [3].
- Iron deficiency accounts for up to 50% of anemic patients and is the most common cause of anemia. Treatment of iron deficiency anemia with iron supplementation has few adverse effects and should be started before surgery [4, 5]. However, oral iron, compared to intravenous iron preparations, is poorly tolerated and less effective when started in the immediate preoperative period as it requires at least 6 weeks to improve hemoglobin level [3].
- Erythropoietin (EPO), a hormone synthesized by the kidneys, can be started several days preoperatively to increase red cell mass in anemic patients treated with iron and provide cytoprotective effects on the heart and kidneys. A combination of EPO and iron supplementation has been shown to decrease adverse outcomes and reduce the number of required transfusions [3].

Nonpharmacologic Interventions

- Preoperative autologous blood donation (PABD) is a method of collecting and storing blood from a patient several weeks before surgery so that it is available for transfusion back to the donor intraoperatively. While this strategy is associated with a lower rate of allogenic transfusions and transfusion-related adverse effects, it is costly and time-intensive to prepare [3].

Preoperative Anticoagulant and Antiplatelet Management

Anticoagulant Management

- An increasing number of cardiac surgery patients now manage comorbidities like atrial fibrillation, stroke, and venous thromboembolism with nonvitamin K oral anticoagulants (NOACs) as opposed to the previously popular vitamin K antagonist, warfarin. This is because NOACs have fixed oral dosing without the need for routine lab monitoring [3].

Table 8.1 Anticoagulant mechanism and reversal [3]

Anticoagulant	Anticoagulation mechanism	Reversal agent	Reversal mechanism
Warfarin	Inhibits vitamin K activation, indirectly decreasing coagulation factor synthesis	Vitamin K, FFP, or prothrombin complex concentrate (PCC) [6]	Restores deficient vitamin K and vitamin K-dependent coagulation factors (II, VII, IX, and X)
Dabigatran	Directly inhibits thrombin (factor IIa)	Idarucizumab[a]	Binds and inhibits dabigatran at its active site
Rivaroxaban, apixaban	Directly inhibits factor Xa	Andexanet alfa[a]	Binds apixaban or rivaroxaban in place of factor Xa molecules

[a] If specific antidotes are not available, PCC can be used as an alternative; however, effectiveness can be variable when used under these circumstances

- Popular NOACs include:
  - Thrombin inhibitors: Dabigatran
  - Factor Xa inhibitors: Apixaban or rivaroxaban
- It is recommended to discontinue NOACs for at least 2 days prior to surgery to minimize bleeding caused by these medications [3].
- If necessary, specific antidotes can be used to reverse anticoagulant effects (see Table 8.1).

Antiplatelet Management

- Dual antiplatelet therapy (DAPT) with P2Y12 inhibitors (ticagrelor, clopidogrel, and prasugrel) and aspirin is used to decrease risk of thrombosis in patients with acute coronary syndrome. These agents prevent the expression of GPIIb/IIIa on the surface of activated platelets, inhibiting platelet adhesion and aggregation [3].
- To minimize bleeding during surgery, P2Y12 inhibitors should be stopped for varying amounts of time preoperatively [3].
  - Ticagrelor: at least 3 days
  - Clopidogrel: at least 5 days
  - Prasugrel: at least 7 days
- It is also reasonable to pursue preoperative platelet function testing as patients with significantly diminished platelet function may require delaying surgery if possible

Intraoperative Blood Management

Pharmacologic Interventions for Bleeding Prevention

- Synthetic antifibrinolytic agents like epsilon aminocaproic acid (EACA or Amicar) or tranexamic acid (TXA) should be used intraoperatively to reduce blood loss during cardiac surgery. These medications bind to the lysine receptors on plasminogen, preventing its activation to plasmin and its subsequent effects on fibrin degradation [4].
  - Care should be taken to monitor for seizures caused by TXA [3]

Nonpharmacologic Interventions for Blood Conservation

- Whenever possible, surgical alternatives with lower risk of bleeding should be considered; this is especially relevant when minimally invasive approaches are viable options.
- Placement of thoracic aortic endografts for thoracic aortic disease can significantly decrease blood loss in an otherwise high-risk population. Transcatheter valve technology can similarly decrease transfusion requirements for patients with structural heart disease. Off-pump coronary surgery compared to on-pump coronary surgery has also been shown to reduce the need for transfusions; however, inferior survival outcomes across patient groups preclude this technique from more widespread use [3].

Intraoperative Hemostasis Testing

- Clotting abnormalities due to inherited defects, anticoagulant and antithrombotic drug use, or intraoperative contact with the extracorporeal bypass circuit can lead to bleeding and/or thrombotic events during surgery. For this reason, point of care (POC) hemostatic monitoring with viscoelastic devices is recommended to better direct hematologic treatment [3, 7].
- Viscoelastic tests, such as a thromboelastography (TEG), have several advantages to routine plasma-based coagulation testing (see Table 8.2) and have become a more popular method of assessing excessive bleeding.
- Interpreting TEGs to guide transfusions requires understanding the individual components of the tracing and recognizing the corresponding blood products used for treating deficiencies (see Fig. 8.1 and Table 8.3).

Table 8.2 Comparison of routine coagulation tests and TEG

Routine coagulation tests	Viscoelastic tests, e.g., TEG
• Only provides the time to initiation of clot formation • Tests are performed in a central laboratory, which requires increased turnover time	• Provides information on platelet–fibrinogen interaction during clot formation • Can be performed at the bedside in the operating room, intensive care unit, or emergency department • Shown to decrease number of unnecessary transfusions, thereby reducing medical costs and the risks of transfusion reactions

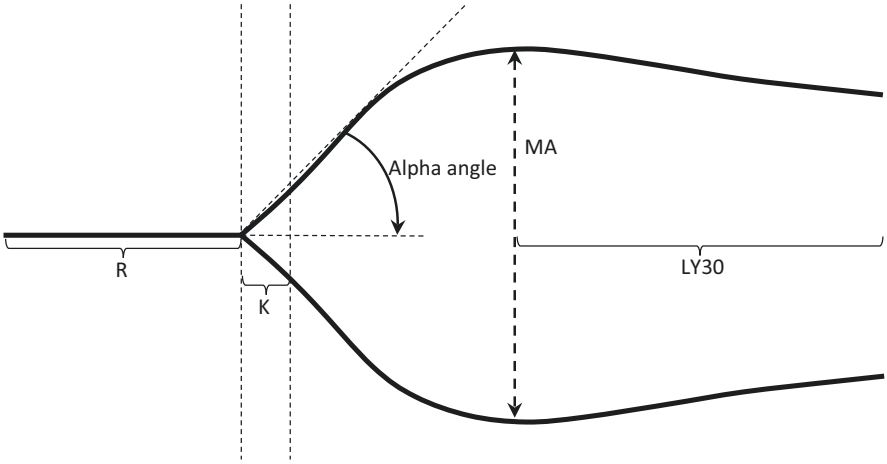


Fig. 8.1 Thromboelastography tracing

Table 8.3 Thromboelastography components

Thromboelastography (TEG)				
Components	Definition	Normal values	Deficiency	Treatment
R time	Time to start forming clot	5–10 min	Coagulation factors	FFP
K time	Time for clot to reach a fixed strength	1–3 min	Fibrinogen	Cryoprecipitate
Alpha angle	Speed of fibrin accumulation	53–72°	Fibrinogen	Cryoprecipitate
Maximum amplitude (MA)	Highest vertical amplitude of the TEG	50–70 mm	Platelets	Platelets/ DDAVP
Lysis at 30 min (LY30)	Percentage of amplitude reduction 30 min after maximum amplitude	0–8%	Excess fibrinolysis	TXA or Amicar

Perfusion Interventions to Reduce Blood Loss

- Acute normovolemic hemodilution (ANH) is a method of limiting blood loss and reducing the amount of allogenic blood transfusions required during cardiac surgery. ANH is performed by replacing whole blood from the patient with crystalloid or colloid intravenous fluids immediately prior to initiation of CPB [8]. The whole blood is then autologously re-infused into the patient either during surgery or after separation from CPB. In doing so, the patient loses blood volume at a lower hematocrit and is transfused with his or her own red blood cells, thereby reducing the risk of adverse effects associated with transfusion of allogenic blood products [3].
  - Other benefits of ANH include:

    Preservation of red blood cells from CPB circuitry
    Preservation of coagulation ability by readministering whole blood containing clotting factors and platelets
    Improved perfusion and oxygenation during CPB by decreasing blood viscosity
    Relatively low-cost and low risk since patients are autologously transfused [8]

  - Naturally, the greater the volume of whole blood that can be removed from the patient without causing hemodynamic instability, the greater the benefit that can be achieved with ANH [3].
  - Factors that limit the use of this strategy include preoperative anemia, lower total blood volume with smaller patients, comorbidities that predispose patients to hemodynamic instability, and patients that are already unstable [3].
- Retrograde autologous priming (RAP) is a method of filling the CPB circuit with the patient's blood to diminish the degree of dilution that would occur if the patient's circulation was connected to a bypass circuit primed with crystalloid solution.
  - Since RAP has been shown to reduce the number of blood transfusions, increase colloid osmotic pressure, and maintain a higher hematocrit at ICU admission and discharge, it should be used whenever possible [3].
- Minimally invasive extracorporeal circulation (MiECC) is a combination of strategies aimed at optimizing patient blood and CPB circuit compatibility. Minimizing priming volumes, using a closed CPB circuit, limiting circuit components to biologically inert material, and opting for a centrifugal pump over a roller pump are all techniques to reduce blood loss and dysfunction [3].
- Cell saver or cell salvage is a technology used intraoperatively to collect shed blood that would have otherwise been lost, wash off debris, and concentrate the red blood cells (RBCs) to be returned to the patient [4, 9].
  - Although there is a danger of bacterial contamination with this technique, this risk is largely outweighed by the benefit of reducing allogenic blood transfusions, especially during cardiac surgeries with large amounts of blood loss.

Post-bypass and Postoperative Blood Management

Laboratory Studies

- Lab studies should be sent at the cessation of CPB and on arrival to the ICU to assess the extent of intraoperative blood loss and coagulation abnormalities that may require correction. Important hematologic lab values that should be prioritized include:
  - Hemoglobin
  - Platelet count
  - PT-INR, PTT, fibrinogen
- Heparin is administered prior to CPB to prevent coagulation as the patient's blood circulates through the bypass machine. On completion of cardiac surgery and after coming off bypass, heparin's effects need to be reversed. This can be accomplished with protamine sulfate (see Chap. 9 for more details). However, if there is ongoing bleeding following heparin reversal with protamine, the activated clotting time (ACT) should be rechecked, and additional doses of protamine based on the ACT should be administered. Utilizing POC hemostatic tests, such as TEG, can also help delineate sources of ongoing bleeding [7].

Transfusion Triggers*

- Anemia:
  - Multiple studies have now established that a more conservative RBC transfusion strategy using a target hemoglobin of 7–8 g/dL reduces the number of blood transfusions without increasing mortality or morbidity rates [2, 3, 10].
  - In fact, allogenic RBC transfusion to a more liberal target hemoglobin of 10 g/dL is not recommended as it is unlikely to improve oxygen transport, prevent infection, or reduce the risk of postoperative ischemic events [3, 10, 11].
  - Recent studies have demonstrated the utility of using factors other than hemoglobin, such as central venous oxygen saturation (SvO_2), as transfusion triggers [12]. Since SvO_2 is a measurement of the body's oxygen delivery and consumption, it can help guide transfusion decisions to match the rise in metabolic demand after cardiac surgery.
- Thrombocytopenia:
  - Platelets should be transfused for thrombocytopenia with ongoing bleeding.
  - In the case of demonstrable platelet dysfunction secondary to uremia, prolonged CPB, or von Willebrand factor (vWF) deficiency, use of desmopressin (DDVAP) 0.3 mcg/kg is reasonable.

- Coagulopathy and fibrinogenemia
  - Fresh frozen plasma (FFP) should be transfused for an elevated INR with ongoing bleeding. However, data does not support giving FFP prophylactically during CPB.
  - If INR remains elevated or if there is ongoing bleeding despite FFP administration, prothrombin complex concentrate (PCC) can be considered as an alternative treatment option [6].
  - In the case of intractable coagulopathy, activated Factor 7 can be considered.
  - Given the prothrombotic risks, the use of factor concentrates should be discussed with the surgical team, hematology, and the blood bank for dosing and appropriateness, particularly in patients who will be managed on mechanical support devices postoperatively.
  - In the case of fibrinogenemia with ongoing bleeding, cryoprecipitate can be administered. Studies regarding the use of fibrinogen concentrates in cardiac surgery are ongoing but may be a reasonable alternative [13].

[*] *Specific transfusion triggers will vary from institution to institution.*

References

1. Padmanabhan H, Brookes MJ, Nevill AM, Luckraz H. Association between anemia and blood transfusion with long-term mortality after cardiac surgery. Ann Thorac Surg. 2019;108(3):687–92. https://doi.org/10.1016/j.athoracsur.2019.04.044.
2. Patel NN, Murphy GJ. Evidence-based red blood cell transfusion practices in cardiac surgery. Transfus Med Rev. 2017;31(4):230–5. https://doi.org/10.1016/j.tmrv.2017.06.001.
3. Tibi P, McClure RS, Huang J, et al. STS/SCA/AmSECT/SABM update to the clinical practice guidelines on patient blood management. Ann Thorac Surg. 2021;112(3):981–1004. https://doi.org/10.1016/j.athoracsur.2021.03.033.
4. Blaudszun G, Butchart A, Klein AA. Blood conservation in cardiac surgery: blood conservation in cardiac surgery. Transfus Med. 2018;28(2):168–80. https://doi.org/10.1111/tme.12475.
5. Hubert M, Gaudriot B, Biedermann S, et al. Impact of preoperative iron deficiency on blood transfusion in elective cardiac surgery. J Cardiothorac Vasc Anesth. 2019;33(8):2141–50. https://doi.org/10.1053/j.jvca.2019.02.006.
6. Karkouti K, Bartoszko J, Grewal D, et al. Comparison of 4-factor prothrombin complex concentrate with frozen plasma for management of hemorrhage during and after cardiac Surgery: a randomized pilot trial. JAMA Netw Open. 2021;4(4):e213936. https://doi.org/10.1001/jamanetworkopen.2021.3936.
7. Fleming K, Redfern RE, March RL, et al. TEG-directed transfusion in complex cardiac surgery: impact on blood product usage. J Extra Corpor Technol. 2017;49(4):283–90.
8. Barile L, Fominskiy E, Di Tomasso N, et al. Acute normovolemic hemodilution reduces allogeneic red blood cell transfusion in cardiac surgery: a systematic review and meta-analysis of randomized trials. Anesth Analg. 2017;124(3):743–52. https://doi.org/10.1213/ANE.0000000000001609.
9. Neef V, Vo L, Herrmann E, et al. The association between intraoperative cell salvage and red blood cell transfusion in cardiac surgery—an observational study in a patient blood management centre. Anaesthesiol Intensive Ther. 2021;53(1):1–9. https://doi.org/10.5114/ait.2021.103735.

10. Mazer CD, Whitlock RP, Fergusson DA, et al. Restrictive or liberal red-cell transfusion for cardiac surgery. N Engl J Med. 2017;377(22):2133–44. https://doi.org/10.1056/NEJMoa1711818.
11. Patel NN, Avlonitis VS, Jones HE, Reeves BC, Sterne JAC, Murphy GJ. Indications for red blood cell transfusion in cardiac surgery: a systematic review and meta-analysis. Lancet Haematol. 2015;2(12):e543–53. https://doi.org/10.1016/S2352-3026(15)00198-2.
12. Zeroual N, Blin C, Saour M, et al. Restrictive transfusion strategy after cardiac surgery. Anesthesiology. 2021;134(3):370–80. https://doi.org/10.1097/ALN.0000000000003682.
13. Callum J, Farkouh ME, Scales DC, et al. Effect of fibrinogen concentrate vs cryoprecipitate on blood component transfusion after cardiac surgery: the FIBRES randomized clinical trial. JAMA. 2019;322(20):1966–76. https://doi.org/10.1001/jama.2019.17312.

Chapter 9
Cardiopulmonary Bypass

Chase C. Marso and Kenneth G. Shann

Basic Components of the Heart-Lung Machine

The heart-lung machine is both intricately complex and at the same time simple in its principles. The core functions of CPB establish mechanisms to (1) drain the body's deoxygenated blood, (2) oxygenate, heat, and pump extracorporeal blood, (3) re-introduce oxygenated blood to the body, and (4) establish cardioplegia while providing the heart with oxygenated blood (Fig. 9.1). The role of the perfusionist in managing the heart-lung machine cannot be overstated and is further discussed in Chap. 33 of this text.

C. C. Marso (✉)
Department of Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: CCMarso@mgh.harvard.edu

K. G. Shann
Division of Cardiac Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: kshann@mgb.org

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_9

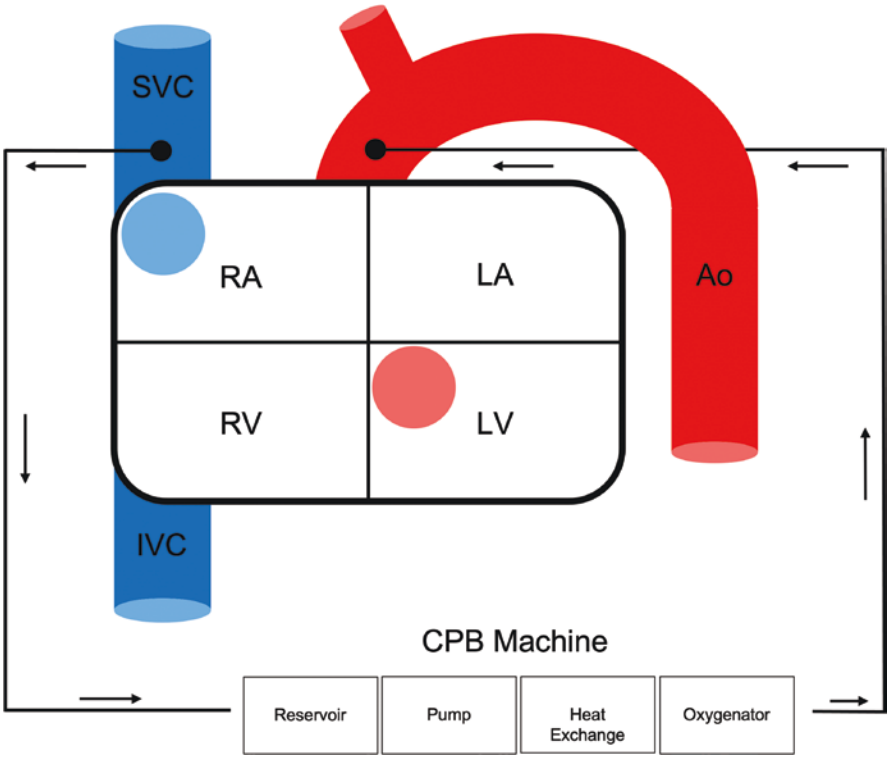


Fig. 9.1 Diagram of cardiopulmonary bypass

Heart-Lung Machine Safety

The heart-lung machine incorporates several safety devices to minimize the two greatest risks of the machine: circuit disruption and gaseous embolism. Standard safety devices include servo-regulated pressure control of the arterial and cardioplegia pumps to prevent high pressure due to kinking or inadvertent clamping of tubing. In addition, servo-regulated bubble and level detection are routinely utilized to avoid drainage of the venous reservoir and introduction of a massive air embolism. If air is introduced into the circuit, or if the reservoir level drops, the arterial pump will stop to allow the perfusionist to remove air.

Cannulation Strategies

Figure 9.1 demonstrates common access sites for cannula when initiating CPB, but multiple access sites are suitable for each cannula. Venous cannulas are often placed in the vena cava and right atrium (also known as dual stage cannulation), but

bicaval, femoral vein, and internal jugular vein access are suitable alternatives. Bicaval cannulation is useful to fully drain the right heart. Oxygenated blood can be returned to the body via aortic, innominate, axillary, or femoral artery access. Common indications for peripheral access include aortic arch surgery and minimally invasive operations. To "vent," or drain, the left ventricle, a cannula may be inserted into the aortic root, the left ventricular apex, the left ventricle via the right superior pulmonary vein, or the pulmonary artery.

Standard Heparin Dosage and Reversal

Anticoagulation via heparin is essential during cardiopulmonary bypass to prevent both extracorporeal coagulation and an intra-operative embolic event. The level of anticoagulation achieved is measured by point of care testing. The Activated Clotting Time (ACT) along with heparin levels (heparin-protamine titration) are the preferred tests. Prior to cannulation, a sample of the patient's whole blood is tested for heparin sensitivity and a heparin bolus is calculated to achieve an ACT ≥400 s and a heparin level ≥2.7 u/mL. During CPB, the ACT is maintained ≥400 s and the heparin level ≥2.0 u/mL. The ACT and heparin level should be checked at least every 30 min, as additional heparin bolus may necessary. At the conclusion of the operation, heparin reversal is achieved through protamine infusion. Protamine dosing is calculated by measuring the heparin level, followed by administration of protamine at 0.8–1.0 mg protamine per 100 units of heparin. Finally, ACT and heparin levels should again be checked to verify the success of reversal [1, 2]. Of note, in rare cases such as in patients with Heparin Induced Thrombocytopenia (HIT), heparin and heparin-specific monitoring cannot be used, and alternative pharmacologic agents and testing must be utilized.

Optimal Flow Rates and MAP

When a patient is on CPB, the heart cannot regulate the body's metabolic needs and accommodate for poor tissue oxygenation by increasing cardiac output. Consequently, patients on CPB must be closely monitored to ensure adequate oxygenation—a task made more difficult due to metabolic changes associated with hypothermia induced during CPB. The most common mechanisms of measuring tissue perfusion include venous oxygen saturation and lactate (a carbon dioxide-derived parameter). However, these markers are imperfect. As a result, optimal CPB flow rates have historically been initiated in the range of 2.2–2.8 L/m^2/min at 35–37 °C and adjusted based on hypothermia (guideline to reduce flow by 7% for each 1 °C reduction) and the aforementioned markers [3]. Recent studies have demonstrated that oxygen delivery—with a goal delivery ≥270 mL/min/m^2—may be used as an adjunct or alternative metric to flow rates calculated from body surface

area [4]. In addition to titrating flow rates, perfusionists closely monitor MAP. Historically, MAP has been maintained at 50–80 mmHg, but sustaining higher MAP (i.e., ≥65 mmHg and up to ≥75 mmHg for patients with cerebrovascular disease) during CPB by increasing the flow rate or administering vasopressors reflects more recent practice patterns in an effort to maintain adequate end organ perfusion. Commonly used vasopressors include α-agonist phenylephrine and norepinephrine as well as the V1 receptor agonist vasopressin. In cases of extreme vasoplegia, methylene blue or hydroxocobalamin may be administered.

Cooling and Warming

During CPB, hypothermia is induced as a cytoprotective measure, particularly for the brain and myocardium. Inducing hypothermia during cardiac surgery is difficult because of the challenges of managing heat transfer through the heart-lung machine. It is therefore critical to monitor temperature closely with multiple temperature probes; the most common sites include the pulmonary artery, nasopharynx, esophagus, and bladder. During CPB, patients are routinely cooled to a core body temperature 32–34 °C. For particularly difficult procedures such as aortic arch surgery, patients may be cooled to 18 °C. During cooling, it is important to maintain a low temperature gradient (<10 °C) between arterial outflow and venous inflow to avoid formation of gas emboli. Approximately 10 minutes prior to the release of the aortic cross-clamp and prior to weaning from CPB, patients are warmed at a rate of ≤0.5 °C, with an arterial-venous gradient ≤4 °C. During rewarming and immediately post-operatively, to avoid cerebral hyperthermia a patient's core body temperature should not exceed 37 °C.

Steps to Initiate and Separate from CPB

Initiating and separating a patient from CPB are processes that demand precise care coordination among the cardiac surgery care team. Both processes are outlined below. In addition, checklists, a crucial asset in surgery [5, 6], are employed during initiating and separating patients from CPB (Fig. 9.2).

Initiation

1. Set up the CPB circuit (Fig. 9.2)
   (a) Perfusionist completes setup, priming, testing
2. Establish anticoagulation
3. Arterial cannulation

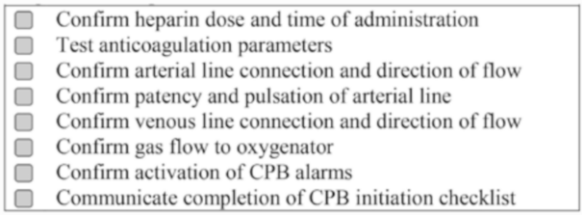

- [ ] Confirm heparin dose and time of administration
- [ ] Test anticoagulation parameters
- [ ] Confirm arterial line connection and direction of flow
- [ ] Confirm patency and pulsation of arterial line
- [ ] Confirm venous line connection and direction of flow
- [ ] Confirm gas flow to oxygenator
- [ ] Confirm activation of CPB alarms
- [ ] Communicate completion of CPB initiation checklist

Fig. 9.2 Example of perfusionist checklist for CPB initiation

4. Venous cannulation
5. Autologous priming
   (a) Retrograde/venous priming
      - Utilizes patient's blood to remove crystalloid priming solution
   (b) Reduces hemodilution to avoid low nadir hematocrit levels
6. Start "On-Pump"
   (a) The patient's heart and lungs remain functional
7. Turn off ventilator and place aortic cross-clamp
8. Induce cardioplegia
   (a) The patient is now dependent on CPB

Separation

1. Restart the heart
   (a) Rewarm the patient (0.3–0.5 °C/min)
   (b) De-air the heart—prevent air embolus
   (c) Resume electrical activity—epicardial pacing wires are used to establish normal sinus rhythm
2. Restart the lungs—ventilate the patient
3. Confirm weaning criteria—see below
4. Wean CPB
   (a) Venous line is gradually clamped, and pump flow is gradually decreased
   (b) Venous cannula is removed
5. Reverse anticoagulation
   (a) Administration of protamine
6. Arterial decannulation

Criteria for Discontinuing CPB

To facilitate CPB, normal physiology is altered. Therefore, when the cardiac procedure is complete and a patient is prepared to be weaned from CPB, the operative team must restore not only mechanical function of the heart and lungs, but also physiologic homeostasis. Criteria can be organized into four categories: heart, lung, metabolism, and anesthesia. Cardiac criteria for discontinuation of the heart-lung machine include restoration of normal sinus rhythm with a normal heart rate and blood pressure (with or without rate control agents, inotropes, and vasopressors). A transesophageal echocardiogram should also confirm an absence of air in the cardiac chambers. Pulmonary physiology is restored through mechanical ventilation and confirmed through PaO_2 monitoring. Metabolically, the patient should be warmed to 35–37 °C, normal acid-base status should be achieved (pH 7.35–7.45), and serum potassium (K^+ 4–5.5 mmol/L), hemoglobin (>7.0 mg/dL), calcium (1.09–1.30 mmol/L) should be normalized. Finally, the anesthesia team should confirm preparedness to resume full control of the patient [1].

Circulatory Arrest and Special Considerations

Aortic arch surgery presents added challenge to cardiac surgery because the surgical field must be free of CPB equipment, including cannulas and clamps. To accommodate aortic arch operations, temporary hypothermic circulatory arrest is induced. Special considerations of circulatory arrest are management of acid-base status and alternative cerebral perfusion techniques.

Alpha Stat and pH Stat

There are two common ways to manage intra-operative pH. Alpha-stat has been traditionally used for CPB (particularly for non-circulatory arrest) and provides a pH that is corrected for the patient's hypothermic state. Accordingly, the patients pCO_2 is maintained in a normal range (35–45 mmHg) at 37 °C, resulting in a low true cerebral pCO_2. Conversely, pH-stat maintains a normal pCO_2 at a patient's true intraoperative temperature. This technique increases cerebral pCO_2, leading to a local cerebral vasodilatory response and increased cerebral perfusion, which promotes complete and homogenous cerebral cooling. pH-stat is also more frequently used in pediatric cardiac surgery.

ACP and RCP

Antegrade cerebral perfusion (ACP) is a CPB technique used in aortic arch surgery to perfuse only the brain. ACP is facilitated through arterial cannulation of either the axillary artery or a branch of the aortic arch. ACP's biggest advantage is neuroprotection through minimization of brain ischemia during hypothermic circulatory arrest.

Retrograde cerebral perfusion (RCP) is another CPB technique that provides blood flow only to the brain. In RCP, the venous cannula is inserted into the SVC and the brain is perfused with oxygenated blood retrograde from the venous system that normally drains the brain. Like ACP, RCP is utilized to minimize brain ischemia during circulatory arrest.

References

1. Ismail A, Semien G, Miskolczi S. Cardiopulmonary bypass. Treasure Island: StatPearls Publishing; 2021.
2. Shore-Lesserson L, Baker RA, Ferraris V, Greilich PE, Fitzgerald D, Roman P, Hammon J. STS/SCA/AmSECT clinical practice guidelines: anticoagulation during cardiopulmonary bypass. J Extra Corpor Technol. 2018;50:5–18.
3. Kirklin J, Barratt-Boyes B. Cardiac surgery. 2nd ed. New York: Churchill Livingstone; 1993.
4. Ranucci M, Johnson I, Willcox T, et al. Goal-directed perfusion to reduce acute kidney injury: a randomized trial. J Thorac Cardiovasc Surg. 2018;156:1918–1927.e2.
5. Gawande A. The checklist manifesto: how to get things right, vol. 1. New York: Henry Holt and Company; 2010. p. 64.
6. Haugen AS, Sevdalis N, Søfteland E. Impact of the World Health Organization surgical safety checklist on patient safety. Anesthesiology. 2019;131:420–5.

Chapter 10
Myocardial Protection

Brittany A. Potz and Pedro del Nido

Cardiac Metabolic Demand in Different States

The heart is an obligate aerobic organ that depends on a continuous supply of oxygen to maintain normal function. Myocardial oxygen consumption (MVO^2) is compartmentalized as oxygen needed for external work of contraction (80–90%) and "unloaded" contraction such as basal metabolism and heat production. Seventy-five percent of the coronary arterial oxygen that is presented to the myocardium is extracted during a single passage through the heart making it highly susceptible to the limitations of oxygen delivery [1].

Under normal conditions, the heart derives its energy from mitochondrial oxidative processes to make adenosine triphosphate (ATP). Oxidation of fatty acids provides the major source of energy production and is used preferentially to carbohydrates [1].

Myocardial ischemia reperfusion injury occurs as a result of cessation of coronary blood flow so that the oxygen delivery to the myocardium is insufficient to meet the basal myocardial oxygen requirements to preserve cellular membrane stability and viability. This injury can be reversible or irreversible. Surgical induced ischemia is a reversible form of myocardial injury [1].

As the partial pressure of oxygen falls in the myocardial tissue, oxidative phosphorylation, electron transport, and mitochondrial ATP production stop. The heart requires a minimum threshold of ATP to prevent irreversible ischemic injury so the

B. A. Potz
Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA
e-mail: bpotz@partners.org

P. del Nido (✉)
Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA

Department of Cardiac Surgery, Boston Children's Hospital, Boston, MA, USA
e-mail: Pedro.Delnido@cardio.chboston.org

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_10

heart begins to depend on energy from glycogenolysis and anaerobic glycolysis [1]. This leads to the accumulation of glycolytic intermediaries, reduced nicotinamide adenine dinucleotide + hydrogen (NADH), and reduction of pyruvate to lactate culminating in severe intracellular acidosis which impairs contractile function, enzyme transport, and cell membrane integrity. The loss of cell membrane integrity leads to cellular loss of potassium and pathologic accumulation of intracellular sodium, calcium, and water [1].

Minimizing myocardial oxygen demand, preventing sodium and calcium entry into the cell, preventing myocardial swelling, and maintaining high-energy phosphates (such as ATP) are the primary principles of cardioplegic induced myocardial arrest.

Basic Elements of Cardioplegia Solution

The application of cardioplegic solutions to provide myocardial protection consists of the following main goals: to induce rapid cardiac arrest to minimize myocardial work, to buffer the acidosis caused by ischemia, to provide the necessary substrates for cell viability during reperfusion, to prevent against myocardial edema, and to cause hypothermia to minimize basal metabolic requirements (Table 10.1) [1].

Table 10.1 Basic elements of cardioplegia

Rapid diastolic arrest	Buffering agents	Metabolic substrates	Isotonic solution	Hypothermia (4°)
Minimize myocardial work	Buffer H^+ accumulation caused by ischemia	Enhance anaerobic metabolism and facilitate hemostasis	Prevent ischemia induced myocardial ischemia	Minimize degradation of energy stores and basal metabolic demands
Mechanisms				
Potassium or lidocaine a sodium channel blocker	Bicarbonate	Glucose	Mannitol	Mild (28–32°)
	Phosphate	Insulin	Sorbitol	Moderate (22–25°)
	Aminosulfonic acid	Adenosine	Glucose	
	THAM	Aspartate	Dextrose	
	Histidine Red blood cells contain carbonic anhydrase	Glutamate	Albumin	

Rapid Cardiac Arrest

Inducing rapid cardiac arrest minimizes the external work of the myocardium contracting against the fixed afterload that is caused by the application of the aortic cross clamp.

Potassium in high concentration is the most common agent used for chemical cardioplegia. As the extracellular potassium concentration increases with the application of cardioplegia, the resting myocardial cell membrane becomes depolarized and the voltage-dependent fast sodium channel is inactivated which arrests the heart in diastole. The slow calcium channel is then activated which results in cytosolic calcium overload [1]. Potassium-based solution produces rapid diastolic arrest at concentrations between 15 and 40 mmol/L. The heart remains arrested until the concentration of extracellular potassium is decreased by noncoronary collateral blood flow so reinfusion of cardioplegia is typically necessary.

Buffering of Cardioplegia Solution

Surgically induced myocardial ischemia leads to intracellular acidosis through the mechanisms described above. Cardioplegia solutions contain a number of buffers to counteract this acidosis including bicarbonate, phosphate, amino sulfonic acid, tris (hydroxymethyl) aminomethane (THAM), and histidine. A pH 6.8 or greater provides adequate myocardial protection [1].

Avoidance of Substrate Depletion

Metabolic substrates can be added to cardioplegic solutions to enhance anaerobic metabolism during ischemia and to provide citric acid cycle intermediates to facilitate homeostasis during reperfusion. In some formulations of cardioplegia these agents include glucose with or without insulin, aspartate, glutamate, and others [1].

Avoidance of Myocardial Edema

Myocardial edema is a consequence of surgically induced ischemia. The extent of myocardial interstitial edema is directly modulated by the osmolarity of the cardioplegia. Isotonic solutions in the range of 290–330 mOsm/L are typically used. Inert sugars such as mannitol and sorbitol and metabolized sugars such as glucose and dextrose are often used to increase osmolarity. Oncotic agents such as albumin can also be used.

Hypothermia

Inducing hypothermia decreases the rate of metabolic degradation of energy stores during surgically induced ischemia. Mild hypothermia (28–32 °C) or moderate (22–25 °C) is chosen based on the length of the procedure, with longer cases cooling to lower degrees [1]. Typically, cardioplegia solution is administered at 4 °Celsius to help cool the heart. The cardioprotective effects of hypothermia have been expressed using the "Q10 rule," which says that for every 10° drop in temperature, metabolic rate decreases by 50%.

Modes of Cardioplegia Delivery

Cardioplegia can be delivered to the myocardium in an antegrade or a retrograde fashion. Antegrade cardioplegia allows cardioplegia to flow following the natural anatomic route from the aorta down the right and left coronary arteries. Antegrade cardioplegia is delivered via a perfusion catheter placed in the aorta just distal to the aortic root but proximal to the aortic cross clamp. The aortic root is pressurized as the cardioplegia is administered in the aorta between the cross clamp and a competent aortic valve. This pressurization allows the cardioplegia to flow down the coronary arteries. Antegrade cardioplegia can also be delivered directly down the coronary arteries via a handheld catheter held to the coronary ostia after the aorta is already open. Antegrade cardioplegia usually induces electrical arrest of the heart in about 30–60 s.

Retrograde cardioplegia is delivered by placing a catheter through the R atrium into the coronary sinus and sending the cardioplegia through the coronary veins. Retrograde cardioplegia takes longer to induce electrical arrest than antegrade cardioplegia (about 2–4 min). One disadvantage of retrograde cardioplegia is that it may induce incomplete protection to the right ventricle because the catheter typically sits in the coronary sinus beyond the site where the first few veins drain the right ventricle. An advantage to retrograde cardioplegia is that it flushes air and emboli out of the coronaries. Additionally, retrograde cardioplegia is particularly helpful in the setting of a redo operation with a previous left internal mammary to left anterior descending bypass graft (LIMA-LAD) as antegrade cardioplegia would perfusion the obtuse marginal and posterior descending artery territories but would not perfuse the left anterior descending territory that is perfused by the left internal mammary artery graft. Additionally, in the setting of aortic insufficiency, retrograde cardioplegia helps to ensure adequate cardioplegia delivery.

Flow and Pressure

Once cardioplegia is started, the surgeon must confirm with the perfusionist that there is adequate line pressure and flow. The initial arresting dose of cardioplegia is typically between 1 and 1.2 L. High line pressure can be a concern for dissection of the coronary arteries. Low line pressure is an indication of poor delivery.

Table 10.2 Cardioplegia flow and pressure

Cardioplegic route	Flow rate	Pressure
Antegrade aortic	Up to 400 mL/min	System pressure ≤ 250 mmHg
Left main coronary	150–250 mL/min	System pressure ≤ 200 mmHg
Right main coronary	50–200 mL/min	System pressure ≤ 200 mmHg
Retrograde	200–400 mL/min	Coronary sinus pressure 20–45 mmHg

For antegrade cannulation, the upper limit of appropriate system pressure at the site of the pump is 250 mmHg for aortic cannulation, which reflects a flow rate of up to 400 mL/min at a typical aortic root pressure of 50–60 mmHg. For direct coronary cannulation, the upper limit of appropriate system pressure at the pump is 200 mmHg, which reflects a flow rate ranging from 50 to 250 mL/min. For retrograde cannulation, the goal flow rate is between 200 and 400 cc/min with a coronary sinus pressure of 20–45 mmHg (Table 10.2).

Achieving Diastolic Arrest

The induction of rapid diastolic arrest after the aorta has been clamped minimizes the depletion of high-energy phosphate moieties by useless mechanical work. The adequacy of cardioplegia is confirmed by the cessation of electrical and myocardial activity by checking the EKG and observation of the heart.

Alternate Formulations

There are many types of cardioplegia solutions but for the purpose of this chapter we will focus on Buckberg, del Nido, and Bretschneider (Table 10.3). All solutions fulfill the basic requirements outlined above. Cardioplegic solutions are either crystalloid or blood based. Historically, cardioplegic solutions were crystalloid based. The addition of blood has been found to serve as a carrier of oxygen and other substrates as well as high buffering capacity, and has been shown to limit reperfusion damage to the myocardium [2, 3]. Cardioplegia is typically given intermittently throughout the case as the substrates get washed away and to provide intermittent cooling to the myocardium.

Buckberg solution uses potassium to induce arrest and is redosed at fairly short intervals (every 15–20 min). Buckberg cardioplegic solution is mixed with fully oxygenated patient blood in a ratio of 1:4, one part crystalloid to four parts blood.

Del Nido solution is calcium free, potassium rich, non-glucose-based solution and has an electrolyte composition similar to the extracellular fluid. The key component in del Nido is that it induces a longer arrest with the use of lidocaine. Lidocaine is a long-acting sodium channel blocker. Sodium channel blockade

Table 10.3 Types of cardioplegia solutions

Buckberg (Blood)		del Nido (Blood)		Bretschneider (Crystalloid)	
Induction dose					
pH	7.2	**pH**	7.4	**pH**	7.02–7.20
Sodium	140 mmol/L	**Plasma-Lyte A**	1200 mL	**Potassium**	10 mmol/L
Potassium	20 mmol/L	**Sodium**	135 mmol/L	**Sodium**	15 mmol/L
Magnesium	13 mmol/L	**Mannitol 20%**	16.3 mL	**Magnesium**	4 mmol/L
Glucose	6 mmol/L	**Magnesium sulfate 50%**	4 mL	**Calcium**	0.02 mmol/L
Maintenance dose		**Sodium bicarb 8.4%**	13 mL	**Histidine/ histidine HCL**	180/18 mmol/L
pH	7.4	**Potassium chloride**	13 mL	**Mannitol**	30 mmol/L
Sodium	140 mmol/L	**Lidocaine 1%**	13 mL	**Tryptophan**	2 mmol/L
Potassium	10 mmol/L			**Alpha-ketoglutarate**	1 mmol/L
Magnesium	9 mmol/L				
Glucose	6 mmol/L				

increases the refractory period of the cardiac mycotic and serves to polarize the cell membrane by preventing sodium and calcium accumulation [4, 5]. It is delivered with fully oxygenated patient blood in a ratio of 4:1, four parts crystalloid to one part blood. Del Nido provides arrest with decreased interval compared to Buckberg (up to 180 min hours if no return of activity), but it is typically redosed at intervals of 40–60 min to provide adequate cooling.

Bretschneider solution is a crystalloid solution that is sodium-poor intracellular cardioplegia solution that acts through depletion of extracellular sodium. It is usually applied as a single infusion but can be reapplied to provide adequate cooling [2].

References

1. Levitsky S, Mccully JD. Chapter 65—Myocardial protection [Internet]. In: Sabiston and Spencer surgery of the chest, 9th ed. Elsevier Inc.; 2022. 1101–1124 p. https://doi.org/10.1016/B978-0-323-24126-7.00065-X.
2. Boening A, Hinke M, Heep M, Boengler K, Niemann B, Grieshaber P. Cardiac surgery in acute myocardial infarction: crystalloid versus blood cardioplegia—an experimental study. J Cardiothorac Surg. 2020;9:1–8.
3. Julia PL, Buckberg GD, Acar C, Partington MT, Sherman MP. Studies of controlled reperfusion after ischemia. J Thorac Cardiovasc Surg [Internet]. 1991;101(2):303–313. https://doi.org/10.1016/S0022-5223(19)36766-2.
4. Matte GS, Del Nido PJ. History and use of del Nido cardioplegia solution at Boston Children's Hospital. J Extra Corpor Technol. 2012;44(3):98–103.
5. Mick SL, Robich MP, Houghtaling PL, Gillinov AM, Soltesz EG, Johnston DR, et al. del Nido versus Buckberg cardioplegia in adult isolated valve surgery. J Thorac Cardiovasc Surg [Internet]. 2015;149(2):626–636.e5. https://doi.org/10.1016/j.jtcvs.2014.10.085.

Chapter 11
Management of Coronary Artery Disease

Dane C. Paneitz and Jennifer S. Lawton

Brief Epidemiology and Pathophysiology

- Cardiovascular disease is the #1 cause of death worldwide with ischemic heart disease (IHD) being its major contributor.
- The overall burden of IHD continues to increase globally with an estimated prevalence of 197 million cases in 2019 [1].
- The development of atherosclerotic lesions underlies the pathophysiology of coronary artery disease [2, 3].
  - The process begins with pathological intimal thickening which involves smooth muscle apoptosis and the accumulation of extracellular matrix components [2].
  - This is followed by the development of a fibroatheroma, which is the result of intimal infiltration by macrophages and the formation of cholesterol clefts [2].
  - Finally, the fibroatheroma cap is thinned out, leaving it vulnerable to rupture and thrombosis [2].
  - The clinical manifestations of coronary artery disease result from flow limitation, rupture, ulceration, or calcification of these fibroatheromas.

D. C. Paneitz · J. S. Lawton (✉)
Division of Cardiac Surgery, Department of Surgery, Johns Hopkins University School of Medicine, Baltimore, MD, USA
e-mail: Dpaneit1@jhmi.edu; jlawton4@jhmi.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_11

Staples of Medical Management for Stable CAD

- Guideline-directed medical therapy (GDMT) includes several components [4]:
  - Lifestyle modifications including increasing physical activity, weight loss, and improving diet.
  - Lipid management with moderate or high dose statin therapy.
  - Blood pressure management with antihypertensive medications for a goal of less than 140/90 mmHg or less than 130/80 mmHg in patients with hypertension AND one of the following: 10-year atherosclerotic cardiovascular disease (ASCVD) of ≥10%, diabetes, or chronic kidney disease.
  - Diabetes management with a targeted hemoglobin A1c of 7% or less in most patients.
  - Smoking cessation.
  - Moderation of alcohol consumption.
  - Antiplatelet medications such as aspirin or clopidogrel.
  - Beta blockers for secondary prevention after myocardial infarction and for initial therapy for relief of symptoms.
  - Sublingual nitroglycerin for immediate relief of angina.

Indications for Revascularization

- Coronary revascularization is indicated to improve symptoms of coronary ischemia, improve survival, and reduce the risk of myocardial infarction, the need for repeat revascularization, and/or the occurrence of cardiovascular events.
- Acute coronary syndromes include ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI), and unstable angina.
  - STEMI is diagnosed with an electrocardiogram (ECG) and treated with emergent reperfusion using percutaneous coronary intervention (PCI) (preferred) or fibrinolytic therapy (much less common) if PCI is not available or possible in addition to medical management.
  - NSTEMI is diagnosed with ECG and elevated troponin level and treated with prompt medical management and coronary angiography with either PCI or surgical revascularization during the same hospitalization for lesions suitable for revascularization.
  - Unstable angina is the ischemic etiology of chest pain or discomfort that does not meet STEMI or NSTEMI criteria, is increased in frequency and/or severity in comparison to stable angina, or is refractory to anti-anginal medical therapy. It is initially managed with antiplatelet medication, coronary vasodilators, and oxygen until the most appropriate revascularization strategy can be determined.

- Stable Ischemic Heart Disease (SIHD) is diagnosed following a workup that typically includes an exercise or pharmacologic stress test, which if positive is concerning for ischemic heart disease, and is followed by a coronary catheterization to identify the sites of the atherosclerotic lesions. For patient who would benefit from revascularization, the optimal method is dependent upon patient and anatomical factors, such as history of diabetes, reduced left ventricular ejection fraction, presence of left main stenosis, multivessel disease, and complex anatomy. Figure 11.1 provides the recommendations for these groups from the 2021 ACC/AHA/SCAI Guideline for Coronary Artery Revascularization. Furthermore, summaries of several important revascularization trials are included in Table 11.1.

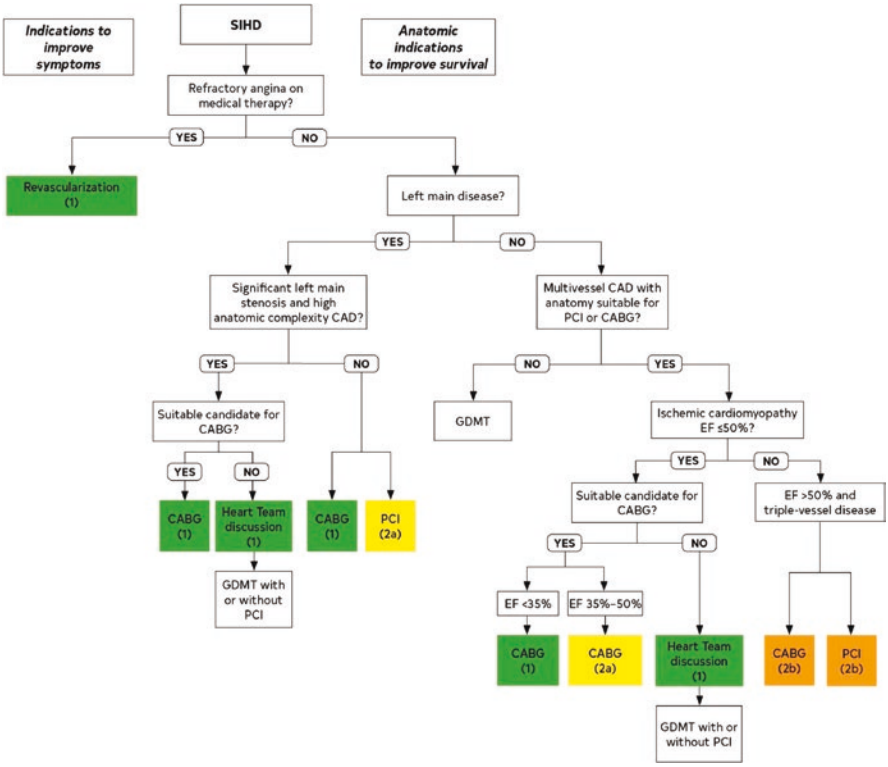


Fig. 11.1 Decision tree for patients with stable ischemic heart disease from the 2021 ACC/AHA/SCAI Guideline for Coronary Artery Revascularization. (Reprinted with permission. Circulation. 2022; 145:e18–e114 © 2021 American Heart Association, Inc.)

Table 11.1 Revascularization trials

Trial	Population	Comparators	Findings
BARI (2007) [12]	Symptomatic patients with multivessel CAD Subgroup analysis in patients with diabetes	CABG (914 patients) vs PTCA (915 patients)	Similar overall 10-year survival: CABG 73.5% vs PTCA 71.0% ($P = 0.18$) Higher subsequent revascularization with PTCA: 76.8% vs 20.3% ($P < 0.001$) Increased 10-year survival in CABG patients with diabetes: CABG 57.8% vs PTCA 45.5% ($P = 0.025$)
FREEDOM (2012) [13]	Patients with diabetes AND multivessel CAD	CABG vs PCI with first- generation DES	Increased incidence of primary outcome (death from any cause, nonfatal MI, or nonfatal stroke) with PCI at median 3.8 years follow-up: 26.6% vs 18.7% ($P < 0.001$) Higher 5-year stroke rate in CABG: 5.2% vs 2.4% ($P = 0.03$)
BEST (2015) [14]	Patients with multivessel coronary disease	CABG (442) vs PCI with everolimus-eluting stents (438)	Increased primary outcome (death, MI, target-vessel revascularization) with PCI at median 4.6 years follow-up: 15.3% vs 10.6% ($P = 0.04$) Similar stroke incidence: CABG 2.9% vs PCI 2.5% ($P = 0.72$)
STITCHES (2016) [15]	Patients with CAD and EF $\leq$35%	CABG plus medical therapy (610) vs medical therapy alone (602)	Increased incidence of death from any cause in medical therapy only at a median 9.8 years follow-up: 66.1% vs 58.9% ($P = 0.02$)
EXCEL (2019) [16]	Patients with left main disease of low or intermediate anatomical complexity	CABG (957) vs PCI (948)	No difference in primary outcome (death, stroke, MI) at 5 years: PCI 22.0% vs CABG 19.2% ($P = 0.13$) Increased all-cause death in PCI: 13.0% vs 9.9% (diff 3.1%, 95% CI 0.2–6.1) Similar stroke incidence: CABG 3.7% vs PCI 2.9% (diff −0.8, 95% CI −2.4 to 0.9) Increased subsequent revascularization in PCI: 16.9% vs 10.0% (diff 6.9%, 95% CI 3.7–10.0)

Table 11.1 (continued)

Trial	Population	Comparators	Findings
SYNTAXES (2019) [17]	Patients with three vessel and/or left main disease Subgroup analyses by three vessel disease, left main, diabetes, and SYNTAX tertiles	CABG (897) vs PCI (903) with first-generation paclitaxel-eluting stents	Similar overall 10-year survival: PCI HR 1.19 ($P = 0.066$) Increased 10-year survival in CABG patients with 3 vessel disease: PCI HR 1.42 (95% CI 1.11–1.81) No difference in left main, diabetes, or SYNTAX subgroup analyses
ISCHEMIA (2020) [18]	Patients with stable but moderate to severe CAD	Angiography and revascularization plus medical therapy versus medical therapy alone	Similar incidence of primary outcome (cardiovascular death, MI, hospitalization for unstable angina, heart failure, resuscitated cardiac arrest) at a median 3.3 years follow-up: invasive 16.4% vs conservative 18.2% (diff −1.8, 95% CI −4.7 to 1.0)
NOBLE (2020) [19]	Patients with left main disease	CABG (603) vs PCI (598)	Increased primary endpoint (MACCE, all-cause mortality, non-procedural MI, repeat revascularization, stroke) with PCI at median 4.9 years follow-up: PCI HR 1.58 (95% CI 1.24–2.01)
PRECOMBAT (2020) [20]	Patients with unprotected left main disease	CABG (300) vs PCI with sirolimus-eluting stents (300)	Similar incidence of primary outcome (any cause death, MI, stroke, ischemia-driven target-vessel revascularization): CABG 24.7% vs PCI 29.8% (PCI HR 1.25, 95% CI 0.93–1.69); importantly however, this study was underpowered
FAME 3 (2022) [21]	Patients with 3 vessel CAD	CABG (743) vs FFR-guided PCI with current generation zotarolimus-eluting stents (757)	Increased 1-year MACCE (any cause death, MI, stroke, repeat revascularization) with FFR-guided PCI: 10.6% vs 6.9% (HR 1.5, 95% CI 1.1–2.2)

CABG coronary artery bypass grafting, *CAD* coronary artery disease, *DES* drug eluting stent, *EF* ejection fraction, *HR* hazard ratio, *MACCE* major adverse cardiovascular and cerebrovascular events, *PCI* percutaneous coronary intervention, *PTCA* percutaneous transluminal coronary angioplasty, *SYNTAX* synergy between PCI with Taxus and Cardiac Surgery score

Conduit Options

- Left internal mammary artery (LIMA) is the preferred conduit for bypass to the left anterior descending artery (LAD). It has excellent 5-year and 10-year patency rates of 95% and 90%, respectively. The right internal mammary artery (RIMA) has similar graft patency rates. Internal mammary artery grafts have been demonstrated to prolong survival and reduce ischemic events. These benefits are derived from the biology of the IMA which has resistance to development of atherosclerosis due to a non-fenestrated internal elastic lamina, high intrinsic production of vasodilators, and resistance to vasoconstriction [5, 6].
- Radial artery is preferred over saphenous vein to bypass a significantly stenosed, non-LAD vessel based on studies showing better long-term patency (80–95% at 5 years) and improved outcomes at 10 years. The radial artery has a fenestrated internal elastic lamina, which makes it more vulnerable to atherosclerosis, and is less resistant to vasospasm than the IMA. It is important that the radial artery be used to bypass a significantly stenosed vessel as competitive flow from the native vessel can compromise the conduit patency [5, 6].
- Multiple (and total) arterial revascularization using bilateral internal mammary arteries (BIMA) and/or radial artery has been found to have improved outcomes including increased survival compared to single arterial revascularization. It should be considered for most patients with only a few exceptions (poorly controlled diabetes, obesity, poor ulnar artery compensation, or prior use for radial access catheterization) [7, 8].
- Greater saphenous vein is the most common venous conduit. Its advantages include ease of harvest and resistance to spasm; however, its disadvantages include a proclivity to intimal hyperplasia, thrombosis, and graft atherosclerosis. Ten-year patency is reported to be around 50%.
- Gastroepiploic artery is an alternative but uncommonly used arterial conduit in the United States.

Basic Steps of Coronary Artery Bypass Grafting (CABG) Using Cardiopulmonary Bypass (CPB) [9]

1. Anesthesia procedures: intubation, general anesthesia, arterial line, central line, Foley catheter with temperature probe, antibiotics
2. Positioning and preparation: supine with both arms tucked unless harvesting radial artery, antiseptic prep from chin to ankles (and arm if radial artery harvested)
3. Sternotomy
4. Conduit harvest: IMA, radial artery, saphenous vein (open vs endoscopic)
5. Pericardial well creation and general inspection of the heart and aorta

6. Heparinization: bolus before conduit harvest and prior to CPB, monitor activated clotting time (ACT) level (goal >480 s during CPB) throughout case
7. Aortic followed by venous cannulation: cannulate aorta with relative systemic hypotension (SBP 90–100 mmHg)
8. Dual lumen aortic root catheter placed in ascending aorta for antegrade cardioplegia and venting
9. Retrograde cardioplegia catheter placed in coronary sinus
10. Commencement of cardiopulmonary bypass
11. Allow mild hypothermia (32 °C) while on CPB
12. Evaluation of distal targets, ordering of anastomoses, and preparation of conduits
13. Aortic cross clamp applied: the "cross clamp time" is the period from clamp placement to removal during which the heart is ischemic and should be minimized as much as possible
14. Administration of antegrade cardioplegia: ensure left ventricular dilatation does not occur
15. Administration of retrograde cardioplegia with aortic root venting
16. Distal anastomoses order: inferior wall followed by distal lateral wall, proximal lateral wall, diagonal, and LAD
17. Systemic re-warming
18. Proximal anastomoses
19. De-airing maneuvers to remove air from grafts: Trendelenburg position, lung inflation, warm retrograde cardioplegia
20. Remove aortic cross clamp
21. Remove retrograde cardioplegia catheter
22. Place epicardial atrial and ventricular pacing wires
23. Wean from CPB
24. Administer protamine to reverse heparinization
25. Remove remaining cannulas
26. Hemostasis and chest tube placement
27. Sternal closure

On Pump Versus Off Pump Coronary Artery Bypass

- While "On Pump CABG" (ONCAB) is the gold standard technique for surgical coronary artery revascularization, off pump CABG (OPCAB) is performed without the use of cardiopulmonary bypass which offers some potential advantages including the avoidance of the inflammatory effects of the cardiopulmonary bypass circuit and the ability to perform bypass without the need for a cross clamp, which is ideal for patients with a porcelain aorta.
- Despite these advantages, OPCAB does not appear to be a better option than ONCAB for most patients. In fact, OPCAB has been associated with decreased survival in addition to a higher incidence of incomplete revascularization and

repeated revascularization [10]. However, this may not be the case for surgeons who routinely perform OPCAB and are able to achieve complete revascularization [10, 11].

References

1. Roth GA, Mensah GA, Johnson CO, et al. Global burden of cardiovascular diseases and risk factors, 1990–2019. J Am Coll Cardiol. 2020;76(25):2982–3021. https://doi.org/10.1016/j.jacc.2020.11.010.
2. Yahagi K, Kolodgie FD, Otsuka F, et al. Pathophysiology of native coronary, vein graft, and in-stent atherosclerosis. Nat Rev Cardiol. 2016;13(2):79–98. https://doi.org/10.1038/nrcardio.2015.164.
3. Suarez-Pierre A, Velez A, Lawton JS. Primary coronary artery bypass surgery. In: Johns Hopkins textbook of cardiothoracic surgery. 3rd ed. McGraw Hill/Medical. 2024.
4. Fihn SD, Gardin JM, Abrams J, et al. 2012 ACCF/AHA/ACP/AATS/PCNA/SCAI/STS guideline for the diagnosis and management of patients with stable ischemic heart disease. J Am Coll Cardiol. 2012;60(24):e44–e164. https://doi.org/10.1016/j.jacc.2012.07.013.
5. Brescia A, Louis C, editors. TSRA review of cardiothoracic surgery. 3rd ed. Independently Published; 2021.
6. Lawton JS, Tamis-Holland JE, Bangalore S, et al. 2021 ACC/AHA/SCAI guideline for coronary artery revascularization: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2022;145(3):e18–e114. https://doi.org/10.1161/CIR.0000000000001038.
7. Taggart DP, Gaudino MF, Gerry S, et al. Effect of total arterial grafting in the arterial revascularization trial. J Thorac Cardiovasc Surg. 2022;163(3):1002–1009.e6. https://doi.org/10.1016/j.jtcvs.2020.03.013.
8. Lawton JS. Why not give all patients the opportunity to have the benefits of BITA grafting? J Am Coll Cardiol. 2021;77(1):27–8. https://doi.org/10.1016/j.jacc.2020.11.015.
9. Lawton JS. Chapter 8: Coronary artery bypass grafting (on pump arrested). In: Cardiac surgery: technique and practice. 2024.
10. Gaudino M, Benedetto U, Bakaeen F, et al. Off- versus on-pump coronary surgery and the effect of follow-up length and surgeons' experience: a meta-analysis. J Am Heart Assoc. 2018;7(21):e010034. https://doi.org/10.1161/JAHA.118.010034.
11. Lawton JS. Off-pump coronary artery bypass grafting: do it often, do it well, and do it completely—or don't do it at all. J Thorac Cardiovasc Surg. 2016;152(5):1331–2. https://doi.org/10.1016/j.jtcvs.2016.07.009.
12. BARI Investigators. The final 10-year follow-up results from the BARI randomized trial. J Am Coll Cardiol. 2007;49(15):1600–6. https://doi.org/10.1016/j.jacc.2006.11.048.
13. Farkouh ME, Domanski M, Sleeper LA, et al. Strategies for multivessel revascularization in patients with diabetes. N Engl J Med. 2012;367(25):2375–84. https://doi.org/10.1056/NEJMoa1211585.
14. Park SJ, Ahn JM, Kim YH, et al. Trial of everolimus-eluting stents or bypass surgery for coronary disease. N Engl J Med. 2015;372(13):1204–12. https://doi.org/10.1056/NEJMoa1415447.
15. Velazquez EJ, Lee KL, Jones RH, et al. Coronary-artery bypass surgery in patients with ischemic cardiomyopathy. N Engl J Med. 2016;374(16):1511–20. https://doi.org/10.1056/NEJMoa1602001.
16. Stone GW, Kappetein AP, Sabik JF, et al. Five-year outcomes after PCI or CABG for left main coronary disease. N Engl J Med. 2019;381(19):1820–30. https://doi.org/10.1056/NEJMoa1909406.

17. Thuijs DJFM, Kappetein AP, Serruys PW, et al. Percutaneous coronary intervention versus coronary artery bypass grafting in patients with three-vessel or left main coronary artery disease: 10-year follow-up of the multicentre randomised controlled SYNTAX trial. Lancet. 2019;394(10206):1325–34. https://doi.org/10.1016/S0140-6736(19)31997-X.
18. Maron DJ, Hochman JS, Reynolds HR, et al. Initial invasive or conservative strategy for stable coronary disease. N Engl J Med. 2020;382(15):1395–407. https://doi.org/10.1056/NEJMoa1915922.
19. Holm NR, Mäkikallio T, Lindsay MM, et al. Percutaneous coronary angioplasty versus coronary artery bypass grafting in the treatment of unprotected left main stenosis: updated 5-year outcomes from the randomised, non-inferiority NOBLE trial. Lancet. 2020;395(10219):191–9. https://doi.org/10.1016/S0140-6736(19)32972-1.
20. Park DW, Ahn JM, Park H, et al. Ten-year outcomes after drug-eluting stents versus coronary artery bypass grafting for left Main coronary disease. Circulation. 2020;141(18):1437–46. https://doi.org/10.1161/CIRCULATIONAHA.120.046039.
21. Fearon WF, Zimmermann FM, De Bruyne B, et al. Fractional flow reserve–guided PCI as compared with coronary bypass surgery. N Engl J Med. 2022;386(2):128–37. https://doi.org/10.1056/NEJMoa2112299.

Chapter 12
Mechanical Complications of Myocardial Infarction

Travis D. Hull and George Tolis Jr

Introduction

- Mechanical complications of acute myocardial infarction (MCAMI) include acute mitral regurgitation (AMR) due to papillary muscle rupture, ventricular septal defect (VSD), free wall rupture (FWR), and left ventricle (LV) aneurysm and pseudoaneurysm (Table 12.1).
- Risk factors for these complications in patients who present with AMI include late hospital presentation, large infarcts, poor tissue reperfusion after percutaneous intervention (PCI) [1], and patient characteristics including older age, female sex, first AMI, or history of heart failure or chronic kidney disease [2, 3].
- The incidence of these mechanical complications is declining largely due to improving reperfusion techniques including the systematic adoption of early percutaneous revascularization strategies. However, the mortality associated with them is high and directly proportional to the amount of time it takes for them to be recognized and treated [4].
- Severely decompensated patients can temporarily be stabilized by supporting end-organ perfusion with mechanical support including an intra-aortic balloon pump (IABP) or extracorporeal membrane oxygenation (ECMO), but surgical repair, usually in the emergent setting, is required to prevent patient death.

T. D. Hull
Division of Cardiothoracic Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: thull1@partners.org

G. Tolis Jr (✉)
Division of Cardiothoracic Surgery, Brigham and Women's Hospital, Boston, MA, USA
e-mail: gtolis@bwh.harvard.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_12

Table 12.1 Mechanical complications of acute myocardial infarction

MCAMI	Incidence/ mortality	Presentation	Diagnosis	Medical management	Surgical management
Acute MR secondary to PM rupture	0.05–0.26% 10–40%	3–5 days after AMI Cardiogenic shock, pulmonary edema	TTE demonstrating severe MR TEE more sensitive for partial PMR	Reduction of LV afterload with pharmacologic agents (nitroglycerin) or mechanical support (IABP)	Emergent (within 24 h) mitral valve replacement
Ventricular septal defect	0.3% 80% with medical management, 40% with surgical management	3–5 days after AMI Varied presentation from murmur to shock	Echocardiography is diagnostic RHC shows step up in oxygenation between the RA and PA	Afterload reduction with IABP ± tMCS to decompress the LV	Emergent to urgent repair as dictated by degree of cardiogenic shock
Free wall rupture	Most common MCAMI, unknown incidence, usually SCD >35% mortality with surgery	3–5 days after AMI Cardiogenic shock with signs of tamponade	High clinical suspicion, signs and symptoms of cardiac tamponade, bedside echocardiography	Not applicable. ECMO can be utilized in the event of cardiovascular collapse as a bridge to the operating room	Immediate emergent surgical repair

Abbreviations: *MCAMI* mechanical complication of AMI, *AMI* acute myocardial infarction, *MR* mitral regurgitation, *TTE* transthoracic echocardiography, *TEE* transesophageal echocardiography, *PMR* papillary muscle rupture, *IABP* intra-aortic balloon pump, *RHC* right heart catheterization, *RA* right atrium, *PA* pulmonary artery, *tMCS* temporary mechanical circulatory support, *SCD* sudden cardiac death, *ECMO* extracorporeal membrane oxygenation

Evaluation

Presentation

- Most patients with MCAMI present in some degree of shock with evidence of end-organ malperfusion from deranged pump function (i.e., cardiogenic shock).
- The differential diagnosis in MCAMI is broad, as it includes other causes of shock including hemorrhagic, hypovolemic, and septic shock as well as a broad array of etiologies for cardiogenic shock including acute pulmonary embolism, aortic dissection, valvular pathology, tamponade, stress-induced cardiomyopathy, or AMI without a mechanical complication.
- A focused history and physical exam should be performed to elucidate risk factors for MCAMI including signs or symptoms of recent or ongoing MI such as chest pain. A personal or family history of coronary artery disease and peripheral vascular disease are additional important clues. Findings on physical exam include a new murmur, pulse irregularities, JVD, or systemic signs of malperfusion.

Diagnostic Studies

- The initial study of choice is a bedside transthoracic echocardiogram (TTE) to assess the pericardial space, pump function and geometry, ventricular loading conditions, valvular pathology, outflow tract obstruction, and regional wall motion abnormalities.
- Additional studies such as CT scan with angiography of the chest, abdomen, and pelvis can be useful, particularly in ruling out extra-cardiac explanations for a presentation of shock.

Initial Management

- If MCAMI is not recognized in a timely manner, diminished cardiac output leads to multi-system organ failure and death, which can only temporarily be curtailed by the institution of inotropes, IABP, and temporary mechanical circulatory support.
- Therefore, the role of the cardiac surgeon in the multi-disciplinary team is imperative, particularly in expediently diagnosing and managing patients with MCAMI.
- Definitive treatment of MCAMI is surgical and includes correction of the structural issue that has occurred secondary to AMI and has resulted in a hemodynamically significant mechanical complication.
- Concomitant surgical revascularization by coronary artery bypass is often of utility but should be assessed on a case-by-case basis and guided by pre-operative coronary angiogram.

Acute Mitral Regurgitation

Pathogenesis

- The mitral valve (MV) is supported by the anterolateral (AL) and posteromedial (PM) papillary muscles.
  - The AL papillary muscle has a dual blood supply from the left anterior descending and left circumflex coronary artery.
  - The PM papillary muscle has a single blood supply from the right coronary artery or left circumflex artery, depending on dominance.
  - Therefore, PM papillary muscle rupture (PMR) is significantly more common and is associated with inferior (RCA) or lateral (left circumflex) infarction.

Diagnosis

- Patients typically present 3–5 days after AMI with evidence of acute pulmonary edema that rapidly progresses to cardiogenic shock.

- On exam, a systolic murmur may be audible at the left lower sternal border radiating to the axilla.
- Diagnosis is made with echocardiography, which shows severe, often eccentric MR.
- Although TTE is readily available and non-invasive, a partial PMR can be missed with this modality, and therefore, sensitivity is improved by transesophageal echocardiography (TEE).

Management

- Medical management is largely temporizing to improve hemodynamics and tissue perfusion due to cardiogenic shock.
- Mechanical circulatory support is used in up to 70% of cases [5, 6].
  - IABP decreases afterload, and thus, the regurgitant volume, in addition to augmenting cardiac output [7].
- Acute MR secondary to PMR is a surgical emergency.
  - Typically, chordal-sparing MV replacement is performed, although MV repair may be an acceptable alternative in some hemodynamically stable patients with a partial PMR [8, 9].
  - Bioprosthetic or mechanical valves can be utilized, with the latter being associated with better long-term, symptom free survival in younger patients [10].
- Revascularization with coronary artery bypass grafting (CABG) should be considered in patients with PMR, although a clear benefit for concomitant CABG targeting the inferior left ventricular wall at the time of MVR has not been clearly demonstrated.
- Only 38–58% of patients with acute severe MR from PMR are offered surgery, likely owning to a patient population with advanced age and significant comorbidities who deteriorate rapidly pre-operatively. In patients who are not surgical candidates due to prohibitively high risk, percutaneous edge-to-edge MV repair with MitraClip can be considered [11].

Post-infarction Ventricular Septal Defect

Pathogenesis

- MCAMI VSDs are characterized as anterior or posterior based on the location of the perforation on the intraventricular septum. This correlates with which coronary artery is occluded and is important in planning the surgical approach.

  - Infarcts in the LAD territory are more common and generally result in anterior and apical VSD. Most occur in the distal half of the septum.
  - Posterior VSDs are caused by inferior infarcts and are often accompanied by RV dysfunction, particularly when the proximal RCA is occluded, which is often accompanied by MR from ischemic tethering of the MV and more complex VSDs [12]. Most occur in the proximal half of the septum.

Diagnosis

- Patients typically present 3–5 days after a transmural MI with signs of heart failure ranging from dyspnea from pulmonary venous congestion to decompensated cardiogenic shock due to left-to-right shunting and overload of the pulmonary circulation.
- A pansystolic murmur is heard at the left lower sternal boarder.
- Echocardiography is the gold standard for diagnosis and can characterize the size and location of the VSD to aid in surgical planning.
- Right heart catheterization can aid in diagnosis by showing a step up in oxygenation between the right atrium and pulmonary artery, but is not necessary.
- Coronary angiography is helpful during initial ischemic presentation as a guide to planning concomitant revascularization.

Management

- Surgical intervention is the standard of care as medical management alone is associated with 80% mortality at 30 days [13]. Hemodynamically stable patients should undergo coronary angiography followed by urgent surgery.
  - Despite operative intervention, mortality approaches 40%.
- In hemodynamically unstable patients, pre-operative temporizing measures include IABP and VA-ECMO.
  - An IABP affords afterload reduction to decrease left-to-right shunting and is utilized in 65–80% of patients [14, 15].
  - VA-ECMO may be utilized in patients with multi-organ failure to allow for end-organ recovery before definitive surgical intervention.
- In patients with multi-vessel disease, the first step in surgical intervention includes coronary revascularization utilizing saphenous vein for the bypass graft while on cardiopulmonary bypass. The coronary artery supplying the ruptured septum should not be bypassed unless there is an apical VSD due to LAD occlusion proximal to the first septal perforator.

Surgical VSD Repair

- Anterior VSDs are repaired with a ventriculotomy parallel to the LAD.
- Posterior VSD are repaired with a ventriculotomy parallel to the posterior descending artery in the infarcted posterior wall of the LV (Fig. 12.1).
- The VSD is repaired with a patch constructed from pericardium or synthetic material using pledgetted mattress or running sutures placed in non-infarcted myocardium.
- Specific techniques for patch repair included primary repair via the Daggett repair or infarct exclusion via the David exclusion technique. Both techniques utilize a pericardial patch. In the Daggett repair, this patch bridges the VSD by fixation to the distal end of the ruptured IVS and free wall of the LV with interrupted, pledgetted mattress sutures. In the David repair, the patch is utilized to exclude the left ventricular side of the septum from the mitral annulus to the anterolateral wall of the LV. The ventriculotomy is then closed in two layers utilizing a buttress of pericardium or felt. In recent years, the David technique has largely replaced the Daggett technique due to its relative technical simplicity and proven reproducibility.
- True apical VSDs can be repaired by amputating the apex and closing it primarily. Some authors have reported successful case studies with this technique without utilizing cardiopulmonary bypass.

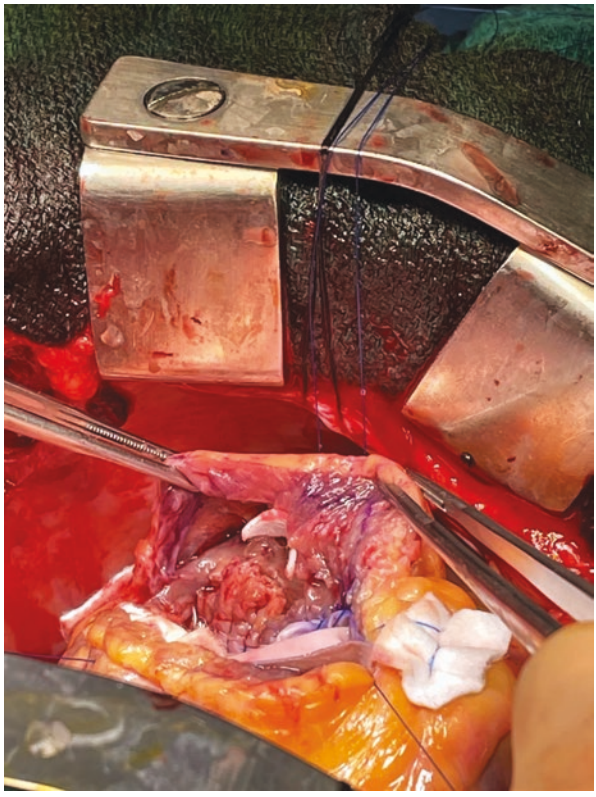


Fig. 12.1 Posterior post-infarction VSD. The VSD has been exposed by a ventriculotomy made parallel to the posterior descending artery (PDA), looking into the left ventricle, through the large VSD and into the right ventricle. The apex of the heart is retracted cephalad and marked by the pledget in the bottom right of the photo

Free Wall Rupture

Pathogenesis

- This MCAMI is rapidly fatal, and not surprisingly, its true incidence is unknown because its most common presentation is out-of-hospital sudden cardiac death to secondary tamponade from massive hemopericardium.
- The major risk factor is delayed reperfusion therapy, and therefore, it should be considered in any patient who suffers sudden hemodynamic collapse after presenting with AMI.

Diagnosis

- Clinical clues include signs of cardiac tamponade including jugular venous distension, muffled heart sounds, and pulsus paradoxus. The rapid progression of this condition to death precludes extensive diagnostic testing before emergent surgical intervention. Echocardiography confirms the diagnosis and may identify cases in which frank rupture has yet to occur, but instead a bloody pericardial effusion is the result of oozing from a transmural area of myocardial infarction and necrosis.

Management

- Emergent surgical repair is indicated in these patients with pre-operative cannulation for ECMO serving as a brief temporizing measure in select patients who have suffered from arrest after cardiovascular collapse.
- The goal of surgical intervention is to relieve the tamponade, repair the defect, and preserve healthy tissue to restore adequate cardiac function.
- Repair techniques continue to evolve and are largely predicated based on infarct location and size.
- Operative approaches include infarctectomy with patch repair, primary patch repair, or a sutureless repair with a patch and biologic glue.
- The sutureless repair option is best suited for cases with bloody oozing from an area of transmural infarction without frank rupture. It is carried out by securing a collagen sheet to the hematoma surrounding the infarct and re-enforcing this with layers of Gelfoam secured with biologic glue. Close follow-up is mandatory with this option as aneurysm and recurrence of the rupture are possible [16].

Left Ventricular Pseudoaneurysm and Aneurysm

LV Pseudoaneurysm

- LV pseudoaneurysms are caused by LV free wall rupture as a consequence of AMI, but their presentation is often sub-acute because the perforation is contained by the pericardium due to adhesions. They are often seen in patients who have undergone prior median sternotomy and have developed adhesions between the ventricular epicardium and the surrounding pericardium. They are most commonly asymptomatic and incidentally discovered.
- Symptomatic patients can present with chest pain, congestive heart failure, or complications specific to their pseudoaneurysm including systemic embolization and arrhythmia [17].
- Diagnosis requires multimodal imaging and a high index of suspicion. Ventriculogram during coronary angiography will show an area of discontinuity, most often in the inferior or lateral cardiac wall with a narrow neck. The diagnosis can be supported with echocardiography and cardiac CT or MRI.
- Incidentally discovered pseudoaneurysms in asymptomatic patients with a history of only remote AMI can be repaired in an urgent manner.
- Symptomatic LV pseudoaneurysms are treated as surgical emergencies due to a theoretical progressive risk of deadly rupture [18].
- Depending on the size of the neck, a buttressed primary repair with pledgetted sutures has been performed successfully, while larger defects with unhealthy myocardial edges can be repaired with a patch.

LV Aneurysm

- Left ventricle aneurysms are comprised of a thin wall of fibrotic, scarred myocardium generally located on the anterior or apical LV most commonly from LAD occlusion.
- They are a delayed MCAMI that causes an increase in LV end-diastolic pressure and increases the risk for thrombus formation, arrhythmia, and heart failure.
- They are managed non-operatively in most patients.
- However, patients with severe refractory ventricular arrhythmias, heart failure despite maximum medical management, or recurrent thromboembolism despite anticoagulation can be considered for aneurysmectomy, especially if they are undergoing another cardiac surgical procedure such as CABG. The goal of this operation is to restore ventricular geometry via aneurysm plication, excision, or ventricular reconstruction with a patch.

References

1. Peterson ED, Shah BR, Parsons L, Pollack CV Jr, French WJ, Canto JG, et al. Trends in quality of care for patients with acute myocardial infarction in the National Registry of Myocardial Infarction from 1990 to 2006. Am Heart J. 2008;156(6):1045–55.
2. Rogers WJ, Frederick PD, Stoehr E, Canto JG, Ornato JP, Gibson CM, et al. Trends in presenting characteristics and hospital mortality among patients with ST elevation and non-ST elevation myocardial infarction in the National Registry of Myocardial Infarction from 1990 to 2006. Am Heart J. 2008;156(6):1026–34.
3. Puerto E, Viana-Tejedor A, Martinez-Selles M, Dominguez-Perez L, Moreno G, Martin-Asenjo R, et al. Temporal trends in mechanical complications of acute myocardial infarction in the elderly. J Am Coll Cardiol. 2018;72(9):959–66.
4. Damluji AA, Forman DE, van Diepen S, Alexander KP, Page RL 2nd, Hummel SL, et al. Older adults in the cardiac intensive care unit: factoring geriatric syndromes in the management, prognosis, and process of care: a scientific statement from the American Heart Association. Circulation. 2020;141(2):e6–e32.
5. DiVita M, Visveswaran GK, Makam K, Naji P, Cohen M, Kapoor S, et al. Emergent TandemHeart-ECMO for acute severe mitral regurgitation with cardiogenic shock and hypoxaemia: a case series. Eur Heart J Case Rep. 2020;4(1):1–6.
6. Bhardwaj B, Sidhu G, Balla S, Kumar V, Kumar A, Aggarwal K, et al. Outcomes and hospital utilization in patients with papillary muscle rupture associated with acute myocardial infarction. Am J Cardiol. 2020;125(7):1020–5.
7. Tehrani BN, Truesdell AG, Psotka MA, Rosner C, Singh R, Sinha SS, et al. A standardized and comprehensive approach to the management of cardiogenic shock. JACC Heart Fail. 2020;8(11):879–91.
8. Lee SK, Heo W, Min HK, Kang DK, Jun HJ, Hwang YH. A new surgical repair technique for ischemic total papillary muscle rupture. Ann Thorac Surg. 2015;100(5):1891–3.
9. Kilic A, Sultan I, Chu D, Wang Y, Gleason TG. Mitral valve surgery for papillary muscle rupture: outcomes in 1342 patients from the Society of Thoracic Surgeons database. Ann Thorac Surg. 2020;110(6):1975–81.
10. Kaneko T, Aranki S, Javed Q, McGurk S, Shekar P, Davidson M, et al. Mechanical versus bioprosthetic mitral valve replacement in patients <65 years old. J Thorac Cardiovasc Surg. 2014;147(1):117–26.
11. Mack MJ, Abraham WT, Lindenfeld J, Bolling SF, Feldman TE, Grayburn PA, et al. Cardiovascular outcomes assessment of the MitraClip in patients with heart failure and secondary mitral regurgitation: design and rationale of the COAPT trial. Am Heart J. 2018; 205:1–11.
12. Crenshaw BS, Granger CB, Birnbaum Y, Pieper KS, Morris DC, Kleiman NS, et al. Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. GUSTO-I (Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries) Trial Investigators. Circulation. 2000;101(1):27–32.
13. Jones BM, Kapadia SR, Smedira NG, Robich M, Tuzcu EM, Menon V, et al. Ventricular septal rupture complicating acute myocardial infarction: a contemporary review. Eur Heart J. 2014;35(31):2060–8.
14. O'Gara PT, Kushner FG, Ascheim DD, Casey DE Jr, Chung MK, de Lemos JA, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation. 2013;127(4):e362–425.

15. Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, et al. [2017 ESC guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation]. Kardiol Pol. 2018;76(2):229–313.
16. Aoyagi S, Tayama K, Otsuka H, Okazaki T, Shintani Y, Wada K, et al. Sutureless repair for left ventricular free wall rupture after acute myocardial infarction. J Card Surg. 2014;29(2):178–80.
17. Frances C, Romero A, Grady D. Left ventricular pseudoaneurysm. J Am Coll Cardiol. 1998;32(3):557–61.
18. Atik FA, Navia JL, Vega PR, Gonzalez-Stawinski GV, Alster JM, Gillinov AM, et al. Surgical treatment of postinfarction left ventricular pseudoaneurysm. Ann Thorac Surg. 2007;83(2):526–31.

Chapter 13
Aortic Valve Repair and Replacement

Nicholas Oh and Douglas Johnston

Overview

- Various valve pathologies contribute to hemodynamically significant aortic stenosis or aortic regurgitation.
- When surgical indications are met, the aortic valve disease can be addressed with aortic valve replacement or repair. However, the choice of valve replacement or type of repair can vary depending on valve anatomy and clinical situation.
- Small aortic roots, prosthetic valve-patient mismatch, and paravalvular regurgitation are additional considerations when addressing aortic valve pathology.

Basic Introduction to Valve Pathologies

Aortic Stenosis

- Aortic Stenosis (AS) is the most prevalent valvular heart disease in adults in developed countries. AS is present in 5% of the population by the age 65 with increasing prevalence with age [1, 2].
- Acquired AS is usually caused by degenerative calcification of the aortic valve. Calcium deposits involve the aortic valve leaflets and may extend into the aortic annulus.
- Bicuspid aortic valves represent the most common form of congenital AS, presenting in 1–2% of the general population. Gradual calcification of the bicuspid AV results in significant stenosis [3].

N. Oh (✉) · D. Johnston
Department of Thoracic and Cardiovascular Surgery, Cleveland Clinic, Cleveland, OH, USA
e-mail: OHN2@ccf.org; JOHNSTD3@ccf.org

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_13

- Rheumatic aortic stenosis is the least common form of AS in adults in the developed world, though prevalence is higher in developing countries. Rheumatic aortic valves are typically thickened and fibrotic with rolled edges and associated cusp fusion.

Aortic Regurgitation

- The pathophysiology of aortic regurgitation (AR) can be differentiated by onset and duration of disease.
- Acute AR can occur in the setting of endocarditis, dissection, or trauma. Hemodynamically significant acute AR usually requires surgery.
- Chronic AR occurs due to distortion of valve leaflets or dilation of the aortic root.
- Distortion of the valve leaflets and improper coaptation can be caused by aortic leaflet calcific degeneration, myxomatous degeneration, infective endocarditis, rheumatic disease, or bicuspid aortic valve.
- Dilation of the aortic root can also disrupt the integrity of the aortic valve. Aortic dissection, trauma, connective tissue disease can dilate the aortic root and annulus, leading to improper coaptation.
- A mixed aortic regurgitation and aortic stenosis are often seen in combination due to calcification or rheumatic disease.

Indications for Repair vs Replacement

- In AS, surgical intervention is considered when severe AS is diagnosed (mean AV gradient >40 mmHg, peak velocity >4 m/s, AVA <1.0 cm^2 dimensionless index <0.25) (Fig. 13.1a).

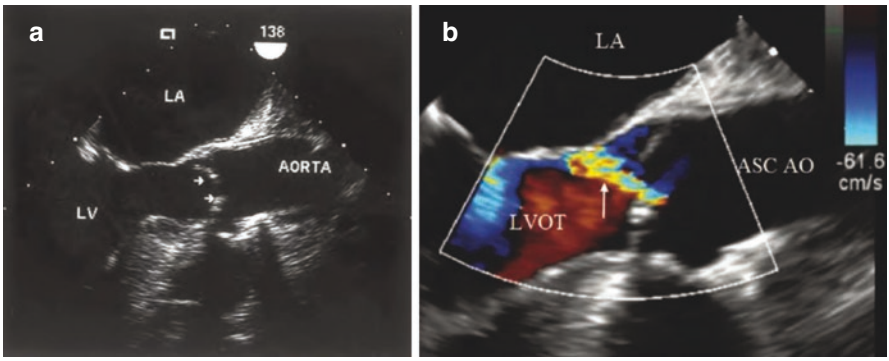


Fig. 13.1 Echocardiogram demonstrates leaflet doming (arrows) indicating severe aortic stenosis (**a**). Color flow doppler shows an eccentric aortic regurgitant jet directed anteriorly (**b**)

- In AR, surgical intervention is considered when severe AR is diagnosed (jet width >65% of LVOT, vena contract >0.6 cm, holodiastolic flow reversal in proximal abdominal aorta, regurgitant volume >60 mL/beat, regurgitant fraction >50%, effective regurgitant orifice >0.3 cm^2, angiographic grade 3+ or 4+) (Fig. 13.1b).
- The ACC/AHA Guidelines for the Management of Valvular Heart Disease provides recommendations for surgical timing based on the severity of disease, presence of symptoms, left ventricular morphology and function, and surgical candidacy. Aortic valve surgery is recommended in the presence for severe disease in symptomatic patients, and in asymptomatic patients with reduced ejection fraction, positive stress test, or other changes in cardiac morphology [4].
- When surgical indications are met, most patients undergo an aortic valve replacement. However, aortic valve repair is an option in selected patients with AR at experienced centers. Successful repair requires careful consideration of the size and quality of the aortic cusps, size of the aortic root, and possible reimplantation.

Repair Techniques

- There are many well-described techniques for aortic valve repair. Each technique must address the underlying pathology and ensure function of the anatomic components of the aortic valve: commissures, leaflets, annulus, sinotubular junction, and sinuses.
- Aortic valve cusp perforation typically occurs in the setting of infective endocarditis or iatrogenic injury. Small perforations can be repaired using a patch of fresh or glutaraldehyde-fixed autologous pericardium.
- Aortic cusp prolapse occurs when free margin of the leaflet is elongated. This can be repaired by plication or suspension of the free margin, or with a commissuroplasty (Fig. 13.2a).
- Dilation of the sinotubular junction from aortic root or ascending aortic aneurysms can lead to increased stress along the free margin of the cusp, causing thinning and stress fenestrations. Small fenestrations can be addressed using a simple stitch.
- The normal valve movement may be restricted by calcification, infective endocarditis, or fibrotic tissue. Aortic cusp restriction can be addressed by removal of calcium, valve extension or extended resection and reconstruction.
- Bicuspid aortic valves can also be repaired when anatomy is favorable. General principles include ensuring good coaptation by creating similar free margin lengths of the bicuspid leaflets and suspension of valve commissures.

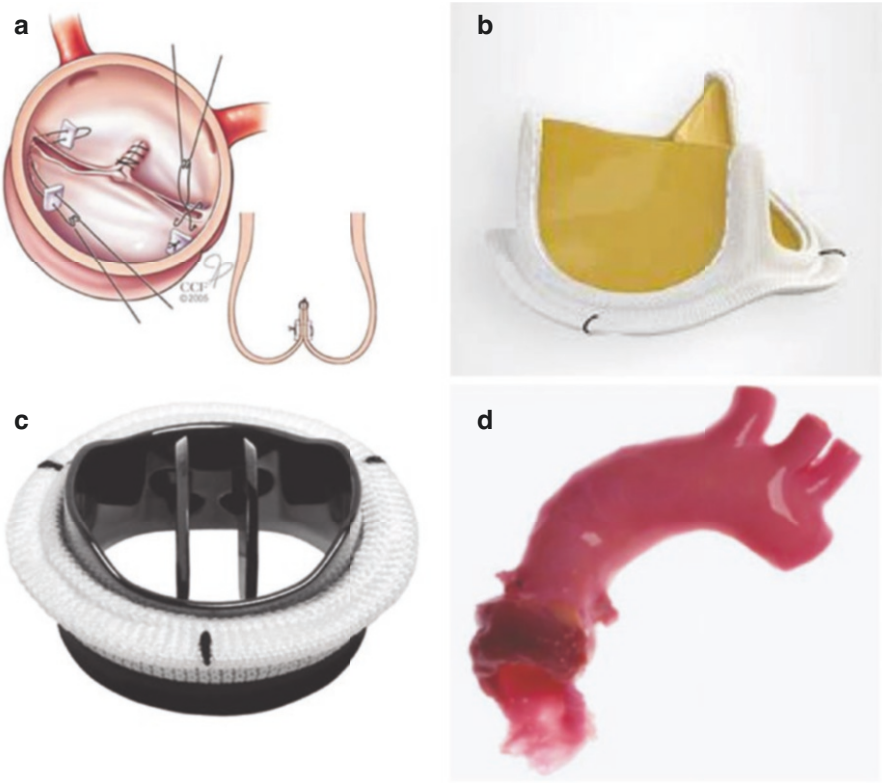


Fig. 13.2 Aortic valve repair of a bicuspid aortic valve with R-L fusion (**a**), bioprosthetic aortic valve (**b**), mechanical aortic valve (**c**), aortic homograft (**d**)

Replacement Techniques

Stented Bioprosthetic Valves

- Stented bioprosthetic valves are constructed using porcine valves or bovine pericardium, mounted on a plastic or metal frame (Fig. 13.2b). These valves are pretreated to prevent extracellular matrix buildup and/or calcium deposition.
- Because of the relative ease of implantation relative to other biological valves, stented valves represent the majority of biological aortic valve replacements.
- Bioprosthetic valves do not require anticoagulation. However, leaflets degrade slowly over time, resulting structural valve deterioration (SVD) and eventual reoperation.
- The advantages and disadvantages of valve type require careful consideration based on patient age, anatomy, risk profile, and patient preference.

Mechanical Valves

- Mechanical valves are constructed from pyrolytic carbon, using two hinged leaflets and have demonstrated very low failure rates related to the valve mechanism (Fig. 13.2c).
- All current mechanical valves require anticoagulation with warfarin. Reoperation for mechanical valves can occur due to infection, formation of thrombus on the valve, or pannus (scar tissue) ingrowth which interferes with leaflet function.
- Reoperation rates for mechanical valves are lower than that for bioprosthetic valves, however rates of hemorrhage and stroke are higher.

Stentless Valves

- Intact preserved porcine aortic roots or cryopreserved aortic homografts constitute stentless valve options (Fig. 13.2d).
- These provide several advantages including excellent hemodynamic profiles, no anticoagulation requirement, and lower risk for prosthetic valve infection.
- The use of stentless valves may help in avoiding patient prosthesis mismatch in the setting of small aortic root.
- Implantation of stentless valves is more complex than stented valves, often requiring reattachment of the coronary arteries as a "full root" replacement.
- The use of homografts is indicated in cases of active aortic valve endocarditis particularly with a root abscess, prosthetic valve infection, or fistula formation.

Ross Procedure

- The Ross procedure uses the autologous pulmonary valve to replace the native aortic valve, and a homograft to replace the pulmonary valve.
- The pulmonary autograft shares the hemodynamic advantages and antithrombotic features of a homograft but has the additional benefit of a fully viable autologous tissue.
- However, the Ross procedure is a technically complex surgery that should be performed in experienced centers. Long-term risks include aortic root dilation, leading to aortic regurgitation and pulmonary homograft dysfunction.
- The Ross procedure is of particular benefit in younger patients with aortic valve pathology, especially in the setting of small aortic root.

Ozaki Procedure

- The Ozaki procedure is a novel technique that uses fixed autologous pericardium to achieve aortic valve neocuspidization.

- This technique requires excision of diseased cusps from the native valve, and shaping new cusps from autologous pericardium, and suturing them into neo-cusps. Studies have demonstrated favorable mid-term outcomes; however, further studies are required to evaluate long-term durability [5].

Prosthesis Selection

- The ACC/AHA Guidelines suggest the use of mechanical valves in those <50 years old, and bioprosthetic valves in >65 years old. For patients between the ages of 50 and 65 years old, it is reasonable to use either valve. Homografts are often considered in infective endocarditis or small aortic roots. Autografts such as the Ross or the Ozaki procedure should be considered on an individual basis with a multi-disciplinary discussion [4].

Special Circumstances

Small Aortic Root

- Small aortic roots pose hemodynamic concerns because of the risk for prosthetic valve-patient mismatch (PPM).
- PPM is used to describe a small aortic valve with a large body surface area, an absolute small valve size, excessive transvalvular gradient post-implantation, increased transvalvular gradient with exercise, or a small indexed effective orifice area.
- The residual stenosis from PPM is thought to hinder reverse remodeling of the left ventricle, may result in limited symptom improvement, and in some cases worse long-term survival.
- Aortic root enlargement techniques can reduce the incidence of PPM by enlarging the aortic annulus to accommodate a larger valve.
- Though the data on PPM are conflicting, it is thought to be associated with adverse early and long-term outcomes, particularly in younger patients.

Paravalvular Regurgitation

- Paravalvular regurgitation occurs when gaps are present between implanted aortic valve and the annulus, leaving portions of the prosthesis unopposed.
- Etiologies include technical error, incomplete decalcification of the anulus, connective tissue disorder, and infective endocarditis.

- Sufficiently large paravalvular regurgitation can cause significant hemolysis or heart failure from aortic regurgitation.
- Clinically significant or symptomatic paravalvular regurgitation typically require surgical intervention, although some cases may be amenable to transcatheter occlusion techniques.

References

1. D'Arcy JL, Coffey S, Loudon MA, et al. Large-scale community echocardiographic screening reveals a major burden of undiagnosed valvular heart disease in older people: the OxVALVE population cohort study. Eur Heart J. 2016;37(47):3515–3522a. https://doi.org/10.1093/eurheartj/ehw229.
2. Brzezinski A, Koprivanac M, Gillinov AM, Mihaljevic T. Pathophysiology of aortic valve disease. In: Cardiac surgery in the adult. 5th ed. McGraw Hill; 2018. p. 633–48.
3. Ward C. Clinical significance of the bicuspid aortic valve. Heart. 2000;83(1):81–5. https://doi.org/10.1136/heart.83.1.81.
4. Otto CM, Nishimura RA, Bonow RO, et al. ACC/AHA guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2021;143:e72. https://doi.org/10.1161/CIR.0000000000000923.
5. Ozaki S, Kawase I, Yamashita H, et al. A total of 404 cases of aortic valve reconstruction with glutaraldehyde-treated autologous pericardium. J Thorac Cardiovasc Surg. 2014;147(1):301–6. https://doi.org/10.1016/j.jtcvs.2012.11.012.

Chapter 14
Mitral Valve Repair and Replacement

Gregory Leya and Serguei Melnitchouk

Overview [1, 2]

- Mitral valve (MV) surgery has advanced dramatically since the first MV repair, a transventricular commissurotomy, was performed by Dr. Elliot Cutler at the Peter Bent Brigham Hospital in 1923 on a 12-year-old girl critically ill from rheumatic mitral stenosis.
- Mitral pathology can be broadly categorized as either regurgitation, further subdivided into primary or secondary, or stenosis.
- Proper management of mitral regurgitation (MR) or stenosis depends on understanding the etiology of the disease, the architectural distortions involved in the ongoing disease process, and the patient's symptomology.

Anatomy [3–7]

- The MV apparatus is composed of the saddle-shaped mitral annulus, the larger anterior and smaller posterior leaflets, the chordae tendinae which attach the leaflets to the anterolateral and posteromedial papillary muscles, and the left ventricle (subvalvular apparatus) (Fig. 14.1).

G. Leya
Department of Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: gleya@mgb.org

S. Melnitchouk (✉)
Division of Cardiac Surgery, Massachusetts General Hospital, Boston, MA, USA

Harvard Medical School, Boston, MA, USA
e-mail: SMELNITCHOUK@MGH.HARVARD.EDU

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_14

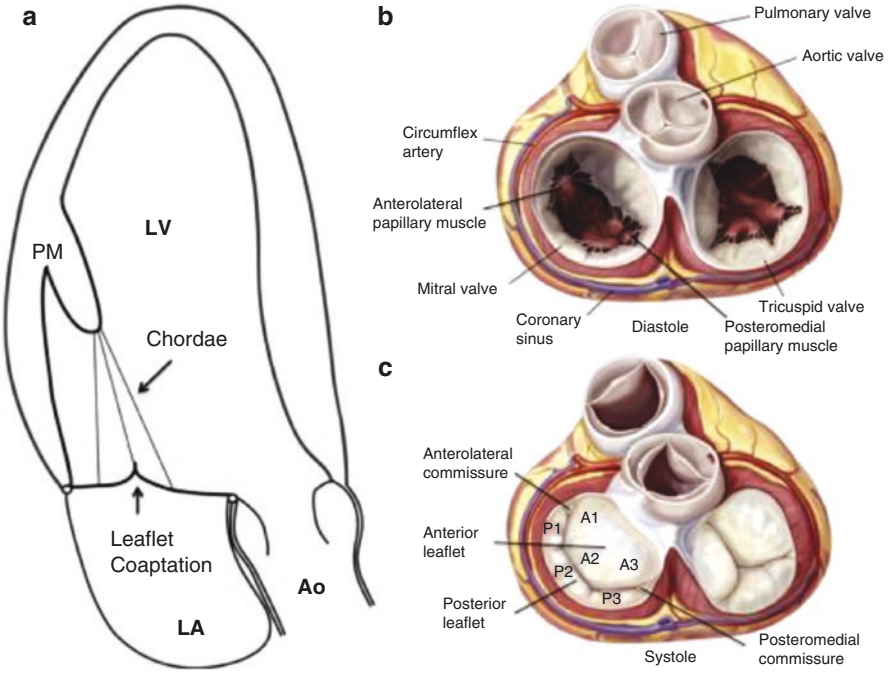


Fig. 14.1 Mitral valve apparatus. (**a**) Schematic drawing of the long-axis view of the heart in systole. (**b**) View of the valves in diastole with atrial walls removed. (**c**) View of the valves in systole. *Ao* aorta, *LA* left atrium, *LV* left ventricle, *PM* papillary muscle. Panels b and c are adapted from Carpentier A et al. Carpentier's Reconstructive Valve Surgery. From Valve Analysis to Valve Reconstruction. 2010 Saunders Elsevier (Duplicated from Dal-Bianco JP & Levin RA with permission [8])

- The right (posteromedial) and left (anterolateral) fibrous trigones of the mitral annulus are dense areas of fibrous continuity between the aortic, mitral, and tricuspid valves on the right and between the aortic and mitral valves on the left.
- The mitral annulus consists of a stronger anterior fibrous portion between the two trigones and a weaker posterior muscular portion that is most often affected with annular dilation.
- The anterior mitral leaflet attaches to 1/3 of the mitral annulus and has a smooth free edge, while the posterior leaflet attaches to 2/3 of the mitral annulus and has a scalloped free edge. The leaflets are divided into three segments each—the posterior leaflet is divided into anterolateral (P1), middle (P2), and posteromedial (P3) segments, while the anterior leaflet is divided into anterior (A1), middle (A2), and posterior (A3) segments.
- The valve leaflets have a marginal rough zone for coaptation and a central smooth zone.
- The primary chordae attach at the leaflet margins to prevent prolapse, the secondary chordae attach centrally on the ventricular side of the leaflets, and the tertiary chordae attach to the leaflet base on the posterior leaflet alone.

- The anterolateral and posteromedial papillary muscles provide chordae to both leaflets.
- At risk structures during a valve repair or replacement include: circumflex coronary artery when placing posterior annular sutures along the anterolateral commissure and P1 scallop, aortic valve (left and non-coronary cusps) when placing anterior annular sutures between two trigones, conduction system when placing annular sutures in the posteromedial trigone commissure/right trigone, and coronary sinus when placing a retraction stitch in the left atrial wall close to the base of the P3 scallop.

Valve Pathophysiology [3–7, 9, 10]

- Mitral regurgitation (MR) is defined as retrograde blood flow from the left ventricle (LV) into the left atrium (LA) during systole, whereas mitral stenosis (MS) is defined as narrowing of the MV orifice leading to an increased flow velocity and pressure gradient across the valve during diastole.
- Affecting 2–3% of US adults, MR secondary to prolapse is a much more common pathology in developed countries than is MS given the paucity of rheumatic fever in developed nations [9].

Mitral Regurgitation

- MR can be categorized as primary (i.e., "organic"), in which there is an intrinsic pathology with the valve itself (e.g., myxomatous degeneration), or secondary (i.e., "functional"), in which a structurally normal valve's architecture is distorted because of pathology affecting the annulus or subvalvular apparatus (e.g., chronic LV overload leading to eccentric hypertrophy and architectural distortion of the LV).
- MR can develop chronically secondary to myxomatous degeneration, rheumatic fever, or dilated ischemic cardiomyopathy, or can occur acutely secondary to chordal rupture, endocarditis, or papillary muscle rupture following MI.
- Degenerative MR is the most common cause of MR (60–70% of cases in developed countries) and is a primary process in which myxomatous degeneration affects the MV and causes valve prolapse, ranging from fibroelastic deficiency, in which the process is isolated to only a part of the valve, to Barlow's disease, in which there is generalized redundancy and thickening of the entire valve.
- Ischemic cardiomyopathy accounts for approximately 20% of MR cases in developed countries and can cause secondary MR via ventricular dilation which causes papillary muscle displacement (e.g., apical displacement of the posteromedial papillary muscle) and chordal tethering of valve leaflets, and by dilation of the MV annulus.

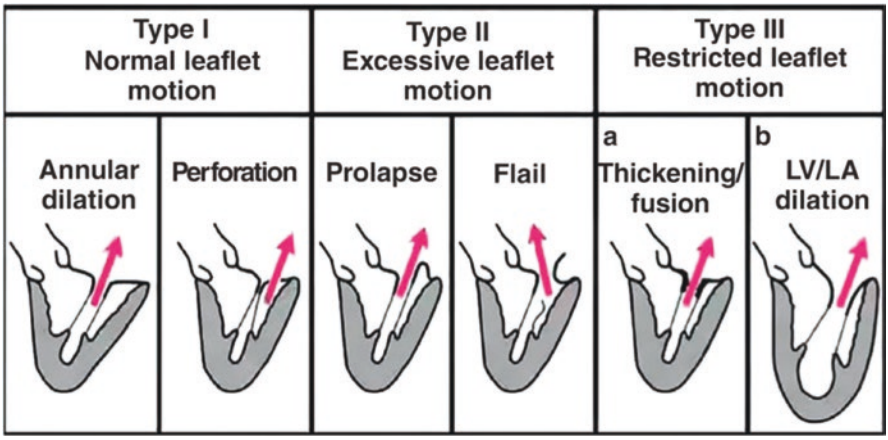


Fig. 14.2 Carpentier classification of functional mechanisms of MR. (Duplicated from Zoghbi et al. with permission [11])

- Endocarditis and rheumatic disease are less common causes of MR in developed countries (each ~2–5% of cases), whereas rheumatic disease is the primary cause of MR in the developing world.
- The functional mechanism of regurgitation can be further categorized according to the Carpentier classification system, which helps to guide operative decision-making (Fig. 14.2).
  - Type I: normal leaflet motion, with regurgitation secondary to annular dilation (e.g., dilated cardiomyopathy) or leaflet perforation (e.g., endocarditis).
  - Type II: excess leaflet motion, with regurgitation secondary to leaflet prolapse (e.g., fibroelastic deficiency, myxomatous degeneration, Barlow's disease) or flail (e.g., ruptured chordae or ruptured papillary muscle secondary to ischemic insult).
  - Type III: restricted leaflet motion, with regurgitation secondary to poor leaflet coaptation.

    IIIa: restricted motion during both diastole (i.e., restricted valve opening) and systole (i.e., restricted valve closure) (e.g., leaflet thickening and calcification or commissural fusion secondary to rheumatic heart disease, radiation, or carcinoid).
    IIIb: restricted motion during systole alone (i.e., restricted valve closure) (e.g., ventricular dilation and papillary muscle displacement leading to chordal tethering in the setting of dilative ischemic cardiomyopathy).
- Patients can remain asymptomatic with MR for an extended period so long as LV function is preserved, but as the LV remodels from increased preload, the LV dilates and hypertrophies, causing increased LV filling pressures, resulting in worsening left atrial distention and pulmonary venous pressures and subsequent

	MR SEVERITY		
	Mild	Moderate	Severe
Color flow jet area	small, central, narrow	variable	>50% of left atrium
Vena contracta width (cm)	>0.3	0.3-0.7	>0.7
Pulmonary vein flow	systolic dominance	normal or systolic blunting	no systolic flow or systolic flow
Regurgitant volume (cc)	<30	30-60	>60
Regurgitant fraction (%)	<30	30-50	>50
Effective regurgitant orifice area (cm2)	<0.2	0.2-0.4	>0.4

Adapted with permission from Zoghbi et al. "Recommendations for Noninvasive Evaluation of Native Valvular Regurgitation." [31]

Fig. 14.3 Grading of MR by echocardiography. (Adapted with permission from Zoghbi et al. "Recommendations for Noninvasive Evaluation of Native Valvular Regurgitation" [11])

pulmonary edema and symptomatic heart failure. Worrisome prognostic features include symptoms of heart failure, atrial fibrillation, advanced age, LV distention, and reduced EF.

- Physical exam findings of MR include a systolic click and a mid- to late-systolic murmur, best heard at the apex and radiating to the axilla.
- MR diagnosis is further aided by ECG demonstrating left atrial enlargement, atrial fibrillation, or a prior ischemic event; a CXR demonstrating cardiomegaly and/or pulmonary edema; and echocardiography. The EF can be misleadingly normal in patients with compensated MR due to the "pop-off" valve effect of MR, and myocardial dysfunction can be present despite a normal EF. Coronary angiography may be helpful in evaluation of CAD in the setting of ischemic MR.
- MR can be graded on transthoracic echo according to severity (Fig. 14.3), which drives operative decision-making as outlined further below.

Mitral Stenosis (MS)

- The primary cause of MS is rheumatic disease, in which mimicry between group A strep antigens and valvular tissue causes autoimmune mediated fibrosis, leading to cardiac damage.
- Rheumatic valve disease affects all parts of the MV apparatus, causing fibrosis and thickening of the leaflets, shortening of the chordae, and fusion of the commissures, producing a characteristic "fish mouth" valve appearance limiting valve motion during both systole and diastole. Concurrent chordae shortening and fibrosis also contribute to MR.
- Nonrheumatic etiologies of MS include senile mitral calcification, congenital deformities, carcinoid, lupus, cardiac neoplasm, prosthetic valve calcification, endocarditis, and mediastinal radiation. While severe mitral annular calcification can cause calcification of leaflets, commissural fusion does not occur as in rheumatic disease.

	MS SEVERITY		
	Mild	Moderate	Severe
Valve area	>1.5	1.0-1.5	<1.0
Mean gradient (mm Hg)	<5	5-10	>10
Pulmonary artery pressure (mmHg)	<30	30-50	>50

Adapted with permission from Baumgartner et al. "Echocardiograpic assessment of valve stenosis." [32]

Fig. 14.4 Grading of MS by echocardiography. (Adapted with permission from Baumgartner et al. "Echocardiographic assessment of valve stenosis" [12])

- Patients with rheumatic disease typically have rheumatic fever before age 20, with MS symptoms not manifesting until one to three decades later. Although patients can remain asymptomatic for an extended period, ultimately the increased mitral gradient cause left atrial distention, pulmonary hypertension, and right heart failure, manifesting as atrial fibrillation, pulmonary edema, and hemoptysis.
- On exam, auscultation can reveal an opening snap after S2, a loud S1, and a mid-diastolic murmur heard loudest at the apex.
- ECG can demonstrate arrythmias (especially atrial fibrillation) and p-mitrale from LA hypertrophy, while CXR can demonstrate LA enlargement, a calcific mitral annulus, or pulmonary congestion.
- Similarly to MR, MS can be graded on transthoracic echo according to severity, which drives operative decision-making as outlined further below (Fig. 14.4). The normal MV area is 4–6 cm^2, and MS severity is classified according to valve area reduction and the transvalvular pressure gradient.

Management of Mitral Regurgitation [2–7, 9, 10, 13–27, 28]

Decision to Operate

- The class I indications for operating on primary degenerative MR include: (1) symptomatic patients with chronic severe primary MR and an LVEF >60%, (2) asymptomatic patients with chronic severe primary MR and LV dysfunction (LVEF 30–60% and/or LV end-systolic diameter >40 mm), or (3) patients with chronic severe MR underdoing another cardiac operation.
- Urgent surgery is indicated for symptomatic patients with acute severe primary MR secondary to papillary muscle rupture (most often the posteromedial papillary muscle owing to its single right or circumflex coronary blood supply, in contrast to the dual left anterior descending and circumflex blood supply of the anterolateral papillary muscle).
- MV repair is recommended over replacement for severe MR, but replacement may be merited in certain circumstances such as heavy calcification of the leaflets or chordae secondary to rheumatic disease, significant leaflet destruction from endocarditis, or acute papillary muscle rupture.

Operative Management of Primary (Degenerative) MR

- The overarching guiding principles of mitral repair include preserving or restoring leaflet motion, restoring an appropriate coaptation surface, and stabilizing the mitral annulus that is often dilated due to underlying pathology.
- Operative steps:
  - The heart is exposed either via median sternotomy or via right anterolateral mini-thoracotomy in the third or fourth interspace, which is preferred for minimally invasive or robotic approaches.
  - There are multiple ways to approach the MV, but the most common is the left atriotomy interatrial/paraseptal approach through the Sondergaard's groove, anterior to the right pulmonary veins. Other approaches include the left atrial dome approach, the vertical transseptal incision via the fossa ovalis, the extended superior transseptal (Guiraudon) incision, and the horizontal transseptal Dubost approach.
  - Once the MV is exposed, it is assessed intra-operatively to evaluate the annulus (dilated or calcified); the degree of leaflet motion (using nerve hooks and a reference point, usually P1); the chordal attachments; and leaflet height.
  - Repairs are achieved through an array of techniques, which can be used in isolation or combination depending on the type of valve dysfunction and the valve lesion. These techniques include resection of excess leaflet tissue (triangular or quadrangular) with limited or extended sliding plasty, artificial chordal replacement to mimic functional chordae tendinae, chordae transfer or transposition, or commissuroplasty.
  - Autologous pericardium (can be treated with glutaraldehyde for better handling) can be used to fix leaflet perforation in endocarditis cases. Autologous pericardium can also be used when the height of either posterior or anterior leaflet needs to be built up (due to paucity of tissue) in order to achieve a better coaptation depth. In such cases, an incision is made along the base of the leaflet and extended from commissure to commissure. Then, either a semilunar shaped patch (for the posterior leaflet) or an oval shaped patch (for the anterior leaflet) is sewn in, thus increasing the corresponding leaflet height and area.
  - Neochordal repair (using the CV-4 PTFE pledgeted sutures) is particularly useful for anterior leaflet repair as well as for posterior leaflet repair in the setting of paucity of tissue, such as the case of fibroelastic deficiency. The length of the neochords is usually adjusted under saline distention of the LV to ensure tight seal of the valve, no residual prolapse of the leaflet, and a full unfolding of the anterior leaflet in order to prevent SAM (systolic anterior motion, further discussed below). Neochordal repair has largely replaced an older technique of chord transposition for anterior leaflet repair, in which a posterior leaflet chord is transposed to the anterior leaflet.

  - Remodeling annuloplasty is then performed in all mitral valve repairs with the goal to restore the size and shape of the mitral annulus and to achieve a durable repair result. This is done by implanting either a complete annuloplasty ring or a partial band implanted from trigone to trigone along the posterior annulus.
  - Annuloplasty sizing is best achieved by measuring the height of the anterior leaflet from its base to the edge of the A2 segment. With chronic MR, the mitral annulus is weakest along the posterior annulus and thus tends to dilate in the antero-posterior dimension.
  - The quality of the repair should be tested with a saline test, distending the LV and monitoring for regurgitation. Not infrequently at this stage, the surgeon needs to close an indentation between neighboring scallops that revealed itself after either resection, neochordal repair, or some other technique.
- Significant advancements are being made in tailoring mitral repair for minimally invasive and robotic approaches.

Systolic Anterior Motion (SAM)

- SAM describes the displacement of the anterior mitral leaflet into the left ventricular outflow tract during systole, causing a Venturi effect and obstruction, secondary to displacement of the leaflet coaptation margin toward the LVOT, and can be seen in up to 3% of mitral repairs.
- SAM occurs secondary to a discrepancy between an excessive amount of valve tissue and a small mitral orifice area—after a mitral repair, excess posterior leaflet tissue can push the anterior leaflet toward the LVOT during systole.
- Specific risk factors include a tall posterior leaflet (>15 mm), an anterior leaflet: posterior leaflet height ratio <1.3, an acute aorto-mitral angle (<120°), presence of an upper septal hypertrophy, short coaptation-septum distance (C-sept <25 mm), a small hyperkinetic ventricle, anteriorly displaced papillary muscles, or a small annuloplasty ring.
- In the immediate post-bypass period, SAM is managed by optimizing ventricular preload/filling through volume and AV pacing, and preventing ventricular hypercontractility by limiting inotropes and adding a beta-blocker. If these measures are unsuccessful, operative re-repair is necessary by reducing the size of the posterior leaflet and/or upsizing the annuloplasty ring.

Operative Management of Ischemic (Secondary) MR

- A remodeling annuloplasty using an undersized complete ring is used to reduce the antero-posterior annulus diameter, thus facilitating leaflet coaptation.
- If the subvalvular apparatus is further tethering leaflets and preventing adequate coaptation, secondary chordae can be divided, primary chordae can be divided and replaced with artificial chords, and papillary muscles can be translocated.

- Mitral leaflets can also be augmented at their bases with pericardial patches sewn in from commissure to commissure in order to improve coaptation depth.
- Concurrent coronary revascularization is often performed to improve ventricular function.
- While undersized annuloplasty repair was long accepted as the normal for management of ischemic MR, a Cardiothoracic Surgical Trials Network study published in 2016 suggests that for severe ischemic MR, at time of CABG, mitral valve replacement (MVR) may be superior to repair. The technique of MVR is outlined further below [26].

Management of Mitral Stenosis [2–7, 9, 10, 29–32]

Decision to Operate

- Operative interventions for patients with MS tend to be deferred until patients become symptomatic, although exceptions can be made; these patients tend to have an MV area <1.5 cm^2, severe LA enlargement, and elevated PA systolic pressures >30 mmHg. Operative interventions include percutaneous mitral balloon commissurotomy, open surgical commissurotomy, or MVR.
- The first-line therapy for MS in appropriate patients is percutaneous mitral balloon commissurotomy. This strategy is particularly suited for symptomatic patients with isolated severe MS, but is contraindicated in individuals with atrial thrombus or significant MR.
- The Wilkins-Palacios score is used to determine patients for whom PMBC has a low chance of success and who would be better served with an open operation. The score considers valve mobility, thickening, calcification, and subvalvular thickening.
- Open commissurotomy and MVR should be delayed until patients have severe symptoms (NYHA III or IV), and are primarily indicated for symptomatic patients with severe MS who are not candidates for, or have failed, previous PMBC. Exceptions include pregnant patients or patients undergoing cardiac surgery for another indication, in whom earlier mitral intervention is appropriate.

Operative Technique

- The primary open operative technique for management of MS is a chordal-sparing MVR, as open commissurotomy is performed much less frequently. Among patients who underwent a PMBC, 60% require repeat intervention within 20 years, 76% of whom undergo a valve replacement [30].

Open Commissurotomy

- Open commissurotomy, which is rarely performed in the modern era, is indicated for patients with pure MS with commissural fusion and with preserved leaflet mobility.
- Open commissurotomy is performed by sharply incising the fused valve commissures, with possible additional splitting of the papillary muscle or fenestration of fused chordae.

Mitral Valve Replacement (MVR)

- Unlike in MR, in which the tissue is relatively pliable and amenable to repair, the significant fibrosis associated with MS often necessitates an MVR if an operation is indicated.
- In a chordal-sparing MVR, attempts are made to preserve the papillary muscles and the posterior leaflet in order to preserve ventricular geometry, which improves post-operative outcomes.
- Operative steps:
  - The MV is accessed as described above for MV repairs.
  - The anterior leaflet must be transferred to prevent LVOT obstruction. It is detached from the annulus and divided into two segments with strong primary chords and the segments are re-anchored with valve sutures to their corresponding commissures.
  - The posterior leaflet is left intact, and the valve leaflet is incorporated into the valve sutures.
  - If the leaflets are too calcified and cannot be preserved, they are excised to prevent outflow tract obstruction.
  - Horizontal mattress sutures are placed circumferentially around the mitral annulus in an everting or non-everting fashion, and the valve is tied down.
  - For mechanical valves, the prosthetic valve should be oriented in an anti-anatomic fashion (valve leaflets perpendicular to the orientation of the native leaflets) to improve leaflet clearance and reduce the risk of a "lazy leaflet." Bioprosthetic valves should be oriented with two posts facing the trigones and one post dividing the posterior annulus.

Mechanical Versus Bioprosthetic MVR

- The decision between a mechanical or bioprosthetic MV depends on durability, risks associated with anticoagulation, and patient preference.
- Both the ACC and AHA recommend mechanical prosthesis for patients younger than 60, and bioprosthetic valves for patients older than 70, primarily guided by higher rates of eventual valve degeneration and re-operation in younger patients.

Between 60 and 70 years of age, either mechanical or bioprosthetic valves can be used depending on the relative risks and benefits of anticoagulation and valve longevity for the individual patient.
- Bioprosthetic valves are chosen if there is any contraindication to anticoagulation, or during pregnancy or women of child-bearing age.

Special Operative Considerations [4–7, 16]

Mitral Annular Calcification

- Mitral annular calcification (MAC) is the phenomenon of significant calcium deposits in the mitral annulus, primarily along the posterior annulus but sometimes circumferentially.
- MAC complicates MV operations since it increases the risks of atrioventricular disruption, paravalvular leak, and valve dehiscence after valve replacement.
- MAC is managed in various ways, depending on tissue quality and age of the patient. In some cases, the sutures can be passed behind the MAC. Because of irregularity of the calcium bar, a felt gasket can be tailored using a bioprosthetic valve sizer. The gasket is then placed between the annulus and the sewing ring of the valve in order to reduce the risk of paravalvular leak in the future. MAC can also be partially debrided either using a rongeur or CUSA (cavitron ultrasonic aspirator), thus allowing placement of the valve sutures through the remaining calcium bar. MAC can also be completely excised, especially in younger patients with stronger tissue quality. Complete excision is achieved by separating the posterior leaflet from the annulus, removing the calcium "en bloc," and repairing the defect with a pericardial patch to mitigate the risk of atrioventricular groove disruption.
- Aggressive MAC debridement or placement of an oversized valve prosthesis can be complicated by AV groove disruption, or separation of the LA and LV due to rupture of the LV near the AV groove posteriorly. This manifests as bright red hemorrhage stemming from behind the heart upon release of the aortic clamp. Management requires going back on bypass, rearresting the heart, removing the old valve prosthesis, and internal repair of the defect using a pericardial patch followed by valve re-replacement.

References

1. Cohn LH. The first successful surgical treatment of mitral stenosis: the 70th anniversary of Elliot Cutler's mitral commissurotomy. Ann Thorac Surg. 1993;56(5):1187–90.
2. Otto CM, et al. 2020 ACC/AHA guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association joint committee on clinical practice. Circulation. 2021;143(5):e35.

3. Carpentier A, Adams D, Filsoufi F. Carpentier's reconstructive valve surgery. St. Louis, MO: Elsevier; 2010.
4. Goldstone A, Woo J. Chapter 80: Surgical treatment of the mitral valve. In: Sabiston & Spencer surgery of the chest. 9th ed. Philadelphia, PA: Elsevier; 2016. p. 1384–429.
5. Anyanwu A, et al. Chapter 92: Ischemic mitral regurgitation. In: Sabiston & Spencer surgery of the chest. 9th ed. Philadelphia, PA: Elsevier; 2016. p. 1624–52.
6. Cohn LH, McClure RS. Chapter 42: Mitral valve repair. In: Kaiser's mastery of cardiothoracic surgery. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2014. p. 407–18.
7. Chan V, et al. Chapter 44: Mitral valve repair for ischemic mitral regurgitation. In: Kaiser's mastery of cardiothoracic surgery. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2014. p. 419–39.
8. Dal-Bianco JP, Levine RA. Anatomy of the mitral valve apparatus. Cardiol Clin. 2013;31(2):151–64.
9. Coffey S, et al. The modern epidemiology of heart valve disease. Heart. 2016;102:75–85.
10. Anyanwu AC, Adams DH. Etiologic classification of degenerative mitral valve disease: Barlow's disease and fibroelastic deficiency. Semin Thorac Cardiovasc Surg. 2007;19:94.
11. Zoghbi WA, et al. Recommendations for noninvasive evaluation of native valvular regurgitation. J Am Soc Echocardiogr. 2017;30(4):303–71.
12. Baumgartner H, et al. Echocardiographic assessment of valve stenosis: EAE/ASE recommendations for clinical practice. Eur J Echocardiogr. 2009;10:1–25.
13. DiBardino D, et al. Four decades of experience with mitral valve repair: analysis of differential indications, technical evolution, and long-term outcome. J Thorac Cardiovasc Surg. 2010;139(1):76–83.
14. Suri RM, et al. Association between early surgical intervention vs watchful waiting and outcomes for mitral regurgitation due to flail mitral valve leaflets. JAMA. 2013;310:609–16.
15. Acker MA, et al. Mitral-valve repair versus replacement for severe ischemic mitral regurgitation. NEJM. 2014;370(1):23–32.
16. Yun KL, et al. Randomized trial comparing partial versus complete chordal-sparing mitral valve replacement: effects on left ventricular volume and function. J Thorac Cardiovasc Surg. 2002;123(4):707–14.
17. David TE, et al. Late outcomes of mitral valve repair for mitral regurgitation due to degenerative disease. Circulation. 2013;127(14):1485–92.
18. Gillinov AM, et al. Valve repair versus valve replacement for degenerative mitral valve disease. J Thorac Cardiovasc Surg. 2008;135(4):885–93.
19. Kaneko T, et al. Mechanical versus bioprosthetic mitral valve replacement in patients <65 years old. J Thorac Cardiovasc Surg. 2014;147(1):117–26.
20. Chan KMJ, et al. Coronary artery bypass surgery with or without mitral valve annuloplasty in moderate functional ischemic mitral regurgitation: finale results of the randomized ischemic mitral evaluation (RIME) trial. Circulation. 2012;126(21):2502–10.
21. DiBardino DJ, et al. Four decades of experience with mitral valve repair: analysis of differential indications, technical evolution, and long-term outcomes. J Thorac Cardiovasc Surg. 2010;139(1):76–83.
22. Bax JJ, et al. Restrictive annuloplasty and coronary revascularization in ischemic mitral regurgitation results in reverse left ventricular remodeling. Circulation. 2004;110(11 Suppl 1):II103–8.
23. Deja MA, et al. Influence of mitral regurgitation repair on survival in the surgical treatment for ischemic heart failure trial. Circulation. 2012;125(21):2639–48.
24. Castillo JG, et al. A near 100% repair rate for mitral valve prolapse is achievable in a reference center: implications for future guidelines. J Thorac Cardiovasc Surg. 2012;144(2):308–12.
25. David TE, et al. Chordal replacement with polytetrafluoroethylene sutures for mitral valve repair: a 25-year experience. J Thorac Cardiovasc Surg. 2013;145(6):1563–9.
26. Goldstein D, et al., for the CTSN. Two-year outcomes of surgical treatment of severe ischemic mitral regurgitation. N Engl J Med. 2016;374(4):344–53.

27. Michler RE, et al., for the CTSN. Two-year outcomes of surgical treatment of moderate ischemic mitral regurgitation. N Engl J Med. 2016;374(20):1932–41.
28. Brown ML, et al. Systolic anterior motion after mitral valve repair: is surgical intervention necessary? J Thorac Cardiovasc Surg. 2007;133(1):136–43.
29. Wilkins GT, et al. Percutaneous balloon dilatation of the mitral valve: an analysis of echo. Br Heart J. 1988;60:299–308.
30. Bouleti C, et al. Reinterventions after percutaneous mitral commissurotomy during long-term follow-up, up to 20 years: the role of repeat percutaneous mitral commissuorotmy. Eur Heart J. 2013;34:1923–30.
31. Marijon E, et al. Rheumatic heart disease. Lancet. 2012;379:953–64.
32. Chandrashekhar Y, et al. Mitral stenosis. Lancet. 2009;374:1271–83.

Chapter 15
Tricuspid Valve Repair and Replacement

Elaine M. Griffeth and Joseph A. Dearani

Learning Objectives
- Basic introduction to valve pathologies
- Indications for repair vs. replacement
- Repair techniques
- Replacement technique
- Prosthesis selection

Introduction

In general, cardiac valvular pathology is characterized as regurgitant, stenotic, atretic, or displaced. Tricuspid valve pathology is predominantly regurgitant due to annular dilation and leaflet tethering secondary to right ventricular dilation. This right ventricular dilation is generally a result of volume or pressure overload, as seen in patients with pulmonary hypertension (primary or secondary due to left-sided heart disease) or dilated cardiomyopathy. Operative management of this condition centers on tricuspid valve repair with annuloplasty, and outcomes are better if surgery is performed prior to the onset of right ventricular systolic dysfunction.

E. M. Griffeth · J. A. Dearani (✉)
Department of Cardiovascular Surgery, Mayo Clinic, Rochester, MN, USA
e-mail: griffeth.elaine@mayo.edu; jdearani@mayo.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_15

A small number of congenital heart disease patients are present with atretic or displaced tricuspid valve pathology, which require more complex repair techniques.

Anatomy

Embryology: The atrioventricular (AV) valves arise from the endocardial cushion. This embryonic structure also gives rise to the semilunar valves and atrial and ventricular septae.

The tricuspid valve is the right-sided AV valve and is composed of a saddle-shaped annulus, three leaflets (anterior, septal, posterior), and subvalvar apparatus including chordae tendineae and papillary muscles.

Please refer to Chap. 2 of this book for more detailed descriptions of valvular anatomy.

Pathology

Primary Tricuspid Regurgitation

Congenital

- Ebstein anomaly—displacement of tricuspid valve posteriorly and septal leaflet inferiorly into the right ventricle, resulting in atrialization of a portion of the right ventricle; associated with atrial septal defects (ASD).
- Congenital tricuspid valve dysplasia—morphologically abnormal valve leaflets and/or subvalvular apparatus (e.g., shortened chordae).
- Endocardial cushion defect (partial or complete atrioventricular septal defect [AVSD])—regurgitant common AV valve resulting from malformation of septae and AV valves from endocardial cushion.

Acquired

- Endocarditis—most common pathogens are staphylococcus and streptococcus; see Chap. 17 for additional information.
- Rheumatic heart disease—valve damage is immune-mediated via a type II hypersensitivity reaction (not due to direct damage from bacteria); can also cause tricuspid stenosis.
- Carcinoid heart disease.
- Myxomatous degeneration (connective tissue disorders).
- Iatrogenic—transvenous pacing leads, endomyocardial biopsy.
- Radiation.
- Trauma.

Secondary (Functional) Tricuspid Regurgitation

- Annular dilation is the most common cause of tricuspid regurgitation; the resultant change in shape of the annulus from oval to circular leads to leaflet tethering and limited coaptation.
  - This can occur in patients with atrial fibrillation in the absence of both pulmonary hypertension and left-sided heart disease.
- Pulmonic stenosis, especially if right ventricular systolic pressure (RVSP) is >55 mmHg.
- Pulmonary hypertension.

Tricuspid Stenosis

- Rheumatic heart disease.
- Carcinoid heart disease.
- Tumor/thrombus (e.g., patients with advanced renal cell carcinoma can have tumor thrombus extending from inferior vena cava (IVC) through right atrium and across the tricuspid valve, effectively narrowing the valve orifice).

Tricuspid Atresia

- Failure of the tricuspid valve to form, resulting in hypoplasia of the right ventricle chamber and outflow tract; associated with ASD and ventricular septal defect (VSD). An ASD is required for post-natal viability with staged palliation in a single ventricle treatment pathway towards the Fontan procedure.

History and Physical Exam

Patients may present with symptoms of right heart failure, which include fatigue, decreased appetite, ascites, peripheral edema, and low albumin. The provider should perform a physical exam, looking for signs of right heart failure and its potential causes such as left heart pathology, right-sided valvular disease, or cor pulmonale.

Physical Exam Findings

- Neck: Jugular venous distension, including a prominent "v" wave indicating tricuspid regurgitation.
- Pulmonary: Signs of pulmonary disease in patients with cor pulmonale, such as increased thoracic anteroposterior diameter and expiratory wheezing in patients with chronic obstructive pulmonary disease (COPD).

- Cardiac:
  - Systolic murmurs:

    Tricuspid regurgitation: holosystolic, high-pitched "blowing" murmur, intensity may increase with inspiration.
  - Diastolic murmurs:

    Tricuspid stenosis: mid to late diastolic, delayed "rumbling" murmur.
  - Right-sided S3 "gallop" indicates right ventricular volume overload and right heart failure.
- Abdomen: Hepatomegaly, ascites.
- Extremities: Peripheral edema, especially in the ankles in ambulatory patients.

Symptoms or signs of right heart failure should raise concern for decompensated tricuspid regurgitation. Due to increased risk for operative mortality, the patient should be admitted and medically optimized prior to surgical intervention. Patients with tricuspid stenosis can present with significantly reduced cardiac output.

Imaging

Echocardiography

- Transthoracic echocardiography (TTE): The main imaging modality used for evaluation of the tricuspid valve; allows for differentiation of primary and secondary causes of tricuspid valve pathology, assessment of the right and left heart function, and measurement of right sided pressures.
- Transesophageal echocardiography (TEE): Used intraoperatively to assess the valve pre- and post-bypass.

Please refer to Chap. 4 for additional details on echocardiography.

Cross-Sectional Imaging

- Cardiac MRI: Used to assess the right ventricle size and function. The tricuspid valve is also assessed but echocardiography is often preferred for valvular anatomic detail.

Please refer to Chap. 5 for additional details on cardiac MRI.

Cardiac Catheterization

- Right heart catheterization: Used to assess hemodynamics and to evaluate the right heart in the case of discordant imaging and/or exam findings.

  Please refer to Chap. 6 for additional details on cardiac catheterization.

In patients presenting with tricuspid regurgitation, always evaluate for left-sided pathology and/or pulmonary hypertension, especially in the setting of structurally normal valves.

Indications for Intervention

Surgical outcomes and survival are better if surgery is performed prior to the onset of right ventricular systolic dysfunction. End-organ damage such as liver or kidney failure markedly affects survival, as does reoperation for severe, isolated tricuspid regurgitation after left-sided valve surgery [1]. Additionally, there is an increased risk of right ventricular failure after operation for patients with severe right ventricular systolic dysfunction or irreversible pulmonary hypertension preoperatively [1]. The above factors are important to consider when evaluating patients for potential surgical intervention.

The indications for intervention are based on the severity of valvular disease and patient symptoms. Tricuspid regurgitation can be categorized into three stages (B-progressive, C-asymptomatic severe, and D-symptomatic severe) based on patient symptoms and valve hemodynamics as measured with echocardiography [1]. Serial measurements must be obtained because tricuspid regurgitation is dynamic and affected by preload. Tricuspid stenosis is categorized as severe (Stages C and D) based on echocardiographic evaluation [2].

Tricuspid Regurgitation

- Surgical treatment of tricuspid regurgitation at the time of surgery for left-sided valve pathology is performed in cases of moderate or severe tricuspid regurgitation (Stages C and D) and to prevent the development of severe tricuspid regurgitation in cases when progressive tricuspid regurgitation can be expected (Stage B) [1].
  - These are the most common indications for tricuspid valve surgery. The previously held belief that right heart dilatation and tricuspid regurgitation would auto-correct following correction of left-sided pathology has been shown to be frequently incorrect [3–7].

- Isolated tricuspid valve surgery is indicated in patients with severe symptomatic tricuspid regurgitation (Stage D) either due to primary disease (e.g., structural or device lead damage) or secondary disease poorly responsive to medical therapy but in the absence of left-sided pathology and pulmonary hypertension, as seen in patients with atrial fibrillation [1].
- Isolated tricuspid valve surgery can be considered on a case-by-case basis for patients with severe asymptomatic (Stage C) primary tricuspid regurgitation and progressive right ventricular dilatation or systolic dysfunction, or severe symptomatic (Stage D) tricuspid regurgitation who have previously undergone left-sided valve surgery but do not have severe pulmonary hypertension or severe right ventricular systolic dysfunction [1].

Tricuspid Stenosis

- Surgical treatment is recommended for patients with severe tricuspid stenosis at the time of operations for left-sided valve disease and for isolated, symptomatic severe tricuspid stenosis [2].

Congenital Heart Disease

- Ebstein anomaly
  - Surgical repair is indicated if there is significant tricuspid regurgitation and one or more of the following are present: heart failure symptoms, objective evidence of worsening exercise capacity, progressive right ventricular systolic dysfunction by echocardiography or cardiac MRI, progressive right ventricular enlargement, systemic desaturation from right-to-left atrial shunt, paradoxical embolism, and/or atrial tachyarrhythmias [8].
- Congenital tricuspid valve dysplasia
  - Surgical repair is considered on a case-by-case basis in patients who have developed symptoms of right heart failure, arrhythmias, or progressive right ventricular dysfunction on imaging. Decisions regarding intervention are also informed by the indications listed above for tricuspid regurgitation.
- Endocardial cushion defects
  - Surgical repair is usually performed early in life (by age 6 months) to prevent irreversible pulmonary vascular disease resulting in Eisenmenger physiology [8].
  - If right AV valve regurgitation and/or stenosis develop later, valve surgery can be considered on a case-by-case basis. However, primary repair of AVSD or closure of residual shunts should not be performed if pulmonary artery systolic pressure is greater than two-thirds systemic, pulmonary vascular resistance is greater than two-thirds systemic, or there is a net right-to-left shunt [8].

- Tricuspid atresia
  - Neonates are treated with prostaglandins to maintain patency of the ductus arteriosus, and surgical intervention is recommended shortly thereafter to create a systemic-to-pulmonary artery shunt (restricted pulmonary blood flow) and/or to enlarge the ASD, or pulmonary artery banding when there is unrestricted pulmonary blood flow. Ultimately these patients are managed with univentricular palliation towards the Fontan procedure due to right ventricular hypoplasia.

Prosthetic Tricuspid Valves

- Repeat tricuspid valve replacement is indicated in patients with symptomatic severe prosthetic valve stenosis or regurgitation. In patients with asymptomatic severe prosthetic valve regurgitation who are low operative risk, surgery can be considered [1].
  - If stenosis is attributable to thrombus, lytic therapy or oral anticoagulation with a vitamin K antagonist is an appropriate first step.
  - If the prosthetic valve is biologic and the anatomic features are amenable to catheter-based valve-in-valve replacement, then percutaneous replacement is reasonable.
- Repeat tricuspid valve replacement is indicated in patients with intractable hemolysis or heart failure attributable to prosthetic transvalvular or paravalvular leak [1].
  - If the anatomic features of the paravalvular leak are amenable to catheter-based therapy, then percutaneous repair is reasonable.
- Repeat tricuspid valve replacement is indicated in patients with infective endocarditis after initiation of intravenous antibiotics. Please see Chap. 17 for a full discussion on management of infective endocarditis.

Tricuspid Valve Surgery

Operative Approach

- Cardiopulmonary bypass and aortic cross-clamp are required for most tricuspid valve surgery.
- Intraoperative transesophageal echocardiography is essential to assess the valve pre-bypass and to assess the adequacy of the repair or replacement post-bypass.
  - The conditions of anesthesia can reduce the degree of tricuspid regurgitation by at least one grade.

- The right atrium is incised parallel to the atrioventricular groove, starting from the right atrial appendage and heading halfway between the IVC and the atrioventricular groove.
  - Avoid being too close to the atrioventricular groove since the right coronary artery runs here.
- Systematically assess the tricuspid valve: evaluate the annulus for dilatation, examine the leaflets for abnormalities (e.g., perforations, thickening), flail segments, etc., and use a blunt hook to lift the leaflets to assess the subvalvular apparatus for ruptured chordae or papillary muscles.
- Perform a leak test by injecting saline into the right ventricle and identify the site of the regurgitation.
  - If the valve appears normal with a central leak, then the cause is likely annular dilatation.

Tricuspid Valve Repair (Fig. 15.1)

- *Tricuspid annuloplasty*: Used for functional tricuspid regurgitation; techniques include banded annuloplasty (most common, Fig. 15.1a) and De Vega suture annuloplasty (Fig. 15.1b) or eccentric inferior annuloplasty [9, 10].
  - Using interrupted sutures, the majority of annular reduction is accomplished in the anterior and posterior annulus.
  - Avoid placing sutures between the anteroseptal commissure and the middle of the septal annulus to protect conduction tissue and avoid heart block.
- *Medial eccentric annuloplasty ("bicuspidization")* (Fig. 15.1c): Used for lesser degrees of annular dilation; technique involves placing pledgeted mattress suture from the center of the posterior leaflet to the commissure between the septal and posterior leaflets [11].
- *Leaflet repair* (Fig. 15.1d): Used for perforations/vegetations from endocarditis to reapproximate injured leaflet edges from trauma, or to augment leaflet size; technique for endocarditis involves excising the vegetation/patching the hole with pericardium, and augmentation involves adding an ellipse of pericardium to the annular side of the anterior leaflet [9, 10].
- *Artificial chordae* (Fig. 15.1e): Used for unsupported leaflets; technique involves suturing unsupported leaflet to a prominent apicoseptal trabeculation using pledgeted 5-0 Gore-Tex suture [9, 10].
- *Pacemaker leads* (Fig. 15.1f, g): If the transvenous ventricular lead is working well, it can be preserved by positioning it adjacent to the annulus in a commis-

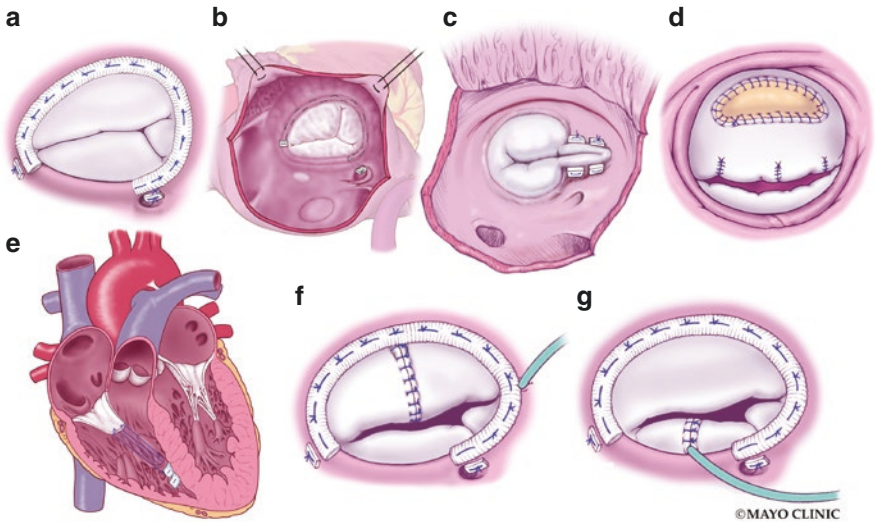


Fig. 15.1 Tricuspid valve repair techniques. (**a**) Banded annuloplasty. (**b**) Suture annuloplasty, sutures would then be cinched down around an appropriately sized Hegar dilator. (**c**) Medial eccentric annuloplasty. (**d**) Anterior leaflet augmentation, generally performed using autologous pericardium. (**e**) Artificial chordae from anterior leaflet to ventricular septum. (**f**) Dissection and removal of pacemaker lead entangled in anterior leaflet chordae, lead moved to anteroinferior commissure followed by suture leaflet repair and banded annuloplasty. (**g**) Dissection of pacemaker lead affecting septal leaflet excursion, moved to hinge point of septal leaflet followed by suture leaflet repair, banded annuloplasty also shown. (From Saran N & Dearani JA. Tricuspid Valve Repair: How I Teach It. Ann Thorac Surg. 2018 Mar;105(3):675–679, used with permission of Mayo Foundation for Medical Education and Research, all rights reserved)

sure; inferoseptal most common. Otherwise, removal and replacement with an epicardial lead should be performed.

- *Cone repair* (Fig. 15.2): Used for Ebstein anomaly; technique involves recruitment of all undelaminated leaflet tissue with complete surgical delamination, which is then anchored to the true annulus creating 360° of a "leaflet cone"; valve repair is accompanied by patch closure or subtotal closure of ASD, plication of atrialized right ventricle, and resection of redundant right atrial wall prior to closing atriotomy [10].
- *Tricuspid stenosis commissurotomy*: Used for tricuspid stenosis not amenable to percutaneous balloon valvuloplasty; technique involves separation of the fused commissures on either side of the septal leaflet [11].
- *Complete AVSD repair* (Fig. 15.3): Reconstruct septum with patch material and divide common AV valve into separate left and right AV valves by incorporating common AV valve tissue into the VSD patch.

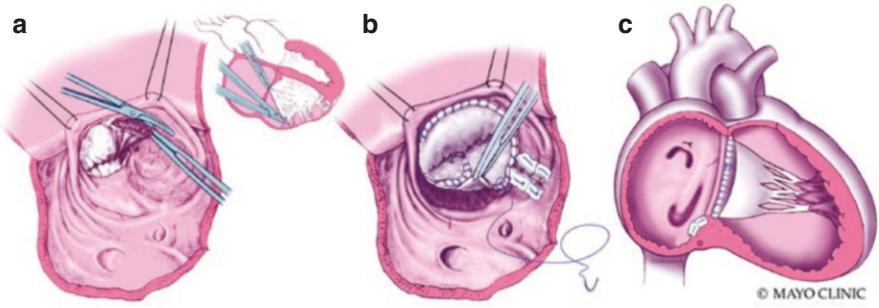


Fig. 15.2 Ebstein repair. (**a**) Surgical delamination of distal anterior leaflet. Fibrous and muscular attachments between the body of the leaflet and right ventricular myocardium are incised, but attachments to the leading edge of the leaflet must be maintained. (**b**) Leaflet tissue is anchored to the true annulus. The inferior annulus has been plicated with pledgeted sutures. (**c**) Completed cone reconstruction of the tricuspid valve for Ebstein anomaly. (From Dearani, J.A., Bacha, E. and Da Silva, J.P. (2008) Cone Reconstruction of the Tricuspid Valve for Ebstein's Anomaly: Anatomic Repair. Operative Techniques in Thoracic and Cardiovascular Surgery, 13, 109–125, used with permission of Mayo Foundation for Medical Education and Research, all rights reserved)

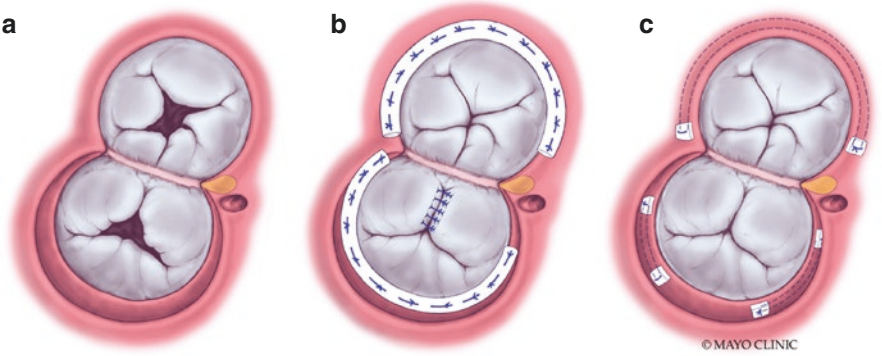


Fig. 15.3 Atrioventricular septal defect repair. (**a**) Anatomy of atrioventricular septal defect. (**b**) Banded annuloplasty of right and left atrioventricular valves, care is taken on right side of valves to protect conduction system. Suture repair of zone of apposition ("cleft") in left atrioventricular valve. (**c**) Right atrioventricular valve with suture annuloplasty. Left atrioventricular valve with eccentric suture annuloplasty, can be performed on the left, right, or both sides as shown here. Leaflet repair can be performed as needed (not shown). (From Patlolla SH, Dearani JA, Connolly HM, Warnes CA, Lahr BD, Schaff HV & Saran N. Repair of Partial Atrioventricular Septal Defects in Adults: A Single Center Experience. Semin Thorac Cardiovasc Surg. 2021 Summer;33(2):469–478, used with permission of Mayo Foundation for Medical Education and Research, all rights reserved)

Tricuspid Valve Replacement

- More commonly performed for patients with marked abnormalities of the leaflets or subvalvular apparatus or massive right ventricular dilatation [12].

- Important to evaluate the annulus and right ventricle size to avoid patient-prosthesis mismatch with undersized valves.
- Poor right ventricular function and contractility cause prosthetic valve dysfunction with poor leaflet motion, leading to valve thrombosis.
- Valves are usually implanted with pledgeted mattress sutures. The native leaflets are typically left in place to preserve the subvalvular anatomy and avoid damage to the conduction system [12].
- Transvenous ventricular device leads are usually removed, and an epicardial lead should be placed at the end of the case.

Prosthesis Selection

Bioprosthetic valves are generally preferred in the tricuspid position due to the low velocity flow through the valve, low right-sided pressures, and often depressed right ventricular function. An additional benefit of bioprosthetic valves is the need for lower dose anticoagulation relative to mechanical valves. Porcine bioprostheses are preferred over pericardial bioprostheses because of their thin and pliable leaflets [12]. Mechanical valves are an option if right ventricular function is preserved and if a left-sided mechanical valve is present.

Postoperative Considerations

- Tricuspid valve replacement has a higher incidence of postoperative heart block and higher late mortality compared to tricuspid valve repair [13].
- Patients with symptoms of right heart failure preoperatively have longer postoperative hospital lengths of stay.
- Patients with preoperative right atrial enlargement are at increased risk of postoperative atrial fibrillation and low cardiac output syndrome.
- Risk factors for mortality include emergent operations, advanced age, male gender, higher numbers of concomitant procedures, longer cardiopulmonary bypass times, prior mitral valve surgery, and signs/symptoms of right heart failure, such as New York Heart Association Class III or greater [13–16].

References

1. Otto CM, Nishimura RA, Bonow RO, Carabello BA, Erwin JP 3rd, Gentile F, et al. 2020 ACC/AHA guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2021;143(5):e72–e227.

2. Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP 3rd, Guyton RA, et al. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation. 2014;129(23):2440–92.
3. McGrath LB, Gonzalez-Lavin L, Bailey BM, Grunkemeier GL, Fernandez J, Laub GW. Tricuspid valve operations in 530 patients. Twenty-five-year assessment of early and late phase events. J Thorac Cardiovasc Surg. 1990;99(1):124–33.
4. Calafiore AM, Gallina S, Iacò AL, Contini M, Bivona A, Gagliardi M, et al. Mitral valve surgery for functional mitral regurgitation: should moderate-or-more tricuspid regurgitation be treated? A propensity score analysis. Ann Thorac Surg. 2009;87(3):698–703.
5. Bianchi G, Solinas M, Bevilacqua S, Glauber M. Which patient undergoing mitral valve surgery should also have the tricuspid repair? Interact Cardiovasc Thorac Surg. 2009;9(6):1009–20.
6. Gammie JS, Chu MWA, Falk V, Overbey JR, Moskowitz AJ, Gillinov M, et al. Concomitant tricuspid repair in patients with degenerative mitral regurgitation. N Engl J Med. 2022;386(4):327–39.
7. Pahwa S, Saran N, Pochettino A, Schaff H, Stulak J, Greason K, et al. Outcomes of tricuspid valve surgery in patients with functional tricuspid regurgitation. Eur J Cardiothorac Surg. 2021;59(3):577–85.
8. Stout KK, Daniels CJ, Aboulhosn JA, Bozkurt B, Broberg CS, Colman JM, et al. 2018 AHA/ACC guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation. 2019;139(14):e698–800.
9. Saran N, Dearani JA. Tricuspid valve repair: how I teach it. Ann Thorac Surg. 2018;105(3):675–9.
10. Saran N, Dearani JA. Strategies for tricuspid valve repair. Indian J Thorac Cardiovasc Surg. 2020;36(Suppl 1):123–30.
11. Fiedler AG, Sullivan J, Walker JD. Tricuspid valve. In: Adult and pediatric cardiac surgery. STS cardiothoracic surgery e-book. Chicago, IL: Society of Thoracic Surgeons; 2020. Available from: https://www.sts.org/online-learning/sts-cardiothoracic-surgery-e-book.
12. Dearani JA. Editorial comment: lessons learned with tricuspid valve replacement. Eur J Cardiothorac Surg. 2014;45(1):90–1.
13. Saran N, Dearani JA, Said SM, Greason KL, Pochettino A, Stulak JM, et al. Long-term outcomes of patients undergoing tricuspid valve surgery. Eur J Cardiothorac Surg. 2019;56(5):950–8.
14. Patlolla SH, Schaff HV, Greason KL, Pochettino A, Daly RC, Frye RL, et al. Early right ventricular reverse remodeling predicts survival after isolated tricuspid valve surgery. Ann Thorac Surg. 2021;112(5):1402–9.
15. Shinn SH, Dayan V, Schaff HV, Dearani JA, Joyce LD, Lahr B, et al. Outcomes of ring versus suture annuloplasty for tricuspid valve repair in patients undergoing mitral valve surgery. J Thorac Cardiovasc Surg. 2016;152(2):406–15.
16. Guenther T, Noebauer C, Mazzitelli D, Busch R, Tassani-Prell P, Lange R. Tricuspid valve surgery: a thirty-year assessment of early and late outcome. Eur J Cardiothorac Surg. 2008;34(2):402–9.

Chapter 16
Pulmonary Valve Repair and Replacement

Elaine M. Griffeth and Joseph A. Dearani

Learning Objectives
- Basic introduction to valve pathologies
- Indications for repair vs. replacement
- Replacement technique
- Repair techniques
- Prosthesis selection
- Special circumstances

Introduction

In general, cardiac valvular pathology is characterized as regurgitant, stenotic, atretic, or displaced. Most cases of pulmonary valve pathology are either stenotic or regurgitant; this may be secondary to congenital heart disease, namely conotruncal anomalies, or pulmonary hypertension. Surgical treatment of pulmonary regurgitation is with valve replacement. In cases of pulmonary stenosis, patients can be treated with a variety of techniques based on the underlying anatomy including balloon valvuloplasty, surgical valvotomy, and replacement. Right ventricle to pulmonary artery conduits can be required for complex conotruncal anomalies. A very small number of congenital heart disease patients present with pulmonary atresia, which requires complex surgical management.

E. M. Griffeth · J. A. Dearani (✉)
Department of Cardiovascular Surgery, Mayo Clinic, Rochester, MN, USA
e-mail: griffeth.elaine@mayo.edu; jdearani@mayo.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_16

Anatomy

Embryology: The semilunar valves arise from the endocardial cushion. This embryonic structure also gives rise to the atrioventricular valves and atrial and ventricular septae.

The pulmonary valve is the right-sided semilunar valve and is composed of the annulus, three cusps (leaflets; anterior, left, right), and sinuses of Valsalva.

Please refer to Chap. 2 of this book for more detailed descriptions of valvular anatomy.

Pathology

Pulmonary Regurgitation and Stenosis

In general, pulmonary stenosis occurs in unrepaired congenital defects (e.g., isolated valvular pulmonary stenosis, conotruncal anomalies, pulmonary atresia) and pulmonary regurgitation occurs in previously repaired congenital defects. Pulmonary regurgitation is the most common late complication after repair of conotruncal defects.

Congenital

- Valvular pulmonary stenosis.
- Conotruncal anomalies.
  - Tetralogy of Fallot—anterosuperior displacement of the infundibular septum resulting in an overriding aorta and pulmonary stenosis; also have ventricular septal defects (VSDs) and right ventricular hypertrophy.

    Pulmonary atresia + VSD (extreme form of Tetralogy of Fallot): Failure of the pulmonary valve to form resulting in hypoplasia of the pulmonary vasculature; pulmonary blood flow can be ductal dependent or dependent on systemic to pulmonary artery collaterals (major aortopulmonary collateral arteries [MAPCAs]).
  - Double outlet right ventricle—both great arteries (aorta and pulmonary artery) arise from right ventricle, also have VSDs (most commonly subaortic).
  - Double chamber right ventricle—type of infundibular right ventricular outflow tract obstruction whereby a fibrous muscle band extends between the right ventricular cavity and infundibulum.
- Rubella infection in-utero.

Acquired

- Pulmonary hypertension—causes pulmonary regurgitation.
- Carcinoid heart disease—can cause pulmonary stenosis and/or regurgitation.
- Rheumatic heart disease—can cause pulmonary stenosis and/or regurgitation; valve damage is immune-mediated via a type II hypersensitivity reaction (not due to direct damage from bacteria).
- Endocarditis—can cause pulmonary stenosis and/or regurgitation.

History and Physical Exam

Patients may present with symptoms of right heart failure, which include fatigue, decreased appetite, ascites, peripheral edema, and low albumin. The provider should perform a physical exam, looking for signs of right heart failure and its potential causes such as left heart pathology, right sided valvular disease, or cor pulmonale.

Physical Exam Findings

- Neck: Jugular venous distension.
- Pulmonary: Signs of pulmonary disease in patients with cor pulmonale, such as increased thoracic anteroposterior diameter and expiratory wheezing in patients with chronic obstructive pulmonary disease (COPD).
- Cardiac:
  - Systolic murmurs:

    Pulmonary stenosis: mid-systolic, harsh ejection murmur.
  - Diastolic murmurs:

    Pulmonary regurgitation: early diastolic, high-pitched "blowing" murmur.
  - Wide splitting of S2 indicates pulmonary stenosis (delayed closure of pulmonary valve).
  - Right sided S3 "gallop" indicates right ventricular volume overload and right heart failure.
- Abdomen: Hepatomegaly, ascites.
- Extremities: Peripheral edema, especially in the ankles in ambulatory patients.

Symptoms or signs of right heart failure should raise concern for decompensated pulmonary regurgitation and associated tricuspid regurgitation. Due to increased risk for operative mortality, providers should consider admitting the patient preoperatively for medical optimization prior to surgical intervention.

Imaging

Echocardiography

- Transthoracic echocardiography (TTE): The main imaging modality used for evaluation of the pulmonary valve; allows for differentiation of causes of pulmonary valve pathology, assessment of the left heart, and measurement of right sided pressures.
- Transesophageal echocardiography (TEE): Used intraoperatively to assess the valve pre- and post-bypass.

Please refer to Chap. 4 for additional details on echocardiography.

Cross-Sectional Imaging

- Cardiac MRI: Used to assess the right ventricle size and function and preferred for chronic surveillance to minimize or avoid radiation exposures. The pulmonary valve is also assessed but echocardiography provides better valvular anatomic detail.
- CT angiography is preferred for planning (re)operation as it provides more detailed vascular anatomy, particularly coronary artery and great vessel anatomy (aorta, pulmonary artery, RV-PA conduit) and other structures that may be abutting the sternum and chest wall making re-entry hazardous.

Please refer to Chap. 5 for additional details on cardiac MRI.

Cardiac Catheterization

- Right heart catheterization: Used to assess hemodynamics and to evaluate the right heart in the case of discordant imaging and/or exam findings.

Please refer to Chap. 6 for additional details on cardiac catheterization.

In patients presenting with pulmonary regurgitation, always evaluate for left sided pathology and pulmonary hypertension, especially in the setting of structurally normal valves. It is important to also assess the tricuspid valve for regurgitation.

Indications for Intervention

In the setting of pulmonary stenosis or pulmonary regurgitation, treatment strategies may include percutaneous approaches, surgical approaches, and occasionally a hybrid approach.

Outcomes and survival are better if intervention is performed prior to the onset of right ventricular systolic dysfunction. End-organ damage such as liver or kidney failure markedly affects survival, and there is an increased risk of right ventricular failure after operation for patients with severe right ventricular systolic dysfunction or irreversible pulmonary hypertension preoperatively [1]. The above factors are important to consider when evaluating patients for potential intervention.

The indications for intervention are based on the severity of valvular disease and patient symptoms. Pulmonary regurgitation and pulmonary stenosis are categorized as severe (Stages C & D) based on echocardiographic evaluation [2].

Pulmonary Regurgitation

- Pulmonary valve replacement is recommended in symptomatic patients with moderate or greater pulmonary regurgitation resulting from previously treated isolated pulmonary stenosis with right ventricular dilatation or dysfunction. It can be considered in asymptomatic patients meeting the same criteria, especially in the setting of progressive right ventricular dilatation or dysfunction and/or a decrease in exercise capacity [3].

Pulmonary Stenosis

- In patients with moderate or severe isolated pulmonary stenosis, balloon valvuloplasty is a safe and effective treatment option [3].
- Surgical treatment is often recommended for adults with moderate or severe valvular pulmonary stenosis and otherwise unexplained symptoms of heart failure, cyanosis, and/or exercise intolerance that are either ineligible for or failed balloon valvuloplasty [3].

Congenital Heart Disease

- Valvular pulmonary stenosis

  - Balloon valvuloplasty is indicated in patients (usually infants and children) with signs/symptoms or imaging consistent with moderate or severe pulmonary stenosis.

- Conotruncal anomalies
  - Due to left to right shunting, repair is indicated early in life (by age 6 months) to prevent irreversible pulmonary vascular disease resulting in Eisenmenger physiology.
  - Neonates with inadequate pulmonary blood flow receive prostaglandin infusions to maintain ductal patency ± atrial balloon septostomy.

- Pulmonary atresia with VSD
  - Neonates are maintained on prostaglandin infusions to maintain ductal patency.
  - In cases of ductal-dependent pulmonary blood flow, surgical repair is indicated in the neonatal period with either complete repair or systemic to pulmonary shunt (e.g., Blalock-Taussig-Thomas shunt).
  - In cases of MAPCA-dependent pulmonary blood flow, the timing and staging of surgical repair is individualized based on underlying anatomy.

Prosthetic Pulmonary Valves

- Repeat pulmonary valve replacement is indicated in patients with symptomatic severe prosthetic valve stenosis or regurgitation. In patients with asymptomatic severe prosthetic valve regurgitation, percutaneous valve-in-valve therapy is applied if anatomy is appropriate or surgery can be considered and may be preferred if additional pathology is present that can also be treated (e.g., tricuspid regurgitation, residual ASD or VSD, etc.) [1].
  - If stenosis is attributable to thrombus, oral anticoagulation with a vitamin K antagonist is an appropriate first step.
  - If the prosthetic valve is biologic and the anatomic features are amenable to catheter-based valve-in-valve replacement, then percutaneous replacement is reasonable.

- Repeat pulmonary valve replacement is indicated in patients with intractable hemolysis or heart failure attributable to prosthetic transvalvular or paravalvular leak [1].
  - If the anatomic features of the paravalvular leak are amenable to catheter-based therapy, then percutaneous repair is reasonable in the absence of infection.

- Repeat pulmonary valve replacement is indicated in patients with infective endocarditis after initiation of intravenous antibiotics. Please see Chap. 17 for a full discussion on management of infective endocarditis.

Please see the Special Circumstances section at the end of this chapter for a discussion on right ventricle to pulmonary artery (RV-PA) conduits.

Pulmonary Valve Surgery

Operative Approach

- Cardiopulmonary bypass is used routinely for pulmonary valve surgery, and aortic cross clamping is used selectively depending on the presence of intracardiac shunts, other intracardiac pathology, or surgeon preference.
- Intraoperative transesophageal echocardiography is essential to assess the valve pre-bypass and to assess the adequacy of the replacement or right ventricular outflow tract intervention post-bypass.
- The pulmonary valve is approached via pulmonary arteriotomy and occasionally extending into the high right ventricular outflow tract or previous RVOT patch.
- Systematically assess the valve, branch pulmonary arteries, and RVOT: evaluate for supravalvar, valvar or subvalvar causes of stenosis.

Pulmonary Valve Replacement

- Pulmonary valve replacement is the mainstay of treatment for pulmonary regurgitation and is required for patients with pulmonary stenosis with marked abnormalities of the leaflets or significant annular hypoplasia.
- It is important to evaluate the annulus, right ventricular size, and main pulmonary artery to avoid patient-prosthesis mismatch with undersized valves. In the adult a 25, 27 or 29 mm stented bioprosthesis is utilized with pulmonary artery patch augmentation to accommodate the largest prosthesis.
- Prosthetic valves (homograft [unstented cadaveric tissue] and stented biologic [porcine or pericardial tissue]) are implanted using interrupted mattress sutures (occasionally continuous) at the level of or just distal to the annulus to avoid compression of the left coronary artery. The high right ventricular outflow tract can be augmented anteriorly with a transannular patch as needed (Fig. 16.1) [4].
  - Moderate oversizing can be beneficial with stented bioprostheses to allow for future percutaneous valve-in-valve replacement (Fig. 16.2).
  - In the case of pulmonary valve re-replacement, some residual sewing ring is often left in-situ posteriorly to facilitate subsequent suture placement and avoid excessive denuding of fragile pulmonary artery tissue. Stitches can be placed through the old sewing ring when sewing in the new valve.
  - Present data indicates no difference in durability between porcine and pericardial bioprostheses.

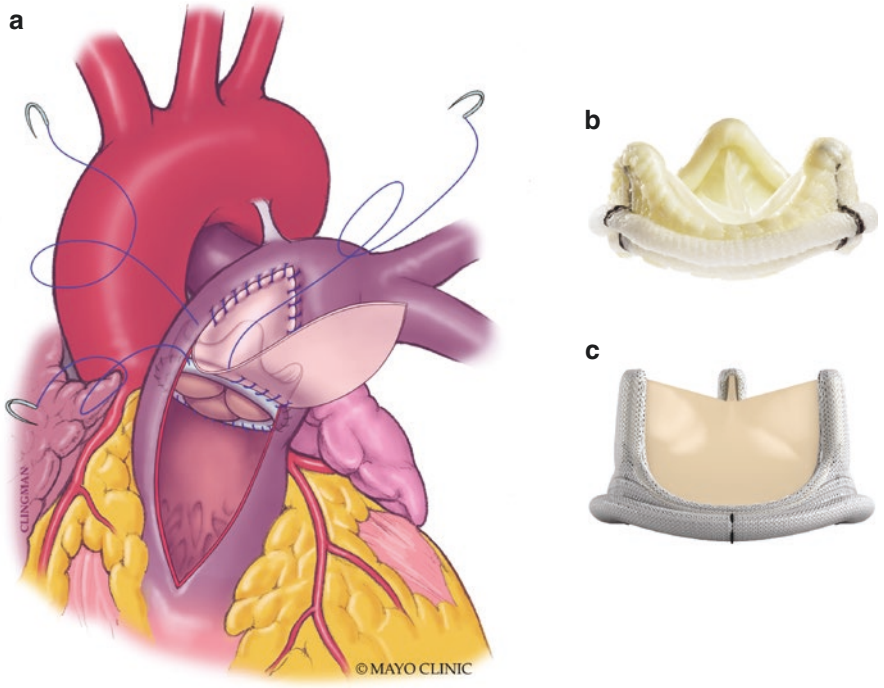


Fig. 16.1 Pulmonary valve replacement. (**a**) Pulmonary valve replacement with transannular patch augmentation to accommodate the largest size prosthesis possible. The incision in the right ventricle should be limited as much as possible (used with permission from Mayo Foundation for Medical Education and Research, all rights reserved). (**b**) Porcine bioprosthesis (image of Epic™ Stented Tissue Valve with Linx™ AC Technology used with permission from Abbott, St. Paul, MN). (**c**) Pericardial bioprosthesis (image of Carpentier-Edwards PERIMOUNT Magna Ease aortic heart valve used with permission from Edwards Lifesciences LLC, Irvine, CA. Disclaimer: Product not indicated for pulmonic use as per the IFU. Edwards, Edwards Lifesciences, Carpentier-Edwards, Magna, Magna Ease are trademarks of Edwards Lifesciences Corporation)

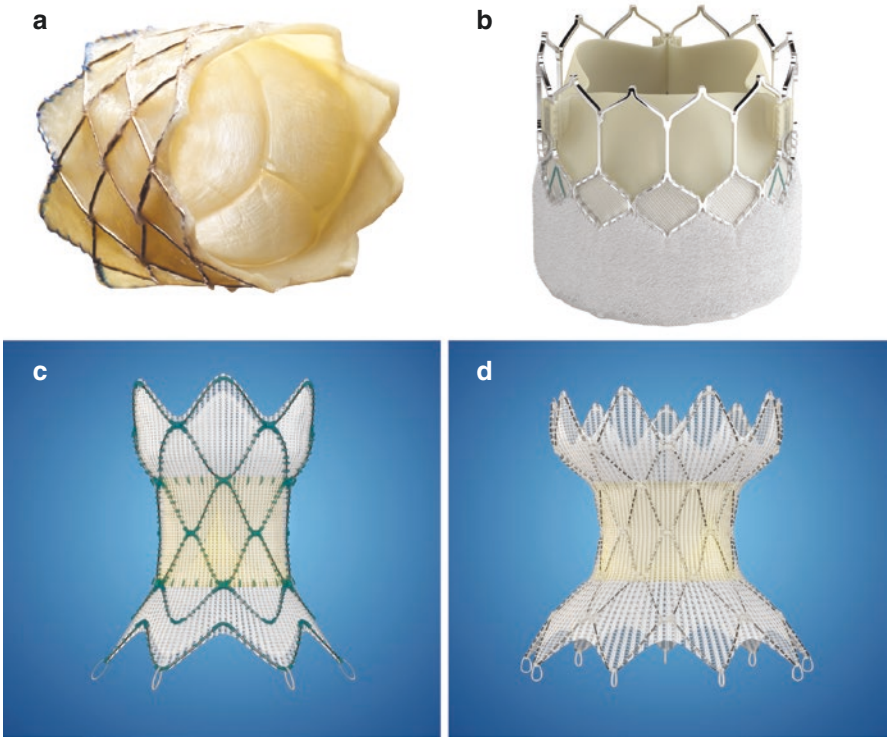


Fig. 16.2 Transcatheter pulmonary valve (TPV) replacement. (**a**) Melody™ TPV valve (reproduced with permission from Medtronic, Inc.); (**b**) SAPIEN 3™ valve (image of Edwards SAPIEN 3™ transcatheter heart valve used with permission from Edwards Lifesciences LLC, Irvine, CA. Edwards, Edwards Lifesciences, Edwards SAPIEN, SAPIEN, SAPIEN 3 are trademarks of Edwards Lifesciences Corporation); (**c**) Harmony™ 22 mm TPV valve (reproduced with permission from Medtronic, Inc.); (**d**) Harmony™ 25 mm TPV valve (reproduced with permission from Medtronic, Inc.)

Right Ventricular Outflow Tract Interventions for Pulmonary Stenosis

- Isolated valvular pulmonary stenosis: balloon valvuloplasty can be performed with successful relief of gradients and symptoms; if unsuccessful, then surgical valvotomy/commissurotomy can be performed [3]. It is typical for there to be some degree of pulmonary regurgitation to develop as a result of balloon valvuloplasty.
- Subvalvar/infundibular pulmonary stenosis: resection of muscle bundles/obstructive fibrous tissue with patch augmentation of right ventriculotomy (not a transannular patch).

- Supravalvar pulmonary stenosis: main pulmonary artery patch angioplasty.
- Severe multilevel pulmonary stenosis:
  - Adults should undergo pulmonary valve replacement with patch augmentation of the right ventricular outflow tract.
  - Pediatric patients undergoing repair of conotruncal anomalies may have transannular patch repair or insertion of RV-PA conduit performed initially; however, they must have long term follow-up because future reintervention for pulmonary regurgitation and/or for the conduit will be required.

    See Chap. 32 for a discussion on adult reoperative surgery for Tetralogy of Fallot, namely pulmonary regurgitation in the setting of transannular patch repair at the initial operation.

RV-PA conduits are covered in a separate section at the end of this chapter.

Congenital Heart Disease

- Conotruncal anomalies: Surgery focuses on complete anatomic repair with establishment of right ventricle to pulmonary artery continuity with relief of pulmonary stenosis and closure of the VSD; there are numerous techniques utilized for these purposes.
- Pulmonary atresia: Surgical repair involves establishing right ventricle to pulmonary artery continuity, patch augmentation of the main and branch pulmonary arteries when hypoplastic, and unifocalization of MAPCAs when present.

Prosthesis Selection

Bioprosthetic valves are preferred in the pulmonary position because of good durability and the lack of need for chronic anticoagulation. Stented bioprosthetic valves provide a landing zone for transcatheter valves if re-replacement becomes necessary later since coronary artery compression can be avoided. Homograft valves are another fully biologic option; they are often preferred in children because of ease with implantation but are frequently avoided in adults with certain congenital abnormalities because of inferior durability compared to stented bioprosthetic valves. Mechanical valves are rarely used in the pulmonary position, but situations where their application is considered include cases of severe pulmonary hypertension, patients who have had multiple failed bioprosthetic valves, and patients who require chronic anticoagulation for left sided prosthetic valves [4].

Postoperative Considerations

- Patients with symptoms of right heart failure preoperatively have longer postoperative hospital lengths of stay.
- Patients with preoperative right atrial enlargement are at increased risk of postoperative atrial fibrillation and low cardiac output syndrome.

Right Ventricle to Pulmonary Artery Conduits

RV-PA conduits are extra-cardiac conduits used to establish right ventricle to pulmonary artery continuity in patients with congenital heart defects that preclude the ability to perform valve replacement with right ventricular outflow tract reconstruction. Reasons for this can be pulmonary atresia, annular hypoplasia, pulmonary artery hypoplasia (especially long-segment), severe multi-level pulmonary stenosis, or the presence of an anomalous left anterior descending crossing the RVOT. Some of the congenital cardiac diagnoses mentioned earlier in the chapter can all potentially require RV-PA conduits for repair, but in general, conduits are avoided if PVR can be performed in the native pulmonary artery and RVOT. Valved conduits are best because they maintain pulmonary valve competency and protect the right ventricle from progressive dilatation and dysfunction due to volume overload.

Challenges associated with RV-PA conduits include the inability of the conduit to grow with patients over time and the need for reoperation due to development of stenosis and/or regurgitation. Reoperation in these patients can be difficult because of the risk of hazardous sternal re-entry due to scarring of the conduit to the underside of the sternum that generally resides close to the midline.

Commonly Used Types of RV-PA Conduits (Fig. 16.3)

- Pulmonary homograft—Fig. 16.3a; cryopreserved cadaveric tissue, includes pulmonary valve and main pulmonary artery; used in pediatric patients and adult patients undergoing the Ross procedure.
- Aortic homograft—Fig. 16.3b; cryopreserved cadaveric tissue, includes aortic valve and ascending aorta and a portion of the arch; more commonly used in neonates and infants since the arc of the aortic homograft lays nicely and reaches the pulmonary confluence.
- Hancock™—Fig. 16.3c; porcine valve in woven Dacron (fabric) conduit; used in older children and adult patients.
- Contegra™—Fig. 16.3d; bovine jugular venous valved conduit (valve is trileaflet); used in pediatric patients.

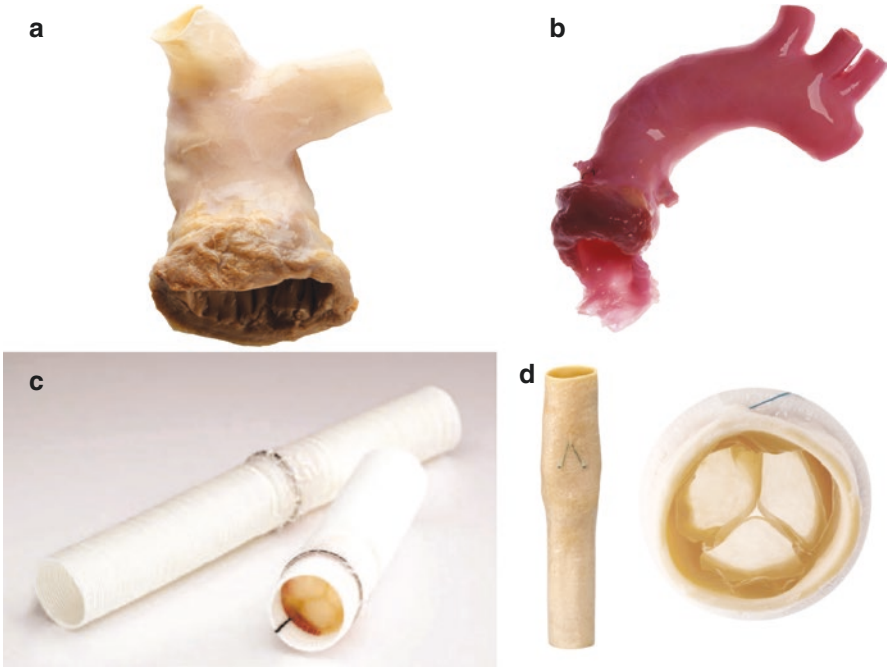


Fig. 16.3 Right ventricle to pulmonary artery conduits. (**a**) Pulmonary homograft (image of CryoValve SG Pulmonary Human Heart Valve used with permission from Artivion, Inc.); (**b**) aortic homograft (image of CryoLife Aortic Homograft Valve used with permission from Artivion, Inc.); (**c**) Hancock™ conduit (reproduced with permission from Medtronic, Inc.); (**d**) Contegra™ conduit (reproduced with permission from Medtronic, Inc.)

Special Circumstances

Some additional congenital cardiac defects with pulmonary valve pathology are listed below.

- Pulmonary atresia with intact ventricular septum: Failure of the pulmonary valve to form resulting in hypoplasia of the pulmonary vasculature; ASD required for postnatal viability; pulmonary blood flow can be ductal dependent or dependent on systemic to pulmonary artery collaterals (major aortopulmonary collateral arteries [MAPCAs]).
- Transposition of the great arteries (TGA)—reversed anatomic relationship of the great arteries whereby the aorta arises anterior to the pulmonary artery and from the right ventricle; associated with VSD.
  - Dextro "D"-looping: normal atrioventricular relationship but discordant ventriculoarterial relationship (i.e., right atrium receives systemic venous return and empties into right ventricle via tricuspid valve which ejects into aorta).

  - Levo "L"-looping (a.k.a. congenitally corrected TGA): Discordant atrioventricular *and* ventriculoarterial relationships (i.e., right atrium receives systemic venous return and empties into left ventricle via mitral valve which ejects into pulmonary artery).

- Truncus arteriosus—single arterial trunk (common arterial origin of aorta and pulmonary artery) overriding the interventricular septum and a VSD, has a single truncal valve; associated with right aortic arch and anomalous coronary arteries.

References

1. Otto CM, Nishimura RA, Bonow RO, Carabello BA, Erwin JP 3rd, Gentile F, et al. 2020 ACC/AHA guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2021;143(5):e72–e227.
2. Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP 3rd, Guyton RA, et al. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation. 2014;129(23):2440–92.
3. Stout KK, Daniels CJ, Aboulhosn JA, Bozkurt B, Broberg CS, Colman JM, et al. 2018 AHA/ACC guideline for the management of adults with congenital heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation. 2019;139(14):e698–800.
4. Dugan ME, Tweddell JS. Pulmonary valve replacement. In: Adult and pediatric cardiac surgery: STS cardiothoracic surgery E-book. Chicago, IL: Society of Thoracic Surgeons; 2020. Available from: https://www.sts.org/online-learning/sts-cardiothoracic-surgery-e-book.

Chapter 17
Transcatheter Therapies for Structural Heart Disease

William Shi and Tsuyoshi Kaneko

Overview

The term "structural heart disease" originally referred to non-coronary interventional procedures such as valve, atrial, or ventricular septal procedures. However, in the past 10–15 years, with the invention and maturation of transcatheter aortic valve replacement (TAVR) and mitral transcatheter edge-to-edge repair (M-TEER), it mainly encompasses the transcatheter management of valvular heart disease, especially for surgeons. These modern transcatheter devices have been investigated thoroughly via major multicenter clinical trials, with further understanding of the clinical outcomes of open valve surgery as a control group [1–5]. The increasing adoption of transcatheter therapies has been fueled by their appeal to patients, given they represent a less invasive treatment modality, allowing shorter hospital stays and faster return to activities. In this chapter, we will discuss transcatheter therapies based on pathology with a specific focus on TAVR.

W. Shi (✉)
Department of Cardiovascular and Thoracic Surgery, Northwell Health System, New York, NY, USA
e-mail: wshi@northwell.edu

T. Kaneko
Department of Cardiovascular and Thoracic Surgery, Northwell Health System, New York, NY, USA

Division of Cardiac Surgery, Barnes Jewish Hospital, Washington University in St Louis, St Louis, MO, USA
e-mail: kaneko@wustl.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_17

Structural Heart Pathologies Treated with Transcatheter Therapies

Aortic Valve Disease

TAVR for aortic stenosis (AS) is the most commonly treated condition in structural heart disease in the United States. Given that AS is common among older patients, TAVR represents an appealing option as it enables valve replacement while avoiding the invasiveness of traditional surgical AVR (SAVR). During TAVR, a bioprosthetic valve is deployed inside the native aortic valve, pushing the native leaflets aside. The presence of aortic valve leaflet calcification is important, as this anchors the TAVR prosthesis to the calcium at deployment. The two valve types currently widely used are the balloon-expandable Sapien (Edwards Lifesciences, Irvine, CA) and self-expanding Corevalve Evolut (Medtronic, Minneapolis, MN) (Fig. 17.1a, b). In patients with isolated aortic insufficiency (AI), TAVR with currently available devices is problematic, as the absence of valve calcification commonly seen in these patients makes valve migration a serious concern. Newer TAVR devices specifically designed for AI are being investigated but are currently not widely available commercially, and TAVR in these patients is currently considered on a case-by-case basis by heart teams.

Aortic Valve Bioprosthetic Failure

Patients who have previously undergone a bioprosthetic aortic valve replacement may present with structural valve degeneration of their prosthesis, resulting in either stenosis or regurgitation. These patients may be suitable for valve-in-valve TAVR, whereby a TAVR valve is deployed within the failed surgical prosthesis (Fig. 17.2). In some cases, the sewing ring of the bioprosthesis may be fractured using an aortic valvuloplasty balloon to facilitate the TAVR valves full expansion to achieve better hemodynamics of the valve [6].

Mitral Valve Disease

Mitral valve regurgitation (MR)—both degenerative (primary) and functional (secondary)—may be managed using transcatheter devices using a method known as TEER. The concept is similar to the edge-to-edge technique (Alfieri suture) in open surgery, which is a repair technique that sutures the anterior and posterior leaflet to create double orifice. In TEER, a clip is placed to replicate this method, thus changing the mitral valve from a single to double orifice configuration (Fig. 17.3). Multiple clips may be placed depending on the anatomy. Care must be taken so as not to create iatrogenic mitral stenosis by overly reducing the valve's orifice area. Currently,

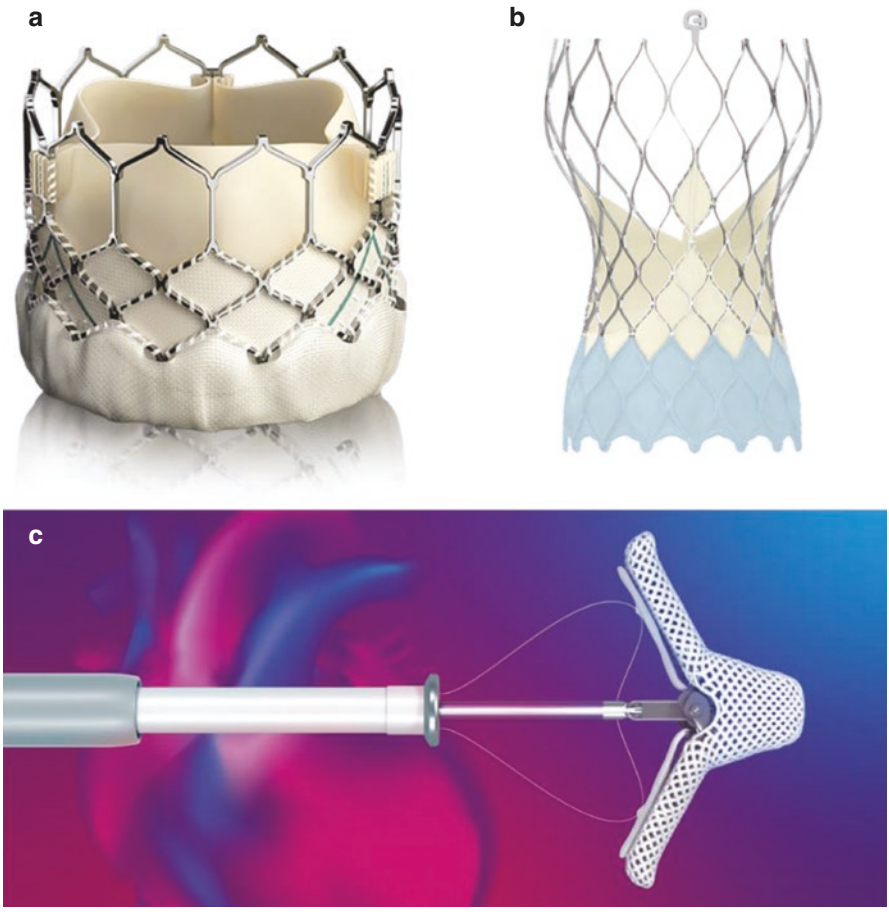


Fig. 17.1 (**a**) Edwards Lifesciences Sapien 3 prosthesis (source: Edwards Lifesciences), (**b**) Medtronic Corevalve Evolut prosthesis (source: Medtronic). (**c**) Abbott Mitraclip (source: Abbott Laboratories)

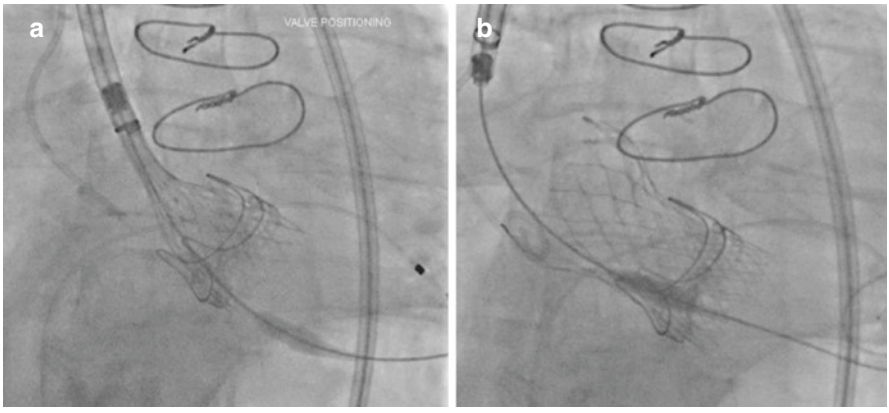


Fig. 17.2 Fluoroscopic images depicting valve-in-valve TAVR. In (**a**), a partially deployed self-expanding Corevalve prosthesis is positioned inside a degenerated bioprosthesis. (**b**) Full deployment of the Corevalve

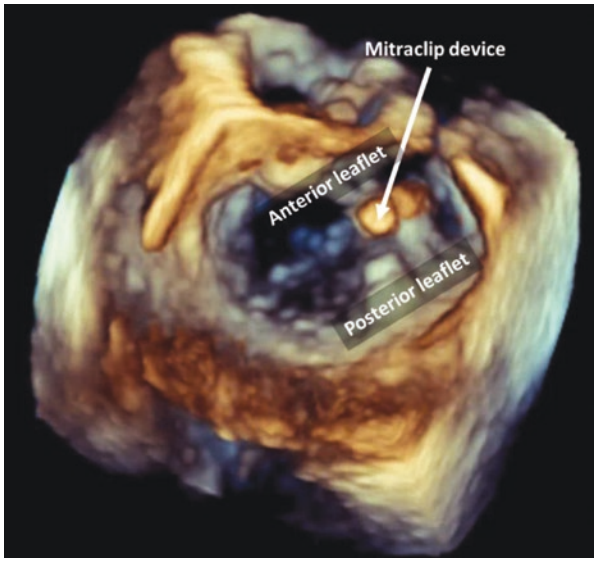


Fig. 17.3 3D TEE image demonstrating deployed Mitraclip device opposing the anterior and posterior mitral leaflets creating a double orifice mitral valve, as seen from the atrial side of the valve

the Mitraclip (Abbott, Chicago, IL) is the only FDA-approved device for TEER (Fig. 17.1c).

The EVEREST II trial compared M-TEER with the Mitraclip device to open surgery for degenerative MR and found M-TEER to be associated with higher rates of >mild MR compared to surgery (M-TEER 12.3% vs. surgery: 1.8%) at 5 years. The trial however did not detect a survival difference between the two strategies at 5 years follow-up. A major randomized clinical trial investigation mitral TEER (M-TEER) for functional MR was the COAPT trial, conducted at 78 sites in the US and Canada. It demonstrated that M-TEER with the Mitraclip device resulted in a reduced likelihood of hospitalization for heart failure (M-TEER: 35.8% per patient year vs. medical therapy: 67.9% per patient year within 2 years) and mortality (M-TEER: 29.1% vs. medical therapy: 46.1% at 2 years). M-TEER was initially approved by the FDA for high-risk patients with degenerative MR, but more recently has been approved for use in functional MR.

Complications of mitral and tricuspid TEER include injury to the subvalvular apparatus. This can occur when the clip becomes entangled in chorda tendinae as it is advanced toward the ventricle prior to leaflet grasping. Single-leaflet clip detachment and clip embolization can also occur, though the rates of such are low (<5%). As the femoral vein is accessed, major access site bleeding is uncommon (2–3%).

Mitral stenosis (MS) has for many years been managed with percutaneous mitral balloon commissuroplasty with reasonable results, though this procedure remains relatively uncommonly performed in the western world given the lower prevalence of rheumatic heart disease.

Transcatheter mitral valve replacement (TMVR) has been slower to be developed compared to TAVR, owing to greater challenges such as valve anchoring mechanisms and the mitral valve's relationship with the left ventricular outflow tract (LVOT).

There are multiple devices currently under clinical trial for TMVR. For patients with a failing mitral bioprosthesis or a failed mitral valve repair which utilized a complete mitral annuloplasty ring, a ViV/valve-in-ring TMVR may be performed whereby a balloon-expanding TAVR valve is deployed inside the failing mitral valve.

In some patients with severe mitral annular calcification, a balloon-expandable aortic valve which is usually used during TAVR can also be deployed in the mitral position, whereby the calcium deposits in the annulus hold the valve in place. While the former is FDA approved, the valve in mitral annular calcification carries significant risk and is being actively researched. For patients undergoing TMVR, the most serious complication is LVOT obstruction. Additional risks include PVL, cardiac injury, and valve embolization [5].

Tricuspid Valve Disease

Devices for managing tricuspid valve regurgitation are currently under clinical trial investigation. Devices exist both to replace valves as well as to perform tricuspid TEER. More data is showing good outcomes in a disease population that does not tolerate surgery well, mainly due to right ventricular dysfunction.

Pulmonary Valve Disease

Pulmonary regurgitation or stenosis is almost always encountered in patients who have previously undergone surgery for congenital disease. Transcatheter pulmonary valve replacement (TPVR) is typically accomplished currently using either a balloon-expandable Sapien valve or the Melody® valve (Medtronic Medtronic, Minneapolis, MN), a trileaflet bovine jugular venous valve 18 mm in diameter, mounted on a stent frame. Once positioned, a balloon is used to inflate the valve to deploy it in the pulmonary position. Most of the experience in placing the Melody valve has been for patients with a previously placed right ventricle to pulmonary artery conduit. There is growing interest in applying the valve to those with a native right ventricular outflow tract (RVOT), which has to date been challenging due to the typically larger size of the native RVOT after congenital repair.

Atrial Septal Defect, Ventricular Septal Defect, and Paravalvular Leaks

Other structural heart diseases that are amenable to transcatheter treatment include atrial/ventricular septal defect and paravalvular leaks. These defects can be closed using transcatheter occluder devices, provided they are of suitable size

and have an adequate rim of tissue to support the device. These occluder devices—are shaped like a disc or double-disc—are available in multiple different sizes and are commonly made of nitinol mesh. They are compressed and introduced into the body via a delivery device and once positioned across the septal defect, they are unsheathed and self-expand, covering the defect. Devices with a double-disc orientation are designed such that one disc sits on the left heart side of the defect, while the other disc lies on the right. Closure of ASD is contraindicated some patients with severe impairment of left ventricular function and advanced pulmonary hypertension. In the former, the presence of a left to right shunt serves to offload the left ventricle. Likewise, in advanced pulmonary hypertension, a right to left shunt offloads the right ventricle. Closure of ASDs in these situations may raise ventricular end-diastolic pressure to a point which may precipitate cardiac failure. Percutaneous closure of ASD is also contraindicated if there is an inadequate rim of tissue on which to anchor the occluder device. In such cases, surgical repair may be required. PVLs from previously implanted surgical valves can also be closed using these occluder devices.

Treatment Options and Considerations in TAVR

Patient Selection: SAVR vs. TAVR

Historically, TAVR was reserved for patients who were deemed to be of high or prohibitive risk with open surgery [3]. However, as the use of TAVR has expanded to younger and healthier patients, the decision of which modality to offer patients has become more complex. At many structural heart programs, patients are evaluated by a cardiologist and cardiac surgeon, as part of multidisciplinary structural heart team. The other members of the heart team include cardiac imaging specialists, radiologists, heart failure specialists, cardiac anesthesiologists, intensivists, nurses, social workers as well as clinical trial coordinators and research staff. The heart team, after multidisciplinary discussion, will make a decision to which modality the patient is best suited for [7].

In general, the decision to proceed with TAVR vs. SAVR hinges on two aspects, the patient factor, and the anatomical factor. Patient factor largely depends on patients' age, surgical risk, life expectancy, and frailty. The Society of Thoracic Surgeons (STS) risk calculator is commonly used to stratify patients' surgical risk (low risk: <4%, intermediate risk: 4–8%, high risk: >8%). While frailty can be judged through clinical assessment, various frailty assessment calculators are also available and may facilitate decision-making.

Anatomical factors include patients who have unfavorable femoral artery anatomy, since alternative-access TAVR has less favorable outcomes compared to those receiving the transfemoral approach. In addition, certain valve morphological features—such as BAV and heavy LVOT calcification—make TAVR higher risk. As such, these patients may be better served with traditional SAVR provided their surgical risk profile is acceptable.

For prohibitive or high-risk patients, TAVR is usually recommended. In intermediate and low surgical-risk patients, TAVR is generally preferred over SAVR for those >80 years of age, or for younger patients with a life expectancy <10 years. As the longevity of TAVR remains somewhat unknown, SAVR remains preferred by most centers for patients under 65 years of age. There is controversy as to the optimal approach in the 65–80 years old age-group. Here, a shared decision-making process is required between physicians and patients. Patients' values and individuals' preferences should be considered, and those who are willing to accept uncertainty about valve durability, a higher risk of receiving a permanent pacemaker and prefer a shorter hospital stay and less post-procedural discomfort may be better suited for TAVR over SAVR.

Prosthesis

Once the decision for TAVR is made, a key consideration is the type of TAVR valve to use. The Sapien (balloon-expanding) and Corevalve Evolut (self-expanding) are the two most commonly used TAVR prostheses. The Sapien valve is deployed by inflating a balloon mounted inside the valve in order to expand it, resulting in a deployment inside the native annulus. This configuration allows only one attempt at valve deployment. For the Corevalve device, the valve is located inside a capsule, which, when released, enables the valve to expand and oppose the aortic annulus without the use of a balloon (Fig. 17.2). This configuration permits recapturing of the valve and repositioning before final deployment. The Corevalve stent frame is made of nitinol— a metal alloy of nickel and titanium—which gives it "shape memory" properties such that it may be deformed outside the body and then recover to its original shape at the temperature inside the body. The decision of which prosthesis to utilize depends on patient factors as well as clinicians' preferences. In patients with a small aortic annulus, the Corevalve may be preferred as the valve's supra-annular position may provide a greater orifice area and improved hemodynamics compared to the Sapien's intra-annular position. On the other hand, the Corevalve's higher position in the aortic root may impair the ability to obtain coronary access for future coronary procedures, which should be factored into decision-making. Newer TAVR prostheses are under clinical trial and may become more commonplace in the future.

Access

One of the most important considerations in TAVR is the access site. Depending on the valve size and access location, the sheath sizes for TAVR range from 14 French to 21 French. As such, arteries must be between 5 and 7 mm in diameter in order to accommodate these. The most common access site for TAVR is via the common femoral artery. Femoral arteries are generally of adequate size, are located relatively superficially and in most cases, provide a relatively straight and unobstructed path toward the aortic valve. In most TAVR programs, 90–95% of TAVRs are generally performed via this route.

In patients whose femoral arteries are not suitable for TAVR, which is commonly due to severe peripheral artery disease, an alternative-access approach is required. The most commonly utilized alternative-access strategies are trans-carotid, trans-axillary, trans-caval, transapical, and transaortic. In trans-carotid and trans-axillary, the valve is introduced via the right carotid and left axillary arteries, respectively. In trans-caval, the valve is delivered via the common femoral vein into the inferior vena cava (IVC). A puncture is made between the IVC and descending or abdominal aorta to allow the valve to pass through this defect, into the aorta and toward the aortic valve. An occluder device is then used to close this defect between the aorta and IVC upon completion of the procedure. In transapical, a small left anterior thoracotomy is performed, exposing the left ventricular apex, through which the valve is delivered. In transaortic, a full or partial sternotomy is performed to gain access to the ascending aorta through which a TAVR is performed. Although transapical and transaortic used to consist the majority of alternative access, these are rarely performed due to the poor outcomes for these patients.

Imaging Evaluation in TAVR

The TAVR computed tomography (CT) represents the most important investigation for TAVR planning. Each center has a specific protocol for a TAVR CT, which images not only the chest but also the abdomen, pelvis, and the proximal lower limbs. The scans are ECG-gated to the cardiac cycle to enhance resolution.

Access

The TAVR CT permits the assessment and determination of the access route. The vessel diameter, presence of calcifications, stenoses, location of major branches, and tortuosity help to inform the feasibility of transfemoral access. If transfemoral is not feasible, then the CT enables the selection of an alternate access route (Fig. 17.4).

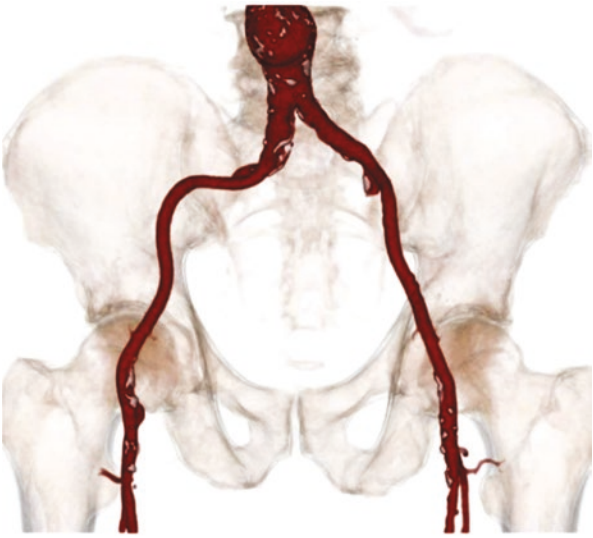


Fig. 17.4 3D reconstruction of the iliofemoral arterial system demonstrating favorable anatomy for transfemoral TAVR

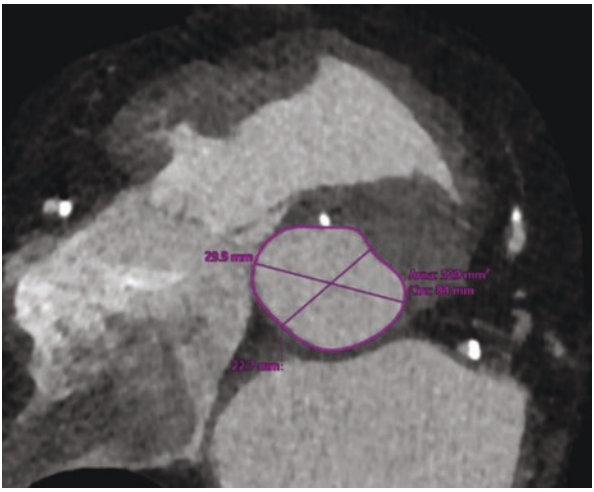


Fig. 17.5 Sizing of the aortic annulus using cardiac CT. TAVR valves are sized based on either the annular area (Sapien) or circumference (Corevalve)

Valve Morphology and Sizing

The selection of TAVR valve size is made based on CT measurements of the native aortic annulus area or perimeter based on advanced reconstruction (Fig. 17.5). The annulus to coronary artery ostia height, sinus of Valsalva diameter, and native valve leaflet length are measured, as these influence the risk of coronary obstruction by the native valve leaflets during TAVR. TAVR CTs will also reveal the presence of coronary calcification or stenoses and the presence of LVOT or aortic calcification that can also influence management. The TAVR CT also provides an estimation of the optimal image intensifier angles to be used to aid valve deployment during TAVR.

Basic Steps of Transfemoral TAVR

The sequence of steps for a transfemoral TAVR can be conceptualized as belonging to three phases: (1) access/preparation, (2) valve-crossing and deployment, and (3) closure. The sequence varies between operators, institutions, and valve prostheses. Below, the steps for placing a Sapien TAVR—a device commonly used at our institution—are summarized.

Access and Preparation

- Obtain US guided access (6 French catheters) of the right common femoral artery (CFA) for the valve delivery sheath and valve, left CFA for the imaging pigtail catheter, and left common femoral vein (CFV) for the temporary transvenous pacemaker.
- Utilize a percutaneous closure device to prepare the femoral artery for closure (Preclose method).
- Dilation and insertion of the large-bore device sheath into the right CFA (14–16 French).
- Heparinize to achieve an ACT >250 s.
- Insert imaging pigtail into the aortic root and connect a contrast injector.
- Perform root aortography and determine the optimal angle of deployment (commonly, a co-planar angle will be utilized whereby non-coronary, right coronary, and left coronary cusps are viewed in line) (Fig. 17.6).
- Insert the temporary transvenous pacing wire via the left CFV into the right ventricular apex and confirm pacing capture and thresholds.

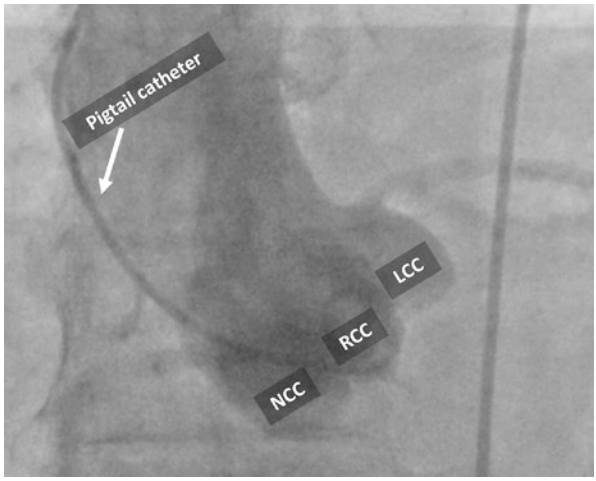


Fig. 17.6 Root aortography demonstrating co-planar alignment of the non-coronary cusp (NCC), right coronary cusp (RCC), and left coronary cusp (LCC) of the aortic valve

Valve-Crossing and Deployment

- Cross the aortic valve utilizing a catheter and guidewire from the right CFA. Once the valve is crossed, a pigtail catheter is placed over the guidewire into the LV apex.
- Perform aortic valve gradient measurements via the right CFA pigtail catheter (in the LV apex) and the left CFA pigtail (in the aortic root) to confirm the presence of severe AS. If the gradient demonstrated very severe AS (>50 mmHg), balloon aortic valvuloplasty can be considered so as to predilate the valve to aid TAVR valve expansion.
- Place a stiff wire (i.e., Safari, Lunderquist) via the right CFA sheath into the LV apex. A stiff wire enables safer passage of a large-bore device such as the valve and delivery system.
- The Sapien valve is prepared and loaded onto the stiff wire and inserted.
- Once the valve is advanced into the descending thoracic aorta, the balloon which expands the valve is then loaded inside the balloon under fluoroscopic vision.
- The valve is advanced into the native aortic valve, and its position is confirmed with fluoroscopy and aortography.
- Once the valve position is deemed satisfactory, rapid pacing is commenced at 180 bpm. Rapid pacing is performed to temporarily reduce cardiac output and minimize the risk that cardiac ejection may cause valve embolization during deployment. Root aortography is performed once again during pacing to confirm correct positioning and the valve. Balloon is inflated, and the valve is deployed (Fig. 17.7).
- The delivery system is retracted out of the body.
- A pigtail catheter is reintroduced into the LV to measure aortic valve gradients post deployment.

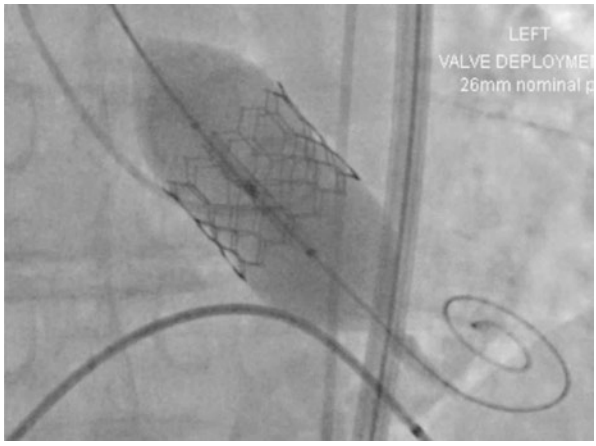


Fig. 17.7 Deployment of the balloon-expanding Sapien prosthesis in the aortic annulus

- A focused transthoracic echocardiogram is performed to examine for PVL, aortic valve gradient, cardiac function, and the presence of pericardial effusion that might suggest cardiac injury and bleeding.
- If there is a concern for PVL, or valve under-expansion causing a residual AV gradient, balloon dilatation of the TAVR valve can be performed here.

Closure

- The right CFA sheath is removed, and the percutaneous closure devices are deployed and secured.
- Angiography of the iliofemoral arterial system can be performed via the left CFA pigtail catheter to confirm vessel patency, with any bleeding reflected by contrast extravasation during angiography.
- The left CFA and CFV catheters and sheaths are removed.
- Lower limb pulses are assessed manually and with a doppler to ensure limb adequate perfusion.

Complications in TAVR

Like in open surgery, the risks of TAVR are dependent on patients' clinical and anatomical factors. In general, the risk of mortality after transfemoral TAVR is approximately 1%. The risk of stroke is also approximately 1%. The risk of a major vascular complication is around 5%. One disadvantage of TAVR is the heightened risk of requiring a permanent pacemaker which is around 5–10%. This risk is heightened in those with pre-existing conduction deficits such as right bundle branch block (RBBB). Fortunately, the risk of annular rupture and aortic injury with the latest generation of TAVR devices is low at <1%.

Unlike in SAVR, TAVR valves are not sewn into the annulus, and as such, there is an inherently increased risk of paravalvular leakage (PVL), though later-generation devices have served to reduce these due to engineering improvements. Patients with bicuspid aortic valve (BAV) stenosis pose a unique challenge, as these valves tend to be more calcified and asymmetrical and in some cases, may limit the effective expansion and sealing of a TAVR prosthesis, leading to residual gradients and PVL. With the latest generation of TAVR valves, the risk of >mild PVL is approximately 5%. Most patients can be discharged on post-operative day 1 after TAVR.

Conclusions

The rise of transcatheter valve technology has changed the way structural heart disease is treated. TAVR has become a popular, non-invasive way to manage aortic stenosis, while the indications for transcatheter mitral and tricuspid procedures also continue to grow. An appreciation of the full spectrum of treatment options for structural heart disease ensures the utmost inpatient care.

References

1. Otto C, Nishimura R, Bonow R, Carabello B, Erwin J Jr, Gentile F, et al. ACC/AHA guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2020;2021(143):e72–227.
2. Carroll J, Mack M, Vemulapalli S, Herrmann H, Gleason T, Hanzel G, et al. STS-ACC TVT registry of transcatheter aortic valve replacement. J Am Coll Cardiol. 2020;76:2492–516.
3. Smith C, Leon M, Mack M, Miller D, Moses J, Svensson L, et al. Transcatheter versus surgical aortic-valve replacement in high-risk patients. N Engl J Med. 2011;364:2187–98.
4. Mack M, Leon M, Thourani V, Makkar R, Kodali S, Russo M, et al. Transcatheter aortic-valve replacement with a balloon-expandable valve in low-risk patients. N Engl J Med. 2019;380:1695–705.
5. Stone G, Lindenfeld J, Abraham WT, Kar S, Lim D, Mishell J, et al. Transcatheter mitral valve repair in patients with heart failure. N Engl J Med. 2018;379:2307–18.
6. Bleiziffer S, Simonato M, Webb J, Rodés-Cabau J, Pibarot P, Kornowski R, et al. Long-term outcomes after transcatheter aortic valve implantation in failed bioprosthetic valves. Eur Heart J. 2020;41:2731–42.
7. Coylewright M, Mack M, Holmes DJ, O'Gara P. A call for an evidence-based approach to the Heart Team for patients with severe aortic stenosis. J Am Coll Cardiol. 2015;65:1472–80.

Chapter 18
Management of Endocarditis

Orit Abrahim, Sary Aranki, and Ashraf A. Sabe

Overview

- Infective endocarditis (IE) occurs when bacteria or fungi in the bloodstream infect the inner lining of the heart (endocardium), typically of a heart valve [1]. It is a relatively rare disease with 2–10 cases per 100,000 people globally [2].
- Risk factors for endocarditis include age greater than 60 years, male sex, structural heart disease (valvular and congenital), prosthetic valves, intravenous drug users (IVDU), chronic hemodialysis, intravascular catheters, indwelling cardiac devices, skin infection, and oral/dental hygiene pathology [1].
- In-hospital mortality remains high, up to 40% in 1 year, with poor outcomes related to left-sided or paravalvular involvement, prosthetic valve endocarditis, multiple comorbidities, CNS involvement, advanced age, and *Staphylococcal* or fungal infections [2].

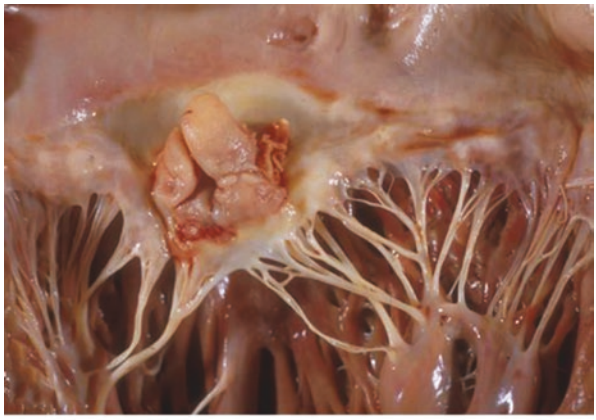
O. Abrahim (✉)
Department of Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA
e-mail: oabrahim@mgb.org

S. Aranki · A. A. Sabe
Department of Surgery, Cardiac Surgery, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA
e-mail: saranki@bwh.harvard.edu; asabe@bwh.harvard.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_18

Common Organisms and Etiology

- The development of IE begins with endothelial damage, followed by the introduction of a pathogen into the bloodstream, adherence of the pathogen to the endothelium, and proliferation of the pathogen.
- Valves may be damaged mechanically (e.g., catheters or electrodes) or by autoantibodies, inflammation, or the pathogen itself. Valves with compromised integrity may be due to such things as mitral valve prolapse, rheumatic heart disease, congenital cardiac malformations, or prosthetic valves.
- Once organisms are able to proliferate, vegetations may form which are infectious mass composed of fibrin, platelets, red and white cells, and bacteria (Fig. 18.1).
- Up to 40–50% of IE cases are caused by *Staphylococcal aureus* [3]. Other common organisms include Viridans group streptococci and enterococci in those with native heart valves. Coagulase-negative staphylococci are common among those with prosthetic valves and cardiac devices. Rarely, endocarditis can be caused by gram-negative bacteria (e.g., HACEK* organisms) and fungus.
- *S. aureus* is the predominant organism responsible for endocarditis among IVDU though at higher proportions nearing 70%, partly attributed to higher rates of colonization [2]. HACEK organisms, polymicrobial infections, and fungus are also more common in these populations.



Source: Lawrence H. Cohn, David H. Adams:
Cardiac Surgery in the Adult, Fifth Edition

Fig. 18.1 Endocarditis of the mitral valve, anterior leaflet

Clinical Presentation

- The clinical manifestations of endocarditis are broad and multisystem. Fever remains the most common symptom (90%) [4].
- From a cardiac perspective, there may be valvular incompetence from direct destruction; rupture of papillary muscles and ventricular walls; pericarditis; myocardial abscess formation; or complete heart block. Cardiac murmurs may not always be heard.
- Septic emboli from vegetations may occur resulting in infarctions or abscess formation in any organ, including the brain, kidney, spleen, mesentery, lung, eyes, and skin. Renal involvement is often universal (i.e., abnormal renal biopsy), even in the absence of clinically apparent disease [5].
- The most common site of embolic disease is the central nervous system. The incidence of stroke has been shown to decline rapidly with the initiation of antibiotic therapy—from 4.82 per 1000 patient days in the first week of therapy to 1.71 per 1000 patient days in the second week [6]. Hallmark physical exam findings suggesting IE may be noted, including Janeway lesions, Osler nodes, Roth's spots, petechiae, and subungual hemorrhages [7].
- IE may also be characterized as acute and subacute based on the virulence of the causative pathogen, severity of infection, progression, and presence of underlying cardiac disease [7]. Acute IE is typically caused by *S. aureus* and is highly destructive to native heart valves with high early mortality if left untreated. Subacute IE is indolent, caused by organisms such as *S. viridans*, and often involves previously damaged valves.
- The side of valvular involvement (left vs. right) results in differences in cardiac manifestations (e.g., heart failure), bacterial density, treatment duration, choice of management (e.g., surgical intervention), and risk of embolus. Left-sided IE accounts for 90% of all IE cases [8]. Of all right-sided IE, 90% are among IVDU [9].

Diagnostic Criteria

- The modified Duke criteria provide a clinical framework for the diagnosis of IE using major and minor criteria. It has a reported sensitivity between 70% and 79% (Table 18.1) [10].
- Definitive endocarditis may also be diagnosed from culture of microorganisms or histologic evidence of active endocarditis from pathologic lesions, vegetations, or intracardiac abscess.

Table 18.1 Modified Duke criteria [1]

Definite IE	Possible IE	Rejected IE
2 major criteria	1 major and 1–2 minor criteria	0 major and 1–2 minor criteria
1 major and ≥3 minor criteria	3–4 minor criteria	1 major and 0 minor criteria
5 minor criteria		
Major criteria		
1. Blood culture positive for IE		
• Typical microorganism for infective endocarditis from two separate blood cultures: *Viridans streptococci*, *Staphylococcus aureus*, *Streptococcus bovis*, HACEK group (*Haemophilus* spp., *Actinobacillus actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella* spp., and *Kingella kingae*) or community-acquired enterococci, in the absence of a primary focus		
• Microorganisms consistent with infective endocarditis from persistently positive blood cultures, defined as follows: – At least 2 positive cultures of blood samples drawn more than 12 h apart – All of 3 or a majority of 4 or more separate cultures of blood (with first and last sample drawn ≥1 h apart) – Single positive blood culture for *Coxiella burnetii* or phase I antibody titer >1:800		
2. Evidence of endocardial involvement		
• Echocardiogram supportive of infective endocarditis		
• Definition of positive findings: Oscillating intracardiac mass, on valve or supporting structures, or in the path of regurgitant jets, or on implanted material, in the absence of an alternative anatomic explanation or myocardial abscess or new partial dehiscence of prosthetic valve		
• New valvular regurgitation (increase or change in preexisting murmur not sufficient)		
Minor criteria		
Predisposing heart condition or intravenous drug use		
Fever ≥38 °C (100.4 °F)		
Vascular phenomena: Major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhage, Janeway lesions		
Immunologic phenomena: Glomerulonephritis, Osler's nodes, Roth spots, rheumatoid factor Positive blood culture not meeting major criterion as noted previously (excluding single positive cultures for coagulase-negative staphylococci and organisms that do not cause endocarditis) or serologic evidence of active infection with organism consistent with infective endocarditis		

Antimicrobial Management

- Treatment of IE begins with empiric antibiotic therapy, ideally after blood cultures are collected if it does not result in significant delays in therapy. Selection of organism specific antibiotic therapy is complex.
- Antibiotic therapy is typically prolonged and dependent on resistance profiles, native vs. prosthetic valves, and type of pathogen.
- Antibiotic selection and duration recommendations are summarized in a set of guidelines by the AHA [11].

Surgical Intervention

- Factors determining whether to operate include the patient's hemodynamic status, left vs. right-sided pathology, risk of thromboembolic complication, and likelihood of source control with non-invasive treatments [2, 12].
- Surgical candidacy and timing can be further complicated by recent stroke/mycotic aneurysms, abscesses/source control, psychosocial issues including IV drug use and non-compliance, and center expertise.

Timing and Indications for Operation on Left-Sided Valves

- Surgical repair of left-sided valves (aortic and mitral) serves to address hemodynamic status/heart failure, obtain source control, and prevent thromboembolic complications.
- Early surgical intervention (within 48 h) for patients with IE and severe valve disease has been shown to be associated with lower rates of death, embolic events, recurrence of IE, and repeat hospitalizations due to the development of CHF [13].
- Several guidelines (AHA, European Society of Cardiology, American Association for Thoracic Surgery (AATS)) exist to guide operative decision-making [12, 14, 15].
- The 2014 AHA guidelines recommend early surgery (defined as during the initial hospitalization before completion of full therapeutic antibiotic course) for otherwise good operative candidates and patients with valve dysfunction resulting in heart failure symptoms, heart block, annular or aortic abscess, destructive penetrating lesions, left-sided IE with highly resistant organisms or persistent infection, prosthetic valve endocarditis, and relapsing infection.
- The AATS emphasizes early operation and minimizing delays before heart failure symptoms are evident [15].

Timing and Indications for Operation on Right-Sided Valves

- Right-sided disease has lower risk of severe sequelae (i.e., lower risk of thromboembolic events and is typically subacute disease).
- Surgery is considered for persistent bacteremia, right heart failure due to tricuspid regurgitation that is refractory to diuresis, and vegetations on the tricuspid valve >20 mm that persist after recurrent pulmonary embolism.
- Common operations in right-sided IE include tricuspid valvectomy, tricuspid valve repair, and tricuspid valve replacement [2].

Complex Multi-Valve Endocarditis

- Complicated endocarditis may require replacement or repair of multiple valves after extensive debridement.
- Commonly, a drop lesion from the aortic valve may infect the anterior leaflet of the mitral valve, or erosive abscesses can invade the intervalvular fibrous body (IFB). [16].
- Replacement or repair of both valves and reconstruction of the IVF can be done through procedures such as the Commando (double-valve replacement with IFB reconstruction) or Hemi-Commando (aortic-valve replacement and mitral valve repair).
- Such procedures are complex, associated with high morbidity and mortality rates, and are typically performed at tertiary care facilities [17–19].
- Similarly, the use of homografts (human tissue grafts) over xenografts or mechanical prostheses requires higher technical expertise. However, the use of homografts has not been shown to be more resistant to reinfection in IE [20].

Timing of Operative Intervention After Acute Stroke

- Cardiac surgeons must balance urgent operation with the risk of converting ischemic stroke to hemorrhagic stroke, worsening existing hemorrhagic stroke, causing hypotension or embolization with cardiopulmonary bypass [21].
- Recently, it was found that early surgery for IE after stroke was not associated with an increased risk of new post-operative stroke and not all operations should be delayed [22–24].
- Patients with intracerebral hemorrhage (ICH), though, may have increased risk of mortality after early surgery.
- Current AATS guidelines recommend (1) early surgery for patients with non-hemorrhagic stroke and cardiac indications for urgent surgery, (2) delaying operations for 3+ weeks for patients with recent ICH, and (3) a neurology consultation for patients with severe or multiple strokes [15].

Special Considerations

Prosthesis Selection

- Prosthesis selection (mechanical vs. tissue) for native valve endocarditis requiring simple valve replacement should be based on normal criteria including age, compliance with anticoagulation, life expectancy, and presence of comorbidities [15, 25].
- Several studies have shown no difference in reinfection rates between patients receiving mechanical vs. tissue prosthetic valves [26–28].
- Recent stroke may limit post-operative anticoagulation mandating a tissue valve.

Prosthetic Valve Endocarditis

- IE can occur in 3–6% of those with prosthetic valves [29].
- PVE is separated into early (first 60 days after surgery), intermediate (60 days-first year after surgery), and late-onset (greater than 1 year) [12].
- Nearly 50% of PVE cases require surgery, though PVE is associated with high mortality and morbidity after surgical intervention [30].
- Surgical debridement and replacement of infected prosthetic valves are associated with improved mortality compared to medical treatment.
- Select cases of PVE—such as patients without signs of congestive heart failure, conduction abnormalities, valvular complications, or annular abscesses or fistulas— may be treated with antibiotic therapy alone [31–34].

Intravenous Drug Use

- Short-term outcomes in patients with IE related to intravenous drug use have been shown to be better than non-IVDU (likely due to age, fewer comorbidities, and selection bias for healthier operative candidates); but, there are high rates of reinfection and re-operation leading to poor long-term outcomes and increased risk of death [35–37].
- Re-do surgeries are complex and costly prompting debate on resource allocation and futility [38].
- IVDUs are a vulnerable population at risk of delayed care, sub-standard care, or refusal of care stemming in part from surgeon bias and stigma within the medical system and greater society [39].
- Coupling addiction treatment with surgical intervention for IE may reduce recidivism and improve outcomes [40]. Psychiatric services including addiction medicine therapy, after-care programs, and medical adjuncts (i.e., buprenorphine, methadone, naltrexone) may be used to reduce the risk of relapse and support IVDUs pre-and post-operatively [41].
- Multi-disciplinary care with cardiac surgery, infectious disease, social work, and addiction medicine is an important part of caring for these populations.

References

1. Wang A, Gaca JG, Chu VH. Management considerations in infective endocarditis: a review. JAMA J Am Med Assoc. 2018;320(1):72–83.
2. Kilic A. Infective endocarditis: a multidisciplinary approach. San Diego: Elsevier Science & Technology; 2021.
3. Vogkou CT, Vlachogiannis NI, Palaiodimos L, Kousoulis AA. The causative agents in infective endocarditis: a systematic review comprising 33,214 cases. Eur J Clin Microbiol Infect Dis. 2016;35(8):1227–45.
4. Cahill TJ, Prendergast BD. Infective endocarditis. Lancet Br Ed. 2015;387(10021):882–93.

5. Holland TL, Baddour LM, Bayer AS, Hoen B, Miro JM, Fowler VG. Infective endocarditis. Nat Rev Dis Primer. 2016;2(1):16059.
6. Dickerman SA, Abrutyn E, Barsic B, Bouza E, Cecchi E, Moreno A, et al. The relationship between the initiation of antimicrobial therapy and the incidence of stroke in infective endocarditis: an analysis from the ICE prospective cohort study (ICE-PCS). Am Heart J. 2007;154(6):1086–94.
7. Cohn LH, Adams DH, Cohn LH. In: Cohn LH, Adams DH, editors. Cardiac surgery in the adult. 5th ed. New York: McGraw Hill Education; 2018.
8. Chahoud J, Sharif Yakan A, Saad H, Kanj SS. Right-sided infective endocarditis and pulmonary infiltrates: an update. Cardiol Rev. 2016;24(5):230–7.
9. Moreillon P, Que YA. Infective endocarditis. Lancet Br Ed. 2004;363(9403):139–49.
10. Shrestha N, Shakya S, Hussain S, Pettersson G, Griffin B, Gordon S. Sensitivity and specificity of Duke criteria for diagnosis of definite infective endocarditis: a cohort study. Open Forum Infect Dis. 2017;4:S550–1.
11. Baddour LM, Wilson WR, Bayer AS, Fowler VG, Tleyjeh IM, Rybak MJ, et al. Infective endocarditis in adults: diagnosis, antimicrobial therapy, and management of complications: a scientific statement for healthcare professionals from the American Heart Association. Circulation (N Y). 2015;132(15):1435–86.
12. Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP, Guyton RA, et al. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation (N Y). 2014;129(23):2440–92.
13. Kang DH, Kim YJ, Kim SH, Sun BJ, Kim DH, Yun SC, et al. Early surgery versus conventional treatment for infective endocarditis. N Engl J Med. 2012;366(26):2466–73.
14. Habib G, Lancellotti P, Antunes MJ, Bongiorni MG, Casalta JP, Del Zotti F, et al. 2015 ESC guidelines for the management of infective endocarditis: the task force for the Management of Infective Endocarditis of the European Society of Cardiology (ESC). Endorsed by: European Association for Cardio-Thoracic Surgery (EACTS), the European Association of Nuclear Medicine (EANM). Eur Heart J. 2015;36(44):3075–128.
15. Pettersson GB, Hussain ST. Current AATS guidelines on surgical treatment of infective endocarditis. Ann Cardiothorac Surg. 2019;8(6):630–44.
16. Goerlich CE, Aziz H, Kilic A. Chapter 19: Complex multivalve operations for infective endocarditis. In: Kilic A, editor. Infective endocarditis. Academic Press; 2022. p. 291–7. Available from: https://www.sciencedirect.com/science/article/pii/B9780128206577000090.
17. Navia JL, Elgharably H, Hakim AH, Witten JC, Haupt MJ, Germano E, et al. Long-term outcomes of surgery for invasive valvular endocarditis involving the aortomitral fibrosa. Ann Thorac Surg. 2019;108(5):1314–23.
18. Davierwala PM, Binner C, Subramanian S, Luehr M, Pfannmueller B, Etz C, et al. Double valve replacement and reconstruction of the intervalvular fibrous body in patients with active infective endocarditis. Eur J Cardiothorac Surg. 2014;45(1):146–52.
19. Kim SW, Park PW, Kim WS, Sung K, Lee YT, Jun TG, et al. Long-term results of aortomitral fibrous body reconstruction with double-valve replacement. Ann Thorac Surg. 2013;95(2):635–41.
20. Kim JB, Ejiofor JI, Yammine M, Camuso JM, Walsh CW, Ando M, et al. Are homografts superior to conventional prosthetic valves in the setting of infective endocarditis involving the aortic valve? J Thorac Cardiovasc Surg. 2016;151(5):1239–48.
21. Tchouta L, Fukuhara S. Chapter 20: Timing of surgical intervention following acute stroke from infective endocarditis. In: Kilic A, editor. Infective endocarditis. Academic Press; 2022. p. 299–306. Available from: https://www.sciencedirect.com/science/article/pii/B9780128206577000168.
22. Ghoreishi M, Foster N, Pasrija C, Shah A, Watkins AC, Evans CF, et al. Early operation in patients with mitral valve infective endocarditis and acute stroke is safe. Ann Thorac Surg. 2018;105(1):69–75.

23. Sorabella RA, Han SM, Grbic M, Wu YS, Takyama H, Kurlansky P, et al. Early operation for endocarditis complicated by preoperative cerebral emboli is not associated with worsened outcomes. Ann Thorac Surg. 2015;100(2):501–8.
24. Yoshioka D, Sakaguchi T, Yamauchi T, Okazaki S, Miyagawa S, Nishi H, et al. Impact of early surgical treatment on postoperative neurologic outcome for active infective endocarditis complicated by cerebral infarction. Ann Thorac Surg. 2012;94(2):489–96.
25. Byrne JG, Rezai K, Sanchez JA, Bernstein RA, Okum E, Leacche M, et al. Surgical management of endocarditis: the society of thoracic surgeons clinical practice guideline. Ann Thorac Surg. 2011;91(6):2012–9.
26. Flynn CD, Curran NP, Chan S, Zegri-Reiriz I, Tauron M, Tian DH, et al. Systematic review and meta-analysis of surgical outcomes comparing mechanical valve replacement and bioprosthetic valve replacement in infective endocarditis. Ann Cardiothorac Surg. 2019;8(6):587–99.
27. Moon MR, Miller DC, Moore KA, Oyer PE, Mitchell RS, Robbins RC, et al. Treatment of endocarditis with valve replacement: the question of tissue versus mechanical prosthesis. Ann Thorac Surg. 2001;71(4):1164–71.
28. Toyoda N, Itagaki S, Tannous H, Egorova NN, Chikwe J. Bioprosthetic versus mechanical valve replacement for infective endocarditis: focus on recurrence rates. Ann Thorac Surg. 2018;106(1):99–106.
29. Habib G, Thuny F, Avierinos JF. Prosthetic valve endocarditis: current approach and therapeutic options. Prog Cardiovasc Dis. 2008;50(4):274–81.
30. Mihos CG, Capoulade R, Yucel E, Picard MH, Santana O. Surgical versus medical therapy for prosthetic valve endocarditis: a meta-analysis of 32 studies. Ann Thorac Surg. 2017;103(3):991–1004.
31. Mahesh B, Angelini G, Caputo M, Jin XY, Bryan A. Prosthetic valve endocarditis. Ann Thorac Surg. 2005;80(3):1151–8.
32. Fonseca JP, Pereiro T, Dos Santos DP, Correia JM, Capelo J, Carragoso A. Successful management of prosthetic valve brucella endocarditis with antibiotherapy alone. Eur J Case Rep Intern Med. 2018;5(4):000808.
33. Akowuah EF, Davies W, Oliver S, Stephens J, Riaz I, Zadik P, et al. Prosthetic valve endocarditis: early and late outcome following medical or surgical treatment. Heart. 2003;89(3):269.
34. Chirillo F, Scotton P, Rocco F, Rigoli R, Pedrocco A, Martire P, et al. Management strategies and outcome for prosthetic valve endocarditis. Am J Cardiol. 2013;112(8):1177–81.
35. Shrestha NK, Jue J, Hussain ST, Jerry JM, Pettersson GB, Menon V, et al. Injection drug use and outcomes after surgical intervention for infective endocarditis. Ann Thorac Surg. 2015;100(3):875–82.
36. Tiako MJN, Mori M, Mahmood SUB, Shioda K, Mangi A, Yun J, et al. Recidivism is the leading cause of death among intravenous drug users who underwent cardiac surgery for infective endocarditis. Semin Thorac Cardiovasc Surg. 2019;31:40–5.
37. Kim JB, Ejiofor JI, Yammine M, Ando M, Camuso JM, Youngster I, et al. Surgical outcomes of infective endocarditis among intravenous drug users. J Thorac Cardiovasc Surg. 2016;152(3):832–841.e1.
38. Fleischauer AT, Ruhl L, Rhea S, Barnes E. Hospitalizations for endocarditis and associated health care costs among persons with diagnosed drug dependence—North Carolina, 2010–2015. MMWR Morb Mortal Wkly Rep. 2017;66(22):569.
39. DiMaio JM, Salerno TA, Bernstein R, Araujo K, Ricci M, Sade RM. Ethical obligation of surgeons to noncompliant patients: can a surgeon refuse to operate on an intravenous drug-abusing patient with recurrent aortic valve prosthesis infection? Ann Thorac Surg. 2009;88(1):1–8.
40. Sacks S, Ries RK. Substance abuse treatment for persons with co-occurring disorders. Treatment improvement protocol (TIP) series 42. Rockville, MD: Substance Abuse and Mental Health Services Administration; 2005.
41. Suzuki J. Medication-assisted treatment for hospitalized patients with intravenous-drug-use related infective endocarditis. Am J Addict. 2016;25(3):191–4.

Chapter 19
Aneurysmal Disease of the Ascending Aorta, Root, and Arch

Bartlomiej R. Imielski and Leonard N. Girardi

Anatomy

- The aortic root is composed of the aortic valve (leaflets, commissures, and annulus), sinuses of Valsalva, right and left coronary ostia, and sinotubular junction (STJ) (Fig. 19.1).
- The aortic valve annulus is anchored by two fibrous trigones to the rigid framework of the heart, and with the STJ provides structural stability to the aortic root.
- The root is centrally located within the heart, such that it is adjacent to all cardiac chambers, which is of importance in aortic valve endocarditis, given that abscess and fistulas may form into any one of these chambers or spaces.
- Diseases affecting the aortic root include intrinsic aortic valve pathology, dilation of the components of the root (annulus, STJ, or sinuses of Valsalva), aneurysms, pseudoaneurysms, acute aortic syndromes (dissections, ruptures), infectious pathology, as well as inherited conditions such as bicuspid aortic valve (BAV and genetically triggered aneurysms).
- The ascending aorta emerges as the main blood vessel from the root, taking off at the STJ and continuing until the aortic arch, just proximal to the innominate artery. Its slight curve lends to the nomenclature of lesser/inner and greater/outer curves (Fig. 19.1).

B. R. Imielski (✉)
Department of Cardiothoracic Surgery, Weill Cornell Medicine, New York, NY, USA

Department of Cardiothoracic Surgery, Wake Forest University School of Medicine, Winston-Salem, NC, USA
e-mail: bimielsk@wakehealth.edu

L. N. Girardi
Department of Cardiothoracic Surgery, Weill Cornell Medicine, New York, NY, USA
e-mail: lngirard@med.cornell.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_19

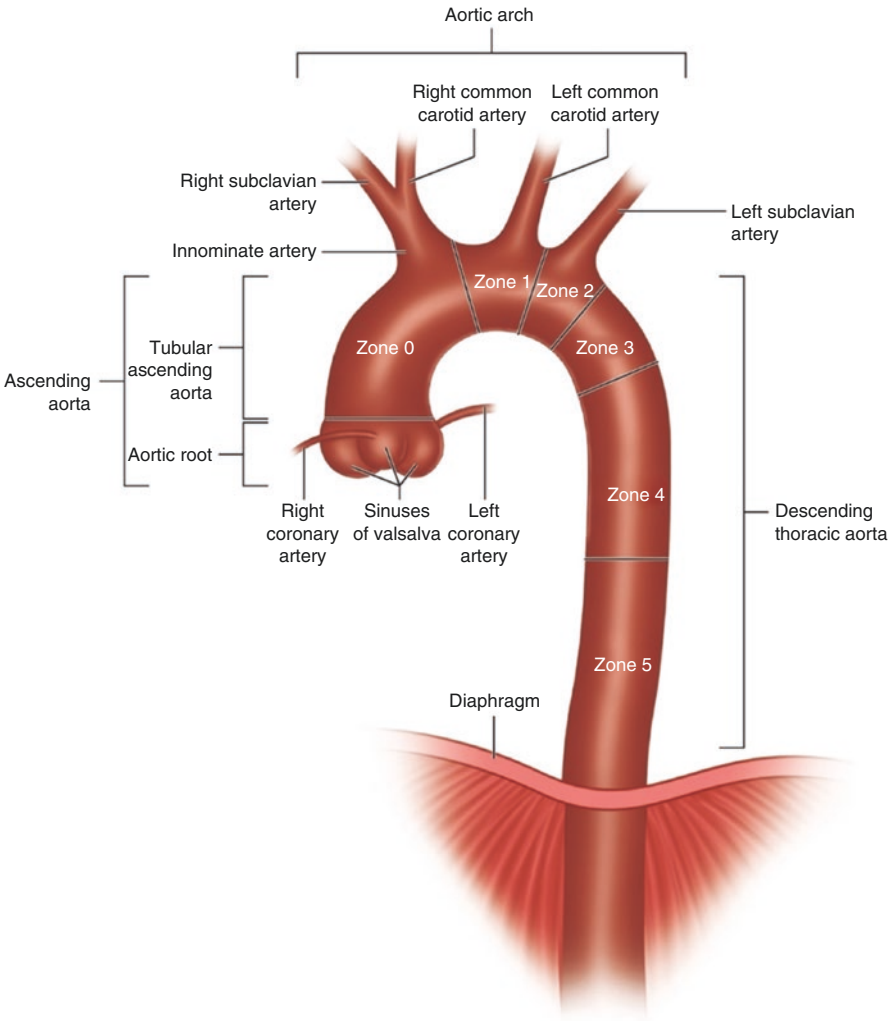


Fig. 19.1 This illustration demonstrates the thoracic aorta, composed of the aortic root, ascending arch and descending aorta. The root contains three billowing sinuses of Valsalva, from which the right and left coronary arteries originate. The root tapers at the sinotubular junction where it transitions to the ascending aorta. A typical great vessel branching pattern is shown

- The aortic arch typically gives off three great vessels: the innominate (aka brachiocephalic), left common carotid, and left subclavian arteries.
- Two common variants are a "Bovine arch" with the left common carotid artery branching off the innominate artery instead of the arch itself, and AA aberrant left vertebral artery that arises directly off of the arch as a fourth arch vessel.
- The ascending and aortic arch segments are subject to dilation, aneurysm and pseudoaneurysm formation, dissection, rupture, aortitis, and entities such as penetrating aortic ulcers, and intramural hematomas.

Etiology of Dilation

- The aortic root and the downstream aorta are subject to dilation given lifelong pressurization and impulse throughout the cardiac cycle.
- Elastin and collagen are the main structural glycoproteins of the aorta, and conditions affecting their synthesis or metabolism contribute to inherited aortic syndromes (Table 19.1).
- Similarly degenerative changes can occur secondary to pro-inflammatory states such as smoking, dyslipidemia, auto-immune conditions, and infections, causing cystic medial degeneration of the aorta (Table 19.2).
- The common consequence is dilation, which once started leads to a vicious cycle according to the Law of Laplace (wall tension = [intraluminal pressure × radius]/2 × wall thickness). As a result, as the vessel dilates and its wall thins, the wall tension further increases exacerbating the process.
- Ectasia refers to dilation beyond normal, whereas aneurysm refers to pathologic dilation. Pseudoaneurysm differs from a true aneurysm in that not all components of the aortic wall are involved in the outpouching.
- The clinical risk associated with dilation is free wall rupture or aortic dissection, events that without surgical intervention are usually fatal. Patients with known aneurysms should be monitored and referred for elective surgery, in order to prevent the aforementioned acute aortic events.

Table 19.1 Inherited aortic conditions

Inherited condition	Genetic defect	Population prevalence	Dissection prevalence
Marfan syndrome	FBN1	1:5000–1:15,000	51%
Loeys-Dietz syndrome	TGFBR1/2, TGFB2	Unknown	20%
Vascular Loeys-Dietz	COL3A1	1:90,000	Unknown
Turner syndrome	45X	1:2000	35%
Bicuspid aortic valve	ROBO4, DATA5, NOTH1	1:5–1:100	35%
Heritable thoracic aortic disease	FBN1, TGFVR1/2, SMAD3, TGFB2, COL3A1, ACTA1, MYH11, MYLK, LOX, PRKG1, FOXE3	Unknown	9–46%

Table 19.2 Causes of aortic aneurysm formation

Acquired	Inflammatory	Inherited	Infections/mycotic
Smoking	Takayasu arteritis	Marfan	Syphilis
Hypertension	Giant cell arteritis	Loeys-Dietz	Traumatic
Hyperlipidemia	Bechet's disease	Turner	Other
Chronic dissection	Ankylosing spondylitis	Bicuspid	

Table 19.3 Surgical indications for aneurysm repair

Aneurysm type/condition	Indication
Degenerative (root, ascending or arch) without genetic predisposition	Aortic diameter >5.5 cm
Degenerative or inherited	Aortic growth >0.5 cm/year
Degenerative or inherited	Aortic diameter >4.5 cm with concomitant surgery on aortic valve
Inherited	Aortic diameter 4–5 cm

- The timing of surgical intervention of asymptomatic aneurysms is based on the size at which the risk of surgery becomes less or equal to the risk of rupture. The 2010 guidelines for Thoracic Aortic Disease are summarized in Table 19.3. In general, aneurysms >5.5 cm in diameter, or with growth rate >0.5 cm/year should be considered for surgery [1].
- Expert consensus does additionally support concomitant root replacement when diameter is <45 mm, by experienced surgeons who routinely perform this complex procedure with exceptionally low mortality. Similarly, when the arch is 45 mm, arch replacement is reasonable; however, for diameters between 40 and 45 mm, the decision is nuanced and should be based on patient age and comorbidity in addition to individual surgeon and center experience [1, 2].
- There remains ongoing debate on the utility of indexed metrics to height, body weight, or BMI, as well as wall strain measurements for surgical prognostication.
- Patients suspected of having inherited aortic conditions should be referred to a geneticist and enrolled in a surveillance program.
- Patients with suspected infectious etiologies should be completely evaluated to determine the source of infection.
- The most common cause of mycotic (infected; bacterial or fungal) aortic degeneration, as seeding of either disrupted aortic intima or mural thrombus can lead to an aggressive local infection with rapid aortic expansion and rupture. Bacterial endocarditis may also contribute to the formation a mycotic aneurysm.
- Patients with mycotic aneurysms should undergo a culture driven anti-microbial treatment course and urgent surgery given the high rate of rapid growth and rupture in this population. Although TEVAR has been used in some series, removal of infected tissue and replacement with graft have been shown to be safe with good long-term outcomes.

Aortic Root Surgery

- One important facet of aortic root aneurysm surgery is whether the aortic valve can be spared. This is largely determined by the integrity of the aortic valve, including any aortic regurgitation and/or stenosis.

- As the aortic root dilates, the aortic valve is prone to becoming regurgitant secondary to one or a combination of the following: aortic annular dilation resulting in decreased free-edge leaflet coaptation, STJ dilation resulting in leaflet tethering, leaflet degeneration and prolapse, and finally free edge or commissural fenestrations.
- If the aortic valve can be spared, there are two types of valve sparring root replacements (VSRR) that can be performed. The first to be described, but less common now, is remodeling (Yacoub), where the aortic annulus and commissures are preserved by cutting out scallops in the sinus of Valsalva to which a scalloped graft is sewn (Fig 19.2).
- The second procedure is reimplantation (David), in which the commissures and annulus are reimplanted within a tube graft (Fig 19.3). One long-term advantage of the reimplantation over the remodeling technique is that the annulus is reinforced by graft material, thereby preventing further dilation, which is especially important in connective tissue disorder patients. Both techniques have undergone multiple iterations to improve patient outcomes.

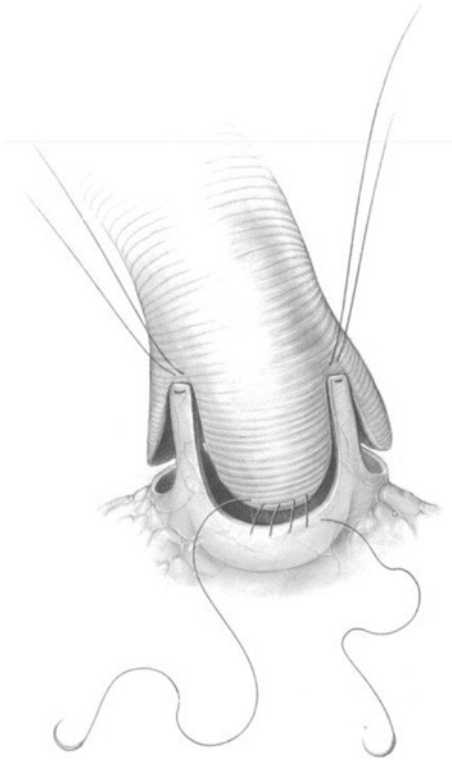


Fig. 19.2 This figure demonstrates a Yacoub remodeling technique of valve sparing root replacement. The conduit is trimmed with three scallops, which are sutured to the base of each sinus of Valsalva

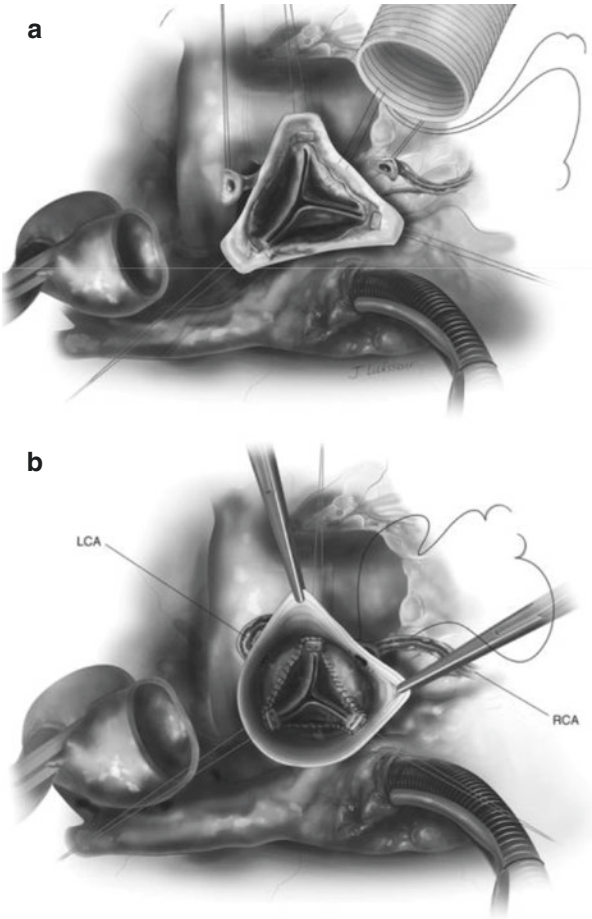


Fig. 19.3 This figure demonstrates a David reimplantation technique of valve sparring root replacement, where each aortic valve commissure is resuspended within the conduit. Unlike Yacoub remodeling, which has one running suture line, the David technique anchors the graft to the basal ring below the anulus of the aortic valve, and then uses a hemostatic suture line to resuspend the commissures and rim of each SoV within the tube conduit **(a)** mobilized root and coronary ostia prior to implantation into a graft conduit **(b)** reimplanted aortic root and coronary ostia within a graft condiut, showing commissural resuspension and a running hemostatis suture line

- If the aortic valve cannot be spared, a composite valve graft/conduit is the most common type of aortic root replacement (Fig. 19.4). This is composed of a bioprosthetic or mechanical aortic valve attached either by the surgeon or prefabricated to a Dacron tube graft.
- One technical challenge that is shared between valve sparring and composite conduits is the need for coronary artery reimplantation.

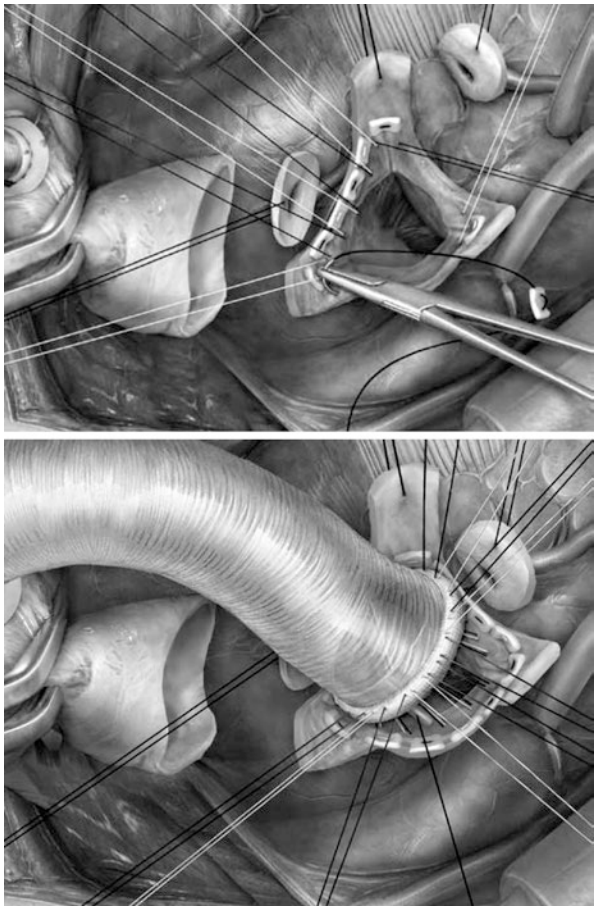


Fig. 19.4 This illustration demonstrates the root dissection for a composite valve conduit root replacement. The coronary buttons have been mobilized, and the aortic valve leaflets excised and debrided. Pledgeted valve sutures are used to secure the CVG to the root prior to anastomosing the coronary buttons

Aortic Arch Surgery

- Operating on the aortic arch poses a technical challenge due to intra-operative management of cerebral perfusion.
- The brain receives blood from the carotid and vertebral arteries which all originate from the aortic arch. As such, replacement of the aortic arch requires interruption of cerebral blood flow.
- In order to address this, surgeons rely on a combination of techniques including hypothermia, pharmacologic adjuncts, circulatory arrest with or without cerebral perfusion (antegrade (ACP) vs retrograde (RCP)), cerebral neural monitoring, and relying on the assumption of an intact cerebral circle of Willis.
- Depending on the extent of arch aneurysm needing to be replaced, surgeons may choose a combination of these cerebral protection modalities. These include deep hypothermic circulatory arrest (DHCA) (18–22 °C) without antegrade or

retrograde perfusion for brief periods of circulatory arrest, DHCA with either ACP or RCP, or moderate hypothermic circulatory arrest (MHCA) (22–26 °C) with ACP or RCP [3, 4].

- Hemiarch replacements are frequently done under deep or moderate circulatory arrest with or without RCP.
- Total arch replacements more likely to be performed under DHCA with ACP, although other strategies include DHCA and RCP, or MHCA with ACP, or DHCA alone [4, 5].
- The duration of maximal hypothermic circulatory arrest remains controversial, with reports showing safety of DHCA/RCP for prolonged periods of circulatory arrest. [6–8].
- When preparing for hypothermic circulatory arrest with retrograde cerebral perfusion, the superior vena cava is cannulated and snared (Fig. 19.5). Upon initiation of circulatory arrest, blood is pumped in a retrograde fashion through the SVC, up through the cerebral venous system and out the arterial system, allowing for washout of debris that may have fallen into the aortic head vessels.

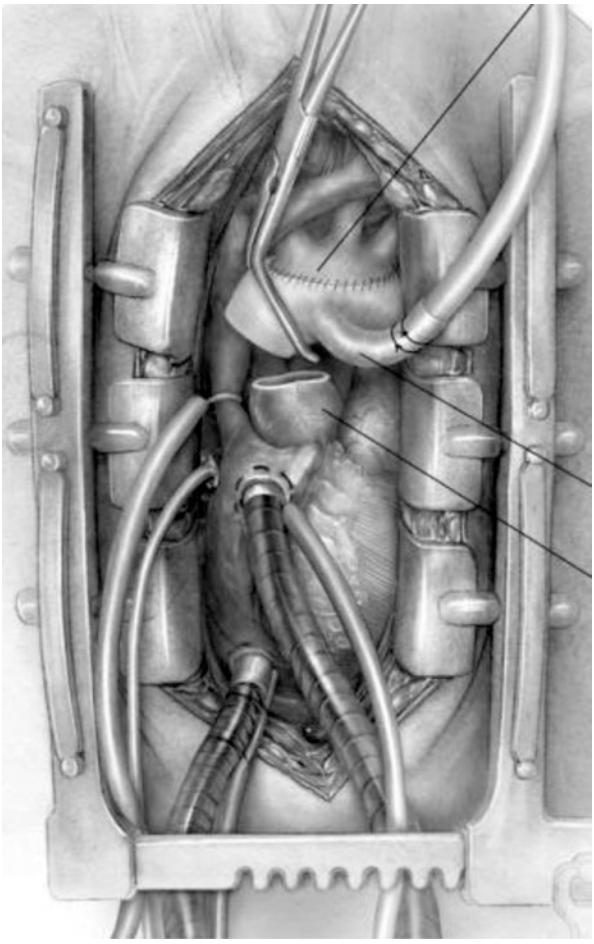


Fig. 19.5 Illustration demonstrating bicaval venous cannulation for cardiopulmonary bypass. One cannula is positioned within the SVC and snared, and the other within the IVC and snared. Retrograde cerebral perfusion can be delivered via the SVC cannula for circulatory arrest

- For antegrade cerebral perfusion, the axillary artery or the innominate artery is cannulated and snared. Upon circulatory arrest, cerebral blood flow is established in an antegrade fashion and relies on an intact cerebral circle of Willis for bilateral perfusion. If neuro monitoring suggests a perfusion mismatch, the surgeon may cannulate the left common carotid artery as well, and provide antegrade flow through it.
- The remains little evidence of any difference in unilateral vs bilateral ACP with regard to temporary or permanent neurological deficits.
- Other adjuncts often used for cerebral protection are hypothermia to decrease cerebral metabolism and oxygen demand (cooling on bypass and ice on head), iso-electric neural function with proper anesthetic management (i.e., propofol, Brevital), steroid, and mannitol administration.

Surgical Principles and Management

- When preparing for aortic aneurysm surgery, you must determine the extent of aneurysm requiring resection, the appropriate cannulation strategy to accommodate for cerebral protection if necessary, plan for cardioplegia and if circulatory arrest is needed, as well as alternative adjuncts such as left ventricular venting for myocardial protection and visualization.
- In older patients with degenerative aneurysms, a more conservative approach may be warranted in which only the aneurysm is resected, whereas ecstatic portions of the aorta are left behind so that cardiopulmonary bypass time, cardiac ischemic time, and cerebral ischemia can be minimized.
- On the other hand, younger patients with connective tissue disorders may undergo more aggressive aortic resection and replacement in hopes of avoiding future surgeries. These may also include standard elephant trunk or frozen elephant trunks, in which unsupported graft material, or a stented graft, respectively, is left distally from a total arch replacement to assist with management of downstream aortic pathology necessitating intervention. This may include subsequent open thoracoabdominal aneurysm surgery or TEVAR.
- The aortic arch is becoming a frontier for endovascular intervention, especially in patients who are poor surgical candidates. However, open surgical repair remains the gold standard. TEVAR traditionally require 2.0–2.5 cm of healthy proximal and distal aortic landing zone to properly anchor the device and prevent endoleaks.
- With this in mind, TEVAR grafts can be deployed more proximally in certain cases such as acute or chronic Type B dissections, or aneurysms covering the left subclavian artery which can be bypassed at the same time, or pre-emptively before stenting.
- In rare cases, a surgeon may choose to debranch the arch, in which the head vessels are bypassed off the main aorta using a side bitter clamp without cardiopulmonary bypass, and a TEVAR graft is deployed across the entire arch.

- Careful patient selection is paramount, as these cases are reserved for poor surgical candidates and have higher incidences of stroke, paralysis, dissection, and other adverse outcomes.
- There are ongoing clinical trials investigating TEVAR deployment across the arch with fenestrated and branched grafts into the great vessels in high-risk patients. These include the NEXUS aortic arch (TRIOMPHE) (NCT02365454), the endovascular treatment of TAAA and aortic arch aneurysms using fenestrated and branched grafts (NCT02323581), and the early feasibility of the branched TAG device in the treatment of aortic arch aneurysms studies (NCT02264977).

References

1. Hiratzka LF, Bakris GL, Beckman JA, Bersin RM, Carr VF, Casey DE Jr, Eagle KA, Hermann LK, Isselbacher EM, Kazerooni EA, Kouchoukos NT, Lytle BW, Milewicz DM, Reich DL, Sen S, Shinn JA, Svensson LG, Williams DM, American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, Society for Vascular Medicine. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with thoracic aortic disease: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. Circulation. 2010;121(13):e266–369. https://doi.org/10.1161/CIR.0b013e3181d4739e. Epub 2010 Mar 16. Erratum in: Circulation. 2010 Jul 27;122(4):e410.
2. Otto CM, Nishimura RA, Bonow RO, Carabello BA, Erwin JP 3rd, Gentile F, Jneid H, Krieger EV, Mack M, McLeod C, O'Gara PT, Rigolin VH, Sundt TM 3rd, Thompson A, Toly C. 2020 ACC/AHA guideline for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2021;143(5):e35–71. https://doi.org/10.1161/CIR.0000000000000932. Epub 2020 Dec 17. Erratum in: Circulation. 2021 Feb 2;143(5):e228. Erratum in: Circulation. 2021 Mar 9;143(10):e784.
3. Girardi LN, DeAnda A Jr, Ikonomidis JS. Feature editor summary: highlighting invited expert opinions on aortic subjects. J Thorac Cardiovasc Surg. 2021;162(1):40–3. https://doi.org/10.1016/j.jtcvs.2021.04.046. Epub 2021 Apr 22.
4. Lau C, Gaudino M, de Biasi AR, Munjal M, Girardi LN. Outcomes of open repair of mycotic descending thoracic and thoracoabdominal aortic aneurysms. Ann Thorac Surg. 2015;100(5):1712–7. https://doi.org/10.1016/j.athoracsur.2015.05.067. Epub 2015 Aug 13.
5. Lau C, Gaudino M, Iannacone EM, Gambardella I, Munjal M, Ohmes LB, Degner BC, Girardi LN. Retrograde cerebral perfusion is effective for prolonged circulatory arrest in arch aneurysm repair. Ann Thorac Surg. 2018;105(2):491–7. https://doi.org/10.1016/j.athoracsur.2017.07.018. Epub 2017 Nov 1.

6. Kuzmik GA, Sang AX, Elefteriades JA. Natural history of thoracic aortic aneurysms. J Vasc Surg. 2012;56(2):565–71. https://doi.org/10.1016/j.jvs.2012.04.053.
7. Brinster DR, Rizzo RJ, Bolman RM. Ascending aortic aneurysms. In: Cohn LH, editor. Cardiac surgery in the adult. New York: McGraw-Hill Medical; 2008. p. 1223–50.
8. Spielvogel D, Mathur MN, Griepp RB. Aneurysms of the aortic arch. In: Cogn LH, editor. Cardiac surgery in the adult. New York: McGraw-Hill Medical; 2008. p. 1251–76.

Chapter 20
Descending Thoracic and Thoracoabdominal Aortic Aneurysms

Srihari K. Lella and Arminder S. Jassar

Anatomy, Definition, and Etiology

- Aneurysms are localized dilations resulting from weakening of the arterial wall and consequent expansion in the vessel size. Generally, a dilation to 1.5 times the normal arterial diameter is defined as an aneurysm.
- Descending thoracic aortic aneurysms (DTAAs) are localized to the descending thoracic aorta while thoracoabdominal aortic aneurysms (TAAAs) can extend variably from distal to the left subclavian artery to the aorto-iliac bifurcation.
- TAAAs are classified according to the Crawford/Safi classification (Fig. 20.1):
  - Type I: distal to left subclavian artery → suprarenal abdominal aorta
  - Type II: distal to left subclavian artery → infrarenal aorta
  - Type III: distal to T6 → infrarenal aorta
  - Type IV: distal to diaphragm → infrarenal aorta
  - Type V: distal to T6 → suprarenal abdominal aorta
- DTAAs and TAAAs can result either due to the medial degeneration of the aortic wall due to atherosclerosis or due to aortic degeneration after acute aortic dissection.

S. K. Lella (✉)
Division of Vascular and Endovascular Surgery, Department of Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: srihari.lella@mgh.harvard.edu

A. S. Jassar
Division of Cardiac Surgery, Department of Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: AJASSAR@mgh.harvard.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_20

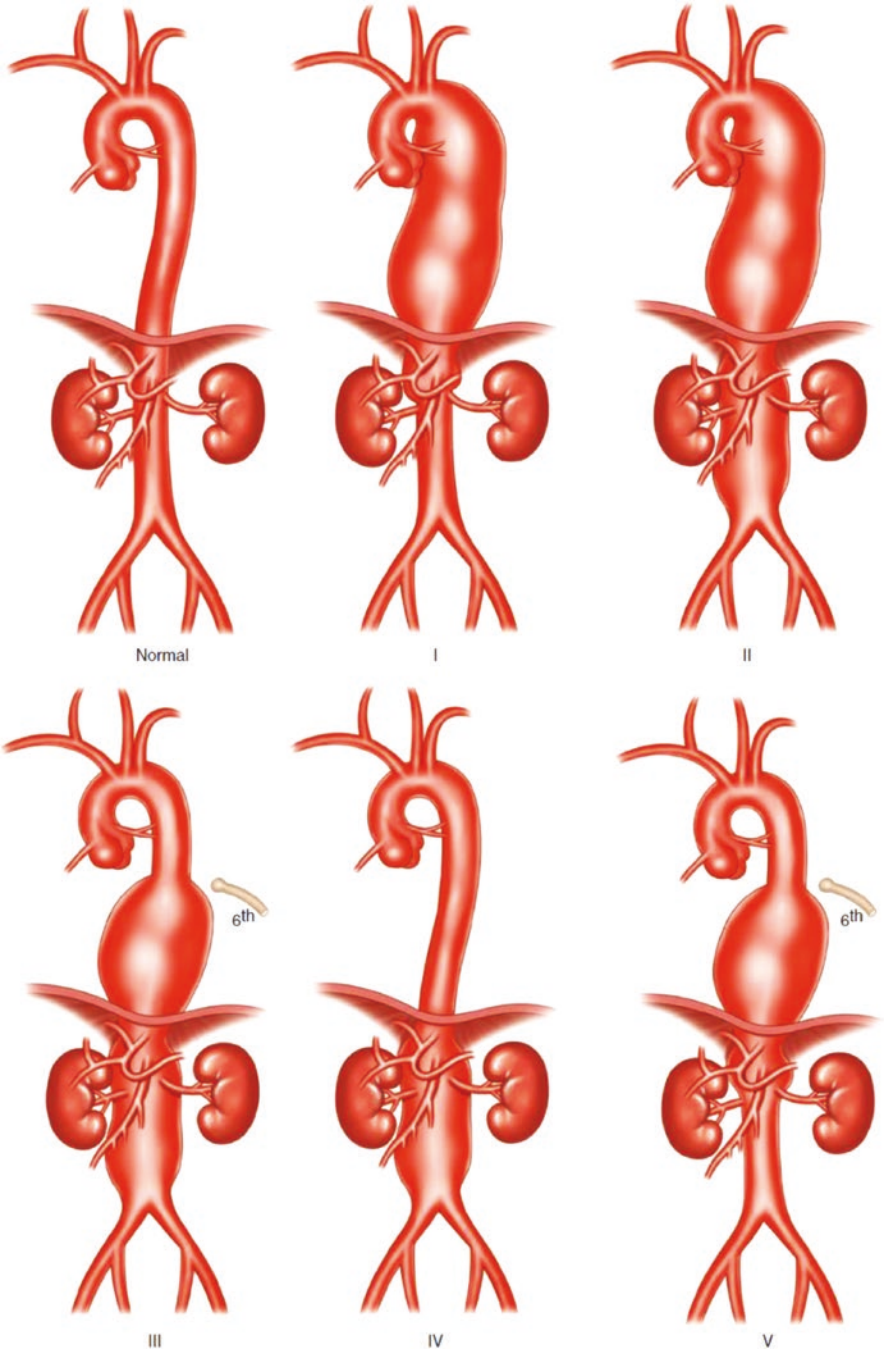


Fig. 20.1 Crawford/Safi classification of thoracoabdominal aortic aneurysms

- Autoimmune disorders, such as giant cell arteritis or Takayasu's arteritis, connective tissue disorders (e.g., Marfan's syndrome, Ehlers-Danlos syndrome), infection, and aortic anomalies can also result in aneurysms.

History and Physical

- Majority of patients do not have symptoms associated with their aneurysms, which are often found incidentally.
- When symptomatic, vague pain involving the chest, abdomen, and/or back/flank is the most common complaint. Pain is an important sign in the setting of aortic aneurysms and may be a harbinger of impending rupture.
- Other symptoms can result from local mechanical effects on nearby structures:
  - Cough or other respiratory symptoms from tracheal deviation.
  - Dysphagia from esophageal compression.
  - Hoarseness from recurrent laryngeal nerve compression or stretching.
- Although rare, erosion of the aneurysm into the esophagus or the airway can result in hematemesis/hemoptysis.
- Large aneurysms can sometimes be palpated as a prominent impulse on abdominal exam, especially in thin patients.
- Evaluate for tracheal deviation, abdominal tenderness, or diminished pulses in the lower extremities.
- Acute dissection or rupture of an aortic aneurysm can present as sudden onset of sharp severe pain, which may localize to the chest, abdomen or the back. Malperfusion to the viscera, kidneys, and lower extremities can also occur from embolization or dissection.

Tests

Imaging Evaluation

- Computed tomographic angiography (CTA)
  - Most used non-invasive imaging modality in defining aortic pathology.
  - Valuable in evaluating etiology (e.g., complex dissection vs. primary degenerative disease) and anatomy (e.g., branch vessels, large intercostals, and significant plaque/calcification) as well as planning surgical repair.
  - Requires intravenous iodinated contrast, which may be restrictive in patients who are allergic to contrast or those with significant renal impairment.

- Magnetic resonance angiography
  - Can provide similar information as CTA with regards to aortic pathology but has lower spatial resolution.
  - Newer 4D MRI offers the ability to assess flow dynamics may assist in identifying particularly weak spots in the aneurysmal wall.
  - Longer study times.
  - Difficult to obtain in claustrophobic patients without pre-medication.
  - Contraindicated in patients with significant metal implants (e.g., certain pacemakers).
  - Costly and less readily available.
- Ultrasound
  - Historically used as primary diagnostic modality for aortic aneurysms but it has now been largely replaced by axial imaging.
  - Today, used for monitoring of smaller sized abdominal aortic aneurysms until the size approaches closer to repair, at which time higher spatial resolution imaging is sought.
  - Limitations:

    Ultrasound technologist variability.
    Vision obstructed by bowel gas and osseous structures.
    Has shown to underestimate diameters (~5 mm) in the anterioposterior direction as compared to CT, with even less accuracy in the lateral direction.
- Positron emission tomography—computed tomography (PET/CT)
  - To assess for inflammatory causes of aneurysms (e.g., aortitis).
  - Helpful in assessing active and inactive vasculitis.
  - Can also be used to detect infectious aortitis, including response to antibiotic therapy.
  - Limited spatial resolution.
- Tagged white blood cell scan
  - Nuclear imaging study, which similarly to PET/CT, can be used to assess for inflamed/infected aneurysms.

Labs

- When less common etiologies are suspected, certain labs and/or markers can be checked to assess for autoimmune or genetic causes, e.g., elevated ESR, CRP, or IgG4 levels may indicate presence of acute aortitis.

Management and Treatment

- No medical treatment currently exists that causes shrinkage or reduction in the size of aortic aneurysms.
- Indications for repair:
  - Aortic diameter is primarily used to determine the need for repair given that the risk of rupture increases with increasing size (i.e., Laplace's Law).

    Current recommended size threshold for aneurysm repair generally ranges from 5 to 6 cm based on aneurysm anatomy, suitability for endovascular repair, and patient's overall health.
    Consider repair at smaller diameters in patients with connective tissue disorders (e.g., Marfan syndrome, Loeys-Dietz syndrome, Ehler-Danlos syndrome).
    Patient comorbidities, body surface area, aneurysm etiology, and morphology can be taken into consideration while determining the timing of aortic repair (e.g., a generally healthy patient with an aneurysm that require a simple repair and with low risk may be considered for repair earlier compared to an extensive aneurysm in a high-risk patient who might be expected to have increased risk of complications from their procedure).
  - ≥5 mm/6 months rate of growth of the aneurysm, regardless of aneurysm size.
  - Symptomatic patients should also be considered for repair, irrespective of aneurysm diameter.
- Patients who do not meet criteria for repair at their initial diagnosis should undergo periodic monitoring with surveillance imaging.
- Medical management and risk factor modification
  - Strict blood pressure management (beta blocker and/or angiotensin-converting enzyme inhibitor or angiotensin receptor blockers).
  - Atherosclerotic risk-reduction (statins).
  - Smoking cessation.
  - Avoidance of heavy, strenuous activities.
- Adjuvant treatments may be necessary for certain aneurysm etiologies:
  - Mycotic aneurysms, which generally warrant repair regardless of the size, require treatment with long-term antibiotics and possibly lifelong suppressive antibiotics.
  - Aneurysms secondary to vasculitis are often found during the acute inflammatory phase, which may necessitate anti-inflammatory medications (e.g., corticosteroids). Surgical repair can be delayed until resolution of the acute phase, if possible.

Operative Repair: Open vs. Endovascular

- Repair is performed to reduce or eliminate the risk of rupture associated with aneurysms.
- Open surgical repair consists of a left thoracotomy (for DTAAs) or left thoracoabdominal (for TAAAs) incision for replacement of the affected aortic segment with an artificial graft. In cases of infection, cryopreserved allografts or pericardial tubes can be utilized in lieu of synthetic grafts.
- If the involved segment involves any major aortic branches (celiac, superior mesenteric artery, renal arteries), these are detached from the aorta and reimplanted on to the aortic graft (described in detail below).
- Endovascular repair utilizes cylindrical endografts to create a neo-lumen with restriction of blood flow into the aneurysm sac
  - Thoracic endovascular aortic repair (TEVAR) has become the mainstay of therapy for DTAA.
  - There are currently no commercially available endografts for TAAA repair in the US. Physician-modified stent grafts for endovascular repair of TAAA require a main body for the aortic portion, with additional branches to maintain perfusion of the reno-visceral vessels. Investigator device exemption trials involving fenestrated and branched stent graft systems are being conducted with potential market availability in the near future. As such, open repair remains the current mainstay of therapy for TAAA.
- Hybrid approach to repair can also be performed for TAAAs, which involves open visceral debranching followed for endovascular aortic stent grafting.

Endovascular Repair of DTAA (TEVAR)

Preoperative planning

- Requires careful assessment of imaging to study aneurysm, branch vessel (i.e., arch and visceral vessels), and access vessel (i.e., iliofemorals) morphology (e.g., tortuosity) and quality (e.g., presence of thrombus, calcifications).
- Assessment of anticipated proximal and distal landing zones (areas of non-aneurysmal aorta on either side of the aneurysm). Generally, at least 2 cm of landing zone in normal aorta is desirable to allow for endograft to aorta apposition ("seal").
  - Proximally, if coverage of the left subclavian artery is necessary to form a seal, then preoperative revascularization via a left subclavian-carotid bypass or transposition can be performed.
  - Distally, when celiac artery coverage is necessary, celiac artery/superior mesenteric artery collaterals should be carefully ascertained to determine the need for celiac artery revascularization.

- Using 3D imaging software, the aorta should be sized (i.e., vessel diameter and length of coverage) orthogonal to the line of blood flow.
- If access vessels are of insufficient size to accommodate the sheaths, a plan for conduits (either endovascular or open) should be determined.

Operative Repair

TEVAR (Fig. 20.2) with the proximal landing zone just distal to the left subclavian artery and the distal landing zone just proximal to the celiac artery is described.

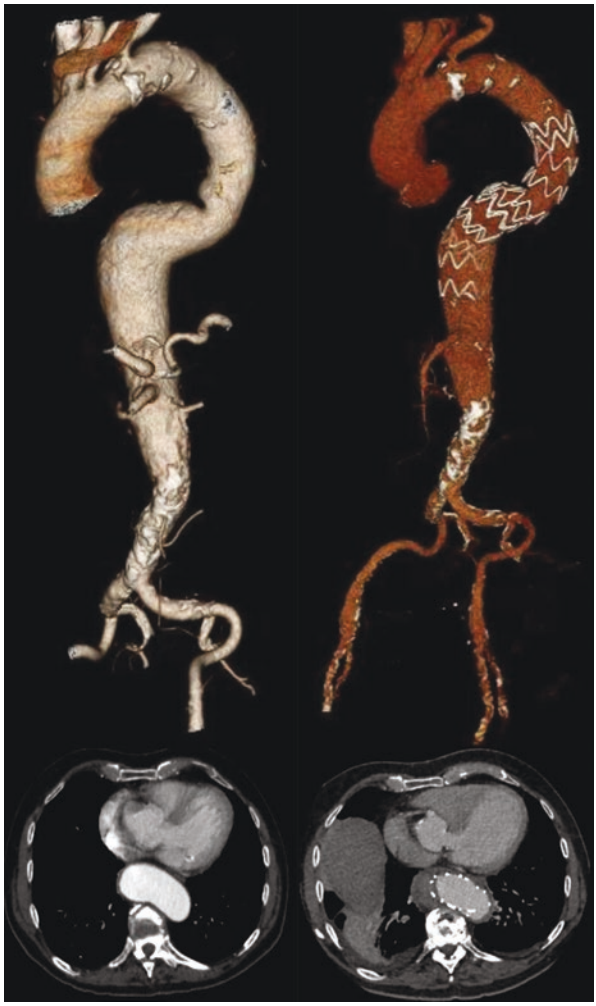


Fig. 20.2 Before and after computed tomographic angiography scan (bottom images) and 3D rendering (top images) of a patient with a TAAA who underwent TEVAR first stage procedure before repair of the TAAA

- Before initiation of anesthesia, a prophylactic cerebrospinal fluid drainage catheter can be considered. As cerebral perfusion pressure (CPP) is defined by the difference between the mean arterial pressure (MAP) and intracranial pressure (ICP), drainage results in a decrease of the ICP (CPP = MAP − ICP). Generally, a CPP ~70 mmHg or higher is maintained.
- The patient is placed in the supine position and bilateral groins are prepped and draped in a sterile fashion.
- Access to the femoral vessels can be obtained either via a surgical cutdown to expose the common femoral artery or percutaneously using ultrasound guidance. Side of graft deployment (usually termed ipsilateral side) is selected based on iliofemoral vessel size (usually need ≥6–7 mm, based on the diameter of the selected stent graft) and anatomy (calcification, tortuosity, etc.). Percutaneous access is obtained on the contralateral side for imaging purposes. Using Seldinger technique, vascular sheaths are placed to maintain vascular access throughout the procedure. A soft wire is inserted into the aorta from the ipsilateral side and advanced proximal to intended proximal landing zone. This is then exchanged over a catheter to a stiff wire, which will be used to guide the stent graft for insertion. A pigtail catheter with radio-opaque markings is inserted on the contralateral side for imaging.
- Angiography is performed via the pigtail catheter for visualization of the landing zones and final graft sizing. The radio-opaque markers on the pigtail are used to determine the length of the stent graft that is required to completely cover the aneurysm. More than one stent graft can be used depending on the diameter of the proximal and distal landing zones and the length of the aorta that needs to be covered.
- The appropriate diameter stent graft (usually 15–20% larger than the aortic diameter in the area of the landing zone) is advanced over the stiff wire to the target location.
- Deployment is device specific, and instructions from manufacturer should be followed; this step should be fully performed under live fluoroscopic visualization. Blood pressure should be controlled (decreased to MAP ~60) during this step to reduce the risk of misplacement due to windsocking effect. Additional stent grafts are deployed ensuring adequate overlap (usually ≥5 cm) with the preceding stent grafts, until the distal stent portion of the stent graft reaches the intended distal landing zone.
- After stent graft deployment, completion angiography is performed to ensure for accurate placement with no evidence of endoleaks. Certain endoleaks can be watched while others (Type I/III) must be treated at the time of the procedure.
- The vascular sheaths are removed, and the arterial access sites are repaired, either with direct repair in case of surgical cutdown, or using one of several commercially available percutaneous vessel closure devices for cases where vessel was accessed percutaneously.

If the patient had a spinal drain placed and there is no evidence of spinal cord ischemia, the drain can be removed on postoperative day 1. If, however, there is evidence of spinal cord ischemia, spinal drain is maintained. Management of spinal cord ischemia is further described below.

Open Repair

Preoperative Planning

- Open aortic repair requires careful preoperative assessment of the anatomy and planning of the anticipated procedure.
- Based on extent of repair, clamp sites and circulatory support strategy should be determined.
- Branch vessel anatomy (including large intercostal/lumbar vessels) should be carefully noted, and method of revascularization should be determined.
- Spinal cord protection (detailed below).
- Operative plan should be communicated with the anesthesia team preoperatively, and further, intraoperative close communication should be maintained.

Physiologic Evaluation

Given the significant physiologic stress for patients undergoing open surgical repair, preoperative evaluation of patient's ability to tolerate surgery should be carefully determined including:

- Overall functional status of a patient.
- Cardiac evaluation with an echocardiogram or stress test, and if necessary, coronary angiography, given that these patients are often elderly with atherosclerotic disease.
- Pulmonary function tests as chronic obstructive pulmonary disease is often prevalent and is a major risk factor for increased rate of complications and mortality after TAAA repair.
- Assessment for chronic renal insufficiency should be conducted with routine lab work, specifically glomerular filtration rate, and if necessary, duplex ultrasonography or cross-sectional imaging. Like COPD, pre-existing renal dysfunction increases risk of mortality after TAAA repair.

Operative Steps

An open extent II thoracoabdominal aortic aneurysm repair is described here. DTAA repair is less extensive and requires a thoracotomy without the abdominal component.

Anesthesia and Positioning

- Before initiation of anesthesia, a prophylactic cerebrospinal fluid drainage catheter can be considered depending on the extent of repair.
- Lung isolation is achieved either with a double lumen endotracheal tube or using a bronchial blocker. Isolated ventilation of the right lung with collapse of the left lung facilitates visualization of the thoracic aorta.

- A right radial arterial line (in case of need for left subclavian artery clamping) and right femoral arterial line (for distal aortic perfusion pressure) should be placed along with a central venous catheter placement.
- Somatosensory and/or motor evoked potentials (SSEP/MEP) can be considered for monitoring for spinal cord ischemia (SCI). Evoked potential monitoring has been shown to be efficacious in detecting SCI and can be used to determine the need for intercostal artery reimplantation.
  - SSEP is conducted via stimulation of the posterior tibial nerves with monitoring at the cervical spinal cord and cortical levels.
  - MEPs are conducted via stimulation of the cervical spinal cord with an assessment for lower extremity motor response.
- The patient should then be placed in a right lateral decubitus position (right side down, left side up), supported by a bean bag, and the left arm should be secured on an arm board.
- A final assessment of all catheters, lines, and probes should be conducted after repositioning and before prepping and draping.

Incision and Exposure

- A thoracic incision is made, usually in the left fifth intercostal space, carefully to avoid the intercostal neurovascular bundle and is carried down on to the abdomen, several centimeters to the left side of the midline, and carried as far down as necessary to achieve the exposure necessary for the operation.
- Left pleural cavity is entered by carrying the dissection through the subcutaneous tissue, chest wall musculature, intercostal muscles, and the pleura. Left lung is deflated to facilitate pleural entry. If needed, the ribs can be incised posteriorly to improve exposure.
- For the abdominal portion, the dissection is carried down to the rectus sheath, which is divided to expose the underlying peritoneum.
- The thoracic and abdominal components are then connected with the division of the anterior costal margin.
- Abdominal aortic exposure can then be proceeded with either a transperitoneal or retroperitoneal approach
  - Transperitoneal approach has a higher risk of splenic injury and increased fluid losses, and it can be performed via a left medial visceral rotation to enter the retroperitoneal space.
  - Retroperitoneal approach makes right iliac system access more difficult and involves mobilizing the peritoneum and intraabdominal contents away from the abdominal wall, while keeping the peritoneum intact, to create a plane toward the aorta.

- The diaphragm is incised circumferentially, leaving an adequate margin on the chest wall for later re-approximation.
- The reno-visceral vessels, distal extent of aneurysm, and planned distal clamp sites should be identified, exposed, and controlled.
- The ureter crosses over the iliac vessels and should be identified to avoid inadvertent injury.
- Attention is then be turned toward the thoracic aorta with dissection of the pleura down to the descending aorta (Fig. 20.3).
- The proximal clamp site is identified and exposed circumferentially and controlled taking care to avoid injury to the left recurrent laryngeal nerve and pericardiophrenic nerve.
- Finally, the aorta is subsequently exposed at the various anticipated clamp sites based on reno-visceral bypasses.

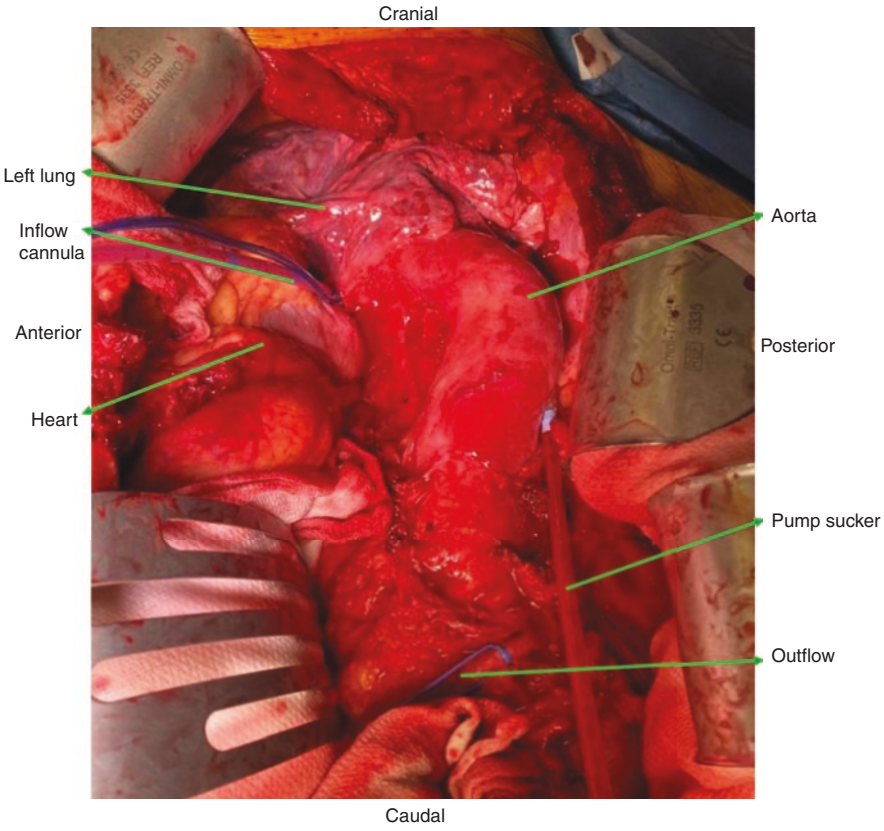


Fig. 20.3 Intraoperative image of thoracoabdominal aorta after complete exposure

Extracorporeal Circulation

- While these operations were historically performed in a clamp and sew manner, some form of distal perfusion strategy is commonly implemented today (Figs. 20.4, 20.5 and 20.6)
  - Passive external bypass (e.g., axillofemoral bypass)
  - Gott shunt
  - Partial heart bypass (left atrial-aortic/femoral bypass)
  - Complete heart bypass (femoral venous, aortic/femoral arterial) with or without deep hypothermic circulatory arrest

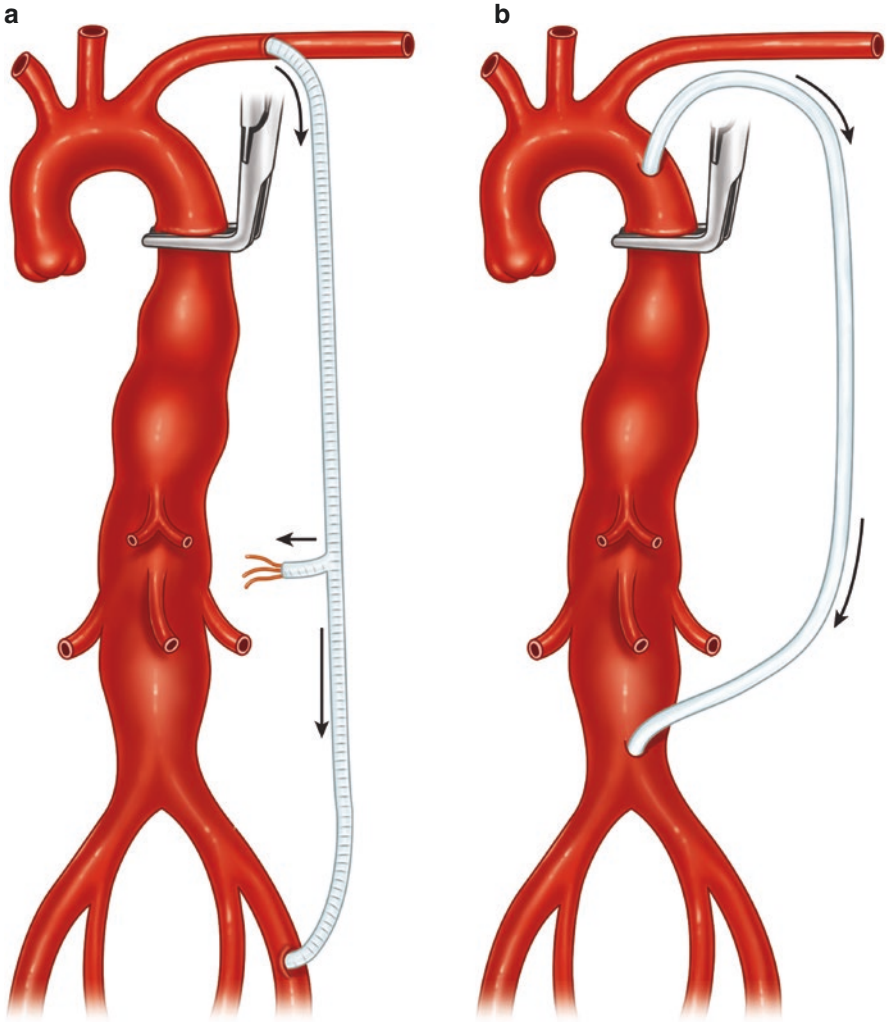


Fig. 20.4 External axillofemoral bypass and Gott shunt

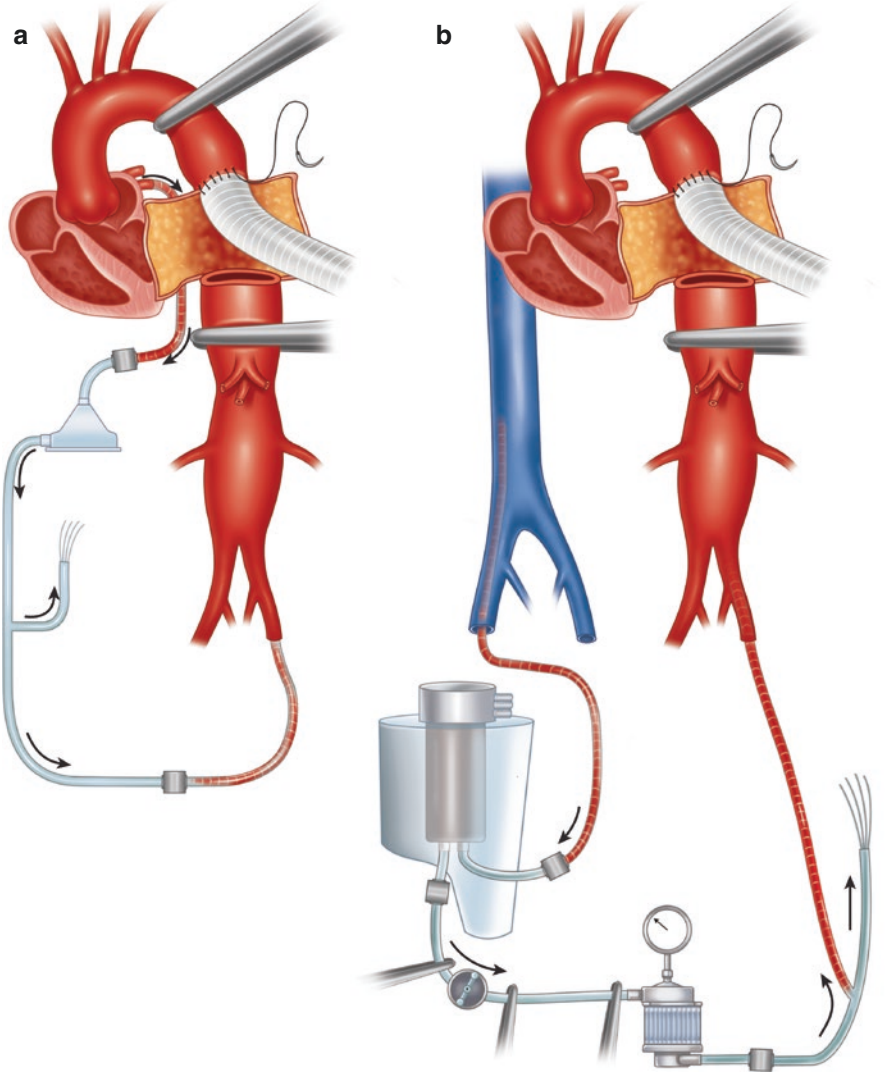


Fig. 20.5 Left and right atrial-femoral bypasses with branch cannulas to reno-visceral vessels

- Method of distal aortic perfusion is selected based on surgeon preference and patient anatomy.
- Regardless of the method chosen, the goal is to reduce ischemic time of the reno-visceral organs, extremities, and the spinal cord, while facilitating various aortic and branch anastomosis.

  Here, we describe the left atrial-femoral artery bypass technique.

- After the aorta and branch vessels have been sufficiency dissected out, the inflow and outflow for distal aortic perfusion should be exposed.

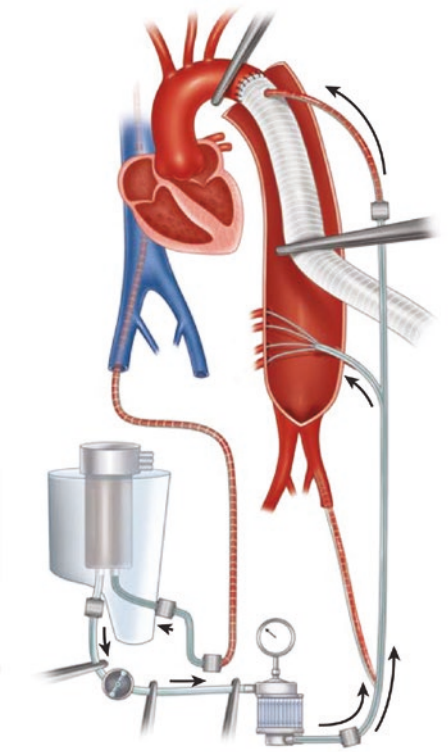


Fig. 20.6 Complete heart bypass with deep hypothermic circulatory arrest

  - Inflow: The left inferior pulmonary vein can be exposed via division of the inferior pulmonary ligament and the pericardium.
  - Outflow: An oblique groin incision can be used to expose the left common femoral artery. Alternately, a portion of the aorta distal to the most distal clamp site can be chosen to provide flow to the lower body. Technique of femoral cannulation is described below.

- Prolene purse-string sutures are then placed at both the inflow and outflow sites.
- The patient is heparinized, although full heparinization is not necessary for partial heart bypass.
- A pulmonary venotomy is made through the purse string suture, and a drainage cannula is inserted and secured via a tourniquet and silk ties.
- The femoral artery is accessed via a micropuncture kit, and serial dilations are performed over a wire to accommodate the arterial cannula, which is similarly secured.
- Both cannulas can then be deaired and connected to the bypass circuit.
- Flow rates from the pulmonary vein to the femoral artery can be adjusted throughout the procedure based on the right femoral arterial line pressure and SSEP/MEP to maintain adequate distal perfusion pressure while the aorta is clamped.

Arterial Reconstruction

- Based on the disease pathology, the visceral and renal vessels can either be re-attached as individual branches or en-bloc as an inclusion patch.
- Prefabricated grafts are available with additional branches for the visceral and renal vessels already attached to the aortic graft.
- Alternately, these grafts can be self-constructed by sewing additional limbs onto the main aortic graft prior to cross-clamping.
  - Main aortic graft is generally made of Dacron as are the celiac/superior mesenteric/and right renal artery bypasses.
  - Ringed (reinforced) polytetrafluoroethylene is commonly used as material for left renal artery bypass to prevent kinking.
- Once ready, the proximal aorta is clamped as the partial heart bypass circuit is started.
- A second aortic clamp is placed on the distal descending aorta. During this time, lower body perfusion is maintained with the bypass circuit that diverts the blood from the left atrium to the femoral artery.
- The isolated portion of the aorta (between the two clamps) is incised, and back-bleeding from small intercostal vessels is controlled by ligating these vessels. Large intercostal vessels are identified and noted for later reimplantation. Back-bleeding from these larger intercostal vessels can be controlled by placing small balloon tipped catheters into their ostia.
- The proximal anastomosis is performed while paying attention to the proper orientation of the branch vessels on the graft to alight with patient's anatomy.
- After completion of the proximal anastomosis, the proximal clamp is shifted on to the graft to allow pressurization and inspection of the proximal anastomosis.
- The distal aortic clamp is sequentially moved distally while each bypass or anastomosis to the reno-visceral vessels is performed.
- During periods of renal ischemia, cold Plegisol® (Pfizer, New York City, New York, USA) solution (contains potassium chloride, sodium chloride, calcium chloride, and magnesium chloride) or cold blood is infused into the kidneys to reduce the risk of renal injury with ischemia.
- Care should be taken to ensure appropriate graft length and orientation to prevent kinking of the branch vessel bypasses in the final resting position of the viscera.
- Finally, the distal clamp is moved to the normal portion of the distal aorta, and the distal anastomosis is performed.
- Selected intercostal arteries can be reimplanted on to the aortic graft, either directly or using a separate 12–14 mm diameter graft.
- Based on the clinical situation, the sequence of the various anastomosis can be altered.

Closure

- Once the reconstruction is complete (Fig. 20.7), the bypass circuit is discontinued. The cannulae are removed. The pulmonary vein opening is closed by tying down the purse string sutures, with additional reinforcement as needed. The femoral arteriotomy is directly repaired to prevent narrowing.
- Protamine can be administered, if desired, for reversal of heparin to help achieve hemostasis.
- The native aorta can then be sewn over the prosthetic graft to reduce the risk of aorto-enteric fistulas.
- Diaphragm is reapproximated to the chest wall.
- Abdominal fascia is closed.

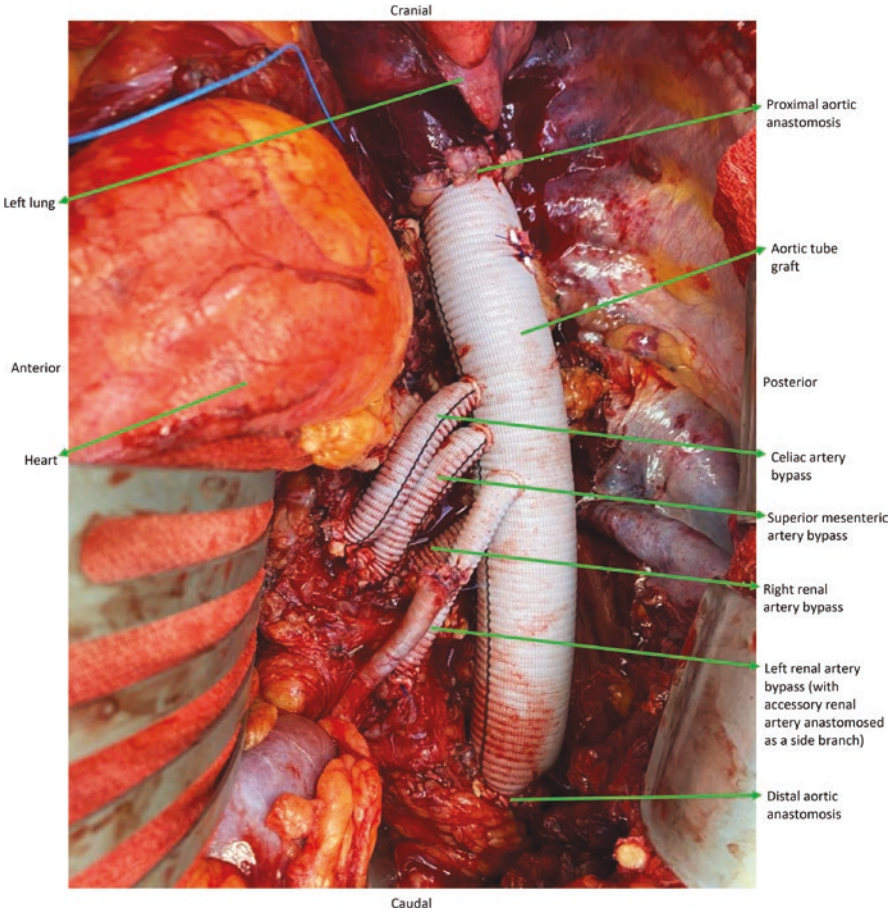


Fig. 20.7 Intraoperative image after replacement of aorta with Dacron tube graft with individual bypasses to the reno-visceral vessels

- Chest tubes are left in the left chest cavity prior to chest closure, which is performed in several layers with reapproximating of the ribs and chest wall musculature.
- Subcutaneous tissue and skin incisions are closed.
- Often, flexible bronchoscopy is performed at the end of the procedure to clear respiratory secretions.
- The patient is transferred to the intensive care unit for further care.

Postoperative Care and Complications

- Patients are initially managed in the ICU postoperatively for close monitoring of hemodynamic, respiratory, and neurologic status.
- Sedation is weaned as tolerated, and a neurologic exam is obtained and monitored to assess for SCI.
- Patients are extubated in the ICU based on clinical progress.
- Cardiac function may need to be optimized with the use of inotropes or vasopressors to maintain adequate cardiac output and perfusion pressure.
- Supplementation with crystalloid/colloids, blood products, and/or vasopressors may be necessary.
- Patients are meticulously monitored for signs of reno-visceral and lower extremity ischemia via physical exam for lower extremity sensation and strength, labs (creatinine, lactate, LFTs, blood glucose levels), development of oliguria/anuria despite adequate resuscitation, and bloody bowel movements.

Spinal Cord Ischemia

- SCI is one of the most feared complications after DTAA and TAAA repairs and requires diligent monitoring for symptoms.
- Patients are at risk of spinal cord ischemia with TEVAR and open surgical repair due to interruption of spinal cord blood supply from the aorta through the segmental intercostal arteries, either due to coverage by the stent graft, or exclusion and ligation during surgical repair.
- The risk for SCI increases as the length of the excluded aortic segment increases due to the increasing number of segmental arteries that are effected.
- Several techniques are implemented to reduce the risk of SCI perioperatively:
  - Augmentation of spinal perfusion pressure (increased MAP goals of at least >90 mmHg and decreased spinal fluid pressure via CSF drainage to maintain a cerebral perfusion pressure (MAP-ICP) of >70 mmHg).
  - Increased oxygen carrying capacity goals (higher hemoglobin levels and oxygen saturation).

  - Intraoperative spinal cord cooling.
  - Although controversial, intravenous corticosteroids and naloxone use.
- Delayed paraplegia can occur (highest 48–72 h postoperatively), so these higher parameters are maintained for several days after the operation.

Bleeding

- Given the complexity of the procedure, with loss of coagulant factors, hemodilution, and possible hypothermia, coagulopathy is not uncommon.
- Chest tube output, hemodynamics and blood hematocrit values should be assessed closely to monitor for bleeding.
- Coagulation profile should be monitored for targeted blood product administration.
- If there is a significant amount of bleeding or continued bleeding despite adequate correction of coagulopathy, return to the OR may be necessary.

Pulmonary

- Pulmonary complications are the most common type of complications after open DTAA and TAAA repairs given the high incidence of smoking history for these patients.
- In addition to appropriately weaning patients toward extubation, they should be optimized with chest physiotherapy, early mobilization, incentive spirometry, and adequate pain control.

Ongoing Surveillance

- All patients who undergo TEVAR or those with remaining dissection or diseased segment of the aorta must be followed lifelong with surveillance imaging.
- Ongoing medical management and risk factor modification must continue.

Further Reading

Acher C, Wynn M. Outcomes in open repair of the thoracic and thoracoabdominal aorta. J Vasc Surg. 2010;52:3S. https://doi.org/10.1016/j.jvs.2010.06.137.

Acher CW, Wynn M. A modern theory of paraplegia in the treatment of aneurysms of the thoracoabdominal aorta: an analysis of technique specific observed/expected ratios for paralysis. J Vasc Surg. 2009;49:1117–24.

Hiratzka LF, Bakris GL, Beckman JA, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/sir/STS/SVM guidelines for the diagnosis and management of patients with thoracic aortic disease. Circulation. 2010;121 https://doi.org/10.1161/cir.0b013e3181d4739e.

Svensson LG, Crawford ES, Hess KR, Coselli JS, Safi HJ. Experience with 1509 patients undergoing thoracoabdominal aortic operations. J Vasc Surg. 1993;17:357–70.

Svensson LG, Kouchoukos NT, Miller DC, et al. Expert consensus document on the treatment of descending thoracic aortic disease using endovascular stent-grafts expert consensus document on the treatment of descending thoracic aortic disease using endovascular stent grafts has been supported by unrestricted educational grants from Cook, Inc. and Medtronic, Inc. Ann Thorac Surg. 2008;85:S1. https://doi.org/10.1016/j.athoracsur.2007.10.099.

Chapter 21
Acute Aortic Syndromes

Alexander A. Brescia and Bo Yang

Definition and Outcomes

The aorta exits the heart at the aortic valve and has both thoracic and abdominal components. The aortic wall consists of three layers. The adventitia is the outermost layer and provides strength through connective tissue cells, the media contains smooth muscle cells, and the intima consists of endothelial cells. The vasa vasorum provides blood supply directly to the aortic wall through tunica media and tunica externa cells.

Acute aortic syndromes cover a spectrum of pathology which includes intramural hematoma (IMH), penetrating aortic ulcer (PAU), and aortic dissection (AD). All three conditions can present as either acute or chronic processes and may affect any part of the aorta. The three pathologies are interrelated with similar characteristics, but varying levels of stability.

AD is the most common subset of acute aortic syndrome and occurs through a tear in the intima into the outer third of the media. The tear results in a separation of the layers of the media, creating a true and false lumen (Fig. 21.1). Pulsatile flow can enter the false lumen which may propagate the dissection either forward (antegrade) or backward (retrograde) from the tear, as well as re-entry tears which may develop elsewhere along the aorta.

IMH and PAU are rarer acute aortic syndromes and usually have absent luminal flow. IMH may arise from spontaneous rupture of aortic vasa vasorum or from a thrombosed aortic false lumen, while PAU occurs secondary to atherosclerotic plaque rupture, similar to a coronary plaque rupture, resulting in an intimal defect and pseudoaneurysm with or without blood flow in it [1].

A. A. Brescia (✉) · B. Yang
Department of Cardiac Surgery, University of Michigan, Ann Arbor, MI, USA
e-mail: abrescia@med.umich.edu; boya@med.umich.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_21

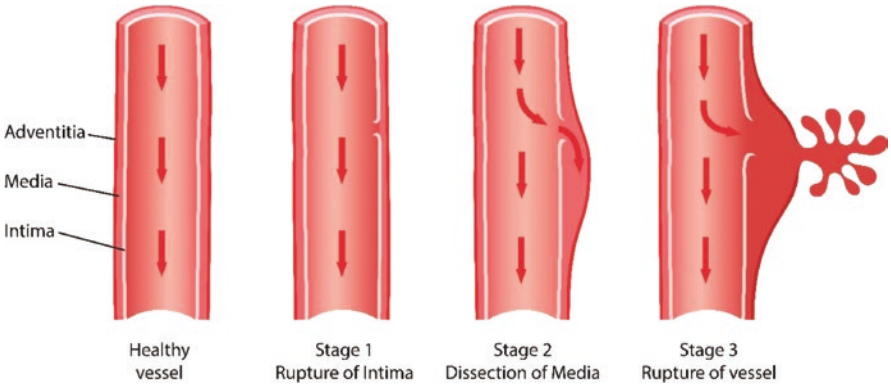


Fig. 21.1 Stages of aortic dissection, initiated by intimal injury (stage 1) and propagation of tear into the outer third of the media (stage 2), potentially progressing to aortic rupture (stage 3)

The establishment of the International Registry of Acute Aortic Dissection (IRAD) database has allowed an international collection of centers to collaborate in assessing the presentation, management, and outcomes of acute AD [2]. The most dangerous, life-threatening acute aortic syndrome is acute type A aortic dissection (ATAAD). ATAAD has an incidence of approximately 10 per 10,000 in the United States and a 1–3% mortality rate/h without surgical repair due to cardiac tamponade, end-organ malperfusion, and aortic rupture. Untreated ATAAD is associated with an over 33% mortality rate in the first 24 h, 50% at 48 h, and 75–90% at 2 weeks. With aggressive medical and surgical treatment, 30-day survival can be as high as 80–90%.

Anatomy and Classification

Classification systems were established for AD but can also be applied to the rarer IMH and PAU based on the location of pathology (Fig. 21.2). While many classification systems exist, the two most common are the Stanford and DeBakey classifications (Table 21.1).

Timing of acute AD is extremely important since it can change management strategy. Chronicity is based on the initial intimal tear and symptom onset, according to consensus North American and European guidelines for thoracic aortic disease [4, 5]:

Acute: ≤14 days
Subacute: 15–90 days
Chronic: >90 days

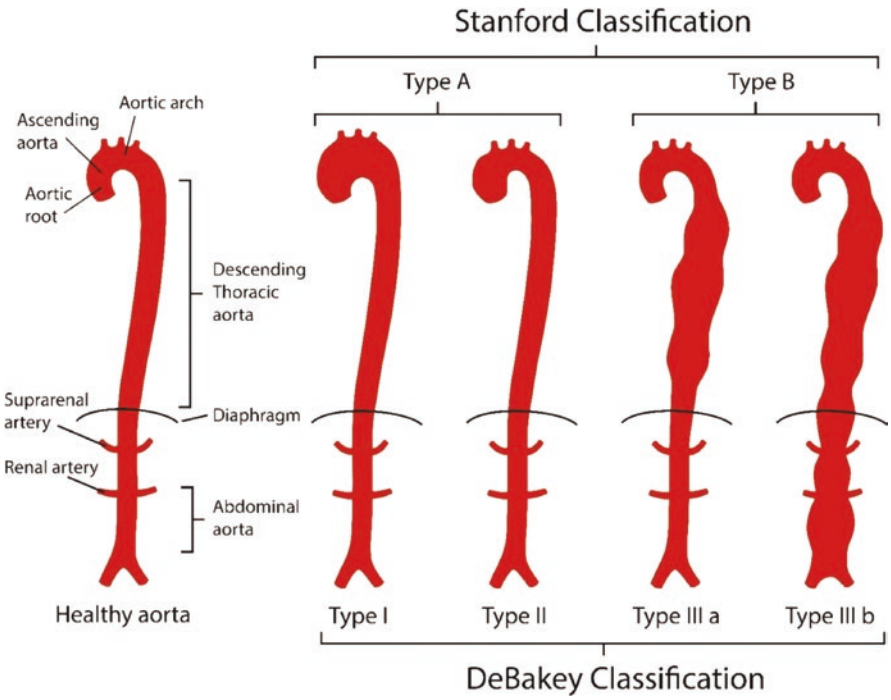


Fig. 21.2 Stanford and DeBakey classifications of aortic dissection (right) and anatomy of a healthy aorta (left)

Table 21.1 Descriptions of Stanford and DeBakey classification systems for aortic dissection

Stanford classification		
Type A	Requires involvement of the ascending aorta	
Type B	Occurring distal to the left subclavian artery	
Non-A, non-B [3]	Dissection involving the aortic arch but not involving the ascending aorta, with or without extension to the descending and abdominal aorta.	
DeBakey classification		
Type I	Involving ascending, arch, and descending aorta	
Type II	Involving ascending only	
Type III	Involving descending aorta only (distal to left subclavian)	
	Type IIIa	Confined to aorta above the diaphragm
	Type IIIb	Extending below the diaphragm

Pathophysiology and Clinical Presentation

A normal aortic diameter is determined by patient age, size, and sex. The ascending, arch, and descending aorta are typically 2–3 cm in diameter. AD occurs as a result of increased wall stress based on Laplace's law, where tension (wall stress) is equal to pressure multiplied by radius, divided by two times the wall thickness ($T = [P \times R]/[2 \times W]$). Any mechanism that increases wall stress beyond its capacity will then predispose that person's aorta to dissection.

AD most commonly occurs in patients aged 60–70 (mean age 63) and has a 3:1 male to female predisposition [1]. The most common risk factor for AD is chronic hypertension, which is present in >75% of cases. Connective tissue diseases are also important risk factors for AD. The three most common are Marfan syndrome, Loeys-Dietz, and Ehlers-Danlos. Connective tissue disorders weaken the elastin and/or collagen layers of the aortic wall media, which predispose the aorta to aneurysm and dissection. Additional risk factors for AD may be divided into direct forces which affect the aortic wall and others that alter the composition of the aortic wall (Table 21.2).

Most commonly the intimal tear in ATAAD begins at the right anterior aspect of the proximal ascending aorta along the greater curvature and may propagate all the way through the iliac arteries. Intimal tears originating in the aortic arch or descending thoracic artery may progress retrograde back to the ascending aorta, which represents an equally if not more dangerous disease. Type B acute ADs most commonly involve an intimal tear just distal to the left subclavian or less frequently from a tear in the abdominal aorta with retrograde progression. ADs tend to occur toward the outer layers of the media, making the outer wall of the false lumen thinner than the intimal flap and dependent upon the adventitia for its strength. All vessels arising

Table 21.2 Risk factors for aortic dissection through mechanisms of impacting wall tension (left) versus altering aortic wall composition (right)

Risk factors for aortic dissection	
Impacting wall tension	Altering aortic wall composition
• Chronic hypertension (↑ pressure) • Increased age • Male sex • Pre-existing aortic aneurysms (↑ radius) • Iatrogenic: cardiac surgery, catheter-based therapies • Pregnancy-related: rare, concomitant connective tissue disorder • Miscellaneous: drugs, trauma, weightlifting	• Marfan syndrome: autosomal dominant (1:5000), fibrilin-1 defect on chromosome 15; Ghent criteria • Ehlers-Danlos: auto dom, type IV most common, mutation in COL3A1 gene which encodes type III collagen • Loeys-Dietz: auto dom, mutations in TGF-β receptors 1 and 2 • Familial thoracic aortic aneurysm and dissection (FTAAD): genes may include MYH11, ACTA2, PRKG1, and TGF-β 1 and 2 • Hereditary causes: bicuspid AV associated with NOTCH-1 gene; aortic coarctation • Turner syndrome: 45X or 45XO • Inflammatory and autoimmune diseases: giant cell arteritis, Takayasu, rheumatoid arthritis, syphilis

from the aorta including the coronaries, aortic arch vessels, intercostal arteries, visceral vessels, and iliac arteries may be sheared off the lumen, occluded by the dissecting media, stay in communication with the false lumen, or remain uninvolved. The false lumen may rupture, re-communicate with the true lumen by re-entry tears, thrombose, or remain intact, leading to future aneurysm formation [1].

The Oxford Vascular Study (OXVASC) found that approximately 50% of acute AD patients died before being diagnosed in a hospital [6]. Heightened suspicion assists early diagnosis as many patients are initially thought to have a different diagnosis. The most common symptom is severe pain, either mid-sternal in location for ascending aortic dissections or inter-scapular for descending aortic dissections, while painless AD can also occur in those with pre-disposing chronic aortic aneurysms. Patients may present with malperfusion corresponding to disruption of any vessels arising from the aorta. This may manifest as myocardial ischemia, stroke, mesenteric ischemia, renal failure, paraplegia, or acute limb ischemia.

IMH most frequently occurs in the descending aorta and in older patients. The most common presentation of acute IMH is chest or back pain, whereas malperfusion and pulse deficits are less likely to be present compared with ADs. PAUs are also most commonly in the descending aorta, which reflects the most common area of the aorta subject to atherosclerotic changes. Patients with acute PAU are typically elderly with hypertension and diffuse, severe atherosclerosis, who present with chest or back pain.

Diagnosis

Most patients with acute aortic syndromes undergo routine testing in the setting of acute chest pain, such as chest X-ray, EKG, and laboratory tests. For acute AD, CXR often shows an abnormality such as a widened mediastinum, right tracheal deviation, or a calcium sign. EKGs are typically normal unless there is coronary involvement. Troponins are frequently mildly elevated, and D-dimers have an extremely high negative predictive value for acute AD in the first 24 h. The mainstays for diagnostic imaging in acute aortic syndromes are as follows:

CT Scan

- Pros: fast, differentiates AD/IMH/PAU and defining anatomy, non-invasive
- Cons: motion/streak artifact, IV contrast, radiation

Transesophageal Echocardiogram (TEE)

- Pros: proximal aorta visualization, fast, LV function assessment
- Cons: semi-invasive, distal asc/prox arch blind spot, operator dependent, sedation

MRI

- Pros: no radiation, differentiates AD/IMH/PAU, gadolinium instead of contrast, assesses AI and LV function
- Cons: availability, PPM contraindication, expensive, slow

Differentiating between AD, IMH, and PAU in the setting of acute aortic syndromes is often achieved by analyzing radiographic differences on CT scan. AD will include an intimal tear and a false and true aortic lumen, with the potential for additional re-entry tears into the true lumen. Diagnosing IMH is based on the presence of thrombus in the aortic wall, but with no blood flow in the false lumen. IMHs are distinguished from AD by the absence of either a definable dissection flap or communication between the true and thrombosed false aortic lumen. Noncontrast CT must be assessed for IMG and will show the thickened aortic wall with a higher density than unenhanced blood on CT and is without enhancement on contrast views of CT or MRI. IMH patients with an ascending aorta >5 cm or IMH thickness of >10 mm is at increased risk of complications and mortality. PAU when viewed tangentially is a mushroom-like outpouching (pseudoaneurysm) of the aortic lumen with overhanging edges, resembling the pedunculated appearance of a gastric ulcer. Young patients (<50 years old) with sudden onset chest pain or upper back pain should be treated as aortic dissection until proven otherwise.

Indications for Surgery

Ascending Aorta

An acute aortic syndrome involving the ascending aorta is classically considered a surgical emergency, with ATAAD the most serious pathology, requiring prompt diagnosis and treatment. Upon diagnosis, medical management should begin immediately, with a focus on limited aortic wall stress, which is affected by the force and rate of contraction (dP/dT). This can be achieved by limited systemic blood pressure typically to a systolic range of 90–110 as well as heart rate. This is most readily addressed with a beta blocker, which is first-line therapy. Esmolol has a shorter half-life and is often chosen as an initial agent. Second- and third-line treatments in addition to beta blocker or instead of them in cases of profound bradycardia include nicardipine, sodium nitroprusside, and fenoldopam. Immediate medical management is paramount to decreasing risk of sudden aortic rupture, false lumen propagation, and dynamic malperfusion. Additional work-up prior to operative repair may include:

1. Blood pressure control (SBP 90–110)
2. Consult to cardiac surgery (prior to arrival, if possible)

3. Confirmation of pathology on cross-sectional imaging, if available
4. IV pain control
5. HPI—medical/surgical history, blood thinners, smoking history, functional status
6. Physical exam—peripheral pulses, murmur suggestive of severe AI, ongoing chest/back/abdominal pain, full neuro exam
7. Blood work—type and cross, CBC, CMP, blood gases with lactate
8. Foley catheter for close urine output monitoring
9. Consent for surgery, if possible

While these steps in work-up should be simultaneously pursued, once a diagnosis of ascending acute aortic syndrome is confirmed, **emergent transport to the operating room must be prioritized above all else.**

The goal of surgery for an acute aortic syndrome of the ascending aorta is to provide life-saving measures by resolving cardiac tamponade, malperfusion, and acute aortic insufficiency as well as preventing aortic rupture. The fundamental approach to achieving these goals is excision of the primary entry tear for AD, followed by reconstruction of the aortic root, ascending aorta, and aortic arch, replacement/repair of the AV to achieve competency, and ensuring patent coronary arteries and aortic arch vessels to maintain myocardial, cerebral, and upper and lower body perfusion (Fig. 21.3). In the absence of cerebral malperfusion, dissected aortic arch branches may not necessarily need to be replaced and reimplanted [7, 8]. The specific operation to achieve these goals depends on each patient's presentation and individual surgeon skill set.

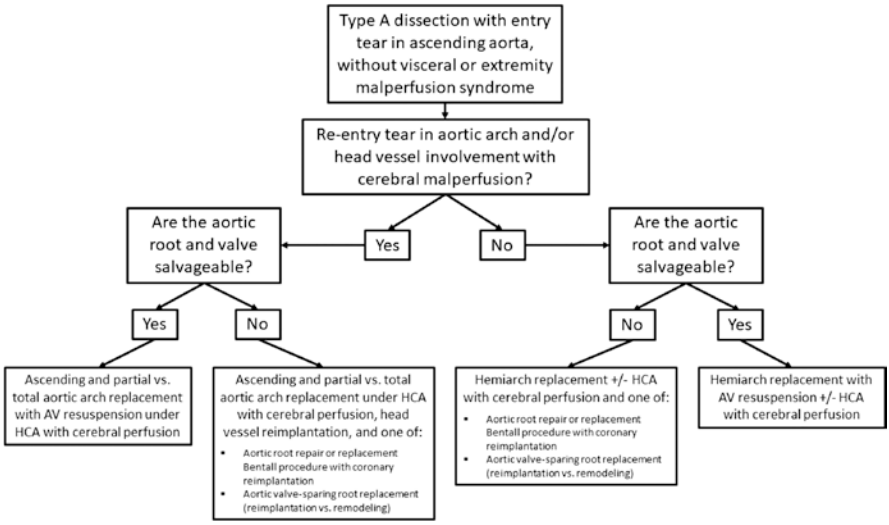


Fig. 21.3 Algorithm for management of type A dissection without extremity or visceral malperfusion

Descending Aorta

Urgent or emergent surgical intervention in the setting of an acute aortic syndrome to the descending aorta is driven by the presence or absence of malperfusion and aneurysm >5.5 cm. Medical management for descending aortic pathology is nearly identical to ascending pathology, with slightly more liberal blood pressure management strategies, often with a goal SBP 90–120 or 90–130. Intervention may be indicated in the setting of continued acute chest, back, or abdominal pain in conjunction with signs of impending rupture, malperfusion, or an aortic aneurysm increasing in size. Acute intervention for descending aortic pathology almost always involves thoracic endovascular aortic repair (TEVAR). The main principle of urgent or emergent TEVAR is to exclude the intimal tear with a covered stent graft, while minimizing serious complications such as paraplegia and renal failure.

Patients with acute aortic pathology distal to the left subclavian artery most frequently undergo aggressive medical management, potential re-imaging, and sometimes eventual elective repair with either TEVAR or an open thoracoabdominal approach. In the absence of acute malperfusion, repair may eventually be indicated due to an aneurysm >6.0 cm, >5.5 cm in patients with connective tissue disease, or growth rate >0.5 cm/year.

Cannulation Strategy

Acute aortic syndromes of the ascending aorta are repaired through a median sternotomy. Venous cannulation for cardiopulmonary bypass is typically achieved with a two-stage cannula through the right atrium, but in some instances may include femoral venous cannulation, particularly in the setting of an unstable or ruptured patient. Arterial cannulation may also depend on hemodynamic stability as well as surgeon preference and patient anatomy and includes many options. In current practice, arch branch vessel cannulation and direct aortic cannulation are becoming more and more popular to achieve antegrade cardiopulmonary bypass compared to femoral artery cannulation.

Femoral

The most common site of arterial cannulation nationwide for ATAAD remains the femoral artery, due to its ease and speed. The non-dissected femoral artery may be chosen based on preoperative CT scan. If imaging is unavailable, then the side with a weaker pulse is more likely to be the true lumen. Drawbacks to femoral cannulation include atheroembolism and stroke due to retrograde cardiopulmonary bypass flow, particularly in the setting of aortic atherosclerosis.

Axillary, Right Subclavian, Innominate, and Other Arch Branch Arterial Cannulation

When intervention on the aortic arch is necessary, arterial cannulation in the right axillary (through a separate axillary cut-down incision) or the innominate or right subclavian artery through median sternotomy access may be preferred. An 8–10 mm Dacron "chimney" or "stovepipe" graft is sutured to the right axillary, intrathoracic right subclavian, or innominate artery which then allows for selective antegrade cerebral perfusion during hypothermic circulatory arrest, required for intervention on the arch. Advantages of this approach include the same access for both arterial cannulation and selective antegrade cerebral perfusion and avoidance of the dissected aorta. Disadvantages include the potentially time-consuming nature of this approach and relative contraindications in the setting of a right aberrant subclavian artery, dissection of these vessels, severe atherosclerosis or calcification, and morbid obesity.

Direct Aortic

Finally, the quickest method of achieving arterial cannulation is through direct ascending aortic cannulation of the true lumen through a modified Seldinger technique over a wire using an elongated one-piece aortic cannula (EOPA). TEE guidance is typically utilized to confirm true lumen cannulation. Selective cerebral perfusion in this setting may then be achieved either with direct antegrade cannulation into arch vessels or retrograde cerebral perfusion through the SVC. Disadvantages include the potential inability to cannulate into the true lumen and the theoretical risk of rupture from cannulating an already dissected aorta, additional time consumption to either establish retrograde cerebral perfusion through the SVC or cannulate arch vessels to establish antegrade cerebral perfusion.

Malperfusion Syndrome

Malperfusion vs. Malperfusion Syndrome

In the setting of acute aortic syndromes particularly ATAAD, it is important to understand the difference between organ malperfusion and malperfusion syndrome. **Malperfusion** is inadequate blood flow to end organs due to dissection-related obstruction of the aorta and its branches, whereas **malperfusion syndrome (MPS)** is tissue necrosis and functional failure of vital organs (e.g., viscera or lower extremity) secondary to late-stage malperfusion [9]. Importantly, the diagnosis of MPS requires both clinical features and laboratory findings compatible with end-organ

Table 21.3 Definition and characteristics of malperfusion syndrome

Malperfusion syndrome (MPS): Clinical features/lab findings compatible with end-organ failure + radiographic findings of dynamic or status obstruction	
Clinical features and laboratory findings	**Radiographic findings**
• Abdominal pain and tenderness • Decreased urine output • Elevated lactate • Abnormal liver/pancreatic enzymes • Abnormal bilirubin/creatinine • Absent peripheral pulses • Motor/sensory deficits in extremities • Neurological deficits • ST segment elevation on EKG	*Dynamic obstruction* • Transient or variable blockage of blood flow due to dissection flap driven by pressure differentials • Resolved through equalization of true and false lumen pressure during open surgical repair of aortic dissection or endovascular fenestration/stenting or TEVAR *Static obstruction* • Fixed blockage of arterial blood flow to an end organ unaffected by differential lumen pressures • Not resolved through open surgical repair of proximal aorta

failure as well as radiographic findings demonstrating dynamic or static findings consistent with low or absent blood flow to the damaged end organs (Table 21.3).

Endovascular Fenestration and Stenting

The Michigan group has developed and pioneered an approach to address life-threatening visceral or extremity malperfusion in the setting of MPS through endovascular fenestration and stenting, then performed delayed open surgical repair of the proximal aorta in the setting of ATAAD [9–11] according to an established algorithm (Fig. 21.4):

The most feared risk to this staged approach is aortic rupture while awaiting resolution of malperfusion-related organ failure prior to open repair. Two decades utilizing this approach has yielded excellent outcomes [9], and other centers have adopted this approach to patients with ATAAD and visceral or extremity MPS [12–17]. However, this approach is only utilized at highly specialized open and endovascular aortic centers and is not standardized across aortic surgical practice, where the typical approach to these patients would still include open surgical repair of the proximal aorta, which continues to result in high operative mortality in this patient population. Both TEVAR and open aortic repair have been used to treat ATAAD with end-organ malperfusion. These strategies in isolation can only resolve dynamic, but not static malperfusion. This is another reason to perform endovascular stenting of branch vessels with static malperfusion in patients with MPS first before open aortic repair.

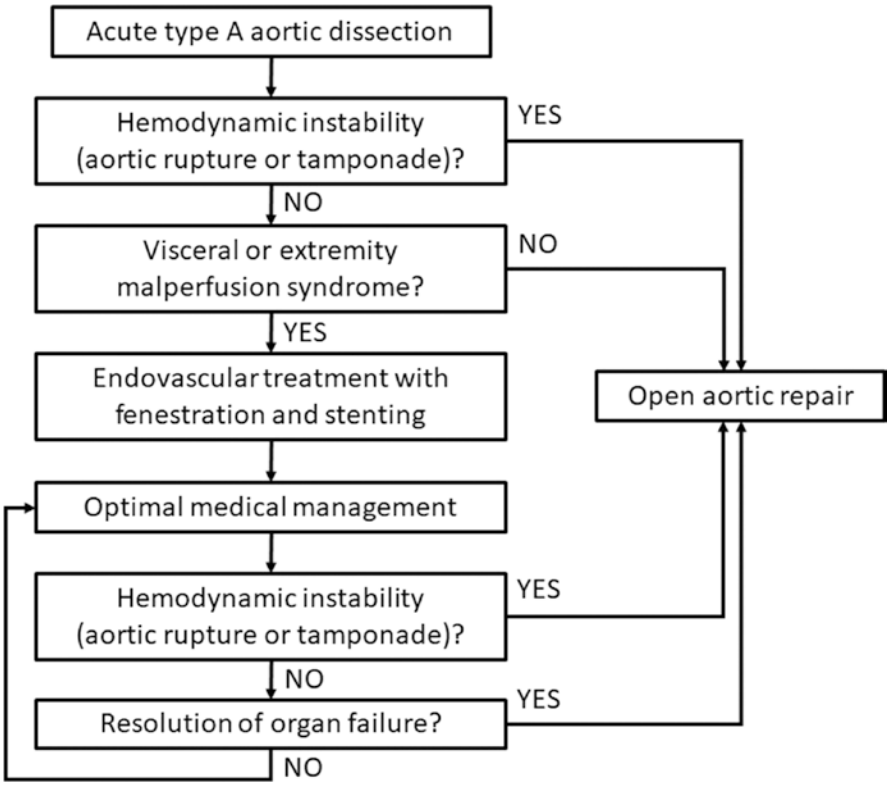


Fig. 21.4 Algorithm for management of acute type A dissection with and without malperfusion

Endovascular Therapy

Endovascular therapy may be utilized to either address the primary or secondary pathology resulting from acute aortic syndromes. Fenestration and stenting to address visceral or extremity MPS in the setting of ATAAD first involve angiographic confirmation of treatable MPS with a significant pressure gradient (>15 mmHg) between the ascending aorta true lumen and a branch artery, then performing fenestration and stenting by creating a tear in the dissection flap to equalize the blood pressure and permit flow between the true and false lumens [10, 11]. If the pressure gradient between the ascending aorta and a dissected branch vessel (e.g., celiac, SMA, or renal arteries) remains >15 mmHg after correction of dynamic obstruction through aortic fenestration/stenting, bare stents may be placed into the branch vessel true lumen past the terminal extent of the dissection to relieve static obstruction, with occasional requirement for thrombolysis, thrombectomy, and embolectomy of true lumen thrombus. Endovascular fenestration and stenting can resolve both dynamic and static malperfusion.

TEVAR therapy has been utilized in both the ascending and descending aorta. For ascending acute aortic syndromes, it is only used in an investigative capacity, due to suboptimal device design, a concerning complication profile (e.g., retrograde dissection, stroke, stent migration, endoleak, and death), and uncertain long-term outcomes [18]. For acute descending aortic syndromes requiring intervention, TEVAR has become the predominant modality of intervention with either straight or branched stent grafts, while open surgical repair is reasonable over TEVAR for patients with connective tissue disorders who have progression of disease despite aggressive medical therapy [19].

References

1. Jabagi H, Brescia AA, Yang B. Type a aortic dissection. In: Brescia AA, Louis C, editors. TSRA review of cardiothoracic surgery. 3rd ed. Chicago: Thoracic Surgery Residents Association; 2022.
2. Evangelista A, Isselbacher EM, Bossone E, et al. Insights from the international registry of acute aortic dissection: a 20-year experience of collaborative clinical research. Circulation. 2018;137:1846–60.
3. Rylski B, Pérez M, Beyersdorf F, Reser D, Kari FA, Siepe M, Czerny M. Acute non-A non-B aortic dissection: incidence, treatment and outcome. Eur J Cardiothorac Surg. 2017;52:1111–7.
4. Hiratzka LF, Bakris GL, Beckman JA, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with thoracic aortic disease. J Am Coll Cardiol. 2010;55:e27–e129.
5. Erbel R, Aboyans V, Boileau C, et al. 2014 ESC guidelines on the diagnosis and treatment of aortic diseases: document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology (ESC). Eur Heart J. 2014;35(41):2873–926.
6. Howard DPJ, Banerjee A, Fairhead JF, Perkins J, Silver LE, Rothwell PM, Oxford Vascular Study. Population-based study of incidence and outcome of acute aortic dissection and premorbid risk factor control: 10-year results from the Oxford Vascular Study. Circulation. 2013;127:2031–7.
7. Norton EL, Wu X, Farhat L, Kim KM, Patel HJ, Deeb GM, Yang B. Dissection of arch branches alone: an indication for aggressive arch management in type A dissection? Ann Thorac Surg. 2020;109:487–94.
8. Norton EL, Wu X, Kim KM, Fukuhara S, Patel HJ, Michael Deeb G, Yang B. Is hemiarch replacement adequate in acute type A aortic dissection repair in patients with arch branch vessel dissection without cerebral malperfusion? J Thorac Cardiovasc Surg. 2021;161:873–884.e2.
9. Yang B, Rosati CM, Norton EL, et al. Endovascular fenestration/stenting first followed by delayed open aortic repair for acute type A aortic dissection with malperfusion syndrome. Circulation. 2018;138:2091–103.
10. Deeb GM, Michael Deeb G, Williams DM, Bolling SF, Quint LE, Monaghan H, Sievers J, Karavite D, Shea M. Surgical delay for acute type A dissection with malperfusion. Ann Thorac Surg. 1997;64:1669–77.
11. Patel HJ, Williams DM, Dasika NL, Suzuki Y, Deeb GM. Operative delay for peripheral malperfusion syndrome in acute type A aortic dissection: a long-term analysis. J Thorac Cardiovasc Surg. 2008;135:1288–95; discussion 1295–6.
12. Di Eusanio M, Trimarchi S, Patel HJ, et al. Clinical presentation, management, and short-term outcome of patients with type A acute dissection complicated by mesenteric malperfusion:

observations from the International Registry of Acute Aortic Dissection. J Thorac Cardiovasc Surg. 2013;145:385–390.e1.
13. Lauterbach SR, Cambria RP, Brewster DC, Gertler JP, Lamuraglia GM, Isselbacher EM, Hilgenberg AD, Moncure AC. Contemporary management of aortic branch compromise resulting from acute aortic dissection. J Vasc Surg. 2001;33:1185–92.
14. Midulla M, Renaud A, Martinelli T, Koussa M, Mounier-Vehier C, Prat A, Beregi J-P. Endovascular fenestration in aortic dissection with acute malperfusion syndrome: immediate and late follow-up. J Thorac Cardiovasc Surg. 2011;142:66–72.
15. Tsagakis K, Konorza T, Dohle DS, Kottenberg E, Buck T, Thielmann M, Erbel R, Jakob H. Hybrid operating room concept for combined diagnostics, intervention and surgery in acute type A dissection. Eur J Cardiothorac Surg. 2013;43:397–404.
16. Yamashiro S, Arakaki R, Kise Y, Inafuku H, Kuniyoshi Y. Management of visceral malperfusion complicated with acute type A aortic dissection. Interact Cardiovasc Thorac Surg. 2015;21:346–51.
17. Goldberg JB, Lansman SL, Kai M, Tang GHL, Malekan R, Spielvogel D. Malperfusion in type A dissection: consider reperfusion first. Semin Thorac Cardiovasc Surg. 2017;29:181–5.
18. Brescia AA, Patel HJ, Likosky DS, et al. Volume-outcome relationships in surgical and endovascular repair of aortic dissection. Ann Thorac Surg. 2019;108(5):1299–306.
19. MacGillivray TE, Gleason TG, Patel HJ, et al. The Society of Thoracic Surgeons/American Association for Thoracic Surgery clinical practice guidelines on the management of type B aortic dissection. J Thorac Cardiovasc Surg. 2022;163:1231–49.

Chapter 22
Aortic Trauma

Jahan Mohebali and H. Davis Waller

- Aortic injury in the trauma patient is highly morbid and confers high mortality—in many cases death occurs at the scene of accident or prior to hospital arrival.
- High mortality in the field results in underestimation of the incidence of major vascular injury in trauma registries.
- A British study of 1203 battlefield injuries in Iraq and Afghanistan found that none of the included personnel who sustained injury to a named vessel in the thorax or abdomen survived; only one survived long enough to undergo surgery [1].
- In one autopsy report, analysis of 552 civilian trauma deaths, penetrating injury was the reported mechanism in 42% of patients, with approximately 80% dying from hemorrhage. The majority of prehospital or immediate deaths from vessel disruption were due to aortic injury (55%), and most (78%) were associated with death within 15 min of injury [2, 3].
- Even in the pediatric population, typically with greater physiologic reserve in the setting of trauma, a thoracic vascular injury with hemodynamic instability has a mortality approaching 100%.
- There has been recent evolution both in the types of aortic trauma seen at major centers and mortality from those injuries. For example, trauma centers are seeing a greater number of patients with blunt thoracic aortic trauma as a result of increased motor vehicle use.
- According to a recent National Trauma Data Base (NTDB) analysis of all trauma admissions from 2002 to 2014, the incidence of all vascular trauma was 2.3%.

J. Mohebali (✉)
Division of Vascular and Endovascular Surgery, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA
e-mail: Mohebali.Jahan@mgh.harvard.edu

H. D. Waller
Department of Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: hdwaller@mgh.org

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_22

10.2% of those patients sustained a thoracic aortic injury and only 2.5% sustained an abdominal aortic injury [4].
- In the same study, mortality for both blunt and penetrating thoracic aortic trauma diminished significantly over the 13 year study period, 46.1–23.7% and 52.4–44.6%, respectively, with a concomitant significant increased use of endovascular stent grafting for both mechanisms of thoracic aortic trauma. There was also improvement in survival after blunt abdominal aortic trauma, 58.3–26.2% with concomitant increased use of endovascular procedures, 1.9–15.9% [4].

Mechanisms of Aortic Trauma

- Mechanisms of aortic trauma can be divided broadly into blunt and penetrating.
- Injuries can be grouped according to aortic zone of injury as we will see for both the thoracic and abdominal aorta.
- Historically and overall, penetrating mechanisms are the most common cause of aortic trauma [5]. In a study by Mattox et al. of 5760 vascular injuries, 14% involved the thoracic aorta and 86% of these injuries were caused by penetrating mechanisms [6].

Mechanisms of Thoracic Aortic Trauma

- In modern civilian practice, the most common cardiovascular injury in the chest is blunt aortic injury (BAI) [5].
- The descending thoracic aorta is the most common location for blunt aortic injury with 98% of these occurring just distal to the left subclavian artery [7].
- 70% of blunt thoracic aortic injuries (BTAI) occur in association with motor vehicle accidents (MVA). Other causes include falls from height and other impacts with a large mass [5, 8].
- These mechanisms create a deceleration whereby a massive shear force is generated as the relatively mobile arch and descending aorta deform around the fixed isthmus.
- In the ascending aorta, blunt force directed to the sternum injures the anterior surface of the vessel, and concomitant aortic injury should be highly suspected in the presence of myocardial contusion, sternal fracture, or dislocation. In these cases, the cardiac apex often rotates posteriorly causing a spiral torsion tear above the aortic valve.
- These injuries ultimately demonstrate a spectrum ranging from slight damage to the aortic intima to full aortic rupture [2] which is captured in a blunt thoracic aortic injury (BTAI) classification scheme as follows: intimal tear (grade 1), large intimal flap or intramural hematoma (grade 2), pseudoaneurysm (grade 3), and rupture (grade 4).

Mechanisms of Abdominal Aortic Trauma

- Among patients undergoing an exploratory laparotomy for traumatic abdominal injuries, vascular trauma was encountered in 14.3% of gunshot injuries, 10% of stabbings, and 3% of blunt injuries [9–11].
- Penetrating trauma remains the most common cause of abdominal vascular injuries, possibly because the abdominal aorta is better tethered and protected from blunt deceleration injury as a result of its retroperitoneal and paraspinous location.
- In urban trauma centers, penetrating injuries account for about 90% of cases and are most often due to gun violence.
- Generally, the vessel is directly injured by a low-velocity missile, whereas blast and high velocity missiles cause injury by both direct contact and shock wave causing transient cavitation [11].
- The infrarenal abdominal aorta in the most commonly injured (45%) followed by suprarenal (37%) and subdiaphragmatic aorta (18%) [12].
- Blunt abdominal aortic injury may be seen more rarely as these injuries likely result in death in the field from hemorrhage and/or associated injuries. A review of blunt abdominal aortic injury in 1997 found that this injury type represents only 5% of aortic injuries [13].
- Generally, three different mechanisms cause blunt vascular trauma in the abdomen:
  - Rapid deceleration as seen with BTAI
  - Direct anteroposterior crushing due to a seat belt or direct impact
  - Lacerations by bony fragments [14]
- Lumbar vertebral chance, compression, or translational fractures should increase the concern for concomitant abdominal aortic injury. Similarly, injury to overlying retroperitoneal organs such as pancreatic transection or duodenal rupture should also raise concern.
- Blunt abdominal aortic injuries were identified in 9% of fatalities associated with MVAs. Of these fatalities, 56% involved transection of the aorta associated with deceleration and hyperflexion with seat belts and nearly all occurred in the infrarenal aorta [13].

Initial Evaluation of Thoracic Aortic Trauma

- The initial evaluation of a patient with thoracic aortic injury who has survived long enough to arrive in the trauma bay begins with the standard A (airway), B (breathing), and C (circulation and access) of the primary survey.
- Important points to note which may raise the specter of aortic injury in the absence of immediately available hard signs are the injury mechanism and con-

comitant injury burden/pattern described above. The vital sign trend from the scene, transport route, and arrival in the bay are also important as injuries that are initially contained may have demonstrated an initial period of hypotension responsive to resuscitation.

- After performing the primary survey, the secondary evaluation must be rapid yet thorough.
- In general, unlike extremity or neck vascular injury which are more likely to present with hard signs (expanding hematoma, thrill or bruit, absent pulse, active hemorrhage), aortic injury which has not resulted in immediate on-scene or en route death is unlikely to present with specific physical exam findings. In most cases, the injury becomes apparent with diagnostic imaging.
- Plain radiography obtained as part of the primary survey is the most commonly utilized initial imaging study following thoracic trauma and should reliably assist in the diagnosis of a large hemo or pneumothorax which can raise suspicion for aortic injury, particularly if present in the left hemithorax. In such cases, tube thoracostomy placement followed by high volume and continuous sanguinous drainage should increase concern for aortic injury.
- BTAI is suggested by a widened mediastinum, apical capping, or loss of the cardiac or aortic arch silhouette. Pathologic mediastinal widening is defined as an 8 cm width mediastinum at the aortic knob or, at the same level, a width that exceeds 25% of the total chest width [15].
  - Consideration must be given to whether the film was obtained as an antero-posterior (AP) or postero-anterior (PA) study with the former exaggerating the size of the mediastinal silhouette, particularly in patients with increased mediastinal adiposity.
  - A chest X-ray is a reasonable screening tool for BTAI, but its sensitivity is only 41% and computed tomographic angiographic (CTA) remains a much better tool for diagnosis in addition to transesophageal echocardiography and intravascular ultrasound (IVUS) in the hybrid operating room.
  - This emphasizes the need to understand the mechanism of injury, as well as the concomitant injury pattern in order to determine whether there is adequate pre-test probability of aortic injury to justify obtaining a CTA.
  - If CTA is equivocal for TAI diagnosis, IVUS is a superior tool for diagnosis when compared to angiography alone [16].

Initial Evaluation of Abdominal Aortic Trauma

- The initial evaluation of a patient with blunt or penetrating abdominal aortic trauma also generally occurs in the trauma bay and may be obvious in cases of free rupture, however, just as in BTAI a high index of suspicion must be maintained in all cases based on the mechanism of injury and concomitant injury pattern.

- A patient may be normotensive on initial evaluation in approximately 18% of cases of penetrating abdominal aortic injuries or blunt injuries where the bleeding is contained in the retroperitoneum or surrounding tissues such as the paraspinous ligaments or diaphragmatic crura [17].
- In unstable patients who are taken straight to the operating room without axial imaging, abdominal aortic injury may first be discovered as a non-expanding or expanding retroperitoneal hematoma on exploratory laparotomy.
- If the patient remains hemodynamically stable after blunt trauma, evaluation should proceed in the typical fashion with performing a trauma survey with focused assessment for trauma (FAST) ultrasound, and ultimately obtaining a CT angiogram which, similar to thoracic aortic injury, has an excellent sensitivity and specificity for abdominal vascular injury.

Indications for Intervention

- The indications for repair of aortic trauma depend on both the mechanism and location of injury.
  - In most cases, penetrating trauma which involves the aorta, regardless of location, will be managed operatively, particularly if there is hemodynamic instability.
  - Blunt traumatic aortic injuries in the thorax and abdomen in a hemodynamically stable patient may be managed nonoperatively in some cases.
  - Specific hard indications for operative intervention in penetrating thoracic trauma include an initial chest tube output of 1500 cc upon placement or persistent chest tube output of 250 cc/h over 3 h.

    In these patients, the decision to proceed to the operating room and, once in the operating room, the chosen surgical exposure may depend on concomitant injuries and a team-based approach and clinical decision-making is paramount.
- For patients with low-risk BTAI (grades 1 and 2), nonoperative management is becoming more widely accepted [18, 19]. However, in any patient with BTAI, aggressive blood pressure control is paramount, whether this is definitive therapy or a bridge to operative intervention.
  - The main goal is to reduce stress on the injured aortic endothelium by suppressing pressure fluctuation. This is accomplished by slowing the heart rate and dampening the pulse pressure.
  - While medical management differs somewhat on an institutional basis, it is generally agreed upon that beta blockade is first line with a goal systolic blood pressure of less than 100 mmHg and a mean arterial pressure (MAP) goal of less than 80 mmHg [20].

  - It is important to note that "permissive hypotension" may be tolerated in patients without evidence of end-organ malperfusion (i.e., adequate urine output, mentating, etc.)
  - If performed adequately, medical management can reduce the risk of rupture after BTAI diagnosis by up to 10.5% [20].

- The Society for Vascular Surgery (SVS) issued guidelines in 2011 for the management of traumatic thoracic aortic injuries.
  - For hemodynamically stable patients with a grade 1 injury (intimal tear), medical management is recommended which includes blood pressure control and serial imaging [21].
  - For all other types of BTAI (grades 2–4), repair is recommended, though in practice, an increasing number of grade 2 injuries is also being managed nonoperatively.
  - In stable patients, thoracic endovascular aortic repair (TEVAR) should be performed within 24 h of admission if possible or at least prior to discharge after consideration of other traumatic injuries [21].

- For abdominal aortic injuries, penetrating trauma usually necessitates operative repair, and the timing is generally either emergent or urgent depending on patient stability.
  - Blunt abdominal aortic injuries are often managed nonoperatively in the hemodynamically stable patient with no other indications for a laparotomy.
  - In a National Trauma Data Bank analysis of 436 patients with blunt abdominal aortic injuries, 90% were managed nonoperatively, 7% were managed with endovascular repair, and only 3% underwent open repair or extra-anatomic bypass [22]. Generally those abdominal aortic injuries managed nonoperatively are small intimal tears, whereas those repaired operatively were either severe aortic injuries with exsanguinating hemorrhage or a large intimal flap/pseudoaneurysm at risk for thrombosis and subsequent embolization or rupture.

- Consideration of abdominal vascular trauma would be incomplete without a thorough knowledge of the anatomy of the abdomen and to that end, the division of the retroperitoneum into three zones:
  - Zone 1 contains the aorta and inferior vena cava (IVC) and runs midline from the aortic hiatus to the sacral promontory.
  - Zone 1 is divided into supramesocolic and inframesocolic areas. These divisions are important because they help to determine the ideal operative incision and exposure.

    The supramesocolic area contains the suprarenal aorta and major branches (celiac axis, superior mesenteric artery, and renal arteries). It also contains the suprarenal cava.
    The inframesocolic area contains the infrarenal aorta and infrarenal IVC.

- Zone 2 is lateral to zone 1 and contains the kidneys, renal vessels, and paracolic gutters.
- Zone 3 is distal to zone 1 and contains the iliac vessels and pelvic retroperitoneum.
- Finally, zone 4 is perihepatic containing the retrohepatic IVC and hepatic veins.

Operative Approach

- The operative approach to a patient with aortic trauma depends on the patient's hemodynamic stability at presentation and the location of injury (thoracic or abdominal).
  - Penetrating thoracic and abdominal aortic trauma is generally managed with an open approach and, depending on patient stability, the operation may be performed in the trauma bay.
  - BTAI is generally managed endovascularly with TEVAR if intervention is deemed necessary.
  - An open approach to a thoracic aortic injury requires either a sternotomy or thoracotomy.
- When deciding which approach to use, one must consider which structures, both vascular and otherwise, may require repair.
  - A median sternotomy will provide access to the heart, proximal great vessels, and anterior mediastinum. The incision can be extended distally to the abdomen or proximally into the periclavicular area or neck. This exposure also allows access to the origin of the innominate and left carotid artery, however, the takeoff of the left subclavian and the distal arch are difficult to reach.
  - The anterolateral thoracotomy is the quickest approach and most likely to be used in the trauma bay. This exposure gives access to the distal arch, proximal left subclavian, and the descending thoracic aorta. Extending this incision across the midline to create a clamshell incision allows access to both pleural spaces, the anterior mediastinum, and nearly all thoracic vascular structures. Extending the lower part of the incision onto paramedian laparotomy can afford access to the entire thoracoabdominal aorta as well.
  - The posterolateral thoracotomy provides exposure of the hemithorax and especially the posterior structures not easily accessible by the anterolateral approach. It is not used as often in trauma as it is in elective thoracic operations.
- An open approach is considered, not only in penetrating thoracic trauma, but also in those patients with BTAI and anatomy unsuitable for TEVAR.
  - The number one reason for TEVAR anatomic incompatibility is an absence of a proximal landing zone for the endograft which is adequate for a "seal" to be obtained.

To achieve this "seal," approximately 40% of patients with BTAI will require coverage of the left subclavian artery which is generally well-tolerated in this population without a subsequent need for carotid-subclavian bypass [23, 24].
Another anatomic consideration which may preclude TEVAR in this population are small diameter, diseased iliofemoral vessels which cannot accommodate the TEVAR delivery device [25].

- If one must proceed to the OR for BTAI, a left thoracotomy through the fourth rib space is usually the optimal incision given the most common location of BTAI is just distal to the left subclavian artery.
  - The initial goal is to obtain proximal and distal control on the injured aorta which can usually be accomplished with a proximal clamp placed between the left common carotid artery and the left subclavian artery; the distal clamp should be placed in the thoracic aorta distal to the injury.
- If possible, a distal aortic perfusion strategy should be employed to maintain arterial blood flow to the abdominal organs and lower extremities.
  - The most common strategy is "left-heart bypass" which is obtained by cannulating the left inferior pulmonary vein to obtain oxygenated blood returning to the heart from the lungs and delivering that blood distally into the thoracic aorta below the distal clamp.
  - If obtaining a proximal clamp position is not possible, then cardiopulmonary bypass with the addition of deep hypothermic circulatory rest may be necessary.
  - Remember, that this patient will likely have polytrauma and is at risk for bleeding from concomitant injuries; as always, the decision to proceed with this repair needs to be made after thorough discussion with all providers in the trauma team because the use of any perfusion strategy requires some degree of systemic anticoagulation and therefore increases the risk of life threatening hemorrhage from other injuries which is even further exacerbated by the need for hypothermia.
- Another consideration when repairing a thoracic aortic injury is protecting the perfusion of the spinal cord to minimize the risk of postoperative paraplegia [26].
  - The most important contributor to spinal cord ischemia is aortic clamping with occlusion of critical arterial branches supplying perfusing the cord.
  - Other considerations include clamp time, aortic segment length excluded by clamping, the level at which the aorta is excluded by clamping, duration of hypotension, CSF pressure, distal aortic pressure, and the volume of intercostal arteries ligated during the repair [27].
  - To combat these variables and lower the rate of paraplegia after open thoracic aortic repair, certain adjuncts like reattachment of intercostal arteries, induced hypothermia, steroid administration, and CSF drainage can be employed,

however, the details of these techniques are beyond the scope of this chapter [25].

- The majority of BTAI in the modern era is and should be managed endovascularly as the risk of spinal cord ischemia and death is lower in all age groups compared with open surgery [28–30].
- A large multicenter retrospective study of 382 patients with BTAI at 9 different level 1 trauma centers found overall aortic-related mortality of 6.5% with aortic-related mortality of 13.1% versus 2.5% of patients treats with an open approach versus TEVAR. In this study, 76.4% of patients who required intervention underwent TEVAR versus 23.6% who underwent an open procedure [31].
- When employing an endovascular approach to thoracic aortic trauma, the procedure should be performed in a hybrid operating room with endovascular imaging capability.
  - Prior to the procedure, the patient is prepped from neck to knees. Femoral access is obtained, and an arch angiogram is shot to confirm the location of injury.
  - An aortogram is obtained to map the anatomy of the arch vessels and identify the proximal landing zone of the graft. Intravascular ultrasound can also be obtained for confirmation and optimal graft diameter sizing.
  - Heparin dosing for systemic anticoagulation depends on concomitant injuries and prior to the procedure should be discussed with all consulting teams.
  - The TEVAR device is then delivered and deployed between seal zones and spanning the injury. The left subclavian artery may be covered if necessary to obtain a proximal seal zone, and additional grafts may be used to extend the aortic coverage distally as indicated.
  - In certain cases, the left subclavian artery may require revascularization; however, in acute emergencies this decision is individualized according to patient anatomy, procedural urgency, and available surgical expertise [21].
- The operative approach to a patient with abdominal aortic trauma depends on the patient's hemodynamic stability and concurrent associated injuries.
  - An extensive study on penetrating abdominal aortic trauma showed that approximately 30% of these patients will present with no measurable blood pressure and up to 21% may require a thoracotomy in the emergency room for proximal control of hemorrhage [5].
  - Another large retrospective study published in 2014 specifically focusing on blunt abdominal aortic injury (BAAI) noted its relatively rare incidence (0.03%) among nearly 400,000 patients with blunt trauma. 47% of these patients are hypotensive on admission with numerous associated injuries including spine fractures, pneumo/hemothorax as well as solid organ, small bowel, and large bowel injuries. The aortic injuries were most commonly without external contour abnormality on CT (intimal injury), followed by free rupture and pseudoaneurysm. Open repair occurred in 43% of all cases with

endovascular repair occurring in 15%. All ruptures were treated with an open operation [32].

- For any patient presenting with abdominal trauma and hemodynamic compromise, peritonitis, or a positive FAST, the operative approach will be open, in most cases without prior axial imaging, and surgeons should be prepared for massive transfusion as well as treating concomitant injuries in the abdomen.
  - Patients are prepped from the neck to knees to accommodate a possible need for thoracotomy or saphenous vein harvest as a conduit in the event of a vascular injury and need for patch or bypass.
  - All of this preparation should be performed, ideally prior to induction of anesthesia, due to the rapid hemodynamic decompensation that may occur following sedation and paralysis.
- There are some advocates of preliminary anterolateral thoracotomy to facilitate a high aortic cross clamp both preserving blood flow to the brain and cardiovascular system; however, in modern practice, with appropriate exposure, a high infradiaphragmatic cross clamp is usually possible even if the left crus of the diaphragm needs to be divided for exposure enhancement.
- There is also the option of using Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA), both in the ED and non-hybrid operative room to obtain proximal aortic control. In ideal circumstances, this balloon should be placed prior to indication allowing for rapid inflation and control should cardiovascular collapse occur as the patient is intubated.
- Once the abdomen has been entered, sites of bleeding should be identified and controlled with either direct pressure or ligation.
- If balloon control is not present, rapid entry into the lesser sac through the gastrohepatic ligament will allow for compression of the supra-celiac aorta against the spine until better exposure is obtained.
- If any prosthetic grafting is planned, enteric spillage should be washed out as best as possible but ultimately in the event of trauma, most vascular surgeons do not consider enteric spillage a contraindication for repair with the most convenient conduit as the time required for autologous vessel harvest may be prohibitive.
- Conversely, for those hemodynamically stable patients with suspected BAAI, a CTA is invaluable for decision-making.
- There are some general rules regarding the choice to intervene and planned intervention depending on which zone of the retroperitoneum contains hematoma.
- Almost all retroperitoneal hematomas due to penetrating abdominal trauma should be explored with an exception made for Zone 4 injuries.
- In the event of blunt abdominal trauma, exploration is rarely required but the retroperitoneal zone classification crystallizes clinical decision-making.
  - Zone 1 injuries should always be explored.
  - Zone 2 and Zone 3 hematomas should rarely be explored due to risk of renal injury and uncontrollable hemorrhage from pelvic fractures.

  - These zones require exploration in the event of blunt abdominal trauma only if the hematoma is expanding, pulsatile, or demonstrates extravasation.

- Knowing the zone of injury also helps with operative planning, specifically which intra-operative maneuvers will be used to facilitate exposure of the injury.
  - A Zone 1 supramesocolic injury to the aorta can be exposed by a left medial visceral rotation (Mattox Maneuver) in which the left line of Toldt (peritoneal reflection) is incised lateral to the descending colon, splenic flexure, and spleen. The stomach, pancreatic tail, spleen, left colon, and kidney are then rotated to the right side of the patient's abdomen to facilitate exposure.
  - If an IVC injury is suspected proximally, the mirror-image exposure is used—the right medial visceral rotation (Cattell-Braasch Maneuver) is accomplished by incising the right-sided peritoneal reflection lateral to the right colon and hepatic flexure and performing a Kocher maneuver of the duodenum. The right and left triangular ligaments as well as the coronary, round and falciform ligaments can also be taken down as part of this exposure to allow for access to the retrohepatic and suprahepatic cava.
  - For Zone 1 inframesocolic injuries, a standard transperitoneal approach can be employed whereby the transverse colon is retracted toward the patient's head, small bowel is placed out of the way on the right side of the abdomen and the base of the mesentery is incised leading to the aorta.

- Endovascular management has a role in selected cases of infrarenal aortic injury.
  - In the immediate management of a patient with blunt abdominal trauma and suspected BAAI in the trauma bay, REBOA has been shown in some studies to be a viable alternative to open aortic occlusion in capable centers [33]. However, these recommendations have been questioned by a recent large multicenter trauma database analysis which found higher rates of acute kidney injury and lower extremity amputations among patients who undergo REBOA when compared to matched patients who do not [34]. Questions remain as to which patient population will benefit from its use and the appropriate timing of use.

- With regard to the decision to employ endovascular treatment for BAAI, there are some helpful classification systems for both identifying the injury and its location in the infrarenal aorta.
  - The identification of injury is based on CT imaging showing the presence of external contour abnormality or no contour abnormality.

    Among those injuries with no contour abnormality, injury is classified as either "intimal tear" or "large intimal flap (LIF)."
    Intimal tears are defects and/or thrombus <1 cm in length or width, whereas LIFs are >1 cm in length or width or intramural hematoma.

  - More severe injuries include pseudoaneurysm with associated external contour abnormality and aortic rupture.

- Zone of injury is classified according to the feasibility of endovascular repair.

  Zone 1 extends from the diaphragm to just proximal to the SMA ostium.
  Zone 2 includes the ostia of the SMA and renal arteries.
  Zone 3 refers to the infrarenal aorta extending to the bifurcation [32].

- In the largest published BAAI series, intimal tears were generally managed nonoperatively. LIFs were managed operatively (55.3%) or nonoperatively (44.7%). Pseudoaneurysms were generally managed operatively in 2/3 of cases. All endovascular repairs were performed in zones 1 (29.4%) or 3(70.5%) [32].The authors of this study concluded with the following recommendations:
  - Timing of repair is based on hemodynamic stability and other injuries. Open repair and endovascular repair with stent graft or branch vessel embolization are appropriate.
  - Intimal tears: manage with antiplatelet agent and beta-blockers with interval CTA within 30 days.
  - LIF: if uncomplicated, i.e., patient stable without thrombus, aneurysmal degeneration, or pseudoaneurysm formation, manage similarly to intimal tear with interval CTA at 48 h.
  - Progression of LIF, i.e., complicated LIF (intraluminal thrombus, aneurysmal degeneration, pseudoaneurysm formation), manage endovascularly when possible.
  - Injuries to the aortic zone 1 and zone 3 are amenable to endovascular repair especially if concomitant gross contamination, which alone should strongly suggest an endovascular repair if possible.
  - Antiplatelet agents should be continued for 6 weeks post-injury if no contraindications exist.
  - If cases are managed nonoperatively, then follow-up CTA should be obtained at 1 month, 6 months, 1 year, and annually thereafter or until injury is resolved [32].

References

1. Stannard A, Brown K, Benson C, Clasper J, Midwinter M, Tai N. Outcome after vascular trauma in a deployed military trauma system. Br J Surg. 2011;98:228–34.
2. Dosios TJ, Salemis N, Angouras D, Nonas E. Blunt and penetrating trauma of the thoracic aorta and aortic arch branches: an autopsy study. J Trauma Acute Care Surg. 2000;49:696–703.
3. MacLeod JB, Cohn SM, Johnson EW, McKenney MG. Trauma deaths in the first hour: are they all unsalvageable injuries? Am J Surg. 2007;193:195–9.
4. Branco BC, Musonza T, Long MA, Chung J, Todd SR, Wall MJ Jr, Mills JL Sr, Gilani R. Survival trends after inferior vena cava and aortic injuries in the United States. J Vasc Surg. 2018;68(6):1880–8.
5. Demetriades D, Theodorou D, Murray J, et al. Mortality and prognostic factors in penetrating injuries of the aorta. J Trauma Acute Care Surg. 1996;40:761–3.
6. Mattox K, Feliciano D, Burch J, Beall A, Jordan G, De Bakey M. Five thousand seven hundred sixty cardiovascular injuries in 4459 patients. Epidemiologic evolution 1958 to 1987. Ann Surg. 1989;209:698.

7. Demetriades D, Velmahos GC, Scalea TM, et al. Diagnosis and treatment of blunt thoracic aortic injuries: changing perspectives. J Trauma Acute Care Surg. 2008;64:1415–9.
8. Mattox KL. Red river anthology. J Trauma Acute Care Surg. 1997;42:353–68.
9. Demetriades D, et al. Selective nonoperative management of gunshot wounds of the anterior abdomen. Arch Surg. 1997;132:178–83.
10. Feliciano DV, et al. Abdominal vascular injury. In: Mattox KL, et al., editors. Trauma. New York: McGraw-Hill; 2000. p. 783–806.
11. Cox EF. Blunt abdominal trauma: a 5-year analysis of 870 patients requiring celiotomy. Ann Surg. 1984;199:467–74.
12. Deree J, Shenvi E, Fortlage D, et al. Patient factors and operating room resuscitation predict mortality in traumatic abdominal aortic injury: a 20-year analysis. J Vasc Surg. 2007;45:493–7.
13. Roth SM, Wheeler JR, Gregory RT, et al. Blunt injury of the abdominal aorta: a review. J Trauma Acute Care Surg. 1997;42:748–55.
14. Demetriades D, Benjamin ER, Inaba K. Vascular trauma: abdominal. In: Rutherford's vascular surgery and endovascular therapy. 10th ed. Saunders Elsevier. 2023;2411–29.
15. Mirvis SE, Bidwell JK, Buddemeyer EU, et al. Value of chest radiography in excluding traumatic aortic rupture. Radiology. 1987;163:487–93.
16. Azizzadeh A, Valdes J, Miller CC, et al. The utility of intravascular ultrasound compared to angiography in the diagnosis of blunt traumatic aortic injury. J Vasc Surg. 2011;53:608–14.
17. Lopez-Viego MA, et al. Penetrating abdominal aortic trauma: a report of 129 cases. J Vasc Surg. 1992;16:332–5.
18. Demetriades D, Velmahos GC, Scalea TM, et al. Blunt traumatic thoracic aortic injuries: early or delayed repair—results of an American Association for the Surgery of Trauma prospective study. J Trauma Acute Care Surg. 2009;66:967–73.
19. Rabin J, DuBose J, Sliker CW, O'Connor JV, Scalea TM, Griffith BP. Parameters for successful nonoperative management of traumatic aortic injury. J Thorac Cardiovasc Surg. 2014;147:143–50.
20. Fabian TC, Davis KA, Gavant ML, et al. Prospective study of blunt aortic injury: helical CT is diagnostic and antihypertensive therapy reduces rupture. Ann Surg. 1998;227:666–76.
21. Lee WA, Matsumura JS, Mitchell RS, Farber MA, Greenberg RK, Azizzadeh A, Murad MH, Fairman RM. Endovascular repair of traumatic thoracic aortic injury: clinical practice guidelines of the Society for Vascular Surgery. J Vasc Surg. 2011;53(1):187–92.
22. De Mestral C, et al. Associated injuries, management, and outcomes of blunt abdominal aortic injury. J Vasc Surg. 2012;56:656–60.
23. DuBose JJ, Leake SS, Brenner M, et al. Contemporary management and outcomes of blunt thoracic aortic injury: a multicenter retrospective study. J Trauma Acute Care Surg. 2015;78:360–9.
24. McBride CL, DuBose JJ, Miller CC, et al. Intentional left subclavian artery coverage during thoracic endovascular aortic repair for traumatic aortic injury. J Vasc Surg. 2015;61:73–9.
25. Arbabi CN, Azizzadeh A. Thoracic vascular trauma. In: Rutherford's vascular surgery and endovascular therapy. 10th ed. Saunders Elsevier. 2023;2397–410.
26. Gharagozloo F, Larson J, Dausmann MJ, Neville RF, Gomes MN. Spinal cord protection during surgical procedures on the descending thoracic and thoracoabdominal aorta: review of current techniques. Chest. 1996;109:799–809.
27. Safi HJ, Miller CC, Carr C, Iliopoulos DC, Dorsay DA, Baldwin JC. Importance of intercostal artery reattachment during thoracoabdominal aortic aneurysm repair. J Vasc Surg. 1998;27:58–66.
28. Xenos ES, Abedi NN, Davenport DL, Minion DJ, Hamdallah O, Sorial EE, et al. Meta-analysis of endovascular vs open repair for traumatic descending thoracic aortic rupture. J Vasc Surg. 2008;48:1343–51.
29. Tang GL, Tehrani HY, Usman A, Katariya K, Otero C, Perez E, et al. Reduced mortality, paraplegia, and stroke with stent graft repair of blunt aortic transections: a modern meta-analysis. J Vasc Surg. 2008;47:671–5.
30. Neschis DG, Scalea TM, Flinn WR, Griffith BP. Blunt aortic injury. N Engl J Med. 2008;359:1708–16.

31. Azizzadeh A, Keyhani K, Miller CC, Coogan SM, Safi HJ, Estrera AL. Blunt traumatic aortic injury: initial experience with endovascular repair. J Vasc Surg. 2009;49:1403–8.
32. Shalhub S, Starnes BW, Brenner ML, Biffl WL, Azizzadeh A, Inaba K, Skiada D, Zarzaur B, Nawaf C, Eriksson EA, Fakhry SM, Paul JS, Kaups KL, Ciesla DJ, Todd SR, Seamon MJ, Capano-Wehrle LM, Jurkovich GJ, Kozar RA. Blunt abdominal aortic injury: a Western Trauma Association multicenter study. J Trauma Acute Care Surg. 2014;77(6):879–85.
33. DuBose JJ, Scalea TM, Brenner M, et al., AAST AORTA Study Group. The AAST prospective Aortic Occlusion for Resuscitation in Trauma and Acute Care Surgery (AORTA) registry: data on contemporary utilization and outcomes of aortic occlusion and resuscitative balloon occlusion of the aorta (REBOA). J Trauma Acute Care Surg. 2016;81(3):409–19.
34. Joseph B, Zeeshan M, Sakran JV, Hamidi M, Kulvatunyou N, Khan M, O'Keeffe T, Rhee P. Nationwide analysis of resuscitative endovascular balloon occlusion of the aorta in civilian trauma. JAMA Surg. 2019;154(6):500–8.

Chapter 23
Acute and Chronic Pulmonary Embolism

Andrea L. Axtell, Cameron D. Wright, and Nathaniel B. Langer

Learning Objectives
- Diagnostic criteria of acute PE.
- Indications for surgery for acute PE.
- Operative technique (brief).
- Etiologies of chronic PE.
- CTEPH.
- Indications for PTE.
- Operative technique (brief).

Introduction

Acute pulmonary embolism (PE) is a major cause of morbidity and mortality resulting in more than 630,000 symptomatic episodes in the United States annually. It is the third most common cause of cardiovascular death among hospitalized adults behind ischemic heart disease and stroke [1]. The majority of PEs are associated with lower extremity deep venous thrombosis (DVT). These blood clots can embolize and enter the pulmonary circulation via the right heart where they lodge in a pulmonary artery (PA). The clot subsequently propagates as a result of stasis and the

A. L. Axtell · C. D. Wright (✉)
Divisions of Thoracic Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: cdwright@mgh.harvard.edu

N. B. Langer
Division of Cardiac Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: NLANGER@mgh.harvard.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_23

activation of platelets and local endothelial cells resulting in pulmonary vasoconstriction and increased pulmonary vascular resistance. This increases right ventricular (RV) afterload, which may cause RV dilation and dysfunction resulting in decreased cardiac output, systemic hypotension, and shock. Several treatment options are available depending on the patient's presentation, ranging from systemic anticoagulation in hemodynamically stably patients to systemic thrombolysis, catheter-directed therapy, or surgical embolectomy in patients with submassive (RV dysfunction or myocardial injury without shock) and massive PE (RV dysfunction with shock and end-organ hypoperfusion).

Patients with prior pulmonary embolism who do not have complete fibrinolysis of their clot may develop chronic thromboembolic pulmonary hypertension (CTEPH). This results from residual clot that remodels into scar and narrows or occludes the pulmonary arteries resulting in progressive pulmonary hypertension and RV dysfunction even in the absence of recurrent pulmonary emboli. The estimated incidence of CTEPH after a documented pulmonary embolism is 4–5% [2]. The gold standard for management of CTEPH is pulmonary thromboendarterectomy (PTE).

Acute Pulmonary Embolism

Presentation and Diagnosis

- The presentation of acute PE is heterogeneous and ranges from asymptomatic to sudden death. Patients may present with dyspnea, tachycardia, hypoxia, pleuritic chest pain, or cough. Hemodynamic stability is evaluated with close attention to signs of shock and RV dysfunction.
- The history should also assess risk factors for thrombosis summarized by Virchow's triad—venous stasis, endothelial injury, and hypercoagulability.
- The Wells and Geneva scoring systems are clinical risk predictive models that may be combined with clinical findings to assess the pretest probability for PE.
- EKG: The most common finding is sinus tachycardia. May also demonstrate non-specific T-wave and ST-segment changes. The classic pattern of S1Q3T3 (S wave in lead I, Q and T waves in lead III) is not common but highly specific.
- D-dimer: A fibrin degradation product that indicates fibrinolysis. The D-dimer will be elevated in acute PE or DVT.
- CT pulmonary angiogram: Considered the gold standard for the diagnosis of acute PE with a sensitivity of 90–95% and specificity of 100%. May be used to further evaluate the location and extent of clot burden (Fig. 23.1).
- Echocardiogram: Can assess right heart strain (RV dilation, septal flattening, IVC congestion) and dysfunction. Transthoracic echo (TTE) cannot image the pulmonary arteries directly but transesophageal echo (TEE) may identify thrombus in the main pulmonary artery or central branches.

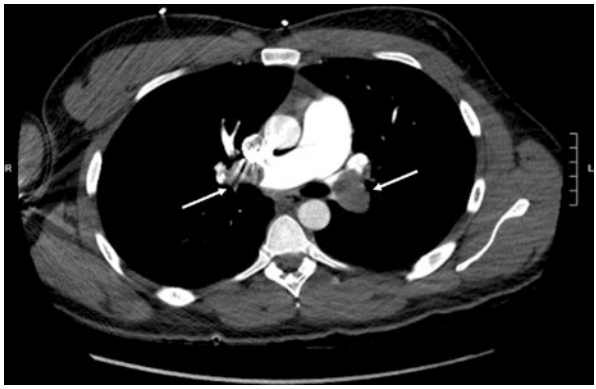


Fig. 23.1 Computed tomography pulmonary angiogram (CT-PA) demonstrating large occlusive clot within the proximal right and left main pulmonary arteries (arrows)

Risk Stratification

- After the diagnosis of acute PE has been established, rapid risk stratification is critical in guiding appropriate management. PE is classified into three main categories: low risk, intermediate risk or submassive PE, and high-risk or massive PE [3].
- Low risk: Patients are normotensive with normal RV function and cardiac biomarkers (troponin, pro-BNP). They have an excellent prognosis once anticoagulation is established.
- Intermediate risk (submassive): Patients have evidence of RV dysfunction and/or myocardial injury (elevated troponin/BNP) but without hypotension or shock.
- High risk (massive): Patients are persistently hypotensive (systolic blood pressure [SBP] <90 mmHG or a decrease in SBP >40 mmHg for ≥15 min), with RV dysfunction and signs of obstructive shock and end-organ hypoperfusion including altered mental status, cold/clammy skin, oliguria, increased lactate, or cardiac arrest. Mortality in this group can be as high as 30–50%.

Treatment of Acute Pulmonary Embolism

- Low risk: Anticoagulation with low-molecular-weight heparin (LMWH) or direct acting oral anticoagulants (DOACs) is considered first-line treatment in patients with low-risk PE.
- Intermediate risk: Systemic anticoagulation is recommended over systemic or catheter-directed thrombolysis in most patients with intermediate risk PE.
  - The pulmonary embolism thrombolysis trial showed that patients treated with thrombolysis had less hemodynamic deterioration but no improvement in mortality and an increased risk of major bleeding (6%) and intracranial hemorrhage (2%) [4].

  - Intermediate risk patients deemed at increased risk of impending clinical deterioration (based on vital signs, severity of RV dysfunction, or gas exchange) may be considered for additional interventions including catheter-directed thrombolysis or catheter embolectomy.

- High risk: Management begins with cardiorespiratory stabilization, which may include extracorporeal membrane oxygenation (ECMO) in patients with profound hemodynamic compromise. Systemic thrombolysis remains the mainstay of therapy. In patients with contraindications to thrombolysis or in whom cardiogenic shock is likely to precede the effect of systemic thrombolysis, catheter-directed therapy or surgical embolectomy can be considered.

Indications for Surgery

- Surgical pulmonary embolectomy should be considered in patients with intermediate or high-risk PE with any of the following:
  - Profound RV failure and cardiogenic shock.
  - Treatment failure of systemic thrombolysis.
  - Contraindication to thrombolysis (history of intracranial hemorrhage, intracranial mass or aneurysm, stroke within the past 3 months, major surgery within the last month, brain or spinal surgery within the past 2 months).
  - Patent foramen ovale.
  - Pregnancy.
  - Thrombus in transit within the right atrium or ventricle.

Operative Technique for Pulmonary Embolectomy

- A median sternotomy is performed, and bicaval and ascending aortic cannulation are established. If additional cardiac procedures are required (i.e., extraction of intracardiac thrombus, PFO closure), cardioplegic arrest and aortic cross-clamping may be required.
- Once cardiopulmonary bypass (CPB) is established, a longitudinal incision is made in the main PA 1–2 cm from the pulmonary valve which can be extended to the proximal left PA.
- Clot is carefully extracted using forceps, suction catheters, or Fogarty balloon catheters (Fig. 23.2).
- A separate incision in the right PA can be made longitudinally between the SVC and the aorta to remove additional thrombus located in the right PA beyond the bifurcation.
- If necessary, additional maneuvers can be performed to remove more peripheral thrombi. This includes opening the pleural spaces bilaterally and manually compressing each lung to mobilize additional clot and retrograde perfusion via the

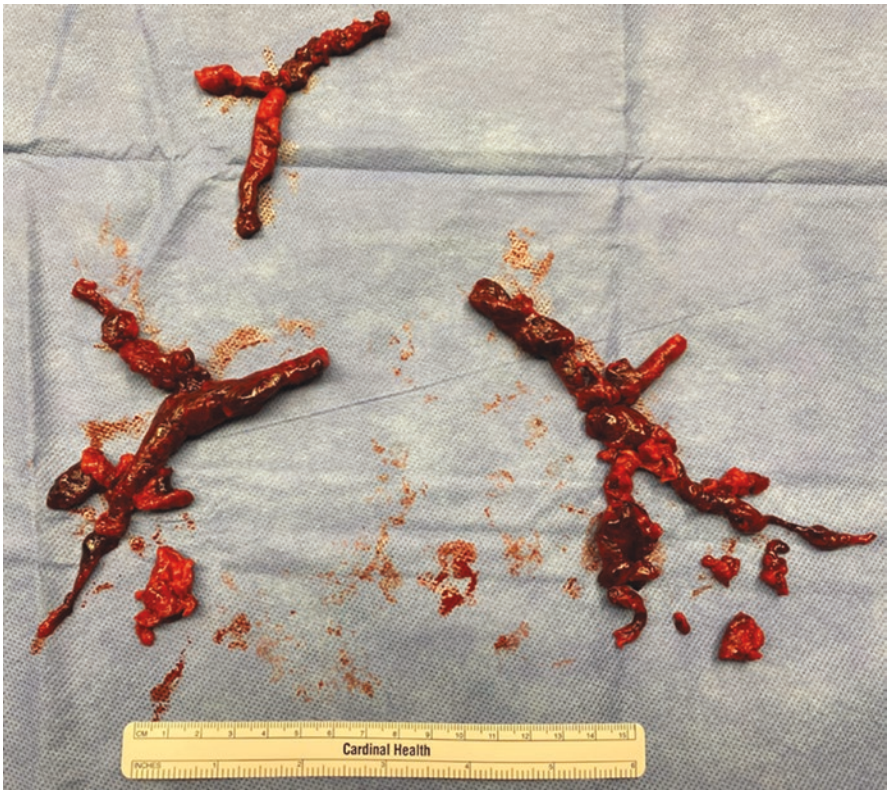


Fig. 23.2 Surgical pulmonary embolectomy specimen

pulmonary veins to retrograde flush thrombotic material into the more proximal pulmonary arteries.
- When sufficient thrombus has been removed, the arteriotomies are closed primarily and CPB is weaned.

Chronic Thromboembolic Disease

Pathogenesis

- CTEPH is a pulmonary vascular disease that results from incomplete thrombolysis of an acute PE. Fibrin within the embolus becomes progressively more cross linked and organized over time leading to incorporation and endothelialization. This leads to pulmonary vascular obstruction, redistribution of blood flow, and accompanying small vessel vasculopathy that drives progressive pulmonary hypertension and eventually right heart failure and death.

- Risk factors for CTEPH include previous PE, younger age, unprovoked PE, splenectomy, venoarterial shunt or infected pacemaker, and antiphospholipid antibody syndrome. While many hypercoagulable disorders have been documented in patients with CTEPH (antithrombin III deficiency, protein C and/or S deficiency, Factor V Leiden, Prothrombin gene mutation), they are not more prevalent than in patients with primary pulmonary hypertension [5].

Presentation and Diagnosis

- The most common presenting symptoms include dyspnea on exertion, atypical exertional angina, exercise intolerance, exertional pre-syncope or syncope, and hemoptysis. Patients commonly have sequelae of right heart failure with lower extremity edema, hypoxemia, and occasionally pulmonary artery flow murmurs.
- Echocardiogram: Can confirm significant pulmonary hypertension, estimate RV size and function, and screen for other cardiac disorders.
- Ventilation-Perfusion (V/Q) scanning: Will show segmental defects in CTEPH. A normal V/Q scan essentially eliminates CTEPH from further diagnostic consideration.
- CT pulmonary angiogram: Will demonstrate pulmonary artery anatomy and can screen for other pulmonary disease processes. Finding suggestive of CTEPH includes a mosaic perfusion pattern of the lung parenchyma, peripheral infarcts, and narrowed pulmonary arteries with pulmonary artery cutoffs.
- Cardiac catheterization: A right heart catheterization is performed to measure the hemodynamics of the pulmonary circulation and to perform a pulmonary angiogram. Typical findings include delayed filling of vessels, branch occlusions, webs, pouches, and narrowed vessels (Fig. 23.3).
- CTEPH is defined as precapillary pulmonary hypertension with a mean pulmonary artery pressure $\geq$25 mmHg and a pulmonary arterial occlusion pressure $\leq$15 mmHg in the presence of organized flow-limiting thrombi or emboli in the pulmonary arteries after at least 3 months of therapeutic anticoagulation [6].

Indications for Surgery

- PTE is the only curative intervention for patients with CTEPH, and 3-year survival approaches 90% [7].
- PTE should be considered in all patients but especially those who are symptomatic, have significant pulmonary hypertension, have proximal or segmental disease, and who do not have prohibitive comorbidities.
- In most cases, if the disease is obstructing the main, lobar, or proximal segmental pulmonary arteries, PTE is feasible and likely successful. If the disease is limited to distal segmental and subsegmental branches, PTE is technically more challenging.

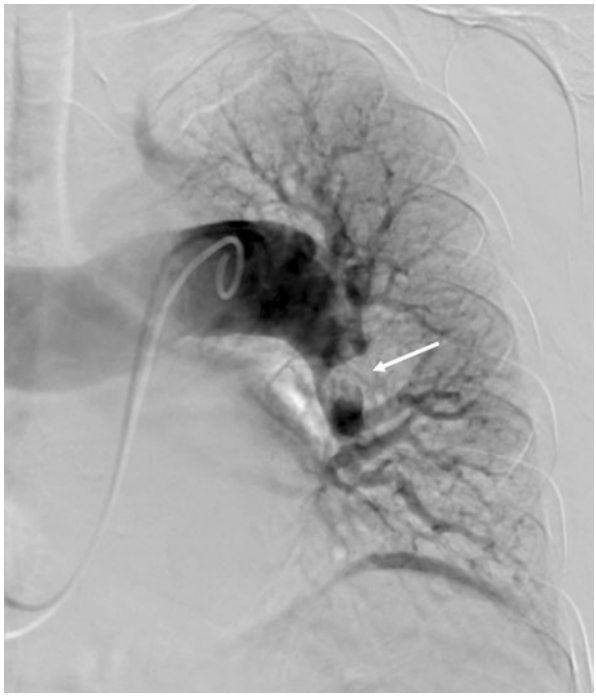


Fig. 23.3 Pulmonary angiogram in a patient with CTEPH demonstrating lower lobe predominant occlusive disease (arrow)

Operative Technique for Pulmonary Thromboendarterectomy

- A median sternotomy is performed, and bicaval and ascending aortic cannulation with left PA and aortic root venting is established. Adequate visualization is accomplished using CPB and periods of circulatory arrest. This allows for a bloodless field by diverting pulmonary arterial blood flow and reducing collateral flow.
- Once cardiopulmonary bypass (CPB) is established, and the desired core temperature (usually 18 °C to 20 °C) has been reached, the aorta is cross-clamped and cardioplegia is administered.
- A longitudinal incision is made in the right main PA between the SVC and the aorta.
- The endarterectomy is undertaken by entering the proper plane between the intima and the media (Fig. 23.4a). The plane should appear smooth and pearly white. The specimen is dissected circumferentially and followed distally as far as possible, following each subsegmental branch individually until it ends in a tail (Fig. 23.4b).
- When blood return obscures the dissection field, deep hypothermic circulatory arrest should be instituted, which improves exposure of the interior of the pulmonary artery. Periods of circulatory arrest are limited to 20 min at a time.

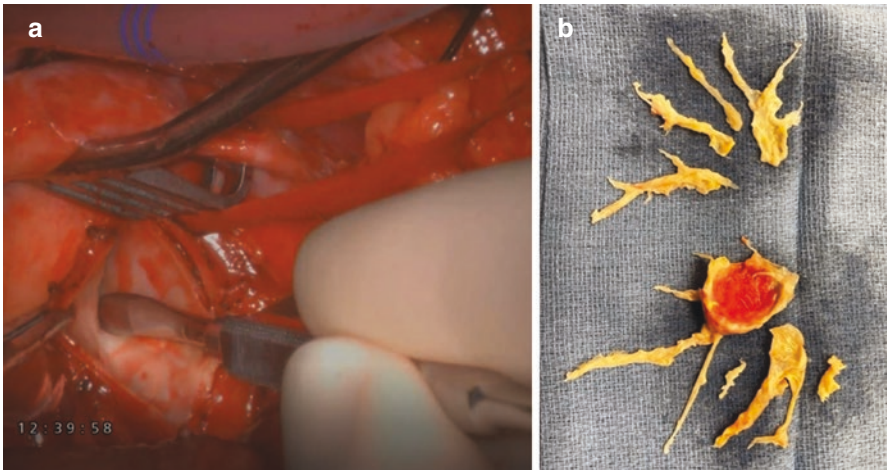


Fig. 23.4 (**a**) Initiation of the endarterectomy plane, (**b**) typical PTE specimen demonstrating abnormal intimal thickening and obstruction of numerous segmental arteries

- Once the right side is completed, hypothermic reperfusion is reinstituted and the pulmonary arteriotomy is closed primarily.
- Next, a left pulmonary arteriotomy is made. The endarterectomy plane is initiated and the specimen dissected into each segmental and subsegmental branch under an additional period of circulatory arrest.
- Once both sides are completed, circulation is reinstituted and full rewarming is begun. If other cardiac procedures are needed (i.e., PFO closure, valve repair), these are performed during the rewarming period.
- When the patient is rewarmed, CPB is weaned and the patient decannulated.

References

1. Licha CRM, McCurdy CM, Maldonado SM, Lee LS. Current Management of Acute Pulmonary Embolism. Ann Thorac Cardiovasc Surg. 2020;26:65–71.
2. LeVarge BL, Wright CD, Rodriguez-Lopez JM. Surgical Management of Acute and Chronic Pulmonary Embolism. Clin Chest Med. 2018;39:659–67.
3. Rivera-Lebron BN, Rali PM, Tapson VF. The PERT concept: a step-by-step approach to managing pulmonary embolism. Chest. 2021;159:347–55.
4. Meyer G, Vicaut E, Danays T, et al. Fibrinolysis for patients with intermediate-risk pulmonary embolism. N Engl J Med. 2014;370:1402–11.
5. Vlahakes GJ, Wright CD. Chronic thromboembolic pulmonary hypertension. In: Atlas of cardiac surgical techniques: a volume in the surgical techniques atlas series. Saunders/Elsevier; 2010.
6. Kratzert WB, Boyd EK, Saggar R, Channick R. Critical Care of Patients after Pulmonary Thromboendarterectomy. J Cardiothorac Vasc Anesth. 2019;33:3110–26.
7. Papamatheakis DG, Poch DS, Fernandes TM, Kerr KM, Kim NH, Fedullo PF. Chronic thromboembolic pulmonary hypertension: JACC focus seminar. J Am Coll Cardiol. 2020;76:2155–69.

Chapter 24
Surgery for Atrial Fibrillation

Sarah M. Nisivaco and James L. Cox

Surgery for Atrial Fibrillation

Intro

Atrial fibrillation (AF) is the most commonly seen cardiac arrhythmia, with a lifetime risk of 25% above 40 years of age. In the US, it accounts for over 500,000 hospital admissions and 160,000 deaths per year [1, 2]. Patients with AF can be asymptomatic or experience a wide range of symptoms including palpitations, lightheadedness, chest pain, shortness of breath, or fatigue.

The morbidity and mortality associated with AF can be largely attributed to 3 main sequelae: (1) symptomatic irregular heartbeat causing patient discomfort, (2) potential for compromised hemodynamics due to atrioventricular (AV) dyssynchrony, and (3) risk for thromboembolism and stroke due to stasis of blood in the left atrium [3].

While various pharmacotherapies exist for the treatment of AF, they have limited success given the lack of efficacy of most medications, as well as bothersome side effects. Given this, there has been great interest in the development of alternative approaches to treating AF including catheter-based and surgical interventions [4].

S. M. Nisivaco (✉) · J. L. Cox
Division of Cardiac Surgery, Bluhm Cardiovascular Institute, Northwestern Medicine and Northwestern University Feinberg School of Medicine, Chicago, IL, USA
e-mail: sarah.nisivaco@nm.org; James.Cox@nm.org

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_24

Atrial Fibrillation Classification

- Paroxysmal: episodes of AF lasting less than 7 days.
- Persistent: episodes of AF lasting longer than 7 days and less than 1 year.
- Long-standing persistent: episodes of AF lasting longer than 1 year.

Surgical Treatment Options

Dr. Cox and his team of researchers began their historical work in the understanding of the electrophysiology of AF in the 1980s. This research formed the groundwork of all present day surgical and catheter-based interventions aimed at treating AF.

The current surgical treatment options for AF include:

1. Cut-and-sew Cox-Maze III procedure
2. Minimally invasive CryoMaze III procedure
3. Cox-Maze IV procedure
4. Modified CryoMaze III procedure
5. Robotic CryoMaze III procedure
6. Hybrid Maze Procedure
7. Robotic CryoMaze III procedure
8. Totally Thoracoscopic (TT) Modified Maze IV procedure
9. TT Modified Maze IV/Catheter Hybrid procedure
10. Convergent Hybrid procedure [5]

Cox-MAZE

The primary surgical treatment of AF, the Cox-Maze procedure, was introduced into clinical practice in 1987 after extensive research investigation on animal models [3]. Through this work, Dr. Cox and colleagues outlined the atrial macro re-entrant circuits that occur in persistent and long-standing persistent atrial fibrillation. The Cox-Maze procedure used knowledge of these re-entrant circuits to create a "maze" of surgical incisions in the atria that interrupt the drivers perpetuating AF. In addition to interrupting these macro-reentrant circuits, this specific maze pattern of surgical incisions allows the SA node to continue to propagate signals throughout the entirety of both atria and then to the AV node, thus maintaining sinus rhythm and AV synchrony and allowing the atria to function properly [4].

The Cox-Maze surgical procedure is successful for both persistent and long-standing persistent AF. The reason for this is behind the pathophysiology of persistent versus paroxysmal AF. In paroxysmal AF, the underlying pathophysiology is focal *triggers* that induce self-limited episodes of atrial fibrillation. The treatment for this (discussed in detail below under "pulmonary vein isolation") is isolation of the triggers in order to lessen the probability of these triggers inducing AF. In

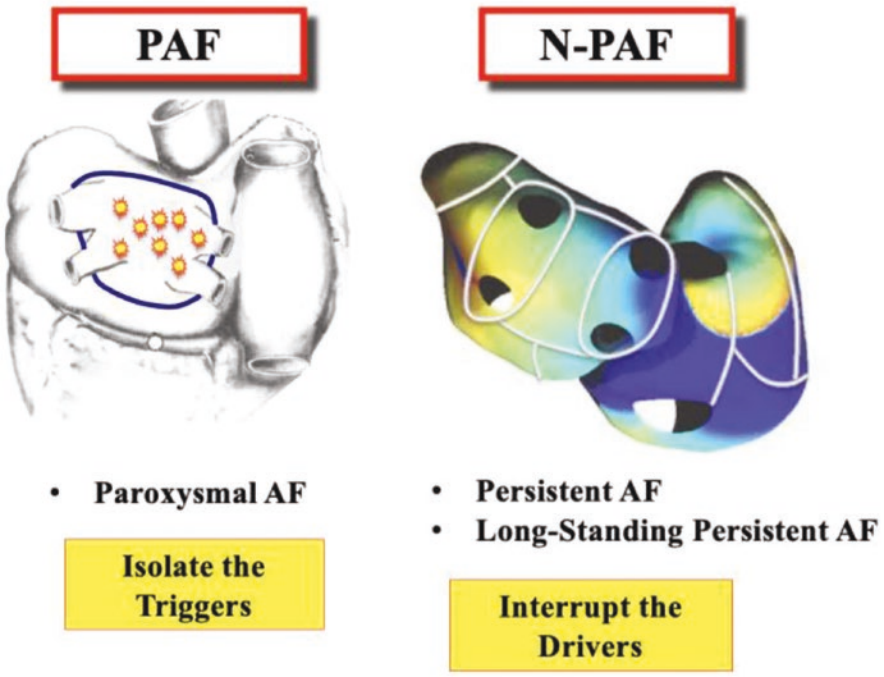


Fig. 24.1 Objectives of AF Surgery. *PAF* paroxysmal atrial fibrillation, *N-PAF* non-paroxysmal atrial fibrillation

contrast, patients with persistent AF have established episodes of AF that are sustained by the aforementioned macro-reentrant *drivers*, and are independent of triggers [5]. Therefore, the treatment for this is a surgical cure that rids the atrium of these drivers and allows the atria to be activated and responsive to sinus rhythm. See Fig. 24.1.

The original Cox-Maze procedure underwent a series of evolutions to result in the current surgical options listed above. The Cox-Maze III procedure is preserved as the gold standard for surgical treatment of AF. It originally created transmural lesions via a "cut-and-sew" method, but eventually transitioned to creation of lesions through a variety of alternative energy techniques. The most common energy techniques used are cryoablation and bipolar radiofrequency ablation. The Cox-Maze IV is the most commonly performed procedure today [6]. It results in the same functional lesion pattern as the Cox-Maze III procedure but the lesions are created with surgical ablation devices to shorten the procedure time.

The Cox-Maze procedure is performed on cardiopulmonary bypass (CPB) and can be done through a median sternotomy, a less invasive right mini-thoracotomy, or the most minimally invasively through a totally endoscopic robotic approach. Of the macro-reentrant drivers present in persistent AF episodes, 70% are located in the left atrium and 30% in the right atrium. Therefore, a bi-atrial lesion set is ideally

performed. However in certain cases—such as higher-risk patients or when trying to minimize CPB time in complex cases—a left atrial lesion set only can be performed. Bi-atrial lesion sets include those in the left atrium, right atrium, and a box lesion around the pulmonary veins (PVs) which acts to electrically "isolate" the pulmonary veins (PVI). All lesions created should be full thickness, transmural lines whether they are created from the endocardium or epicardium.

The left atrial set includes the PVI set, as well as lines connecting to the mitral valve annulus, LAA, and coronary sinus.

The right atrial lesion set includes lines to the SVC, IVC, TV annulus, and RAA.

Pulmonary Vein Isolation (PVI)

PVI is an option for surgical treatment of paroxysmal AF. The pulmonary veins have been well established as a common trigger point in a majority of AF cases. The PVs can be electrically isolated as "islands" with independent lesion sets around the right and left PVs, with or without a connecting lesion between the two islands. More commonly, the PVs are isolated as a combined "box" with a single lesion set going around all four PVs and the posterior left atrial wall. Unlike the Cox-Maze procedure, PVI does not require opening of the left atrium or any part of the heart, thus allowing for avoidance of CPB. Like the Cox-Maze procedure, it can be performed with minimally invasive techniques such as robotically or thoracoscopically. Surgical PVI, often combined with a left atrial appendage occlusion as discussed below, is often performed for patients with concomitant AF who are undergoing cardiac surgery for other reasons. In patients not already undergoing cardiac surgery, a catheter PVI can be performed which is successful in decreasing the likelihood that these triggers will induce episodes of paroxysmal AF. Finally, this surgical

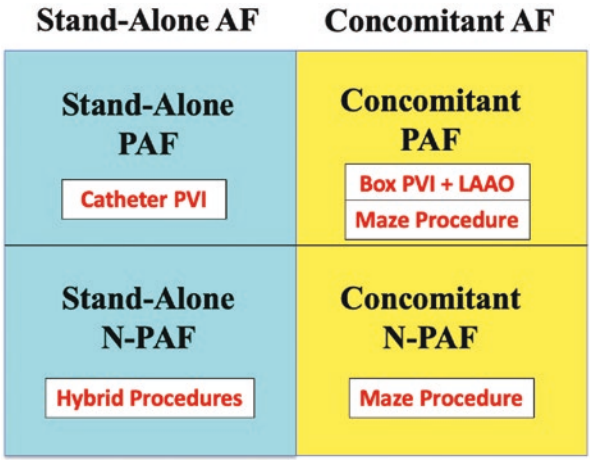


Fig. 24.2 Overview of interventional treatment of AF. *PAF* paroxysmal atrial fibrillation, *N-PAF* non-paroxysmal atrial fibrillation, *PVI* pulmonary vein isolation, *LAAO* left atrial appendage ligation

approach can be combined with various catheter ablation techniques resulting in a "hybrid" approach [7, 8]. See Fig. 24.2.

Management of the Left Atrial Appendage (LAA)

In patients with AF, the most dreaded complication is stroke as a result of thrombus formation in the heart due to stasis of blood from the abnormal rhythm. In AF, it has been well established that the LA appendage (LAA) is the main source of thromboembolism that can result in stroke. This is the reason for "occlusion" or ligation of the LAA in patients with AF undergoing cardiac surgery. This is accomplished through a variety of surgical techniques including amputation of the LAA and suture closure, stapler occlusion, double-layer suture closure from inside the left atrium, or epicardial occlusion with an FDA-approved device. In a recent randomized trial, patients with atrial fibrillation undergoing cardiac surgery were randomized to either have LAA occlusion or not. This trial of over 4000 patients showed a lower risk of ischemic stroke with occlusion compared to no LAA occlusion [9].

References

1. Centers for Disease Control and Prevention, National Center for Health Statistics. In: About multiple cause of death, 1999–2019. CDC WONDER online database website. Atlanta, GA: Centers for Disease Control and Prevention; 2019.
2. Cohn LH. In: Cohn LH, editor. Cardiac surgery in the adult. 4th ed. McGraw-Hill Medical; 2012.
3. Benjamin EJ, Muntner P, Alonso A, Bittencourt MS, Callaway CW, Carson AP, et al. Heart disease and stroke statistics—2019 update: a report from the American Heart Association. Circulation. 2019;139(10):e56–528.
4. Ferguson TB Jr, Cox JL. Surgery for atrial fibrillation. In: Zipes DP, Jalife J, editors. cardiac electrophysiology: from cell to bedside. 2nd ed. Philadelphia: Saunders; 1995. p. 1567.
5. Cox JL. The surgical treatment of atrial fibrillation. IV. Surgical technique. J Thorac Cardiovasc Surg. 1991;101:584.
6. Cox JL, Boineau JP, Schuessler RB, et al. Electrophysiologic basis, surgical development, and clinical results of the maze procedure for atrial flutter and atrial fibrillation. Adv Card Surg. 1995;6:1.
7. Bhadwar V, Rankin JS, Damiano R Jr, et al. The Society of Thoracic Surgeons 2017 clinical practice guidelines for the surgical treatment of atrial fibrillation. Ann Thorac Surg. 2017;103:329–41.
8. Brescia AA, Louis C. TSRA review of cardiothoracic surgery. 3rd ed. Independently published; 2021.
9. Whitlock RP, Belley-Cote EP, Paparella D, et al. Left atrial appendage occlusion during cardiac surgery to prevent stroke. N Engl J Med. 2021;384(22):2081–91.

Chapter 25
Pericardial Disease

Adam Paine, Akash Premkumar, and Thoralf M. Sundt

Learning Objectives
- Natural history and etiology.
- Diagnostic criteria.
- Distinguishing between constrictive pericardial disease vs restrictive cardiomyopathy.
- Indications for pericardiectomy.
- Operative technique.

Anatomy

There are two layers to the pericardium, the visceral and parietal, folded onto one another creating a closed space between which there is normally a small amount of serous fluid (Fig. 25.1) [1]. The visceral layer, more commonly referred to as the epicardium, is normally thin and translucent, although it becomes markedly thickened in the setting of pericarditis. The parietal pericardium is more substantial, normally 1 mm thick, and what is more commonly referred to as "the pericardium." The cephalad border of the parietal pericardium is its reflection onto the superior vena cava (SVC), aorta, and pulmonary artery (Fig. 25.2). The caudal border is opposed to the surface of the diaphragm. The lateral borders abut the bilateral pleural spaces with the pericardium reflected around the four pulmonary veins. The anterior border abuts the sternum and the posterior border abuts the posterior

A. Paine (✉) · A. Premkumar · T. M. Sundt
Division of Cardiac Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: apaine@partners.org; apremkumar@mgh.harvard.edu; tsundt@mgh.harvard.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_25

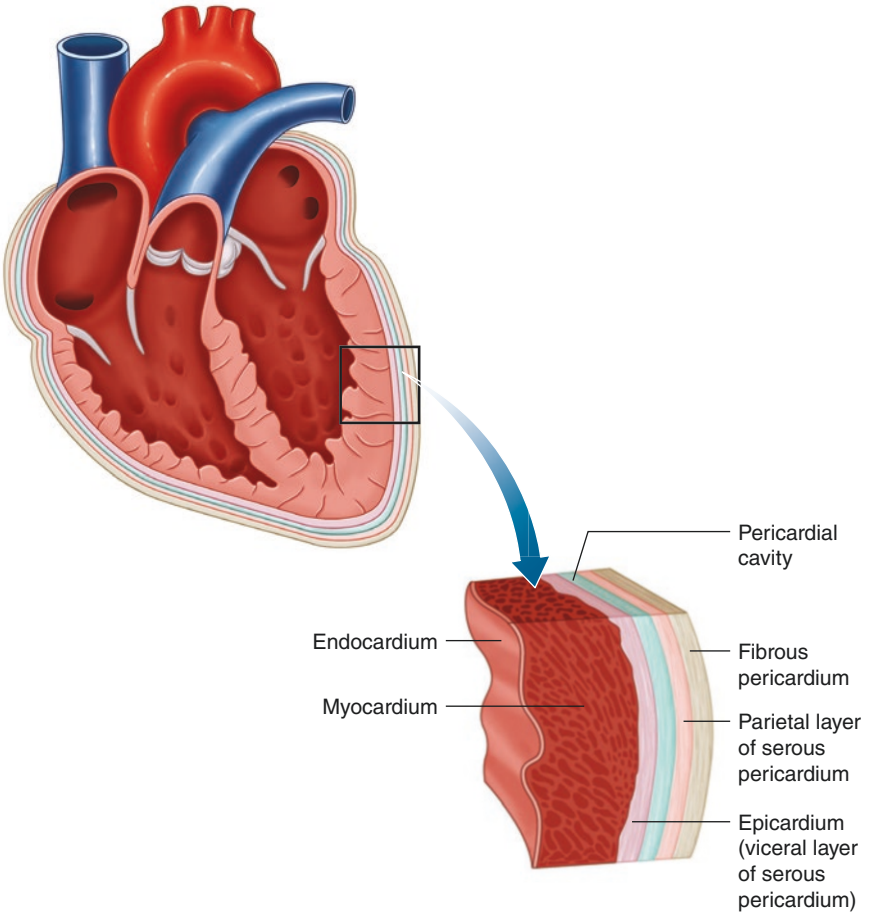


Fig. 25.1 Layers of the pericardium from the outermost fibrous pericardium with its adherent inner parietal serous pericardium that lines the pericardial cavity and is in continuity with the visceral serous pericardium, synonymous with the epicardium

mediastinal structures including the descending thoracic aorta and the esophagus. There are two potential spaces within the pericardial sac: the transverse sinus, which is posterior to the aorta and the main pulmonary artery and anterior to the parietal pericardium, and the oblique sinus, which is posterior to the left atrium, anterior to the parietal pericardium, and flanked by the four pulmonary veins. The transverse sinus is of note to the cardiac surgeon as it can be used as a tunnel for a right internal thoracic artery bypass graft to the lateral wall of the heart (see Chap. 11—Management of Coronary Artery Disease).

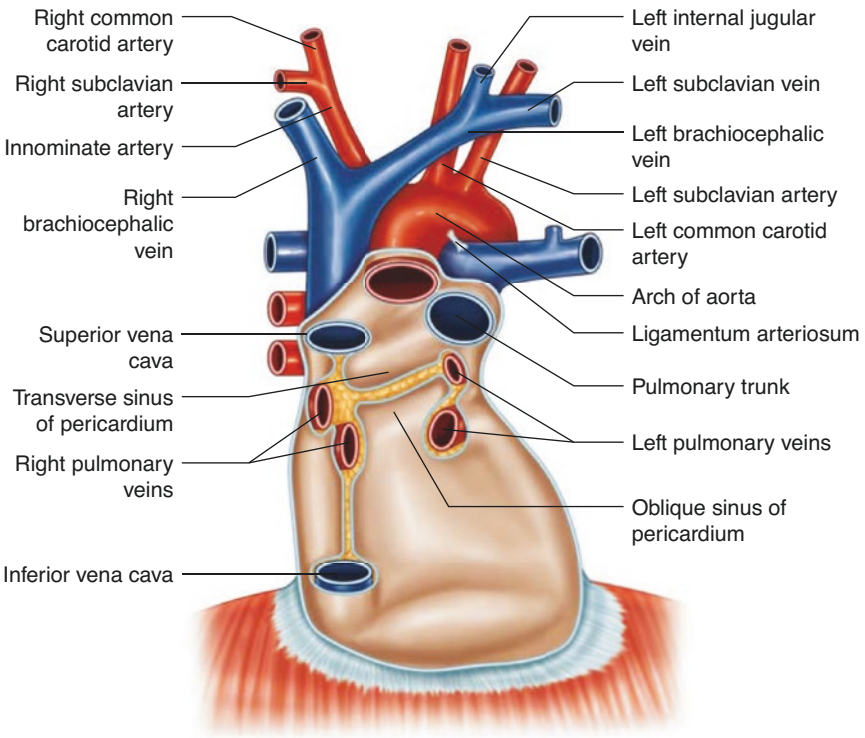


Fig. 25.2 Cephalad, caudal, lateral, and posterior borders of the pericardium including the oblique and transverse sinuses

Etiology

Acute pericarditis is the most common disease of the pericardium and is most frequently caused by a systemic viral infection. Other causes include uremia, autoimmune disorders, and less commonly bacterial infections. The inflammation of the pericardium can lead to a reactive increase in the volume of pericardial fluid termed effusive pericarditis. Occasionally, if the effusion accumulates rapidly enough, it can lead to tamponade physiology. Less than 1% of patients with pericarditis will eventually go on to develop constrictive pericarditis [2].

Constrictive pericarditis is a bit of a misnomer as it occurs consequently to pericarditis but frequently exists absent active inflammation. In the developing world, constriction is most commonly caused by tuberculous pericarditis, however in regions with a low prevalence of tuberculosis, constriction is commonly idiopathic with or without a recognized episode of acute pericarditis. It also occurs in a small percentage of cases following cardiac surgery and may become evident many years after mediastinal radiation [3]. In this latter case, radiation cardiomyopathy often

coexists making it difficult to determine if a patient's signs and symptoms are secondary to constriction, restriction, or both. Rather than an external force on the myocardium, restrictive cardiomyopathy is a condition inherent to the myocardium itself that renders it non-compliant. Other causes of restrictive cardiomyopathy are amyloidosis, sarcoidosis, and endomyocardial fibrosis [4].

Physiology

Due to the relative non-compliance of the parietal pericardium, an increase in fluid volume within the pericardial sac results in reduced diastolic filling of the cardiac chambers and subsequent reduced cardiac output (cardiac output = heart rate × stroke volume). When this results in hemodynamic compromise, it is referred to as tamponade. Cardiac tamponade may be the result of fluid collection secondary to acute inflammation, uremic pericarditis, or malignancy among non-surgical patients, or secondary to postoperative bleeding in the post-surgical patient (See Chap. 34—Principles of Perioperative Care). Pericardial constriction similarly restricts cardiac filling but in a slowly progressive manner that makes its diagnosis often difficult and delayed. Both constriction and restriction impair diastolic filling of the heart, thereby reducing cardiac output and leading to symptoms of heart failure. It is critical to make the distinction of constrictive versus restrictive etiology of symptoms as only constrictive pericardial disease will benefit from pericardiectomy.

History

Patients with acute pericarditis present with severe chest pain, classically made worse by leaning forward. There may or may not have been a preceding episode of viral illness. Patients with constrictive pericarditis most commonly present with slowly progressive symptoms of right-sided heart failure. Signs and symptoms include dyspnea on exertion, orthopnea, ascites, and peripheral edema. Some will complain of a sense of fullness in their head, especially when bending over, or may notice jugular venous distension. Given the aforementioned causes of constriction, a thorough history including inciting infections, mediastinal radiation, and cardiac surgery should be elicited as well as potential causes of restrictive cardiomyopathy such as sarcoidosis, cardiotoxic chemotherapy, etc.

Physical Exam

Acute pericarditis classically causes a loud friction rub best heard with the patient leaning forward. In cases complicated by a significant effusion (effusive pericarditis), there may be co-existing signs consistent with constrictive physiology

including evidence of right-sided heart failure such as peripheral edema, hepatomegaly, and ascites. Kussmaul's sign, a paradoxical increase in jugular venous distention with inspiration, can be seen in patients with restricted right ventricular filling including both constriction and restriction. Patients with constriction may have a pericardial knock on auscultation, which is caused by abrupt cessation of ventricular filling during diastole once the point of physical constriction has been reached.

Imaging

The diagnosis of acute pericarditis is principally clinical with supportive laboratory evidence of elevated inflammatory markers and potentially echocardiographic evidence of an effusion. The diagnosis of tamponade or constriction, however, is often made by imaging studies. Enlargement of the cardiac silhouette may be seen in the presence of an effusion, although echocardiography is the diagnostic test of choice. In the setting of constriction, pericardial calcification seen on *chest X-ray* is essentially diagnostic, although it is present in less than 25% of cases [5] (Fig. 25.3). Cross-sectional imaging with *computed tomography (CT)* and *magnetic resonance imaging (MRI)* may demonstrate pericardial calcification, but almost invariably demonstrates pathologic thickening (>4 mm) (Fig. 25.4). Cardiac MRI can be helpful in identifying tethering of the myocardium by pericardial adhesion as well as evidence of active inflammation of the pericardium, infiltrative myocardial processes causing restriction, and can demonstrate impaired diastolic filling [6, 7].

Echocardiography is a required diagnostic test in all patients presenting with heart failure symptoms as the differential is broad. While constriction is uncommon, it is important to consider as a potentially curable cause. When tamponade is

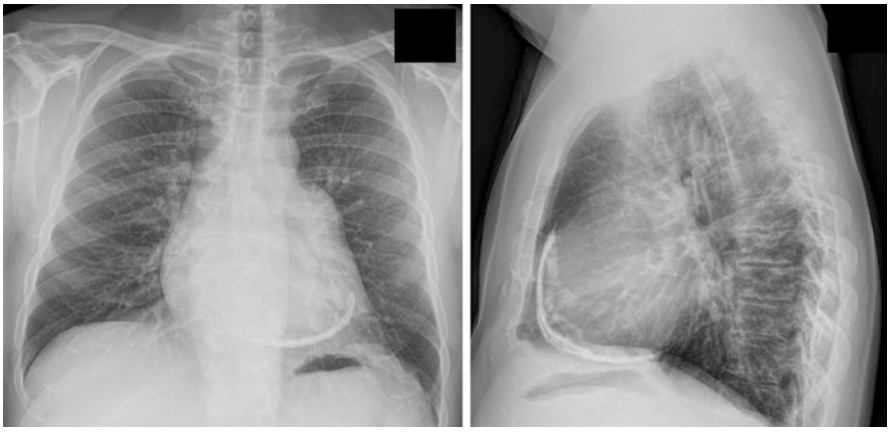


Fig. 25.3 Pericardial calcification on chest X-ray, although seen in less than 25% of cases, is highly suggestive of constrictive pericarditis in the appropriate clinical context

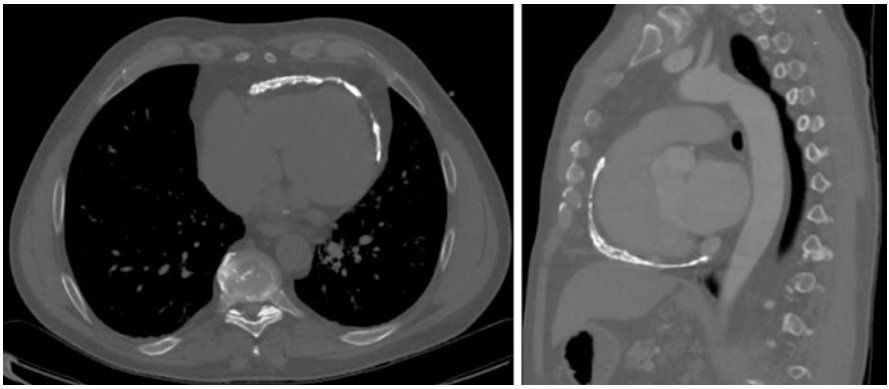


Fig. 25.4 Computed tomography demonstrating pericardial calcification and pathologic thickening (>4 mm) is highly suggestive of constrictive pericarditis in the appropriate clinical context

present, in addition to obvious fluid in the pericardial space, there will be right ventricular collapse and inferior vena cava dilatation without respiratory variation. Evidence of right heart failure, including inferior vena cava distention with decreased respiratory variation, can also be seen on the echocardiogram of patients with either constriction or restrictive cardiomyopathy. Echocardiography may identify pericardial thickening and will pathognomonically demonstrate increased *ventricular interdependence* in constriction. Given a fixed volume that can be accommodated within the non-compliant pericardium, as one cardiac chamber volume increases, another chamber must have a corresponding decrease in volume. Ventricular interdependence is demonstrated on echocardiography as interventricular *septal bounce* where the septum bows toward the left ventricle during inspiration as the pulmonary venous return declines with pooling of blood in the lungs permitting rapid filling of the right ventricle at the expense of left. Conversely, during expiration, the left fills at the expense of the right, resulting in reversal of hepatic venous flow [8] (Fig. 25.5).

Cardiac catheterization is invasive but definitive. It is not always necessary to diagnose constriction; however, in cases where imaging and history do not adequately differentiate constriction and restriction, it is indicated. Ventricular pressure tracings in constriction demonstrate the *square root sign* which corresponds to a ventricle that fills rapidly in early diastole until meeting the fixed resistance of the non-complaint pericardium (Fig. 25.6). Additional specific findings supportive of constriction include equalization of left ventricular end diastolic pressure (LVEDP) and right ventricular end diastolic pressure (RVEDP), both of which are abnormally elevated [9]. Most characteristic of constriction as compared with restriction is systolic discordance, with a decline in systemic pressure and increase in pulmonary artery pressure during inspiration and the reverse in expiration in the setting of constriction due to the changes in filling noted above.

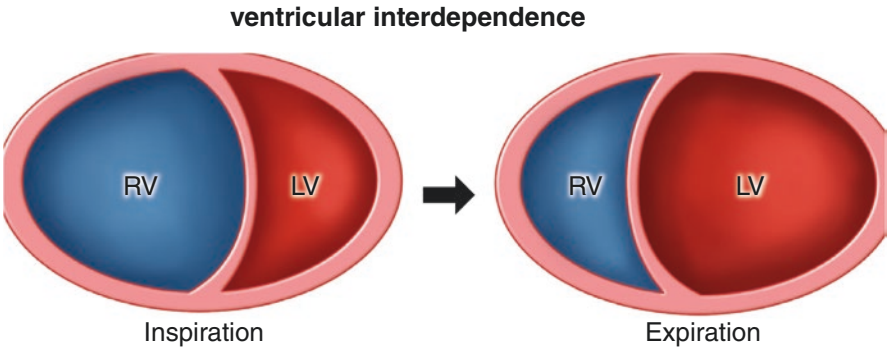
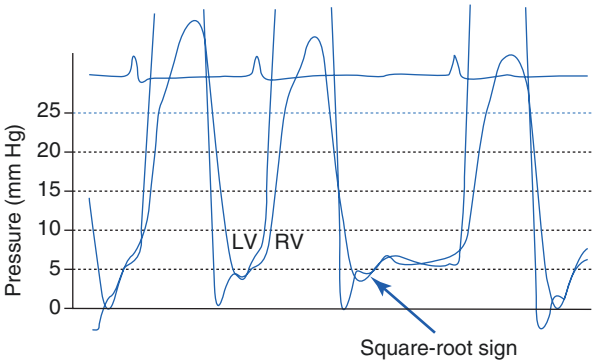


Fig. 25.5 Echocardiography demonstrating ventricular interdependence is seen in constrictive pericarditis. Ventricular interdependence is demonstrated on echo by the interventricular septum bowing toward the left ventricle during inspiration and toward the right ventricle on expiration

Fig. 25.6 Ventricular pressure tracing demonstrating the square root sign consistent with a diagnosis of constrictive pericarditis. This tracing results from a ventricle that fills rapidly in early diastole until meeting the fixed resistance of the non-complaint pericardium



Indication for Surgery

Surgical intervention is not indicated in acute pericarditis unless there is a significant effusion with evidence of tamponade. In this case either a subxiphoid or lateral thoracoscopic window may be therapeutic, although increasingly percutaneous drainage is employed if possible. In the setting of constriction, there is no effective medical management. Optimizing volume status with diuretics may improve symptoms; however, medications will not reverse the fixed constriction of the pericardium. In properly selected patients, pericardiectomy improves functional status with an acceptable risk profile with most series reporting an operative mortality between 2.5 and 9% [10, 11]. Factors that are associated with poor outcomes following pericardiectomy include prior radiation (as these patients commonly have a component of radiation-induced restrictive cardiomyopathy which will not benefit

from pericardiectomy) renal insufficiency, reduced ventricular function, high pulmonary artery pressures, and NYHA class IV symptoms.

Operative Technique

The standard approach to pericardiectomy is via a median sternotomy. The tenet to a successful outcome is near complete removal of the pericardium leaving only a small portion of pericardium posterior to the right phrenic nerve, a narrow strip along the left phrenic nerve, and the inaccessible portion of posterior pericardium within the oblique sinus. As removal of certain parts of the pericardium requires manipulation of the heart that can lead to significant hemodynamic changes, the use of cardiopulmonary bypass (CPB) may be necessary. With an experienced surgeon and cardiac anesthesiologist, a complete pericardiectomy can often be performed without the use of CPB, however it is typically necessary in a reoperative setting and one should not compromise on the adequacy of pericardiectomy just for the sake of avoiding CPB. It is critical to preserve the bilateral phrenic nerves which innervate the diaphragm as injury can result in respiratory failure. Accordingly, the patient should not be paralyzed during pericardiectomy. The phrenic nerves can be tested with a nerve stimulator both for identification and to determine their integrity post-pericardial resection. Finally, it is critical to remove both the parietal and visceral pericardium as both can contribute to impaired diastolic filling. There is often a very appealing plane of dissection between the layers, however this will fail to treat the disease. The visceral pericardium requires meticulous dissection off the myocardium, as it can be quite adherent. Adequate visceral pericardial dissection is demonstrated by clearly visible epicardial coronary arteries at the conclusion of the pericardiectomy [12] (Fig. 25.7).

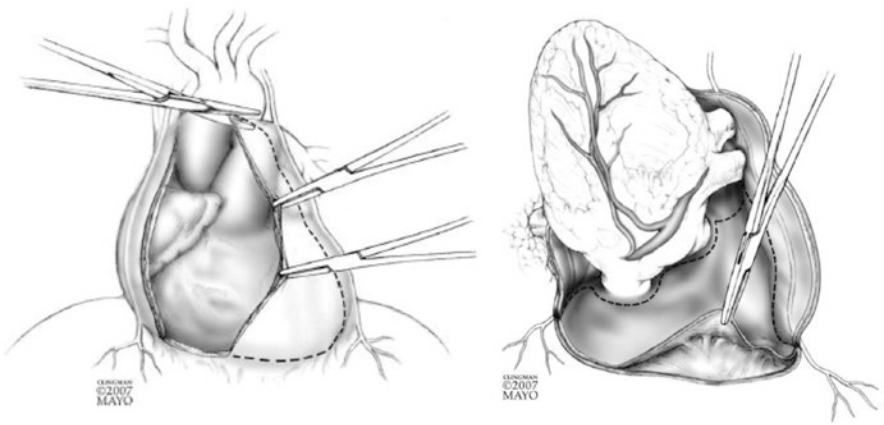


Fig. 25.7 Pericardiectomy requires near complete removal of the visceral and parietal pericardium leaving only a small portion posterior to the right phrenic nerve, a narrow strip along the left phrenic nerve, and the inaccessible portion of posterior pericardium within the oblique sinus (Copyrighted and used with permission of Mayo Foundation for Medical Education and Research)

References

1. Rodriguez ER, Tan CD. Structure and anatomy of the human pericardium. Prog Cardiovasc Dis. 2017;59(4):327–40.
2. Adler Y, Charron P, Imazio M, Badano L, Barón-Esquivias G, Bogaert J, et al. 2015 ESC guidelines for the diagnosis and management of pericardial diseases: the task force for the diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC)endorsed by: the European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J. 2015;36(42):2921–64.
3. Myers RBH, Spodick DH. Constrictive pericarditis: clinical and pathophysiologic characteristics. Am Heart J. 1999;138:219–32.
4. Muchtar E, Blauwet LA, Gertz MA. Restrictive cardiomyopathy: genetics, pathogenesis, clinical manifestations, diagnosis, and therapy. Circ Res. 2017;121(7):819–37.
5. Cosyns B, Plein S, Nihoyanopoulos P, Smiseth O, Achenbach S, Andrade MJ, et al. European Association of Cardiovascular Imaging (EACVI) position paper: multimodality imaging in pericardial disease On behalf of the European Association of Cardiovascular Imaging (EACVI) and European Society of Cardiology Working Group (ESC WG) on Myocardial and Pericardial diseases.
6. Masui T, Finck S, Higgins CB. Constrictive pericarditis and restrictive cardiomyopathy: evaluation with MR imaging. Radiology. 1992;182(2):369–73.
7. Gupta A, Singh Gulati G, Seth S, Sharma S. Cardiac MRI in restrictive cardiomyopathy. Clin Radiol. 2012;67(2):95–105.
8. Yang R, Smith J, Mokadam NA. Pericardial disease. In: Baumgartner WA, Jacobs JP, Darling GE, editors. STS cardiothoracic surgery E-book. Chicago: Society of Thoracic Surgeons; 2020.
9. Feins EN, Walker JD. In: Cohn LH, Adams DH, editors. Cardiac surgery in the adult, pericardial disease. 5th ed. McGraw Hill Education; 2017. p. 1225–42.
10. Vistarini N, Chen C, Mazine A, Bouchard D, Hebert Y, Carrier M, et al. Pericardiectomy for constrictive pericarditis: 20 years of experience at the Montreal heart institute. Ann Thorac Surg. 2015;100:107–13.
11. Gillaspie EA, Stulak JM, Daly RC, Greason KL, Joyce LD, Oh J, et al. A 20-year experience with isolated pericardiectomy: analysis of indications and outcomes. J Thorac Cardiovasc Surg. 2016;152:448–58.
12. Villavicencio MA, Dearani JA, Sundt TM. Pericardiectomy for constrictive or recurrent inflammatory pericarditis. Oper Tech Thorac Cardiovasc Surg. 2008;13(1):2–13.

Chapter 26
Cardiac Neoplasms

Fernando Ramirez Del Val and Michael J. Reardon

Incidence

Neoplasms of the heart can be primary (benign or malignant) or secondary (metastatic). Secondary neoplasms are many times more common than primary tumors. Primary cardiac tumors have an incidence of 1% and a prevalence of 0.001–0.03 in autopsy series [1, 2]. As many as 20% of patients with terminal metastatic disease have cardiac involvement. In these patients, surgery is mainly palliative and often limited to drainage of pericardial effusions [3]. Common sites of metastasis to the heart are shown in Table 26.1. Primary heart tumors can require cardiac surgery of varying complexity to achieve complete resections. In adults, the most common cardiac neoplasms are atrial myxoma and papillary fibroelastoma [4, 5]. Up to 25% of all primary cardiac neoplasms are malignant and 75% of these are sarcomas [6, 7]. Table 26.2 shows a list of primary cardiac tumors as well as the structures from which they most often arise.

F. R. Del Val (✉)
Harvard Medical School, Massachusetts General Hospital, Boston, MA, USA
e-mail: framirezdelval@mgh.harvard.edu

M. J. Reardon
Cardiothoracic Surgery, Houston Methodist Hospital, Houston, TX, USA
e-mail: mreardon@houstonmethodist.org

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_26

Table 26.1 Common sites of primary tumors in metastatic heart disease

Breast carcinoma
Esophageal carcinoma
Gastric carcinoma
Germ cell tumors (malignant)
Hepatocellular carcinoma
Leukemia/lymphoma
Lung carcinoma
Melanoma
Mesothelioma
Renal cell carcinoma
Sarcoma

Table 26.2 Types of primary cardiac tumors

Benign		
	Valves	
		Papillary fibroelastoma
	Atria	
		Myxoma
		Lipomatous hypertrophy
		Paraganglioma
		Schwannoma
	Ventricle	
		Rhabdomyoma
		Fibroma
		Hemangioma
		Granular cell tumor
		Epithelioid hemangioendothelioma
		Hamartoma of mature cardiac myocytes
		Inflammatory myofibroblastic tumor
	Pericardium	
		Lipoma
		Germ cell tumor
Malignant		
	Atria	
		Undifferentiated pleomorphic sarcoma
		Angiosarcoma
		Osteosarcoma
		Myxofibrosarcoma
	Ventricles	
		Rhabdomyosarcoma
		Liposarcoma
	Pericardium	
		Mesothelioma
		Lymphoma
		Synovial sarcoma
		Solitary fibrous tumor
	Vasculature	
		Leiomyosarcoma

Workup

An important differential diagnosis of a cardiac mass is tumor versus thrombus. Cardiac MRI looking for perfusion of the mass can help differentiate tumor from thrombus [8]. Thrombus is more likely to be associated with indwelling catheters, pacemakers, mechanical devices, and hypercoagulable syndromes. The predominant symptoms in patients with cardiac neoplasms result from embolic events, mechanical disruption of normal blood flow, alterations in the normal conduction, or systemic constitutional symptoms. Symptoms, therefore, depend on tumor location, size mobility, and friability.

Both atrial and ventricular tumors can impede normal blood flow from the atrium to the ventricle or the ventricular outflow tract. Atrial tumors may mimic valvular stenosis and are more likely to produce intermittent obstruction depending on the tumor's position. Arrhythmias are seen due to direct tumor infiltration or myocardial irritation. Hemopericardium is more often observed in malignant neoplasms. Sudden cardiac death secondary to obstruction of blood flow or ventricular arrhythmias may occur, thus most neoplasms (benign and malignant) benefit from resection when possible.

On physical examination, signs and symptoms of left (chest pain, orthopnea, dyspnea) or right heart failure (peripheral edema, ascites, hepatomegaly) are often observed. Left heart failure is a more common presentation both because left heart tumors are more common and because large right heart tumors are generally malignant and tend to grow exophytically rather than into the right atrium. A characteristic neoplasm plop (early diastolic low pitched sound after S2) has been described in association with cardiac tumors. It is thought to be caused as the neoplasm strikes the myocardium or as the mass prolapses from the atrium to the ventricle [9].

Transthoracic echocardiography is often the initial imaging study in the assessment of cardiac tumors as it is readily available, inexpensive, and provides good spatial and temporal resolution [10]. Transesophageal echocardiography provides a better definition of cardiac structures and is less limited by poor acoustic windows, thus playing an integral part during preoperative and intraoperative planning. The role of cardiac computer tomography (CCT) and cardiac magnetic resonance (CMR) imaging in the assessment of cardiac tumors has grown over the last two decades [11].

Both CCT and CMR provide a multiplanar reconstruction of the heart and adjacent structures but CCT provides the best special resolution but limited tissue characterization and no functional assessment of the heart. In contrast, CMR has the highest tissue characterization and provides a comprehensive functional assessment of the cardiovascular structures [12].

Differential Diagnosis

Myxoma

Myxomas are the most common primary cardiac neoplasm in adults [13]. These gelatinous benign neoplasms originate in the sub-endocardium and are formed by lepidic cells (polygonal/stellate myxoma cells with abundant eosinophilic

cytoplasm) [14]. They are most commonly solitary and are located in the left atrium (within the fossa ovalis) in >80% of the time, but can also be present in the right atrium or rarely in the ventricles [15]. Myxomas are more common in women (2:1) and present between the third and sixth decade of life. Most are sporadic and single, but familial syndromes such as Carney syndrome are well described and are associated with multiple or recurrent myxomas [16].

The classic presentation of systemic symptoms such as weight loss and fatigue, valvular obstruction, and embolization occurs in 30% of the patients. Smaller, irregular, more friable tumors have a higher risk of embolization [17]. The most common sequela of these embolic events are neurologic, most of which result in permanent deficits, although they can also result in visceral or lower extremity ischemia.

Myxomas can cause outflow obstruction and will mimic right- or left-sided valvular disease depending on their location. Left atrial myxomas may present with elevated left atrial and pulmonary pressures raising concerns for mitral stenosis which leads to imaging workup and diagnosis of the tumor. Myxomas that completely occlude the mitral valve result in syncopal episodes (if transient) or sudden cardiac death. Tumors located in the right atrium mimic tricuspid stenosis and can produce paradoxical embolization if a patent foramen ovale is present. Similarly, left and right ventricular outflow tract obstructions can be seen when these neoplasms arise from the ventricles.

Surgical resection with a negative margin is recommended in all myxomas due to the risk of embolization. Recurrence is rare in sporadic myxomas (5%) and is associated with positive margins. Resection of these tumors is achieved via median sternotomy with aortic and bi-caval cannulation or using minimally invasive or robotic approaches. Exposure of the tumor varies depending on the cavity it is located. Left atrial lesions are approached by incising the anterior wall of the left atrium via Sondergaard's groove. Biatrial exposure through an additional parallel right atrial incision is generally reserved for large tumors. Venous cannulation can be challenging in right atrial tumors. If the size or location of the myxoma precludes central venous cannulation, peripheral femoral and jugular venous cannulas can be placed to initiate cardiopulmonary bypass.

Papillary Fibroelastoma

Papillary fibroelastomas (PFE) account for 10% of all primary cardiac tumors [14] and are the most common valve tumor but can present anywhere in the endocardium. Histologically, they are formed by papillary folds and reassemble valvular chordae tendinea [18]. PFE are more common in patients in their seventh decade of life [19]. They most commonly occur in the aortic valve, but can also occur in right-sided vales, are generally small (<1 cm), and present as a single lesion in 80% of the patients [20]. They are most commonly asymptomatic and incidentally found during cardiac workup for other disease processes occasionally present with embolic events. Surgery is indicated in all left-sided lesions given the risk of systemic

embolization. The timing of resection is driven by the size of the tumor (>1 cm), embolic events, or mobile masses. Valve sparing resections are generally possible by employing conservative resection margins with excellent outcomes [14].

Sarcoma

Primary cardiac malignancies are exceedingly uncommon, angiosarcomas and rhabdomyosarcomas account for more than half of all primary cardiac sarcomas. These tumors are most common in women during their fifth decade of life. Most sarcomas arise from the atrium and pulmonary vessels, followed by the ventricles, mitral valve, and epicardium [21] Surgical resection has been associated with improved survival for primary cardiac sarcoma [6].

Angiosarcomas, are often found in the right atrium, are more common in men, and often present between 20 and 50 years of age [22]. A majority of patients present with metastatic disease (to the lung, liver, and brain) with poor 12-month survival. Rhabdomyosarcomas are equally distributed among the right and left chambers of the heart.

The overall prognosis for cardiac sarcomas is poor because both metastatic disease and mechanical circulatory collapse from local spread are common [17]. One- and five-year survival for these tumors are 47% and 16%, respectively [23]. The most common cause of death without surgery is local disease progression. The most common cause after surgical resection is distant metastatic disease [7, 24].

Surgical Resection of Primary Malignancies

Treatment is individualized as complete surgical resection is not always possible. Multimodality management includes surgical resection, chemotherapy, palliative radiation for non-resectable tumors, and heart transplantation in selected cases [17].

Right-sided sarcomas tend to be bulky, metastasize early, and grow exophytically (resulting in a lower incidence of heart failure or mechanical circulatory collapse). These characteristics provide a valuable time window for neoadjuvant chemotherapy [25, 26]. This strategy aims to decrease the size of the tumor, thereby facilitating R0 resection in as many as one-third of the patients [26]. Ride-sided lesions are easily accessible via a median sternotomy and are amendable to standard resection techniques.

Left-sided sarcomas are less likely to metastasize early, are less infiltrative, and are more likely to present with heart failure or circulatory collapse compared to their right-sided counterparts. A common presentation is that of a patient with an incomplete first resection where the tumor was misdiagnosed as a myxoma, followed by early recurrence. These tumors are often managed with primary resection followed by adjuvant chemotherapy as heart failure symptoms at presentation often

preclude the use of neoadjuvant treatment [26]. Best outcomes are seen with R0 resection but this is particularly challenging in posterior left atrial wall tumors, where exposure limits adequate excision. Autotransplantation (excision of the native heart from its anatomical position to remove the tumor ex vivo) has been employed to improve exposure and facilitate complete resection [27]. The procedure is done via midline sternotomy with bi-caval and aortic cannulation. The temperature goal is set for 28 °C. The inferior and superior vena cava, great vessels, and left atria are divided. The heart is placed on a back table with iced saline to remove the tumor and reconstruct the atrium when necessary followed by re-implantation of the heart [28, 29]. Pulmonary artery sarcoma has been treated with both total excision and endarterectomy. Total excision when possible provides a better oncologic resection [7]. These tumors usually arise from the level of the pulmonary valve and extend distally along the arteries. Pulmonary root replacement is often necessary for which we use pulmonary root allograft.

Role of Cardiac Surgery in the Management of Metastatic Disease

The incidence of cardiac metastasis is 9.1%. Mesothelioma, melanoma, lung cancer, and breast cancer are the most common primary tumor sites [30, 31]. The pericardium, followed by the epicardium are the most frequently involved sites of metastasis. Endocardial metastases are most common on the right side and are associated with tumors with endovascular growth such as liver and renal cancers [31].

Only 14% of all surgically resected cardiac tumors are metastatic [32]. These are usually located on the right side. They result from direct hematogenous extension from the cava or as hematogenous spread from distant tumors. Resection is palliative for the latter. Direct cavo-atrial spread in renal cell carcinoma is rare (<10%) and well documented [33, 34]. These patients have a survival benefit from complete oncologic resection as survival is not dependent on the presence of direct hematogenous spread [35].

Lung cancer can metastasize to the heart by local invasion, lymphatic, and hematogenous spread [36]. Resection of the great vessels and the atrium in patients with pathological N0–1 and T4 lung cancer to obtain R0 resection has been described with a median survival of 14 months [37, 38].

References

1. Lam KY, Dickens P, Chan AC. Tumors of the heart. A 20-year experience with a review of 12,485 consecutive autopsies. Arch Pathol Lab Med. 1993;117(10):1027–31.
2. Sütsch G, et al. Heart tumors: incidence, distribution, diagnosis. Exemplified by 20,305 echocardiographies. Schweiz Med Wochenschr. 1991;121(17):621–9.

3. Smith C. Tumors of the heart. Arch Pathol Lab Med. 1986;110(5):371–4.
4. Silverman NA. Primary cardiac tumors. Ann Surg. 1980;191(2):127–38.
5. Reynen K. Cardiac myxomas. N Engl J Med. 1995;333(24):1610–7.
6. Yin K, et al. Survival outcomes in patients with primary cardiac sarcoma in the United States. J Thorac Cardiovasc Surg. 2021;162(1):107–115.e2.
7. Chan EY, et al. Surgical Management of Primary Pulmonary Artery Sarcoma. Semin Thorac Cardiovasc Surg. 2021;35(1):53–64.
8. Wyler von Ballmoos MC, Chan EY, Reardon MJ. Imaging and surgical treatment of primary pulmonary artery sarcoma. Int J Cardiovasc Imaging. 2019;35(8):1429–33.
9. Keren A, et al. The etiology of tumor plop in a patient with huge right atrial myxoma. Chest. 1989;95(5):1147–9.
10. Auger D, et al. Cardiac masses: an integrative approach using echocardiography and other imaging modalities. Heart. 2011;97(13):1101–9.
11. Shenoy C, et al. Cardiovascular magnetic resonance imaging in suspected cardiac tumour: a multicentre outcomes study. Eur Heart J. 2021;43(1):71–80.
12. Hoey ET, et al. MRI and CT appearances of cardiac tumours in adults. Clin Radiol. 2009;64(12):1214–30.
13. Burke, A. and R. Virmani, Atlas of tumor pathology: tumors of the heart and great vessels. 1996.
14. Abu Saleh WK, et al. Cardiac papillary Fibroelastoma: single-institution experience with 14 surgical patients. Tex Heart Inst J. 2016;43(2):148–51.
15. Kuon E, et al. The challenge presented by right atrial myxoma. Herz. 2004;29(7):702–9.
16. Carney JA. Differences between nonfamilial and familial cardiac myxoma. Am J Surg Pathol. 1985;9(1):53–5.
17. Burke A, Jeudy J Jr, Virmani R. Cardiac tumours: an update: cardiac tumours. Heart. 2008;94(1):117–23.
18. Heath D. Pathology of cardiac tumors. Am J Cardiol. 1968;21(3):315–27.
19. Gowda RM, et al. Cardiac papillary fibroelastoma: a comprehensive analysis of 725 cases. Am Heart J. 2003;146(3):404–10.
20. Reynen K. Frequency of primary tumors of the heart. Am J Cardiol. 1996;77(1):107.
21. Zhang PJ, et al. Primary cardiac sarcomas: a clinicopathologic analysis of a series with follow-up information in 17 patients and emphasis on long-term survival. Hum Pathol. 2008;39(9):1385–95.
22. McAllister HA Jr, Hall RJ, Cooley DA. Tumors of the heart and pericardium. Curr Probl Cardiol. 1999;24(2):57–116.
23. Oliveira GH, et al. Characteristics and survival of malignant cardiac tumors: a 40-year analysis of >500 patients. Circulation. 2015;132(25):2395–402.
24. Chan EY, et al. Primary cardiac sarcomas: treatment strategies. J Thorac Cardiovasc Surg. 2022;166(3):828–838.e2.
25. Abu Saleh WK, et al. Improved outcomes with the evolution of a neoadjuvant chemotherapy approach to right heart sarcoma. Ann Thorac Surg. 2017;104(1):90–6.
26. Blackmon SH, Reardon MJ. Surgical treatment of primary cardiac sarcomas. Tex Heart Inst J. 2009;36(5):451–2.
27. Blackmon SH, et al. Cardiac autotransplantation for malignant or complex primary left-heart tumors. Tex Heart Inst J. 2008;35(3):296–300.
28. Conklin LD, Reardon MJ. Autotransplantation of the heart for primary cardiac malignancy: development and surgical technique. Tex Heart Inst J. 2002;29(2):105–8; discussion 108.
29. Ramlawi B, et al. Autotransplantation for the resection of complex left heart tumors. Ann Thorac Surg. 2014;98(3):863–8.
30. Paraskevaidis IA, et al. Cardiac tumors. ISRN Oncologia. 2011;2011:208929.
31. Bussani R, et al. Cardiac metastases. J Clin Pathol. 2007;60(1):27–34.
32. Murphy MC, et al. Surgical treatment of cardiac tumors: a 25-year experience. Ann Thorac Surg. 1990;49(4):612–7; discussion 617–8.

33. Zustovich F, et al. Cardiac metastasis from renal cell carcinoma without inferior vena involvement: a review of the literature based on a case report. Two different patterns of spread? Int J Clin Oncol. 2008;13(3):271–4.
34. Kearney GP, et al. Results of inferior vena cava resection for renal cell carcinoma. J Urol. 1981;125(6):769–73.
35. Sidana A, et al. Determinants of outcomes after resection of renal cell carcinoma with venous involvement. Int Urol Nephrol. 2012;44(6):1671–9.
36. Tamura A, et al. Cardiac metastasis of lung cancer. A study of metastatic pathways and clinical manifestations. Cancer. 1992;70(2):437–42.
37. Fukuse T, Wada H, Hitomi S. Extended operation for non-small cell lung cancer invading great vessels and left atrium. Eur J Cardiothorac Surg. 1997;11(4):664–9.
38. Tsuchiya R, et al. Extended resection of the left atrium, great vessels, or both for lung cancer. Ann Thorac Surg. 1994;57(4):960–5.

Chapter 27
Hypertrophic Cardiomyopathy

Boateng Kubi and Thoralf M. Sundt

Clinical Presentation

- HCM may or may not be symptomatic. When symptoms occur, they may be due to left ventricular outflow tract (LVOT) obstruction secondary to systolic anterior motion (SAM) of the mitral valve. They may also be due to inflow impairment secondary to chamber hypertrophy and decreased compliance of the left ventricle (diastolic dysfunction).
- A patient with HCM is considered to have significant LVOT obstruction when the maximal instantaneous subaortic pressure gradient is ≥30 mmHg either at rest (basal obstruction) or with physiologic provocation (labile obstruction). The conventionally accepted threshold for intervention (surgical or percutaneous) is ≥50 mmHg (Fig. 27.1).
- Dyspnea and chest pain are the most common presenting symptoms of HCM. Other symptoms include syncope, heart palpitations, paroxysmal nocturnal dyspnea, pedal edema, and sudden cardiac death.
- Due to impaired compliance of the left ventricle in HCM, physical exam may reveal a normal s1, split s2, and audible s3 in the setting of decompensated heart failure. Patients may also have a double apical impulse due to forceful left atrial contraction against the low-compliance ventricle.
- HCM is typically diagnosed in the fifth decade of life.

B. Kubi · T. M. Sundt (✉)
Department of Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: bkubi@mgb.org; tsundt@mgh.harvard.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_27

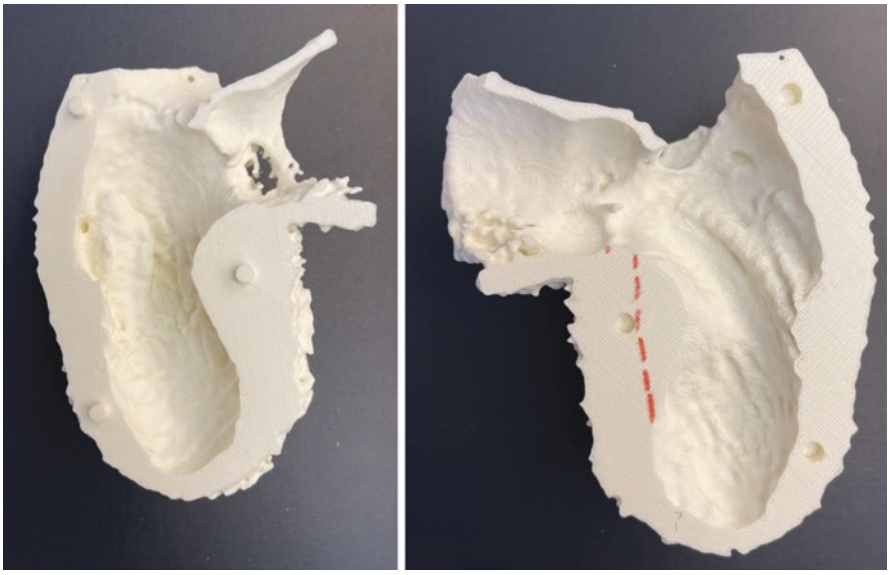


Fig. 27.1 3-D printed model demonstrating focal hypertrophy at interventricular septum. Dashed red line demonstrates the extent of myectomy that would be performed to correct LVOT obstruction

Diagnostic Evaluation of HCM

- After a comprehensive history and physical exam, an EKG should be obtained. In 75–95% of cases of HCM, a 12-lead EKG demonstrates changes consistent with left ventricular hypertrophy [1]. However, a normal EKG does not exclude HCM.
- Conventional 2-dimensional echocardiography is the most utilized imaging modality in HCM due to its widespread availability.
- Cardiac magnetic resonance (CMR) is increasingly used to confirm the diagnosis particularly when echocardiographic images are inconclusive. In addition to aiding preoperative planning, CMR can identify myocardial ischemia in the absence of epicardial coronary arterial disease—an important feature of HCM [2].
- An LV wall thickness > 15 mm on imaging (in the absence of another etiology) is generally considered diagnostic of HCM in adults, especially if associated with a family history of HCM.

Management of HCM

- Medical management with beta-blockers, verapamil, and/or disopyramide is the recommended initial therapy for obstructive HCM (Fig. 27.2) [3].

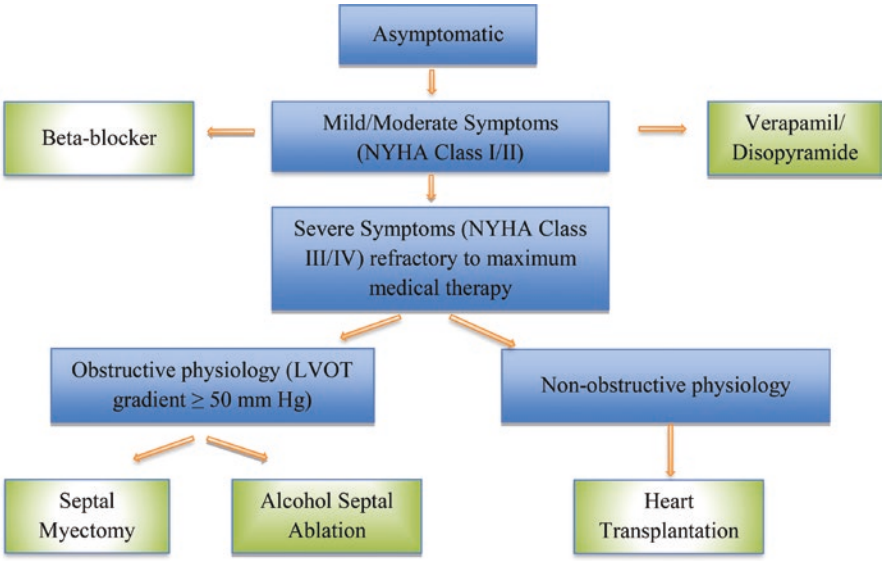


Fig. 27.2 Treatment algorithm for HCM. LVOT indicates left ventricular outflow tract; NYHA indicates New York Heart Association

- For patients who remain symptomatic despite maximal medical management, septal reduction therapy by alcohol septal ablation or septal myectomy is indicated (Fig. 27.1).
- Surgical intervention to alleviate LVOT obstruction is most performed via the transaortic approach, although transatrial and transapical myectomy techniques have been described.
- Percutaneous septal ablation is appropriate for patients who have failed medical management but may be poor surgical candidates and have favorable coronary anatomy.
- A single-chamber transvenous or subcutaneous implantable cardioverter-defibrillator (ICD) is indicated for all HCM patients who experience cardiac arrest or sustained ventricular tachycardia. For HCM patients with LV wall thickness > 30 mm, an apical aneurysm, a history of syncope, a family history of SCD, or an ejection fraction <50%, placement of an ICD is recommended as a Class II recommendation by the American Heart Association [4].

Transaortic Septal Myectomy

- Transaortic septal myectomy has been the standard surgical management for patients symptomatic from septal hypertrophy since Morrow and colleagues introduced it in the 1960s [5].

- Patients with isolated LVOT obstruction or mid-cavitary obstruction are typically excellent candidates for this technique.
- Key steps of this approach include: [6] a low oblique aortotomy, taking care to identify the origin of the right coronary artery to prevent inadvertent trauma, [7] initiation of the myectomy just right of the nadir of the right coronary cusp, avoiding the membranous septum, and carrying it leftward (for the right-handed surgeon) to the commissure between right and left cusps, [8] the proximal extent of the myectomy should remain 5 mm below the surgical annulus of the aortic valve, [1]. distally, it must extend well beyond the endocardial scar indicating the mitral valve contact point. This may be 5–7 cm into the ventricle. The thickness resected will depend upon the thickness of the ventricle.
- Anomalous papillary muscle attachments to the septum are common and may require division.
- Postoperatively, patients commonly develop left bundle branch block [9]. Complete heart block is uncommon unless a right bundle branch block (common after prior septal ablation) is present.
- Postoperative care should focus on maintenance of atrial-ventricular synchrony.
- Symptom relief can be expected in 90% of cases. Recurrence is uncommon if initial myectomy was adequate.

Transatrial Myectomy

- For HCM patients with concomitant mitral valve and sub-valvular apparatus pathology, transatrial septal myectomy may be a more appropriate approach as it offers a broader view of the ventricular septum and affords the opportunity for intervention upon pathology of both the septum and the mitral valve and its apparatus [10].
- Compared to the transaortic approach, the transatrial approach reduces risk of injury to the aortic valve cusps and associated coronary vessels.
- Characteristics of a successful repair for both approaches include achievement of septal thickness of 8-10 mm, absence of secondary ventricular septal defect, and separation between the septum and the anterior leaflet of the mitral valve throughout the cardiac cycle.

Benefits and Risks of Septal Myectomy

- In experienced centers, perioperative mortality after isolated septal myectomy is <1% [11].
- The residual inducible gradient across the LVOT should be less than 10 mmHg [12].

- Myectomy has been well demonstrated to be beneficial to patients with obstructive HCM, with a superior overall survival than medically managed patients [13].
- Satisfactory myectomy should relieve mitral regurgitation secondary to SAM among patients without primary mitral valve pathology.
- Patients who have had myectomy are less likely to have discharges from their implantable cardioverter-defibrillator (ICD), pulmonary hypertension, sudden cardiac death, and have even been demonstrated in some studies to have reverse myocardial remodelling [14, 15].
- Postoperative complications include ventricular septal perforation, conduction abnormalities, and injury to the aortic or mitral valves [9–11, 16].

Alcohol Septal Ablation

- Alcohol septal ablation is another therapeutic option for symptomatic HCM patients for whom surgery may be contraindicated, if their coronary anatomy is favorable.
- In this technique, approximately 1 to 3 cc of ethanol is injected into the septal branches supplying the hypertrophied aspects of the myocardium. Through this iatrogenic chemical necrosis, contractile dysfunction of the injected portion of the myocardium is induced and the septum atrophies over time.
- While current studies have not demonstrated any differences in overall survival between patients undergoing alcohol ablation and those undergoing myectomy, freedom from reintervention and sustained reduction of LVOT gradient is greater in the myectomy group [17].

References

1. Maron BJ. The electrocardiogram as a diagnostic tool for hypertrophic cardiomyopathy: revisited. Ann Noninvasive Electrocardiol. 2001;6(4):277–9.
2. Shirani J, Dilsizian V. Nuclear cardiac imaging in hypertrophic cardiomyopathy. J Nucl Cardiol. 2011;18(1):123–34.
3. Authors/Task Force members, Elliott PM, Anastasakis A, Borger MA, Borggrefe M, Cecchi F, et al. 2014 ESC guidelines on diagnosis and management of hypertrophic cardiomyopathy: the Task Force for the diagnosis and Management of Hypertrophic Cardiomyopathy of the European Society of Cardiology (ESC). Eur Heart J. 2014;35(39):2733–79.
4. Ommen SR, Mital S, Burke MA, Day SM, Deswal A, Elliott P, et al. 2020 AHA/ACC guideline for the diagnosis and treatment of patients with hypertrophic cardiomyopathy: A report of the American College of Cardiology/American Heart Association joint committee on clinical practice guidelines. J Am Coll Cardiol. 2020;76(25):e159–240.
5. Morrow AG, Brockenbrough EC. Surgical treatment of idiopathic hypertrophic subaortic stenosis: technic and hemodynamic results of subaortic ventriculomyotomy. Ann Surg. 1961;154:181–9.

6. Maron MS, Hellawell JL, Lucove JC, Farzaneh-Far R, Olivotto I. Occurrence of clinically diagnosed hypertrophic cardiomyopathy in the United States. Am J Cardiol. 2016;117(10):1651–4.
7. Maron BJ, Maron MS. A discussion of contemporary nomenclature, diagnosis, imaging, and management of patients with hypertrophic cardiomyopathy. Am J Cardiol. 2016;118(12):1897–907.
8. Maron BJ, Maron MS, Semsarian C. Genetics of hypertrophic cardiomyopathy after 20 years: clinical perspectives. J Am Coll Cardiol. 2012;60(8):705–15.
9. Ralph-Edwards A, Vanderlaan RD, Bajona P. Transaortic septal myectomy: techniques and pitfalls. Ann Cardiothorac Surg. 2017;6(4):410–5.
10. Wehman B, Ghoreishi M, Foster N, Wang L, D'Ambra MN, Maassel N, et al. Transmitral septal Myectomy for hypertrophic obstructive cardiomyopathy. Ann Thorac Surg. 2018;105(4):1102–8.
11. Maron BJ, DearaA OSR, Maron MS, Schaff HV, Nishimura RA, et al. Low operative mortality achieved with surgical septal Myectomy: role of dedicated hypertrophic cardiomyopathy centers in the Management of Dynamic Subaortic Obstruction. J Am Coll Cardiol. 2015;66(11):1307–8.
12. Kotkar KD, Said SM, Dearani JA, Schaff HV. Hypertrophic obstructive cardiomyopathy: the Mayo Clinic experience. Ann Cardiothorac Surg. 2017;6(4):329–36.
13. Orme NM, Sorajja P, Dearani JA, Schaff HV, Gersh BJ, Ommen SR. Comparison of surgical septal myectomy to medical therapy alone in patients with hypertrophic cardiomyopathy and syncope. Am J Cardiol. 2013;111(3):388–92.
14. Wang J, Sun X, Xiao M, Zhang M, Chen H, Zhu C, et al. Regional left ventricular reverse remodeling after Myectomy in hypertrophic cardiomyopathy. Ann Thorac Surg. 2016;102(1):124–31.
15. Smedira NG, Lytle BW, Lever HM, Rajeswaran J, Krishnaswamy G, Kaple RK, et al. Current effectiveness and risks of isolated septal myectomy for hypertrophic obstructive cardiomyopathy. Ann Thorac Surg. 2008;85(1):127–33.
16. Kwon DH, Smedira NG, Thamilarasan M, Lytle BW, Lever H, Desai MY. Characteristics and surgical outcomes of symptomatic patients with hypertrophic cardiomyopathy with abnormal papillary muscle morphology undergoing papillary muscle reorientation. J Thorac Cardiovasc Surg. 2010;140(2):317–24.
17. Nguyen A, Schaff HV, Hang D, Nishimura RA, Geske JB, Dearani JA, et al. Surgical myectomy versus alcohol septal ablation for obstructive hypertrophic cardiomyopathy: A propensity score-matched cohort. J Thorac Cardiovasc Surg. 2019;157(1):306–315.e3.

Chapter 28
Temporary Mechanical Circulatory Support

Stanley B. Wolfe and Eriberto Michel

Intra-Aortic Balloon Pump

Intra-aortic balloon pump is a catheter with a balloon on the tip that is placed in the descending thoracic aorta (Fig. 28.1). The balloon inflates during diastole to increase coronary artery perfusion and deflates during systole to decrease left ventricular afterload [1].

Indications

- Stabilize patients awaiting heart transplant (i.e., bridge to transplant).
- Bridge to durable left ventricular assist device.
- Congestive heart failure exacerbation with hemodynamic instability.
- Myocardial infarction with hemodynamic instability.
- Prophylactic placement before high-risk coronary angioplasty.
- Acute severe mitral valve regurgitation.

S. B. Wolfe (✉) · E. Michel
Division of Cardiac Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: stanleywolfe@icloud.com; EMICHEL2@mgb.org

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_28

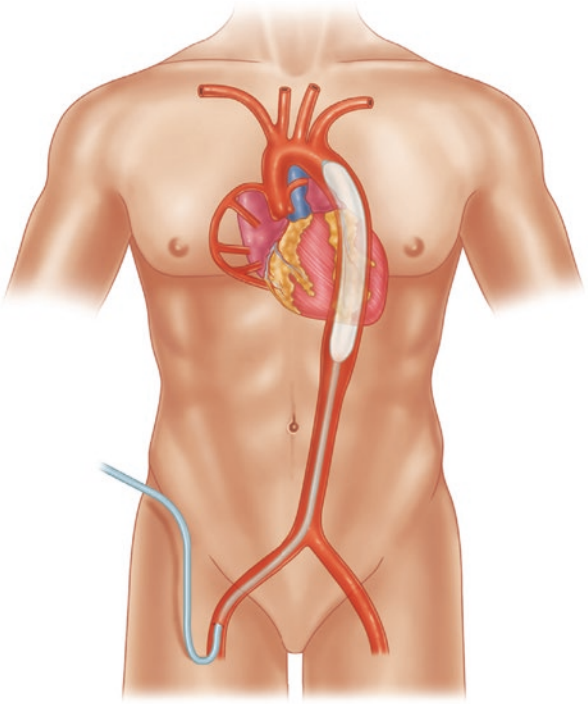


Fig. 28.1 Intra-aortic balloon pump inserted through the right femoral artery

Placement/Positioning

- The balloon catheter is inserted through the common femoral artery into the descending thoracic aorta under fluoroscopic guidance. Care must be taken to ensure the catheter is distal to the left subclavian artery but proximal to the renal arteries to prevent malperfusion.
- Alternatively, in patients who will need support for a more extended period, it can be inserted through a graft into the axillary artery.
- The catheter is connected to the console and electrocardiogram (ECG) leads.
- Heparin is not required but is often used, especially when decreasing inflation frequency.

Settings and Augmentation Assessment

- Triggering inflation: The balloon is inflated during diastole as determined by the T wave and deflated at the start of systole, which is indicated by the peak of the R wave. When the ECG quality is poor or not available, aortic pressure is used as a backup to trigger inflation and deflation of the balloon.
- Augmentation ratio: This is the ratio of supported cardiac cycles to total cardiac cycles. 1:1 augmentation indicates the balloon inflates during diastole of each

cardiac cycle. 1:2 augmentation indicates the balloon inflated during diastole of every second cardiac cycle.
- Augmentation is assessed when set to a 1:2 augmentation ratio. The arterial waveform is evaluated for the following features:
  - Diastolic augmentation wave: The tallest wave indicating inflation of the balloon. Should start just following the peak of the previous unassisted systole wave.
  - Assisted systole wave: Peak immediately following diastolic augmentation wave. It should be lower than the unassisted systole peak.

Weaning

- Decrease the augmentation ratio from 1:1 to 1:2 for 30 min and monitor the patient for ECG changes, hypotension, and reduced cardiac index.
- If the patient tolerates 1:2, a second trial further decreasing the augmentation ratio to 1:3 can be attempted.
- The intra-aortic balloon pump should never be turned off and back on due to the risk of clot formation and subsequent embolization.

Removal

- Once the patient passes a weaning trial, the balloon catheter can be removed. This should be done with the patient in the supine position. If the balloon is inserted via the axillary approach, the axillary incision must be reopened and the graft ligated.
- Confirm that the patient is not anticoagulated prior to removal.
- Turn off the system console and disconnect the catheter.
- Ensure complete deflation of the balloon by aspirating the balloon inflation port with a syringe.
- Remove the balloon catheter and sheath together (balloon will not fit through sheath after initial inflation).
- Apply direct pressure to the femoral artery just proximal to the puncture site for 30 min to ensure hemostasis.
- The patient should remain on bed rest for 6 hours (or per institutional policy).

Impella®

Impella® is a type of temporary left ventricular assist device (tLVAD). They are catheter-based continuous flow pumps placed into the left ventricle via the aorta [2]. They function similar to an Archimedes screw by pumping blood from the distal

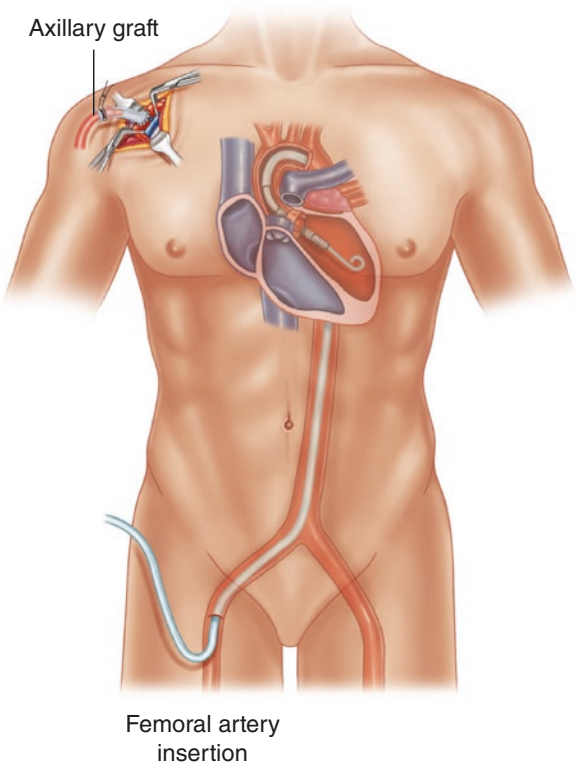


Fig. 28.2 The Impella® pump is sitting across the aortic valve with the distal end in the left ventricle and proximal end in the ascending aorta. Both axillary and femoral artery insertion approaches are demonstrated

end of the catheter, which sits in the left ventricle, to the proximal end of the pump portion of the catheter, which sits in the aorta (Fig. 28.2).

Indications

- Left ventricular failure.
- Bridge to heart transplant.
- Bridge to durable left ventricular assist device.
- Congestive heart failure exacerbation with hemodynamic instability.
- Myocardial infarction with hemodynamic instability.
- Elevated left ventricular end-diastolic pressure in patients on ECMO.
- Procedural or peri-procedure support for high-risk coronary interventions in the catheterization lab or cardiac surgery patients with depressed LV function.

Placement/Positioning

- There are four different types of Impella®, and placement approach varies by device. Fluoroscopic and echocardiographic guidance is used during the placement of all devices.
- Impella® 2.5 (2.5 L/min) and CP (3.5 L/min): Percutaneous access of the femoral artery.
  - The femoral artery is accessed using the Seldinger technique, and a sheath is placed.
  - A guidewire is advanced into the apex of the left ventricle.
  - The Impella® is advanced over the guidewire into the femoral artery, through the aortic valve, and into the middle of the left ventricle.
  - The guidewire is removed, and placement is confirmed via fluoroscopy.
- Impella® 5.0 (5.0 L/min) and 5.5 (5.5 L/min): Surgical cutdown on the axillary artery or central insertion directly into the ascending aorta.
  - The axillary artery is exposed, and a vascular graft is anastomosed to its side.
  - An introducer is inserted in and secured to the graft.
  - A guidewire is advanced through the introducer and into the left ventricle.
  - The Impella® is advanced over the guidewire into the graft, through the axillary artery, and into the middle of the left ventricle.
  - The guidewire and sheath are removed, and placement is confirmed via fluoroscopy and echocardiography.
  - The Impella® is secured to the vascular graft, the wound is closed, and the Impella® is secured to the skin.
- Patients are anticoagulated while on Impella® support, both via a device purge system and systemically.

Settings

- Impella® devices can provide varying levels of support.
- P levels indicate the level of flow provided by the device, with P-0 being 0 L/min and P-9 being the highest flow supported by that device.
- The lowest level of support to provide the desired cardiac output should be used as higher flow levels result in more hemolysis.
- Power settings below P2 may not overcome the amount of aortic insufficiency created by the catheter and lead to patient decompensation.

Weaning

- Reduce the level of support by incrementally decreasing the P level while monitoring for stability in the patient's hemodynamic profile.
- Do not decrease the level of support lower than P2 until the Impella® is ready to be removed.

Removal

- Decrease the level of support to P1 and pull the catheter out of the ventricle into the aorta.
- Reduce the level of support to P0.
- The Impella® device is carefully removed and the arteriotomy repaired.

Protek Duo/LifeSparc® System

Protek Duo® cannula and LifeSparc® pump are a system that can be used as a right ventricular assist device that pumps blood from the right atrium into the pulmonary artery (Fig. 28.3) [3].

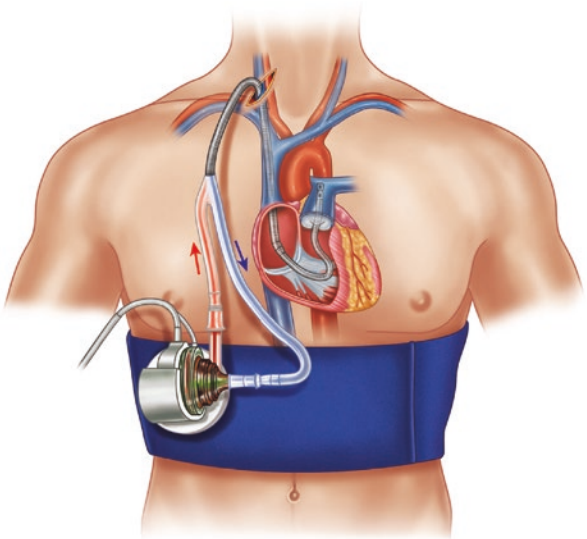


Fig. 28.3 The Protek Duo cannula is inserted through the right internal jugular vein with the tip of the catheter terminating in the pulmonary artery

Indications

- Right ventricular failure.
- Venovenous extracorporeal membrane oxygenation.

Placement/Positioning

- The pump is primed with heparinized saline.
- Protek Duo® catheter is placed under fluoroscopic guidance.
- A pulmonary artery (PA) catheter is placed via the right internal jugular vein under ultrasound guidance.
- A guidewire is advanced through the PA catheter into the PA, and the catheter is then removed.
- The patient is anticoagulated with unfractionated heparin.
- The internal jugular vein is dilated, and the cannula is inserted over the guidewire.
- The distal tip of the cannula sits in the main pulmonary artery, and the proximal port is in the right atrium.
- Once positioning is confirmed, the cannula is sutured into place.
- The cannula is connected to the pump while the assistant adds heparinized saline as needed to prevent air from being introduced into the circuit.
- The pump is turned on, and flows are adjusted as needed.
- Patients are anticoagulated while on Protek Duo® support.

Settings

- The LifeSparc® pump console allows for adjustment of pump speed to achieve an appropriate flow rate.
- Flow rates should be adjusted to avoid overflowing the lungs, which can lead to pulmonary hypertension and pulmonary edema. Echocardiographic evaluation to ensure midline position of the intra-atrial and intra-ventricular septa is an important adjunct.
- An oxygenator can be added to the pump circuit for patients with concomitant respiratory failure. In this case, the pump functions like venovenous extracorporeal membrane oxygenation while providing right ventricular support.

Weaning

- When right ventricular function recovers—as evidenced by minimal inotrope/pressor requirements, normalization of liver transaminase, and return to baseline renal function—Protek Duo® support can be weaned.

- Weaning is achieved by sequentially decreasing flow rates and observing for hemodynamic stability and steady central venous pressure (CVP).
- To prevent clot formation, the pump should not be completely stopped until the Protek Duo® cannula is ready to be removed.

Removal

- A purse-string suture is placed around the RIJ cannulation site.
- The pump is turned off.
- The Protek Duo®cannula is carefully removed.
- The purse-string suture is tied, and manual pressure is applied to the site for 15 min.

Temporary Implantable Biventricular Assist Devices

Temporary implantable biventricular assist devices (BiVAD) provide left and right ventricular support [4]. This is accomplished through two separate circuits, one for the left ventricle and one for the right ventricle (Fig. 28.4). CentriMag® and

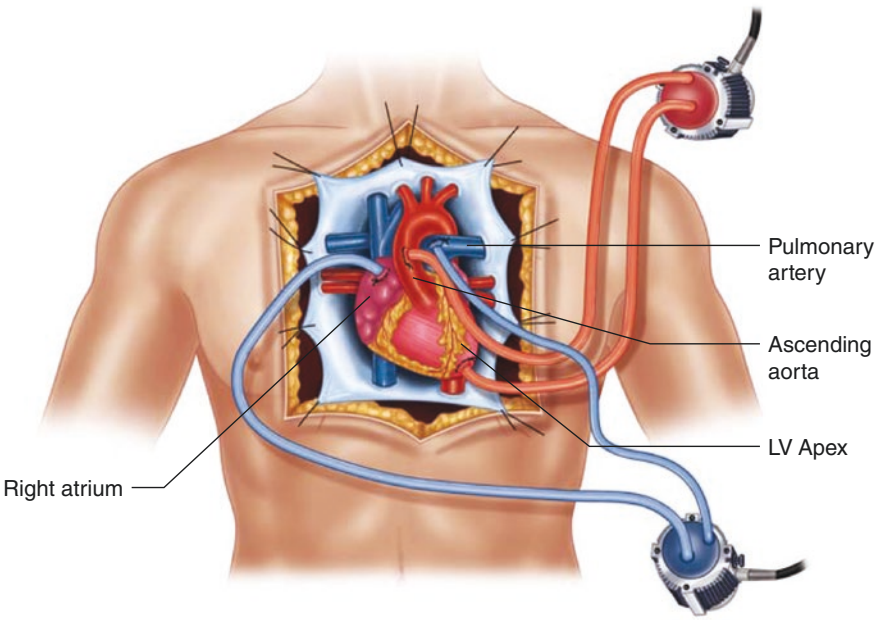


Fig. 28.4 Biventricular assist device. Right ventricular support (blue) is provided by a drainage cannula in the right atrium and a return cannula in the pulmonary artery. Left ventricular support (red) is provided by a drainage cannula in the LV apex and a return cannula in the ascending aorta

Rotaflow® are examples of extracorporeal pumps that can be used as temporary implantable biventricular assist devices.

Indications

- Biventricular heart failure.
- Bridge to transplant.
- Bridge to durable LVAD.

Placement/Positioning

- BiVADs are placed via median sternotomy with the use of cardiopulmonary bypass.
- Tunneled cannulas are directly placed in the left ventricular apex (LVAD inflow), aorta (LVAD outflow), right atrium (RVAD inflow), and main pulmonary artery (RVAD outflow).
- Importantly, the LVAD is started first, followed by the RVAD.
- The sternum can be closed in standard fashion if appropriate.

Settings

- Extracorporeal pump consoles allow pump speed adjustment to achieve an appropriate flow rate.
- The left ventricular flow should be greater than the right ventricular flow to avoid overflowing the lungs, which can lead to pulmonary hypertension and pulmonary edema.

Weaning

- Typically, BiVADs serve as bridge devices and are not weaned.
- If weaning is attempted and appropriate, the RVAD flows are weaned first to prevent overflowing the lungs.

Removal

- Removal requires reopening the sternotomy.

Other Devices

TandemHeart®

- Provides support for patients with left heart failure by unloading the left ventricle.
- The drainage catheter is placed under fluoroscopic guidance via the femoral vein and directed from the right atrium through the interatrial septum into the left atrium.
- The reperfusion catheter is placed into the femoral artery.
- Requires anticoagulation.

Impella® RP

- Provides support for patients with right ventricular failure by pumping blood from the inferior vena cava to the pulmonary artery.
- The femoral vein is accessed, and the Impella RP is advanced into the pulmonary artery under fluoroscopic guidance.
- Requires anticoagulation.

Extracorporeal Pumps

- CentriMag® and Rotaflow® are examples of extracorporeal pumps that can be used to support the left and/or right ventricles.
- Left ventricular support is provided by placing a drainage cannula in the left atrium and a reperfusion cannula in the ascending aorta or axillary artery.
- Right ventricular support is provided by placing a drainage cannula in the right atrium and a reperfusion catheter in the pulmonary artery.
- Requires anticoagulation.

References

1. Khan TM, Siddiqui AH. Intra-aortic balloon pump. In: StatPearls. StatPearls Publishing; 2022. http://www.ncbi.nlm.nih.gov/books/NBK542233/. Accessed 16 Feb 2022.
2. Ellison T, Kilic A, Choi C, Bush E. Extracorporeal membrane oxygenation and short-term mechanical circulatory support | adult and pediatric cardiac. In: STS Cardiothroacic Surgery E-Book. https://ebook.sts.org/sts/view/Cardiac-and-Congenital/1864050/all/Extracorporeal_Membrane_Oxygenation_and_Short_Term_Mechanical_Circulatory_Support?refer=true. Accessed 15 Apr 2022.

3. Kapur NK, Esposito ML, Bader Y, et al. Mechanical circulatory support devices for acute right ventricular failure. Circulation. 2017;136(3):314–26. https://doi.org/10.1161/CIRCULATIONAHA.116.025290.
4. Shehab S, Newton PJ, Allida SM, Jansz PC, Hayward CS. Biventricular mechanical support devices—clinical perspectives. Expert Rev Med Devices. 2016;13(4):353–65. https://doi.org/10.1586/17434440.2016.1154454.

Chapter 29
Extra-Corporeal Membrane Oxygenation

Philicia Moonsamy and Jerome Crowley

ECMO Configurations [1]

There are three main ECMO configurations: veno-venous (V-V), veno-arterial (V-A), and veno-pulmonary arterial (V-P). Each differ in the type of support that is provided to the patient. By convention, the letters listed before the hyphen refer to the cannula draining blood from the patient to the ECMO circuit, and letters listed after the hyphen refer to the cannula returning blood to the patient. ECMO does not correct the underlying patient pathophysiology, but rather is used to provide oxygenation and hemodynamic support as a bridge to recovery, decision, or transplantation. The ECMO circuit is comprised of four main components: a drainage cannula that removes blood from a large central vein, a pump, a membrane oxygenator to provide gas exchange, and a return cannula that delivers oxygenated blood to the patient. There are many types of pumps including centrifugal, roller, and peristaltic. There is no reservoir in the ECMO circuit, unlike the cardiopulmonary bypass circuit.

P. Moonsamy (✉)
Department of Cardiac Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: pmoonsamy@mgb.org

J. Crowley
Department of Cardiac Anesthesia, Massachusetts General Hospital, Boston, MA, USA
e-mail: jccrowley@mgh.harvard.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_29

Indications

V-V ECMO [1–3]

V-V ECMO is used to treat respiratory failure that is refractory to maximal medical therapy and mechanical ventilation [2]. It is used for oxygen delivery and carbon dioxide removal. Deoxygenated blood is drained from the venous system, pumped through an oxygenator, and returned to the right atrium. It does not provide any cardiovascular support, therefore requires intact cardiac function. However, correcting the respiratory acidosis and reversing hypoxemia may improve myocardial function, particularly on the right side of the heart. The main indications for V-V ECMO are listed in Table 29.1. The extracorporeal life support organization (ELSO) registry reports 60% survival to discharge for adult V-V ECMO patients. Outcomes differ by indication.

V-A ECMO [1, 4]

The main difference between V-V and V-A ECMO is that V-A ECMO provides cardiovascular support in addition to oxygen delivery and carbon dioxide removal. Concurrent respiratory failure is not mandatory for V-A ECMO cannulation but may necessitate the choice of V-A ECMO over other temporary mechanical circulatory support options in these patients. Indications are not based on prospective randomized controlled trials, and cannulation is determined by clinical judgment in an emergency setting in a patient that is unstable with a risk of imminent death from cardiopulmonary failure. Common indications for V-A ECMO are listed in Table 29.2 and can be grouped into those that are related to primary cardiac dysfunction vs. those that secondarily cause cardiac dysfunction.

Table 29.1 Indications for V-V ECMO

Indications for V-V ECMO
• Pneumonia or aspiration
• Acute Respiratory Distress Syndrome (ARDS)
• Bridge to lung transplant
• Primary graft dysfunction (PGD) post lung transplant
• Volume overload
• Bronchopleural fistula and severe air leak syndromes
• Status asthmaticus
• Drowning
• Transfusion related lung injury
• Thoracic Surgery

Table 29.2 Indications for V-A ECMO

Indications for V-A ECMO
• Primary cardiac dysfunction
• Acute coronary syndromes
• Fulminant myocarditis
• Post-cardiotomy shock
• Unstable arrhythmia
• Bridge to heart transplant or durable support (class IV heart failure)
• Graft failure after heart transplant
• Extracorporeal CPR
• Secondary cardiac dysfunction
• Acute massive pulmonary emboli or pulmonary hypertension
• Sepsis associated cardiomyopathy
• Drug overdose
• Amniotic fluid emboli
• Hypothermia

The ELSO registry reports an overall 46% survival to discharge for adult V-A ECMO patients [5]. As with V-V ECMO, observational studies have shown differences in survival with respect to indication. The most favorable short-term outcomes are in patients requiring ECMO for fulminant myocarditis or primary graft failure after cardiac transplant with 70% and 80% survival to hospital discharge, respectively.

V-P ECMO

V-P ECMO is used to support a failing right ventricle in addition to veno-venous ECMO support. This configuration is not commonly used but is becoming more prevalent due to the availability of percutaneous dual lumen cannula. Advantages include a low rate of recirculation (since drainage is from the RA and return is into the pulmonary artery, the tricuspid and pulmonic valves serve to minimize mixing) and the ability to support a failing right heart as well as potentially help decongest the liver and kidneys in the setting of right ventricular failure. It is important to remember that this configuration is not ideal in the setting of any degree of left ventricular dysfunction as the independent right sided support will rapidly overwhelm the failing left ventricle leading to severe pulmonary edema.

The cannula is placed via the right IJ vein and must be placed under fluoroscopic guidance. A flow directed catheter with a balloon at its tip is placed into the pulmonary artery using Seldinger technique. Placement can't be readily adjusted so all

Table 29.3 Contraindications to ECMO

Contraindications to ECMO
• To all forms of ECMO
• Unrecoverable multi-organ failure or neurologic disease
• Unwitnessed cardiac arrest or CPR[a] >30 min without return of spontaneous circulation
• Active severe bleeding
• Contraindication to anticoagulation or refusal to receive blood products
• Intracranial bleeding or neurosurgical procedure within 10 days
• Contraindications to V-V ECMO
• Cardiogenic shock
• Contraindications to V-A ECMO
• BMI>40[b]
• Aortic dissection
• Severe aortic valve regurgitation
• End-stage renal and liver disease

[a] Cardiopulmonary resuscitation
[b] Body mass index

efforts should be made to ensure satisfactory location before leaving the fluoroscopy suite. ECMO support is initiated as above with additional attention paid to the left ventricular function to avoid overloading the pulmonary circulation. In the setting of isolated right ventricular dysfunction, improvement in cardiac output should be noted with a reduced need for inotropic support.

Contraindications [3, 6]

Contraindications to all forms of ECMO are listed in Table 29.3. In general, contraindications are relative, and physicians must balance the risks of the procedure vs. the potential benefits. ECMO is resource intensive, and its futile use carries a host of ethical issues for patients. Use in patients with unrecoverable multi-organ failure or untreatable systemic disease such as metastatic cancer is contraindicated. For the same reason, patients with who had an unwitnessed cardiac arrest or prolonged CPR should not be cannulated given their uncertain neurologic status. Given the need for systemic heparinization, use in patients with active severe bleeding, patients who have a contraindication to anticoagulation and those who underwent a recent neurosurgical procedure is contraindicated.

Use of V-V ECMO in patients with severe cardiogenic shock is contraindicated since they likely require V-A ECMO. Use of V-A ECMO in patients with significantly high body mass index (>40) is contraindicated as it is difficult to achieve sufficiently high flow rates. Furthermore, patients with high body mass index suffer higher cannulation site and limb ischemia complications leading to worse outcomes. The integrity of the V-A ECMO circuit is dependent on a competent aortic valve and intact aorta, therefore use in severe aortic regurgitation and aortic dissection is contraindicated.

Cannulation Strategies

Nomenclature

ECMO cannulas are designated with respect to the pump (not the patient). "Inflow" cannulas bring blood to the pump, and "outflow" cannulas send blood away from the pump. Therefore, drainage cannulas are inflow cannulas and cannulas that return oxygenated blood to the patient are outflow.

V-V ECMO [1, 3, 7]

The circuit is set up to drain de-oxygenated blood from the vena cava and return oxygenated blood to the right atrium. The artificial lung is placed in series with the native lung. Oxygenated and decarboxylated blood from the ECMO circuit is pumped systemically by the native heart. Cannulas are usually placed percutaneously at the bedside, ideally using ultrasound guidance to identify a safe location to access the vein. There are various configurations for cannulation. We favor the femoral-internal jugular configuration which drains deoxygenated blood from the inferior vena cava (IVC) via the right femoral vein and returns oxygenated blood to the superior vena cava(SVC)/right atrial (RA) junction via the right internal jugular vein (IJ). The right femoral vein is preferred for the drainage cannula over the left since it has a straighter path to the IVC. To achieve optimal drainage, the tip of the drainage cannula is placed at the IVC/right atrial junction. The RA and hepatic veins stent open the IVC in this location which helps prevent collapse of the IVC around the drainage cannula and can cause chatter in the circuit. However, care must be taken to avoid placing the drainage cannula into the hepatic veins. This can be assessed using trans-esophageal echocardiography (TEE), trans-thoracic echocardiography, or fluoroscopy. If these imaging modalities are not available, a plan X-ray film may suffice to confirm appropriate placement.

Cannula size is determined by the size of the patient. In general, 25Fr drainage cannulas are tolerated by patients over 40 kg and are sufficient to achieve adequate flows. 19Fr return cannulas (into the IJ) are used in average sized patients with a body surface index (BSA) close to 2 m^2. If either inflow or outflow cannulas are placed too far into the RA so that they are next to each other, there is increased risk of recirculation. Recirculation is a phenomenon whereby reinjected oxygenated blood is withdrawn by the drainage cannula without passing through the systemic circulation. This decreases the efficiency and effectiveness of the circuit. 8–10 cm between cannulas is usually required to decrease recirculation.

The femoral-femoral configuration is less favorable as it has a higher risk of recirculation, allows for lower maximal flow than other cannulations strategies and has a high rate of deep vein thrombosis around the cannulas. The drainage cannula

is placed in the proximal IVC, and the return cannula is placed at the IVC/RA junction. It is preferable to place the return outflow cannula in the right femoral vein since the cannula may not reach the RA from the left femoral vein in larger patients.

Finally, a dual lumen bicaval cannula can be introduced from the right IJ, passing through the RA with the tip positioned in the IVC. Deoxygenated blood is drained from the drainage ports which are located in the SVC and IVC and oxygenated blood is returned through the reinjection port which is carefully positioned at the RA and directed toward the tricuspid valve. The cannula must therefore be placed under both TEE and fluoroscopic guidance to ensure the correct positioning of the various sideholes and to prevent catastrophic injury to the right heart. The dual lumen cannula offers various advantages including minimal recirculation, prevention of limb ischemia complications and allows patients to ambulate freely. However, the cannula is expensive and therefore may be best suited for patients who will require long-term V-V ECMO therapy and rehabilitation such as those awaiting lung transplant. Dual lumen cannulation is also more time consuming and therefore not recommended as first line for an emergent cannulation.

Common to all forms of cannulation is meticulous avoidance of air in the ECMO circuit using "wet to wet" connections and robust securing of cannula with multiple sutures to prevent inadvertent dislodgement. After cannulation, the ECMO circuit is appropriately connected ensuring no air in the lines. It is recommended that an "ECMO Initiation Checklist" be performed to ensure appropriate direction of tubing, adequate fresh gas supply, and appropriate levels of oxygen are confirmed. Support should be initiated slowly to minimize abrupt temperature shifts since rapid infusion of cold fluid can cause the heart to fibrillate. Cannulation can trigger a vasodilatory response so vasopressors should be available to support the blood pressure. Conversely, correction of the respiratory acidosis/hypoxia may lead to rapid improvement in vasomotor tone and cardiac function, so close monitoring is essential. Once ECMO support is established satisfactorily, ventilatory support should be weaned to lung protective settings to minimize further lung injury.

V-A ECMO [1, 4]

Cannulation for V-A ECMO can be achieved centrally (in the chest) or peripherally. Peripheral cannulation is much more expeditious since it does not require a sternotomy and is therefore used in emergencies. Both open and percutaneous peripheral techniques can be used to access the femoral vessels. The open cutdown technique has the advantage of higher success rate, easier method of ensuring limb perfusion, and potentially easier removal of the cannulas. Drawbacks include slower cannulation times, higher rates of bleeding, and infection. We favor ultrasound-guided percutaneous cannulation. In either strategy, the common femoral artery is accessed below the inguinal ligament but above the bifurcation of the superficial and deep femoral arteries. A cannula is placed using Seldinger technique. The venous drainage cannula is placed into the femoral vein below the inguinal ligament

with the tip located at the IVC/RA junction. The venous cannula can be placed on the same or contralateral side to the arterial cannula. Cannula size is determined by the vessel size/patient's body surface area and by the goal flow. In general, 17Fr arterial cannulas and 25Fr venous drainage cannulas are used in average sized patients. When the femoral arterial vessels cannot be accessed, the axillary artery can be used as an alternate for the arterial return cannula, however this carries higher rates of bleeding complications. The carotid artery is also commonly used in pediatrics because it can be simply ligated with decannulation. This is not possible in adults.

Central cannulation is achieved via sternotomy and has the advantages of offering antegrade arterial flow which is more physiologic and therefore avoids North-South Syndrome (discussed below). The drainage cannula is placed directly into the right atrium, and the arterial cannula is placed antegrade into the aorta. Central cannulation carries a higher risk of infection since the patient's chest must remain open during the ECMO run, therefore every effort should be made to close the skin as much as possible after cannulation. Central cannulation also allows support for higher flows and allows for easy placement of a left ventricular vent in order to prevent left ventricular dilation (discussed below).

As with V-V ECMO cannulation, the ECMO circuit is appropriately connected ensuring no air in the lines, an "ECMO Checklist" is performed and support is initiated slowly. Cannulation can trigger a vasodilatory response so vasopressors should be available to support the blood pressure. Conversely, correction of the low output state may allow for rapid weaning of vasopressors and close monitoring of blood pressure is critical.

Circuit Function and Monitoring While on ECMO [8]

The amount of ECMO support delivered to the patient is described by three variables: the flow through the circuit, the percentage of oxygen delivered by the gas in the oxygenator known as the delivered oxygen fraction (FdO_2), and the flow rate of the gas in the oxygenator known as the sweep flow. The FdO_2 is analogous to the fraction of inspired oxygen (FiO_2) on the ventilator and is one of the determinants of the oxygenation of blood. The sweep flow rate determines the rate of carbon dioxide removal from the blood and is analogous to the minute ventilation. The flow through the circuit is determined by several factors: the rotations per minute of the pump (RPMs), the resistance to drainage through the venous cannula, and the resistance to return through the return cannula. Increasing the RPMs will increase the flow; however, it is important to remember that the ECMO pump is both preload dependent and afterload sensitive so the flow may vary at the same RPMs due to different loading conditions.

Oxygenation of the blood via the ECMO circuit is related to two factors: the FdO_2 and the total flow of the ECMO circuit. Approximately 70% of blood must participate in efficient gas exchange to maintain an arterial saturation of 90–94%.

Therefore if 1.0 FdO_2 is delivered, then the ECMO circuit flow must be ~70% of the cardiac output to achieve this goal. This is particularly relevant for V-V ECMO when deciding the goal flow rates.

Monitoring the ECMO patient includes firstly a full physical exam assessing the cannulation sites, neurologic status of the patient (stroke is a common complication especially in anticoagulated patients) and assessing distal extremities especially in peripherally cannulated patients. The native hemodynamic function of the patient is also important to continually assess and is indicated by mean arterial pressure, vasopressors, and inotropes. The ventilator settings and the FdO2, sweep gas and flow (RPMs) delivered by the ECMO circuit should be noted with the intent to continuously wean the patient from the circuit. Frequent laboratory monitoring is also necessary including complete blood count (CBC) to monitor infection (pneumonia is a common complication), hemoglobin, platelets, and coagulation factors to prevent bleeding complications. The kidneys do not tolerate long ECMO runs due to relative ischemia, and the patient's creatinine must be judiciously monitored. CVVH should be initiated early if the patient's renal function starts to decline. In addition to laboratory monitoring, daily chest X-rays and frequent echo are important to assess cannula location, pulmonary edema, and left ventricular size.

Weaning ECMO [1]

The patient must have sufficient recovery of native lung function in order to liberate from the V-V ECMO circuit. This can be detected by improvements in imaging, lung compliance, and resolution of the underlying disease process. As the patient's native lung function improves, the required sweep gas flow and FdO_2 will decrease. Once these are minimal, then the patient can be placed on standard ventilatory settings, and the sweep gas turned to 0LPM. This effectively means the patient is off ECMO. Flow is maintained in the ECMO circuit in order to prevent thrombosis; however, no gas exchange is occurring. The patient's tidal volumes should be >4–5 cc/kg, indicating good lung compliance. The FiO_2 delivered by the ventilator should also be weaned to whatever the clinician is willing to tolerate to come off the ECMO circuit and just use the ventilator for oxygenation. If the patient tolerates this "capping trial" for a prescribed period of time (6-24 hrs depending on the fragility of their respiratory status), then they can be decannulated. Decannulation from V-V ECMO can usually be performed at the bedside with cannula removed and hemostasis obtained with sutures at the skin site and manual pressure. Patients who do not show signs of lung recovery should be considered for referral to a lung transplant center that has experience with prolonged ECMO weans and the possibility of lung transplant if the patient fails to wean.

Weaning from V-A ECMO is more complicated as the sweep flow can never be reduced to zero because this would create a large shunt with deoxygenated blood returning to the arterial system. First and foremost, the patient's underlying problem should have been addressed and treated before weaning and the patient's other end organ function should be recovering. The pulsatility of the native heart

should be assessed with TTE, examination of the arterial line waveform and should be >20 mmHg on low dose inotropes. Once this has been achieved, a "ramp trial" can be attempted. During this trial, the ECMO flows are slowly decremented to 2LPM over a period of 12–24 h and an echocardiogram is obtained. If this study is promising, then the patient can be further anticoagulated, and the flows further reduced until the circuit is clamped and the patient is observed off of ECMO. Caution must be taken in the setting of right ventricular failure as often the right ventricle only needs partial unloading and failure to wean may not be apparent immediately. If the wean is successful, the patient can be decannulated from V-A ECMO which is commonly done via surgical cut down and direct repair of the femoral artery.

Complications and Troubleshooting [4, 9]

North-South Syndrome

Also known as Harlequin Syndrome, North-South Syndrome (or differential hypoxia) is a complication specific to peripheral V-A ECMO. The phenomenon is caused by the retrograde direction of the arterial flow from the cannula up the aorta toward the heart. The oxygenated "ECMO blood" meets the "native blood" that is ejected by the heart after it has started to recover and regain pulsatility. If the patient's native lung function is severely compromised, then this blood has the potential to be significantly hypoxic. This is problematic as the coronaries and the cerebral circulation are more likely to see native blood and consequently will suffer hypoxic injury despite adequate performance of the ECMO circuit. Close monitoring for this phenomenon is critical and includes arterial blood gas sampling from the right upper extremity as this reflects the blood entering the cerebral circulation and will give warning of differential hypoxia.

Troubleshooting depends on the severity of the lung injury. As a first maneuver, ventilatory support can be optimized to improve native blood oxygenation. However, this is not ideal if it results in injurious settings that can potentiate further lung injury and reduce the likelihood of recovery. Negative inotropes and diuretics can also be used to depress cardiac function and decrease ejection. If the patient's cardiac function has recovered significantly, one could consider also converting to V-V ECMO. Another option is to create a hybrid circuit, known as veno-arteriovenous ECMO (V-AV). Here, an additional return cannula is placed in one of the jugular or subclavian veins and y-connected to the arterial cannula so that some oxygenated blood is returned to the venous system, "pre-oxygenating" the blood before it passes into the native lungs and therefore before it is ejected by the heart. In effect, the patient will then be on both V-V and V-A ECMO. This configuration can be challenging to maintain as it requires higher total drainage to support the flows needed as well as a partial occluding clamp to adjust the relative venous vs arterial flow. As a last resort, the patient can be converted to central cannulation (to achieve antegrade arterial flow) or biventricular support can be initiated with separate temporary

left and right ventricular assist devices and an oxygenator in order to preoxygenate all the blood going through the right heart.

Left Ventricular Distension [1]

An additional complication specific to peripheral V-A ECMO is left ventricular (LV) distension which results from an impaired left ventricle facing the elevated afterload resulting from retrograde arterial ECMO flow up the aorta. Distension of the left ventricle will lead to further left ventricular stress in the setting of increased wall tension as well as the potential for significant pulmonary edema which will reduce the likelihood of liberation from ECMO. Frequent echocardiography should be used to assess for aortic insufficiency, LV function, mitral regurgitation, and LV size. Chest X-rays can also be used to look for pulmonary edema progression, and consideration of a pulmonary artery catheter to monitor LV filling pressures.

If left untreated, this can lead to irreversible LV injury, pulmonary edema, and stasis in the LV leading to clot formation. An initial maneuver can be to increase inotropic support to encourage LV contractility. In addition, ECMO flow can be increased further to decrease blood returning to the LV. Additional invasive methods of decompressing the left ventricle can be used such as placement of an intra-aortic balloon pump, placement of a percutaneous left ventricular assist device (Impella), atrial septostomy, or placement of a surgical vent via the left ventricular apex or the left superior pulmonary vein.

Thrombosis

In all forms of ECMO, it is optimal to maintain a reasonable degree of anticoagulation to reduce the risk of thrombosis in the circuit and thrombotic complications in the patient. The oxygenator and circuit tubing should be monitored closely to check for clot or fibrin formation, and the circuit should be switched out if large clots form. Targeted levels of anticoagulation vary by institution and usually involve targeting a aPTT range, a Xa range, or an activated clotting time. If the risk of bleeding is too high for a particular patient, just a bolus dose can be used for cannulation (which carries the highest initial risk of thrombosis due to stasis in the cannula), or anticoagulation can be avoided all together. Cannulation and running ECMO without anticoagulation have been reported, so in the appropriate patient, this is reasonable acknowledging the likely increased risk of thrombotic complications. Other strategies to prevent thrombosis include preferentially flowing the circuit at higher rates, having two providers perform cannulation simultaneously to avoid prolonged periods where a cannula is left clamped with no flow, and flushing of cannulas with saline to avoid any blood sitting in them while ECMO is being prepared.

Limb Ischemia

Limb ischemia complications with peripheral V-A ECMO are extremely common, and frequent physical examination of the distal extremities is very important. Doppler assessment of distal arterial flow can be challenging given the non-pulsatile nature of the ECMO circuit. Given that the ECMO cannulas are large and can completely occlude the vessel at the insertion site, patients usually experience ischemia complications in the limb that was cannulated. We therefore advocate for the routine placement of a distal perfusion cannula into the superficial femoral artery (SFA) on the same side that the femoral arterial cannula is placed. A 5–7Fr cannula is placed antegrade into the SFA under ultrasound guidance. It is easier to place the distal perfusor before the larger arterial cannula is placed because the SFA can go into vasospasm after cannulation which makes it difficult to identify.

References

1. Squiers JJ, Lima B, DiMaio JM. Contemporary extracorporeal membrane oxygenation therapy in adults: fundamental principles and systematic review of the evidence. J Thorac Cardiovasc Surg. 2016;152(1):20–32.
2. Zapol W. Extracorporeal membrane oxygenation in severe acute respiratory failure. JAMA. 1979;242(20):2193.
3. Akoumianaki E, Jonkman A, Sklar MC, et al. A rational approach on the use of extracorporeal membrane oxygenation in severe hypoxemia: advanced technology is not a panacea. Ann Intensive Care. 2021;11:107.
4. Rao P, Khalpey Z, Smith R, et al. Venoarterial extracorporeal membrane oxygenation for cardiogenic shock and cardiac arrest. Circ Heart Fail. 2018;11(9):e004905.
5. https://www.elso.org/registry/internationalsummaryandreports/internationalsummary.aspx. Accessed 23 Jun 2023.
6. Axtell AL, Funamoto M, Legassey AG, et al. Predictors of neurologic recovery in patients who undergo extracorporeal membrane oxygenation for refractory cardiac arrest. J Cardiothorac Vasc Anesth. 2020;34(2):356–62.
7. Osho AA, Moonsamy P, Hibbert KA, et al. Veno-venous extracorporeal membrane oxygenation for respiratory failure in COVID-19 patients: early experience from a major Academic Medical Center in North America. Ann Surg. 2020;272(2):e75–8.
8. Mazzeffi MA, Rao VK, Dodd-O J, et al. Intraoperative Management of Adult Patients on extracorporeal membrane oxygenation: an expert consensus statement from the Society of Cardiovascular Anesthesiologists-Part I, technical aspects of extracorporeal membrane oxygenation. Anesth Analg. 2021;133(6):1459–77.
9. Sidebotham D. Troubleshooting adult ECMO. J Extra Corpor Technol. 2011;43(1):P27–32.

Chapter 30
Durable Mechanical Circulatory Support

Lynze Franko and David D'Alessandro

Left Ventricular Support

Indications [1–7]

- Initially, left ventricular assist devices (LVADs) were utilized as a temporary treatment for patients listed for transplant until a heart transplant became available. This was deemed bridge to transplant (BTT).
- Destination therapy (DT) is a term utilized for the treatment of patients with end-stage heart failure refractory to optimal medical management who are not candidates for heart transplant. Reasons for these patients not to be a heart transplant candidate often include age, active substance use such as smoking, insulin-dependent diabetes with end organ damage, recent cancer history, chronic renal failure, or other significant comorbidity. There is current FDA approval for both BTT and DT indications (now referred to as short term and long term, respectively).
- Alternative designations for LVAD implementation include bridge to decision and bridge to candidacy for patients who still have tests or treatments required before they can officially be evaluated or listed for heart transplantation. There is also bridge to recovery, though this indication is less common. Bridge to recovery patients are often identified after improvement in cardiac function following

L. Franko (✉)
Department of Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: lfranko@mgb.org

D. D'Alessandro
Division of Cardiac Surgery, Department of Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: dadalessandro@mgh.harvard.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_30

LVAD implantation. Careful evaluation can identify a small subset of patients in whom the LVAD can be safely removed.

- Approximately 50% of LVAD utilization is for DT, while 26% is for BTT and 23% is for bridge to candidacy. However, DT or long-term LVAD therapy continues to grow as the primary designation for LVAD treatment.
- Indications for LVAD implantation include:
  - New York Heart Association (NYHA) Class IV congestive heart failure symptoms refractory to maximal medical therapy and conventional surgical interventions.

    NYHA Stage IV = unable to carry on any physical activity without symptoms of heart failure or having symptoms at rest.
    These patients continue to have this level of symptoms despite already being on maximal medical therapy and having undergone needed surgical interventions, such as coronary artery bypass grafting or valve repair/replacement.
  - Ejection fraction <25%.
  - Reduced functional capacity with a maximal oxygen consumption (VO_2) <14 mg/kg/min or inability to complete the test.
  - Dependency on IV inotropes or temporary mechanical circulatory support.
  - Estimated mortality over 50% at 1 year with medical management.
- Contraindications to LVAD implantation include limited life expectancy due to malignancy or concomitant end organ failure, pulmonary HTN, significant right heart failure, or lack of social support.
- The interagency registry for mechanically assisted circulatory support (INTERMACS) collects data from government and private organizations regarding the utilization and outcomes of FDA-approved mechanical circulatory support devices. There are also INTERMACS profiles or levels that further classify patients with advanced heart failure (NYHA III–IV) to better assess their need for mechanical circulatory support and risks related to intervention. INTERMACS class 1 is identified as critical cardiogenic shock, while class 7 is identified as advanced NYHA III symptoms.
  - Classically, most patients selected for LVADs are INTERMACS class 2–3. Highest risks of complications and mortality of LVAD implantation are seen in INTERMACS class 1. Growing evidence shows improved outcomes and reduction in complications for patients with ambulatory heart failure (class 4–7) prior to significant decompensation with LVAD implantation. However, there are concerns that this relatively healthy population can experience devastating complications with LVAD implantation.
- Selection for LVAD therapy is complex as the patient will need to be adherent to medical therapy, manage the machine/batteries around the clock, and have substantial support systems in place throughout treatment. Further, these patients can face significant adverse events and re-hospitalizations. For this reason, selection of patients for LVAD therapy is reviewed by a multi-disciplinary team, who can help optimize patient selection in order to maximize patient benefit.

Devices [3, 5, 8–10]

- Devices can either have pulsatile flow or continuous flow. Initial pulsatile devices were pneumatic and were associated with high rates of mechanical failure. Given this, devices were instead focused on continuous flow. In 2016, greater than 95% of implanted devices were continuous flow. One of the newer devices, HeartMate3 (HM3; Abbott Labs, Chicago, IL), was created with an artificial pulse created with rhythmic pump speed modulation. This is thought to mimic natural blood flow to theoretically reduce bleeding and thromboembolic events. The HM3 is currently the only FDA-approved, durable LVAD available in the US.
- Another delineation in devices is between axial flow and centrifugal flow. Axial flow is created by a propeller within a pipe with blood flow parallel to the axis of rotation. Centrifugal flow is created by a disk spinning within a cavity with blood flow perpendicular to the axis of disc rotation. Newer centrifugal pumps also use magnetic levitation of the disc to reduce friction and hemolysis.
- There are several different LVAD devices. Three commonly utilized LVAD devices include HeartMate II (HMII; Abbott Labs), HeartWare HVAD (Medtronic, Minneapolis, MN), and the HM3.
  - HeartMate II: FDA approved for BTT 2008 and DT 2010. Axial flow device with the motor placed within a preperitoneal pocket. The HMII is no longer being produced and has been replaced by the HM3.
  - HVAD: FDA approved for BTT 2012 and DT 2017. Centrifugal flow device with magnetic levitation. Motor placed within the pericardium. Recently, evidence of increased risk of significant neurological adverse events was identified in HVAD when compared to HM3. Medtronic has stopped distribution and sale of the HVAD.
  - HeartMate3: FDA approved for BTT 2017 and DT 2018. Centrifugal flow device with magnetic levitation. Motor placed within the pericardium. This is currently the most common LVAD device implanted in the US. The MOMENTUM 3 trial by Mehra et al. (2019) demonstrated survival similar between HMII and HM3; however, HM 3 had higher rates of survival at 2 years free of reoperation and disabling stroke.

REMATCH Clinical Trial [1]

- Randomized Evaluation of Mechanical Assistance for the Treatment of Congestive Heart Failure (REMATCH) is a randomized multicenter trial evaluating outcomes comparing patients with end-stage heart disease who were not cardiac transplant candidates treated with maximum medical therapy versus long-term LVAD.
- The study included 129 patients with NYHA stage IV heart failure (68 LVAD vs 61 medical management). The LVAD utilized in this study was the pneumatic HeartMate VE (Thoratec, Pleasanton, CA). Survival rates were 52% in the LVAD

group and 25% in the medical management group at 1 year. Additionally, at 1 year the LVAD group reported improved quality of life compared to the medical management group. However, the LVAD group did have 2.35 times more serious adverse events, including infection, bleeding, and device malfunction. This was one of the leading studies which indicated survival benefit for DT over medical management.

Surgical Implantation Technique [11–13]

- Classically, inflow of the LVAD is within the apex of the left ventricle (LV). The most common outflow location for LVADs is within the ascending aorta, though placement within the descending aorta has been utilized with minimally invasive techniques. The placement of the pump itself is either within the pericardial pocket (HVAD and HM3) or within a preperitoneal pocket (HMII). The drive line is tunneled within the abdominal wall, which attaches to a power supply.
- The classic approach for LVAD implantation is through a median sternotomy, though some less invasive approaches have been developed. These alternative minimally invasive approaches are particularly beneficial in patients with prior coronary bypass grafts as it can help avoid accidental injury to bypass grafts during redo sternotomies. Further, these approaches can help reduce the formation of adhesions, which can complicate cardiac transplant in the future. Even with the classic approach, it is recommended to avoid dissection in areas critical to cardiac transplantation, like between the aorta and pulmonary artery, in order to avoid adhesion formation. Cardiopulmonary bypass (CPB) is most often required with LVAD implantation, though some new techniques do not require this support.
  - Minimally invasive techniques are increasingly utilized. One approach is the left thoracotomy combined with either mini-sternotomy versus a right mini-thoracotomy. The inflow cannula is implanted via the left thoracotomy. Then, the outflow cannula is most often implanted via a right mini-thoracotomy or mini-sternotomy. Minimally invasive techniques avoid the need for a complete sternotomy, which has greater morbidity and longer recovery.
- Below is a description of the general surgical technique for a HM3 implantation:
  - Cannulate the patient for CPB. Bi-caval cannulation suggested if placing a temporary RVAD or need for mitral valve repair/replacement.
  - Placing the inflow cannula in the LV can be performed by two methods: (1) Sew then cut (described below) or (2) Cut then sew.

    The LV apex is elevated to allow for appropriate inflow placement. The inflow cannula should point toward the mitral valve. If the inflow cannula is directed toward the septum (placed too laterally), it can create obstruction of blood flow, which can lead to long-term issues. Transesophageal echo-

cardiogram can be utilized to assist in identifying the best position of the inflow tract. It is important to avoid the left anterior descending artery during placement.

The sewing ring is then secured—either with interrupted U-stitches or a running stitch. Some techniques utilize a continuous felt strip or placing stitches deeply as the myocardium has poor strength and a tendency to allow sutures to pull through the muscle.

Myocardium within the sewing ring is then removed utilizing a coring device. Myocardium or papillary muscle obstructing the inflow tract is then excised.

Next, the inflow cannula is placed within the sewing ring and secured. The LVAD devices are then secured to the inflow cannula.

– The outflow cannula is sutured to the ascending aorta on the greater curvature just above the sinotubular junction.

Measuring the outflow cannula length is important. It is recommended to measure the outflow cannula length with a full heart. The Dacron is expected to length slightly with time. It is important to avoid kinking of the outflow cannula.

A partial aortic clamp in a side-biting fashion can be utilized at this stage. De-airing is critical before the anastomosis is complete.

Clamping the outflow cannula after the anastomosis is completed prevents retrograde flow into the LV while on CPB.

– The drive line is then tunneled in the midclavicular line within the subcutaneous tissue, though some techniques recommend tunneling deep to the rectus abdominis muscle. The location where the drive line exits the skin is secured with a suture in a purse-string fashion. Of note, the velour should be completely within the driveline tunnel with a silicone only interface at the skin. Depending on technique, the drive line can be tunneled before or after LVAD implantation.
– After CPB is lowered, the LVAD flow is then slowly increased to avoid sudden increasing of flow and demand on the right ventricle (RV).

Post-operative Complications [4, 5, 8, 10, 11]

- Overall survival for end-stage heart failure after LVAD implantation, with or without RVAD implantation, is approximately 80% at 1 year and 70% at 2 years; however, heart transplant still has better long-term outcomes than LVAD.
- Pump thrombosis is a difficult problem instigated by turbulent flow and inadequate anticoagulation. The exact frequency differs depending on the study and device, though is somewhere around 5–10%. Pump thrombosis has been found to be less frequent with the HM3. This complication can lead to clinical decompensation and require pump exchange.

- Bleeding is the most common complication of LVADs long term, particularly gastrointestinal bleeds. This is a particular problem in BTT patients as it exposes the patient to more transfusions and the potential for developing additional antibodies.
- Right heart failure develops in approximately 25% of LVAD patients. Optimizing LVAD flow is critical to prevent or avoid worsening right heart failure. If the LVAD flow is too high, it can decompress the LV. The decompressed LV then shifts the interventricular septum, which can worsen right ventricular function due to changes in the right ventricle geometry and uncoupling interdependence. This is critically important as the septum can provide a majority of the right ventricle's function. Further, RV dysfunction can be unmasked by LVAD placement as the RV is placed under additional demand for increased flow with the increased cardiac output supplied by the LVAD.
  - Right ventricle function can be supported by interventions like RV pacing or medications (milrinone, dobutamine, inhaled nitric/epoprostenol). Temporary RVADs and ensuring adequate right-sided coronary perfusion can also assist with RV function.
- Significant neurological adverse events, including transient ischemic attacks and stroke, are of particular concern due to its impact on the patient's quality of life. Depending on patient population and device, this complication can occur in about 10–30% of patients.
- Additional complications include driveline infections and aortic insufficiency (AI). Of note, AI can worsen with time with LVAD therapy. This is a significant concern because AI allows for recirculation of blood from the inflow cannula back to the LV directly. This decreases systemic perfusion while increasing pump flow. Possible interventions include aortic valve replacement at the time of LVAD implantation in patients with AI; however, bioprosthetic valves can degenerate over time in this setting. The aortic valve can be over sewn to prevent recirculation.

Right Ventricular Support [3, 11, 14, 15]

- Long term, or durable, right ventricular assist device utilization is complex and currently uncommon. RVADs are most often utilized in a temporary setting with durable LVAD implantation. They are most often extracorporeal or percutaneous. These temporary RVAD devices can be placed at the time of LVAD implantation in patients with known right heart failure. Alternatively, temporary RVADs can be placed after LVAD implantation in those who develop right heart failure after LVAD implantation. In these cases, the goal is to wean the patient off of these temporary RVADs, relying on the LVAD for long-term support.
- Some centers will use 2 VADs in one patient—one for right heart support and one for left heart support. These instances are referred to as biventricular assist

devices (BiVADs). Unfortunately, this configuration can be difficult for the patient as it requires two drive lines and battery packs. Further, it is difficult to titrate pump flow for the RV, which is more compliant than the LV and susceptible to suction events.

  - RVADs have an inflow cannula placed in the RV or right atria. The outflow cannula is in the pulmonary artery.

- Because LVAD devices have superior outcomes, clinically BiVADs are most often employed in critical cases for patients without other options. Often, these patients are then confined to the hospital given needed for significant monitoring and interventions. There are also a higher rate of complications associated with BiVADs. There are additional BiVADs and durable RVADs under development, though none are currently routinely utilized in the US.

References

1. Rose EA, Gelijns AC, Moskowitz AJ, Heitjan DF, Stevenson LW, Dembitsky W, et al. Long-term use of a left ventricular assist device for end-stage heart failure. N Engl J Med. 2001;345(20):1435–43.
2. Cohn LH, Adams DH. Cardiac surgery in the adult. 5th ed. New York: McGraw-Hill Education; 2017.
3. Molina EJ, Shah P, Kiernan MS, Cornwell WK 3rd, Copeland H, Takeda K, et al. The Society of Thoracic Surgeons Intermacs 2020 annual report. Ann Thorac Surg. 2021;111(3):778–92.
4. Han JJ, Acker MA, Atluri P. Left ventricular assist devices. Circulation. 2018;138(24):2841–51.
5. Kirklin JK, Pagani FD, Kormos RL, Stevenson LW, Blume ED, Myers SL, et al. Eighth annual INTERMACS report: special focus on framing the impact of adverse events. J Heart Lung Transplant. 2017;36(10):1080–6.
6. Heidenreich Paul A, Bozkurt B, Aguilar D, Allen Larry A, Byun Joni J, Colvin Monica M, et al. 2022 AHA/ACC/HFSA guideline for the management of heart failure: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. J Am Coll Cardiol. 2022;79(17):e263–421.
7. Drakos SG, Kfoury AG, Stehlik J, Selzman CH, Reid BB, Terrovitis JV, et al. Bridge to recovery: understanding the disconnect between clinical and biological outcomes. Circulation. 2012;126(2):230–41.
8. Cho SM, Mehaffey JH, Meyers SL, Cantor RS, Starling RC, Kirklin JK, et al. Cerebrovascular events in patients with centrifugal-flow left ventricular assist devices: propensity score-matched analysis from the Intermacs Registry. Circulation. 2021;144(10):763–72.
9. Hosseinipour M, Gupta R, Bonnell M, Elahinia M. Rotary mechanical circulatory support systems. J Rehabil Assist Technol Eng. 2017;4:2055668317725994.
10. Mehra MR, Uriel N, Naka Y, Cleveland JC, Yuzefpolskaya M, Salerno CT, et al. A fully magnetically levitated left ventricular assist device—final report. N Engl J Med. 2019;380(17):1618–27.
11. Whitson BA. Surgical implant techniques of left ventricular assist devices: an overview of acute and durable devices. J Thorac Dis. 2015;7(12):2097–101.
12. Beyersdorf F, Scheumann J, Siepe M. Implantation of the HeartMate 3—description of the surgical technique. Oper Tech Thorac Cardiovasc Surg. 2017;22(3):173–85.
13. Maltais S, Anwer LA, Tchantchaleishvili V, Haglund NA, Dunlay SM, Aaronson KD, et al. Left lateral thoracotomy for centrifugal continuous-flow left ventricular assist device

placement: an analysis from the mechanical circulatory support research network. ASAIO J. 2018;64(6):715–20.
14. Shimada S, Nawata K, Kinoshita O, Ono M. Mechanical circulatory support for the right ventricle in combination with a left ventricular assist device. Expert Rev Med Devices. 2019;16(8):663–73.
15. Shehab S, Hayward CS. Choosing between left ventricular assist devices and biventricular assist devices. Card Fail Rev. 2019;5(1):19–23.

Chapter 31
Heart Transplantation

Antonia Kreso, Akash Premkumar, and David D'Alessandro

Introduction

- Since the first reported human heart transplantation in 1967 [1], the field has grown as a result of surgical and medical advances. Heart transplantation is the gold standard for selected patients with advanced stages of heart failure [2].
- The donor pool is the limiting factor to increasing the number of heart transplants.
- The process of listing a patient for a heart transplantation is complex and requires multidisciplinary expertise to ensure that a patient meets the criteria.
- Careful donor selection is a prerequisite to successful outcomes in heart transplantation.
- Meticulous surgical techniques of donor cardiectomy and recipient implantation are key steps in the successful outcome of a patient following transplantation.
- Patients are maintained on life-long immunosuppression following transplantation, and the graft is monitored for rejection regularly.
- The outcomes following heart transplant have been excellent and continue to improve despite sicker recipients.

Pre-transplantation Assessment

- In general, heart transplantation is reserved for patients who have advanced heart failure and remain symptomatic despite medical optimization.

A. Kreso (✉) · A. Premkumar · D. D'Alessandro
Division of Cardiac Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: akreso@mgh.harvard.edu; apremkumar@mgh.harvard.edu; dadalessandro@mgh.harvard.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_31

– The severity of symptoms, potentially reversible medical factors, and medical therapy optimization are assessed in each potential transplant recipient.
– An important measure is the VO_2 which measures the amount (volume) of oxygen the body uses while exercising over a fixed time.
– Pulmonary function tests are important in determining the forced expiratory volume in 1 s (FEV1), which is a marker of underlying obstructive lung disease.
– A comprehensive, system-based examination is necessary for each potential recipient, and evaluation is generally completed by the medical, surgical, psychiatric, and social work teams. This multidisciplinary committee reviews blood work, imaging, and functional status of potential recipients.
– While the criteria vary between centers, patients must demonstrate no medical contraindications, ability to participate in guideline directed medical therapy, have no evidence of infection or malignancy, and have a good support system in place.

Stages of Heart Failure

– The New York Heart Association (NYHA) functional class system of congestive heart failure is typically used to classify patients into four different severity classes (Table 31.1).
– Transplant recipients fall into class IV.

Indications for Heart Transplantation

– The International Society for Heart and Lung Transplantation (ISHLT) listing criteria are guidelines to assist in patient selection for heart transplantation [3].
– Based on the ISHLT consensus statement, the indications for heart transplantation are [3] as follows:

1. Cardiogenic shock requiring intra-venous inotropic support or mechanical circulatory support (see mechanical support chapter).
2. Persistent NYHA class IV symptoms despite optimal medical therapy. Peak VO_2 ≤12–14 mL/min or <55% predicted.

Table 31.1 NYHA functional class system of heart failure

Class	Functional capacity
I	No physical limitations
II	Slight limitation of physical activity in the form of moderate exertion
III	Marked limitation of physical activity in the form of minimal exertion
IV	Inability to exert because of symptoms of heart failure at rest

3. Intractable or severe angina symptoms not amenable to percutaneous coronary intervention or coronary artery bypass grafting.
4. Intractable life-threatening arrhythmias unresponsive to therapy.
5. Select patients with restrictive and hypertrophic cardiomyopathies.
6. Arrhythmogenic right ventricular cardiomyopathy or left ventricular non-compaction.
7. Corrected or non-corrected symptomatic congenital heart disease not amenable to palliative or corrective surgery.
8. History of prior cardiac transplant with developing cardiac allograft vasculopathy or symptomatic graft dysfunction without evidence of active rejection.

Contraindications for Heart Transplantation

– The ISHLT also provides guidance on contraindications for heart transplantation.
– The relative contraindications are [3] as follows:

1. Systemic illness with life expectancy <2 years despite heart transplantation. This includes active or recent solid organ or blood malignancy, irreversible kidney or liver dysfunction, and severe obstructive pulmonary disease (FEV1 less than 1 L/min).
2. Severe cerebrovascular or peripheral vascular disease.
3. Irreversible pulmonary hypertension (pulmonary vascular resistance greater than 6 Wood units).
4. Active substance abuse.
5. Inability to comply with drug therapy.
6. Multisystem disease (i.e., amyloidosis) with severe extracardiac organ dysfunction.

– The relative contraindications are [3] as follows:

1. Age >70 years.
2. Active infection (with exception of device-related infection for patients with ventricular assist devices).
3. Morbid obesity (body mass index >35) or cachexia (body mass index <18).

Criteria for Medical Urgency

– The Organ Procurement and Transplantation Network (OPTN) was created because of the National Organ Transplant Act of 1984 to ensure equitable distribution of donor organs in the United States.
– The United Network for Organ Sharing (UNOS) is a nonprofit organization that is contracted to oversee the activities related to transplantation.

Table 31.2 Tiers for heart allocation

Status	Characteristics
1	– VA ECMO – Non-dischargeable surgically implanted non-endovascular biventricular support device – Mechanical circulatory support with life-threatening ventricular arrhythmia
2	– Intra-aortic balloon pump – Non-dischargeable surgically implanted non-endovascular left ventricular assist device – Ventricular tachycardia/ventricular fibrillation, mechanical support not required – Mechanical circulatory support with device malfunction or mechanical failure – Total artificial heart – Percutaneous endovascular mechanical circulatory support device
3	– Dischargeable left ventricular assist device up to 30 days – Multiple inotropes or single high-dose inotropes with continuous hemodynamic monitoring – VA ECMO after 7 days, percutaneous endovascular circulatory support device or IABP after 14 days – Non-dischargeable surgically implanted non-endovascular left ventricular support device after 14 days – Mechanical circulatory support with device infection, thromboembolism, hemolysis, right ventricular failure, mucosal bleeding, aortic insufficiency
4	– Dischargeable left ventricular assist device without discretionary 30 days – Inotropes without hemodynamic monitoring – Re-transplant – Diagnosis of congenital heart disease, ischemic heart disease with intractable angina, hypertrophic cardiomyopathy, restrictive cardiomyopathy, amyloidosis
5	– Approved combined organ transplants: heart–lung; heart–liver; heart–kidney
6	– All remaining active candidates
7	– Inactive/not transplantable

– The OPTN revised the US adult heart allocation policy in 2018 (Table 31.2) [4].
– This policy categorizes transplant candidates into status levels based on illness severity, with Status 1 being the sickest and Status 6 being the least sick.

Donor Selection [5]

- Criteria for donor selection and matching to recipient include:
  - ABO type.
  - Age (generally less than 55 years old).
  - Body size (want a similar BMI between donor and recipient).
- The pool of donors is limited and generally there are two categories of donation: donation after brain death (DBD) and donation after circulatory death (DCD).
- This reflects the two legal ways by which death can be pronounced. Death may be pronounced when a person's heart stops beating (circulatory death) or when the person's brain stops functioning (brain death).

DBD

- Brain death is the complete, irreversible loss of all brain function due to lack of blood supply to the brain.
- Brain death is diagnosed by a persistent coma and absence of brainstem reflexes. Additional testing, such as radionucleotide brain scans, transcranial Doppler ultrasound, or cerebral angiography can be used to confirm brain death.
- Once a patient is declared brain dead, evaluation for cardiac donation begins.
- Heart beating brain dead donors represent a large portion of the hearts that are used for transplantation purposes within the US.
- The advantage of having a beating heart is that the ischemia time is limited. As well, the cardiac damage that results from the agonal withdrawal period is avoided.

DCD [6]

- Since the original case series on the effective use of DCD hearts, this source of donors has been increasingly used [7].
- Determination of death criteria varies among hospitals and states.
- There is an obligatory hypoxemic time that the heart suffers after circulatory death.
- To use DCD hearts, these hearts need to be assessed and resuscitated before implant.
- There are two competing techniques to retrieve DCD hearts: normothermic regional perfusion (NRP) or direct procurement and perfusion (DPP).
- During NRP, perfusion is restored to the arrested heart within the donor. After the declaration of death, ECMO or cardiopulmonary bypass (CPB) is established to resuscitate and evaluate the heart in the patient. The great vessels to the head are occluded to prevent cerebral circulation. The heart is evaluated in the loaded state and if found suitable, it is removed and maintained on ice for transportation.
- During DPP, the heart is removed directly. After the declaration of death, blood is collected from the patient and used to prime the organ care system (OCS, Transmedics). The heart is then connected to the OCS machine, where it is perfused. The left ventricle is fully decompressed.
- The heart can be assessed by measuring lactate as the heart should be consuming lactate, rather than generating lactate. This serves as a surrogate to heart function.
- For the original DCD trials, patients had to meet death criteria within 30 min of warm ischemia time (WIT).
- WIT in the DCD trial was defined as time from when the mean systolic blood pressure is less than 50 mmHg or peripheral saturation is less than 70% to aortic cross clamp and administration of cold cardioplegia to the donor.

Surgical Techniques [8]

Preservation Options

- General preservation strategies to protect the allograft during procurement, transport, and implantation are as follows:
  1. Topical hypothermia: using ice and iced saline with goal temperature of 4 °C.
  2. Hypothermic perfusate: cardioplegia infusion is delivered during recovery. The commonly used solutions are as follows: Stanford, modified EuroColins, and University of Wisconsin solution.
- Historically, other preservation options were utilized, including donor systemic cooling via cardiopulmonary bypass and allograft continuous perfusion (e.g., autoperfusion).

Donor Cardiectomy: General Sequence During DBD Procurement

1. Large incision and sternotomy.
2. Pericardiotomy and pericardial well creation.
3. Dissect the aorta from the pulmonary artery and encircle.
4. Dissect superior and inferior venae cavae circumferentially.
5. Once the abdominal team is ready, give heparin.
6. Place a cardioplegia catheter into ascending aorta.
7. Clamp the aorta and start cardioplegia.
8. Vent the left heart by either incising the LA (if lungs will be procured) or by dividing the pulmonary vein.
9. Transect the inferior vena cava (location discussed with liver team).
10. Transect the superior vena cava.
11. Transect the aorta.
12. Transect the pulmonary artery near its bifurcation.
13. Transect the left atrium (location discussed with lung team).

Donor Cardiectomy in DCD

- Heparin is administered prior to withdrawal of life-supportive therapy.
- WIT is measured and if the patient has a loss of pulse, an institution specific observation period is respected (generally 5 min) before confirming death according to national guidelines.
- The portable machine perfusion device (currently OCS Transmedics) is prepared if DPP protocol is used.

NRP Protocol

- After declaration of death, sternotomy is performed.
- The head vessels are tied off.
- Cannulas can be inserted into the ascending aorta and right atrium to restore perfusion to the body.
- Alternatively, the patient can be placed on peripheral extracorporeal membrane oxygenation and the head vessels tied off.
- Functional assessment of the heart can be done using a pulmonary artery line and transesophageal echocardiogram.
- If found suitable, the heart is arrested with cardioplegia and donor cardiectomy performed (as described above).
- Generally, the heart is transported back to the transplant center on ice.

DPP Protocol

- As soon as death is confirmed, median sternotomy is performed.
- A large cannula is inserted into the right atrium and blood is drained for priming of the OCS.
- Ideally, only after 1–1.5 L of blood is collected, the abdominal team can deliver preservation solution.
- Thereafter, cold cardioplegic solution is administered into the aortic root before retrieval of the heart and instrumentation on the OCS.
- Once the heart is removed from the donor, it is placed onto the OCS device (Fig. 31.1).

Implant Technique

- Prior to the donor heart arriving, the recipient cardiectomy would have proceeded in a similar manner to the donor cardiectomy.
- Bicaval and central aortic cannulation is employed, and the patient is placed on cardiopulmonary bypass and cooled.
- The major vessels are transected and prepared for implant (Fig. 31.2).
- The donor heart is inspected, and the heart is implanted in an expedited fashion.
- The sequence of anastomoses varies but generally it proceeds in the following manner:

  1. Left atrium.
  2. Inferior vena cava.
  3. Superior vena cava (backwall).
  4. Pulmonary artery.

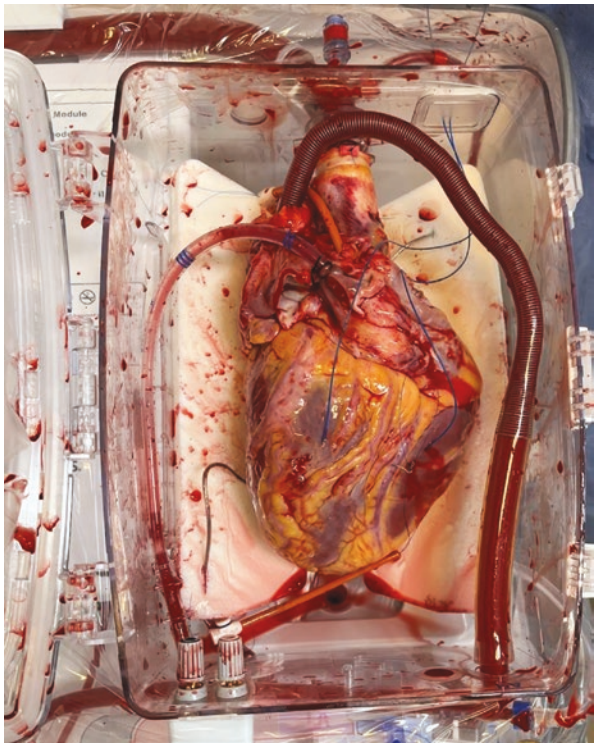


Fig. 31.1 DCD heart preservation. The aorta is connected to the inflow, and the pulmonary artery is cannulated and connected to an outflow port. A left ventricular vent is placed. The cavae are snared, and pacing wires are attached. The machine is turned on, and the heart is perfused with the left ventricle unloaded

5. Aorta.
6. The cross-clamp is then removed, and the front wall of the SVC is completed.

– The patient is weaned from cardiopulmonary bypass, and the chest is closed.

Immunosuppression

– Generally, a three-drug therapy regimen is employed:

1. Calcineurin inhibitor (cyclosporine or tacrolimus).
2. Antiproliferative agent (mycophenolate mofetil).
3. Corticosteroid.

– Some centers use selective induction agents, such as basiliximab, that target the interleukin-2 receptors on T cells, or nonselective immunosuppressive agents such as antithymocyte globulin, in the perioperative period to decrease the incidence of early rejection.

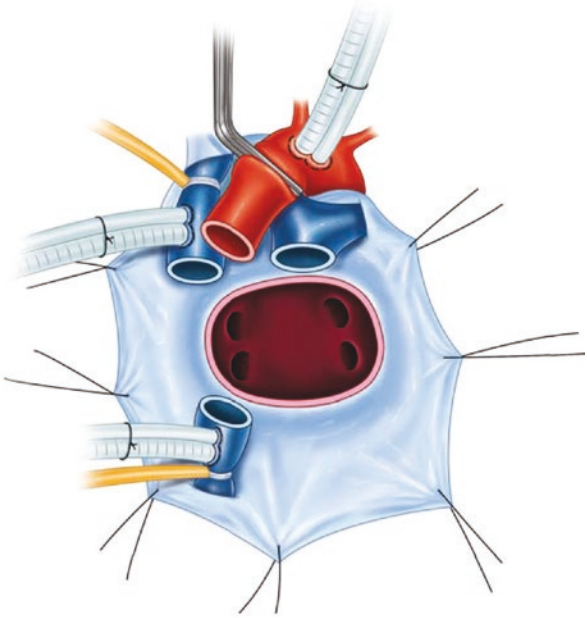


Fig. 31.2 Recipient cardiectomy. The recipient heart has been removed. The cavae are snared with cannulas in place. The aorta has been transected distal to an aortic clamp and aortic cannula. The pulmonary artery and left atrial cuffs are created

Post-operative Care Considerations

- Maintaining adequate end organ perfusion is critical in the early post-operative period.
- The donor heart lacks autonomic nerves (sympathetic and parasympathetic), and the intrinsic heart rate in a transplanted heart is typically 90–110 beats per minute and is dependent on circulating catecholamines.
- The elevated heart rate should be maintained with chronotropic agents or epicardial pacing wires to reduce the likelihood of right ventricular failure by reducing end-diastolic volume.
- Newly transplanted hearts typically require inotropic support for several days.

Outcomes

- The OPTN and the Scientific Registry of Transplant Recipients (SRTR) publish annual data from the registry about mortality related to heart transplantation.
- Each transplant center is required to share outcome data with the patient and compare this to the national outcome data (www.srtr.org).
- The most recent registry report shows that the 1-year survival post heart transplantation exceeds 90% in the US [9].

– The survival outcome trends over the years show that survival is improving despite sicker patients receiving the heart transplants.
– Mortality following heart transplantation is usually secondary to primary graft failure (within the first 30 days), opportunistic infections (6 months–1 year), acute allograft rejection (first 3 years), cardiac allograft vasculopathy, and malignancy (late) [10].

References

1. Bernard CN. The operation. A human cardiac transplant: an interim report of a successful operation performed at Groote Schuur Hospital, Cape Town. S Afr Med J. 1967;41(48):1271–4.
2. Lower RR, Shumway NE. Studies on orthotopic homotransplantation of the canine heart. Surg Forum. 1960;11:18–9.
3. Mehra MR. Guidelines for listing candidates for heart transplant: a 10-year update. JAMA Cardiol. 2017;2(1):98–9.
4. Meyer DM, Rogers JG, Edwards LB, Callahan ER, Webber SA, Johnson MR, Vega JD, Zucker MJ, Cleveland JC Jr. The future direction of the adult heart allocation system in the United States. Am J Transplant. 2015;15(1):44–54.
5. Copeland H, Hayanga JWA, Neyrinck A, MacDonald P, Dellgren G, Bertolotti A, Khuu T, Burrows F, Copeland JG, Gooch D, Hackmann A, Hormuth D, Kirk C, Linacre V, Lyster H, Marasco S, McGiffin D, Nair P, Rahmel A, Sasevich M, Schweiger M, Siddique A, Snyder TJ, Stansfield W, Tsui S, Orr Y, Uber P, Venkateswaran R, Kukreja J, Mulligan M. Donor heart and lung procurement: a consensus statement. J Heart Lung Transplant. 2020;39(6):501–17.
6. Messer S, Page A, Axell R, Berman M, Hernández-Sánchez J, Colah S, Parizkova B, Valchanov K, Dunning J, Pavlushkov E, Balasubramanian SK, Parameshwar J, Omar YA, Goddard M, Pettit S, Lewis C, Kydd A, Jenkins D, Watson CJ, Sudarshan C, Catarino P, Findlay M, Ali A, Tsui S, Large SR. Outcome after heart transplantation from donation after circulatory-determined death donors. J Heart Lung Transplant. 2017;36(12):1311–8.
7. Dhital KK, Iyer A, Connellan M, Chew HC, Gao L, Doyle A, Hicks M, Kumarasinghe G, Soto C, Dinale A, Cartwright B, Nair P, Granger E, Jansz P, Jabbour A, Kotlyar E, Keogh A, Hayward C, Graham R, Spratt P, Macdonald P. Adult heart transplantation with distant procurement and ex-vivo preservation of donor hearts after circulatory death: a case series. Lancet. 2015;385(9987):2585–91.
8. Liao K, Ranjit J. Orthotopic heart transplantation. Oper Tech Thorac Cardiovasc Surg. 2010;15(2):138–46.
9. Colvin M, Smith JM, Ahn Y, Skeans MA, Messick E, Bradbrook K, Gauntt K, Israni AK, Snyder JJ, Kasiske BL. OPTN/SRTR 2020 annual data report: heart. Am J Transplant. 2022;22(Suppl 2):350–437.
10. Lund LH, Edwards LB, Kucheryavaya AY, Benden C, Dipchand AI, Goldfarb S, Levvey BJ, Meiser B, Rossano JW, Yusen RD, Stehlik J. The Registry of the International Society for Heart and Lung Transplantation: Thirty-second Official Adult Heart Transplantation Report—2015; Focus Theme: Early Graft Failure. J Heart Lung Transplant. 2015;34(10):1244–54.

Chapter 32
Lung Transplantation

Eliza D. Hompe and Asishana A. Osho

Indications for Transplant and Recipient Selection [1, 2]

- Lung transplantation is indicated for end-stage lung disease that is refractory to medical or surgical management. The type of lung disease can be categorized as obstructive, restrictive, infectious, or pulmonary vascular disease.
- The most common indications for lung transplantation are chronic obstructive pulmonary disease (COPD) and interstitial lung disease, specifically idiopathic pulmonary fibrosis (IPF), which is a type of restrictive lung disease (Fig. 32.1). In addition, other lung pathologies that frequently progress to transplantation are cystic fibrosis/bronchiectasis, alpha-1 antitrypsin deficiency (obstructive), and primary pulmonary hypertension (vascular) (Fig. 32.1).
- Given the paucity of organ donors, it is important to carefully select appropriate candidates for lung transplantation. There have been several iterations of consensus guidelines for recipient selection, last updated in 2015 by the International Society for Heart and Lung Transplantation (ISHLT).
- Generally a patient with end-stage lung disease should meet the following three criteria:
  - High (>50%) risk of death from lung disease within 2 years without transplantation
  - High (>80%) likelihood of surviving at least 90 days after lung transplantation
  - High (>80%) likelihood of 5-year post-transplant survival from a general medical perspective.

E. D. Hompe (✉) · A. A. Osho
Department of Cardiac Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: EHOMPE@MGH.HARVARD.EDU; Asishana.Osho@MGH.HARVARD.EDU

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_32

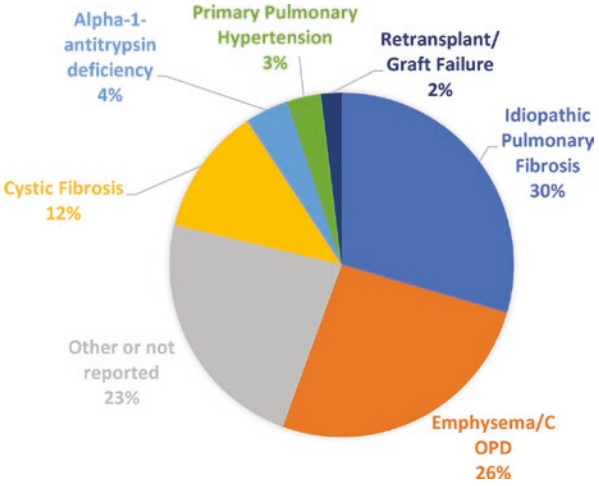


Fig. 32.1 Indications for lung transplantation in recipients. Based on Organ Procurement and Transplantation Network (OPTN) data for lung transplants performed in the US from January 1, 1988 to March 31, 2022, as of April 30, 2022

- Absolute contraindications to transplant are untreatable malignancy, severe uncorrectable dysfunction of another organ system (i.e., coronary artery disease not amenable to revascularization or renal failure), obesity (BMI >35 kg/m^2), nonadherence to medical therapy, substance abuse, and poor social support.
- Relative contraindications to transplant include age >65 years, prior cardiothoracic surgery, or HIV, hepatitis B or hepatitis C infection.

Lung Allocation Score [3]

- Once a patient is determined to be an appropriate transplant candidate based on the progression of his or her lung disease and comorbidities, the candidate is placed on the waitlist.
- Each candidate (age 12 and older) then receives a lung allocation score (LAS). Similar to the MELD score for liver transplantation, the LAS helps to determine priority for lung transplantation when a donor organ becomes available, combined with other factors such as blood type and geographic area.
- The LAS is based on patient characteristics including age and type of lung disease, as well as objective parameters such as BMI, 6-min walk distance, and pulmonary capillary wedge pressure (Table 32.1).
  - It is used to prioritize a candidate for transplant based on their wait list mortality and projected post-transplant survival.
  - It is measured on a scale of 0–100 and if a patient has a higher LAS, the patient is more likely to benefit from a transplant and thus will receive higher priority in the allocation process.
- Since the LAS was implemented in 2005, there have been fewer waitlist deaths, more overall transplants, and an increase in transplants for patients with IPF.

Table 32.1 Components of the Lung Allocation Score (LAS)

Age
BMI
Lung diagnosis code[a]
Functional status[b]
Assisted ventilation[c]
Supplemental O_2 requirement
Pulmonary artery systolic pressure (mmHg)
Mean pulmonary artery pressure (mmHg)
Cardiac index (L/min/m^2)
6-min walk distance (feet)
Total bilirubin
Serum creatinine
PCO_2 (mmHg)

[a]Lung diagnosis code is the etiology of the candidate's lung disease, e.g., alpha-1 antitrypsin deficiency or sarcoidosis
[b]Functional status is defined as performing activities of daily living with no, some or total assistance
[c]Types of assisted ventilation include BiPAP (bilevel positive airway pressure), CPAP (continuous positive airway pressure), mechanical (intermittent or continuous), or none

Single vs. Double Lung Transplant [4–6]

- Both lungs from a donor can be transplanted into a single individual (double lung transplant) or can be split to benefit two recipients (each receives a single lung transplant).
- Importantly, patients with infectious lung disease such as cystic fibrosis are only eligible for a double lung transplant, given the high risk of contamination of the transplanted lung by organisms in the remaining native lung. Alternatively, patients with COPD or IPF can be eligible for single or double lung transplants.
- Single lung transplants are technically easier to perform, resulting in shorter operative time and decreased perioperative morbidity. Because a set of donor lungs can benefit two recipients, there is also greater societal benefit provided by single lung transplants.
- Multiple studies have demonstrated that double lung transplantation leads to higher long-term survival and improved quality of life in patients with COPD and IPF. A double lung transplant is also optimal for a patient with a high LAS.

Donor Selection [7, 8]

- There are two types of deceased organ donors—DBD, or donation after brain death, and DCD, donation after cardiac death.
- In donation after brain death, a patient is declared brain dead and their organs are procured while their heart and lungs continue to function and perfuse their body, supported by a ventilator.

- In donation after cardiac death, the heart stops functioning prior to organ procurement. This can occur in the setting of cardiac arrest or planned withdrawal of life support.
- The introduction of DCD donors has expanded the donor pool at the expense of increased warm ischemia time.
- "Ideal" donor criteria have been established but, in reality, few donors satisfy all of the requirements and "expanded" criteria are often used.
  - Ideal donor criteria include age <55 years old, ABO compatibility, PaO_2 >300 mmHg on 100% FiO_2, and PEEP 5 cm H_2O of PEEP, no smoking history, no evidence of pulmonary infection on chest X-ray, bronchoscopy or gram stain of sputum, and no prior cardiothoracic surgery.
  - Studies have demonstrated that even if one or more of these criteria is not met, there are comparable postoperative outcomes and survival in transplant recipients.

Donor Pneumonectomy [9, 10]

- Once a potential donor is screened and selected, the patient is brought to the operating room for organ procurement.
- At that time, the lung procurement team first performs a bronchoscopy to examine the airway anatomy and confirm that there are no signs of infection (i.e., purulent secretions).
- The steps of the donor pneumonectomy operation are as follows:
  - Exposure and dissection

    A median sternotomy is performed. The pericardium is opened with electrocautery. Silk sutures are used to retract the pericardium bilaterally. The bilateral pleural spaces are then opened. The lungs are inspected carefully and manually palpated for any abnormalities. Any regions of the lung with atelectasis are recruited. Confirmatory arterial blood gases are sent centrally and from each individual pulmonary vein after recruitment maneuvers while the lung is being ventilated on FiO_2 100% and positive end-expiratory pressure (PEEP) 5 mmHg.

    Next, the superior vena cava (SVC) is identified, dissected out bluntly, and encircled with suture or umbilical tape. A separate suture is passed around the azygous vein which may be ligated at this time but does not need to be divided. The inferior vena cava (IVC) is also dissected out circumferentially.

    Dissection is then carried out to separate the ascending aorta from the pulmonary artery (PA). The aorta may also be encircled with umbilical tape.

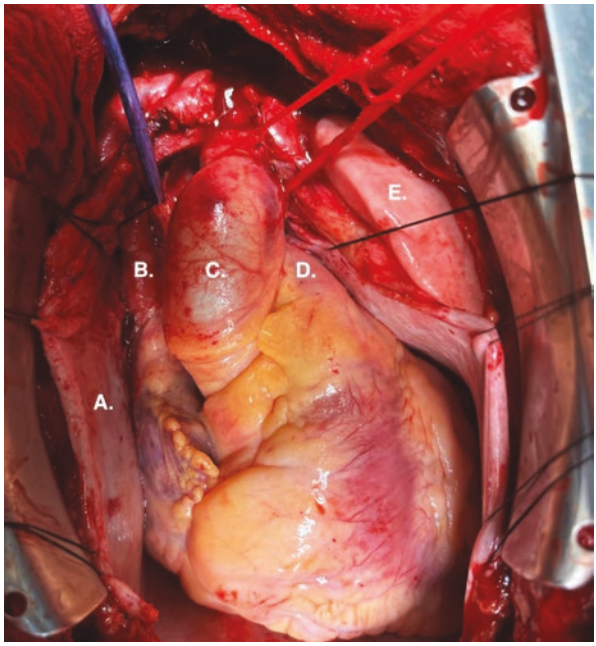


Fig. 32.2 Donor pneumonectomy exposure. (**a**) Opened pericardium with silk stay sutures for retraction. (**b**) Superior vena cava (SVC). (**c**) Ascending aorta encircled with umbilical tape. (**d**) Pulmonary artery. (**e**) Left lung

The posterior pericardium is then incised to expose the trachea, and it is freed from its attachments manually.

– Cannulation

Once the dissection is complete, purse string sutures (4–0 polypropylene) are placed on the ascending aorta and main PA just proximal to its bifurcation. These sutures are used to secure cannulas which are needed for infusion of preservation solutions.

Prior to cannulation, systemic heparin is administered to the donor. The first cannula is inserted into the ascending aorta and will administer cardioplegia to the heart after aortic cross-clamp. A second perfusion cannula is inserted into the PA and will be used for antegrade lung flush with preservation solution (Fig. 32.2).

– Aortic cross-clamp

Prostaglandin E_1 is injected into the PA for pulmonary vasodilation. This agent can cause significant systemic hypotension so all procurement teams must be notified prior to administration.

Blood flow to the heart is stopped by ligation of the SVC and division of the IVC.

The aorta is cross-clamped, and cardioplegia solution is administered.

The left atrial appendage is incised to vent the left heart.

Lung preservation solution is flushed through the PA cannula, and a mixture of ice and saline is poured into the pleural spaces bilaterally to cool the lungs.

– Organ extraction

The heart is then extracted, with a cuff of left atrium left around the orifices of the pulmonary veins.
Each lung is retracted anteriorly and cephalad to expose the inferior pulmonary ligament which is divided.
The posterior pericardium is opened and a plane created anterior to the esophagus. Mediastinal tissue on either side is separated from the esophagus up to the hilum.
The lungs are then retracted inferiorly and further dissected off the esophagus and descending aorta.
The trachea is stapled and divided at least 2 rings above the carina after withdrawing the endotracheal tube high into the airway. The lungs are then removed, triple-bagged with cold preservation solution, and placed on ice.

- The most common method of lung preservation is cold static preservation, in which the organ is flushed with preservation solution (low-potassium dextran solution) and kept on ice. Traditionally, lungs that undergo cold static preservation are considered viable for up to 8 h after procurement. Recent studies suggest however that the lung may be able to tolerate even longer cold ischemic times.

Recipient Procedure [11]

- Lung transplantation is commonly performed via a "clamshell" incision, which is a bilateral anterior thoracotomy in the fourth intercostal space with a transverse sternotomy. Other incisions used for lung transplantation include thoracotomy (unilateral or bilateral without transverse sternotomy) and median sternotomy.
- Lung transplantation may be performed without cardiopulmonary support, with extra-corporeal membrane oxygenation (ECMO) or with full cardiopulmonary bypass (CPB). Choice between these strategies is generally a matter of experience and center preference although certain clinical situations may necessitate the use of ECMO or CPB.
- Explant technique
  - After a clamshell incision is made, two rib spreaders, or Finochietto retractors, are inserted to expose the heart and lungs. Any adhesions between the lungs and parietal pleura are taken down, with careful attention to hemostasis during adhesiolysis.
  - The inferior pulmonary ligaments are divided, and the pulmonary artery, veins, and bronchus are dissected free and mobilized. The pulmonary artery, veins, and bronchus are sequentially stapled to complete the pneumonectomy.

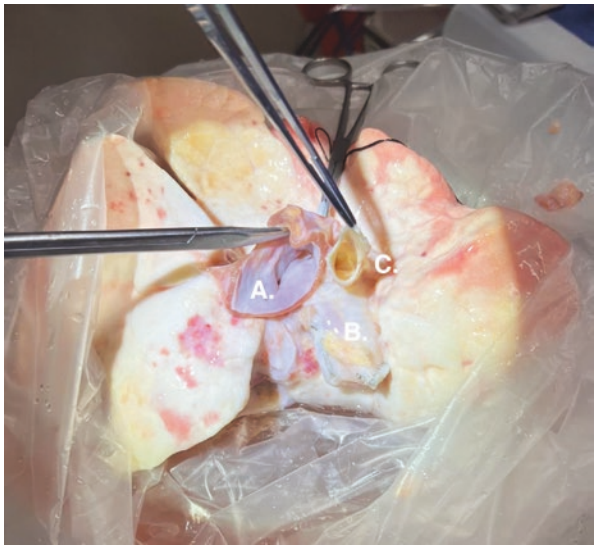


Fig. 32.3 Lung hilum dissection. The donor lungs, transported in cold preservation solution and triple-bagged, are dissected on the back table prior to implantation. The hilar structures— left atrial cuff/pulmonary veins (**a**), bronchus (**b**), and pulmonary artery (**c**)—are identified and dissected out

  - During all aspects of dissection and explanation of the lungs, it is crucial to protect the phrenic, vagus, and recurrent laryngeal nerves.

- Implant technique.
  - The donor lungs are split on the back table if harvested en bloc and prepared for implantation (Fig. 32.3). The lungs are sequentially transplanted. The lung with worse function is removed first, with single lung ventilation via the contralateral lung. If there is a discrepancy in lung function, the right lung is implanted first because the anatomy is more favorable.

– There are three anastomoses in a lung transplant—the bronchial, pulmonary artery, and left atrial cuff/pulmonary vein anastomoses. The order of anastomoses and surgical technique varies across institutions. Generally, the bronchial anastomosis (most posterior) is done first, followed by the pulmonary artery anastomosis and, lastly, the pulmonary vein anastomosis.

  The bronchial anastomosis is an "end-to-end" anastomosis, sewn with running absorbable suture (PDS) around the entire circumference of the airway. To prevent ischemia, the donor airway is cut back to within one ring of the upper lobe take off as it is devascularized during explant. The bronchial anastomosis is often reinforced with an intercostal muscle or pericardial flap to separate it from the vascular anastomoses and prevent the formation of a fistula between the airway and the vasculature.
  The pulmonary artery anastomosis is similarly sewn with a running prolene suture, and the pulmonary vein anastomosis with a running prolene suture.
  After the anastomoses are complete, the lung is carefully deaired and gradually reperfused over the course of 10–15 min as rapid exposure of the

implanted lung to significant cardiac output can lead to ischemia reperfusion injury. The surgical field is carefully inspected for hemostasis.

– Two or more chest tubes are placed in each pleural cavity at the conclusion of the case. Sternal wires are used to reapproximate the sternum and the incision closed in layers. A postoperative bronchoscopy is performed to inspect the patency and caliber of the bronchial anastomoses and perform pulmonary toilet to eliminate accumulated blot and mucus.

Immunosuppression [12, 13]

- Patients who undergo lung transplantation are started on immunosuppression to prevent rejection. At the time of transplant, high-dose immunosuppressive agents are administered to dampen the T-cell immune response and, over time, the immunosuppression is gradually down titrated to maintenance dosing.
- High-dose glucocorticoids (IV methylprednisolone) are administered prior to lung perfusion, usually during completion of the first vascular anastomosis, to decrease reperfusion injury.
- An induction immunosuppression agent may be given at the time of transplant to reduce the risk of acute organ rejection. Most patients who undergo lung transplantation in the United States receive induction therapy (80.9%).
- The most common induction agents used are basiliximab (monoclonal antibody (mAb) against the interleukin-2 receptor), rabbit anti-thymocyte globulin (T-cell depleting agent), and alemtuzumab (mAb against CD52 and T-cell depleting agent).
  - One important contraindication to giving induction therapy is if the patient is at high risk for certain postoperative infections, such as in cases of CMV mismatch (CMV+ organ is transplanted into a CMV− recipient).
- For maintenance immunosuppression, most patients are on tacrolimus (a calcineurin inhibitor), mycophenolate mofetil (a nucleotide blocking agent), and a glucocorticoid such as prednisone.
  - Patients are monitored closely for side effects of these medications and take prophylaxis against potential opportunistic infections.

Outcomes [13–15]

- In the most recent report on outcomes of lung transplantation in the United States, post-transplant survival was 89.4% at 1 year, 74.8% at 3 years, and 61.2% at 5 years. The incidence of acute rejection was reported to be 14.6%, but multiple other studies describe higher rates of acute rejection, approaching 30–40%.

Chronic lung allograft dysfunction (bronchiolitis obliterans) was reported in 5.7% of recipients at 1 year and 40.2% at 5 years.

- The average time to transplant for candidates on the waitlist was 1.4 months. In recent years, there has been a notable increase in transplant rates for adults 65 years and older and patients with restrictive lung disease.
- Other contributors to post-transplant morbidity and mortality include infections, particularly in the first year after transplant, the development of renal insufficiency (often secondary to calcineurin inhibitors) and malignancies (post-transplant lymphoproliferative disorder, skin cancer, and solid organ tumors) in the long term.
- With the increase in the number of DCD donors, it is important to compare outcomes and survival rates in patients who receive DBD versus DCD lungs. Recent data showed no difference in 5-year survival in DCD recipients compared to DBD recipients. In addition, while primary graft dysfunction was shown to be worse for DCD lungs in the immediate postoperative setting, it was noted to improve rapidly and there was ultimately no difference in graft function at 72 h.
- Innovations to address the donor shortage, such as increased use of DCD donors, organ engineering and xenotransplantation, as well as strategies to prevent and treat chronic lung allograft dysfunction, will ultimately be needed to make meaningful progress in the field.

References

1. Weill D. Lung transplantation: indications and contraindications. J Thorac Dis. 2018;10(7):4574–87.
2. Weill D, Benden C, Corris PA, Dark JH, Davis RD, Keshavjee S, et al. A consensus document for the selection of lung transplant candidates: 2014—an update from the Pulmonary Transplantation Council of the International Society for Heart and Lung Transplantation. J Heart Lung Transplant. 2015;34(1):1–15.
3. Lyu DM, Goff RR, Chan KM. The lung allocation score and its relevance. Semin Respir Crit Care Med. 2021;42(3):346–56.
4. Puri V, Patterson GA, Meyers BF. Single versus bilateral lung transplantation: do guidelines exist? Thorac Surg Clin. 2015;25(1):47–54.
5. Thabut G, Christie JD, Ravaud P, Castier Y, Brugière O, Fournier M, et al. Survival after bilateral versus single lung transplantation for patients with chronic obstructive pulmonary disease: a retrospective analysis of registry data. Lancet. 2008;371(9614):744–51.
6. Weiss ES, Allen JG, Merlo CA, Conte JV, Shah AS. Survival after single versus bilateral lung transplantation for high-risk patients with pulmonary fibrosis. Ann Thorac Surg. 2009;88(5):1616–25.
7. Chaney J, Suzuki Y, Cantu E III, van Berkel V. Lung donor selection criteria. J Thorac Dis. 2014;6(8):1032–8.
8. Lardinois D, Banysch M, Korom S, Hillinger S, Rousson V, Boehler A, et al. Extended donor lungs: 11 years experience in a consecutive series. Eur J Cardiothorac Surg. 2005;27(5):762–7.
9. Puri V, Patterson GA. Adult lung transplantation: technical considerations. Semin Thorac Cardiovasc Surg. 2008;20(2):152–64.

10. Sundaresan S, Trachiotis GD, Aoe M, Patterson GA, Cooper JD. Donor lung procurement: assessment and operative technique. Ann Thorac Surg. 1993;56(6):1409–13.
11. Gust L, D'Journo XB, Brioude G, Trousse D, Dizier S, Doddoli C, et al. Single-lung and double-lung transplantation: technique and tips. J Thorac Dis. 2018;10(4):2508–18.
12. Chung PA, Dilling DF. Immunosuppressive strategies in lung transplantation. Ann Transl Med. 2020;8(6):409.
13. Valapour M, Lehr CJ, Skeans MA, Smith JM, Miller E, Goff R, et al. OPTN/SRTR 2020 annual data report: lung. Am J Transplant. 2022;22(Suppl 2):438–518.
14. Van Raemdonck D, Keshavjee S, Levvey B, Cherikh WS, Snell G, Erasmus M, et al. Donation after circulatory death in lung transplantation-5-year follow-up from ISHLT Registry. J Heart Lung Transplant. 2019;38(12):1235–45.
15. Villavicencio MA, Axtell AL, Spencer PJ, Heng EE, Kilmarx S, Dalpozzal N, et al. Lung transplantation from donation after circulatory death: United States and single-center experience. Ann Thorac Surg. 2018;106(6):1619–27.

Chapter 33
Adult Congenital Heart Disease

Selena S. Li and Jordan P. Bloom

Adult Presentation of Common Congenital Heart Defects

In the modern era, over 90% of children with congenital heart defects survive into adulthood, and the prevalence of CHD has shifted from infancy/childhood to adulthood [1]. The presentation of adults with congenital heart defects encompasses both those who diagnosed and treated in childhood, as well as new diagnoses. Over 75% of ACHD patients have had prior palliative surgeries and interventions and have established care with cardiology and cardiac surgery. However, the remaining 25% can present with either new symptoms of a known defect or a previously undetected diagnosis [2]. The symptoms of CHD that manifest later in life differ from those that present in childhood and can often be misdiagnosed.

S. S. Li · J. P. Bloom (✉)
Division of Cardiac Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: ssli@mgb.org; jpbloom@mgh.harvard.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_33

	Definition	Presenting symptoms in the adult
Atrial septal defect (ASD) and patent foramen ovale (PFO)	ASD = membranous defect in interatrial septum, resulting in connection between the right and left atria PFO = failure of closure of foramen ovale, with persistent connection between the right and left atria	Isolated ASD/PFO • Exam – Fixed, split S2 From delayed closure of the pulmonary valve (PV) – Systolic murmur at upper left sternal border Increased blood flow across pulmonary valve (PV) – Mid-diastolic murmur at lower left sternal border Increased flow across tricuspid valve • Paradoxical embolism – Stroke – Peripheral embolism – Mesenteric ischemia • Right heart failure – Elevated jugular venous pressure (JVP) – Peripheral edema – Hepatic congestion • Eisenmenger syndrome (right-to-left shunt) – Cyanosis – Shortness of breath – Clubbing – Hemoptysis *May also present associated with other congenital syndromes
Partial anomalous pulmonary venous return (PAPVR) [3]	Pulmonary veins drain erroneously to right heart (SVC, IVC, RA) instead of to LA	*Depends on degree of left-to-right shunting • Shortness of breath • Peripheral edema • Chest pain/discomfort • Palpitations
Ventricular septal defect (VSD)	Defect in membranous or muscular interventricular septum, resulting in connection between RV and LV	*Most common congenital heart defect • Exam – Loud S1 with wide, fixed split S2 – Holosystolic murmur at left sternal border Left-to-right shunt • Right heart failure • Elevated JVP • Peripheral edema • Orthopnea Eisenmenger syndrome (right-to-left shunt) • See above
Coronary anomalies [4]	May include anomalies of coronary vessel: – Origin – Course – Termination – Collateral vessels	• Incidental finding on CT/CTA • Chest pain/angina • Dyspnea on exertion • Syncope • Sudden cardiac death – Higher risk in anomalous LEFT coronary artery

Tetralogy of Fallot

Tetralogy of Fallot (ToF) is the most common cyanotic congenital heart disease, and historically, the first to be palliated surgically.

Anatomy/Pathophysiology

- Infundibular portion of ventricular septum is displaced anteriorly into the right ventricular outflow tract (RVOT)—remember the mnemonic "PROVe" (Fig. 33.1).
  - Pulmonary stenosis.
  - Right ventricular (RV) hypertrophy (resulting from pulmonary stenosis).
  - Overriding aorta.
  - VSD.

Initial Operation

- Neonatal repair (Fig. 33.2).
  - Principles:
    1. VSD closure.
    2. Relief of RVOT obstruction.

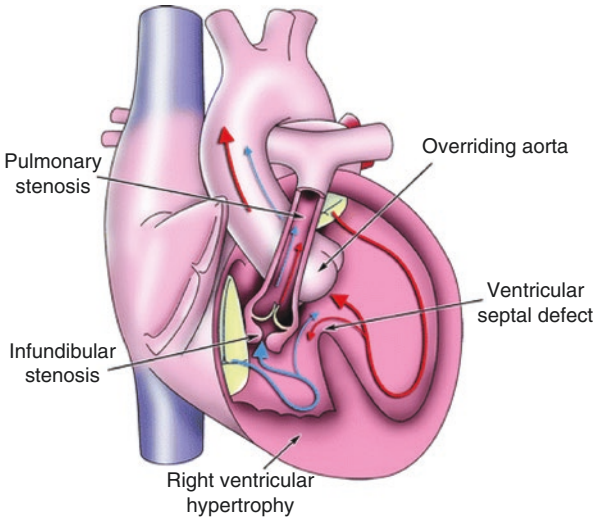


Fig. 33.1 Tetralogy of Fallot anatomy

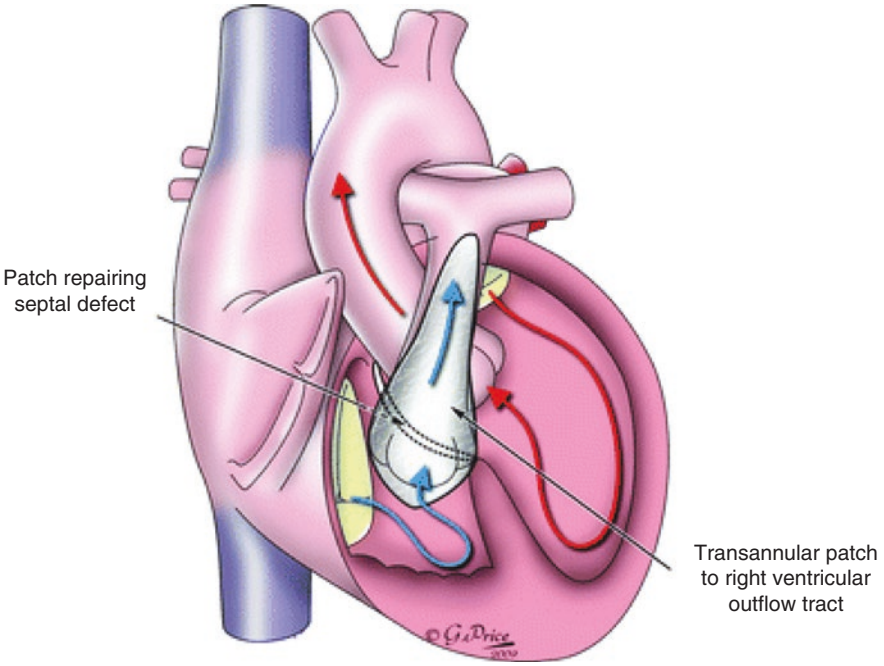


Fig. 33.2 Surgical repair of tetralogy of Fallot with transannular patch

- Pulmonary valvotomy and augmentation of the infundibulum.
- Transannular patch.
- RV to PA conduit.

Common Complications

- Severe pulmonary regurgitation.
  - RV dilation or dysfunction (usually much later in life).
- Risk for arrhythmia and sudden cardiac death.
- Heart failure.
- Aortic root dilation and aortic regurgitation.

Reoperative Surgery

- Principles:
  - Prior surgical repair techniques (transannular patch) result in PR which leads to risk of RV dilation and arrhythmia in adulthood.

  - Reoperative surgery involves pulmonary valve replacement (PVR).
- Indications for PVR.
  - Severe PR and symptoms or decreased exercise tolerance.
  - Asymptomatic severe PR and any of the following:

    Moderate to severe RV dysfunction.
    Moderate to severe RV enlargement.
    Symptomatic or sustained arrhythmia.
    Moderate to severe TR.
- Surgery versus catheter-based intervention.
  - Percutaneous PVR.

    Current studies have focused on use in patients with previous surgical RVOT conduits [5].
    Short-term results comparable to surgical cohorts in terms of reductions in pulmonary regurgitation and RV volume.
    No randomized trials that directly compare the two.

Coronary Artery Anomalies

Coronary artery anomalies can broadly be categorized as anomalies of origin, course, or termination of the artery, or abnormal collateral vessels. Discussed here are anomalous coronary arteries originating from the wrong sinus as it is the anomaly most frequently associated with sudden cardiac death.

Anomalies of Origin

	Anomalous right coronary (RCA)	Anomalous left coronary (LCA)
Epidemiology [6]	– 0.28% originating from left sinus of Valsalva – 0.003% from pulmonary artery [4] – Increased risk with ToF or double outlet RV	– 0.03% from right sinus of Valsalva – 0.008% from pulmonary artery [4]
Workup	– Imaging (CT, MRI, intravascular US, left heart catheterization) – Nuclear stress test	– Imaging (CT, MRI, intravascular US, left heart catheterization) – Nuclear stress test
Treatment	Asymptomatic, negative stress test: – Regular follow-up Symptomatic or positive stress: – PCI w/stent – Surgery	Age <35 – Surgery (regardless of symptoms) Age ≥35 – Surgery if symptomatic or positive stress

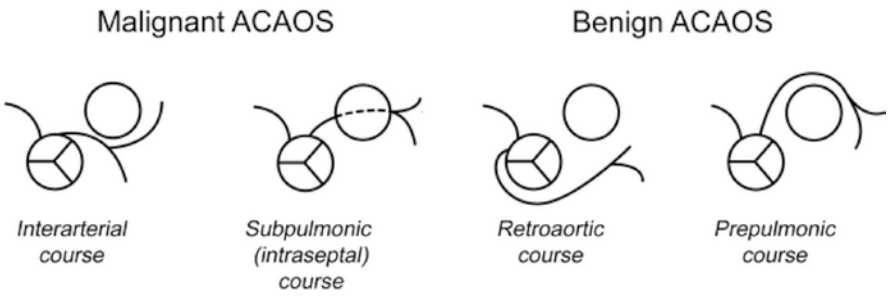


Fig. 33.3 Courses of anomalous coronary arteries

Imaging

- CT superior to MRI for definition of course of anomalous coronary artery (Fig. 33.3).

Prepulmonary (anterior)	– Courses anterior to pulmonary artery
Retroaortic	– Courses posterior to aorta
Transseptal	– Courses through interventricular septum
Interarterial	– Courses between aorta and pulmonary artery – Higher risk of compression—>ischemia

- Increasing role of intravascular US to define anomalous origins.

Risk of Sudden Cardiac Death [4]

- Higher risk in:
  - Anomalous LCA from right sinus.
  - Interarterial course. (There is debate about whether this leads to higher risk)
  - Intramural course (in which the coronary artery courses through the media of the aorta) [7]
  - Slit-like orifice opening.
  - Anomalous LCA from pulmonary artery
  - Atresia of LCA.

Surgical Repair

- Coronary artery unroofing (excising common wall between aorta and anomalous coronary, Fig. 33.4).

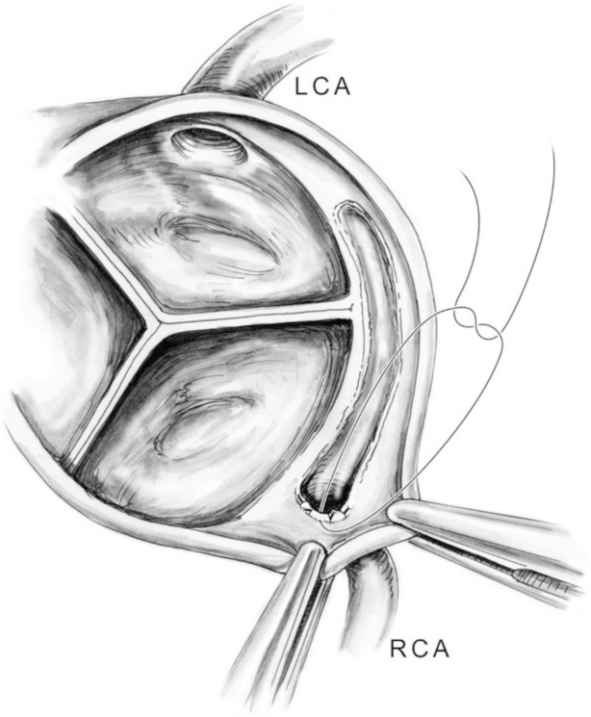


Fig. 33.4 Unroofing technique for repair of anomalous coronary artery from the right pulmonary artery

- Other less commonly used techniques.
  - Osteoplasty (creation of new ostium at the end of the ectopic artery's intramural segment).
  - Direct reimplantation of ectopic artery at aortic root.

Coronary bypass is NOT preferred due to high risk of graft failure due to competitive flow [8].

Vascular Rings

A vascular ring is a malformation of the aorta and its major branch vessels in which the vessels partially or completely encircle the aerodigestive tract. In the normal embryologic development of the aorta, the 6 major aortic (branchial) arches involute or migrate to form the aortic arch and major branch vessels. Abnormalities in this development can lead to aberrancies in laterality and origin of branch vessels, causing the formation of vascular rings.

Anatomy/Pathophysiology [9]

Normal Anatomy/Development (Fig. 33.5)

- 6 aortic (branchial) arches bilaterally.
- Migration and involution result in final anatomy of aortic arch and major branch vessels.

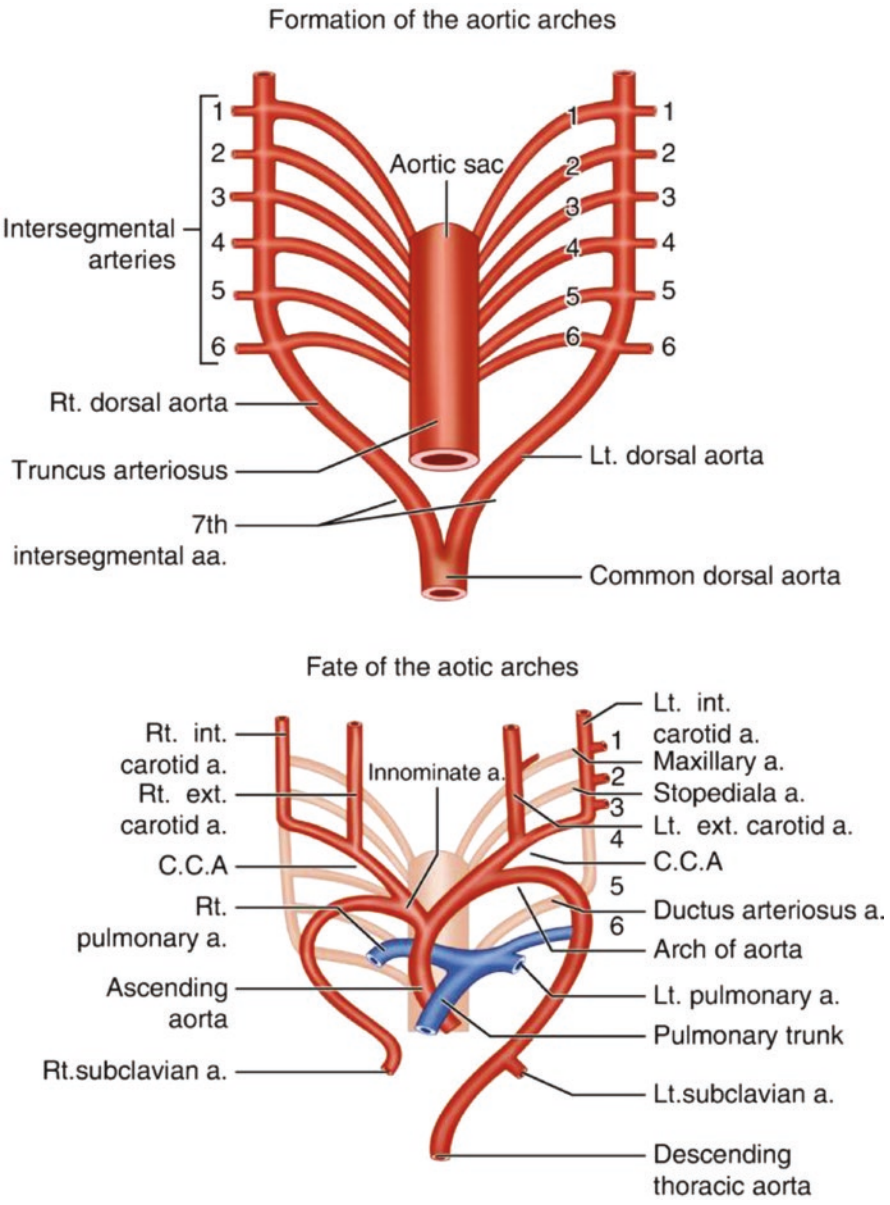


Fig. 33.5 Normal embryological development of the aorta

– Left fourth aortic arch—>normal left-sided adult aorta.
– Right fourth aortic arch—>right subclavian artery.

Embryonic vessel	Outcome
Truncus arteriosus	Proximal ascending aorta Pulmonary root
Aortic sac	Distal ascending aorta Brachiocephalic artery Arch up to origin of left common carotid artery
1st arch	Maxillary artery
2nd arch	Stapedial artery
3rd arch	Common carotid artery Proximal internal carotid artery
4th arch	Right: proximal right subclavian artery Left: aortic arch (segment between left common carotid and left subclavian arteries)
5th arch	Involutes
6th arch	Right proximal: right pulmonary artery Right distal: involutes Left proximal: left pulmonary artery Left distal: ductus arteriosus
Right dorsal aorta	Cranial portion: right subclavian artery Distal portion: involutes
Left dorsal aorta	Aortic arch distal to left subclavian artery

Vascular Ring Anatomy

Type IA: double aortic arch	– Right dorsal aorta fails to involute – Persistence of right and left fourth aortic arches – Descending aorta on the left – Each subclavian and common carotid arises from its respective arch
Type IB: vascular rings	
Right aortic arch + retroesophageal left subclavian artery	– Left fourth arch involutes while right fourth arch persists – Left subclavian artery arises aberrantly behind the esophagus from the right-sided arch – Left-sided ligamentum arteriosum joins left pulmonary artery to complete ring
Right aortic arch + mirror image branching	– Left fourth arch involutes while right fourth arch persists – Left innominate arises anteriorly – Ligamentum arteriosum arising from aorta (behind esophagus) to left pulmonary artery—>forms ring
Left aortic arch + aberrant right subclavian artery	– Right subclavian artery originates distal to left subclavian artery with retroesophageal course

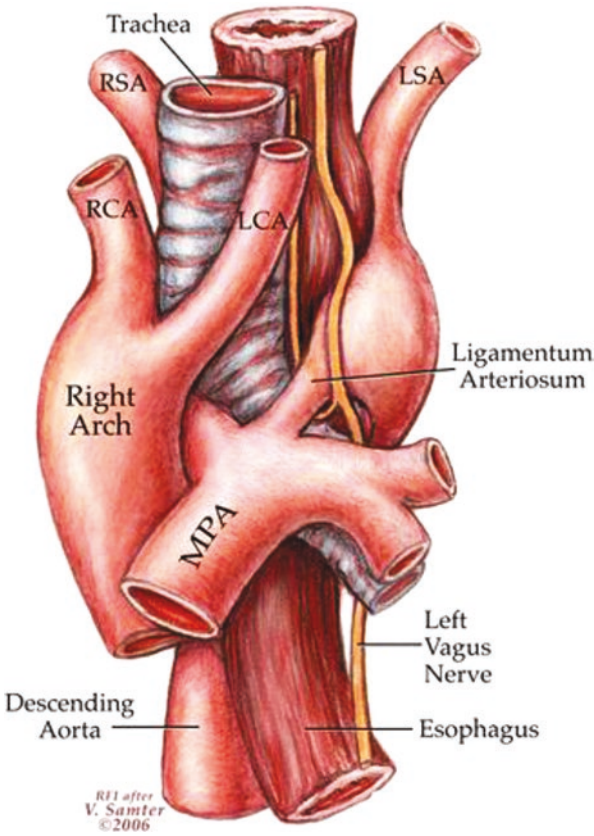


Fig. 33.6 Compression of aerodigestive tract from vascular ring

Presentation

- Compression of trachea and esophagus (Fig. 33.6).
  - Dysphagia lusoria.
  - Stridor.
  - Recurrent respiratory infections.

Kommerell's Diverticulum

- Arises from a remnant fourth dorsal aortic arch.
- Can occur in both left and right aortic arch anatomy.
  - Aberrant subclavian artery rises to the contralateral side.
- Consider surgery when symptomatic or diverticulum orifice >3 cm or descending aorta >5 cm.
- Options for repair.

- Open surgical repair.
- Hybrid endovascular repair.
- Total endovascular repair.

Adult Coarctation of the Aorta (CoA)

While most patients with CoA are diagnosed in childhood, about 10% of patients will present in adulthood [10]. Unlike pediatric coarctation in which surgery is the treatment of choice, adult coarctation is often managed with transcatheter approaches.

Presentation

- Symptoms:
  - Hypertension (most common).
  - Leg claudication.
  - Mesenteric angina.
  - Headache.
  - Tinnitus.
  - Epistaxis.
- Exam:
  - Delayed or weak femoral pulses.
  - Continuous systolic–diastolic murmur between scapulae (blood flow through collateral vessels).

Surgical Repair [11]

- Indications for intervention:
  - BP difference >20 mmHg between upper and lower limbs (regardless of symptoms) with:

    Upper limb hypertension (>140/90 mmHg).
    Pathologic BP response during exercise.
    Significant LV hypertrophy.
  - Hypertension with ≥50% aortic narrowing relative to aortic diameter at the level of the diaphragm.
- Surgery versus catheter-based interventions (Fig. 33.7):

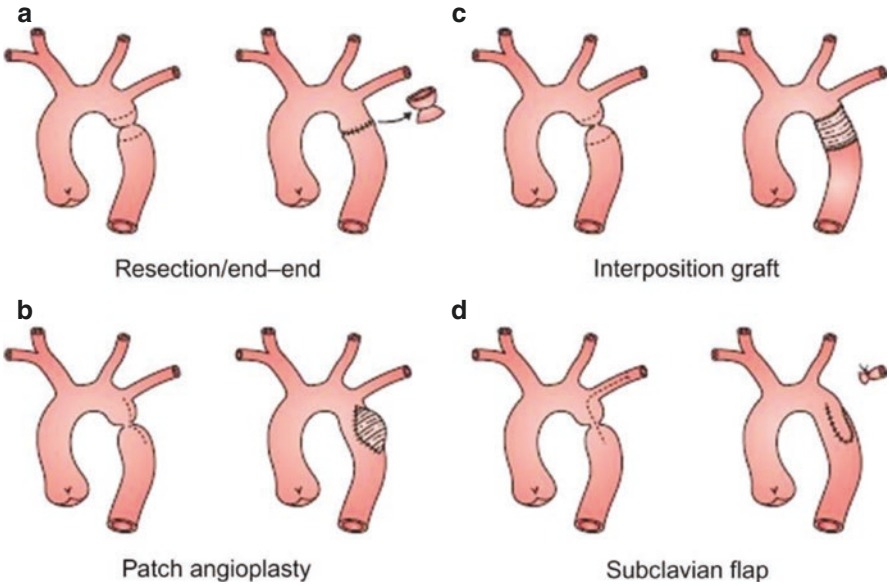


Fig. 33.7 Surgical interventions for aortic coarctation: (**a**) resection with end-to-end anastomosis, (**b**) patch angioplasty enlargement, (**c**) resection with interposition graft, and (**d**) subclavian flap enlargement

	Key points	Types of repair
Surgery	Preferred in pediatric patients – In adults, increased mortality risk due to degenerate changes in aortic wall	Resection with end-to-end anastomosis – High rates of re-coarctation due to narrowing of the suture line Patch aortoplasty – PTFE patch is sewed at the level of the coarctation to expand the diameter of the aorta – Aneurysms form in 20–40% of cases opposite to patch – Only used in complex arch reconstruction Subclavian flap aortoplasty – Subclavian artery is sacrificed and the orifice enlarged to create a flap using intrinsic tissue – Avoids the use of patch or graft – Low re-coarctation rate (3%) Interposition graft – Graft will not grow with the patient – Preferred in adult-sized patients
Catheter-based	Preferred in adults – Treatment of both native coarctation and re-coarctation or aneurysm after initial repair	Balloon angioplasty – High incidence of re-stenosis when used alone Stenting – Sustained hemodynamic benefit – Reducing risk of dissection and aneurysm by tacking intimal flaps to aortic wall ***Balloon angioplasty + stenting is preferred treatment in adults**

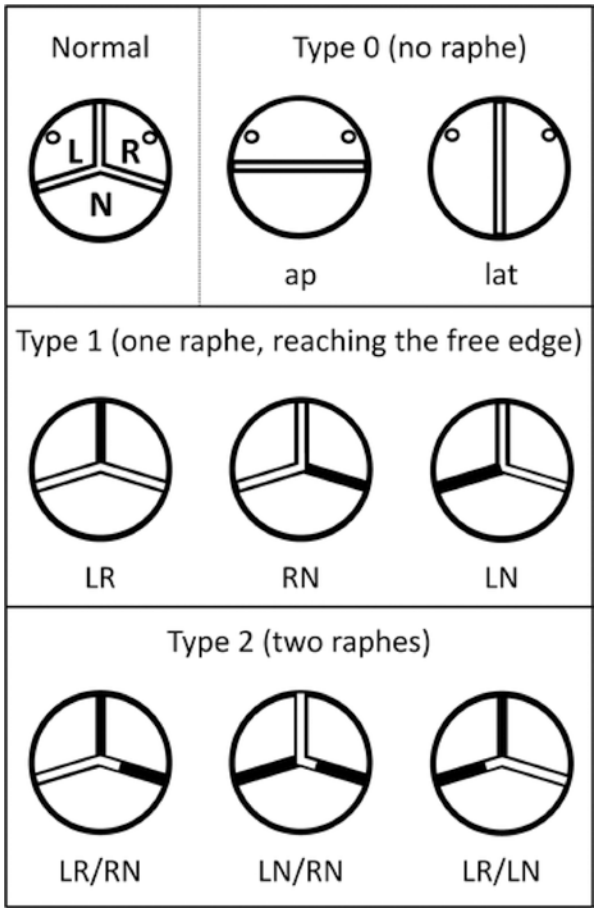


Fig. 33.8 Morphologic classification of bicuspid aortic valves. Type 0: no raphe, type 1: one raphe, type 2: two raphes

Bicuspid Aortic Valve

Bicuspid aortic valves are one of the most common congenital abnormalities in adults and are found in 1–2% of the population. The management of patients with bicuspid aortic valves do not significantly differ from the general public, although may require more frequent monitoring for valve degeneration or aortic dilatation.

Anatomy

- Bicuspid aortic valves arise when adjacent leaflets fail to separate—>only 2 cusps rather than the usual 3 (right, left, and noncoronary).
- Morphology varies according to which commissures are fused (Fig. 33.8).
- Associated with coronary artery anomalies in 2% of patients.

Presentation

- Increased risk of valvular degeneration.
  - Aortic stenosis.

    Harsh systolic murmur at left upper sternal border.
    Fatigue, dyspnea, syncope.
  - Aortic regurgitation.

    Diastolic murmur.
    Pulmonary congestion and right heart overload.
- Increased risk of aortic aneurysms.
  - Theories for the etiology of this risk include intrinsic tissue weakness versus abnormal flow dynamics leading to skewed stress on the aortic wall.

Surgery

- Options:
  - Aortic valve replacement.

    Considered in severe aortic stenosis or damaged valves.
    Mechanical or bioprosthetic options depending on patient age, likelihood of medication compliance and bleeding risk (as patients with mechanical valves will require lifelong anticoagulation).
  - Aortic valve repair.

    Can be considered when leaflets themselves are salvageable (i.e., not thickened or sclerotic).
    Involves resuspension of the commissures, oversewing aortic clefts, or plication of floppy leaflets (Fig. 33.9).
- Aortic root or ascending aorta replacement if diameter >5.5 cm.
  - Previously, bicuspid aortic valves were considered higher risk for aortic dissection in patients with aortic aneurysms, and the threshold for intervention was lower (>4.5–5 cm). Current guidelines recommend standard thresholds for aortic root/ascending replacement.

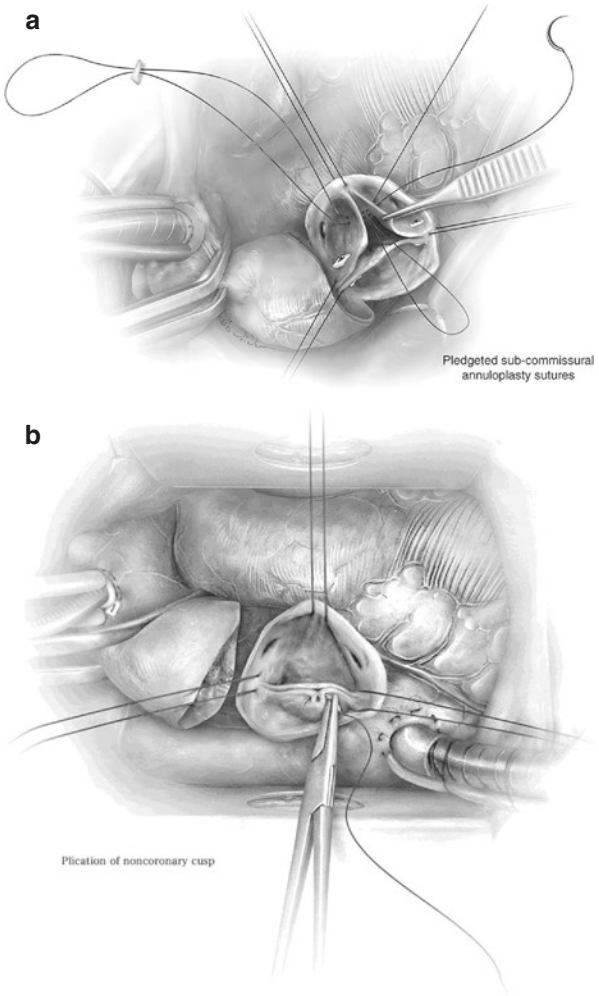


Fig. 33.9 Aortic valve repair with resuspension of commissures (**a**) and plication of cusps (**b**)

Cor Triatriatum Dexter [12]

Cor triatriatum is an abnormal septation within the atrium that leads to inflow obstruction to the ventricle. While cor triatriatum can occur on either side, obstruction on the left leads to a cor triatriatum sinister (CTS) which, as its name suggests, is a more severe variant presenting in childhood. Cor triatriatum dexter (CTD) has a milder presentation and may remain undetected until adulthood.

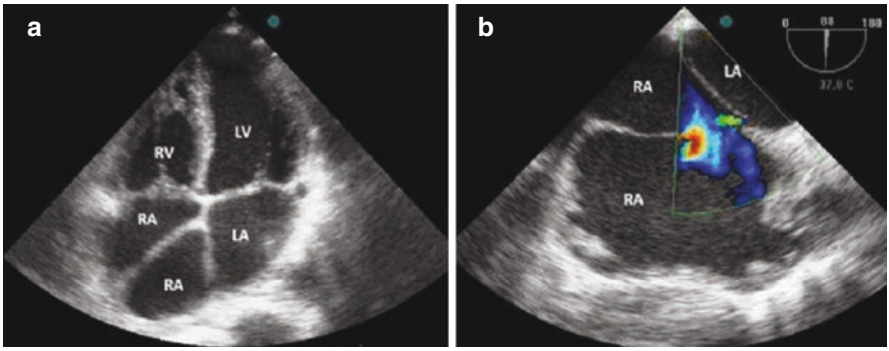


Fig. 33.10 (**a**) Transesophageal echocardiography demonstrating cor triatriatum dexter (CTD) (**b**) with Doppler demonstrating area of flow through a foramen in the membrane

Anatomy

- Abnormal septation in the atrium (can be complete, incomplete, or fenestrated) leads to obstruction of blood flow passing from the atrium to the ventricle (Fig. 33.10).
- Membrane exists between the smooth and trabeculated parts of the atrium.
  - Arises due to persistence of the right valve of the right horn of the sinus venosus.
- Most common location of the membrane:
  - To the right of the SVC, IVC, and coronary sinus.
  - To the left of the coronary sinus and right of SVC and IVC.

Presentation

Cor triatriatum is a rare congenital heart disease, and CTD less common than CTS. Presentation and symptomatology depend on location of the membrane, degree of septation, concomitant ASD, and associated cardiac lesions.

- Common presentations:
  - Systemic venous congestion (symptoms mimic tricuspid stenosis).

    Elevated JVP
    Hepatic congestion
    Peripheral edema
    Clubbing

- Increased risk of thrombosis due to proximal chamber dilation

  Paradoxical embolism in patients with concomitant ASD

  - Can present with myocardial infarction due to paradoxical embolism to coronary arteries [13]

Surgical Repair

- Perioperative management:
  - Increased risk of desaturation and hypoxemia due to reduced pulmonary blood flow
  - Avoid tachycardia and maintain sinus rhythm to ensure diastolic filling of RV
  - CVP will be persistently high due to inflow obstruction

    Volume administration should be guided by transesophageal echocardiography.
- Principles of surgical repair:
  - Excision of interatrial membrane.
  - Correction of associated anomalies.

Complex Adult Congenital Heart Defects

Single Ventricle [14]

- Single ventricle physiology refers to a group of congenital defects that result in one functioning ventricle.

Type	Definition
Hypoplastic left heart syndrome (HLHS)	• Underdeveloped left ventricle with atretic aortic and mitral valves • Aorta atresia results in decreased coronary blood flow • Interatrial septum is usually thickened, and PFO is necessary for survival
Tricuspid atresia	• Absence or agenesis of tricuspid valve—>dilated RA and underdeveloped RV • RV is unable to support pulmonary circulation • ASD or PFO is necessary for survival

Type	Definition
Double-inlet left ventricle	• Main ventricle is a morphologic LV with an outlet chamber of RV morphology • Great arteries are frequently transposed – Aorta arises from hypoplastic RV – PA arises from LV
Complete atrioventricular septal defect (AVSD)	• Defect in both the atrial and ventricular septae lead to a large inlet VSD contiguous with an ASD • Can be balanced or unbalanced – Unbalanced can be LV or RV dominant (the latter is more common)
Others	• Mitral atresia • Pulmonary atresia with intact ventricular septum • Ebstein's anomaly of the tricuspid valve

Single Ventricle Physiology

- Systemic and pulmonary blood flow mix in the single functioning ventricle which provides both systemic and pulmonary circulation.
  - Systemic and pulmonary circuits are in parallel instead of in series.
- Mixed blood provides lower systemic oxygen saturation (between 75 and 85%).

Adults with Single Ventricle CHD

- Most patients with single ventricle defects who survive into adulthood have undergone some form of surgical palliation.
  - Ex. Fontan repair.
- Progressive heart failure and effects on multiple organs.
  - Cardiac arrhythmias.
  - Pulmonary vascular remodeling.
  - Hepatic congestion.
  - Renal hypoperfusion.
  - Chronic venous stasis.

Fontan Repair

- Fontan repair in childhood is used for single ventricle anatomy.
  - Creates circulation in series (right and left circulation) without 2 pumping chambers (Fig. 33.11).

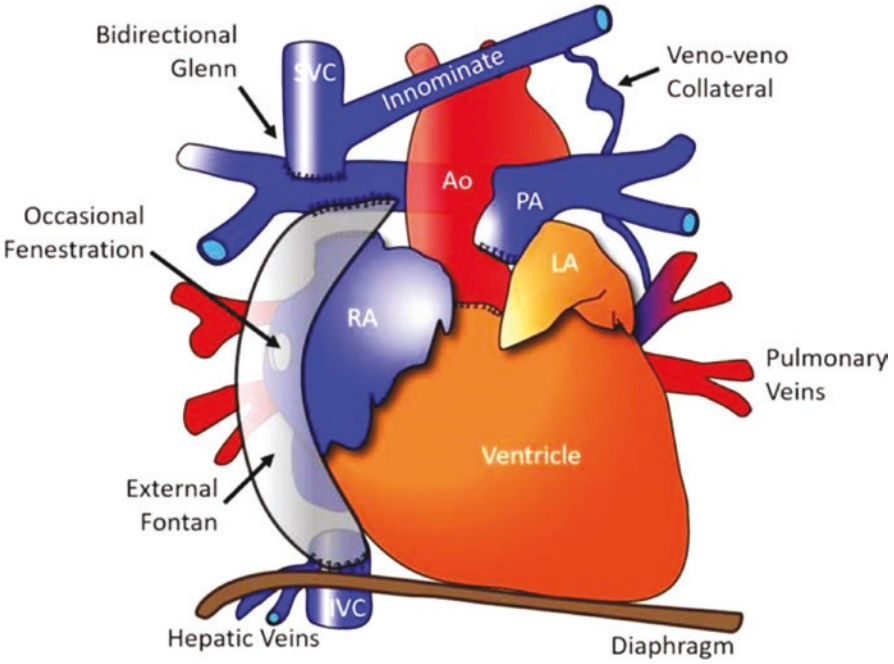


Fig. 33.11 Fontan repair of tetralogy of Fallot

- Classic Fontan = conduit between RA and PA.

  Directs vena caval flow to pulmonary arteries.

- Modifications include fenestration in the Fontan baffle (between Fontan and atrium), allowing right-to-left shunting.

- Leads to chronic state of low cardiac output.
  - Passive flow of caval blood to pulmonary arteries.
  - Inability to augment cardiac output during exercise.
- Fontan physiology may lead to:
  - Elevated CVP.
  - Elevated pulmonary vascular resistance.
  - Systolic and diastolic dysfunction.
  - Cyanosis.
  - Atrial arrhythmias.
- "Failing Fontan" patients are at risk for protein-losing enteropathy, plastic bronchitis, and liver disease, along with various forms of cardiac failure
- Surgical intervention depends on symptomatology [15]:
  - Fontan conversion to extracardiac Fontan.
  - Cardiac pacing.

- Fontan fenestration.
- Thoracic duct ligation.
- Rerouting innominate vein to left atrium.
- Heart or heart-liver transplant.

Transplant and Mechanical Circulatory Support

- Heart failure is the leading cause of morbidity and mortality in ACHD patients [16].
  - Approximately 50% of Fontan patients die or need a heart transplant by age 40 [17].
- Drug therapy (ACE-I, ARBs, SGLT-2 inhibitors, neprilysin inhibitors) have proven effects on LV failure but are poorly demonstrated in Fontan patients and RV failure.
- Unlike acquired heart disease which relies heavily on pharmacologic treatments for symptoms and survival in heart failure, ACHD patients with heart failure require early structural support.
  - Early identification of progressive heart failure and referral for transplantation is key to improving survival.
  - Mechanical circulatory support (MCS) is less commonly used as a bridge to transplant in ACHD patients given their prior surgeries and altered anatomy.

References

1. Brida M, Gatzoulis MA. Adult congenital heart disease: past, present and future. Acta Paediatr. 2019;108(10):1757–64. https://doi.org/10.1111/apa.14921.
2. Angelini A, di Gioia C, Doran H, Fedrigo M, Henriques de Gouveia R, Ho SY, et al. Autopsy in adults with congenital heart disease (ACHD). Virchows Arch. 2020;476(6):797–820. https://doi.org/10.1007/s00428-020-02779-8.
3. Pendela VS, Tan BE, Chowdhury M, Chow M. Partial anomalous pulmonary venous return presenting in adults: a case series with review of literature. Cureus. 2020;12(6):e8388. https://doi.org/10.7759/cureus.8388.
4. Kastellanos S, Aznaouridis K, Vlachopoulos C, Tsiamis E, Oikonomou E, Tousoulis D. Overview of coronary artery variants, aberrations and anomalies. World J Cardiol. 2018;10(10):127–40. https://doi.org/10.4330/wjc.v10.i10.127.
5. Ansari MM, Cardoso R, Garcia D, Sandhu S, Horlick E, Brinster D, et al. Percutaneous pulmonary valve implantation: present status and evolving future. J Am Coll Cardiol. 2015;66(20):2246–55. https://doi.org/10.1016/j.jacc.2015.09.055.
6. Blissett S, Lin S, Mahadevan V, Ordovas K. Adult presentation of congenital heart disease. Semin Roentgenol. 2020;55(3):251–63. https://doi.org/10.1053/j.ro.2020.06.008.

7. Zimmerman SL. Intramural versus septal course for anomalous interarterial coronary arteries. In: Earls and pitfalls in cardiovascular imaging: pseudolesions, artifacts and other difficult diagnoses. Cambridge: Cambridge University Press; 2015. p. 113–6.
8. Krasuski RA, Magyar D, Hart S, Kalahasti V, Lorber R, Hobbs R, et al. Long-term outcome and impact of surgery on adults with coronary arteries originating from the opposite coronary cusp. Circulation. 2011;123(2):154–62. https://doi.org/10.1161/CIRCULATIONAHA.109.921106.
9. Mascio CE, Austin EH. Vascular rings, slings, and other arch anomalies. 2016. https://thoracickey.com/vascular-rings-slings-and-other-arch-anomalies/. Accessed 7 Apr 2022.
10. Liberthson RR, Pennington DG, Jacobs ML, Daggett WM. Coarctation of the aorta: review of 234 patients and clarification of management problems. Am J Cardiol. 1979;43(4):835–40. https://doi.org/10.1016/0002-9149(79)90086-9.
11. Alkashkari W, Albugami S, Hijazi ZM. Management of coarctation of the aorta in adult patients: state of the art. Korean Circ J. 2019;49(4):298–313. https://doi.org/10.4070/kcj.2018.0433.
12. Jha AK, Makhija N. Cor triatriatum: a review. Semin Cardiothorac Vasc Anesth. 2017;21(2):178–85. https://doi.org/10.1177/1089253216680495.
13. Hussain ST, Mawulawde K, Stewart RD, Pettersson GB. Cor triatriatum dexter: a rare cause of myocardial infarction and pulmonary embolism in a young adult. J Thorac Cardiovasc Surg. 2015;149(3):e48–50. https://doi.org/10.1016/j.jtcvs.2014.11.078.
14. Rao PS. Single ventricle—a comprehensive review. Children (Basel). 2021;8(6):441. https://doi.org/10.3390/children8060441.
15. Geoffrion T, Fuller S. Surgery for adult congenital heart disease. Cardiol Clin. 2020;38(3):435–43. https://doi.org/10.1016/j.ccl.2020.04.013.
16. Menachem JN, Schlendorf KH, Mazurek JA, Bichell DP, Brinkley DM, Frischhertz BP, et al. Advanced heart failure in adults with congenital heart disease. JACC Heart Fail. 2020;8(2):87–99. https://doi.org/10.1016/j.jchf.2019.08.012.
17. Dipchand AI, Honjo O, Alonso-Gonzalez R, McDonald M, Roche SL. Heart transplant indications, considerations and outcomes in Fontan patients: age-related nuances, transplant listing and disease-specific indications. Can J Cardiol. 2022;38(7):1072–85. https://doi.org/10.1016/j.cjca.2022.02.019.

Chapter 34
Teamwork in the Cardiac Surgical Operating Room

Sameer Hirji and Marco Zenati

Roles/Responsibilities of Cardiac OR Teams

Cardiac Surgery

The cardiac surgery team often includes 1 or 2 primary surgeons (also known as the "attending surgeon(s)") who are assisted by either 1 or 2 operators (known as the "first assistant" or "second assistant"). The primary surgeon is viewed as the "captain" of the team who is board certified in the field of cardiothoracic surgery. His/her main responsibilities include:

- To perform the highly specialized procedure based on his/her technical skills and experience (e.g., perform sternotomy, perform valve repair or replacement, perform the coronary bypass, etc.)
- To communicate effectively with the other OR teams during various aspects of the procedure as needed (e.g., obtaining surgical instruments, inquiring about patient anesthesia, understanding hemodynamics during cardiopulmonary bypass, requesting for surgical implants, etc.)
- To hold each member of the OR team accountable for their roles to ensure patient safety is upheld at all times (e.g., working with perfusion throughout cardiopulmonary bypass).
- To help facilitate patient transitions of care from the OR to the intensive care unit once the procedure is concluded.

S. Hirji (✉)
Division of Cardiac Surgery, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA
e-mail: shirji@mgb.org

M. Zenati
Veterans Affairs Boston Healthcare System, West Roxbury, MA, USA

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_34

The first or second assistant can be a surgical fellow/resident in training (if at an academic center), or a specialized physician assistant who is trained to assist with various aspects of the procedure (e.g., harvesting of vein conduits for bypass, assisting with groin access for cardiopulmonary bypass). These individuals work closely with the primary surgeon and are familiar with the needs and preferences of the primary surgeon to ensure smooth conduct of the procedure.

Anesthesia

The anesthesia team often includes 1 or 2 primary anesthesiologists (also known as the "attending surgeon(s)") who are assisted by either a resident/fellow in training or nurse anesthetist. Their combined responsibilities include:

- To safely provide adequate anesthesia for the patient during the perioperative period.
- To perform key adjunct procedures (e.g., transesophageal echocardiography) that provide clinical insight and enhance clinical decision-making.
- To work closely with the cardiac surgeon and perfusion team during periods of high cognitive load (e.g., initiation of cardiopulmonary bypass, weaning off bypass, providing systemic heparinization and reversal, etc.)
- To supervise the bypass heart-lung machine to ensure adequate systemic ventilation.
- To assist with the delivery of drugs and medications that prevent arrythmia's or systemic hypotension.
- To assist with the transport of the cardiac surgical patient safely to the cardiac intensive care unit for post procedure recovery.

Perfusion

This team often includes 1 or 2 individuals whose primary responsibility is as follows

- To select patient-specific equipment that will support the cardiopulmonary needs of the patient.
- To operate the heart-lung machine during cardiac surgery that allows regulating blood flow and blood temperature during surgery.
- To coordinate with the cardiac surgery and anesthesia teams regarding the appropriate timing of initiation and weaning off cardiopulmonary bypass.
- To manage metabolic demands of the patient during surgery by analyzing the blood chemistry and making adjustments as needed.
- To deliver drugs (or "cardioplegia") used to arrest the heart to allow the cardiac surgeon to perform the procedure. This allows the surgeon to manage the physi-

ologic demands of the patient under the direction of the surgeon or anesthesiologist.

Nursing

The nursing team remains an essential component of the cardiac OR with several key roles

- To ensure the OR is set up appropriately for the cardiac procedure.
- To coordinate and obtain all surgical supplies (instruments, implants, imaging platforms) that will ensure the smooth conduct of the procedure.
- To assist in patient positioning and transfer to and from the OR table.

Scrub Person

The scrub person plays a huge role in ensuring the smooth conduct of the procedure. Their responsibility include:

- To ensure all surgical instruments are sterile and available for the cardiac surgery team based on a-priori specified surgeon preferences.
- To anticipate the needs of the surgeon during the procedure and provide the correct instruments in the timely fashion.
- Assist in patient positioning before and after the procedure.
- To ensure sterile procedures are followed at all times during the procedure.

Variations in Cognitive Load During Cardiac Surgery

In recent years, the role of human cognition in contributing to errors in complex environments is increasingly recognized across the various teams in the cardiac OR, which is a high-impact complex and dynamic healthcare environment where the majority of human errors leading to preventable patient harm can potentially occur [1]. Cognitive workload, or the level of measurable mental effort put forth by an individual in response to a mental task, varies substantially throughout a particular procedure and can impact the different teams differentially [2]. One recent study, for instance, investigated individual measures of cognitive load over time during cardiopulmonary bypass for various stakeholders (surgeon, anesthesiologist, and perfusionist) [2]. The study found that perceived cognitive load varied throughout the procedure. Furthermore, while on bypass, the anesthesiologists experienced significantly lower levels of perceived cognitive load than both surgeons and perfusionists. Correlational analyses also reveal that perceived cognitive load of both the surgeon

and the team had significant positive associations with bypass length and surgery length. Another novel preliminary study attempted to identify and capture dynamic changes in heart rate variability as a proxy for cognitive workload among perfusionists while operating the cardiopulmonary bypass pump during real-life cardiac surgery [3]. Cognitive workload was at its highest during the time between initiating bypass and clamping the aorta (preclamping phase during bypass), and decreased over the course of the bypass period [3].

The nature, timing, and extent of high cognitive load can also vary substantially across the different cardiac surgical procedures. Thus, the different teams have to mentally train and adopt to the various perioperative circumstances to ensure that patient safety is upheld and individual/team performance is not compromised during periods of cognitive overload. Likewise, the adoption of non-technical skills via simulation-based training to enhance surgical team performance is essential to improve patient safety in OR.

Impact of Preoperative Briefing ("Huddle")

Preoperative briefing or "huddle" remains an essential and proven framework that is designed to promote situation awareness, teamwork, and error prevention especially given the high acuity nature of cardiac surgery [4]. Implementation of surgical safety checklists during preoperative briefing also improves perceptions of surgical safety and facilitates a shared adoption of mental models to provide a safe environment to raise/highlight mutual awareness of any patient safety-related concerns [5]. The shared model also enables an individual to develop a higher level abstraction about the expertise and responsibilities of other team members prior to the surgical incision. This is particularly relevant in the context of high turnover of staff in the OR (e.g., different trainees, new nurses). Overall, this process enhances team closed-loop communication that is essential throughout the surgical procedure.

Promoting a Culture of Safety in the OR

Given the integrated nature of collaboration between the various teams in the cardiac OR, the importance of developing and maintaining a "culture of safety" cannot be over-emphasized given the strong correlation between patient safety climate and patient safety [4]. This concept is common to the commercial aviation industry which have consistently demonstrated the virtue of open communication, and root-cause analyses in a blame-free environment to prevent catastrophic failures [4, 6]. Each team member, rather than the surgeon alone, is expected to play a pivotal role in advocating for patient safety and reducing errors as the stakes are high even though the leadership style of the attending surgeon has a significant impact on the function of the entire OR. Ultimately, although variable, the behavior of the surgical team has a substantial impact on patient outcomes in the context of surgical safety.

References

1. Kennedy-Metz LR, Barbeito A, Dias RD, Zenati MA. Importance of high-performing teams in the cardiovascular intensive care unit. J Thorac Cardiovasc Surg. 2022;163(3):1096–104.
2. Kennedy-Metz LR, Wolfe HL, Dias RD, Yule SJ, Zenati MA. Surgery task load index in cardiac surgery: measuring cognitive load among teams. Surg Innov. 2020;27(6):602–7.
3. Kennedy-Metz LR, Dias RD, Srey R, Rance GC, Conboy HM, Haime ME, et al. Analysis of dynamic changes in cognitive workload during cardiac surgery perfusionists' interactions with the cardiopulmonary bypass pump. Hum Factors. 2021;63(5):757–71.
4. Wilson JL, Whyte RI, Gangadharan SP, Kent MS. Teamwork and communication skills in cardiothoracic surgery. Ann Thorac Surg. 2017;103(4):1049–54.
5. Kennedy-Metz LR, Dias RD, Zenati MA. The cognitive relevance of a formal pre-incision time-out in surgery. ECCE. 2021;2021:2867.
6. Wiegmann DA, Zhang H, von Thaden TL, Sharma G, Gibbons AM. Safety culture: an integrative review. Int J Aviat Psychol. 2009;14:117–34.

Chapter 35
Principles of Postoperative Care

Lynze Franko and Kenneth Shelton

Assessment and Goals for the Immediate Postoperative Period [1–4]

- The first step when the ICU receives a patient from the operating room (OR) is connecting the patient to monitoring and the ICU ventilator to ensure safety. Next, the handoff occurs. This is a critical moment in time when all members of the team should be present, focused, and alert. This includes anesthesia, ICU providers, nurses, and a cardiac surgical team member. Checklists are often utilized, which have been shown to reduce errors and gaps in communication.
- Important points for the cardiac surgery team to convey are pertinent patient history, preoperative cardiac disease, operative findings, surgical interventions, operative complications, bypass configuration, location and number of tubes/drains, placement of pacing wires, underlying cardiac rhythm, need for defibrillation/cardioversion, and desired postoperative care. The surgical team should also discuss expected postoperative course, possible complications to watch for, and desired interventions, including antibiotic needs, blood pressure goals, and anticoagulation plan.
- The anesthesia team conveys information about pre/postop echocardiogram, airway concerns, lines, clamp time, CPB time, circulatory arrest, any difficulty coming off CPB, transfusions, fluids, urine output, medications (inotropes, vasopressors, antibiotics, analgesics, paralytics), and other pertinent details.

L. Franko (✉)
Department of Surgery, Massachusetts General Hospital, Boston, MA, USA
e-mail: lfranko@mgb.org

K. Shelton
Heart Center Intensive Care Unit, Department of Anesthesia, Critical Care, and Pain Medicine, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA
e-mail: kshelton@mgh.harvard.edu

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8_35

- Critical first assessments after handoff include:
  - Vital signs: It is important to assess for hypothermia upon arrival to the ICU. Blood pressure is closely monitored. The ventilator settings can be changed to obtain appropriate oxygen saturations immediately upon arrival. Additionally, telemetry should be applied, and a 12-lead EKG obtained.
  - Chest X-ray: Evaluate for chest tube position, hemothorax, pneumothorax, and endotracheal tube position.
  - Laboratory values: A full set of labs should be sent upon arrival to the ICU. This should include a CBC, BMP, LFTs, ionized calcium, magnesium, phosphorus, INR, aPTT, fibrinogen, lactate, arterial blood gas, and mixed venous oxygen saturation.
  - Tubes and lines assessment: All tubes and lines should be assessed upon arrival. Central and peripheral intravenous line access should be assessed for patency. Additionally, chest tubes should be assessed for output and drainage. Immediate interventions should be employed if a chest tube appears to be obstructed by clot. Without drainage, blood can collect around the heart or lungs leading to tamponade or hemothorax.
- In cardiac surgical ICU patients, the focus is often on optimizing the patient's cardiac output in the postoperative period in order to supply sufficient end organ perfusion.
  - Cardiac Output = Stroke Volume × Heart Rate.

    Stroke volume is a function of preload, afterload, and contractility. Interventions focused on modifying these variables can improve cardiac output and end organ perfusion.

  - Preload of the right side is often measured by central venous pressure. Left-sided preload can be assessed with pulmonary capillary wedge pressures. More accurate assessments of preload utilize respiratory arterial pulse pressure variation and stroke volume variation as well. Increasing preload often focuses on giving the patient volume in the form of fluid or transfusions.
  - Afterload is "the workload imposed by any factor that resists ejection of blood from the ventricle" [4]. Afterload is influenced by vascular resistance and wall stress. For example, vasoconstriction often increases afterload, and vasodilation decreases afterload. Afterload is important to maintain blood pressure, though can also be detrimental in decreasing cardiac output and increasing myocardial demand.
  - Contractility is based on the hearts ability to eject blood. This can be modified with inotropic medications.

Hemodynamic Evaluation [2, 3, 5–16]

- Standard hemodynamic monitoring in the ICU is based on blood pressure. For cardiac surgery patients, this is often through arterial line monitoring and intermittent cuff pressures. However, blood pressure alone is insufficient as a marker of perfusion.
  - The first step when a significant blood pressure change occurs is to check the arterial wave form to ensure it is not dampened (false hypotension) and does not have an exaggerated wave form/whip (false hypertension). A cuff blood pressure should be done to make sure there is not an error in arterial line monitoring.
  - Avoiding significant hypertension is important, particularly in cases with an aortic anastomosis, to prevent bleeding and excessive stress on a new suture line. Hypotension is important to avoid as it can lead to reduced cardiac and end organ perfusion. Blood pressure goals after specific surgeries are often quite surgeon dependent. Commonly, blood pressure goals include mean arterial pressures (MAP) greater than 60–65 or a systolic blood pressure between 100 and 140 mmHg.
  - Baseline blood pressure of the patient and presence of kidney disease is important to understand when setting blood pressure and MAP goals for patients. Chronically hypertensive patients or those with kidney disease may require higher blood pressures for sufficient end organ perfusion.
- Pulmonary Artery Catheters (PACs): PACs provide information regarding right and left heart function. There is controversy regarding utilization of PAC given few studies showing improvement in patient outcomes utilizing this device. Because of this, PACs are not often utilized for non-cardiac surgery patients who are critically ill. Further, PACs are no longer utilized for all cardiac surgeries. However, PACs are utilized in cardiac surgery patients with high risk of cardiac dysfunction, including those with preoperative reduced EF, pulmonary HTN, or right ventricular failure. Additionally, a Class 1A recommendation for heart transplant patients is intermittent right heart catheterization to evaluate pulmonary artery pressures, which can be assessed with a PAC.
  - The PAC is placed through a sheath in the central line. Right-sided internal jugular lines can lead to easier placement of PAC than left. In extreme situations, the PAC can be placed through a femoral sheath though this is significantly more difficult. To place a PAC, the balloon at the catheter tip will be inflated after the PAC is inserted approximately 15–20 cm. The inflated balloon at the tip of the catheter allows it to flow with blood through the right atrium, tricuspid valve, right ventricle, and pulmonic valve into the pulmonary artery without injuring these structures. Location of the balloon is identified based on

transduced wave form and pressures. For example, lower diastolic pressures are seen in the right ventricle compared to the pulmonary artery. Placement is confirmed by monitoring transduced wave forms. It is important never to withdraw a PAC with the balloon inflated as this can lead to injury, so the balloon is always deflated before withdrawing during placement or manipulation.

- Once the PAC is in the many pulmonary artery, the catheter is advanced until it "wedges" in a branch of either the right (more common) or left pulmonary artery. Wedging is identified by changing from an arterial wave form to a venous wave form (reduction in systolic pressure and loss of a dicrotic notch). It is often around a distance of 50 cm when placed. The catheter should have a wedged wave form when the balloon is inflated and pulmonary artery waveform when deflated. If the PAC has a wedged wave form when the balloon is deflated, it needs to be slightly withdrawn (with the balloon deflated). Having the balloon inflated for a significant amount of time or too deep can lead to ischemia and complications. PAC placement can be monitored by transducing the PAC and X-ray. On X-ray, the catheter should not be 1–2 cm past the mediastinum.
- Complications related to PAC include valve injury, subclavian vein thrombosis, arrhythmias, pulmonary infarct, and pulmonary artery rupture. Pulmonary artery rupture is a life-threatening complication. This should be suspected in patients with a PAC who have hemoptysis. This can be caused by distal migration of the catheter with subsequent balloon over inflation or perforation of the artery by the tip of the catheter. If significant, this can require interventional radiology embolization or surgical resection. The initial step in treatment is often inflating the balloon to tamponade the bleeding.
- Pulmonary capillary wedge pressure (PCWP, normal = 4–12 mmHg) measures the filling pressure of the left heart, which is equivalent to left atrial and therefore left ventricle end diastolic pressure in patients without mitral stenosis. This is particularly helpful in a patient with rising CVP and filling pressures to help identify if the source of increased pressure is the right heart, lungs, or left heart.

- Cardiac Output (CO, normal = 4–8 L/min)/Cardiac Index (CI, normal = 2.2–4.0 L/min/m^2).
  - Cardiac Index = Cardiac Output/Body Surface Area.
  - CO/CI are most often calculated utilizing data from PAC. Information regarding CI can be helpful in monitoring postoperative patients and managing inotrope support. While there are newer techniques to monitor CI continuously, most often CI is measured at specific intervals in the initial postoperative period and with changes in the patient's condition.
  - Thermodilution is one way to measure cardiac output with a PAC. Most often, a small volume of room temperature or cool fluid is injected into the catheter at a proximal port (often the RA port). It then measures resulting temperature change in the pulmonary artery with a sensor near the end of the catheter. It utilizes a temperature curve to calculate the cardiac output. This method can be inaccurate in patients with tricuspid regurgitation with the CO being falsely underestimated.
  - Fick's principle is another way to calculate cardiac output. This also requires a PAC. However, in this instance, the PAC is used to obtain a mixed venous

oxygen saturation (SvO_2). In addition, arterial oxygenation saturation (SaO_2) is collected from arterial blood gas samples. A calculation utilizing this information is then performed based on estimated oxygen consumption over the arteriovenous oxygen difference.

- Systemic vascular resistance (SVR): SVR is a calculated measurement based on CO and MAP to describe that the amount of systemic (non-pulmonary) resistance blood flow is experiencing. An increase in SVR can be equated to be an increase in afterload and myocardial demand. Given it is a calculated number, SVR can often be misleading; however, it can be beneficial to trend.
- Central venous pressure (CVP, normal 0–8 mmHg): CVP is hemodynamic assessment tool used to monitor preload or filling pressures of the heart. Extremes of CVP (high or low) or overall trends can be helpful in determining fluid status and evaluate overall right heart function. Relative trends in CVP are often more helpful than exact values.
- EKG/Telemetry: Telemetry allows for continuous monitoring. A 12-lead EKG is beneficial as it can be used to identify underlying conduction abnormalities and evidence of acute ischemia. A 12-lead EKG is best obtained with the pacer on pause, if hemodynamically possible, to allow for an accurate assessment of the underlying rhythm.
  - Conduction delays can be seen immediately after cardiac surgery, particularly with valve procedures. These abnormalities can often improve with time as the stunned tissue recovers. Evidence of ischemia is particularly important to monitor for after coronary artery bypass grafting.

- Pacing: A patient's cardiac rhythm after surgery can be abnormal due to injury or stunning of the conduction system. Often times, this abnormality is temporary and improves with time. Given this, temporary epicardial pacing wires are often placed during cardiac surgery. Pacing can be beneficial in cases of heart block, bradycardia, tachyarrhythmias, and bradycardia-induced arrhythmias. Atrial-ventricular pacing has the benefit of maintaining the atrial kick, which can contribute up to 25% of the cardiac output. Ventricular pacing is often utilized as a backup mode.
  - Pacer wires should evaluated every shift. This includes assessment of underlying rhythm, current pacer settings, and settings required for atrial and ventricular capture. Caution should be applied when checking underlying rhythms as patients with heart block or bradycardia can develop significant hypotension with even a brief pause in pacing.

- Echocardiogram: In the event of decreased cardiac output or changes in patient condition, an echocardiogram can be particularly useful. An echocardiogram can be either transthoracic (TTE) or transesophageal (TEE). In postoperative patients, TEE is often more useful due to difficulty getting good views with TTE. Evaluation for overall ventricular function, localized wall motion abnormalities, valve dysfunction, or pericardial fluid collection can be helpful in identifying the underlying cause of hemodynamic instability.

- Vasopressors/Inotropes: Vasopressors are medications that cause vasoconstriction, which can assist in increasing blood pressure. However, these medications must be used cautiously as increasing afterload without supporting cardiac function can lead to reduced cardiac output. Inotropes are medications that improve cardiac contractility, which work to raise cardiac output. A list of commonly utilized vasopressors, inotropes, and other common medication drips in the cardiac surgery patient can be seen in Table 35.1.

Table 35.1 Commonly utilized vasopressors, inotropes, and other common medication drips in the cardiac surgery

Medication	Mechanism of action	Effect	Side effects/adverse effects
Vasopressor			
Vasopressin	Vasopressin 1 and 2 receptor agonist	– Vasoconstriction – Increases afterload	– Decreases splanchnic flow – Decreases cardiac output through increased afterload
Phenylephrine	Alpha agonist		– Decreases cardiac output through increased afterload
Angiotensin II	Angiotensin II agonist		
Vasopressor and inotrope			
Norepinephrine	Strong alpha agonist, moderate beta 1 agonist	– Vasoconstriction – Increases afterload – Increases contractility – Increases heart rate	– Tachycardia – Arrhythmia – Increases myocardial oxygen demand
Epinephrine	Strong alpha agonist, strong beta 1 agonist, moderate beta 2 agonist	– Vasoconstriction – Increases afterload – Increases contractility – Increases heart rate – Bronchodilation	
Dopamine	Low dose—dopamine agonist Intermediate dose—dopamine agonist, beta 1 agonist High dose—alpha 1, beta 1 agonist, dopamine agonist	– Low dose—increases renal blood flow – Low dose—systemic vasodilation – Intermediate dose—increases contractility – Intermediate dose—increases heart rate – High dose—vasoconstriction – High dose—increases afterload – High dose—increases contractility – High dose—increases heart rate	– Tachycardia – Arrhythmia – Increases myocardial oxygen demand
Inotrope			

Table 35.1 (continued)

Medication	Mechanism of action	Effect	Side effects/adverse effects
Milrinone	Phosphodiesterase 3 inhibitor	– Increases contractility – Pulmonary and systemic vasodilation – Increases relaxation (Lusitropy)	– Hypotension – Tachycardia – Arrhythmia
Dobutamine	Strong beta 1 agonist, moderate beta 2 agonist, faint alpha 1 agonist	– Increases contractility – Increases heart rate – Pulmonary vasodilation	– Arrhythmia and tachycardia
Isoproterenol	Strong beta 1 and beta 2 agonist	– Increases contractility – Increases heart rate – Pulmonary vasodilation – Bronchodilation	– Increases myocardial oxygen demand – Arrhythmia and tachycardia – Hypotension
Other			
Inhaled nitric oxide	Direct delivery of nitric oxide to pulmonary vascular endothelium	– Selective pulmonary vasodilation – Reduces RV afterload – Minimal systemic vasodilation – Decreases ventilation/perfusion mismatch	– High cost – Production of nitrogen dioxide with direct lung tissue damage – Rebound pulmonary hypertension – Methemoglobinemia
Inhaled epoprostenol	Prostaglandin I_2 (prostacyclin)		– Rebound pulmonary hypertension – Inhibition of platelet aggregation
Nitroglycerin	Converts to nitric oxide in blood stream	– Arterial and venous vasodilation – Decreases preload and afterload	– Headache
Nitroprusside	Releases nitric oxide upon breakdown		– Cyanide toxicity (treated with hydroxocobalamin and sodium thiosulfate) – Methemoglobinemia
Nicardipine	Dihydropyridine calcium channel blocker	– Arterial vasodilation – Decreases afterload	– Rebound hypertension

Laboratory Evaluation [2, 17, 18]

- Lactate levels are utilized in cardiac surgery as a marker of perfusion. A lactate greater than 2, slow clearance, or a rising lactate can be a marker of global hypoperfusion. Of note, some patients on epinephrine have been shown to have elevated lactate levels without hypoperfusion thought to be due to sympathetic beta-2 mediated stimulation. Lactate is less reliable marker in patients with significant liver dysfunction.
- Mixed venous oxygen saturation (SvO_2) describes the amount of oxygen saturation of hemoglobin remaining after all blood has returned to the heart. This is drawn from the distal port of a PAC. This provides a true mixed venous sample from the entire body, including the superior vena cava, inferior vena cava, and coronary veins. This can be used as a marker of perfusion. A level greater than 60% is often associated with good perfusion. The trend is also helpful as a marker of recovery if improving. SvO_2 is utilized to calculate CO via the Fick's principle. Alternatively, central venous oxygen saturation ($ScvO_2$) is drawn from a central line and is only indicative of the venous oxygen present within the superior vena cava. This is a less accurate marker of perfusion.
- Monitoring for anemia, coagulopathy, and thrombocytopenia is important postoperatively. Trending hemoglobin and hematocrit is important to monitor for bleeding and to guide transfusions. Additional frequently monitored labs include PT/INR, PTT, fibrinogen, and platelets. In the event of bleeding, transfusions can be targeted based on deficits identified.
- Electrolytes, including potassium, magnesium, calcium, and phosphorus, should also be monitored and replaced to avoid cardiac arrhythmias. Blood glucose monitoring and treatment of hyperglycemia are important. Monitoring of the kidney function, liver function tests, and lipase can monitor for organ function and/or the development of complications. LDH level trends can be helpful in mechanical circulatory support patients as a marker of hemolysis. Arterial blood gases can be used to titrate ventilator settings in addition to provide more information about the acid/base balance of the patient.
- Of note, postoperative troponin levels are not often followed due to troponin release during standard cardiac surgery; however, if concerned for ischemia, troponin levels can be followed. Persistently high levels of troponin had been associated with increased mortality.

Chest X-ray Evaluation [2, 19]

- Chest radiographs (chest X-rays) are a critical assessment tool. Immediately upon arrival to the ICU, a chest X-ray should be obtained to assess endotracheal tube and line positions to quickly identify malposition. Chest X-rays should also be performed when there is a change in the patient's condition to aid in identifi-

cation of the problem. While some institutions do perform chest X-rays daily, some experts would suggest only performing chest X-rays when clinically indicated rather than routinely after the first 24 h.
- Several abnormalities can be seen on chest X-rays including widening cardiac silhouette (potential cardiac tamponade), pneumothorax, hemothorax, atelectasis, or consolidation. Further, fluid status can be evaluated by identifying signs of pulmonary edema.

Warming, Weaning Sedation, Waking [2, 3]

- Obtaining normothermia is an important first step in recovery before further steps toward hemodynamic stability and extubation can be taken. Hypothermia is associated with decreased cardiac function, arrhythmias, and coagulopathy. Forced air warming devices are often utilized in the ICU to treat hypothermia. Ideal body temperature is greater than 36.0 °C.
- Weaning of sedation starts after the patient has been stabilized, and normothermia has been obtained. It is important to ensure paralysis has been reversed or worn off prior to waking. Further, the patient must be hemodynamically stable. It is important to treat pain as sedation is weaned. Quick stabilization and weaning of sedation support early extubation as described below.

Fluid Management [2, 3, 10, 20]

- Administration of fluid is often done to optimize preload and support CO to ensure end organ perfusion. Fluid administration should be individualized based on markers of perfusion, though most patients after cardiac surgery receive approximately 2–3 L of fluid in the immediate postoperative period.
- Determining if fluid will be beneficial to the patient versus harmful can be difficult. Both hypovolemia and fluid overload should be avoided. Fluid overload is associated with pulmonary edema, heart failure, delayed intestinal motility, and prolonged hospital stays. It is important to determine which patients are fluid responsive. Assessments such as monitoring blood pressure and pulse pressure during passive leg raise or fluid boluses can be helpful. A patient who is not responsive to fluid can be managed with vasopressors and inotropes, which can help to avoid fluid overload.
- Goal directed resuscitation, or fluid administration, is often utilized in the cardiac ICU in order to optimize cardiac output and oxygen delivery. Resuscitation in goal-directed therapy is based on signs of hypovolemia, including lactate trends, SvO_2 less than 60%, cardiac index (CI) less than 2.0, MAP <60, and urine output less than 0.5 mL/kg/h.

- The choice of which type of fluid, crystalloid, or colloid to administer is debated. Crystalloids are generally preferred for early resuscitation. The use of normal saline has decreased given high sodium and chloride content relative to physiologic levels, which can be associated with hyperchloremic acidosis and increased risk of acute kidney injury. For this reason, lactate ringers is often utilized as the crystalloid of choice. Albumin is a frequently utilized colloid in fluid resuscitation. Colloids theoretically have the benefit of increasing oncotic pressure and increasing plasma volume with less third spacing of fluid. However, there are not large-scale clinical trials to specifically support an exact fluid resuscitation protocol for cardiac surgery patients regarding crystalloid versus colloid at this time. Albumin does have an increased cost compared to crystalloids, so some experts suggest against its routine use.

Use of Sodium Bicarbonate [21–23]

- Initial studies identified a reduction of cardiac surgery associated acute kidney injury with the utilization of sodium bicarbonate loading and continuous infusions in patients undergoing cardiopulmonary bypass. However, further research found no benefit to sodium bicarbonate infusions. There was even association of this intervention with increased ventilator time, higher risk of alkalemia, and longer ICU length of stay.
- Sodium bicarbonate is still utilized as needed to buffer the pH to prevent significant acidosis during the immediate postoperative recovery.

Goals and Criteria for Extubation [2, 24, 25]

- The goal for patients after cardiac surgery is for early extubation, which is defined as extubation within 6 hours after surgery. Early extubation is used as a quality marker. This goal was developed as prolonged ventilation is associated with higher costs, increased complications, and longer hospital stays. Given the benefits of early extubation, many institutions have developed multidisciplinary protocols to assist in fast-track extubation pathways.
- Criteria for extubation first rely on decreased ventilator support. Patients should be able to maintain oxygenation with FiO_2 at or below 40% while on relatively low ventilator support. Once this is obtained, the patient's readiness for extubation can be further assessed. The patient should have spontaneous breathing without significant tachypnea, awake mental status, ability to follow commands, hemodynamic stability, and no evidence of significant bleeding. Often prior to extubation, a pressure support trial with decreased ventilator settings is completed to ensure the patient can maintain oxygenation and ventilation without ventilator support.

Complications After Cardiac Surgery

Postoperative Bleeding [6, 26–28]

- Excessive postoperative bleeding is associated with increased blood transfusions, hospital length of stay, and cost. Postoperative hemorrhage occurs in approximately 2–6% of cardiac surgery patients. Management of postoperative bleeding starts before even leaving the OR. Utilization of a checklist to ensure common areas of bleeding are checked and treated prior to closing has been associated with reduced rates of re-exploration. Often, time will be spent in the OR after the chest is closed monitoring chest tube output to identify patients who require surgical interventions to obtain hemostasis.
- An important focus in the immediate postoperative period is maintaining chest tube patency. If the chest tubes become clogged with clotted blood, this can lead to cardiac tamponade or clinically significant hemothorax, which can require invasive interventions.
- Close monitoring for coagulopathy and thrombocytopenia is critical in cardiac surgery patients as is treatment with transfusions to correct these abnormalities. Treating hypothermia can also help to reverse coagulopathy. Anti-fibrinolytic agents, like epsilon-aminocaproic acid (Amicar) and tranexamic acid (TXA), have been found to be associated with reduced transfusion requirements in cardiac surgery patients without significant increase in morbidity. Red blood cell transfusions can replace blood loss while providing time for correction of coagulopathies.
- If the above interventions are not successful in reducing blood loss, re-exploration and washout in the OR are definitive treatments for postoperative blood loss.

Tamponade [2, 3, 6, 29–31]

- Cardiac tamponade occurs when the chambers of the heart become compressed by external forces, often fluid or blood. This leads to an equalization of pressures between the four chambers and reduced venous filling. This reduces end diastolic volume and subsequently cardiac output. Tamponade can occur in the cardiac surgery patient when there is bleeding without sufficient drainage. This is often signaled by tachycardia, narrowed pulse pressure, increased CVP, and reduced cardiac output. Pulsus paradoxus can also been seen. Pulsus paradoxus is a reduction in systolic blood pressure greater than 10 mmHg during inspiration. Tamponade is also associated with increased vasopressor requirement and worsening markers of perfusion.
- In non-surgical patients, TTE is often utilized to be the gold standard of diagnosis for cardiac tamponade. However, TEE has a greater ability to visualize pericardial fluid/blood in patients after cardiac surgery. This is because TTE can be

limited by chest tubes, patient position, incision, and ventilation. Further, TEE also allows for visualization of the posterior pericardium, where fluid or clot may have accumulated.

- Echocardiographic evidence of cardiac tamponade includes collapse of the atria during systole or ventricles during diastole, respiratory variation of mitral and tricuspid flow, and enlargement of the inferior vena cava with reduced pulsatility. A chest X-ray can show an enlarged cardiac silhouette, and EKG can show low voltage. However, classic signs of tamponade after cardiac surgery may not be initially present so tamponade should be suspected in patients with hemodynamic instability and a pericardial collection.
- Maintaining chest tube patency is critical in the postoperative period to prevent the accumulation of blood around the heart. Additionally, avoiding significant coagulopathy can reduce bleeding postoperatively. In the immediate postoperative period, a definitive measure to treat tamponade is re-exploration in the OR, including opening of the chest and washout with identification and treatment of any areas of bleeding. In decompensating patients, the chest can be opened at the bedside. In patients who are several days from surgery, pericardial drain placement can relieve tamponade.

Atrial Fibrillation [2, 5, 6, 32–34]

- Atrial fibrillation is common after cardiac surgery. Rates of atrial fibrillation are between 10 and 50% after cardiac surgery, depending on the patient population and surgery. High rates of atrial fibrillation are associated with increased age, mitral valve disease, COPD, and valve surgery. Of note, medications likely epinephrine and norepinephrine can increase the risk of atrial fibrillation because of sympathetic stimulation. Atrial fibrillation in the postoperative period is associated with significant morbidity and increased length of stay.
- Beta blockers have been utilized at some institutions prophylactically to prevent postoperative atrial fibrillation. Amiodarone has also been used prophylactically, but often second line at many institutions due to possible toxicities and adverse effects.
- Pericardial effusions have been associated with increased rates of atrial fibrillation, thought to be due to the effusion causing local inflammation and oxidative damage. Posterior left pericardiotomy has been shown to reduce rates of atrial fibrillation without increasing complications. This procedure involves an incision in the posterior pericardium that allows postoperative pericardial fluid to drain into the left pleural space.
- The decision to anticoagulant postoperative patients with atrial fibrillation is somewhat controversial though most patients with persistent atrial fibrillation and higher CHA_2DS_2 VASc score are recommended to start on anticoagulation.

Stroke [6, 35, 36]

- The overall risk of stroke with neurological deficits after cardiac surgery is approximately 2–3%. The risk of stroke is associated with increased age, diabetes, known preoperative cerebrovascular disease, and emergent operations. The risk of stroke is highest in aortic and mitral valve surgery. Stroke is associated with increased morbidity and mortality after cardiac surgery.

References

1. Chatterjee S, Shake JG, Arora RC, Engelman DT, Firstenberg MS, Geller CM, et al. Handoffs from the operating room to the intensive care unit after cardiothoracic surgery: from the Society of Thoracic Surgeons Workforce on Critical Care. Ann Thorac Surg. 2019;107(2):619–30.
2. Stephens RS, Whitman GJ. Postoperative critical care of the adult cardiac surgical patient. Part I: routine postoperative care. Crit Care Med. 2015;43(7):1477–97.
3. Cohn LH, Adams DH. Cardiac surgery in the adult. 5th ed. McGraw-Hill Education; 2017.
4. Annich G, Lynch W, Maclaren G, Wilson J, Bartlett R, editors. ECMO: extracorporeal cardiopulmonary support in critical care. 4th ed. Ann Arbor: Extracorporeal Life Support Organization; 2012.
5. DiMarco RF Jr. Postoperative care of the cardiac surgical patient. In: Surgical intensive care medicine. Boston: Springer; 2010. p. 535–66.
6. Stephens RS, Whitman GJ. Postoperative critical care of the adult cardiac surgical patient: part II: procedure-specific considerations, management of complications, and quality improvement. Crit Care Med. 2015;43(9):1995–2014.
7. Tomita A, Takada S, Fujimoto T, Iwasaki M, Hayashi Y. Analysis of difficulty in placement of pulmonary artery catheter through the left internal jugular vein. JA Clin Rep. 2020;6(1):63.
8. Hadian M, Pinsky MR. Evidence-based review of the use of the pulmonary artery catheter: impact data and complications. Crit Care. 2006;10(3):S8.
9. Neerukonda T, Gibson WJ, Abicht T, Sauer A, Flynn BC. Pulmonary artery rupture management with a single lumen endotracheal tube: old tricks that should be revisited. Am J Case Rep. 2018;19:342–6.
10. Johnston LE, Thiele RH, Hawkins RB, Downs EA, Jaeger JM, Brooks C, et al. Goal-directed resuscitation following cardiac surgery reduces acute kidney injury: a quality initiative pre-post analysis. J Thorac Cardiovasc Surg. 2020;159(5):1868–77.e1.
11. Yelderman ML, Ramsay MA, Quinn MD, Paulsen AW, McKown RC, Gillman PH. Continuous thermodilution cardiac output measurement in intensive care unit patients. J Cardiothorac Vasc Anesth. 1992;6(3):270–4.
12. Reade MC. Temporary epicardial pacing after cardiac surgery: a practical review: part 1: general considerations in the management of epicardial pacing. Anaesthesia. 2007;62(3):264–71.
13. Feldman D, Pamboukian SV, Teuteberg JJ, Birks E, Lietz K, Moore SA, et al. The 2013 International Society for Heart and Lung Transplantation guidelines for mechanical circulatory support: executive summary. J Heart Lung Transplant. 2013;32(2):157–87.
14. Klijian A, Khanna AK, Reddy VS, Friedman B, Ortoleva J, Evans AS, et al. Treatment with angiotensin II is associated with rapid blood pressure response and vasopressor sparing in patients with vasoplegia after cardiac surgery: a post-hoc analysis of angiotensin II for the treatment of high-output shock (ATHOS-3) study. J Cardiothorac Vasc Anesth. 2021;35(1):51–8.
15. Bojar R. Manual of perioperative care in adult cardiac surgery. 5th ed. Wiley-Blackwell; 2011.

16. Overgaard CB, Dzavík V. Inotropes and vasopressors: review of physiology and clinical use in cardiovascular disease. Circulation. 2008;118(10):1047–56.
17. Totaro RJ, Raper RF. Epinephrine-induced lactic acidosis following cardiopulmonary bypass. Crit Care Med. 1997;25(10):1693–9.
18. Devereaux PJ, Lamy A, Chan MTV, Allard RV, Lomivorotov VV, Landoni G, et al. High-sensitivity troponin I after cardiac surgery and 30-day mortality. N Engl J Med. 2022;386(9):827–36.
19. Tolsma M, Kröner A, van den Hombergh CL, Rosseel PM, Rijpstra TA, Dijkstra HA, et al. The clinical value of routine chest radiographs in the first 24 h after cardiac surgery. Anesth Analg. 2011;112(1):139–42.
20. Mwaura L, Vuylsteke A. Fueling the debate on albumin after cardiac surgery. J Cardiothorac Vasc Anesth. 2019;33(11):2928–9.
21. Kim JH, Kim HJ, Kim JY, Ahn H, Ahn IM, Choe WJ, et al. Meta-analysis of sodium bicarbonate therapy for prevention of cardiac surgery-associated acute kidney injury. J Cardiothorac Vasc Anesth. 2015;29(5):1248–56.
22. Haase M, Haase-Fielitz A, Bellomo R, Devarajan P, Story D, Matalanis G, et al. Sodium bicarbonate to prevent increases in serum creatinine after cardiac surgery: a pilot double-blind, randomized controlled trial. Crit Care Med. 2009;37(1):39–47.
23. Tie HT, Luo MZ, Luo MJ, Zhang M, Wu QC, Wan JY. Sodium bicarbonate in the prevention of cardiac surgery-associated acute kidney injury: a systematic review and meta-analysis. Crit Care. 2014;18(5):517.
24. Chan JL, Miller JG, Murphy M, Greenberg A, Iraola M, Horvath KA. A multidisciplinary protocol-driven approach to improve extubation times after cardiac surgery. Ann Thorac Surg. 2018;105(6):1684–90.
25. Fitch ZW, Debesa O, Ohkuma R, Duquaine D, Steppan J, Schneider EB, et al. A protocol-driven approach to early extubation after heart surgery. J Thorac Cardiovasc Surg. 2014;147(4):1344–50.
26. Elassal AA, Al-Ebrahim KE, Debis RS, Ragab ES, Faden MS, Fatani MA, et al. Re-exploration for bleeding after cardiac surgery: revaluation of urgency and factors promoting low rate. J Cardiothorac Surg. 2021;16(1):166.
27. Christensen MC, Dziewior F, Kempel A, von Heymann C. Increased chest tube drainage is independently associated with adverse outcome after cardiac surgery. J Cardiothorac Vasc Anesth. 2012;26(1):46–51.
28. Gerstein NS, Brierley JK, Windsor J, Panikkath PV, Ram H, Gelfenbeyn KM, et al. Antifibrinolytic agents in cardiac and noncardiac surgery: a comprehensive overview and update. J Cardiothorac Vasc Anesth. 2017;31(6):2183–205.
29. Imren Y, Tasoglu I, Oktar GL, Benson A, Naseem T, Cheema FH, et al. The importance of transesophageal echocardiography in diagnosis of pericardial tamponade after cardiac surgery. J Card Surg. 2008;23(5):450–3.
30. Price S, Prout J, Jaggar SI, Gibson DG, Pepper JR. 'Tamponade' following cardiac surgery: terminology and echocardiography may both mislead. Eur J Cardiothorac Surg. 2004;26(6):1156–60.
31. Carmona P, Mateo E, Casanovas I, Peña JJ, Llagunes J, Aguar F, et al. Management of cardiac tamponade after cardiac surgery. J Cardiothorac Vasc Anesth. 2012;26(2):302–11.
32. Boons J, Van Biesen S, Fivez T, de Velde MV, Al Tmimi L. Mechanisms, prevention, and treatment of atrial fibrillation after cardiac surgery: a narrative review. J Cardiothorac Vasc Anesth. 2021;35(11):3394–403.
33. Gaudino M, Sanna T, Ballman KV, Robinson NB, Hameed I, Audisio K, et al. Posterior left pericardiotomy for the prevention of atrial fibrillation after cardiac surgery: an adaptive, single-centre, single-blind, randomised, controlled trial. Lancet. 2021;398(10316):2075–83.
34. Arsenault KA, Yusuf AM, Crystal E, Healey JS, Morillo CA, Nair GM, et al. Interventions for preventing post-operative atrial fibrillation in patients undergoing heart surgery. Cochrane Database Syst Rev. 2013;2013(1):Cd003611.

35. Sultan I, Bianco V, Kilic A, Jovin T, Jadhav A, Jankowitz B, et al. Predictors and outcomes of ischemic stroke after cardiac surgery. Ann Thorac Surg. 2020;110(2):448–56.
36. Karunanantham J, Ali JM, Evans NR, Webb S, Large SR. Impact of stroke on outcomes following cardiac surgery: propensity matched analysis. J Card Surg. 2020;35(11):3010–6.

Index

J. P. Bloom, T. M. Sundt (eds.), *Cardiac Surgery Clerkship*, Contemporary Surgical Clerkships, https://doi.org/10.1007/978-3-031-41301-8