Cardiac Anesthesia

The Basics of Evaluation and Management

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"In loving memory of my mother and father. I'm so grateful and proud to be your son!

To my wife, Afaf, for her love, support, and never-ending friendship; to our children, Rami, Nancy, and Adam, for their never-ending support and patience."

Ahmed Awad

"Dedicated to my mother, who introduced me to anesthesiology and medicine; and to my family, mentors, residents, and fellows, who make it all worthwhile."

Lou Mitrev

"This effort is dedicated to all past, present, and future trainees who devote their professional careers to the care of surgical patients with cardiovascular disease."

Gord

"Dedicated to the anesthesiologists who I have had the privilege of working with through the years. They tolerated me, taught me, respected me, and most of all kept me out of trouble. They have been my dear friends and colleagues throughout the years. It is my

true hope that this book helps them to comprehend my immense gratitude and helps the next generation anesthesiologists understand how important they are to the team."

Michael Rosenbloom

"Dedicated to the memory of my dad, Peter Solina, who lived his life to make others happy."

Al

Foreword

Cardiac Anesthesia. The Basics of Evaluation and Management edited by Ahmed Awad, MD with Drs. Mitrev, Morewood, Rosenbloom, and Solina as associate editors is quite a remarkable textbook. This book should be part of the library of every anesthesiology resident and adult cardiac anesthesiology fellow. It is a down-to-earth, nuts and bolts overview of state-of-the art adult cardiovascular vascular anesthesiology. It is beautifully illustrated and basic essential topics are masterfully and concisely explained. The style of presentation is extremely consistent, which is a major accomplishment for a multi-authored textbook. Trainees and anyone needing a refresher on basic topics essential to the practice of cardiovascular anesthesia will find this book indispensable.

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Preface

Cardiovascular anesthesia has evolved over the last six decades since the first successful use of the cardiopulmonary bypass machine, in 1953, for closure of atrial septal defect. Since then, as cardiac surgery has become more specialized and complex, cardiac anesthesia has correspondingly developed as a subspecialty.

Trainees starting their education in cardiovascular anesthesia can easily be overwhelmed. They face a daunting task: acquiring the vast knowledge base of this anesthesia sub-specialty; the major textbook (Joel Kaplan) alone has nearly 1700 pages.

Despite the availability of widespread online information, the presence of several comprehensive textbooks, and easy access to extensive review articles, anesthesia trainees often need a concise and relevant text to support their everyday clinical activities.

Cardiac Anesthesia: The Basics of Evaluation and Management is such a concise, practical, evidence-based, and clear educational resource. Also, in this book, trainees will not find just one expert's voice, but many expert voices, all with valuable information for them.

This book is divided into ten parts, dealing with all aspects of the care of patients undergoing cardiovascular surgery. The text of each chapter is simple and clear, and the illustrations provide clear descriptions of the concepts presented.

I hope that this book—devoted to clinical and practical application—meets your needs for simplified fundamentals and will assist you in becoming an accomplished cardiovascular anesthesia provider.

I would like to hear from you if you have corrections, or cardiovascular-anesthesia questions (all contributors will be credited). You can reach me via email in care of Springer Publisher.

Camden, NJ, USA — Ahmed S. Awad

Acknowledgments

I cordially acknowledge all associate editors, authors, and co-authors of this book, most of whom are practicing cardiac surgeons, cardiologists, hospitalists, and cardiovascular anesthesiologists, for their excellent contributions.

A medical illustrator must understand the concepts and techniques of the scientist as well as those of the artist. Thus, special thanks go to Mr. Glenn Calhoun and Ms. Soojung Moon, medical illustrators, for creating illustrations that allow the reader to visualize and understand the basic concepts and intellectual substance of cardiac anesthesia.

I also thank Dr. William Brown and his team at *International Medical Editing Service* for their valuable review and modification of the manuscript.

Disclaimer

Cardiac Anesthesia: The Basics of Evaluation and Management is a practical clinical guide. Dosages of medications are presented for quick reference only. Although the dosages have been meticulously reviewed, we cannot guarantee their complete accuracy. Before using the information in this book for patient care, readers should check package inserts for each drug, as approved by the FDA, and other resources.

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Part I

General Concepts

Basic Cardiovascular Anatomy

1

Magdy Takla, David Youssef, Angelo Andonakakis, and Irwin Gratz

Bernard was right. The germ is nothing, the terrain is everything

—Louis Pasteur

Anatomy of the Heart

Position of the Heart in the Thorax

The heart is a hollow, cone-shaped muscular organ in the middle mediastinum of the thoracic cavity, oriented slightly left of the sternum. The heart lies between the lungs and rests on the superior surface of the diaphragm, with its apex pointing inferiorly toward the left hip and its base pointing toward the right shoulder. The apex normally is formed by the left ventricle, and the base is formed by the atria and the origin of great vessels.

The Borders and Surfaces of the Heart

Because of its cone shape, the heart has three borders and three surfaces:

- The right border is mainly formed by the right atrium.
- The left border is mainly formed by the left ventricle and partially formed by the left atrial appendage.
- The lower border is formed largely by the right ventricle and partially by the right atrium.

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- The anterior surface is formed mainly by the right atrium and right ventricle.
- The diaphragmatic surface is formed by the right and left ventricles.
- The posterior surface is formed by the left atrium and the four pulmonary veins that drain into it.

Layers of the Heart Wall

The heart wall is composed of three basic layers: pericardium, myocardium, and endocardium. The myocardium is the muscle layer; the endocardium is the smooth inner endothelial lining; the pericardium is the covering sac.

The Pericardium

The pericardium is a double-walled fibroserous sac that wraps the heart and the roots of the great vessels. It has two layers: an outer fibrous layer and an inner serous layer. The pericardium supports and protects the heart. The fibrous layer is a tough layer that encompasses the heart and has openings to allow the superior vena cava, pulmonary trunk, and aorta to pass through it. The fibrous layer is tightly bound to the central tendon of the diaphragm. The serous layer of the pericardium is subdivided into two segments: The outer parietal pericardium lines the inner surface of the fibrous layer and the internal visceral layer that forms the outer layer of the heart, the epicardium. The pericardial space is the potential space between the parietal and visceral layers; it contains less than 50 mL of serous fluid. The pericardium and its fluid lubricate the dynamic, moving surface of the heart. Two pericardial sinuses (channels) pass through the pericardial cavity. These lines of reflection are marked on the posterior aspect of the heart by:

1. *The transverse sinus*, which is positioned above the left atrium, anterior to the superior vena cava but posterior to the pulmonary trunk and ascending aorta.
2. *The oblique sinus,* which is situated posteriorly to the heart and behind the left atrium; it is bordered by the two right and two left pulmonary veins.

The pericardium is supplied by several internal thoracic arteries and is innervated by the phrenic nerve. The visceral layer of the serous pericardium is innervated by branches of the sympathetic trunk and vagus nerves. Pain originating from the pericardium is felt as diffuse retrosternal pain often made worse by lying down.

Chambers of the Heart

The heart is a four-chamber system with dual pumps and two circuits. The first pump is a low-pressure system that drives blood to the lungs; the second pump is a high-pressure pump that drives blood to the whole body. The four chambers are the left and right atrium and two larger bottom chambers, the left and right ventricle.

The atria and ventricles are divided from one another by the interatrial septum and the interventricular septum, respectively.

The Right Atrium

The right atrium is in the right upper corner of the heart above the right ventricle. It has four anatomically distinct parts: a venous component, the vestibule, the right atrial appendage, and a septum.

The venous component is formed by convergence of the superior and inferior caval veins into the atrium, forming the posterior, smooth wall of the right atrium. A valve of the inferior vena cava (IVC), the Eustachian valve, guards the orifice of the IVC; during fetal life it directs oxygen-rich blood into the left atrium, thus bypassing the nonfunctioning lung during fetal life. This region has a network of filamentous strands, the Chiari network. The Eustachian valve and Chiari network are normal variants within the right atrium. The valve of the coronary sinus, the Thebesian valve, covers the opening of the coronary sinus to prevent blood backflow during systole.

The vestibule, the second component of the atrium, is just proximal to the opening of the tricuspid valve. It has a smooth area of musculature that converges into the leaflets of the tricuspid valve, as indicated in Fig. 1.1 below. The right atrial appendage, the third component of the atrium, is a thin-walled, triangular structure

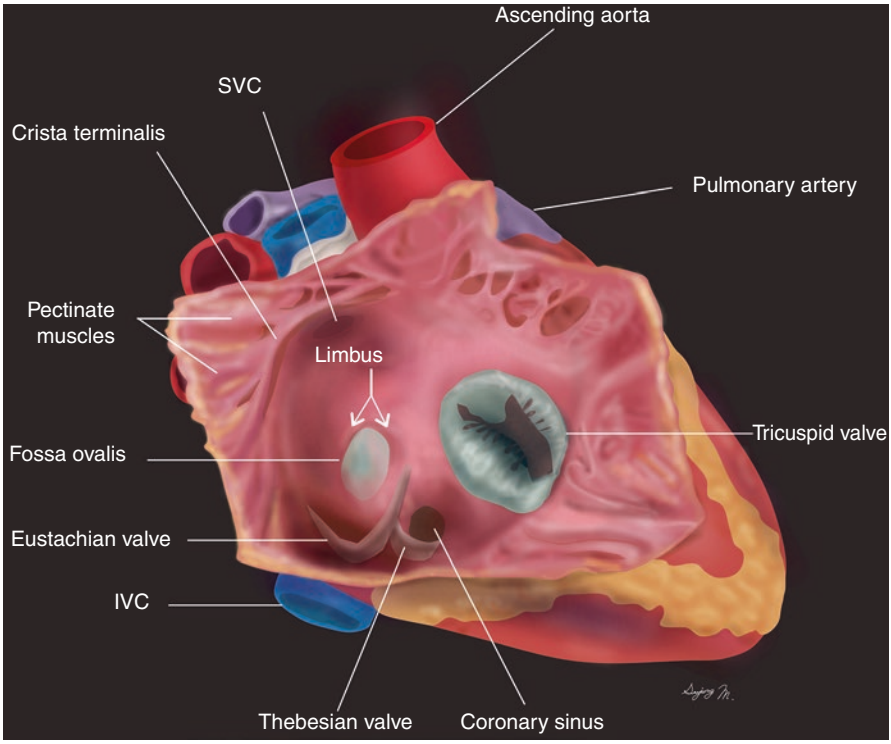

Fig. 1.1 Illustration of exposure of the right atrial cavity, schematic drawing

that is larger than the left atrial appendage. The junction with the vestibule is demarcated by a prominent muscular band, the crista terminalis. The septal wall is the fourth component of the atrium. It is a muscular wall that separates the two atria with a small, oval-shaped depression on the posterior wall called the fossa ovalis, which is a remnant of an interatrial septum opening, the foramen ovale. A prominent oval rim around the fossa ovalis is called the limbus fossa ovalis.

The Tricuspid Valve

The tricuspid valve separates the right atrium and right ventricle. The valve area, of 4–6 cm^2, makes it the largest of the four valves in the heart.

The tricuspid valve has three leaflets: anterolateral, posterolateral, and septal, as illustrated in Fig. 1.2, b. It has two to nine papillary muscles, which hold the chordae tendineae.

The Right Ventricle

The right ventricle (Fig. 1.3) is in the right lower corner of the heart. A crescent-shaped chamber that wraps around the left ventricle, it forms most of the anterior surface. It consists of three components: the inflow (sinus) component, a trabecular part, and the outflow tract. The inflow component consists of the tricuspid valvular apparatus, which includes the tricuspid valve, chordae tendineae, and papillary muscles. The second component is the coarse, trabeculated apical myocardium. The most protruding of the trabeculae is the septomarginal trabecula (moderator band); it connects the ventricular septum to the base of the anterior papillary muscle and transports part of the right bundle branch of the conduction system. The third part of the right ventricle is the smooth outflow tract, which contains the infundibulum, or conus. The infundibulum gives rise to the pulmonic valve and supports it. The inlet and outlet of the right ventricle are separated by a muscular ridge, the crista supraventricularis.

The Pulmonic Valve

The pulmonic valve divides the right ventricle from the pulmonary artery. It has three semilunar cusps: anterior, right, and left, as seen in Fig. 1.4. The cusps are thin and are not attached to the muscular wall of the pulmonary artery; they are supported by freestanding musculature.

The Left Atrium

The left atrium consists of an appendage, the main body, a septal portion, and the vestibule. The vestibule is the outlet of the atrial chamber, which surrounds the mitral valve. The interatrial septal wall is flat and has a flap valve of the fossa ovalis. The left atrium has a smooth, free wall. The left atrial appendage is multilobulated and significantly smaller and narrower than the atrial appendage on the right side.

The Mitral Valve Apparatus

The mitral valve apparatus is a complex structure composed of several parts working in synchrony to open during diastole and close in systole. The apparatus consists of six anatomic parts: an annulus, two leaflets, chordae tendineae, two papillary

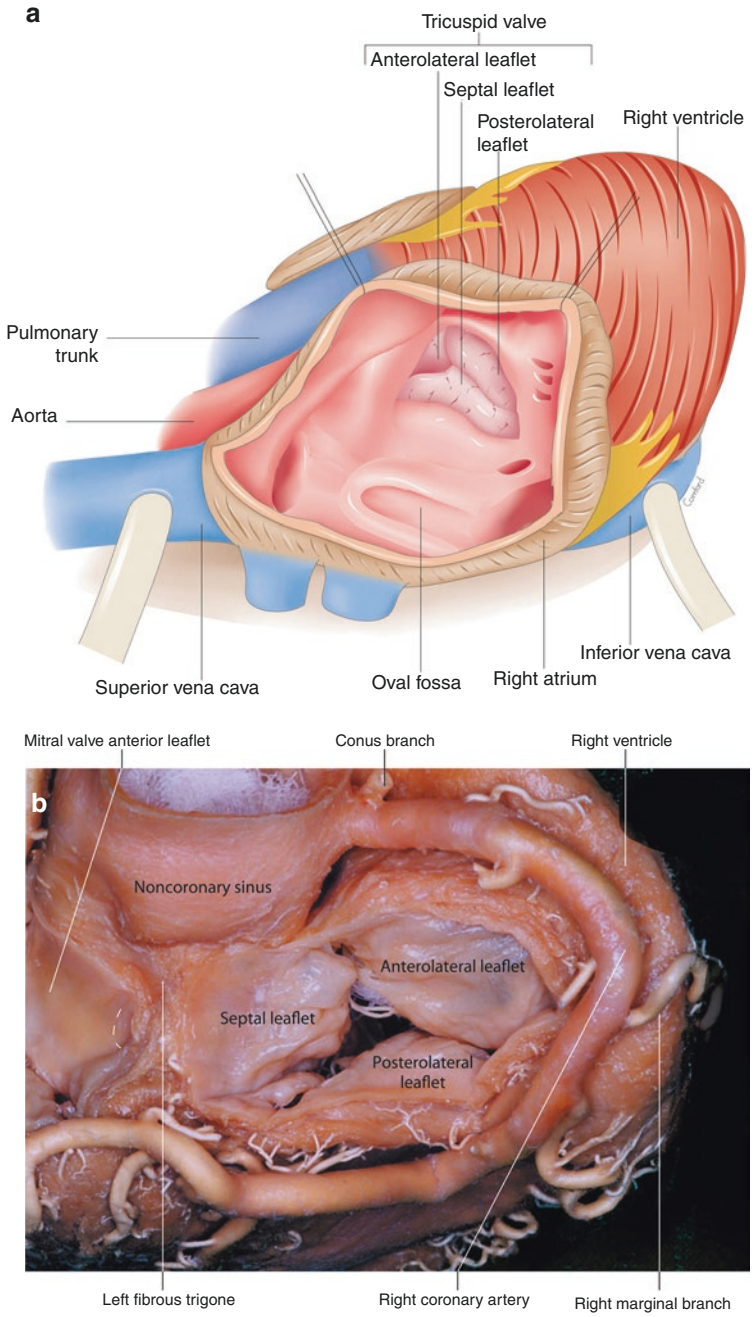

Fig. 1.2 (**a**) Illustration of the leaflets of the tricuspid valve in the diastolic position, schematic drawing. (Berdajs and Turina (2011), *Operative anatomy of the heart. Springer, Berlin, Heidelberg).* (**b**) Image of dissected portion of the leaflets of the tricuspid valve. (Berdajs and Turina (2011), *Operative anatomy of the heart. Springer, Berlin, Heidelberg*)

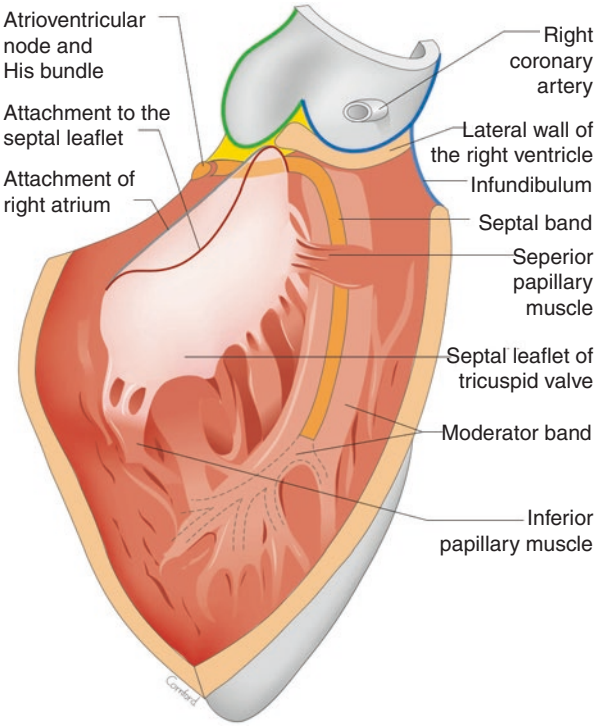

Fig. 1.3 Illustration of drawing of the right ventricle. (Berdajs and Turina (2011), *Operative anatomy of the heart. Springer, Berlin, Heidelberg*)

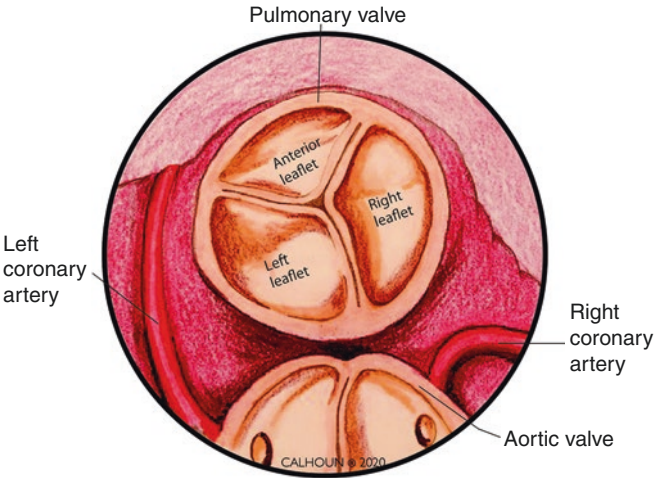

Fig. 1.4 Illustration of superior view of the pulmonary root

muscles, the posterior left atrial wall, and the left ventricular wall. Malfunction of any of these parts will disturb the synchrony of the valvular apparatus and render the valve incompetent. For instance, left atrial enlargement, dilatation of the mitral annulus, ischemia of the left ventricle wall or papillary muscle, or rupture of the chordae tendineae will directly alter the normal mitral valvular mechanism.

Leaflets

The mitral valve has an anterior leaflet and a posterior leaflet. The anterior leaflet is trapezoidal, is much broader than the posterior leaflet, and covers one-third of the circumference; the posterior leaflet contributes to the remaining two-thirds. The posterior leaflet is subdivided into three scallops, each demarcated by a cleft. The subdivisions are designated P1, P2, and P3. P1 is adjacent to the anterolateral commissure, and P3 is adjacent to the posteromedial commissure. P2 is the middle scallop. Figure 1.5a illustrates the typical arrangement of mitral valve anatomy. The anterior leaflet does not have clefts; however, it is divided into three segments, designated A1, A2, and A3, to correspond to P1, P2, and P3.

Annulus

The annulus is the junctional area that separates the left atrium and left ventricle and gives attachment to the mitral valve.

Papillary Muscles

The two papillary muscles with their chordae tendineae support the mitral valve leaflets and help anchor the leaflets to prevent them from protruding back into the atrium during systole. The posteromedial papillary muscle has cords that attach to A3, P3, and half of A2 and P2. The anterolateral papillary muscle has cords that attach to P1, A1, and half of A2 and P2.

The blood supply for the anterolateral papillary muscle comes from a branch of the left anterior descending coronary artery and, sometimes, also from a branch of the circumflex artery (first obtuse marginal artery). The posterolateral papillary muscle has one source of blood supply, depending on the dominance of coronary circulation, which can either be a branch of the posterior descending artery or a branch from the obtuse marginal artery.

Clinical Pearls

The dual blood supply protects the anterolateral papillary muscle from ischemic damage. However, the posterolateral papillary muscle is more prone to damage because of its single blood supply.

Chordae Tendineae

The chordae tendineae are fan-shaped chords extending from the papillary muscles and attached to the leaflets. There are three types of chordae tendineae, depending on the site of insertion. The primary chordae are thin and attach to the free margins

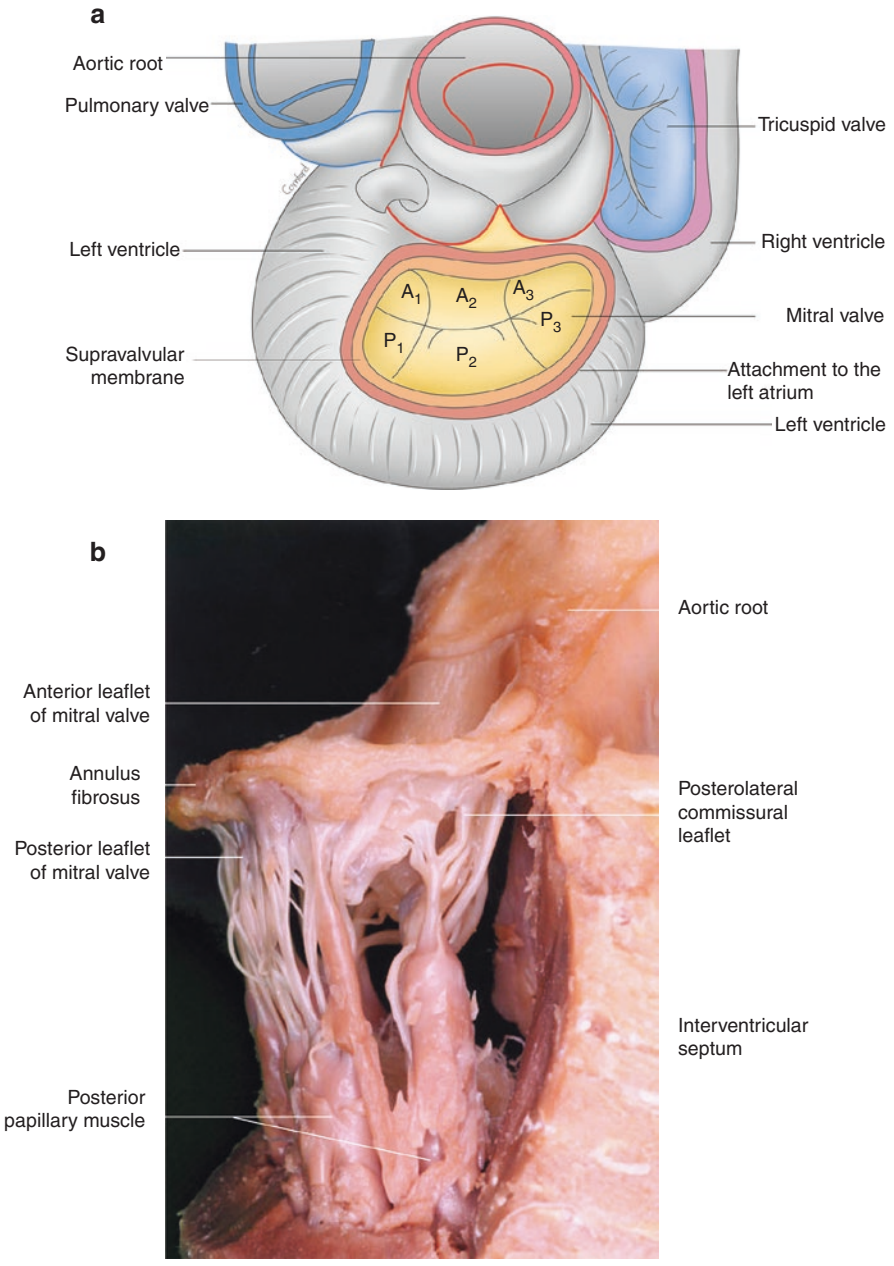

Fig. 1.5 (**a**) Illustration of posterior superior view of the mitral valve, schematic drawing. (Berdajs and Turina (2011), *Operative anatomy of the heart. Springer, Berlin, Heidelberg*). (**b**) Image of dissected portion of the leaflets of the mitral valve with papillary muscles and chordae tendineae. (Berdajs and Turina (2011), *Operative anatomy of the heart. Springer, Berlin, Heidelberg*)

of both leaflets. The secondary chordae or strut chordae are thick and attach to the body of both leaflets from the ventricular surface. The tertiary chordae are the thickest; they stem directly from the ventricular wall and attach only to the posterior leaflet (Fig. 1.5b).

The Left Ventricle

The left ventricle is in the left lower corner of the heart. In the anatomical position of the heart, it forms most of the lateral and posterior surface. It is a conical chamber, with a thick wall tapering to a rounded apex. It is comprised of three components:

- Inflow (inlet) component – guarded by the mitral valve
- Apical myocardium – made up of fine trabeculation
- Smooth outflow tract – leads to the aortic valve (Fig. 1.6)

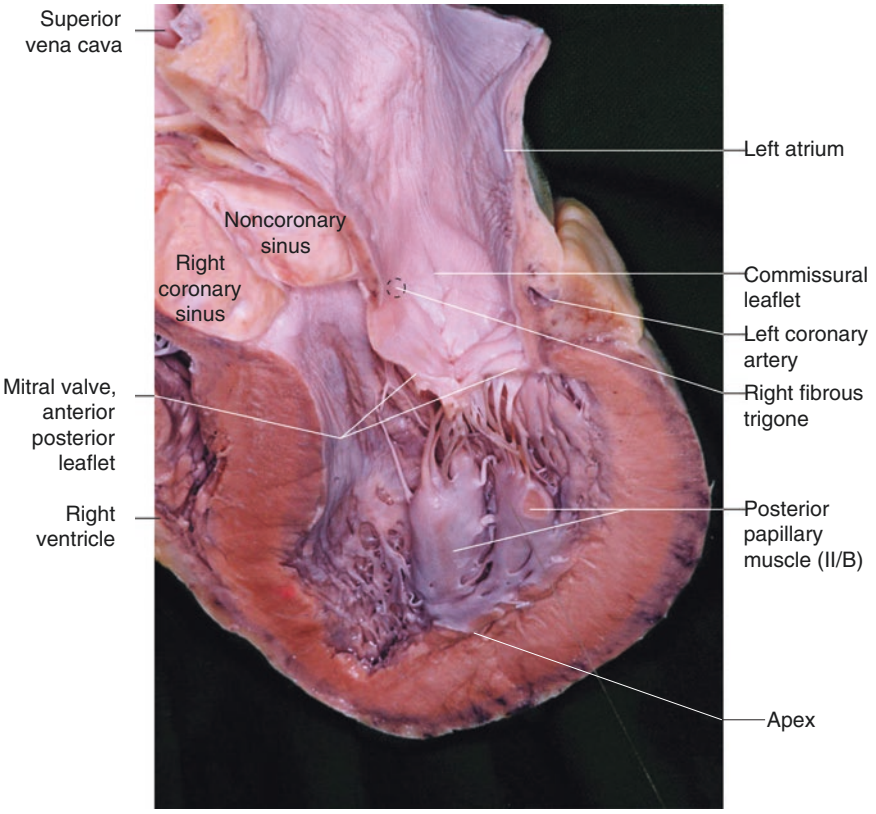

Fig. 1.6 Image of dissected portion of the left ventricle revealing the outlet "LVOT" and inlet "mitral valve." (Berdajs and Turina (2011), *Operative anatomy of the heart. Springer, Berlin, Heidelberg*)

The Interventricular Septum

The interventricular septum separates the right and left ventricles. It has a tiny fibrous part, the membranous septum, measuring about 0.5 cm^2, located immediately inferior to the commissure of the anterior and septal tricuspid cusps. The muscular portion is formed by myocardial-cell fibers from the left and right ventricle along with circumferentially arranged fibers derived from the left ventricle. Blood supply to the anterior two-thirds of the septum is by the septal perforators of the left anterior descending artery; blood supply to the posterior one-third is by the distal branches of the right coronary artery.

The Aortic Valve and Root

The aortic root is a complex structure made of four key components: the aortic annulus, the aortic valve, the aortic sinuses, and the sinotubular junction. The root extends between the left ventricle outlet and the ascending aorta longitudinally and lies between the anterior mitral valve leaflet and interventricular septum transversely.

Aortic Annulus

While there is no true anatomic annulus, histologically, the ventriculoaortic junction is considered the anatomic aortic annulus. This annulus is a collagenous ring and constitutes the tightest portion of the aortic root. It provides structural support to the aortic valve. A virtual basal ring, termed the "surgical annulus" by surgeons and echocardiographers, is formed by an imaginary line joining the hinge points of each aortic cusp. The surgical annulus is proximal to the anatomic aortic annulus, as illustrated below (Fig. 1.7) in relation to other anatomic structures. Typically, the diameter of the aortic annulus is between 16 and 30 mm.

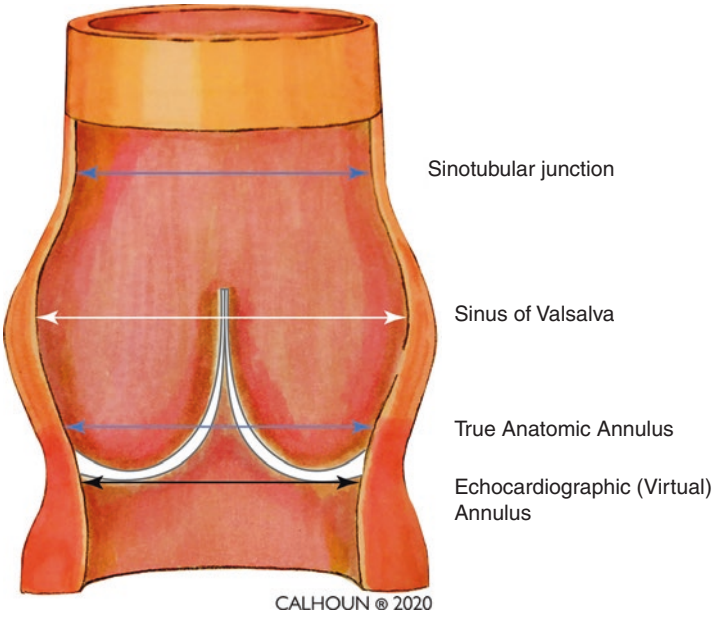

Fig. 1.7 Illustration of sinotubular junction as a superior border of the aortic root, schematic drawing

Aortic Valve Leaflets and Sinuses

The aortic valve is in the center of the heart, anterior to the tricuspid and mitral valves and posterior and medial to the pulmonary valve. The valve is composed of three semilunar cusps and has an area of 2.5–3.5 cm^2.

The three semilunar cusps are named by the sinuses that overlie them: the right coronary cusp, left coronary cusp, and noncoronary cusp. The sinus of Valsalva is the space between the luminal surface of the bulge in the wall of the aorta and its respective valvular cusp. The right coronary cusp is the most anterior cusp and has the right sinus of Valsalva overlying it, from which the right coronary artery emanates. The left coronary cusp contains the left sinus of Valsalva and gives rise to the left coronary artery. The noncoronary cusp is the most posterior and is slightly larger than the other two cusps. It lies adjacent to the interatrial septum. At the center edge of each cusp is a fibrous bulge called the nodule of Arantius. The normal triangular shape of the aortic valve leaflets is illustrated in Fig. 1.8a, b.

Clinical Pearls

In cardiopulmonary bypass, the right coronary artery, due to its anterior location, frequently is susceptible to air embolism after the aortic clamp is removed or immediately after weaning from the pump. This adverse event can result in intractable cardiac arrhythmia, right ventricle myocardial infarction, hypotension, and consequent low cardiac output syndrome.

Sinotubular Junction

The sinotubular junction is the transition zone in which the tubular portion of the ascending aorta joins the sinus of Valsalva portion (Fig. 1.9). The tip of each semilunar cusp is attached to the wall of the aorta at the superior border of the aortic root,

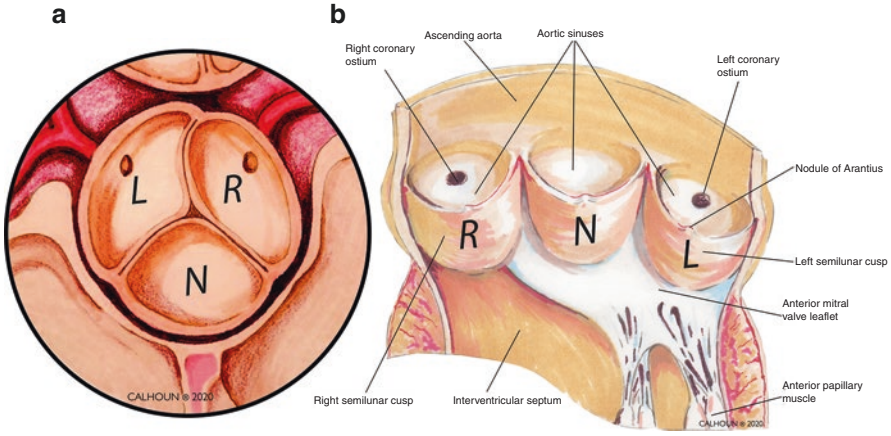

Fig. 1.8 (**a**) Illustration of the aortic valve (**b**) opened aortic valve with three cusps

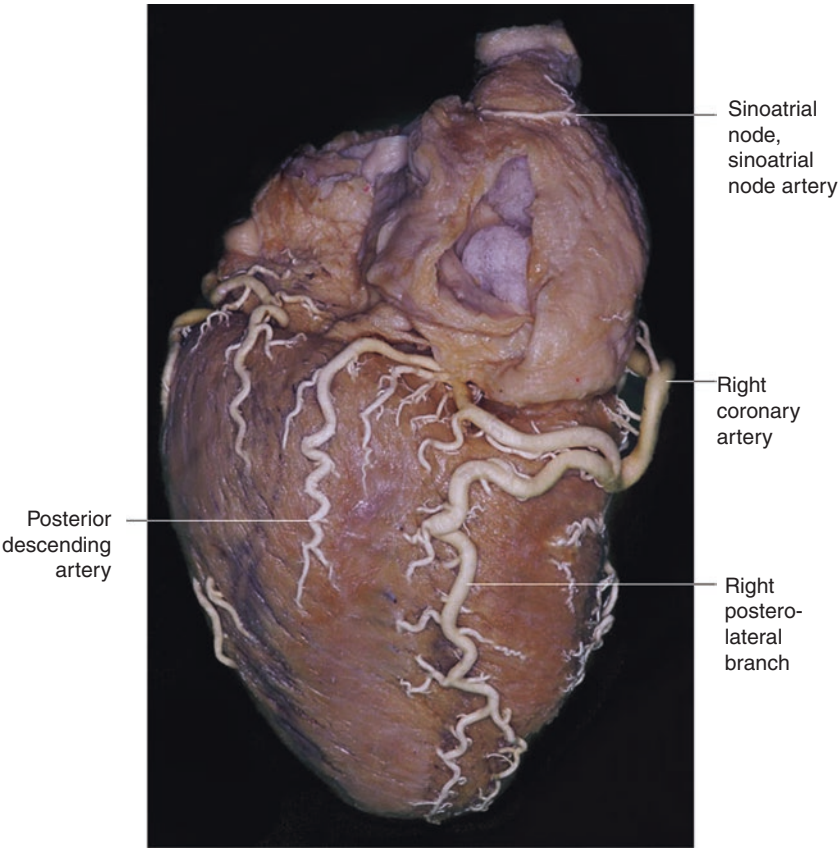

Fig. 1.9 Image of branches of the right coronary artery. (Berdajs and Turina (2011), *Operative anatomy of the heart. Springer, Berlin, Heidelberg*)

creating a raised ridge and forming the sinotubular junction. The diameter of the sinotubular junction usually exceeds the diameter of the surgical annulus by 1:1.6

Vascular Supply of the Heart

Coronary Artery Circulation

The heart is supplied by the right and left coronary arteries. They arise from the aortic root in the corresponding coronary sinus just below the level of the sinotubular junction. The right coronary artery (RCA) emerges anteriorly between the pulmonary trunk and the right atrial appendage and then descends in the right atrioventricular groove between the right atrium and right ventricle. It encircles the annulus of the tricuspid valve as it curves around the inferior border to reach the right ventricular diaphragmatic surface, where it descends into the posterior

interventricular groove and terminates as the posterior descending artery (Fig. 1.9). The RCA typically supplies the sinoatrial (SA) node, the right atrium, a portion of the left atrium, right ventricle, and posterior interventricular septum. In 60% of the population, it supplies the SA node, and in 80%, it supplies the atrioventricular (AV) node.

The left coronary artery emerges from the left coronary sinus and travels leftward between the pulmonary trunk and the left atrial appendage. It has a short main stem that divides under the left atrial appendage into the circumflex branch, which runs in the atrioventricular groove on the left and the left anterior descending artery that runs in the anterior interventricular groove. The left anterior descending artery supplies the anterior wall of the left ventricle through the diagonal branches and the anterior two-thirds of the interventricular septum through septal perforators.

The circumflex artery travels under the left atrial appendage to enter the left AV groove. It encircles the annulus of the mitral valve as it curves around the lateral border of the heart. It branches into obtuse marginal arteries, which supply the lateral aspect of the left ventricle, including the posteromedial papillary muscle (Fig. 1.10).

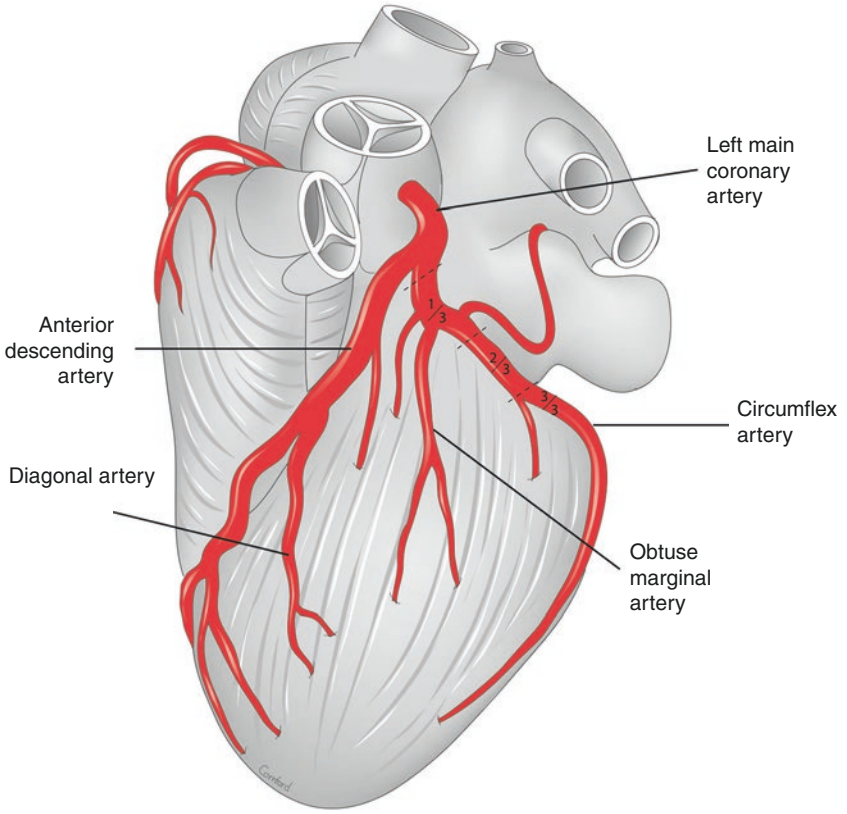

Fig. 1.10 Illustration of branches of the left main artery. (Berdajs and Turina (2011), *Operative anatomy of the heart. Springer, Berlin, Heidelberg*)

Venous Drainage of the Heart

Venous drainage of the heart occurs mostly through two systems: the coronary sinus and the Thebesian veins. The coronary sinus is the major venous drainage system. It starts when the great cardiac vein joins the oblique vein of the left atrium, and it runs transversely from left to right in the AV groove on the posterior surface of the heart. The coronary vein drains all the venous blood from the heart except for the anterior cardiac veins which drain directly into the right atrium, thus a total of 65–85% of the heart's venous drainage. The small, middle, and great cardiac veins, together with the oblique vein of the left atrium, empty into the coronary sinus, which opens into the right atrium. The Thebesian venous network is the lesser cardiac venous drainage; it constitutes 15–35% of the venous drainage of the heart. These veins are small valveless vessels embedded in the walls of the heart, which drain the inner layers of the myocardium directly into the lumens of heart chambers through openings in the endocardial surface, thus contributing to the right-to-left physiologic shunting.

The great cardiac vein is the largest vein that drains through the sinus; it begins at the apex of the heart and ascends upward toward the anterior interventricular groove accompanying the LAD, as illustrated in Fig. 1.11 below.

The middle cardiac vein drains most of the heart supplied by the RCA. It begins at the apex and ascends in the posterior interventricular groove accompanying the PDA leading to the coronary sinus. The small cardiac vein travels in the coronary groove and drains venous blood supplied by the marginal branches of the RCA. The figure below illustrates the posterior aspect of the heart and the cardiac veins draining into the coronary sinus.

Autonomic Innervation of the Heart

Autonomic innervation regulates cardiac function. The heart is innervated by parasympathetic vagal (cranial nerve X) and sympathetic fibers. These fibers originate in the medulla of the brainstem, which receives sensory input from receptors (e.g., baroreceptors and chemoreceptors) through afferent fibers. Autonomic outflow through efferent fibers modulates the activity of the heart. The left parasympathetic vagus nerve innervates the AV node, and the right innervates the SA node. The postganglionic sympathetic fibers travel along the arteries to innervate the SA and AV nodes. Sympathetic stimulation increases heart rate (chronotropy), contractility (inotropy), and conduction velocity (dromotropy), whereas parasympathetic stimulation decreases these activities. The nerve supply of the pericardium is from the phrenic nerve.

Anatomy of the Great Vessels

Five vessels leave and enter the heart: the superior vena cava (SVC), inferior vena cava (IVC), pulmonary trunk, pulmonary veins, and the aorta, as illustrated below in Fig. 1.12.

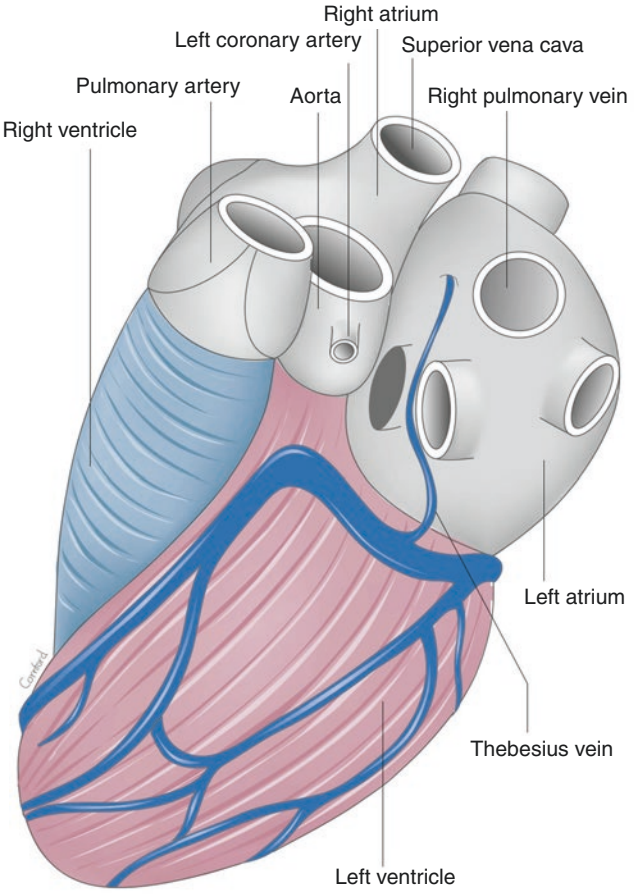

Fig. 1.11 Illustration of presentation of the heart venous system, schematic drawing. (Berdajs and Turina (2011), *Operative anatomy of the heart. Springer, Berlin, Heidelberg*)

The SVC and the IVC return deoxygenated blood from the body. The SVC is formed by merging of the left and right innominate veins (formed by the merger of each corresponding subclavian vein and jugular vein) behind the first right costal cartilage. It descends along the right side of the aorta and trachea and drains into the right atrium. The IVC is formed by the merger of the common iliac veins in the pelvis; it ascends in the abdomen along the right side of the vertebral column and abdominal aorta, where the renal veins and the hepatic veins drain directly into it. It enters the thorax through the central tendon of the diaphragm, where it drains into the right atrium.

The pulmonary trunk carries blood from the right ventricle to the lungs. It ascends from the conus arteriosus of the right ventricle and travels at an oblique

angle along the left ascending aorta, passing anterior to the left ventricle outflow and the aortic root. After the pulmonary trunk emerges from the pericardium, it divides into the left and right pulmonary arteries, which enter each corresponding lung.

The two right pulmonary veins travel behind the right atrium and SVC. The two left pulmonary veins travel in front of the descending thoracic aorta. The right and left pulmonary veins coalesce at the left atrium.

The aorta carries blood from the left ventricle to the systemic circulation. It is divided into the ascending aorta, arch of the aorta, and descending portion until it bifurcates into the common iliac arteries at the L4 level. First, the ascending aorta emerges from the left ventricle at an angle to the right and then curves to the left and backward as it becomes the aortic arch. The arch curves upward and backward over the right pulmonary artery, passing along the left side of the trachea and esophagus. The arch is defined as the segment of the aorta between the ascending and descending aorta. It starts proximal to the origin of the innominate artery and ends distal to the origin of the left subclavian artery. Finally, it enters the posterior mediastinum as the descending thoracic aorta. The descending thoracic aorta begins at the T4 vertebral body and descends on the left side of the fifth through twelfth thoracic vertebral bodies (T5–T12) and enters the abdomen through the aortic hiatus at T12. The thoracic aorta has several branches; the first branches are the coronary arteries.

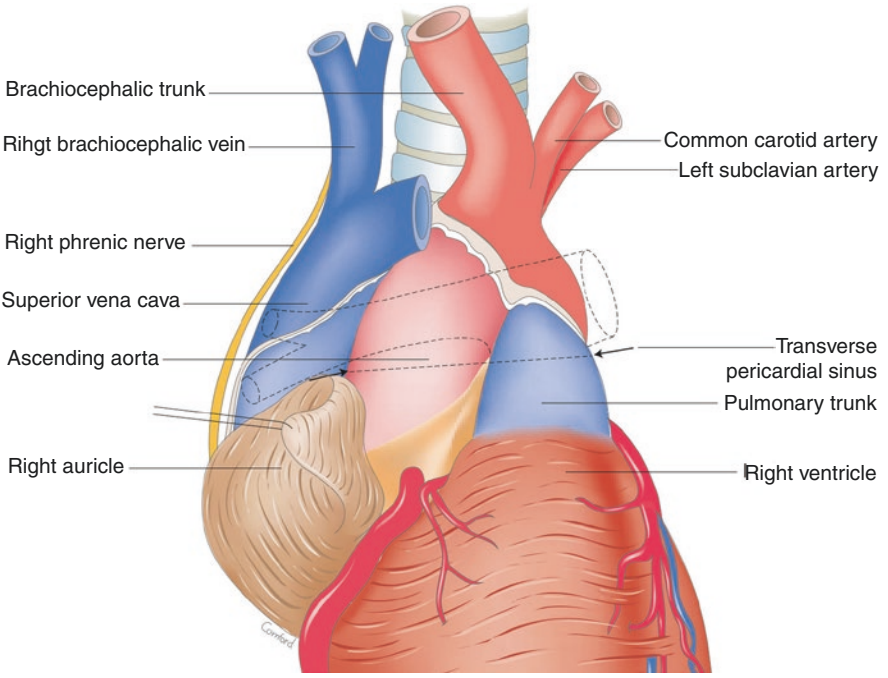

Fig. 1.12 Illustration of schematic view of the great vessels. (Berdajs and Turina (2011), *Operative anatomy of the heart. Springer, Berlin, Heidelberg)*

Next, three branches arise from the arch (proximal to distal): the innominate, left common carotid, and left subclavian arteries.

The aortic arch contains peripheral baroreceptors and chemoreceptors that relay blood pH and pressure information to the spinal cord medulla. The descending aorta in the thorax gives off branches to supply the intercostal, pericardial, bronchial, and mediastinal arteries. The abdominal aorta, a direct continuation of the descending aorta, gives blood to the entire abdomen, including the kidneys, foregut, midgut, and hindgut. It also carries the artery of Adamkiewicz, which arises from the left posterior intercostal artery at the level of the ninth to twelfth thoracic vertebrae and is the largest anterior segmental medullary artery supplying the lower two-thirds of the spinal cord. The artery of Adamkiewicz has significant variability and is the only major arterial supply of the anterior spinal artery to the lower thoracic, lumbar, and sacral cord.

Further Reading

Alston P, Myles P. Oxford textbook of cardiothoracic anaesthesia. Anatomy and pathology of the heart and major vascular system. Oxford: Oxford University Press; 2015.

Berdajs D, Turina M. Operative anatomy of the heart. Berlin, Heidelberg: Springer; 2011.

Erdman A. Concise anatomy for anaesthesia. The cardiovascular system. London: Greenwich Medical Media; 2001.

Erdmann A. Concise anatomy for anesthesia. New York: Cambridge University Press; 2006. ISBN 1841100692.

Farag E, et al. Basic sciences in anesthesia. Cardiovascular anatomy and pharmacology. Berlin, Heidelberg: Springer; 2018.

Filipoiu F. Atlas of heart anatomy and development. Notions of clinical anatomy of the heart: definition, situation, and position. London: Springer, Verlag; 2014.

Ho SY, Cabrera JA, Sanchez-Quintana DS. Left atrial anatomy revised. Arrhyth Electrophysiol. 2012;5:220–8.

Basic Cardiovascular Physiology

2

Robert Morris, Muhammad Ahmed, Spencer Drotman, and Y. Yuliana Salamanca-Padilla

The more the ECG resembles the EEG, the sicker the heart.

—Stephen J. Prevoznik

Introduction

The cardiovascular system consists of the heart, blood vessels, and circulating blood. The heart, by contracting, provides energy for forward movement of blood to perfuse the body's organs. Blood vessels are distribution channels through which blood flows from the heart to the entire body and returns to the heart. The heart functions as two pumps in series: one forces blood to the pulmonary circulation, where exchange of oxygen and carbon dioxide occurs between blood and alveoli, and the other forces blood to the systemic circulation, where it fulfills the metabolic requirements of body tissues by distributing metabolites and oxygen and collecting wastes and carbon dioxide; it also is responsible for hormone distribution and thermoregulation. Blood pumped by the heart is quantified as a unit of volume per minute and is called "cardiac output." Cardiac output can be influenced by many factors, including heart rate, stroke volume, preload, afterload, diastole, and systole. The body pairs cardiac output with the demands of the organs being perfused; these factors work together to closely pair oxygen supply with demand.

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This chapter contains a brief presentation of these topics:

- Excitation contraction coupling
- Cardiac cycle
- Control of cardiac output
- Arterial system and control of blood pressure
- Principles of oxygen transport and consumption
- Factors affecting myocardial metabolism

The Conduction System

Please refer to Chap. 4 for discussion of the conduction system.

Cardiac Excitation–Contraction Coupling

Cardiac excitation–contraction coupling is the chain of events that links the propagation of an electrical impulse through the conduction system to the myocardial cells; it achieves well-organized, synchronized myocardial contraction and relaxation. Excitation–contraction coupling is accomplished by the interaction of three components: a mechanical unit (cardiac myocyte), electrical excitation (action potentials), and the *coupling of electrical and mechanical activity.*

Cardiac Myocytes (the Mechanical Unit)

Cardiac myocytes are a specialized muscle fiber composed of myofibrils, which consist of actin and myosin proteins organized as sarcomeres. Unique to cardiac muscle tissue is the organization of sarcomeres via intercalated discs, which enables synchronized contraction. Cardiac myocytes contain a network of transverse tubules (*T*-tubules), which are invaginations of the sarcolemma that permeate the myocardial filaments, allowing for rapid, uniform contraction (Fig. 2.1).

The Electrical Activity

The creation and propagation of electrical impulse are initiated by a sequence of ion movements across the cardiac cell membrane. There are two types of the action potential, depending on the type of myocardial cell: cardiac myocyte potential and pacemaker cell potential.

1. *Cardiac myocyte action potential*

The cardiac myocyte has a resting membrane potential of −90 mV. Action potential is triggered by stimulation from neighboring cells and is divided into five phases: 0 through 4 (Fig. 2.2).

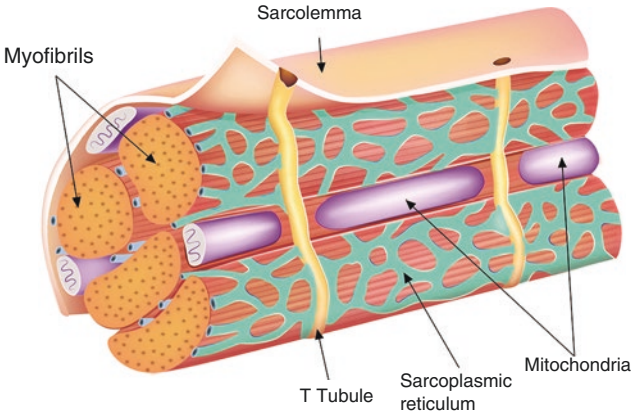

Fig. 2.1 Illustration of action potential profiles for cardiac myocyte and pacemaker cells. Cardiac tissues composed of myofilaments arranged in sarcomeres, *T*-tubules, and numerous mitochondria for energy

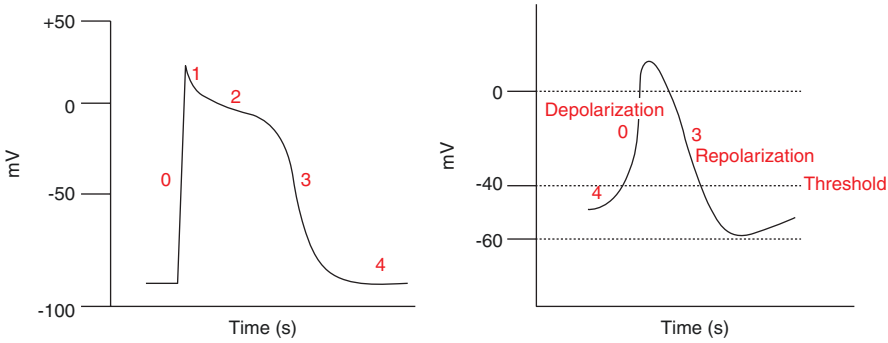

Fig. 2.2 Illustration of action potential profiles for cardiac myocyte and pacemaker cells

- Phase 0 starts by the opening of fast sodium channels, thus allowing for rapid depolarization.
- Phase 1 is rapid partial repolarization that is carried out by outward flow of potassium ions.
- Phase 2 is the plateau phase, in which there is a balance of inward flux of calcium ions and outward flow of potassium ions.
- Phase 3 is considered repolarization, in which potassium continues to efflux while influx of calcium stops.
- The action potential concludes in phase 4, in which the resting membrane potential is maintained by potassium efflux that is balanced by sodium and calcium influx.

2. *Pacemaker cell action potential*

The heart also has pacemaker cells with different types of action potentials; they are present in the His–Purkinje system, atrioventricular node, and sinoatrial node. These cells possess self-initiated depolarization or rhythmic firing, which is a unique quality of automaticity. They can spontaneously trigger an action potential. The sinoatrial node normally fires at a rate of 60–100 beats per minutes, and the atrioventricular node at a rate of 40–60 beats per minute. The action potential is slower than myocardial non-pacemaker action potentials and consists of only three phases (Fig. 2.2). Phase 4 is the spontaneous depolarization phase owing to slow influx of sodium, leading to rise of the membrane potential from −60 mV to about −40 mV. In phase 0, when the threshold potential is reached at around −40 mV, an upstroke phase occurs due to calcium influx into the cell, allowing for a slower rate of depolarization than in other cardiac cells. Phase 3 is the repolarization phase and consists of potassium channels that open and allow an efflux of potassium ions.

Events that Lead to Myocardial Fiber Contraction (Coupling)

Depolarization of the action potential is sensed by a *T*-tubule voltage sensor that allows for a conformational change of calcium channels along the tubules. This action permits release of calcium from the sarcoplasmic reticulum. Calcium binds with troponin C, allowing interaction between actin and myosin to facilitate muscle contraction (Fig. 2.3).

Phases of the Cardiac Cycle

The cardiac cycle is a finely coordinated series of events that occurs in a single heartbeat. A common tool to help understand the cardiac cycle is the Wiggers diagram (Fig. 2.4). This diagram temporally relates blood pressures (aortic,

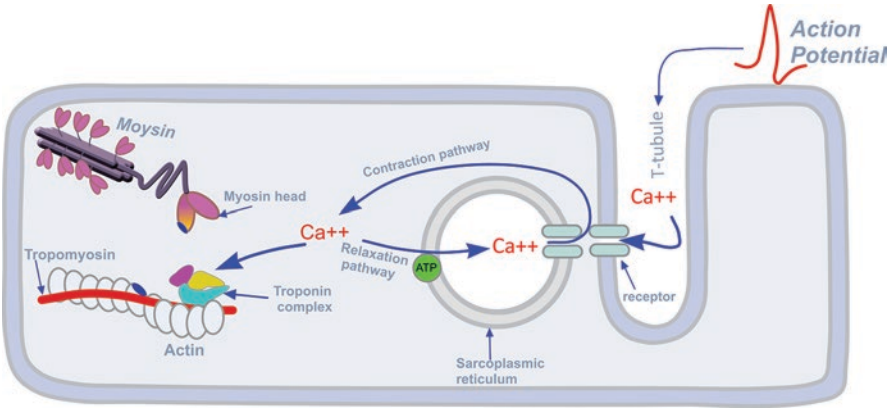

Fig. 2.3 Illustration of excitation–contraction coupling in systole

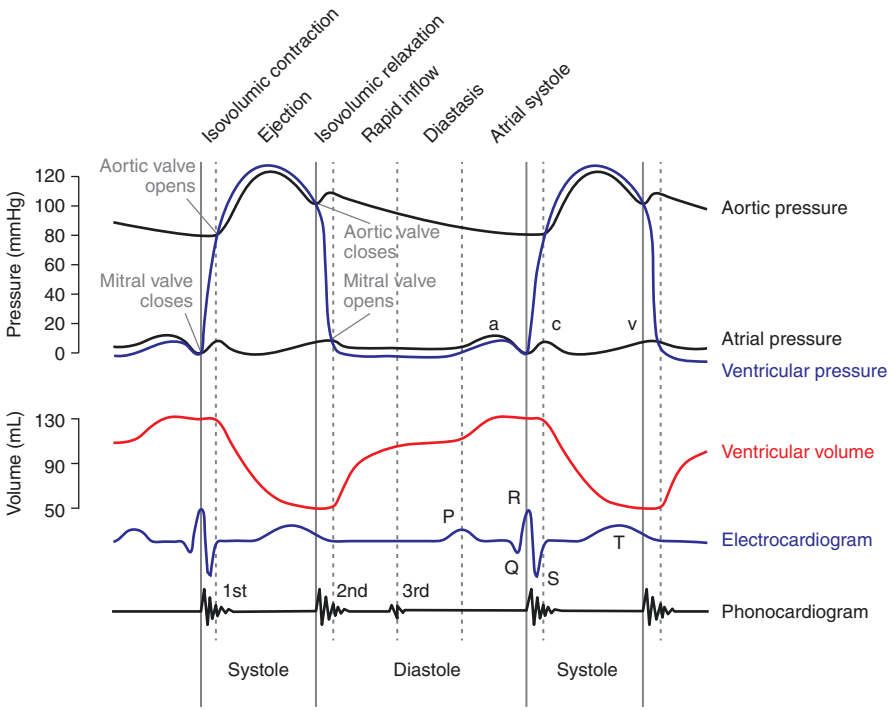

Fig. 2.4 Illustration of action potential profiles for cardiac myocyte and pacemaker cells. Wiggers diagram depicting the electrical, mechanical, and audible events of the cardiac cycle. (Ref: P. Colli Franzone et al., Mathematical Cardiac Physiology)

ventricular, and atrial), with ventricular volumes and an electrocardiographic (ECG) tracing. The cardiac cycle has seven phases, four with diastole and three with systole.

1. *Diastolic diastasis*

Near the end of diastole, both the atria and the ventricles are relaxed. The tricuspid and mitral valves are open, so blood flows from the atria into the ventricles. The pulmonary and aortic valves are closed. Slow filling, the longest duration of the cardiac cycle, provides about 5% of diastolic filling. With increase in the heart rate, the duration of this phase is compromised.

2. *Atrial systole and late diastole*

Atrial systole and late diastole are initiated by atrial depolarization, represented by the P wave of the ECG, followed by right and left atrial contraction. Blood is pumped across the open tricuspid and mitral valves into the ventricles. At rest, atrial contraction contributes about 15–20% of diastolic ventricular volume, which is often known as the "atrial kick." As atrial pressure rises and retrograde atrial blood

flows into the venous vessels, a wave pressure tracing on the jugular and left atrium appears and reflects atrial systole. The ventricular volume at the end of this phase is called the "end-diastolic volume."

3. *Isovolumetric contraction*

Systole is the stage of the cardiac cycle that consists of ventricular contraction and the ejection of ventricular blood. It is initiated by ventricular depolarization, represented by the beginning of the QRS complex (a combination of the Q, R, and S waves) of the ECG followed by isovolumetric contraction. In this phase, the ventricular contraction increases the pressure of the ventricles above that of the atria, forcing the closing of the atrioventricular valves, but not enough to open the pulmonic and aortic valves (semilunar valves), so all valves are closed. c-wave pressure tracing of the jugular and left atrium appears as the atrioventricular valve cusps bulge back into the atria.

4. *Rapid ejection phase*

In this phase, ventricular contraction raises intraventricular pressure to its maximum until it exceeds the aortic or pulmonic arterial pressure, forcing open the semilunar valves and causing a rapid ejection of ventricular blood. Also, atrial filling starts. The T wave of the ECG marks the end of the rapid ejection phase. With downward movement of the atrioventricular valves, pressure in the atria drops as the valves are pulled away from the atria. x descend appears on the jugular and left atrial pressure tracings.

5. *Reduced ejection phase*

The reduced ejection phase is initiated by ventricular repolarization, represented by the T wave of the ECG, followed by a drop in ventricular pressure; ejection of ventricular blood continues but at a slow rate. Filling of the atria persists, and the atrial pressure increases just before the atrioventricular valves close, forming the v wave on the jugular and left atrial pressure tracings. The ventricular volume at the end of this phase is called the end systolic volume.

6. *Isovolumetric relaxation*

Diastole, or ventricular relaxation, begins with isovolumetric relaxation, in which intraventricular pressure decreases before blood can enter the ventricles. Once intraventricular pressure becomes less than atrial pressure, the atrioventricular valves (tricuspid and mitral valves) open to allow flow of blood from the atria into the ventricles.

7. *Early diastolic/rapid filling*

The initial rapid inflow stage provides about 80% of the diastolic volume. A drop in pressure in the atria occurs when the atrioventricular valves open, and y descend appears on the jugular and left atrial pressure tracings.

Pressure–Volume Loops

The ventricular pressure curve, coupled with the volume curve, during a complete cardiac cycle creates the ventricular pressure–volume loop (Fig. 2.5). The shape of the loop is influenced by ventricular contractility and compliance. The loop allows easy evaluation and interpretation of left ventricular systolic and diastolic function. It can be thought of as composed of four phases: diastole, isovolumetric contraction, ejection, and isovolumetric relaxation.

Diastole Occurs Along the End-Diastolic Pressure–Volume Relationship (EDPVR)

The EDPVR represents compliance of the ventricles and is represented graphically by the lower curve of the pressure–volume relationship. The slope of the EDPVR is the reciprocal of compliance; thus, anything that increases compliance (such as a dilated cardiomyopathy) decreases the slope of the EDPVR, and anything that decreases compliance (such as ventricular hypertrophy) increases the slope.

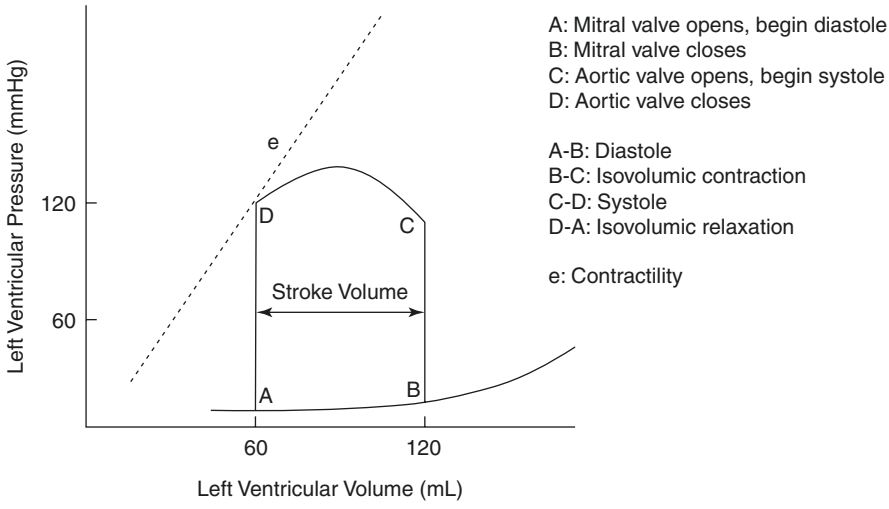

Fig. 2.5 Illustration of pressure volume diagram of a single cardiac cycle. (Ref: Iaizzo, Paul A. Handbook of cardiac anatomy, physiology, and devices)

The end-systolic pressure–volume relationship (ESPVR) is related to contractility of the ventricle. It represents the maximal pressure that can be generated at any volume within the left ventricle. The ESPVR slope is directly related to cardiac muscle contractility, with a steeper slope indicating increased contractility and a flatter slope indicating decreased contractility.

Determinants of and Control of Cardiac Output

Cardiac output is the volume of blood pumped by the heart per minute. It is a product of stroke volume and heart rate. Stroke volume is determined by three factors: preload, afterload, and contractility. More discussion about this subject can be found in Chap. 9, "Intraoperative hemodynamic monitoring for the cardiac surgery patient."

Preload

At the cellular level, preload is the degree of stretch of individual myocytes at maximal ventricular distension, just before systole. Ventricular volume is used as a surrogate of myocyte stretch. Left ventricular end-diastolic volume or left ventricular end-diastolic pressure can be used for quantitative measures of preload. Left ventricular end-diastolic volume can be determined with echocardiography.

The relationship between myocardial stretch and myocardial contraction can be explained with the Frank–Starling curve (Fig. 2.6), which illustrates how changes in venous return affect stroke volume.

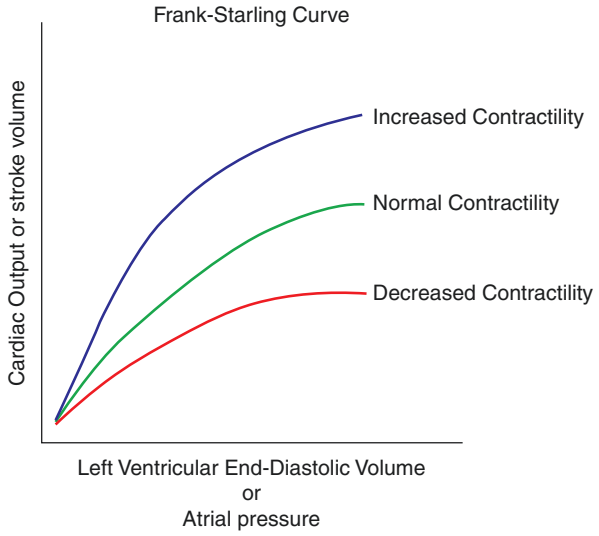

Fig. 2.6 Illustration of the Frank–Starling curve. If end-diastolic volume increases, cardiac output also increases. Excessive volume and muscle stretch will result in decreased cardiac output

Increases in preload result in increased stretch on myocardial sarcomeres, which results in increased force generation and thus increased cardiac output.

Afterload

Afterload refers to the pressure against which a ventricle must contract to eject blood. In the right heart, this refers to pulmonary artery pressure, and in the left heart it refers to aortic pressure. Afterload is primarily determined by pulmonary and systemic arterial vascular resistance. Afterload and cardiac output are inversely related, such that as afterload increases, output decreases.

Contractility

Contractility, or inotropism, refers to the heart's innate ability to generate force at a given sarcomere length. It correlates directly with intracellular calcium concentrations, which are dependent on sarcoplasmic calcium stores and the inward calcium current. The autonomic nervous system exerts the greatest effect on contractility. Sympathetic inotropic stimulation of the cardiac myocyte exerts its effects through β_1 receptors.

Arterial System and Control of Blood Pressure

The primary goal of the cardiovascular system is delivery of oxygen and nutrients to the tissues and removal of carbon dioxide and waste products. Forward flow in the system is determined by the pressure gradient between the arterial and venous systems. Maintaining an adequate mean arterial pressure, which is determined by cardiac output and systemic vascular resistance, is necessary for tissue perfusion. Fortunately, the cardiovascular system has many mechanisms for regulating blood pressure. The two most important mechanisms are a fast mechanism, that is, the baroreceptor mechanism, which is controlled by the central nervous system, and a slow mechanism, that is, the renin–angiotensin–aldosterone mechanism, which is controlled through hormones.

The Baroreceptor Reflex

The baroreceptor reflex is a suppressive system that regulates blood pressure in response to intra-arterial pressures (Fig. 2.7). The receptors are highly innervated stretch receptors in the carotid sinus and aortic arch. They respond to elevated blood pressure by increasing the impulse firing rate, which is transmitted via the glossopharyngeal nerve and the vagus nerve to the vasomotor centers in the brain stem. Stimulation of the vasomotor centers responds with inhibition of sympathetic

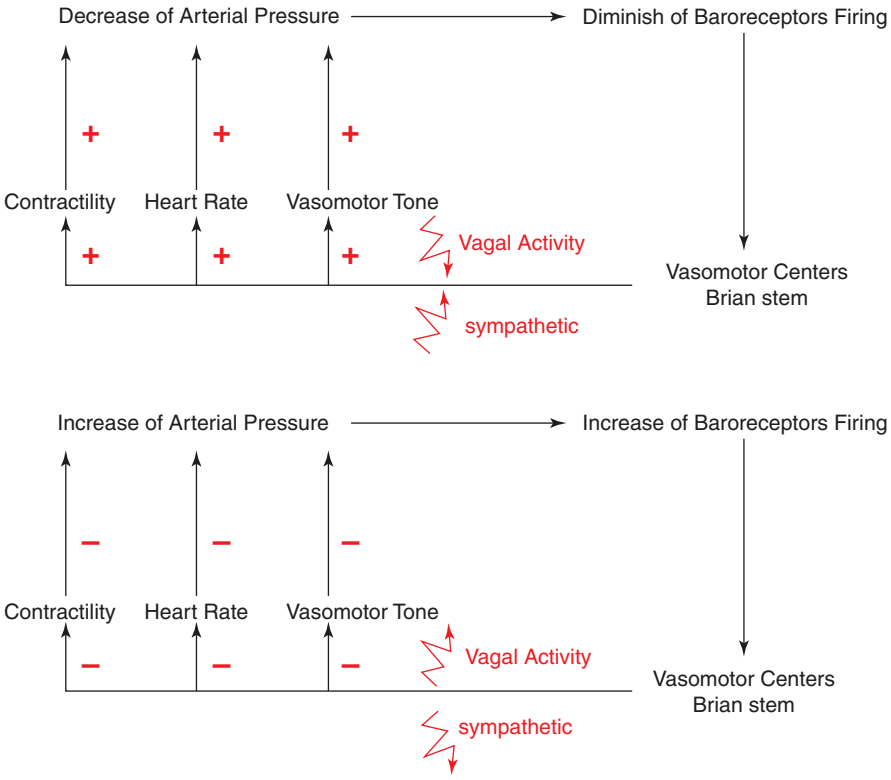

Fig. 2.7 Illustration of the negative feedback control of blood pressure

output. Thus, increased arterial pressure increases the frequency of baroreceptor firing and increases sympathetic inhibition, whereas reduction in blood pressure diminishes the frequency of firing. Increase in arterial pressure inhibits vasomotor areas, resulting in decreased heart rate, myocardial contractility, and vasodilation, thus lowering blood pressure. Also, stimulation of vagal regions via the vagus nerve contributes to the bradycardia and further lowers blood pressure by exerting its effects on the sinoatrial node.

The Renin–Angiotensin–Aldosterone System

The system regulates blood pressure primarily by increasing blood volume through hormonal control. Release of renin into the systemic circulation is triggered by low renal perfusion pressure or by decrease in tubular sodium content (Fig. 2.8). Released renin cleaves angiotensinogen (a hepatic protein) to angiotensin I. Angiotensin I is converted to angiotensin II by angiotensin-converting enzyme,

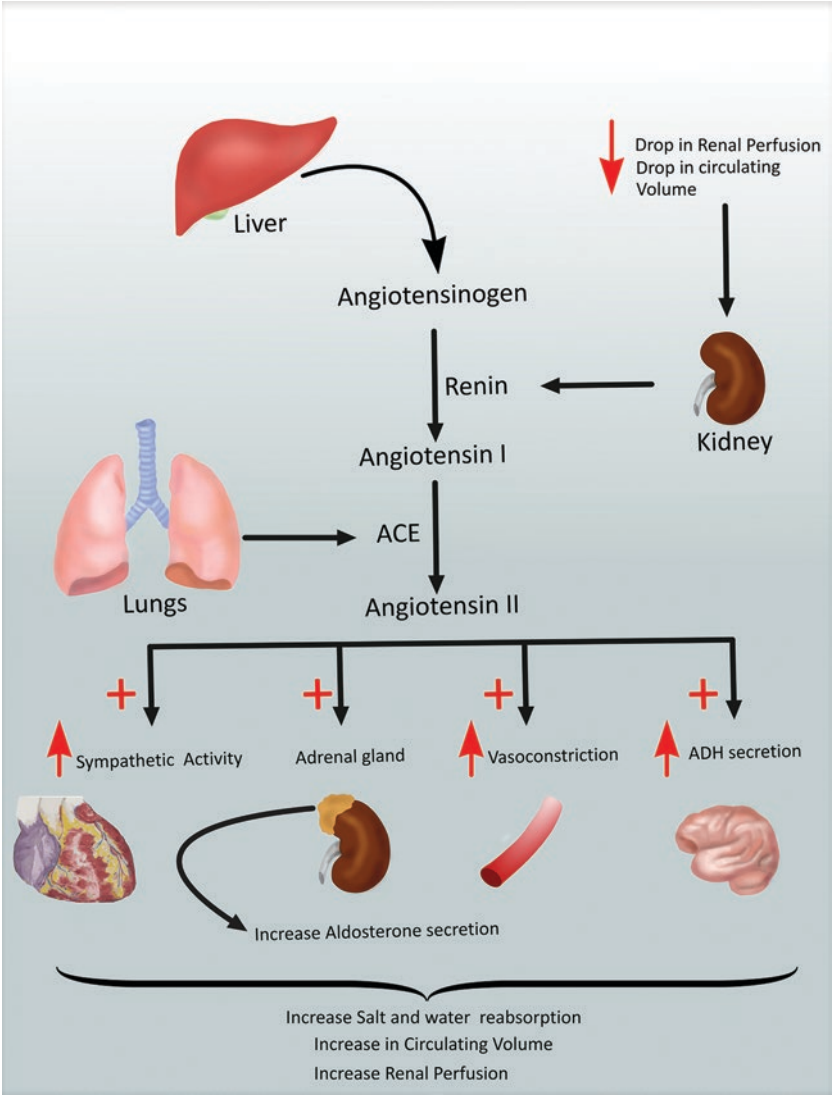

Fig. 2.8 Illustration of the renin–angiotensin System

which is present mainly in the lungs. Angiotensin II exerts its effects on multiple sites: It stimulates the release of adrenaline, noradrenaline, and aldosterone from the adrenal gland and promotes the secretion of vasopressin from the pituitary gland. Whereas aldosterone increases Na^+ and Cl^- reabsorption, leading to water retention, adrenaline and noradrenaline cause arteriolar vasoconstriction. Vasopressin release leads to antidiuresis.

Principles of Oxygen Transport and Consumption

Tissue oxygenation is dependent on delivery of oxygen and oxygen consumption of tissues. Oxygen delivery is determined by the product of cardiac output and arterial oxygen concentration, as expressed by the equation

$$DO_2 = CO \times CaO_2,$$

where DO_2 is oxygen delivery, CO is cardiac output, and CaO_2 is arterial oxygen concentration.

Arterial and venous oxygen concentrations can be determined with the formulas

$$CaO_2 = \left(Hgb \times 1.39 \times SaO_2\right) + \left(PaO_2 \times 0.003\right)$$
$$CvO_2 = \left(Hgb \times 1.39 \times SvO_2\right) + \left(PvO_2 \times 0.003\right),$$

where Hgb represents hemoglobin concentration of the blood, SaO_2 represents arterial oxygen saturation, and SvO_2 represents venous oxygen saturation. Each gram of hemoglobin can carry 1.39 mL of oxygen. PaO_2 represents the oxygen tension in arterial blood; 0.003 is the solubility coefficient of oxygen in human plasma. The primary determinant of arterial oxygen concentration is the amount of oxygen bound to hemoglobin.

The amount of oxygen in blood that is bound to hemoglobin is primarily determined by plasma pH, temperature, and the concentration of 2,3-diphosphoglycerate. Anything that increases hydrogen ion concentration (decreased pH), increases temperature and increases 2,3-diphosphoglycerate. 2,3-diphosphoglycerate facilitates oxygen dissociation from hemoglobin, as would be expected in catabolic states where there is higher oxygen utilization by tissue and a right shift in the oxygen hemoglobin dissociation curve. The reverse is true for physiologic conditions that decrease hydrogen ion concentration (increased pH), decrease temperature, and decrease 2,3-diphosphoglycerate, with a left shift in the oxygen hemoglobin dissociation curve (Fig. 2.9).

Oxygen consumption can be calculated with the Fick Principle as the difference between arterial and venous O_2 content:

$$VO_2 = CO \times \left(CaO_2 - CvO_2\right),$$

where VO_2 represents oxygen consumption, which is the product of cardiac output and the difference between arterial oxygen concentration and venous oxygen concentration.

The Fick Principle can be used further as a determinant of either cardiac output or oxygen consumption when applied to the mixed venous oxygen saturation by combining the above equations:

$$SvO_2 = SaO_2 - \left[\left(VO_2\right) / \left(Hgb \times 1.36 \times CO\right)\right]$$

This equation helps elucidate that mixed venous oxygen saturation depends on arterial oxygen saturation, oxygen consumption, hemoglobin concentration, and

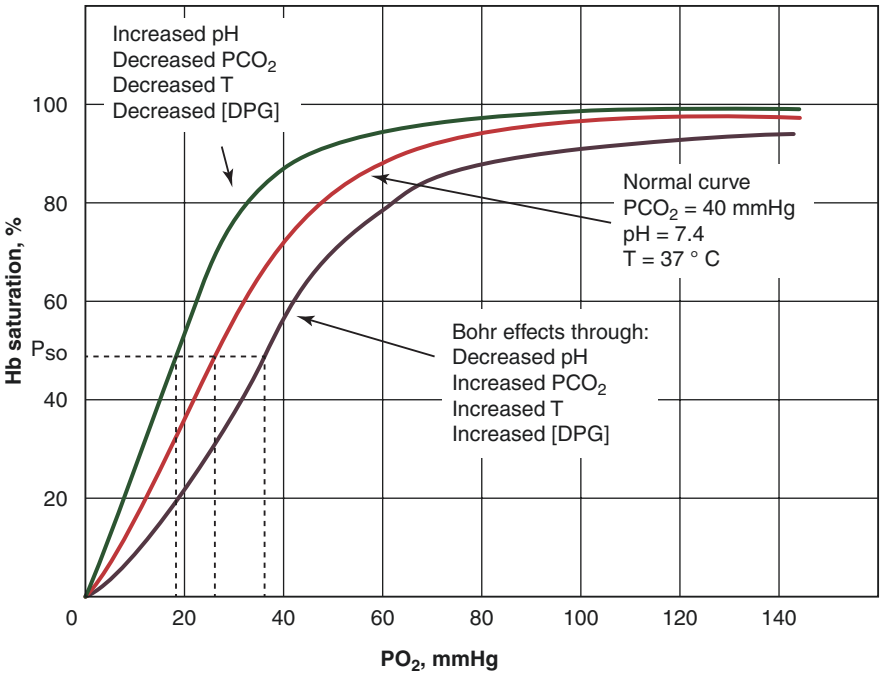

Fig. 2.9 Illustration of the oxygen–hemoglobin dissociation curve can be shifted to the right by several factors. The increased P50 in these cases indicate a lower hemoglobin–oxygen affinity. (Ref: Springer Nature Singapore Pte Ltd. 2018. H. M. Cheng and F. Jusof, Defining physiology: principles, themes, concepts)

cardiac output. Normal SvO_2 is approximately 75%, representing a tissue extraction of one molecule of oxygen per hemoglobin (normal hemoglobin has four oxygen-binding sites). Therefore, in a steady state of tissue metabolism and hemoglobin concentration, changes in venous oxygen concentration are directly related to changes in cardiac output.

Factors Affecting Myocardial Metabolism

Myocardial Oxygen Supply

Myocardial oxygen supply is dependent on two factors:

1. Coronary blood flow: Coronary blood flow is proportional to change in pressure divide by resistance.
   Coronary circulation has a high degree of autoregulation over wide ranges of pressure ranges.

Table 2.1 Factors affecting myocardial oxygen supply

Increase myocardial oxygen supply		Decrease myocardial oxygen supply	
↑Coronary blood flow	↑Arterial oxygen content	↓Coronary blood flow	↓Arterial oxygen content
Increased aortic diastolic pressure	Increased hemoglobin concentration	Decreased aortic diastolic pressure	Decreased hemoglobin concentration
Decreased left ventricular end-diastolic pressure (LVEDP)	Increased arterial oxygen partial pressure	Decreased time in diastole	Decreased arterial oxygen partial pressure
Increased diastolic time		Increased LVEDP	
Decreased coronary vascular resistance		Increased coronary vascular resistance	

$$\text{Coronary perfusion pressure} = \text{aortic diastolic pressure} - \text{left ventricular end-diastolic pressure}$$

Coronary vascular resistance is primarily determined by local metabolic control in response to hypoxia, as mediated by adenosine. Decreased myocardial oxygen tension leads to adenosine-mediated vasodilation. There is some autonomic control as well, with sympathetic-mediated coronary vasodilation and parasympathetic-mediated vasoconstriction.

2. Arterial Oxygen Concentration
   See principals of O_2 transport above
   The factors affecting myocardial oxygen supply are summarized in Table 2.1.

Myocardial Oxygen Consumption

Myocardial oxygen consumption is most strongly determined by three factors: heart rate, contractility, and wall tension. Heart rate may be an especially important determinant of myocardial oxygen consumption because it influences both oxygen supply and demand, as increase in heart rate decreases the time in diastole. Contractility affects oxygen consumption in that more powerful contractions require more antitachycardia pacing (ATP), and thus oxygen consumption. Wall tension is determined by the Law of Laplace, which states that wall tension is directly proportional to the pressure and radius of the heart, and inversely proportional to the wall thickness:

$$\text{Wall tension} = (\text{pressure} \times \text{radius}) / (2 \times \text{wall thickness})$$

Factors Affecting Myocardial Oxygen Consumption

Factors that increase myocardial oxygen demand:

- Increased heart rate
- Increased contractility
- Increased wall tension
- Increased basal metabolic rate

Factors that decrease myocardial oxygen demand:

- Decreased heart rate
- Decreased contractility
- Decreased wall tension
- Decreased basal metabolic rate

Through a balance of myocardial oxygen supply and demand the heart is capable of adequate perfusion and the avoidance of myocardial ischemia.

Clinical Pearls

- Heart rate, preload, afterload, and myocardial activity are the main determinants of pump performance.
- Preload is the quantity of blood that a cardiac chamber contains immediately before contraction begins.
- Afterload is the external resistance to emptying that confronts the chamber after the onset of contraction.
- Oxygen delivery: $DO_2 = CO \times CaO_2$
- Arterial oxygen content of blood: $CaO_2 = (Hgb \times 1.39 \times SaO_2) + (PaO_2 \times 0.003)$
- Oxygen hemoglobin dissociation curve:
  - Right shifts are due to increased H^+, pCO_2, temperature, and 2,3-diphosphoglycerate.
  - Left shifts are due to decreased H^+, pCO_2, temperature, and 2,3-diphosphoglycerate.

Further Reading

1. Iaizzo PA. Handbook of cardiac anatomy, physiology, and devices. 3rd ed. Cham: Springer International Publishing; 2015.
2. Kaplan JA. Kaplan's cardiac anesthesia: in cardiac and noncardiac surgery. 7th ed. Philadelphia: Elsevier Inc; 2017.
3. Klabunde RE. Cardivascular physiology concepts. 2nd ed. Philadelphia: Lippincott Williams Wilkins, a Wolters Kluwer Business; 2012.
4. Mohrman DE, Heller LJ. Cardiovascular physiology. 9th ed. New York, USA: McGraw-Hill Education; 2018.
5. Reves JG, Reeves Scott T, Abernathy III, James H. Atlas of cardiothoracic anesthesia. 2nd ed. Philadelphia: Current Medicine Group; 2009.

Basic Cardiovascular Pharmacology

3

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Medicine heals doubts as well as diseases.

—Karl Marx

Abbreviations

BP	Blood pressure
cAMP	Cyclic adenosine monophosphate
CPB	Cardiopulmonary bypass
cGMP	Cyclic guanosine monophosphate
HIT	Heparin-induced thrombocytopenia
LV	Left ventricle or left ventricular
MAO	Monoamine oxidase
MAP	Mean arterial pressure
PCC	Prothromobin complex concentrate
PDA	Phosphodiesterase
SVR	Systemic vascular resistance

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Overview

The pharmacologic agents commonly used in the treatment of cardiothoracic surgery patients are agents that (1) have a direct or indirect effect on the heart and circulatory system (e.g., vasopressors, inotropes, vasodilators, antiarrhythmic medications, and medications used to treat myocardial ischemia) and (2) those that do not have a direct effect on the heart and circulation (e.g., hematologic agents, diuretics, anesthetic agents, and antihyperglycemic agents). This chapter will describe briefly the pharmacology of the most commonly used medications in the perioperative management of cardiac surgery patients used including (1) the pharmacodynamics, i.e., the effect of the drug on the body, such as the cardiovascular and central nervous systems; (2) the pharmacokinetics, i.e., the effect of the body on the drug, such as absorption and distribution; and (3) the side effects of the pharmaceutical agents used.

Vasopressors

Vasopressors are a class of short-acting drugs that constrict blood vessels, leading to an increase in systemic vascular resistance (SVR) and consequent increase in mean arterial pressure (MAP). Through this hemodynamic effect, vasopressors help maintain organ perfusion when the vascular resistance is inadequate. The drugs are given intravenously, preferably through a central line, thus having an immediate effect. Vasopressors are different from inotropes, which only increase myocardial contractility; some vasopressors increase both SVR and myocardial contractility. The mechanism of action of vasopressors is mediated by alpha receptors, beta receptors, or vasopressin-1 receptors on the smooth muscle of vessels; some vasopressors affect multiple receptors. Activation of these receptors increases cytosolic calcium, with consequent vasoconstriction.

Phenylephrine HCl (Neosynephrine)

Mechanism of action:	Powerful direct-acting alpha$_1$-adrenergic agonist. A vasoconstrictor without a direct cardiac effect
Dose:	Bolus: 50–100 μg every 1–2 min as needed Infusion dose: 25–200 μg/min or 0.3–2 μg/kg/min
Onset of action:	Immediate onset
Dilution:	Dilute 10 mg of phenylephrine (1 mL) with 100 mL of the chosen diluent to get 100 μg/mL concentration.
Half-life:	2–3 h. Metabolized by monoamine oxidase (MAO). Clinical effect of bolus administration is brief, with need to re-administer after several minutes.
Hemodynamic effects:	Increases SVR and blood pressure (BP); decreases heart rate (reflex bradycardia)
Clinical usage:	Hypotension, nasal decongestion, mydriasis
Side effects:	Reflex bradycardia, severe hypertension if injected in patients using MAO inhibitors (e.g., tricyclic antidepressants); tissue and skin necrosis with extravasation at the site of injection

Ephedrine Sulfate

Mechanism of action:	Adrenergic agonist agent with mixed effects. Mainly at alpha and $beta_1$ receptors. Acts indirectly through the release of catecholamines. Longer acting and less potent than epinephrine
Dose:	Bolus: 5–10 mg IV every 5–10 min
Dilution:	Dilute 50 mg of ephedrine sulfate (1 mL) with 9 mL of the chosen diluent to get 5 mg/mL concentration.
Onset of action:	1 min
Duration of action:	1 h
Half-life:	3–6 h. Eliminated by liver and kidney
Hemodynamic effects:	Increases systolic, diastolic, and MAP. Increases myocardial contractility, heart rate, and cardiac output
Clinical usage:	Treatment of hypotension, bradycardia, bronchospasm, and urinary incontinence
Side effects:	Repeated doses can result in tachyphylaxis from depletion of catecholamine stores.

Clinical Pearls

1. Crosses the blood-brain barrier, resulting in dose-dependent side effects of anxiety, restlessness, nervousness, and tachycardia.
2. Severe hypertension if injected in patients who are using drugs or medications that inhibit reuptake of norepinephrine (e.g., cocaine) or affect the degradation of norepinephrine (e.g., MAO inhibitors). Chronic cocaine users may be catecholamine depleted and therefore not respond to ephedrine.

Norepinephrine (Levophed)

Mechanism of action:	Sympathomimetic, with direct $alpha_1$ and $beta_1$ agonist properties
Dose:	Bolus: 4–16 μg IV push Infusion dose: 4–10 μg/min or 0.05–0.15 μg/kg/min
Onset of action:	30 s
Dilution:	Dilute 4 mg with 250 mL of 5% dextrose to achieve a concentration of 16 μg/mL.
Half-life:	2 min. Primarily metabolized by MAO and catechol-O-methyltransferase at the synaptic cleft
Hemodynamic effects:	Increases SVR, heart rate, and contractility, thereby raising BP. Has more inotropic effects than chronotropic effects. Causes pulmonary artery vasoconstriction. Sometimes infused through a left atrial line in hypotensive patients who have pulmonary hypertension (minimizes the vasoconstrictor effect in the pulmonary vasculature)
Clinical usage:	Low BP caused by low SVR, as in septic shock. Used when less potent drugs are inadequate
Side effects:	Tachyarrhythmias and ischemia (myocardial, mesenteric, renal, extremities); tissue and skin necrosis, with extravasation at the site of injection

Vasopressin (Pitressin)

Mechanism of action:	A hormone synthesized in the hypothalamus and released from the pituitary into the circulation. Two primary functions: antidiuretic action via activation of the vasopressin-2 receptors and vasoconstriction via activation of smooth muscle vasopressin-1 receptors
Dose:	Bolus: 0.1–0.2 units Infusion dose: 0.01–0.06 units/min (vasodilatory shock)
Onset of action:	Peak effect within 15 min
Dilution:	Dilute 50 units with 250 mL of 0.9% normal saline to get 0.2 units/mL concentration or dilute 100 units with 100 mL of 0.9% normal saline to get 1 unit/mL.
Half-life:	10–20 min; eliminated by liver and kidney
Hemodynamic effects:	Increases SVR
Clinical usage:	Vasodilatory catecholamine-resistant shock; vasoplegia syndrome in patients who are hypotensive after weaning from cardiopulmonary bypass; diabetes insipidus; upper gastrointestinal bleed
Side effects:	Angina pectoris, atrial fibrillation, bradycardia

Clinical Pearls

1. There are no vasopressin receptors in the brain or lung vasculature. Therefore, vasopressin is a good option in patients with pulmonary hypertension who are catecholamine depleted and do not respond to ephedrine. Vasopressin does not increase pulmonary vascular resistance.
2. Effective in hypotensive patients on ACE inhibitors.
3. High risk for intestinal ischemia complications.
4. May decrease urine output.

Angiotensin II (Giapreza)

Angiotensin II is a naturally occurring protein hormone.

Mechanism of action:	Raises blood pressure by two mechanisms: direct action on the adrenal cortex, increasing aldosterone secretion; and activation of angiotensin II type 1 receptor on vascular smooth muscle
Dose:	Bolus: no bolus Infusion dose: initial – 20 ng/kg/min IV. Titrate every 5 min by increments of up to 15 ng/kg/min to achieve target blood pressure; limit the dose to 80 ng/kg/min in the first 3 h of treatment.
Dilution:	Dilute 2.5 mg vial with 250 mL of 0.9% normal saline to achieve a concentration of 10,000 ng/mL
Duration of action:	Short duration of action, metabolized by aminopeptidase A and angiotensin-converting enzyme in plasma and erythrocytes
Half-life:	< 1 min
Hemodynamic effects:	Increases SVR and aldosterone release, thus raising BP
Clinical usage:	Septic or distributive shock
Side effects:	Thromboembolic events, hyperglycemia, peripheral ischemia

Clinical Pearls
Angiotensinogen is a precursor protein produced in the liver and cleaved by renin to form angiotensin I. Angiotensin I is then converted to angiotensin II by angiotensin-converting enzyme (ACE) present in the renal endothelium, lungs, and capillary endothelium (see Fig. 2.8).

Inotropic Support Therapies

Inotropes are medications that increase myocardial contractility. In this section, we discuss the inotropic pharmacologic agents that are used in the management of patients with acute heart failure during cardiac surgery and/or low cardiac output syndrome after cardiac surgery. Low cardiac output syndrome has an incidence of about 10% for isolated coronary artery bypass graft or valve surgery to 27% for combined coronary artery bypass graft and mitral valve surgery. Inotropic support in cardiac surgery may begin before terminating bypass in cases of long cross-clamp periods, preexisting left ventricular (LV) dysfunction, LV hypertrophy, recent infarction, or incomplete revascularization. Support should be maintained for at least 6–8 h or until cardiac contractility and cardiac output improve. Inotropes are also used to maintain hemodynamics in off-pump surgery.

Mechanisms of Action

There are four classes of inotropes with different mechanisms of action:

1. Calcium and calcium-releasing agents. These agents lead to an increase in cytosolic calcium, which improves calcium-dependent myocardial contractility.
   (a) Adrenergic agents: activation of $beta_1$-adrenergic receptors leads to the formation of cyclic adenosine monophosphate (cAMP), which increases intracellular calcium.
   (b) Phosphodiesterase (PDE) inhibition agents: cAMP is metabolized in the cell by the PDE enzyme. Agents that inhibit this enzyme indirectly increase the availability of intracellular calcium (e.g., PDE III inhibitors, such as milrinone).
   (c) Sodium-potassium ATPase inhibition: Agents that inhibit this pump cause an increase in intracellular sodium, which leads to an increase in intracellular calcium (e.g., digoxin).
2. Calcium sensitizers. These agents increase the responsiveness of receptors to calcium (e.g., levosimendan, a calcium synthesizer/K^+-ATP activator that is not yet approved for use in the United States).
3. Cardiac myosin activators. These agents enhance the actin-myosin cross-bridge formation, thus increasing the force generated by cross bridges. The agents also

lengthen the duration of the contraction (e.g., omecamtiv mecarbil, which is not approved in the United States).
4. Lusitropic drugs. These drugs promote diastolic cardiac relaxation through activation of the intracellular calcium uptake system or sarco/endoplasmic reticulum Ca^{2+}-ATPase SERCA2a (e.g., istaroxime, which is not approved in the United States).

Positive inotropic agents commonly used in the cardiac operating room:

1. Calcium
2. Catecholamines
   (a) Dopamine
   (b) Dobutamine
   (c) Dopexamine
   (d) Epinephrine
   (e) Isoproterenol
   (f) Norepinephrine
3. Calcium sensitizers: Levosimendan
4. Cardiac myosin activators: Omecamtiv
5. Cardiac glycosides: Digoxin
6. PDE III inhibitors:
   (a) Enoximone
   (b) Piroximone
   (c) Milrinone
   (d) Amrinone
7. Glucagon

Calcium Chloride ($CaCl_2$); Calcium Gluconate(Kalcinate)

Two forms of intravenous calcium are available: 10% calcium chloride and 10% calcium gluconate.

Mechanism of action:	The electrolyte calcium increases the extracellular and intracellular ionized calcium concentrations. Calcium is the final common pathway necessary to trigger muscle contraction; thus, it may increase periphral vascular resistance (PVR) and cardiac contractility and enhance the effect of inotropic agents.
Dose:	0.5–1 g (5–10 mL of a 10% solution) given IV over a few minutes. IV bolus can cause profound hypertension.
Onset of action:	Immediate onset, peaking at 5 min; eliminated by the gastrointestinal tract and kidney
Duration of action:	10–20 min
Hemodynamic effects:	Increases SVR, MAP, and cardiac output
Clinical usage:	Hypocalcemia associated with hemodilution from priming the cardiopulmonary bypass (CPB) circuit; hyperkalemia, which is a common problem during CPB (secondary to impaired urinary potassium excretion and the use of hyperkalemic cardioplegia solutions); hypomagnesemia; and calcium channel blocker toxicity
Side effects:	Potentiates ischemia-reperfusion injury

Clinical Pearls

1. Calcium chloride is irritating and can cause tissue and skin necrosis with extravasation at the site of injection; thus, administration through a central line is preferred. Calcium gluconate does not cause skin irritation and can be given in a peripheral IV.
2. Calcium chloride has three times more elemental calcium than does calcium gluconate.

Catecholamines

Dopamine, norepinephrine, and epinephrine are endogenous catecholamines, produced by the adrenal medulla. The sequence of synthesis is tyrosine to L-dopa ➔ dopamine ➔ norepinephrine ➔ epinephrine.

Types of Adrenergic Receptors

Alpha-Adrenergic Receptors

(a) $Alpha_1$-adrenergic effects
  (i) Vascular smooth muscle contraction
(b) $Alpha_2$-adrenergic effects
  (i) Vascular smooth muscle relaxation

Beta-Adrenergic Receptors

(a) $Beta_1$-adrenergic effects
  (i) Inotropy (improved cardiac contractility)
  (ii) Chronotropy (increased heart rate)
(b) $Beta_2$-adrenergic effects
  (i) Vasodilation
  (ii) Bronchodilation
(c) $Beta_3$-adrenergic effects
  (i) Enhancement of lipolysis in adipose tissue
  (ii) Thermogenesis in skeletal muscle
  (iii) Urinary bladder relaxation

Dopaminergic Receptors

(a) DA_1 – Mediates vasodilation in kidney, intestine, and heart
(b) DA_2 – Located presynaptically. Inhibits the release of norepinephrine and dopamine. Associated with the antiemetic action of droperidol

Epinephrine HCl (Adrenaline)

Epinephrine is a naturally occurring sympathomimetic catecholamine produced in the adrenal medulla.

Mechanism of action:	At lower doses, beta effects predominate, leading to increased heart rate. As the dose increases, alpha receptor effects dominate, resulting in vasoconstriction.
Dose:	Bolus: 4–10 μg Infusion dose: 1–8 μg/min (0.01–0.05 μg/kg/min) Cardiac arrest: 1 mg every 3–5 min
Onset of action:	Immediate onset
Duration of action:	Short duration, peak at 5 min
Half-life:	2 min; rapid metabolism and neuronal uptake, as it is metabolized by catechol-O-methyltransferase and MAO
Hemodynamic effects:	Increases cardiac output and heart rate at low doses (0.01–0.03 μg/kg/min); increases SVR, with higher doses potentially leading to decreased cardiac output because of increased myocardial afterload
Clinical usage:	Bradyarrhythmia: improving atrial responsiveness to pacing at the conclusion of bypass (stimulation of the sinus node) Bronchospasm: Epinephrine has a robust bronchodilator action and can be used after weaning from CPB to treat bronchospasm. Anaphylactic shock: can be used to treat protamine-induced hypotension Cardiogenic shock: In cardiac surgery, epinephrine can be used to support and improve cardiac output. Administration can begin before terminating bypass in cases of long cross-clamp time, preexisting LV dysfunction, LV hypertrophy, recent infarction, and incomplete revascularization. Drug of choice in heart transplant patients with denervated heart. Works directly on $beta_1$ receptors of the denervated heart, increasing the BP and heart rate
Side effects:	Tachycardia and tachyarrhythmia Increases myocardial work and oxygen consumption; can induce myocardial ischemia Associated with metabolic acidosis, hyperglycemia, hypokalemia, and increased lactate level Severe vasoconstriction with high dose can cause bowel ischemia, pulmonary hypertension, and acute kidney injury.

Clinical Pearls

1. Correction of acidosis is vital, as acidosis decreases epinephrine's effectiveness.
2. Combination of low-dose epinephrine and low-dose milrinone is synergistic, while decreasing the side effects of both drugs.

Dopamine (Intropin)

Dopamine is an endogenous sympathomimetic catecholamine and immediate precursor to norepinephrine.

Mechanism of action:	Dopamine is a positive inotropic and chronotropic agent. It stimulates the adrenergic receptors directly and indirectly. At low doses, dopamine stimulates DA_1 in the central nervous system and renal vascular beds. With moderate doses, it shifts to stimulate $beta_1$-adrenergic receptors in the heart, and then alpha1-adrenergic receptors at high doses.
Dose:	1–20 μg/kg/min continuous infusion
Onset of action:	5 min after initiating IV infusion
Duration of action:	Short, less than 10 min
Half-life:	2 min; through rapid metabolism and neuronal uptake; metabolized by catechol-O-methyltransferase and MAO
Hemodynamic effects:	Dose-dependent Low dose: 1–4 μg/kg/min – increased renal blood flow and urine output Moderate dose: 4–10 μg/kg/min – beta-adrenergic effects with positive inotropic and chronotropic effects, where heart rate, cardiac contractility, cardiac output, and blood pressure increase High dose: >10 μg/kg/min – considered a vasopressor, where alpha-adrenergic effects begin to predominate, with vasoconstriction and increased BP
Clinical usage:	Hemodynamic instability in shock, especially septic shock Cardiogenic shock and low cardiac output syndrome after cardiac surgery Bradycardia
Side effects:	Tachycardia and tachyarrhythmia Increases myocardial work and oxygen consumption; can induce myocardial ischemia Severe vasoconstriction with high-dose dopamine infusion can cause limb ischemia. Symmetrical peripheral gangrene is known as purpura fulminans.

Clinical Pearls

1. Contraindications include pheochromocytoma, uncorrected tachyarrhythmias, or ventricular fibrillation.
2. Extravasation of dopamine during peripheral IV administration can lead to tissue necrosis and should be treated with local injection of diluted phentolamine 5 to 10 mg.

Dobutamine (Dobutrex)

Dobutamine is a synthetic derivative of isoproterenol.

Mechanism of action:	Dobutamine is a positive inotropic and chronotropic medication. It stimulates $beta_1$ in the heart and $beta_2$ in the vascular smooth muscle adrenergic receptors. Unlike dopamine, it matches higher myocardial oxygen demand by increasing coronary blood flow.
Dose:	1–20 μg/kg/min (initial dose is usually 2–5 μg/kg/min)
Onset of action:	Within 5 min after intravenous infusion
Duration of action:	Short duration, 3–5 min
Half-life:	2 min; metabolized in the liver by catechol-O-methyltransferase
Hemodynamic effects:	Decreases SVR and reduces LV afterload, decreases mean arterial pressure, increases cardiac output and heart rate, exerts a moderate pulmonary vasodilator effect, reduces LV wall stress
Clinical usage:	Hemodynamic instability (shock and low cardiac output heart failure) with a mild elevation in SVR; dobutamine stress echocardiography for the detection of functional coronary artery stenosis
Side effects:	Dose-dependent tachycardia and tachyarrhythmia; theoretically, it can cause coronary steal; decreases mean arterial pressure if the increase in cardiac output does not offset the decrease in SVR; tachyphylaxis if used after several days of a continuous infusion

Clinical Pearls

1. Dobutamine is contraindicated in patients with hypertrophic cardiomyopathy and diastolic dysfunction.
2. Alkalinization inactivates catecholamines, including dobutamine, so avoid injecting sodium bicarbonate or any alkaline solutions in the same intravenous line.
3. Dobutamine has little vasoconstrictor activity, so the risk of extravasation and skin necrosis is low. Thus, it can be infused in a peripheral IV.
4. Synergistic effect in improving cardiac output with the use of milrinone.

Isoproterenol (Isuprel)

Isoproterenol is a synthetic sympathomimetic.

Mechanism of action:	Isoproterenol is a positive inotropic and chronotropic agent through nonselective beta-adrenoceptor agonism.
Dose:	Bolus: 1–4 μg Infusion: 1–10 μg/min (0.01–0.06 μg/kg/min) titrated to heart rate and BP goals
Onset of action:	1–5 min after IV infusion
Duration of action:	10–15 min

Half-life:	2.5–5 min; drug is conjugated in hepatic and pulmonary tissue.
Hemodynamic effects:	Isoproterenol decreases SVR, increases cardiac output and heart rate, and decreases pulmonary vascular resistance.
Clinical usage:	Refractory torsades de pointes (a type of polymorphic ventricular tachycardia that can be fatal); refractory symptomatic bradycardia (heart block) or beta-blocker overdose; refractory electrical storm (3 or more sustained episodes of ventricular tachycardia or ventricular fibrillation); post-heart transplantation for heart rate optimization; Brugada syndrome (ST-segment elevation in the right precordial leads of ECG associated with an increased chance of sudden cardiac death due to ventricular fibrillation without apparent structural heart disease); bronchospasm during anesthesia; right ventricular dysfunction
Side effects:	Dose-dependent arrhythmia; coronary steal; hypoxia, by increasing ventilation-perfusion mismatch; reduced MAP

Calcium Sensitizers

Levosimendan (Simdax)

Levosimendan is a positive inotropic agent with ATP-dependent potassium-channel-opening and calcium-sensitizing effects. It improves myocardial contraction and improves relaxation during diastole, which aids ventricular filling. Levosimendan has not been approved by the Food and Drug Administration for use in the United States.

Cardiac Myosin Activators

Omecamtiv mecarbil

Omecamtiv mecarbil is a first-in-class cardiac myosin activator that increases the proportion of myosin heads that are tightly bound to actin and creates a force-producing state that is not related to cytosolic calcium accumulation. Phase I and II trials have been completed in patients with ischemic cardiomyopathy and acutely decompensated heart failure. Phase III studies are underway.

Cardiac Glycosides

Digoxin (Lanoxin)

Digoxin inhibits Na-K-ATPase, which results in increased intracellular calcium concentrations. It is indicated for supraventricular arrhythmias and exhibits positive inotropic effects, but it is rarely used due to its slow onset of action (4–6 h), narrow therapeutic index, and unpredictable dose-response dynamics.

Phosphodiesterase (PDE III) Inhibitors

Enoximone (Perfan)

Enoximone is a specific inhibitor of PDE III in cardiac and smooth muscle. Enoximone was recently evaluated in on-pump cardiac surgery (MOSEC study) and improved 30-day post-surgery renal function. Further studies are needed to validate these findings and delineate therapeutic utility.

Piroximone

Piroximone is a specific inhibitor of PDE III in cardiac and smooth muscle that was studied in animal models in the mid-1980s. This drug is not available in the United States.

Milrinone (Primacor)

Mechanism of action:	Non-catecholamine inodilator that inhibits PDE III and prevents cAMP breakdown, leading to increased intracellular calcium
Dose:	Bolus: 25–75 μg/kg Infusion: 0.375–0.75 μg/kg/min
Onset of action:	5–15 min after intravenous infusion
Duration of action:	2.7 h
Half-life:	2.3 h; 83% is unchanged in urine; 12% is metabolized in the liver by glucuronidation. Active tubular secretion is a major elimination pathway. Half-life is prolonged in renal failure.
Hemodynamic effects:	Decreases SVR, reduces myocardial oxygen demand, increases cardiac output and heart rate, decreases pulmonary vascular resistance, reduces LV end-diastolic pressure
Clinical usage:	Low cardiac output syndrome post-cardiac surgery, right ventricular dysfunction associated with high pulmonary vascular resistance
Side effects:	Decreased MAP; bronchospasm; torsades de pointes; cardiac arrhythmias; increased concentrations in renal dysfunction

Clinical Pearls

1. It has additive effects with epinephrine, dopamine, and dobutamine.
2. The patient's myocardial function should be monitored for a few hours after the drug is administered because of its long half-life.

Glucagon (GlucaGen)

Mechanism of action:	Glucagon has positive chronotropic and positive inotropic effects in low cardiac output failure after cardiac surgery.
Dose:	Bolus: 2–5 mg IV every 30–60 min Infusion: 1–10 mg/h
Onset of action:	5–10 min after IV bolus
Duration of action:	20–30 min for bolus
Half-life:	3–6 min; eliminated by liver and kidney
Hemodynamic effects:	Increases cardiac output, decreases pulmonary vascular resistance
Clinical usage:	Low cardiac output syndrome after cardiac surgery; severe hypoglycemia; gastrointestinal smooth muscle relaxation for endoscopy; treatment of beta-adrenergic blocker toxicity
Side effects:	Severe hyperglycemia; hypokalemic syndrome; contraindicated in pheochromocytoma, glucagonoma, diabetes mellitus, and insulinoma

Methylene Blue (Methylthionine Chloride, Urolene Blue)

Mechanism of action:	It inhibits nitric oxide synthase and guanylate cyclase, thus decreasing vascular smooth muscle relaxation.
Dose:	Bolus: 1–2 mg/kg IV injection slowly over 5 min; may repeat in 1 h
Onset of action:	Almost immediate
Duration of action:	30 min
Half-life:	5–6 h
Hemodynamic effects:	Increases MAP, SVR Increases pulmonary vascular resistance
Clinical usage:	Vasoplegia syndrome post CPB refractory to catecholamines
Side effects:	Methemoglobinemia at higher doses; hemolysis in patients with glucose-6-phosphate dehydrogenase deficiency

Vasodilator Drugs

Vasodilator drugs treat hypertension by decreasing systemic vascular resistance through many mechanisms. Arterial vasodilators reduce afterload, which results in increased cardiac output. Additionally, they improve myocardial oxygen demand dynamics. Venous dilators reduce preload; they are used to treat systemic hypertension if cardiac output is satisfactory. If the cardiac index is 2.0–2.2 L/min/m^2, accompanying inotropic support should be administered to improve myocardial function.

Nitroprusside (Nipride, Nitropress)

Mechanism of action:	Relaxes smooth vascular muscles by converting to nitric oxide, which increases cyclic guanosine methyl phosphatase (cGMP)
Dose:	Infusion: 0.5–10 μg/kg/min
Onset of action:	0.5–1 min
Duration of action:	1–10 min
Half-life:	3–6 min. Sodium nitroprusside is broken down in red blood cells to cyanide and nitric oxide. Cyanide is changed to thiocyanate in the liver by rhodanese.
Hemodynamic effects:	Reduced preload and afterload; decreased cardiac output; dose-dependent decrease in systemic vascular resistance and pulmonary vascular resistance; reflex tachycardia
Clinical usage:	Hypertensive emergencies
Side effects:	Prolonged administration leads to accumulation of thiocyanate and cyanide and resultant metabolic acidosis and methemoglobinemia; negative inotropes and inhaled anesthetics potentiate its hypotensive effects; severe renal or hepatic dysfunction increases the risk of thiocyanate and cyanide toxicity.

Clinical Pearls

1. Appropriate antidote therapy should be administered if thiocyanate and cyanide toxicities develop (hydroxocobalamin and methylene blue, respectively).
2. Dose reduction is indicated in patients with renal or hepatic disease.
3. Do not use in cases of compensatory hypertension (aortic coarctation, AV shunting), surgery in moribund patients, those with inadequate cerebral circulation, or defective or absent rhodanese.

Nitroglycerin (Tridil)

Mechanism of action:	Acts as a nitric oxide donor and exerts its vasodilator effects primarily in the venous system by increasing cGMP
Dose:	Bolus: dilute to 40 μg/mL; give 40–80 μg (1–2 mL) at a time. Infusion: 0.5–10 μg/kg/min Sublingual: 400–500 μg; may be used in the holding area if patient develops chest pain
Onset of action:	1 min
Duration of action:	1–10 min
Half-life:	1–3 min; eliminated in urine as inactive metabolites
Hemodynamic effects:	Coronary vasodilator, as it increases collateral coronary flow; venodilator that reduces preload and decreases cardiac output; improves diastolic function; improves subendocardial perfusion; decreases pulmonary vascular resistance
Clinical usage:	Perioperative hypertension; hypertension with myocardial ischemia or high filling pressures; angina and coronary spasm; prevention of radial artery spasm; pulmonary hypertension
Side effects:	Excessive or prolonged administration may result in tolerance or methemoglobinemia; increased pulmonary V/Q mismatch.

Clinical Pearls

1. Antidote therapy with methylene blue should be administered if methemoglobinemia develops.
2. Contraindicated in pericardial tamponade, constrictive pericarditis, restrictive cardiomyopathy, and in the presence of phosphodiesterase inhibitors.

Hydralazine (Apresoline)

Mechanism of action:	Direct arterial vasodilation
Dose:	Bolus: 10–40 mg IV every 4–6 h (perioperative); 5 mg every 15 min (intraoperative) Infusion: 1.5 μg/kg/min (intraoperative)
Onset of action:	10–80 min; peak effect: 20 min
Duration of action:	Up to 12 h depending on acetylator status; metabolites eliminated in the urine
Half-life:	3–7 h
Hemodynamic effects:	Acts as a direct arteriolar vasodilator, resulting in afterload reduction
Clinical usage:	For hemodynamically stable postoperative patients with hypertension when oral administration of an antihypertensive is not possible or when there is an urgent need to lower the BP
Side effects:	Rarely, hydralazine causes a systemic lupus erythematosus with glomerulonephritis syndrome, especially in slow acetylators. It can lead to coronary steal in patients with suspected coronary artery disease, and it can lead to angina attack and myocardial ischemia.

Phentolamine (Regitine, OraVerse)

Phentolamine is an alpha-blocker used in hypertension associated with pheochromocytoma and for the management of alpha agonist vasopressor extravasation. After stopping the vasopressor infusion, the catheter tip should be left in place so that aspiration of fluid from the extravasated area may be attempted. Ten milligrams of phentolamine is mixed in a 10-mL syringe, and 1 mL is injected into the catheter before removal.

Fenoldopam (Corlopam)

Mechanism of action:	DA_1 receptor agonist without adrenergic receptor agonist
Dose:	0.01–0.3 μg/kg/min IV infusion; may increase by 0.05–0.1 μg/kg/min every 15 min until target BP is achieved 0.03–0.1 μg/kg/min to optimize renal perfusion in patients with preoperative renal dysfunction
Onset of action:	10 min (adult)

Duration of action:	1 h
Half-life:	5 min
Hemodynamic effects:	Decreases BP without increasing heart rate or contractility; increases renal perfusion
Clinical usage:	Severe hypertension requiring prompt control to optimize renal perfusion in patients with preoperative renal dysfunction
Side effects:	Dose-related tachycardia, especially in doses >0.1 μg/kg/min; increase in intraocular pressure; hypokalemia (monitor potassium within 6 h of administration)

Clinical Pearls

1. Use caution in patients with coronary artery disease or ongoing angina pectoris, as it can increase myocardial oxygen demand due to dose-dependent tachycardia.
2. May transiently increase intraocular pressure (caution in patients with glaucoma).
3. Avoid concomitant use with beta-blockers due to their negative chronotropic effects.

Prostaglandin E2 (Iloprost)

Prostaglandin E2 dilates systemic and pulmonary arterial vascular beds. It has been used in cardiothoracic surgical patients who have pulmonary hypertension, hypoxemia, or right heart dysfunction to decrease mean pulmonary artery pressure without altering MAP.

Epoprostenol (Flolan, Veletri)

Epoprostenol is a naturally occurring prostacyclin. It dilates systemic and pulmonary arterial vascular beds and inhibits platelet aggregation. It is used for long-term intravenous treatment of primary pulmonary hypertension and, in the nebulized form, in cardiothoracic surgical patients with pulmonary hypertension and right heart dysfunction. The aerosolized dose is 3–50 ng/kg/min.

Nitric Oxide, inhaled (NO, iNO, INOmax)

Nitric oxide is a naturally occurring vasodilator made by vascular endothelial cells.

Mechanism of action:	Decreases vascular smooth muscle tone by increasing intracellular cGMP through activation of guanylate cyclase
Dose:	20–40 parts per million

Onset of action:	10 min (adult)
Duration of action:	1 h
Half-life:	5 min
Hemodynamic effects:	Lowers right ventricular afterload; selective pulmonary vasodilation
Clinical usage:	Pulmonary hypertension associated with acute right ventricular dysfunction post CPB; treatment of right ventricular failure after heart transplantation

Antiarrhythmics

Antiarrhythmic drugs are used to prevent and treat cardiac conduction abnormalities. Antiarrhythmic drugs are classified by mechanism of action into four groups (Table 3.1). They alter the velocity of conduction and the duration of the refractory period, and they suppress abnormal automaticity. Cardiac surgery patients often develop supraventricular arrhythmias, such as atrial fibrillation, whereas ventricular arrhythmias usually occur in the presence of injury to the cardiac muscle. Continuous electrocardiographic monitoring is necessary, especially during the parenteral administration of these drugs.

Sodium Channel Blocker

Procainamide (Pronestyl)

Procainamide is a class Ia antiarrhythmic that blocks sodium channels. It is the antiarrhythmic of choice in preexcitation syndromes with accessory pathways, such as Wolff-Parkinson-White syndrome. It is contraindicated in the prolonged QT syndrome.

Table 3.1 Vaughan-Williams classification of antiarrhythmic drugs according to their mechanism of action

Mechanism of action	Class	Drug
Fast sodium channel blocker	Ia	Procainamide
	Ib	Lidocaine
Beta-blocker	II	Metoprolol
Potassium channel blocker	III	Amiodarone
Calcium channel blocker	IV	Diltiazem
Other	N/A	Adenosine
		Magnesium
		Atropine/glycopyrrolate
		Digoxin

Lidocaine (Xylocaine)

Mechanism of action:	Class Ib antiarrhythmic that blocks sodium channels and decreases automaticity
Dose:	1–1.5 mg/kg (usually 100 mg dose)
Onset of action:	45–90 s
Duration of action:	10–20 min
Half-life:	90–120 min; eliminated by hepatic metabolism
Hemodynamic effects:	Therapeutic doses do not affect myocardial contractility and rarely reduce BP, but high doses depress myocardial contractility.
Clinical usage:	Indicated for ventricular arrhythmias and is the preferred medication in patients with ventricular arrhythmias that have a prolonged QT interval
Side effects:	Lidocaine central nervous system toxicity manifests as nausea, vomiting, mental status changes, and seizure.

Clinical Pearls

1. Lidocaine is contraindicated in patients with documented immune-mediated hypersensitivity to an amide local anesthetic.
2. Lidocaine has antiarrhythmic activity in ischemic myocardial tissue.
3. Contraindicated in Wolff-Parkinson-White and Stokes-Adams syndromes.

Beta-Blockers (Class II Antiarrhythmics)

Beta-blockers bind to beta-adrenergic receptors and have negative inotropic and chronotropic effects, and they slow AV conduction. Thus, they reduce BP and myocardial oxygen demand. Beta-blockers suppress renin activity and can reduce insulin release in hyperglycemia and mask initial symptoms of hypoglycemia, especially in labile diabetics. In cardiac surgery, indications include control of postoperative hypertension with a satisfactory cardiac output. Intravenous beta-blockers should be avoided when hypertension is accompanied by impaired cardiac output. Contraindications include cardiogenic shock, metabolic acidosis, hypotension, bradycardia, second- or third-degree heart block, sick sinus syndrome, and pheochromocytoma that has not been treated with alpha-blocking agents.

Atenolol (Tenormin)

Mechanism of action:	Selective beta$_1$ adrenoceptor antagonist
Dose:	Bolus: 2.5–10 mg IV push at a rate not to exceed 1 mg/min
Onset of action:	Effects are seen in 1 h with maximum effects in 2–4 h
Duration of action:	Up to 24 h
Half-life:	6–7 h; significantly prolonged in renal dysfunction

Hemodynamic effects:	Decreases BP by decreasing heart rate
Clinical usage:	In patients undergoing cardiac surgery, postoperative prevention of atrial fibrillation is important. Atenolol is used in hypertension, angina, acute myocardial infarction (with stable BP and heart rate), and tachyarrhythmias.
Side effects:	Hypotension, heart block, bradycardia, bronchospasm, heart failure

Esmolol (Brevibloc)

Mechanism of action:	Cardioselective $beta_1$ receptor blocker with a very short duration of action
Dose:	Bolus: 150 μg/kg over 30 s; then start infusion at 50 μg/kg/min; titrate to a maximum dose of 300 μg/kg/min for desired heart rate
Onset of action:	30 s
Duration of action:	10–30 min; metabolized by erythrocyte cholinesterase
Half-life:	2–9 min
Hemodynamic effects:	Decreases BP by decreasing heart rate
Clinical usage:	Perioperative hypertension; treatment of supraventricular tachycardia; atrial fibrillation (ventricular rate control); acute myocardial ischemia
Side effects:	Hypotension, heart block, bradycardia, bronchospasm, heart failure

Clinical Pearls

1. Contraindications include sinus bradycardia and heart block greater than first degree. Use with extreme caution in hypertensive patients who have marginal cardiac output.
2. Esmolol competitively prolongs neuromuscular blockade by succinylcholine due to the cholinesterase metabolism of both drugs.

Labetalol (Normodyne, Trandate)

Mechanism of action:	Selective $alpha_1$-adrenergic blocker and nonselective $beta_1$, $beta_2$-adrenergic blocker with a ratio of 1:7 IV
Dose:	Bolus: 5–10 mg IV push Infusion: 1–4 mg/min
Onset of action:	Rapid onset of action with maximal effects after 5 min
Duration of action:	2–6 h
Half-life:	6–8 h
Hemodynamic effects:	Negative inotropic and chronotropic effects; therefore, reduces BP
Clinical usage:	Used as a long-acting antihypertensive medication
Side effects:	Contraindications include overt cardiac failure, greater than first-degree heart block, and severe bradycardia.

Clinical Pearls

Labetalol is the only intravenous beta-blocker with alpha blocking activity.

Metoprolol (Lopressor)

Mechanism of action:	Selective beta$_1$ receptor blocker
Dose:	Bolus: 2.5–5 mg IV push
Onset of action:	1–5 min
Duration of action:	IV: 5–8 h
Half-life:	3–4 h; prolonged to 7–9 h in those with poor CYP2D6 metabolism
Hemodynamic effects:	Decreases heart rate and therefore BP
Clinical usage:	Intravenous use is indicated with myocardial ischemia and slowing of the ventricular response to atrial arrhythmias.
Side effects:	Hypotension, heart block, bradycardia, bronchospasm, heart failure

Potassium Channel Blocker (Class III Antiarrhythmic)

Amiodarone (Cordarone)

Mechanism of action:	Amiodarone is a class III antiarrhythmic with many pharmacodynamic effects. Amiodarone blocks sodium and potassium channels and exhibits calcium channel and adrenergic-blocking effects. Amiodarone prolongs the action potential by delaying repolarization.
Dose:	Malignant ventricular arrhythmia: 150 mg IV push followed by an infusion of 1 mg/min x 6 h Pulseless ventricular arrhythmia: 300 mg IV push
Onset of action:	Rapid control of ventricular rates; rhythm control requires higher cumulative doses.
Duration of action:	A bolus of amiodarone is rapidly distributed out of the intravascular space, resulting in a duration of action that is significantly shorter than the pharmacologic half-life (approximately 10–15 min).
Half-life:	IV single dose: mean range 9–36 days
Hemodynamic effects:	Slows conduction throughout the sinoatrial and atrioventricular nodes, resulting in negative chronotropic and dromotropic (conduction speed) effects
Clinical usage:	Indicated for the treatment of atrial fibrillation, polymorphic ventricular tachycardia without a prolonged QTc, and ventricular fibrillation
Side effects:	Prolonged QT, hypotension, atrioventricular block

Clinical Pearls

Hypotension associated with amiodarone administration may be mitigated by lengthening the infusion time.

Calcium Channel Blockers (Class IV Antiarrhythmics)

Nicardipine (Cardene)

Mechanism of action:	Inhibits calcium influx into vascular smooth muscle cells, including coronary arteries; no sinoatrial/atrioventricular nodal activity
Dose:	5 mg/h IV infusion; titrate by 2.5 mg/h increments every 5–15 min to a maximum infusion of 15 mg/h.
Onset of action:	1–2 min
Duration of action:	8 h; upon discontinuation of continuous infusion, a 50% reduction in effect occurs after 30 min, with a gradual reduction in effect over 50 h.
Half-life:	Alpha half-life of 3 min, with a rapid early distribution phase, an intermediate phase; beta half-life of 45 min; and terminal half-life of 14 h
Hemodynamic effects:	Reduces BP without negative inotropic effects or influence on sinoatrial or atrioventricular nodal conduction
Clinical usage:	Postoperative hypertension in the presence of reduced ventricular function; prevention of radial artery spasm if used in coronary artery bypass graft as an arterial conduit; coronary vasodilator
Side effects:	Hypotension, reflex tachycardia, vasodilation and flushing, headache

Clinical Pearls

1. Nicardipine is contraindicated in patients with advanced aortic stenosis.
2. Use with caution in patients with angina and heart failure.
3. Drug should be administered through large peripheral veins or central veins. Change site every 12 h to reduce the risk of venous thrombosis and local irritation.
4. Do not dilute with sodium bicarbonate or lactated Ringer's solution.

Clevidipine (Cleviprex)

Mechanism of action:	Short-term calcium channel blocker; inhibits calcium influx into vascular smooth muscle cells, producing a substantial reduction in BP; no sinoatrial/atrioventricular nodal activity
Dose:	1–2 mg/h, doubling the dose at 90 s intervals to achieve BP goal. Usual maintenance dose: 4–6 mg/h; maximum 21 mg/h
Onset of action:	1–4 min
Duration of action:	5–15 min
Half-life:	Biphasic: initial, 1 min; final, 15 min
Hemodynamic effects:	Potent BP reduction; no effect on heart rate
Clinical usage:	Emergency control of BP
Side effects:	Hypotension, atrial fibrillation, nausea, fever

Clinical Pearls

1. For every 1–2 mg/h increase in dose, an approximate reduction of 2–4 mm Hg in systolic blood pressure may occur.
2. Drug metabolism and elimination are not affected by hepatic or renal disease, and there are no significant drug-drug interactions.
3. Use with caution in patients with angina and heart failure.
4. Formulated in an oil-in-water emulsion containing 200 mg/mL of lipid (2 kcal/mL); contains soybean oil, egg yolk, phospholipid, and glycerin; contraindicated in patients with hypersensitivity to any of the components.

Diltiazem (Cardizem)

Mechanism of action:	Benzothiazepine calcium channel blocker that acts by preventing the influx of calcium into the slow channels of vascular smooth muscle and myocardium during depolarization
Dose:	Bolus: 0.25 mg/kg over 2 min. If no adequate response is noticed after 15 min, a repeat bolus of 0.35 mg/kg over 2 min is given. Infusion: start at 5–15 mg/h IV, using a 125 mg/125 mL mix for up to 24 h.
Onset of action:	3 min (IV)
Duration of action:	1–3 h (IV bolus); 0.5–10 h (after discontinuation of continuous infusion)
Half-life:	3–4 h (IV bolus); 4–5 h (continuous infusion). Administration with midazolam or triazolam can increase effects.
Hemodynamic effects:	Reduces BP; reduces heart rate
Clinical usage:	Used for slowing the ventricular response to atrial fibrillation and flutter (pacemaker backup must be readily available); treatment of systemic hypertension; prevention of arterial graft spasm and radial artery and coronary artery spasm
Side effects:	Arrhythmia, atrioventricular block, hypotension

Clinical Pearls

1. Avoid in cases with marginal cardiac output due to effects on slowing heart rate.
2. Contraindicated in patients with sick sinus syndrome, second- or third-degree AV block (except if functioning ventricular pacemaker is present), hypotension SBP <90 mm Hg, and ventricular tachycardia.
3. Concomitant use with beta-blockers, digoxin, or clonidine can lead to additive effects on cardiac conduction. Avoid administration in proximity of other conduction-altering agents.

Endogenous Nucleoside

Adenosine (Adenocard)

Mechanism of action:	Adenosine is a nucleoside that acts by slowing the conduction through the atrioventricular node.
Dose:	Bolus: 6 mg rapid IV push over 1–2 s followed immediately by a saline flush; may repeat 12 mg twice within 1–2 min
Onset of action:	10–20 s
Duration of action:	3–7 s
Half-life:	Metabolized in blood in less than 10 s
Hemodynamic effects:	Reduces BP and heart rate
Clinical usage:	Used for treatment and conversion to sinus rhythm of paroxysmal supraventricular tachycardias with atrioventricular reentry
Side effects:	Adenosine can produce a short heart block; do not give the second dose if a high-grade block develops after the first dose.

Electrolytes

Magnesium Sulfate

Hypomagnesemia is common after CPB due to dilution and diuresis and is associated with an increased risk of cardiac arrhythmias. While the efficacy of magnesium for the prevention of supraventricular and ventricular arrhythmias is controversial, the administration of magnesium at the conclusion of CPB (2 g over 15 min) and on the first morning after surgery is common practice, given the detrimental effects of postoperative hypomagnesemia. In patients with torsades de pointes, administer magnesium sulfate 25–50 mg/kg IV push.

Potassium Chloride (KCl)

Mechanism of action:	Crucial for resting membrane potential and action potential that are necessary for myocardial contraction and nerve conduction
Dose:	Infusion: 20–40 mEq IV over 30–60 min
Onset of action:	Immediate
Duration of action:	Variable
Half-life:	Variable; depends on the kidney for elimination
Hemodynamic effects:	At high concentration, decreases atrioventricular conduction and myocardial contractility
Clinical usage:	Prevention and treatment of arrhythmias associated with hypokalemia (serum potassium <3.5 mEq/L)
Side effects:	Phlebitis in the peripheral IV, so central line administration is preferable; rapid administration may cause cardiac arrest in diastole; caution in end-stage renal disease, as it accumulates in the circulation.

Clinical Pearls

1. Metabolic and respiratory alkalosis exacerbate hypokalemia, with intracellular shift of potassium. There is a tendency to hyperventilate the patient with the Ambu bag, causing respiratory alkalosis during transport to the intensive care unit; caution is warranted since hypokalemic patients can develop a serious cardiac arrhythmia.
2. Potassium should be administered to hypovolemic patients only after hydration and diuresis are established.
3. Chronological ECG changes of hyperkalemia (serum potassium >5.5 mEq/L): tall tented T-waves, prolongation of PR interval, widening QRS complex, flattening and disappearance of P-waves, development of a sine wave pattern, and, finally, asystolic cardiac arrest.

Anticholinergic

Atropine sulfate (Atropine)

Atropine is an anticholinergic antimuscarinic agent.

Mechanism of action:	It antagonizes the muscarinic actions of acetylcholine at smooth muscle parasympathetic locations, salivary glands, and the central nervous system. It stops the effects of acetylcholine on the sinoatrial and atrioventricular nodes.
Dose:	Bolus: 0.01 mg/kg IV (usually 0.5–1 mg IV); can be administered intramuscularly
Onset of action:	1–4 min
Duration of action:	15–30 min
Half-life:	3–4 h; metabolized by the liver and excreted renally
Hemodynamic effects:	Low dose may cause bradycardia, but higher dose produces tachycardia.
Clinical usage:	Indicated for treatment of bradyarrhythmia; sinus bradycardia if atrioventricular pacing wires fail or were not placed at the end of cardiac surgery; adjunct to the reversal of neuromuscular blockade; bronchodilation
Side effects:	Acute glaucoma; pyloric obstruction. In patients with prostatic hypertrophy, it can lead to complete urinary retention. Central anticholinergic syndrome is associated with delirium, stupor, and coma.

Glycopyrrolate (Robinul)

Glycopyrrolate is an anticholinergic antimuscarinic agent with a longer duration than atropine. It does not cross the blood-brain barrier.

Mechanism of action:	Antagonizes the muscarinic actions of acetylcholine at smooth muscle parasympathetic locations and salivary glands
Dose:	0.1–0.2 mg/dose IV every 2–3 min; can be administered intramuscularly
Onset of action:	Within 1 min
Duration of action:	2–3 h as a vagal blockade; 7 h as anti-sialagogue
Half-life:	0.5–1.5 h
Hemodynamic effects:	Tachycardia
Clinical usage:	Bradyarrhythmia; premedication as anti-sialagogue; adjunct to the reversal of neuromuscular blockade
Side effects:	Severe allergic reactions, mental confusion, urinary retention

Cholinergic

Neostigmine (Prostigmin)

Neostigmine, a reversal agent of non-depolarizing neuromuscular blocking agents, can be used in cardiac surgery to slow the heart rate in supraventricular tachycardia without affecting myocardial contractility, especially in patients coming off CPB. In this situation, caution should be exercised (pacing wires at the time of administration of neostigmine should be functional) as neostigmine might cause severe bradycardia or asystole. Also, when using neostigmine to slow the HR, redosing of neuromuscular blocking agents is necessary.

Diuretics

Numerous elements play a role in the development of kidney injury during CPB, including exposure to nephrotoxins, either exogenous from administered drugs or endogenous from iron or heme pigments released from traumatized red blood cells. Also, factors such as ischemia-reperfusion injury, embolization, hemodynamic perturbation, neurohormonal activation, non-pulsatile flow, and the activation of the systemic inflammatory response may impair renal function. Oliguric acute kidney injury is associated with more severe injury than is nonoliguric acute kidney injury. However, intraoperative diuretic administration in oliguric states does not reduce the risk of postoperative renal dysfunction, although diuretics can be used in the short term for volume control through their effects on urine output. Diuretics decrease preload and reduce vascular congestion. Given the potential for adverse effects, use of these medications should be reserved for compelling indications, such as hyperkalemia or significant volume overload.

Loop Diuretic

Furosemide (Lasix)

Mechanism of action:	Furosemide prevents sodium and chloride reabsorption in the loop of Henle and proximal and distal tubules by slowing or stopping the action of the sodium-potassium-chloride transport protein (symporter).
Dose:	Incremental doses from 10 mg IV (max 200 mg/dose). Consider higher initial doses in patients on chronic diuretic therapy or with poor renal perfusion.
Onset of action:	5 min; peak effect at 30 min
Duration of action:	1.5–2 h
Half-life:	0.5–2 h; prolonged up to 9 h in end-stage renal disease
Hemodynamic effects:	Hemodynamic effects should be minimal, although excessive diuresis resulting in hypovolemia may cause hypotension.
Clinical usage:	Increases urine output in severe volume overload, increases potassium excretion in hyperkalemia
Side effects:	Electrolyte disturbances: hypokalemia, hypomagnesemia; ototoxicity

Clinical Pearls

1. The maximum concentration for IV administration is 10 mg/mL.
2. Infusion rate should not exceed 4 mg/min to minimize the risk of ototoxicity.

Osmotic Diuretic

Mannitol (Osmitrol)

Mechanism of action:	Mannitol is an osmotic diuretic that reduces cell swelling and tissue edema during hemodilution by increasing oncotic pressure. It is an oxygen-free radical scavenger that reduces ischemia-reperfusion injury in vital organs.
Dose:	Mannitol 25% solution – 12.5 g added to the CPB circuit prime
Onset of action:	Diuresis: 1–3 h
Half-life:	0.5–2.5 h; 6–36 h in renal failure
Hemodynamic effects:	Hypotension if given quickly
Clinical usage:	Often added to the CPB priming solution. It reduces cell swelling after cardioplegic arrest and improves urine output.
Side effects:	Exacerbation of congestive heart failure by expanding intravascular volume

Clinical Pearls

1. Do not use solutions that have visible crystals.
2. Administer via a ≤5-micron filter set to avoid administration of crystallized particles.
3. Crenation and agglutination of red blood cells may occur if mannitol is administered with whole blood.

Anticoagulants, Prothrombotics, and Pharmacological Blood Conservation

Normal coagulation is a delicate equilibrium between the procoagulant pathway, which is responsible for thrombus formation at the damaged or exposed surface of the vascular system, and the processes that keep thrombus from forming elsewhere. There is a high incidence of hematological complications in the perioperative period of cardiac surgery because of the imbalance of the coagulation system. The goal is to prevent and treat coagulopathies. Many patients are at risk for developing coagulopathy, which leads to increased perioperative bleeding. The causes of coagulopathy can be either patient-related or procedure-related. Causes of patient-related coagulopathies are advanced age, preoperative anemia, liver disease, renal insufficiency, and antiplatelet or long-acting anticoagulant therapy at the time of surgery.

The use of hypothermic CPB necessitates full anticoagulation. Additionally, the hemodilution effects of coagulation factors and exposure of the blood elements to the artificial CPB circuit lead to platelet consumption and activation of the coagulation cascade, including fibrinolysis. The hematologic agents discussed in this section variably affect the coagulation cascade, either promoting or inhibiting coagulation.

Before the initiation of CPB, adequate anticoagulation must be accomplished and confirmed by a whole-blood test of anticoagulation, i.e., the activated clotting time. Activated clotting time goals vary, depending on the anticoagulant used and whether the patient is off-pump or on CPB. After the patient is weaned from CPB, anticoagulation must be reversed; heparin is fully reversed with the use of protamine sulfate. Newer drugs that reverse other anticoagulants are now approved by the Food and Drug Administration and commercially available. The risk of excessive perioperative bleeding for the cardiac surgical patient is high, and the anesthesia provider must have pharmacological strategies for blood conservation.

Anticoagulants:

- Heparin
- Bivalirudin
- Argatroban

Prothrombotic agents:

- Protamine
- Coagulation factors
  - Antithrombin III
  - Prothrombin complex concentrates
  - Factor VII
- Antifibrinolytic agents
  - Tranexamic acid
  - ε-Aminocaproic acid
- Reversal of direct oral anticoagulants
  - Idarucizumab
  - Andexanet alfa

Anticoagulants

Indirect Thrombin Inhibitors

Unfractionated Heparin

Mechanism of action:	Heparin binds and activates antithrombin III, causing inactivation of thrombin (factor II) and factor Xa. Heparin also prevents the conversion of fibrinogen to fibrin.
Dose:	300 units/kg prior to the initiation of CPB. Monitor and maintain target activating clotting time per institution standards for CPB and non-CPB operations.
Onset of action:	Immediate. Check activated clotting time 3 min after administration.
Duration of action:	4–6 h
Half-life:	1.5–2.5 h Eliminated via the liver and the reticuloendothelial system
Clinical usage:	Anticoagulation for CPB
Side effects:	Bleeding and heparin-induced thrombocytopenia (HIT), a life-threatening disorder caused by the formation of IgG antibodies to heparin and platelet factor 4

Direct Thrombin Inhibitors (DTIs)

Direct thrombin inhibitors are anticoagulants that inhibit both soluble thrombin and fibrin-associated thrombin. Consequently, they inhibit fibrin formation, activation of factors V, VIII, and XIII, protein C, and platelet aggregation. Since the discontinuation of lepirudin and desirudin, only bivalirudin and argatroban are available in the United States.

Bivalirudin (Angiomax)

Mechanism of action:	Direct thrombin inhibitor
Dose:	Bolus: 1 mg/kg initially Infusion (initiation of 2.5 mg/kg/h, targeting activated clotting time of more than 2.5 times of baseline). Fifty milligrams are added to the priming solution. Additional boluses of 0.1–0.5 mg/kg may be given to maintain an activated clotting time in the therapeutic range. Discontinue infusion 10–15 min prior to weaning from CPB.
Onset of action:	Immediate
Duration of action:	Coagulation times return to baseline about 1 h after the cessation of infusion.
Half-life:	Twenty-five minutes with normal renal clearance. Drug is eliminated by enzymatic deactivation in plasma.
Clinical usage:	Anticoagulation in patients who cannot be treated with heparin (e.g., HIT)
Side effects:	Bleeding

Argatroban (Acova)

Mechanism of action:	Direct thrombin inhibitor
Dose:	Bolus: 6.5 mg initially (0.1 mg/kg) followed by infusion – 5–10 μg/kg/min, with a target activated clotting time more than 500 s. Dosing of argatroban as an alternative to heparin during CPB is associated with failure to provide adequate anticoagulation and clotting of the oxygenator. Argatroban should be restricted to circumstances where other thrombin inhibitors are contraindicated.
Onset of action:	Immediate
Duration of action:	Coagulation times return to baseline about 90 min after infusion.
Half-life:	Dose-dependent, 1.5–2.5 h; eliminated by hepatic metabolism
Clinical usage:	Anticoagulation in the setting of HIT
Side effects:	Bleeding

Antithrombin III (ATryn, Thrombate III)

Antithrombin III is a naturally occurring anticoagulant. Two types of antithrombin are available in the United States: human antithrombin III pooled from human serum (Thrombate III) and recombinant human antithrombin III (ATryn).

Mechanism of action:	Serine protease inhibitor binds to thrombin, XIIa, IXa, and XI to deactivate them.
Dose:	In patients with known antithrombin III deficiency prior to surgery, dosing is individualized according to baseline levels, using formulas provided in the package labeling to achieve antithrombin III levels at 120%. In patients with suspected heparin resistance with inadequate activated clotting time after repeated doses of heparin, antithrombin doses of 500–1000 units have been suggested.
Onset of action:	5 min
Duration of action:	7 min
Half-life:	Plasma-derived antithrombin III, 2.5–3.8 days Recombinant human antithrombin III, 12–18 h
Hemodynamic effects:	Reduces blood pressure; vasodilation
Clinical usage:	Acquired heparin resistance; treatment of hereditary antithrombin III deficiency; prophylaxis of deep-vein thrombosis or pulmonary embolism during surgical or obstetrical procedures
Side effects:	Viral infection; goat milk protein hypersensitivity

Clinical Pearls

In hospitals that do not stock antithrombin III concentrates, fresh-frozen plasma may be used to provide antithrombin III in suspected heparin resistance.

Antiplatelet Agent

Cangrelor (KENGREAL)

Cangrelor, a platelet P2Y12 inhibitor, is a rapidly acting intravenous antiplatelet drug, used in patients with HIT.

Mechanism of action:	Reversibly blocks ADP-induced platelet activation and aggregation
Dose:	Bolus: 30 μg/kg IV; 4 μg/kg/min continuous infusion for 2 h
Onset of action:	2 min after administration by IV bolus
Duration of action:	Normal platelet function is restored in 1 h.
Half-life:	3–6 min; deactivated in the circulation by dephosphorylation
Clinical usage:	Percutaneous coronary intervention
Side effects:	Bleeding, bronchospasm, anaphylactoid reactions, angioedema

Clinical Pearls

HIT results from the production of IgG antibodies directed to heparin-platelet factor 4 complex. A new option used as an alternative to anticoagulation with bivalirudin and argatroban is administration of cangrelor, which inhibits platelets; heparin then can be used for anticoagulation during CPB, since there will be no sites for the HIT antibody to attach.

Procoagulants

Protamine Sulfate

Mechanism of action:	Protamine sulfate is a highly positively charged basic protein molecule that combines with and neutralizes polyacidic negatively charged heparin molecules to form inactive salt aggregates. Historically, protamine sulfate was prepared from salmon sperm, but it is now produced via recombinant biotechnology.
Dose:	1–1.3 mg protamine reverses 100 units of heparin. Alternatively, dosing is calculated by a heparin-protamine titration device (Hepcon). Protamine should be given slowly after a test dose. The dose of protamine should not exceed a ratio of 2.6 mg protamine:100 units heparin, as higher doses can prolong impaired platelet function and paradoxically raise the activated clotting time.
Onset of action:	0.5–1 min
Duration of action:	2 h
Half-life:	Metabolized in blood in less than 10 s
Hemodynamic effects:	Reduces BP (hypotension from histamine release if administered quickly); reduces heart rate (bradycardia)
Clinical usage:	Heparin reversal
Side effects:	Four types of protamine reactions: 1. Hypotension from rapid administration, mediated by nonimmunologic histamine release and direct myocardial depression. Hypotension usually is transient and only necessitates brief support of BP. 2. True anaphylaxis is mediated by antiprotamine IgE antibody in patients sensitized to protamine. Treatment is early recognition followed by discontinuation of protamine and administration of diphenhydramine 25–50 mg IV, famotidine 20 mg IV, epinephrine 10–100 μg IV, hydrocortisone 100 mg IV, and an inhaled bronchodilator. 3. Anaphylactoid-like reactions are mediated by activation of the complement system and direct degranulation of mast cells. Anaphylactoid-like reactions are less dramatic than anaphylaxis, but treatment is the same. 4. Acute pulmonary vasoconstriction with pressure overload results in acute right ventricular failure, pulmonary edema, and circulatory collapse. This is the most severe clinical complication of protamine reactions and is mediated by a massive discharge of thromboxane A_2, in the lungs. Treatment is early recognition followed by discontinuation of protamine, full heparinization, and administration of epinephrine, nitroglycerin, and milrinone. If all fails, hand massage of the heart, re-cannulation, and resumption of bypass are indicated. Mechanical support should be considered. For subsequent CPB, direct administration of protamine into the aorta (bypassing the pulmonary circulation) or letting the heparin metabolize without heparin reversal can be considered.

Clinical Pearls

1. Heparin rebound occurs after adequate neutralization of heparin; 2–3 h after administration, protamine is cleared from the circulation, and non-neutralized heparin dissociated from plasma protein reappears in the circulation, causing anticoagulation and bleeding.
2. Patients who are allergic to fish, diabetics who have been previously treated with insulin preparations containing protamine, and vasectomized men have increased risk of protamine allergic reactions because of the presence of antiprotamine antibodies in the serum.

Prothrombin Complex Concentrates (Kcentra)

Prothrombin complex concentrate (PCC) is indicated for significant bleeding caused by vitamin K antagonist therapy (e.g., warfarin). There are two formulations on the market: 4-factor and 3-factor. All PCCs contain factors II, IX, and X. 4-factor PCCs contain therapeutic levels of factor VII as well as proteins S and C. Activated PCCs contain factor VII in the activated form. Unactivated 4-factor PCC is sometimes used off-label to treat patients with severe coagulopathic hemorrhage after cardiac surgery that involved CPB. PCC restores vitamin K-dependent clotting factors and promotes coagulation.

Mechanism of action:	Administration of 4-factor PCCs rapidly increases plasma levels of vitamin K-dependent coagulation factors II, VII, IX, and X and the anticoagulant proteins C and S.
Dose:	Use in cardiac surgery to a maximum of 50 units/kg or 5000 units
Onset of action:	Rapid; significant INR reduction within 10 min
Duration of action:	6–8 h
Half-life:	Depending on the clotting factor, it ranges from 1.5 to 60 h.
Hemodynamic effects:	Hypotension if given quickly
Clinical usage:	Often added to the CPB priming solution. It reduces cell swelling after cardioplegic arrest and improves urine output.
Side effects:	Thromboembolic complications; 4-factor PCC contains heparin and may provoke HIT.

Clinical Pearls

1. Potency of the available PCC agent is measured by the content of factor IX units.
2. 4-factor PCC contains heparin and is contraindicated in patients with known heparin-induced thrombocytopenia or heparin hypersensitivity.
3. Should not be administered in disseminated intravascular coagulopathy, hypercoagulable disease states, or with other prothrombotic agents, such as antifibrinolytic agents, as administration may predispose to thromboembolic complications.

Factor VII (Novoseven)

Mechanism of action:	rFVIIa attaches to the surface of activated platelets and promotes factor X activation and rapid thrombin generation from prothrombin on the activated platelet surface.
Dose:	Dosing not well-established; 35–70 μg/kg/dose has been recommended in non-prospective studies. Lower doses (10–20 μg/kg) may be preferred to reduce thromboembolic events in patients with left ventricular assist devices.
Dilution:	Reconstitute with histidine diluent provided by the manufacturer according to prescribing information.
Onset of action:	Immediate
Duration of action:	Up to 6 h
Half-life:	2.8–3.1 h
Clinical usage:	Consider off-label use for management of intractable nonsurgical bleeding that is unresponsive to routine hemostatic therapy after surgery involving CPB.
Side effects:	Thrombotic complications, including stroke

DESMOPRESSIN ACETATE (DDAVP)

Desmopressin is a synthetic analog of antidiuretic hormone produced in the posterior pituitary gland.

Mechanism of action:	Releases factors VIII and XII and von Willebrand factor from vascular endothelium and increases their levels by three- to fivefold
Dose:	Bolus: 0.3 μg/kg IV slowly over 20 min
Onset of action:	Within minutes; peak in 15–30 min
Duration of action:	4–12 h
Half-life:	Biphasic; initial is 7.8 min, and terminal is 75.5 min; renal elimination
Hemodynamic effects:	Increases MAP; tachycardia
Clinical usage:	Platelet dysfunction associated with uremia or recent antiplatelet agent administration in patients undergoing cardiac surgery to reduce bleeding and transfusion; von Willebrand disease; hemophilia A
Side effects:	Thrombotic events, water intoxication, hyponatremia, seizures, allergic reactions, and anaphylaxis

Antifibrinolytic Agents

Contact of blood with the extracorporeal surfaces during CPB initiates a total systemic inflammatory response characterized by activation of inflammation, coagulation, and fibrinolysis. Fibrinolysis occurs from the release of endothelial plasminogen activators and fibrin formation that leads to activation of plasmin. Plasmin then breaks down fibrin into fibrin degradation products. Use of prophylactic antifibrinolytic therapy is one approach that has been used to reduce nonsurgical-related

bleeding. Antifibrinolytics preserve clot integrity through inhibition of fibrinolysis by binding to plasminogen lysine binding sites. Antifibrinolytic agents include tranexamic acid and ε-aminocaproic acid. Both agents increase the risk of arterial and venous thrombotic events. Therefore, antifibrinolytics should not be administered in disseminated intravascular coagulopathy or with other prothrombotic agents, such as factor VIIa or PCC.

Aminocaproic Acid (Amicar)

Aminocaproic acid is a synthetic lysine analog.

Mechanism of action:	Stabilizes clot formation, as explained above
Dose:	Bolus: 100 mg/kg (typically 5–10 g) followed by 5 mg/kg added to CPB circuit priming solution Infusion: 10–15 mg/kg/h (typically 1–1.5 g/h) during cardiac surgery
Onset of action:	Immediate
Duration of action:	3–5 h
Half-life:	2 h; eliminated by kidneys
Clinical usage:	Treatment of bleeding associated with systemic hyperfibrinolysis during cardiac surgery
Side effects:	Increased risk of arterial and venous thrombotic events, hypotension, bradycardia, and renal failure. Contraindicated in disseminated intravascular coagulopathy or with other prothrombotic agents, such as factor VIIa or PCC

Tranexamic Acid (TXA, Cyklokapron)

Tranexamic acid is a synthetic lysine analog that is ten times more potent than ε-aminocaproic acid.

Mechanism of action:	Stabilizes clot formation, as explained above
Dose:	High dose: 30 mg/kg bolus, 2 mg/kg added to CPB circuit priming solution and continuous infusion of 16 mg/kg/h during surgery Low dose: 10 mg/kg bolus, 1–2 mg added to CPB circuit priming solution and continuous infusion of 1 mg/kg/h during cardiac surgery
Onset of action:	Immediate
Duration of action:	3–5 h
Half-life:	3 h; eliminated by renal clearance
Clinical usage:	Bleeding associated with systemic hyperfibrinolysis during cardiac surgery; spine surgery; trauma
Side effects:	Increased risk of arterial and venous thrombotic events, hypotension with rapid infusion, seizures, and renal failure. Contraindicated in disseminated intravascular coagulopathy or with other prothrombotic agents, such as factor VIIa or PCC

Aprotinin (Trasylol)

Aprotinin is a serine protease inhibitor. After a research trial (the BART study) reported increased mortality with aprotinin, the Food and Drug Administration issued a warning, and its use was discontinued in the United States.

Reversal of Direct Oral Anticoagulants

Patients taking oral anticoagulants may need urgent or emergent cardiac surgery. Reversal of anticoagulation is necessary to reduce bleeding and the need for transfusion. Two recently developed agents that are indicated for life-threatening bleeding due to direct oral anticoagulants are discussed here.

Idarucizumab (Praxbind)

Idarucizumab is a monoclonal antibody Fab fragment that is indicated for reversal of the oral direct thrombin inhibitor dabigatran (Pradaxa). Idarucizumab attaches to free and thrombin-bound dabigatran to neutralize its activity. The recommended dose of idarucizumab is 5 g. Side effects include thromboembolic risk and hypersensitivity reactions.

Andexanet alfa (Andexxa)

Andexanet alfa is a modified recombinant factor Xa protein approved for reversal of oral factor Xa inhibitors, including apixaban (Eliquis) and rivaroxaban (Xarelto). Andexanet alfa may be used off-label for reversal of edoxaban (Savaysa), betrixaban (Bevyxxa), and enoxaparin (Lovenox). Depending on the dose and time since the last administration of factor Xa inhibitor, a low-dose (400 mg IV bolus followed by a 4 mg/min continuous infusion for up to 2 h) or high-dose (800 mg IV bolus followed by 8 mg/min continuous infusion for up to 2 h) regimen may be indicated. Side effects include increased thromboembolic risk.

For common cardiac drips, please see Chap. 8.

Further Reading

1. Abbott TR. The use of glucagon following open heart surgery in children. Br J Anesth. 1972;44(8):854–8.
2. Bojar RM. Manual of perioperative care in adult cardiac surgery. Chichester: Wiley-Blackwell; 2011.
3. Cheng DCH, David TE, Cheng DC. Perioperative care in cardiac anesthesia and surgery. Philadelphia: Wolters Kluwer Health; 2015.
4. Goodman LS, Brunton LL, Chabner B, Knollmann BC. Goodman & Gilman's pharmacological basis of therapeutics. 12th ed. New York: McGraw-Hill; 2011. Print

5. Hemmings HC, Talmage E. Pharmacology and physiology for anesthesia. 2nd ed. Philadelphia: Elsevier; 2019. Print
6. Kazama T, Ikeda K, Morita K. Reduction by fentanyl of the Cp50 values of propofol and hemodynamic responses to various noxious stimuli. Anesthesiology. 1997;87:213.
7. MacKenzie M, Zed PJ, Ensom MH. Opioid pharmacokinetics-pharmacodynamics: clinical implications in acute pain management in trauma. Ann Pharmacother. 2016;50:209.
8. Shore-Lesserson L. The Society of Thoracic Surgeons, The Society of Cardiovascular Anesthesiologists, and The American Society of ExtraCorporeal Technology: clinical practice guidelines – anticoagulation during cardiopulmonary bypass. Ann Thorac Surg. 2018;105:650–62.
9. Short TG, Plummer JL, Chui PT. Hypnotic and anaesthetic interactions between midazolam, propofol, and alfentanil. Br J Anaesth. 1992;69:162.

Basic ECG and Common Arrhythmias for the Cardiac Surgery Patient

4

Matthew Ortman

Everyone has a plan until they get punched in the mouth.

—"Iron" Mike Tyson

Introduction

Arrhythmias are quite common during and following cardiac surgery, related either to direct manipulation of the cardiac conduction system or the electrolyte, metabolic, and ischemic alterations that can complicate the perioperative period. It is crucial that the treatment team have a clear understanding of what is benign, what is pathological, and what requires immediate intervention. Knowledge of the anatomy and electrophysiology of the conduction system is a prerequisite for the correct interpretation of 5- or 12-lead ECG.

The Conduction System of the Heart

The mechanical activity of the heart is dependent upon the initiation, propagation, and synchronicity of electrical signals through the specialized conduction system (Fig. 4.1).

1. *The anatomy of the conduction system*:
   - *Sinus node (SA)*: The sinoatrial node, or "pacemaker" of the heart, is a specialized cluster of cells with inherent automaticity that initiates the cardiac

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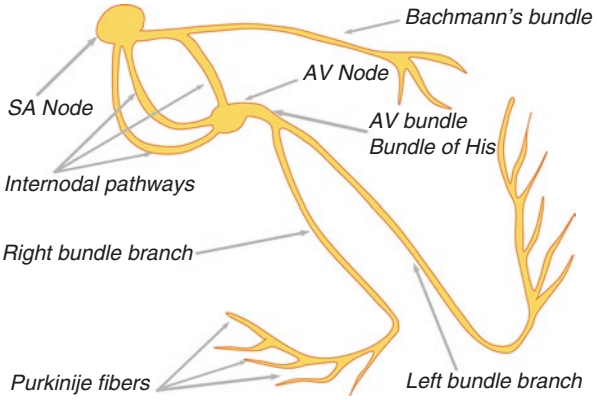


Fig. 4.1 The anatomy of the conduction system of the heart

cycle. It is located in the superior-posterior right atrium near its junction with the superior vena cava.

- *Atrial conduction channels*: The electrical impulse spreads inferiorly and leftward through the right atrium via internodal pathways and into the left atrium via Bachmann's bundle and direct muscular connections with the coronary sinus. The P wave on the ECG tracing is a reflection of atrial depolarization.
- *Atrioventricular node (AVN)*: The atrioventricular (AV) node is a cluster of specialized cells in the anterior portion of the interatrial septum that mediates electrical conduction from the atrium into the His-Purkinje system. The PR interval on the ECG tracing is a reflection of electrical delay between atrium and ventricle, the majority of which is usually due to the decremental (slowly conducting) properties of the AVN.
- *His-Purkinje system (HPS)*: The compact His bundle originates anterior and inferior to the compact AV node before transitioning into the right and left bundle branches, a subendocardial network of rapidly conducting cells located along the interventricular septum. The bundle branches then arborize broadly into the Purkinje cells to allow for the rapid, diffuse depolarization of the myocardium. The QRS complex on the ECG tracing is reflection of ventricular depolarization.

General Overview of the ECG

The surface ECG represents a composite recording of the sum of all cardiac action potentials which represent various phases of cardiac stimulation over a period of time.

The Normal ECG Tracing

The ECG is composed of waveforms (P, T, and U), intervals (PR, QT), segments (ST), and complexes (QRS). A cardiac cycle is represented on the ECG as one PQRST wave complex. The different components of the ECG waveform correspond to various phases of the cardiac cycle. The ECG wave returns to the isoelectric line between cardiac cycles.

Waveforms

- *P wave*: atrial depolarization
- *T wave*: ventricular repolarization

Complexes

- *QRS complex*: ventricular depolarization

Intervals

- *PR interval*: onset of the P wave to onset of the QRS complex
- *QT interval*: onset of the QRS complex to the end of the T wave

Segments

- *PR segment*: end of the P wave to onset of the QRS complex
- *ST segment*: end of the QRS complex to the start of the T wave

The following is an example of a basic ECG morphology (Fig. 4.2).

Surface ECG Monitoring System

- *Modified 5-lead ECG system*: The 5-lead system is a standard monitor for detecting abnormalities in rate and rhythm and identifying myocardial ischemia. Leads I and V_5 provide 80% sensitivity for the detection of ischemia. This system is capable of monitoring 7 leads, the 3 standard limb leads (I, II, and III), the 3 augmented limb leads (aVR, aVL, and aVF), and 1 precordial lead, usually V_5. Electrodes are placed as follows: white on the right arm, black on the left arm, red on the left leg, and green on the right leg. To avoid the surgical field, V_5 (brown) is placed lateral to the left anterior axillary line of the chest wall.
- *Standard 12-lead ECG system*: The 12-lead ECG system is the standard method for detecting postoperative cardiac events. It consists of 3 standard limb leads (I, II, and III), 3 augmented limb leads (aVR, aVL, and aVF), and 6 precordial leads (V1–V6). The 4 limb leads are placed as in the 5-lead system. The 6 precordial leads are placed as follows: V_1 and V_2 are positioned on either side of the sternum at the fourth intercostal space, V_3 is positioned between V_2 and V_4, V_4 is positioned at the fifth intercostal space along the midclavicular line, V_5 is placed between V_4 and V_6, and V_6 is positioned in the fifth intercostal space along the midaxillary line (Fig. 4.3).

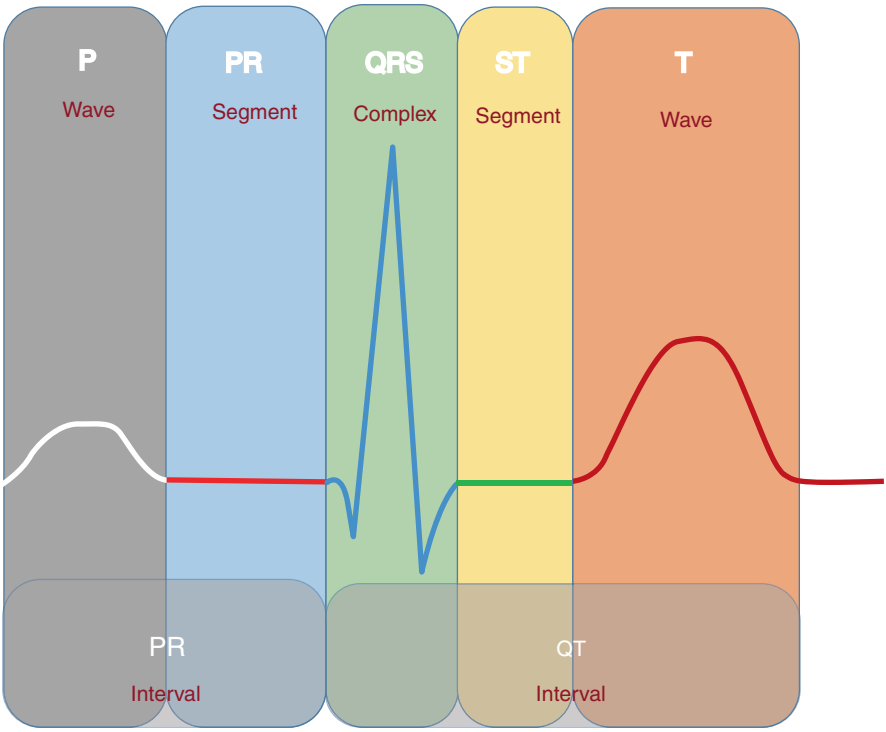


Fig. 4.2 Illustration of ECG breakdown with labeled components; waveforms, intervals, segments, and complexes

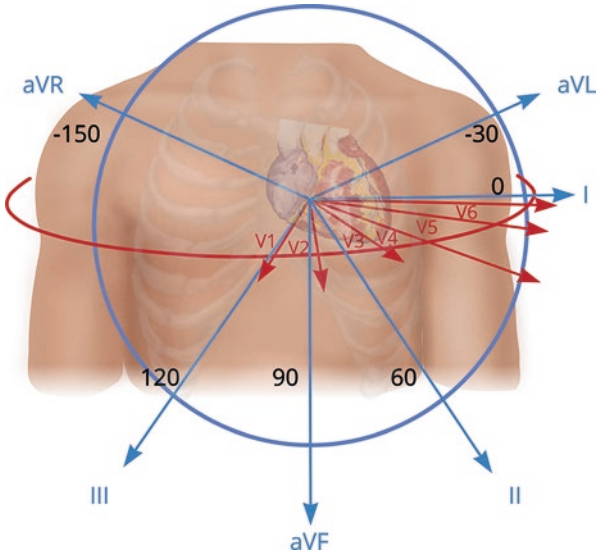


Fig. 4.3 The 12-lead ECG-precordial (red) and limb (blue) leads-system

Ischemia Detection

The ST segment and T wave morphology are the most critical parts of the ECG tracing for assessing myocardial ischemia. The ST segment is normally isoelectric. During myocardial ischemia, the affected myocardial cells are incapable of transmitting electrical signals. This results in distinctive changes in the shape of the action potential and the resting membrane potential. The end result is a characteristic deviation of the ST segment from isoelectric baseline. The ECG changes during myocardial ischemic differ substantially depending on the length of time of the ischemic event, the extent of the myocardium at risk, and the location of the ischemic myocardium. Real-time intraoperative monitoring of myocardial ischemia poses a challenge for the anesthesia provider depending solely on visual inspection.

- Subendocardial ischemia is most often due to a mismatch in oxygen supply and demand. It usually manifests as ST segment depression (see Fig. 4.4).
- Transmural ischemia is most often due to thrombotic occlusion of a native coronary artery or bypass graft that results in full thickness injury. It usually manifests as ST elevation (see Figs. 4.5 and 4.12).

ECG Interpretation for Abnormal Rhythms

AV Block

Any components of the cardiac conduction system described above can be transiently or permanently effected by open heart surgery. AV block – that is, a failure of electrical communication between the atria and ventricles – is most often seen after valve surgery because of the proximity of the conduction system to the cardiac skeleton. The septal leaflet of the tricuspid valve, the anterior leaflet of the mitral valve, and the noncoronary cusp of the aortic valve are adjacent to the interatrial

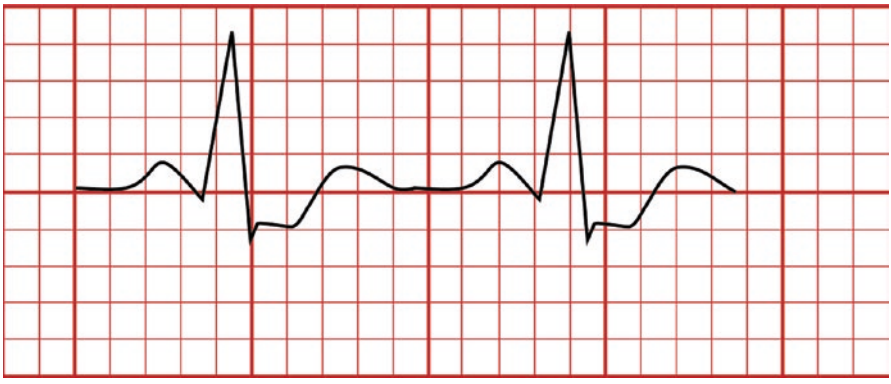


Fig. 4.4 ECG tracing showing Subendocardial ischemia ST segment depression

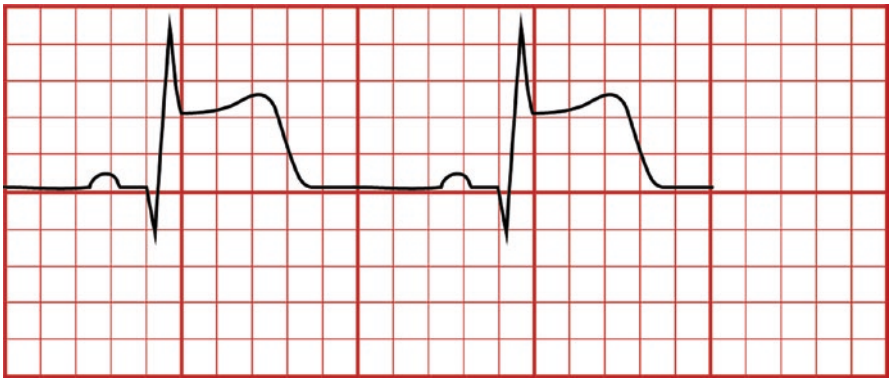


Fig. 4.5 ECG tracing showing Transmural ischemia ST segment elevation

septum, the location of the compact AV node and proximal portion of the HPS, so it is not difficult to understand why there is a risk of postoperative AV block in this patient population. For that reason, the cardiac surgeon routinely places epicardial pacing wires at the conclusion of surgery, typically threading a wire from the right atrium and right ventricle to an external pulse generator which serves as a temporary backup in the postoperative period. In the majority of cases, postoperative AV block resolves over the course of a few days with a predictable trajectory, and permanent pacing is not necessary. Until that happens, it is imperative that the patient's underlying rhythm and epicardial pacing system be reassessed on a daily basis, so that small issues can be identified, and corrected, promptly.

Benign Versus Pathological

An intrinsic failure of AV conduction is associated with progressive sinus acceleration, reflecting the withdrawal of parasympathetic influence during the episode (Fig. 4.6). The episode *may* resolve spontaneously with a junctional or ventricular escape complex but can degenerate into asystolic arrest without intervention and should be treated as an emergency. If pacing is not immediately available, a precordial thump can reset the electrical system and abort the episode. Transcutaneous, transvenous, or epicardial pacing should be arranged as soon as possible as a bridge to permanent pacemaker placement.

An intrinsic failure of conduction ("bad wiring") can cause AV block, but so, too, can extrinsic factors – electrolyte abnormalities, acid-base disturbances, hypoxia, drugs, and even hypervagatonia. It is important to distinguish an intrinsic failure of conduction from an extrinsic failure of conduction because the latter may resolve

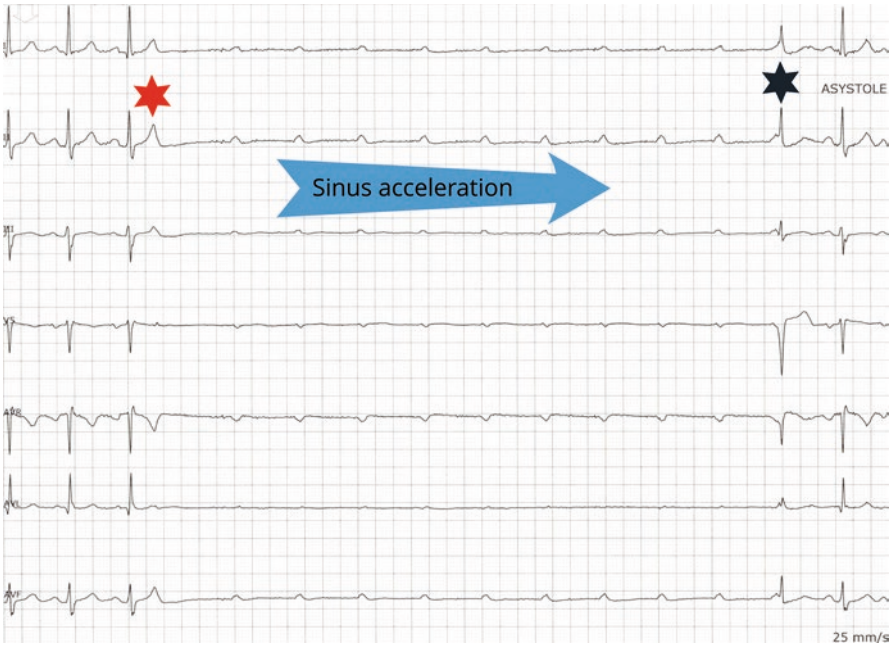


Fig. 4.6 Telemetry monitoring shows a premature atrial complex embedded on the T wave (red star) followed by a brief, non-compensatory pause and then high-grade AV block. Note that the sinus rate accelerates through the tracing before the pause finally ends with a junctional escape (black star)

with correction of the underlying problem. It is also worth mentioning that severe hyperkalemia, acidemia, or hypoxia can increase pacing thresholds and thereby compromise an otherwise normal epicardial pacing system, putting a pacemaker-dependent patient at risk. In these circumstances, increasing the pacing output or placing a transvenous pacemaker wire may not help, but correcting the underlying abnormality will.

Vagally mediated AV block *usually* follows a classic pattern, reflecting the parasympathetic influence of the vagus nerve on the SAN and AVN – sinus slowing, PR prolongation, and then either sinus arrest or high-grade AV block (Fig. 4.7). The whole episode usually resolves within 15–30 s without intervention, often with a reflex sinus tachycardia.

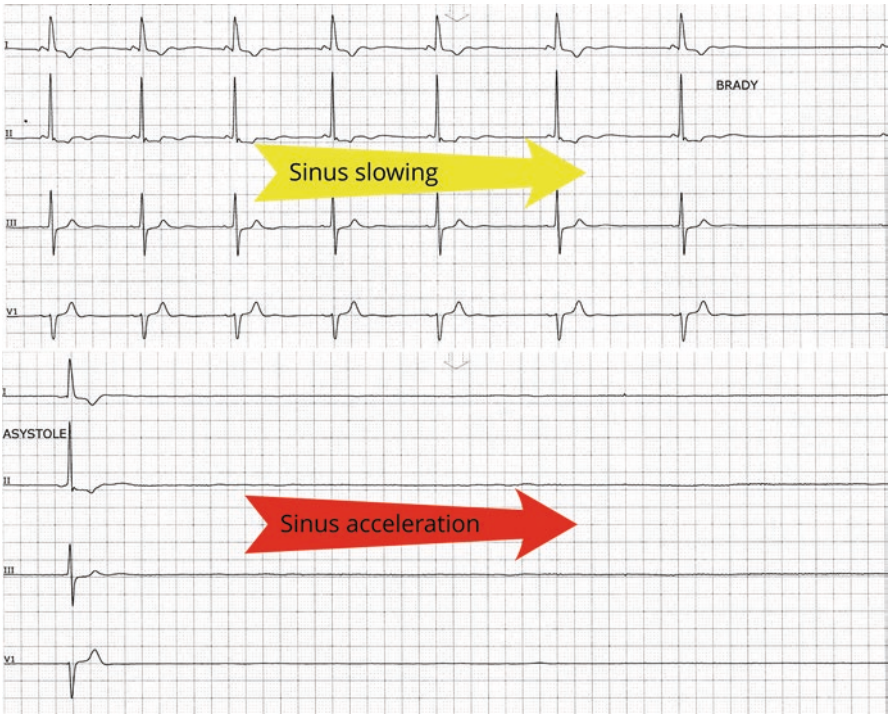


Fig. 4.7 Telemetry monitoring shows progressive sinus slowing followed by sinus arrest due to hypervagatonia. There is no evidence of AV block

Atrial Fibrillation and Flutter

Atrial arrhythmias complicate up to 50% of cardiac surgery. The diagnosis is not usually difficult, but 12-lead ECG and telemetry can be difficult to interpret.

Atrial Flutter

Atrial flutter is a macro-reentrant atrial arrhythmia that utilizes different regions of the right and/or left atrium for propagation. In the case of typical atrial flutter, the proximity of the circuit to the epicardial pacing wire can facilitate pace termination; this can be quite useful in a patient with rapidly conducted arrhythmias or bleeding issues that contraindicate anticoagulation. How does pace termination work? Think of a dog chasing its tail. The dog's mouth represents the leading edge of the arrhythmia, and the dog's tail represents the trailing edge of the arrhythmia. The open space that separates the two – and keeps the dog in motion – represents the arrhythmia's "excitable gap." Rapid atrial pacing in the immediate vicinity of the circuit may penetrate this gap, leaving it electrically

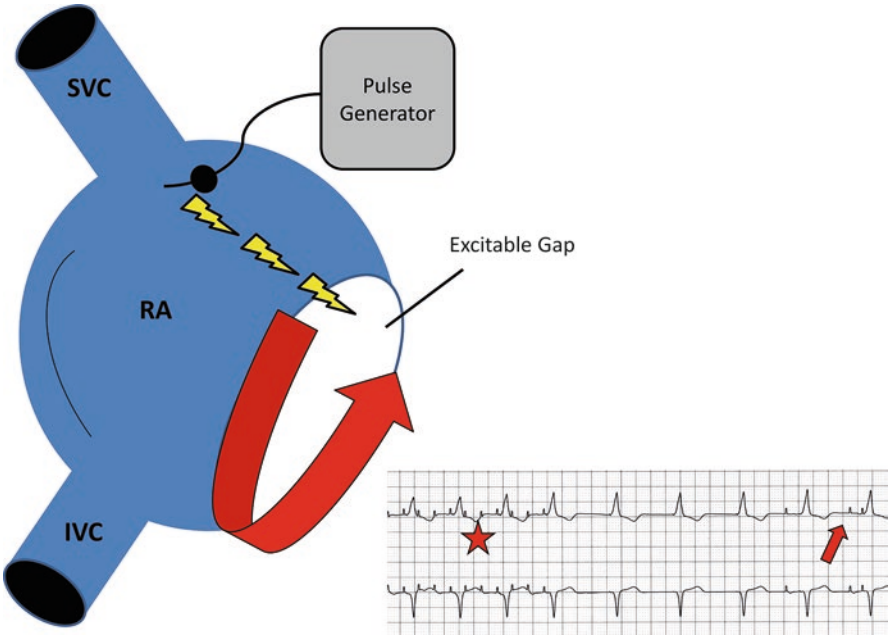


Fig. 4.8 Pace termination of atrial flutter. Manual burst atrial pacing (red star) restores sinus rhythm with evidence of AV sequential pacing (red arrow)

refractory and thereby terminating the arrhythmia. This is done by selecting "rapid atrial pacing" on the external pulse generator and then delivering high output pacing (10–20 mA) at a rate that exceeds the tachycardia rate, typically for 5–10 s. If pace termination is successful, it will restore sinus rhythm immediately (Fig. 4.8).

Pace termination is unfortunately not a panacea. It may temporarily restore sinus rhythm, but atrial flutter can obviously recur. It may also cause atrial flutter to degenerate into atrial fibrillation or have no effect whatsoever if the arrhythmia circuit arises from the left atrium, far from the epicardial pacing wire. With rare exception, pace termination is usually worth trying since it may spare the patient the need for electrical cardioversion.

Atrial Fibrillation

Atrial fibrillation is a much faster, more disorganized arrhythmia, characterized by the absence of discrete P waves *and* an irregularly irregular rhythm. Recognition of atrial fibrillation may be difficult if the patient's epicardial pacing is programmed DDD and unable to sense low-amplitude fibrillatory waves. Normal pacemaker function depends upon the ability to sense and pace (please see Chap. 32 to read more about temporary epicardial pacemaker therapy). Failure to sense low-amplitude atrial fibrillatory waves

can lead to atrial pacing which then masks the diagnosis. It is always useful to assess telemetry or 12-lead ECG for the presence of a well-defined P wave after the atrial pacing spike as that signifies atrial capture. If there is no capture, that may signify that the pacing output needs to be increased (Fig. 4.9) or that the pacemaker is pacing inappropriately into atrial fibrillation.

In some cases, attaching the epicardial atrial wire to the 12-lead ECG can clarify the rhythm (Figs. 4.10 and 4.11). Unlike atrial flutter, atrial fibrillation cannot be terminated by burst pacing.

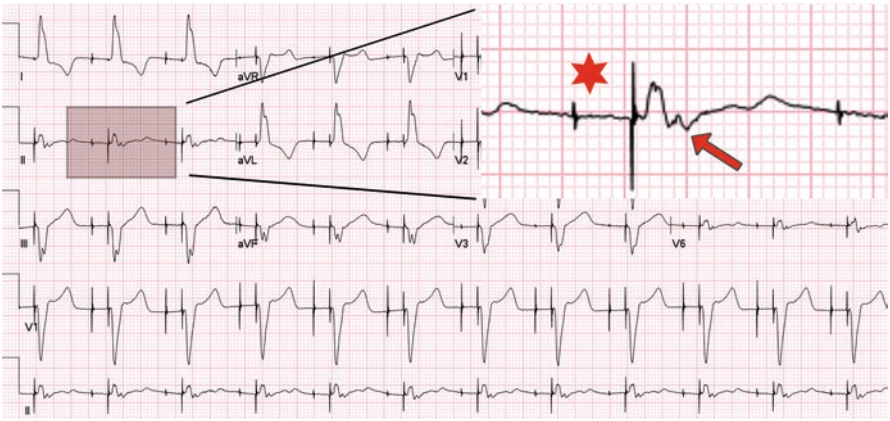


Fig. 4.9 12-lead ECG demonstrating loss of atrial capture. There is no P wave after the atrial pacing spike (inset, red star). The retrograde P wave after the paced QRS complex (inset, red arrow) proves that the underlying rhythm is either sinus or junctional, rather than atrial fibrillation or flutter

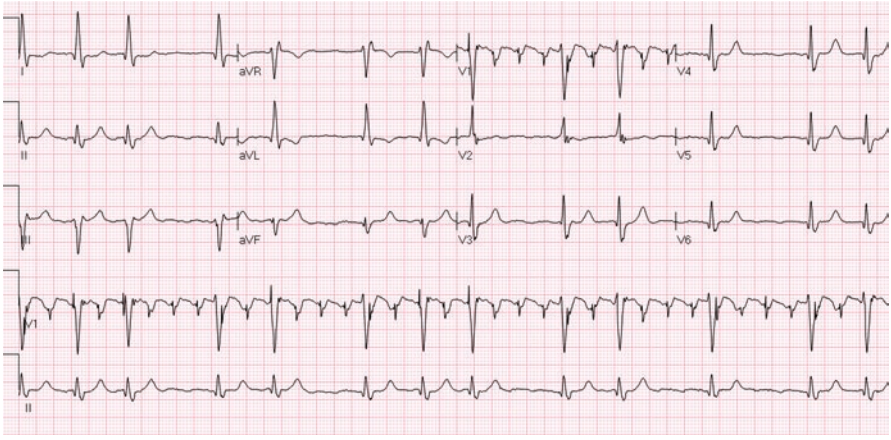


Fig. 4.10 12-lead ECG of atrial flutter. Lead V1 has been attached to the epicardial atrial wire, proving that the underlying rhythm is atrial flutter with variable conduction

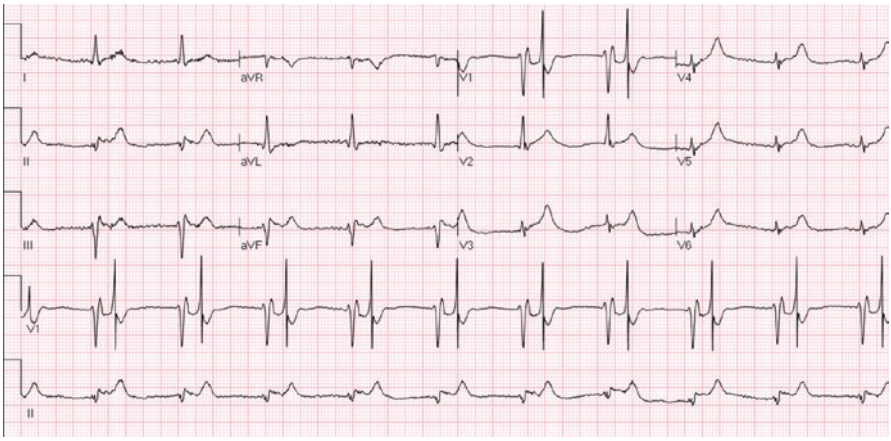


Fig. 4.11 12-lead ECG of junctional rhythm. Lead V1 has been attached to the epicardial atrial wire, proving that the underlying rhythm is junctional with retrograde AV nodal conduction

Ventricular Arrhythmias

In the immediate postoperative period, polymorphic ventricular tachycardia (PMVT) and/or ventricular fibrillation (VF) should raise concern about acute graft failure, particularly if ischemic ST-T changes are noted (Fig. 4.12).

Not all PMVT is mediated by ischemia, however. The combination of QT prolongation and pause-dependent PMVT is the hallmark of torsades de pointes (Fig. 4.13).

Whereas acute graft failure may require emergency coronary angiography and/or surgical exploration, TdP may respond to magnesium infusion (usually 1–2 g over 5–10 min) or an increase in the base pacing rate which will shorten the QT and eliminate ventricular ectopy (Figs. 4.13, 4.14, and 4.15).

Amiodarone is generally useful in the management of polymorphic ventricular arrhythmias but should absolutely be avoided in the setting of TdP.

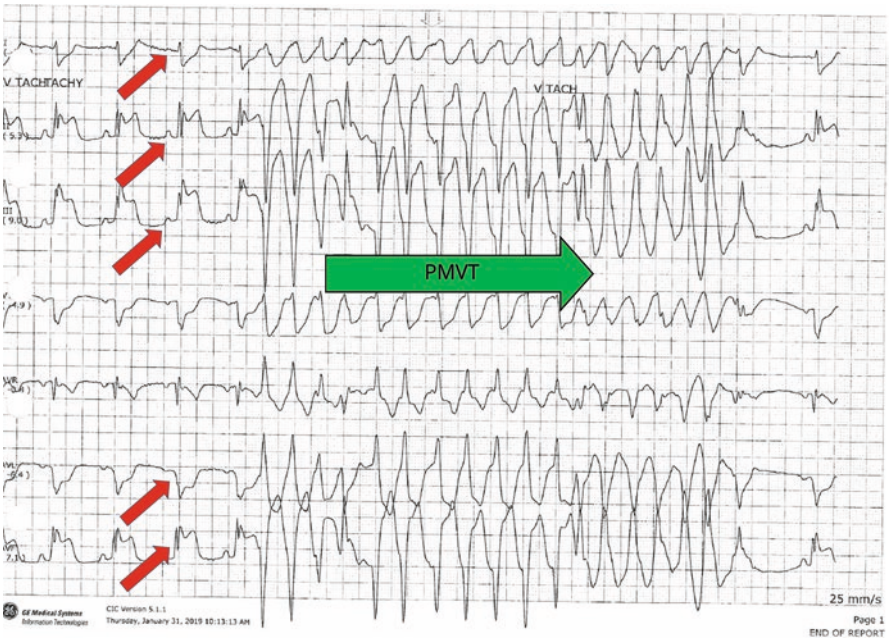


Fig. 4.12 Telemetry strip of polymorphic ventricular tachycardia (PMVT). There are profound ST-T segment shifts in the distribution of the right coronary artery, signaling the presence of coronary artery vasospasm with underlying ischemia (red arrows). The QTc is normal

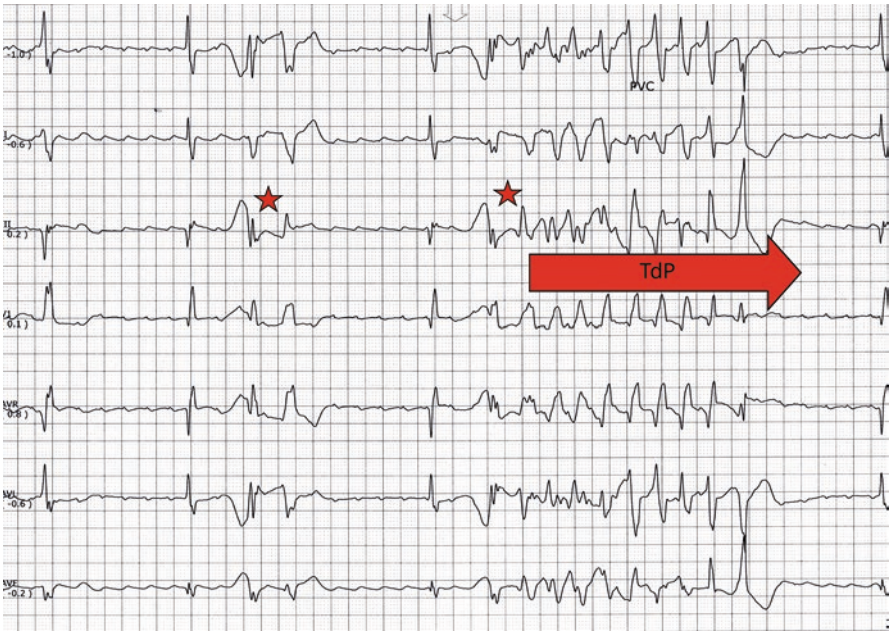


Fig. 4.13 Telemetry strip of torsades de pointes (TdP). The underlying rhythm is atrial flutter with slow ventricular response and severe QT prolongation. Early after depolarizations (red stars) eventually trigger TdP which self-terminates within a few seconds

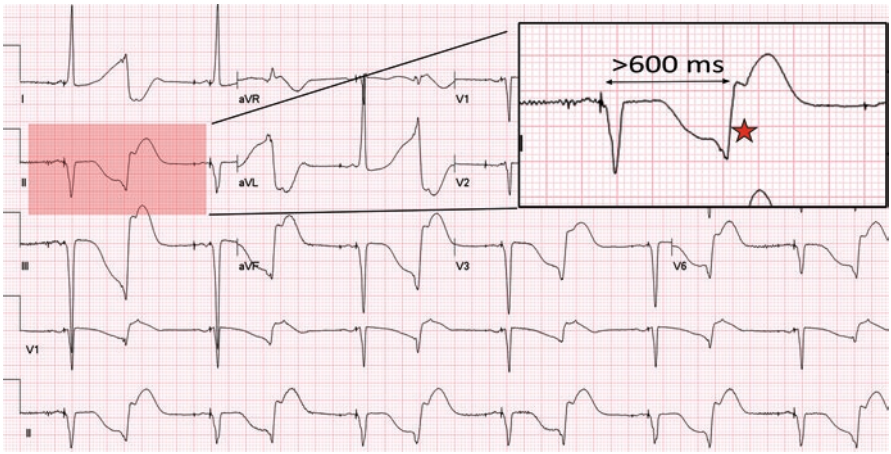


Fig. 4.14 12-lead ECG of an AV paced rhythm with severe QT prolongation and ventricular bigeminy, secondary to early after depolarizations (red star, inset)

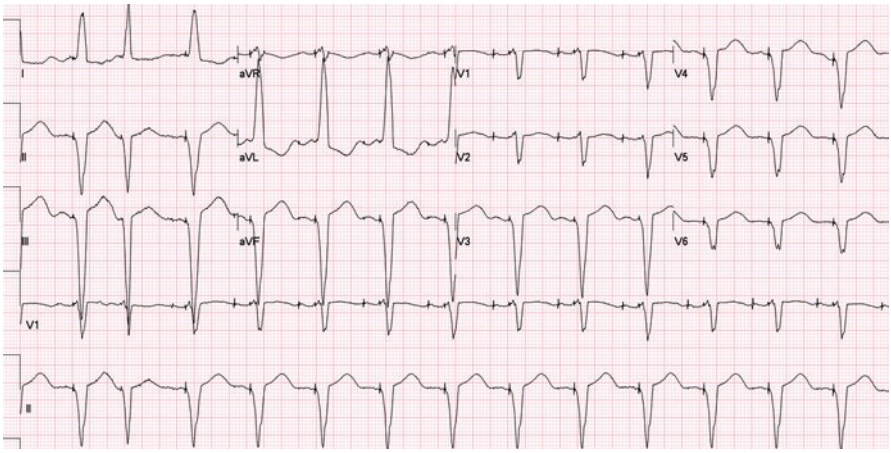


Fig. 4.15 The same patient in Fig. 4.11. A faster pacing rate has shortened the QT interval, eliminating ventricular ectopy

ECG Strips and Atlas of the Most Common ECG Abnormalities

This section will provide a quick reference and summary of the most common ECG abnormalities.

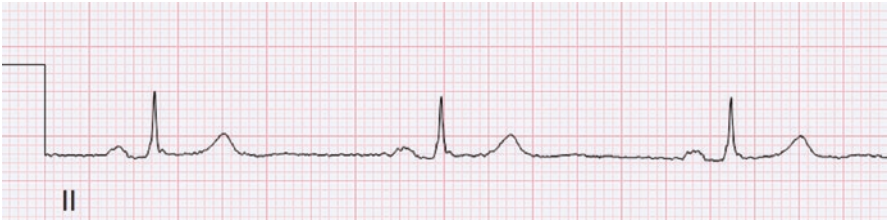
Basic Steps for Analyzing an ECG Rhythm Strip

Steps for ECG Rhythm Strip Analysis

1. Estimate the HR.
2. Verify regularity (rhythm).
3. Identify and inspect P waves (atrial activity).
4. Measure PR interval (atrioventricular conduction).
5. Evaluate and measure QRS complex (ventricular activity).

Estimate the HR	HR is calculated by counting the number of large boxes between R's and divide by 300
Verify regularity (rhythm)	Rhythm = regular or irregular = regular (R-R's are equal)
Identify and inspect P waves	P wave before every QRS/P wave upright and uniform
Determine PR interval	PR interval length = normal 0.12–0.20 s (3–5 small squares)
Examine and measure QRS complex	QRS length and shape = normal ≤0.12 s (1½ to 3 small squares)

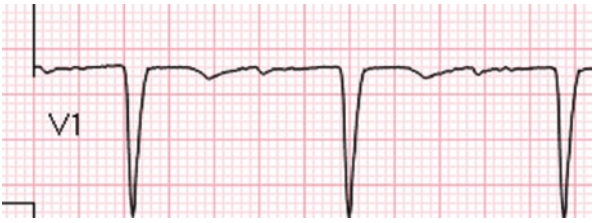
Sinus bradycardia



ECG features

Rate	Below 60/min
Rhythm	Regular; atrial and ventricular
P wave	Normal in shape, upright, size, and each P wave followed by QRS
PR interval	Normal 3–5 small squares (0.12–0.20 s)
QRS	Normal width: 1½ to 3 small squares (≤0.12 s)

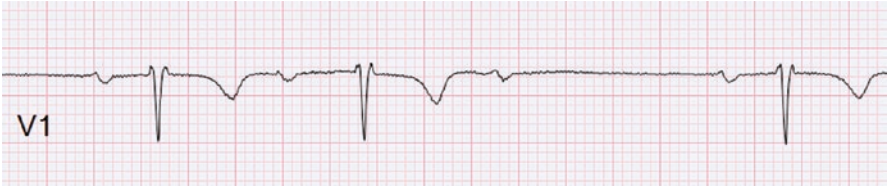
First-degree AV block



ECG features

Rate	Variable
Rhythm	Regular; atrial and ventricular
P wave	Normal in shape, size, upright, and each P wave followed by QRS
PR interval	Prolonged more than 5 small squares (0.20 s)
QRS	Normal width: 1½ to 3 small squares (≤0.12 s)

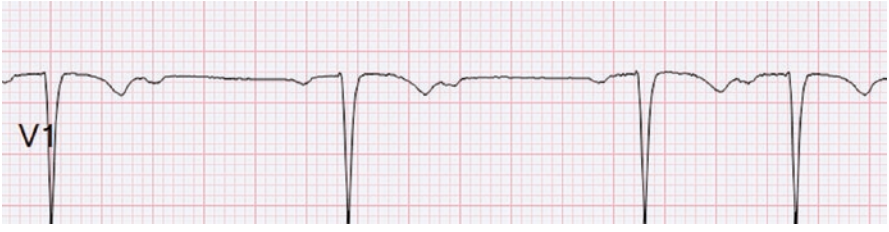
Second-degree AV block
Mobitz type I



ECG features

Rate	Variable
Rhythm	Regular atrial; irregular ventricular
P wave	Normal in shape, size, upright, but some P waves not followed by QRS
PR interval	Progressively lengthens until a QRS drops
QRS	Normal width: 1½ to 3 small squares (≤0.12 s)

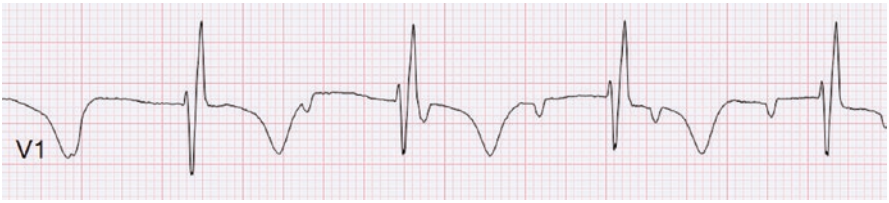
Mobitz type II



ECG features

Rate	Atrial rate higher than ventricular rate
Rhythm	Regular; atrial but variable ventricular
P wave	Normal in shape, size, upright, but every P wave not followed by QRS
PR interval	Variable; however, conducted beats are regular
QRS	Normal width: 1½ to 3 small squares (≤0.12 s)

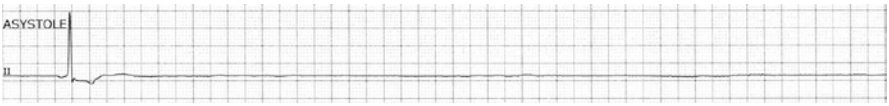
Complete or third-degree heart block



ECG features

Rate	Atrial rate normal but higher than ventricular rate
Rhythm	Regular, but both atrial and ventricular are independent
P wave	Normal in shape, size, upright, but marching through QRS complexes
PR interval	Variable and no correlation between P and QRS
QRS	May be normal or wide

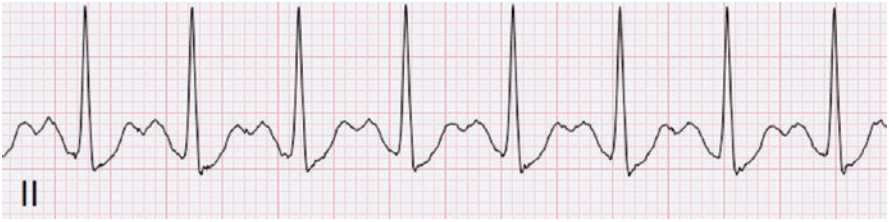
Sinus arrest



ECG features

Rate	Variable
Rhythm	Regular then irregular during arrest
P wave	Normal then absent during arrest
PR interval	Normal then absent during arrest
QRS	Normal then absent during arrest

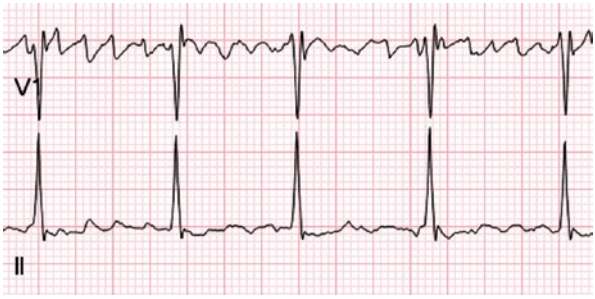
Sinus tachycardia



ECG features

Rate	Rapid above 100/min
Rhythm	Regular
P wave	Normal but may be merged with T wave at fast rate
PR interval	Normal 3–5 small squares (0.12–0.20 s)
QRS	Normal width: 1½ to 3 small squares (≤0.12 s)

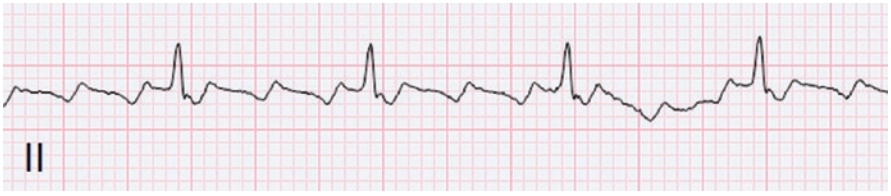
Atrial fibrillation



ECG features

Rate	Atrial rate between 350 and 400 ventricular rate variable
Rhythm	Ventricular irregularly irregular
P wave	No P waves, just fibrillatory waves
PR interval	Not measurable
QRS	Normal width: 1½ to 3 small squares (≤0.12 s)

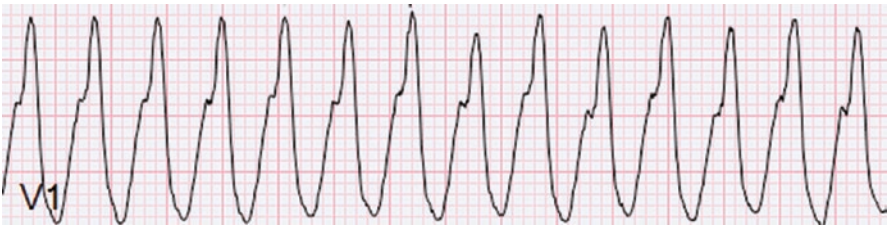
Atrial flutter



ECG features

Rate	Atrial rate between 250 and 350 ventricular rate depends on the AV block
Rhythm	Regular or irregular
P wave	Sawtooth waves
PR interval	Not measurable
QRS	Normal width: 1½ to 3 small squares (≤0.12 s)

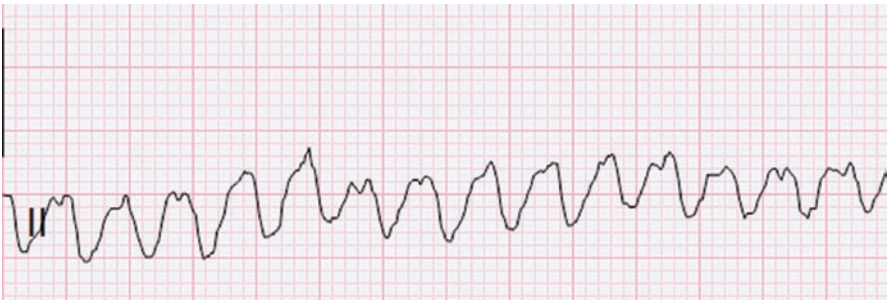
Ventricular tachycardia (VT)



ECG features

Rate	Between 140 and 250/min
Rhythm	Regular
P wave	No P waves
PR interval	Not measurable
QRS	Wide bizarre shape QRS >0.12 s

Ventricular fibrillation (VF)



ECG features

Rate	None
Rhythm	None; chaotic waves
P wave	Absent irregular chaotic waves
PR interval	Not measurable
QRS	Absent irregular chaotic waves

Further Reading

1. Kibos AS. Cardiac arrhythmias from basic mechanism to state-of-the-art management: © Springer-Verlag London; 2014. edition.
2. Olshansky B. Arrhythmia essentials. 2nd ed. Philadelphia, PA: Elsevier; 2016.

Part II

Patient Evaluation

5 Preoperative Evaluation and Risk Assessment for the Cardiac Surgery Patient

Ronak Desai, Vinay Kudur, Keyur Trivedi, Irwin Gratz, and Kinjal Patel

Declare the past, diagnose the present, foretell the future.

—Hippocrates

Introduction

The fundamental objective of preoperative anesthetic evaluation and risk stratification is to reduce the patient's surgical and anesthetic risk of perioperative morbidity and mortality. This includes obtaining crucial history and testing and diagnostic imaging results about the patient and developing a perioperative anesthetic care plan. In 2012, the American Society of Anesthesiologists (ASA) published their latest practice advisory for preanesthesia evaluation. They recommend that the preoperative anesthetic evaluation encompass a patient interview and thorough medical history, performing a focused physical examination, reviewing pertinent medical records, and, when appropriate, ordering and checking preoperative laboratory tests, and requesting specialist consultations.

Besides the ASA recommendations, a more specific evaluation is required for the cardiac surgical patient that involves determining the severity of cardiac disease. This includes assessment of the myocardial ischemic burden, evaluating the

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presence and severity of valvular disorders, measuring the degree of the right and left ventricular dysfunction, and determining whether congestive heart failure (CHF) is compensated or not.

Perioperative risk assessment should also be conducted as part of the preanesthesia evaluation. The Society of Thoracic Surgeons (STS) utilizes a national clinical outcome database for adult patients undergoing cardiac surgery, and they have developed a scoring tool for aiding in the calculation of perioperative risk assessment. This predicted risk score is used as a guide to balance the risk-benefit ratio and to provide accurate tailored recommendations and informed consent for each patient.

Preanesthesia Medical History

A comprehensive medical history is the first step for obtaining extensive information about the patient and should precede the ordering, requiring, or performance of specific preanesthesia tests. The preoperative evaluation is not meant for "clearance." Rather, it is intended for medical optimization, risk stratification, and postoperative care planning. It is also critically important to obtain and review any relevant prior anesthetic records that may uncover previous unexpected complications.

The opportunity to interview and assess each cardiac surgical patient will depend on the urgency of the surgery (i.e., elective, urgent, or emergent). If the operation is elective, the preoperative evaluation should be performed in the preanesthesia clinic, while for urgent or emergent surgery, this assessment is completed in the hospital setting and possibly inside the operating room when under time constraints.

It is important that the anesthesia provider establish a favorable rapport with each patient during the initial few minutes of the preanesthesia assessment to allow for a comprehensive analysis of the medical history and to help allay any anxiety. Techniques that favor effective communication skills and questioning techniques include active listening, empathy, open-ended questions, and the avoidance of external distractions.

Prior to the Interview

- It is critical to examine the patient's previous medical records and laboratory results.
- When reviewing the previous anesthetic records, take note of the airway management techniques and tools employed, whether there was documented difficult tracheal intubation, and any unexpected intensive care unit (ICU) admission after surgery.
- Additionally, review all specialist consult notes and the results of diagnostic imaging and laboratory tests.

History of Present Illness

The patient interview generally begins by discussing the history of present surgical illness. This is a chronological description of the progression of the patient's present illness from the first sign and symptom to the present. The proposed surgery is generally confirmed within this history.

Past Surgical History

- The patient should be asked about past surgical history with specific emphasis on:
- Prior sternotomy, thoracotomy, saphenous vein stripping, or coronary revascularization
- Note that in patients in whom prior thoracic surgery was performed, a computed tomography (CT) of the thorax is required to identify the anatomy of the right ventricle (RV), previous bypass grafts, and the position of the aorta in relation to the sternum.
- Prior percutaneous coronary intervention (PCI) and timing of any coronary stents

Anesthetic History

The patient should be questioned about previous anesthetic history and any related complications including difficult airway, prolonged mechanical ventilation, and familial anesthetic syndromes.

Medications History

A complete listing and dosages of current prescription, herbal preparations, dietary supplements, and over-the-counter medications should be obtained. A multidisciplinary plan should be in place for the discontinuation of any prior anticoagulant use.

Family History

Inquiry into a family history of anesthetic complications, especially inherited syndromes such as malignant hyperthermia, should be performed.

Social History and Habits

Smoking

Actively smoking patients are predisposed to perioperative pulmonary complications, and they should be advised to cease smoking at least 4–8 weeks before surgery.

Alcohol Misuse

- Heavy alcohol consumption is usually associated with alcoholic liver disease.
- Hepatic dysfunction or liver cirrhosis may increase the risk of excessive intraoperative bleeding. This coagulopathy is generally due to platelet dysfunction and a deficiency in vitamin K-dependent clotting factors.
- Any abnormality in coagulation may affect the selection of prosthetic valve(s). Bioprosthetic valves are preferred in such patients as postoperative anticoagulation may be avoided.
- Excessive preoperative alcohol intake may lead to postoperative withdrawal, including delirium tremens, which carries a high mortality.

Illicit Drug Use

- Intravenous drug abusers (IVDAs) have a higher risk of acquiring infective endocarditis.
- HIV and hepatitis infections are more prevalent in IVDAs.

Support System

A discussion surrounding the patient's current living conditions and support network will be important during postoperative rehabilitation.

Jehovah's Witness

- Such patients should be identified preoperatively and counselled about the dangers and consequences of transfusion rejection.
- Clearly document the patient wishes for transfusion (e.g., cell salvage, recombinant clotting factors, albumin, etc.).
- Employ a preoperative optimization protocol to increase hematocrit $\geq$40%.
- Discontinue any medications or food supplements that may increase the risk of perioperative bleeding.

Allergy

The anesthesia provider should identify the patient's medication and food allergies. Additionally, inquiry into a history of latex allergy or hypersensitivity reaction should be established to avoid latex-containing products in the operating room.

Past Medical History and Review of Systems

Cardiac surgery patients commonly have associated comorbidities. A systematic approach to the review of systems, including detailed descriptions of symptoms and chronicity, is encouraged. Such methodology may reveal previously uncovered or inadequately controlled disease states.

Cardiac History

In reviewing the cardiac history, the goals are to ascertain the indications for surgery and to evaluate the stability and severity of the current cardiac disease processes. Also, one must identify the potential risk for further end-organ damage.

Ischemic Heart Disease (IHD)

- In patients with known ischemic coronary artery disease, the most important aspects to expose are the degree of severity, the ischemic threshold, and the frequency of angina. This can be accomplished using the Canadian Cardiovascular Society (CCS) Functional Classification.
- Document previous myocardial infarctions (MI).

Congestive Heart Failure (CHF)

- The patient should be questioned about the presence, extent, and the rate of progression of symptoms.
- The signs and symptoms of decompensated left or right heart failure include dyspnea on exertion, frequent respiratory infection, and wheezing. More specifically, chest X-ray may reveal pulmonary congestion for left-sided CHF, while lower extremity edema, jugular venous distention, anorexia, nausea, and abdominal distension may indicate right-sided CHF.
- A detailed history may provide clues to the etiology of CHF.
- The New York Heart Association (NYHA) system can be used to characterize the severity of CHF.
- The presence of an AICD or biventricular pacemaker device may suggest reduced ventricular function.

Arrhythmia

- The patient should be questioned about symptoms suggestive of arrhythmia, such as palpitations, panic attacks, lightheadedness, and vasovagal symptoms.
- A detailed description of the palpitations and the circumstances and precipitating factors that correlate with the symptoms should be elicited.
- Prior electrophysiology intervention, including cardioversion and pacemaker insertion, should be determined. The location, the manufacturer, and the model of the implanted device should be documented.
- Patients with previously undiagnosed atrial dysrhythmias may require a preoperative catheter ablation or surgical maze procedure and/or intraoperative left atrial appendage ligation.

Peripheral Vascular Disease

- The patient should be questioned about manifestations of venous and arterial diseases.
- Severe aortoiliac occlusive or aneurysmal disease makes transfemoral placement of an intra-aortic balloon pump or femoral arterial cannulation technically unfeasible.
- In patients with severe ascending aortic atheroma, cross-clamping of the aorta can be challenging and may require epiaortic echocardiography.
- Patients with renal artery stenosis are prone to developing acute kidney injury (AKI) postoperatively.
- Patients with lower extremity ischemia will have poor skin and wound healing after vein harvesting for coronary bypass grafts.

Neurological Disease

- The patient should be questioned about prior cerebrovascular events, specifically transient ischemic attacks (TIAs) and cerebrovascular accidents (CVA). The patient with a prior CVA should be asked about residual motor or sensory deficits, as well as any history of cognitive impairment and any change or loss in vision or speech.
- Severe dementia may be a contraindication for surgery.
- A history of dizziness and syncope with exertion may suggest critical aortic stenosis, while similar symptoms in recumbency may imply arrhythmia.

Table 5.1 Obstructive sleep apnea STOPBANG screening tool

	STOP	Score		Score	
S	Snoring	Yes	+1	No	0
T	Tired	Yes	+1	No	0
O	Observed apnea	Yes	+1	No	0
P	Blood pressure	Yes	+1	No	0
	BANG				
B	BMI › 35 kg/m^2	Yes	+1	No	0
A	Age › 50	Yes	+1	No	0
N	Neck circumference	Yes	+1	No	0
G	Gender	Male	+1	Female	0

Airway

- A thorough dental examination should be routine, particularly for valve surgery as the spread of organisms from the oral cavity has been linked to the occurrence of infective endocarditis.
- A history of radiation and surgical procedures to the head and neck area may increase the risk of altered airway anatomy and a subsequent difficult intubation.

GI History

- A history of esophageal or gastric conditions like dysphagia, esophageal stricture, motility disorders, prior gastric surgery, or tumors that might affect the transesophageal echocardiography (TEE) probe placement, should be investigated.
- Inquire about gastroesophageal reflux symptoms as they may increase the risk of pulmonary aspiration.

Pulmonary History

- Any active respiratory infection should be resolved before surgery.
- A history of asthma should encourage further discussion into the severity of the disease (e.g., prior intubation, ICU admission, and corticosteroid therapy).
- All relevant patients should be screened for obstructive sleep apnea (OSA) using the STOPBANG questionnaire. A STOPBANG score of 5–8 is associated with a high probability of moderate to severe OSA as noted in Table 5.1.
- Symptoms consistent with a diagnosis of chronic obstructive pulmonary disease (COPD) such as dyspnea, cough, and mucus production should be investigated. If the disease is present, one should assess the severity by inquiring about home oxygen use, nebulizer treatment, and inhalers.
- Patients with significant COPD and ventilation/perfusion mismatch may not be fit for intraoperative lung isolation techniques used in minimally invasive cardiac surgery.

- Patients on amiodarone should be questioned about symptoms of pulmonary toxicity.

Renal History

- Pre-existing renal insufficiency predisposes the cardiac surgical patient for postoperative acute kidney injury. Medication dosing should be adjusted accordingly in the presence of renal dysfunction.
- Patients with a history of pelvic radiation or prostatic hypertrophy may require placement of a suprapubic catheter intraoperatively prior to surgery.
- Dialysis patients should be dialyzed the day of or the day before surgery. A serum potassium level should be checked immediately prior to surgery. The presence of an arteriovenous fistula will affect placement of invasive monitors and will require enhanced vigilance when positioning the arms prior to surgery.

Metabolic History

Diabetes Mellitus

- Patients with a history of diabetes should be assessed for end-stage complications including neuropathy, retinopathy, and nephropathy.
- Bilateral internal mammary artery (IMA) bypass grafting is not recommended in diabetics as there is an increased risk of sternal wound infection.
- Patients with diabetes may be more prone to develop phrenic nerve dysfunction due to ligation of the IMA branches to the nerve.
- Patients using protamine-containing insulin preparations preoperatively may develop protamine reactions with heparin reversal at the end of cardiopulmonary bypass.

Thyroid

- Thyroid disease may affect the selection of antiarrhythmic drugs. For example, amiodarone is avoided in patients with thyroid disease as it can induce severe thyroid dysfunction.
- Long-standing goiters may increase the risk for airway obstruction, tracheomalacia, and altered airway anatomy.
- Thyroid disease may induce arrhythmias.

Assessment of Functional Capacity and Exercise Tolerance

A comprehensive history requires the assessment of one's functional capacity or exercise tolerance. Functional capacity or exercise tolerance is a measure of the patient's physiologic reserve. A variety of methods can be used. Metabolic equivalents (METs) are a method that can be used to estimate the functional capacity of

the patient. One MET is equal to the oxygen consumption of a 70 kg, 40-year-old man at resting state (~3.5 cc oxygen/kg/min). The ACC/AHA considers patients unable to climb two flights of stairs (>4 METs) as having poor functional capacity.

Another method used to quantify exercise tolerance is the Duke Activity Status Index (DASI). It was originally developed for cardiac rehabilitation and is a self-assessment questionnaire tool that can be processed during the preoperative interview. Scores from this tool range between 0 and 58.2. Patients may also be questioned regarding their ability to perform activities of daily living (ADLs) and instrumental activities of daily living (IADLs). The ADL questionnaire examines routine daily activities like bathing, dressing, and toileting, while the IADL probes into activities like telephone use, meal preparation, and housework.

Physical Examination

The next step in the preoperative evaluation for patients undergoing cardiac surgery is to perform a focused physical examination. The goal is to identify any underlying processes that might require further optimization prior to surgery.

Vital Signs

Pulse

- Measure the heart rate. Tachycardia might suggest cardiopulmonary and autonomic impairment. Bradycardia might suggest heart block.
- Assess the rhythm. An irregular heart rhythm suggests cardiac arrhythmia. An irregularly irregular pulse is a sign of atrial fibrillation.
- Assess the volume or amplitude of the pulse to determine the best site for arterial line placement. A faint pulse may suggest a low cardiac output syndrome or severe arterial atherosclerotic disease. A bounding pulse may suggest aortic regurgitation.

Blood Pressure

- Measure the blood pressure in both arms if an aortic dissection or subclavian arterial stenosis is suspected.
- Uncontrolled hypertension needs immediate attention as it increases the risk of CVA or aortic dissection.
- Hypotension may suggest a low cardiac output syndrome and is associated with perioperative organ hypoperfusion.

Respiratory Rate

- Assess the respiratory rate, pattern, and work of breathing.
- Tachypnea may be manifestation of an undiagnosed lung disease or heart failure.

Head and Neck

- Inspect closely for loose teeth. For an elective surgery, extraction of the loose tooth preoperatively may be required. Prior to emergency surgery, a silk suture may be passed around the loose tooth and knotted at its base. This technique will allow retrieval of the tooth if it gets dislodged.
- Assess for tooth decay and periodontal disease as it is associated with an increased risk for infective endocarditis, postoperative infections, and delayed wound healing. Any dental infection should be treated preoperatively.
- A complete airway evaluation is particularly important to predict potential difficult mask ventilation or intubation. Independent predictors of difficult mask ventilation are the presence of a beard, macroglossia, edentulous, BMI › 26, and a neck circumference › 50 cm. Predictors of difficult intubation are the presence of prominent upper incisors, restricted mouth opening, reduced neck range of motion, Mallampati class › II, or short thyromental distance.
- Post-induction hypoxia and/or hypercarbia in the cardiac surgical patient will lead to prompt and profound cardiovascular collapse.
- Examine the neck veins and inspect for visible jugular venous distention (JVD) while the patient is at 45 degrees. JVD is a sign found in CHF, superior vena cava syndrome, cardiac tamponade, constrictive pericarditis, and pulmonary hypertension.

Height and Weight (BMI)

- Height and weight measurements are necessary to calculate BMI and ideal body weight (IBW). These measurements are further required for determining optimal drug dosages and ventilator settings.
- Formula for $\text{BMI} = \dfrac{\left(\text{Weight in pounds}\right)}{\left(\text{Height in inches}\right)^2} \times 703.$
- Formula for IBW male: IBW = 50 kg + 2.3 kg for every inch over 60 inches.
- Formula for IBW female: IBW = 45.5 kg + 2.3 kg for every inch over 60 inches.
- Patients with an increased BMI have an increased risk of pulmonary aspiration.

Neurologic

- A baseline neurologic assessment should be performed prior to cardiac surgery and can be used as a reference should there be postoperative neurological deterioration.
- The baseline neurologic exam should include mental status, function of the cranial and peripheral nerves, symmetry, muscle tone, and strength.

Chest

- The chest should be inspected for scars from previous sternotomy or thoracotomy incisions, chest tube sites, and implantable cardiac devices.

- The lungs are auscultated to identify any pulmonary rales, rhonchi, or wheezing that may suggest pneumonia, CHF, or COPD, respectively.
- The heart is auscultated for murmurs, rubs, or an S3 gallop that may suggest valvular disease, pericarditis, or CHF, respectively.
- Patients with implantable cardiac devices will require interrogation and reprogramming on the day of surgery.

Vascular

- The carotid artery is auscultated bilaterally for bruits. If present, it may be associated with significant carotid occlusive disease and will require further investigation.
- A weak pulse and/or a prolonged capillary refill time of the extremity may indicate severe peripheral vascular disease.

Extremities

- Inspect closely for cyanosis, clubbing, edema, varicose veins, rash, and/or the presence of an active cellulitis.
- The presence of these signs may suggest vascular insufficiency, respiratory, or cardiac decompensation.

Scoring Systems and Risk Stratification Models

The American Society of Anesthesiologists (ASA) physical status classification system was created to evaluate the fitness of patients prior to surgery. Patients undergoing cardiac surgical procedures are generally classified as an ASA 3–4. However, many organizations have developed more complex models to predict operative mortality and morbidity after adult cardiac and noncardiac surgery based on a variety of factors including patient demographics and comorbid conditions (e.g., reduced LV function, obesity, age, emergency surgical intervention, and the complexity of the procedure).

Common Scoring Systems for Cardiac Surgery

At a minimum, 19 risk models exist for adult cardiac surgery. However, the two most commonly used models with good predictive risk are The Society of Thoracic Surgeons Predicted Risk of Mortality (STS PROM) score and European System for Cardiac Operative Risk Evaluation (EuroSCORE II). Compared to the STS scoring system, EuroSCORE II does not take into consideration morbidity risk prediction such as CVA, acute renal failure, prolonged ventilation, reoperation, hospital length of stay, and deep sternal wound (DSW) infection.

Society of Thoracic Surgeons Predicted Risk of Mortality (STS PROM)

The Society of Thoracic Surgeons (STS) model was developed from an outcome database utilizing data from hospitals within the USA. This validated model estimates the perioperative risk of mortality at 30 days based on a number of different clinical variables including height, weight, age, race, gender, ejection fraction, renal failure, cardiac arrhythmias, hypertension (HTN), lung disease, peripheral artery disease, diabetes, cerebrovascular disease, and many others. The STS considers a 4–8% risk of mortality an intermediate risk and >8% high risk. Overall, the STS approximates the mortality rate of cardiac surgery over the last decade to be 2.3% for CABG, 3.4% for isolated valve procedures, and 6.8% for combined valve/CABG procedures. The STS model also incorporates several additional endpoints including a patient's percent risk for AKI, CVA, prolonged ventilation, DSW infection, reoperation, and length of stay.

An example calculation utilizing the STS PROM scoring system is seen below:

A 70-year-old white male presents with worsening shortness of breath. His past medical history includes type 2 diabetes, OSA, hypertension, obesity, and coronary artery disease with two bare metal stents placed 5 years ago. His weight is 120 kg, LV ejection fraction is 40%, and his laboratory studies demonstrate a hematocrit of 36%, white blood cell count of 12,000, creatinine of 1.2, and platelet count of 150,000. He has a 40-pack-year smoking history and is scheduled for elective CABG surgery.

The STS risk calculator can be accessed at http://riskcalc.sts.org/stswebriskcalc/calculate. The above values are submitted into the calculator and the risk results are shown below:

Risk of mortality:	1.592%
Renal failure:	2.472%
Permanent stroke:	0.858%
Prolonged ventilation:	9.691%
DSW infection:	0.304%
Reoperation:	2.164%
Morbidity or mortality:	14.158%
Short length of stay:	34.192%
Long length of stay:	5.513%

European System for Cardiac Operative Risk Evaluation (EuroSCORE II)

The EuroSCORE II permits the calculation of mortality risk after cardiac surgery by evaluating 17 patient clinical variables, the current cardiac state, and the proposed operation. Scores are stratified into low risk (0–2 points; estimated mortality 1.3%), medium risk (3–5 points; estimated mortality 2.9%), and high risk (≥6 points;

estimated mortality 10.9–11.5%). The EuroSCORE II is the most widely used mortality risk score in Europe for cardiac surgery.

Common Scoring Systems for Noncardiac Surgery

In noncardiac surgery, the most commonly used risk stratification models are the Lee Revised Cardiac Risk Index (RCRI), the Gupta Myocardial Infacrtion or Cardiac Arrest (MICA) risk calculator, and the American College of Surgeons National Surgical Quality Improvement Program (NSQIP) risk-prediction model.

The Lee Revised Cardiac Risk Index (RCRI)

The RCRI is a simple cardiac risk scoring tool that estimates the risk of postoperative major adverse cardiac events (MACE) – e.g., MI, pulmonary edema, cardiac arrest, and complete heart block. It incorporates six similarly weighted variables: CHF, CAD, creatinine >2.0, IDDM, cerebrovascular disease, and supra-inguinal vascular surgery. Despite its limitations, the RCRI is the most widely used metric in practice guidelines and research to assess high-risk patients.

Gupta Myocardial Infarction or Cardiac Arrest Calculator (MICA)

The Gupta MICA score was created utilizing the NSQIP database to improve on the accuracy of the RCRI. Five factors were distinguished as predictors of MICA: type of surgery, dependent functional status, abnormal serum creatinine level, ASA status, and old age. This verified risk model has a better predictive performance than the commonly used RCRI.

The American College of Surgeons National Surgical Quality Improvement Program (NSQIP) Risk Calculator

The National Surgical Quality Improvement Program was initially developed to compare surgical outcomes after risk adjustment of collected data. The NSQIP database allows for identification of variables that can be used to estimate unfavorable outcomes after surgery, including MACE, death, and eight others. Using the NSQIP registry, the American College of Surgeons (ACS) developed a surgical risk calculator (http://riskcalculator.facs.org) which incorporates 20 patient-specific variables (age, BMI, sex, dyspnea, previous MI, functional status, etc.) plus the surgical procedure to identify the risk of unfavorable postoperative outcomes.

Preoperative Labs

Blood Tests

- Prior to any cardiac surgery, routine blood tests performed are complete blood count (CBC), assessment of coagulation (prothrombin time, activated partial thromboplastin time, international normalized ratio), basic metabolic panel (BMP), and type and crossmatch.
- Patients maintained on heparin preoperatively should have their platelet count measured daily to rule out heparin-induced thrombocytopenia (HIT).
- Serum creatinine should be checked after cardiac catheterization to rule out contrast-induced nephropathy (CIN).
- Abnormalities on test results should be evaluated for reversible causes and may require consulting a specialist or the rescheduling of elective surgery.
- Liver function tests (LFTs) are required when there is right-sided CHF or a history of liver dysfunction.
- Patients are cross-matched for two units of packed red blood cells (PRBCs) for routine cardiac surgical procedures and four units of PRBCs, fresh frozen plasma (FFP), and platelets in redo sternotomy or thoracoabdominal aortic surgery.

Urinalysis

Generally performed only in symptomatic patients.

Cardiac Imaging and Studies

After a thorough history and physical evaluation are performed, diagnostic imaging studies will be required to delineate cardiac anatomy, valvular pathology, myocardium at risk, and targets for revascularization. Commonly ordered studies include ECG, CXR, echocardiograms, nuclear stress tests, and cardiac catheterization. Chest X-Ray (CXR) a standard posteroanterior (PA) view CXR. Any abnormalities identified in this view will guide further testing (e.g., CT chest). Electrocardiogram (ECG) a baseline 12-lead ECG is obtained and may be used as a reference to compare with postoperative ECGs.

Echocardiography

- Transthoracic echocardiography (TTE) is routinely performed for patients scheduled to undergo CABG or valve repair/replacement. Echocardiography will allow assessments of valve stenosis and/or insufficiency, pulmonary hypertension, and regional wall motion abnormalities. It can also quantify the patient's

ejection fraction. Other entities that may be recognized are mural thrombi, septal defects, or aneurysms.

- Transesophageal echocardiography (TEE), which provides better imaging quality than TTE, is performed for patients in whom TTE could not yield adequate information to answer a clinical question. For example, a TEE may be more beneficial in ruling out the presence of an atrial appendage thrombus in an atrial fibrillation patient or to grade the severity of mitral valve regurgitation. The indications and contraindications for TEE placement are discussed in the TEE chapter.

Preoperative Stress Testing for Myocardial Ischemia

When possible, exercise stress testing is the preferred method to uncover myocardial ischemia. One can determine functional capacity, ischemic regions, and arrhythmias that may develop. Radionuclide stress imaging and myocardial perfusion imaging use intravenous radioisotopes to help diagnose coronary artery disease and evaluate viable myocardium. Positron emission tomography (PET) is occasionally used as it can allow for a better understanding of regional blood flow and myocardial metabolism.

Magnetic Resonance Imaging (MRI)

MRI can be useful in providing a high-resolution 3-dimensional image of cardiac structures. This test will also demonstrate perfusion and atherosclerosis of coronary arteries by detecting lipid accumulation, edema, fibrosis, the rate of phosphate turnover, and intracellular pH in ischemic areas.

Cardiac Catheterization

Nearly all patients scheduled for either CABG or valve surgery have undergone diagnostic coronary angiography, as it is considered the gold standard for the diagnosis of cardiac pathology. Cardiac catheterization will be discussed in detail in Chap. 6.

Perioperative Medication Management

The management of preoperative medications is a coordinated effort requiring careful planning between surgeons and anesthesiologists as it may serve to reduce perioperative complications. Table 5.2 summarizes the general recommendations for the management of a specific drug class in the perioperative

Table 5.2 Recommendations for perioperative medication management[a]

Name or drug class	Recommendation
Cardiovascular medications	
Beta blockers	Continue perioperatively
Calcium channel blockers	Continue perioperatively
Diuretics	Hold morning of surgery
ACE inhibitors or ARB	Hold morning of surgery
Alpha-2 antagonist (clonidine)	Continue perioperatively
Statins	Continue perioperatively
Non-statin lipid-lowering therapy (niacin, fibrates)	Hold morning of surgery
Antiarrhythmic	Continue perioperatively
Digoxin	Continue perioperatively
Pulmonary medications	
Inhaled beta agonists or anticholinergics	Continue perioperatively
Theophylline	Discontinue evening before surgery
Leukotriene inhibitors	Continue perioperatively
Endocrine medications	
Prandial insulin	Hold morning of surgery
Long-acting insulin	Continue perioperatively[b]
Non-insulin oral diabetic agents	Hold morning of surgery
Thyroid medication for hypo- and hyperthyroidism	Continue perioperatively
Oral contraceptive pills	Discontinue prior to surgery
Glucocorticoids	Continue perioperatively[c]
Bisphosphonates	Hold morning of surgery
Drugs affecting hemostasis	
Aspirin	Depends on surgery. Continue for CABG
$P2Y_{12}$ inhibitors (clopidogrel, ticagrelor, etc.)	Hold 5 days prior to surgery
Anticoagulants (warfarin, apixaban)	Hold 5 days prior to surgery
NSAID	Hold 5 days prior to surgery
Psychotropic medications	
Tricyclic antidepressants	Continue perioperatively[d]
SSRIs	Continue perioperatively
MAO inhibitors	Hold 2 weeks prior to surgery
Benzodiazepines	Continue perioperatively
Antipsychotics	Continue perioperatively
Stimulants	Hold morning of surgery

ACE angiotensin-converting enzyme, *ARB* angiotensin II receptor blocker, *NSAID* nonsteroidal anti-inflammatory drug, *SSRI* selective serotonin reuptake inhibitor, *MAO* monoamine oxidase

[a]These are general recommendations; exceptions may apply based on the indication of the medication and type of surgery

[b]If prolonged NPO status is anticipated, recommendations are to decrease the dose by 50% for type 2 diabetics if taking more than 50 units and by 20% for type 1 diabetics

[c]Patients on chronic steroids will require stress-dose steroids preoperatively to prevent adrenal insufficiency

[d]Most textbooks recommend continuing TCA perioperatively; the US FDA recommends holding certain TCAs (amitriptyline, nortriptyline, imipramine, and desipramine) the day before surgery

setting. As each drug class may be used for several different clinical indications, maintenance of such medications should be individualized to the patient and type of surgery. For example, it is generally recommended to hold aspirin on the morning of elective noncardiac surgery. However, in patients undergoing CABG operations, they should continue aspirin therapy in the perioperative setting.

Further Readings

Committee on Standards and Practice Parameters, et al. Practice advisory for preanesthesia evaluation: an updated report by the American Society of Anesthesiologists Task Force on Preanesthesia Evaluation. Anesthesiology. 2012;116:522–38.

Cohen NH. Medically challenging patients undergoing cardiothoracic surgery. 2009.

Weisberg A. Preoperative evaluation and preparation of the patient for cardiac surgery. Med Clin North Am. 2009;93(5):979–94.

A Guide to Interpreting Preoperative Cardiac Studies

6

Simon Topalian, Rady Ho, and Georges Kaddissi

Introduction

Patients undergoing cardiac surgery are at risk for perioperative morbidity and mortality. The degree of risk is related to the complexity of surgery and patients' comorbidities.

Comprehensive assessment of cardiac surgery patients starts with a thorough medical history and physical examination. All pertinent clinical, laboratory, and imaging data are reviewed by the anesthesia provider in order to formulate a good anesthetic plan.

Several risk calculators can be used to predict the outcome of cardiac surgery.

Preoperative evaluation and risk assessment of cardiac surgery patients are discussed in Chap. 5. Systematic review and interpretation of preoperative imaging studies is a key component in managing these patients. This chapter provides a guide to interpreting preoperative cardiac studies.

Preoperative Cardiac Studies

Since all patients undergoing coronary artery bypass grafting (CABG) or valve surgery receive a 12-lead electrocardiogram (ECG), a transthoracic echocardiogram (TTE), and a cardiac catheterization, anesthesia providers should know how to interpret these studies.

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Electrocardiogram

A preoperative ECG is useful in the evaluation of electrical and conduction abnormalities as well as dynamic ischemic ST-segment changes. The preoperative ECG is a valuable baseline study for comparison if new ECG changes develop postoperatively. Concerning preoperative ECG findings are the presence of Q waves, which are evidence of old myocardial infarction; ischemic ST-T changes; arrhythmias, such as atrial fibrillation; high-degree atrioventricular block; left ventricular hypertrophy; QT prolongation; and bundle-branch block. Often, the anesthesia provider places a pulmonary artery catheter to enable hemodynamic monitoring. When the balloon-directed pulmonary artery catheter is advanced through the right ventricle, the right bundle branch can be injured. It is critical that the preoperative ECG be examined as a pre-existing left bundle-branch block would pose a risk for complete heart block. If left bundle-branch block is present, the catheter should be passed after the surgeon has placed the temporary epicardial pacing wires. Please refer to Chap. 4 for the basic ECG and common ECG abnormalities found in cardiac surgery patients.

Echocardiogram

Preoperative left ventricular (LV) dysfunction and New York Heart Association heart failure class are important predictors of outcome following cardiac surgery. TTE is valuable for assessing the baseline LV function and regional wall motion. A step-by-step practical approach to TTE and TEE, from basic ultrasonography and image acquisition to common pathophysiologic findings on TTE and FAST examination, is discussed in Chaps. 10, 11, and 12; this section focuses on the preoperative assessment of ventricular function and the severity of major valvular disorders. Several variables are measured for assessing the LV function:

- Chamber dimension in systole and diastole, in M-mode and 2-D mode.
- Assess the contractility during systole of each wall in reference to diastole. In the parasternal long axis view, the posterior and septal walls are well seen. In the parasternal short axis views, the LV looks like a "donut." In that view, the anterior, septal, lateral, and inferior wall contractility can be easily assessed. The apical views complete the full assessment of LV wall contractility (Fig. 6.1).
- LV function can be assessed with volumetric measurement, such as with Simpson's method (Fig. 6.2).
- TTE also provides assessment of the right ventricular (RV) size and function. RV systolic pressure can be estimated with Doppler interrogation of the tricuspid valve and the size of the inferior vena cava.
- TTE and TEE are used to assess the severity of stenotic or regurgitant valves. They provide anatomical and morphological assessment of the valves, i.e.,

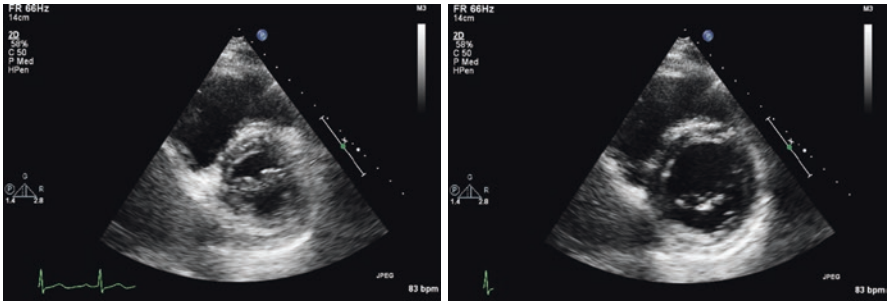

Fig. 6.1 TTE image of parasternal short axis view for assessing the LV in systole and diastole

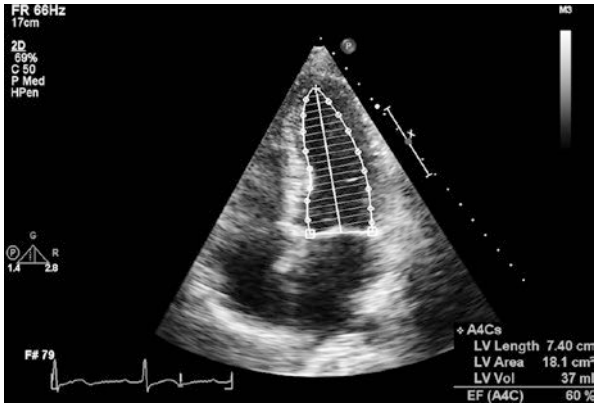

Fig. 6.2 TTE image of volumetric assessment of LV function by Simpson's method

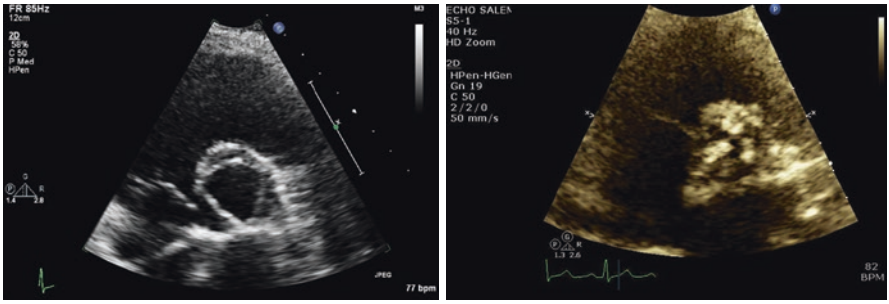

Fig. 6.3 TTE image of (**a**) normal aortic valve morphology and opening and (**b**) heavily calcified aortic valve in severe aortic stenosis

presence and extent of calcification, leaflet mobility, and excursion (Figs. 6.3, 6.4, 6.5, 6.6, and 6.7).

- Table 6.1 summarizes the echocardiographic criteria for severe valvular heart disease.

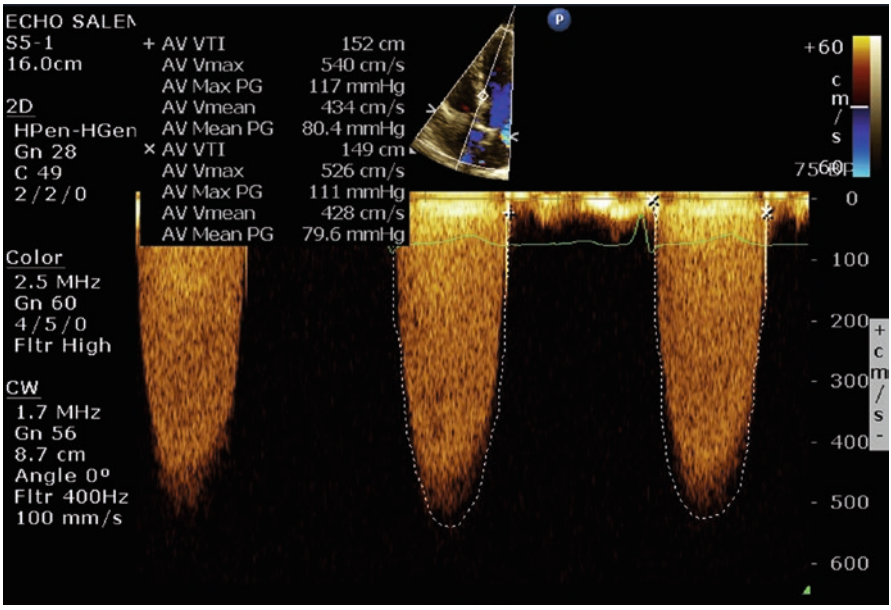

Fig. 6.4 TTE image of Doppler evaluation of critical aortic stenosis. Velocity > 5 m/sec, mean gradient 79.6 mm Hg

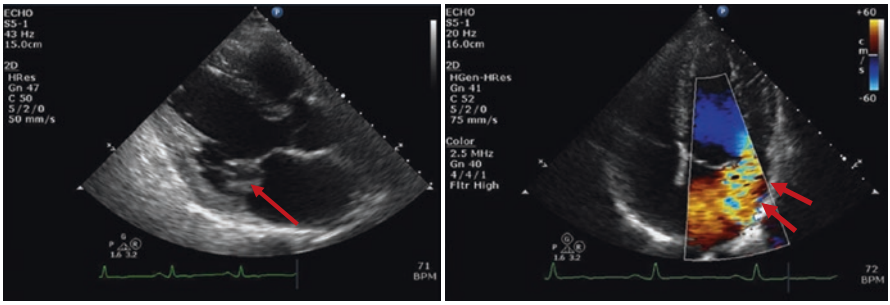

Fig. 6.5 TTE image of an example of flail posterior mitral valve leaflet (red arrow), resulting in severe posteriorly directed mitral regurgitation, as seen by color Doppler

Computed Tomography

There are three types of CT scanning: conventional, spiral, and multi-slice. Chest CT has a major role in the management of certain cardiac surgery conditions (Fig. 6.8):

(a) Planning of reoperation heart surgery. Identification of the positions of the ascending aorta, RV, and bypass grafts in relation to the sternal wall to avoid damage during re-sternotomy.
(b) Assessment and evaluation of aortic aneurysms and dissections.

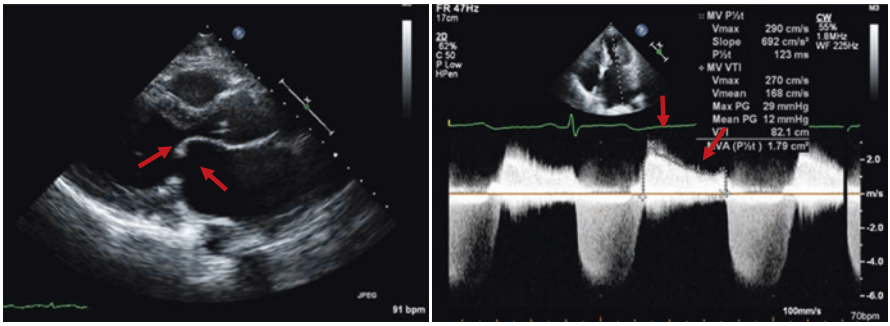

Fig. 6.6 TTE image of "Hockey stick" appearance of the mitral valve, specific for rheumatic mitral stenosis, with Doppler assessment of the gradient across the mitral valve

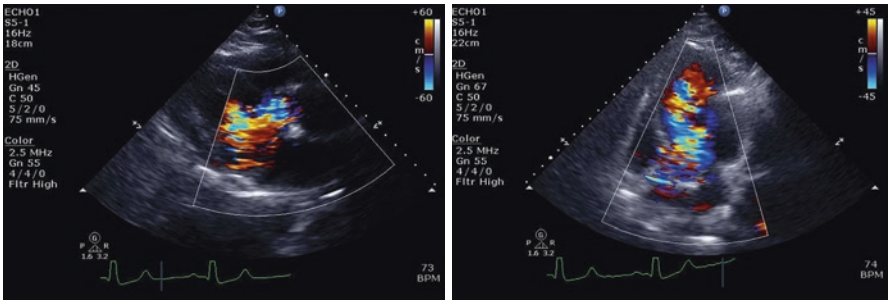

Fig. 6.7 TTE image of color Doppler example of severe aortic regurgitation

Table 6.1 The echocardiographic criteria for severe valvular heart disease (Nishimura et al. JACC 2017)

Severe aortic stenosis	Severe aortic regurgitation	Severe mitral stenosis	Severe mitral regurgitation
Aortic $V_{max} \geq 4$ m/s or mean $\Delta P \geq 40$ mm Hg	Doppler jet width ≥ 65% of LVOT	MVA ≤ 1.5 cm² (MVA ≤1 cm² with very severe MS)	Central jet MR >40% LA or holosystolic eccentric jet MR
AVA typically is ≤1 cm² (or AVAi ≤0.6 cm²/m²)	Vena contracta >0.6 cm	Diastolic pressure half-time ≥ 150 msec	Vena contracta ≥0.7 cm
Critical AS: $V_{max} \geq 5$ m/s, or mean $\Delta P \geq 60$ mm Hg	Holodiastolic flow reversal in the proximal abdominal aorta	Diastolic pressure half-time ≥ 220 msec with very severe MS	Regurgitant volume ≥ 60 cc
	RVol ≥60 mL/beat		Regurgitant fraction ≥50%
	RF ≥50%		ERO ≥0.40 cm²
	ERO ≥0.3 cm²		
	Chronic, severe AR requires evidence of LV dilation		

AVA aortic valve area, *AVAi* aortic valve area index, *AS* aortic stenosis, *LVOT* left ventricular outflow tract, *RVol* regurgitant volume, *RF* regurgitant fraction, *ERO* effective regurgitant orifice, *MVA* mitral valve area, *MS* mitral stenosis, *MR* mitral regurgitation, *LA* left atrium

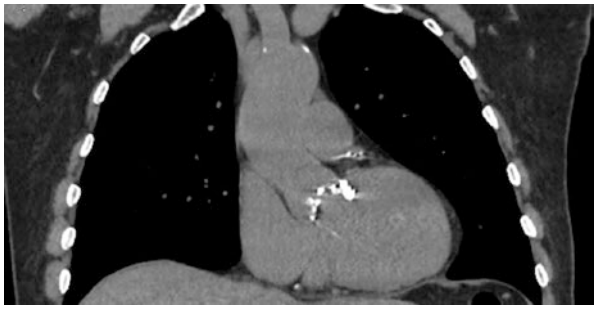

Fig. 6.8 An image of CT scan for a patient with severe aortic valve stenosis scheduled for minimally invasive aortic valve replacement showing moderate aortic valvular calcifications but no significant aortic calcification

(c) Assessment of vascular access and aortic root anatomy in pre-procedural evaluation for transcatheter aortic valve replacement.
(d) Assessment of aortic valve calcification and accurate measurement of the height of coronary artery ostia from the aortic annulus.
(e) Accurate measurement of the aortic annulus as needed in choosing the appropriate-size valve.
(f) Occasional assessment of the extent of aortic calcification (i.e., porcelain aorta) before CABG.
(g) Preparation for minimally invasive valve and robotic surgery.

Cardiac MRI

CMRI is a valuable tool for cardiac imaging since it provides high-quality anatomic and functional imaging without exposing the patient to ionizing radiation.

I It is the most used tool in evaluating myocardial viability by identifying scarring (with gadolinium contrast).
II It can assess the size and function of the right and left ventricles and accurately measure their ejection fraction.
III It can be used with pharmacological stress to replace stress echocardiography.
IV It is especially useful in quantification of valvular regurgitation.
V It provides accurate three-dimensional reconstruction analysis of the complex cardiac and extracardiac anatomy.

CT scan and CMR are also helpful in the evaluation of congenital abnormalities of the heart and/or great vessels.

Radionuclide Myocardial Imaging

Radionuclide myocardial imaging is uniquely valuable in assessing patients undergoing cardiac surgery: It reveals coronary perfusion, which is an indicator of the extent of myocardial ischemia and myocardial viability. With ECG-gated images, an ejection fraction can be generated.

Cardiac Catheterization

Preoperative diagnostic cardiac catheterization with angiography is an invasive procedure that is accomplished via percutaneous access to the heart and its vessels. Guided by fluoroscopy, cardiac catheterization allows imaging of the coronary arteries and cardiac chambers by injecting radio-opaque contrast media. Measurement of intracardiac filling pressures and hemodynamics is obtained during cardiac catheterization. It also permits sampling of blood for oxygen saturation analysis which aids in calculating cardiac output and intracardiac shunting. Except for young patients (less than 35 years old) who have no significant cardiac risk factors, the procedure is performed routinely in patients undergoing cardiac surgery. For the anesthesia provider, knowing whether there is significant left main coronary artery stenosis is important during the pre-surgical part of the procedure because of the notoriously high risk of hemodynamic collapse that might occur during anesthesia induction (switching to positive pressure ventilation driving preload down instead of negative pressure ventilation and the effect of medications on the cardiovascular system). Prolonged hypotension must be avoided since the patient may become ischemic with ensuing cardiac arrest. For proper interpretation of cardiac catheterization, the anesthesia provider needs basic knowledge of the coronary anatomy (discussed in Chap. 1).

Another important determination made by cardiac catheterization is the presence of a dominant circumflex coronary artery, which course is in close proximity to the posterior segment of the mitral valve annulus. Narrowing or occlusion of this artery by iatrogenic injury during repair or replacement of the mitral valve can precipitate severe ventricular dysfunction in these patients during weaning from bypass.

Cardiac catheterization can be either for the left heart, right heart catheterization, or both.

Left Heart Catheterization

A pigtail catheter is inserted percutaneously through the femoral or radial artery. The catheter is advanced retrograde over a wire, and under fluoroscopic guidance, it is passed across the aortic valve into the LV. Ventriculography of the left ventricle and aortography of the aortic root are performed with large volumes of contrast media injected using a power injector. Ventriculography reveals the anatomy and systolic function of the LV, and it depicts segmental wall-motion abnormalities. It can also reveal the presence and severity of mitral regurgitation. The areas of the aortic and mitral valves, in stenotic lesions, can be calculated from the intracardiac and right heart pressures using the Gorlin formula. Blood samples can be taken via the catheter to assess oxygen saturation.

Pre-shaped coronary catheters are advanced retrograde and over a wire to selectively engage the coronary ostia. Contrast medium is manually injected into the ostium of each coronary for visualization of the vessels.

Right Heart Catheterization

A balloon-tipped flow directed catheter is inserted percutaneously through the femoral, brachial, subclavian, or internal jugular vein and advanced antegrade. Similar to floating a Swan-Ganz catheter, the balloon-tipped catheter is advanced to the right atrium, RV, and pulmonary artery. This is achieved using fluoroscopic guidance and monitoring of the pressure waveform tracing.

Right heart catheterization provides diagnostic information from the right side of the heart. Filling pressures of the right atrium, RV, pulmonary artery, and occlusion pressure of the pulmonary artery can be measured. Blood can be sampled for measurement of mixed venous oxygen saturation. Cardiac output, peripheral and pulmonary vascular resistance, and shunt ratio can be then calculated.

Cardiac Catheterization Equipment

Evaluation of a three-dimensional complex structure such as the heart from two-dimensional cine angiographic views, on a flat screen that lacks depth, can be difficult and confusing to the anesthesia provider. Next is a brief description on how images are acquired and an explanation of the angiographic view's nomenclature. The equipment used consists of:

I Cardiac catheterization X-ray table where the patient is positioned.
II Real-time projection X-ray imaging system.
  (a) The X-ray tube (or generator) is positioned below the table. The X-ray beam traverses the patient and the signal is captured on the image intensifier or flat digital panel, positioned above the patient. The image intensifier and X-ray source are connected by the C-arm (like the letter C).
  (b) Image display monitor.
Ill Angiographic and hemodynamic monitoring display panels.
IIII Lead shields.
IIIII Control room.

Image Acquisition

For acquisition of real-time angiographic images, the operator rotates the C-arm and moves the table, so the X-ray beam is centered on the patient's heart. The C-arm can be moved around the patient in two planes:

I Looking from the patient's feet, the image intensifier can be moved left or right giving the LAO (Left Anterior Oblique) or RAO (Right Anteior Oblique) views (Fig. 6.9a)
II Looking from the patient's side, the image intensifier can be angulated towards the head or the feet giving the cranial (CRA) or caudal (CAU) views (Fig. 6.9b)

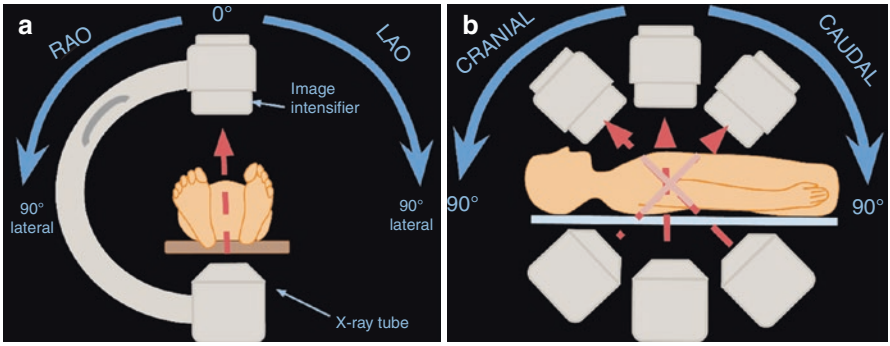

Fig. 6.9 Illustration of (**a**) the image intensifier moving left or right to create the LAO or RAO views, respectively, and (**b**) the image intensifier moving cranial or caudal to create CRA or CAU views, respectively

The image intensifier can rotate up to 90° to either the right or left side. At 90° to the right, the view is called right lateral view; at 90° to the left, it is called left lateral view. Rotation towards the right side of the patient is called right anterior oblique (RAO), and rotation towards the left is called left anterior oblique (LAO). The degree of rotation is added, so RAO 40° means the image intensifier is at 40° angle to the right. The same angulation applies to the cranial (CRA) or caudal (CAU) views. So, CRA 30° means the image intensifier was angulated 30° towards the head of the patient. There is a limit to how far the image intensifier can move in a cranial or caudal direction before it collides with the patient or the table.

Coronary Angiography

To assess the left coronary artery, four or five projections are usually obtained, but more views with different angulations may be needed for complete assessment.

- The RAO caudal view reveals the left main, left circumflex, and proximal left anterior descending arteries well. In this view, the left anterior descending artery is located to the right (Fig. 6.10).
- The LAO caudal view, known as "spider view," is the best view for assessing the left main artery and ostia of the left anterior descending and left circumflex arteries. In this projection, the left anterior descending artery is on the left of the screen and the left circumflex artery is to the right (Fig. 6.11).
- The RAO cranial view reveals the left anterior descending artery in its entirety, from the proximal to the distal segments (Fig. 6.12).
- In the LAO cranial views, the left anterior descending artery is on left side, and the spine is slightly to the right side (Fig. 6.13).

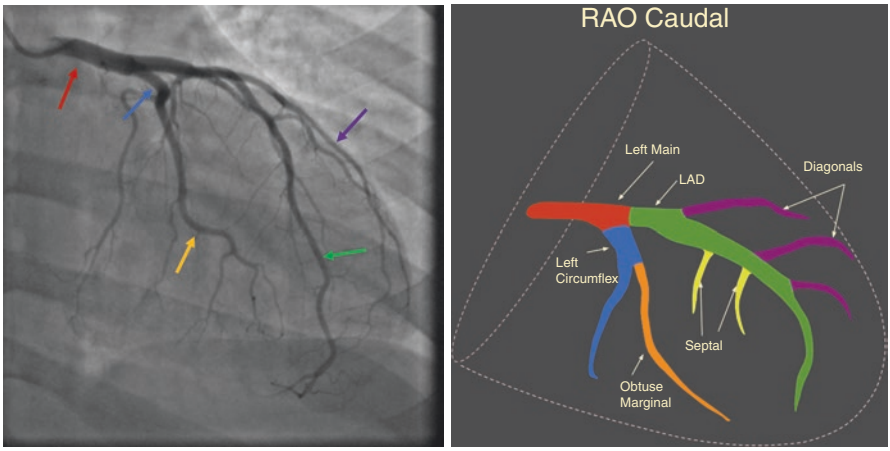

Fig. 6.10 An image and illustration of left coronary artery in the RAO caudal view (red arrow "segment," left main coronary; green, left anterior descending artery; blue, left circumflex artery; orange, obtuse margin; purple, diagonal branches; yellow, septal perforators)

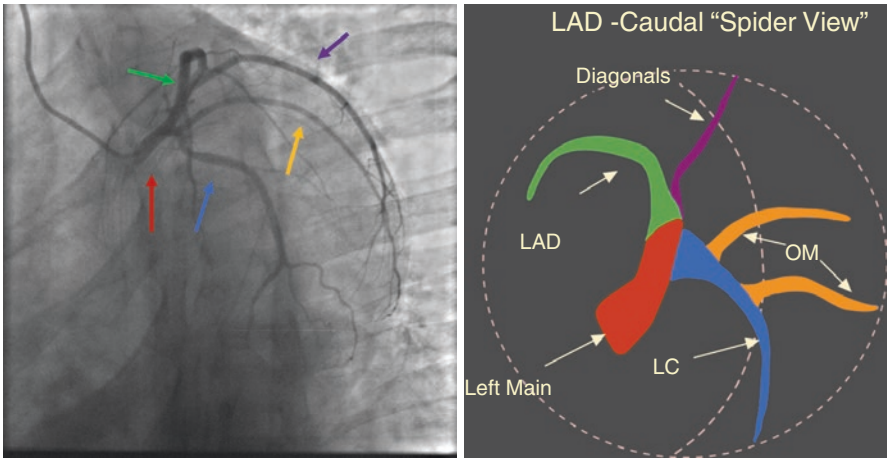

Fig. 6.11 An image and illustration of left coronary artery in the LAO caudal view, known as "spider view" (red arrow "segment," left main coronary artery; green, left anterior descending; blue, left circumflex; orange, obtuse margin; purple, diagonal branches)

For the right coronary artery (RCA), two projections are usually obtained (Fig. 6.14):

- The LAO view with mild cranial angulation. In this view, the RCA looks like a "C." The entire segments of the RCA can be seen well.
- The RAO view is useful for assessing the mid-segment of the RCA and the entire posterior descending artery.

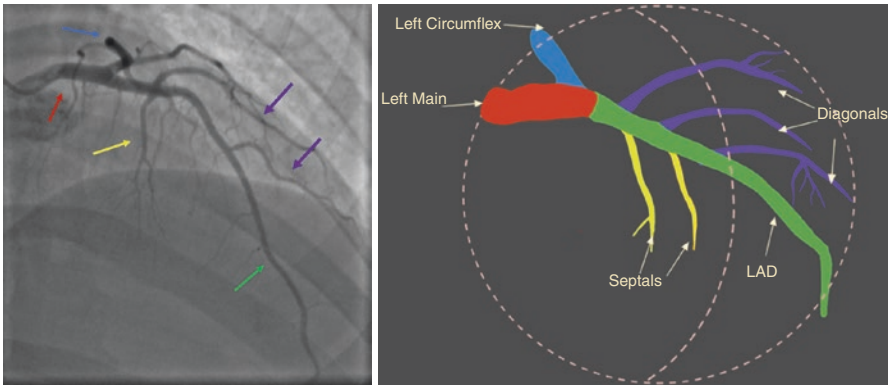

Fig. 6.12 An image and illustration of left coronary artery in the RAO cranial view (red arrow "segment," left main coronary artery; blue, left circumflex; green, left anterior descending; yellow, septal perforator; purple, diagonal branches)

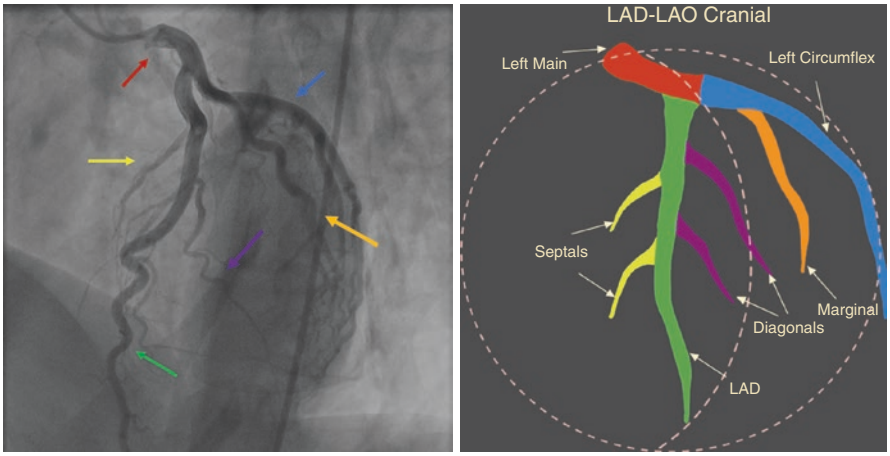

Fig. 6.13 An image and illustration of left coronary artery in the LAO cranial view (red arrow "segment," left main coronary artery; blue, left circumflex; green, left anterior descending; yellow, septal perforator; purple, diagonal branches; orange, obtuse margin)

- At times, a straight anteroposterior view with cranial angulation is used to evaluate the bifurcation of the posterior descending artery and the posterolateral branch.

Modern computerized software systems help with quantitative coronary analysis of the degree of the luminal narrowing. However, subjective assessment by visual estimation remains the most widely used method to assess coronary stenosis. With trained eyes, one compares the degree of narrowed segment to the next "normal" segment to determine the percentage of stenosis (Fig. 6.15). The degree of coronary calcification and the extent of coronary atherosclerosis should be assessed during coronary angiography.

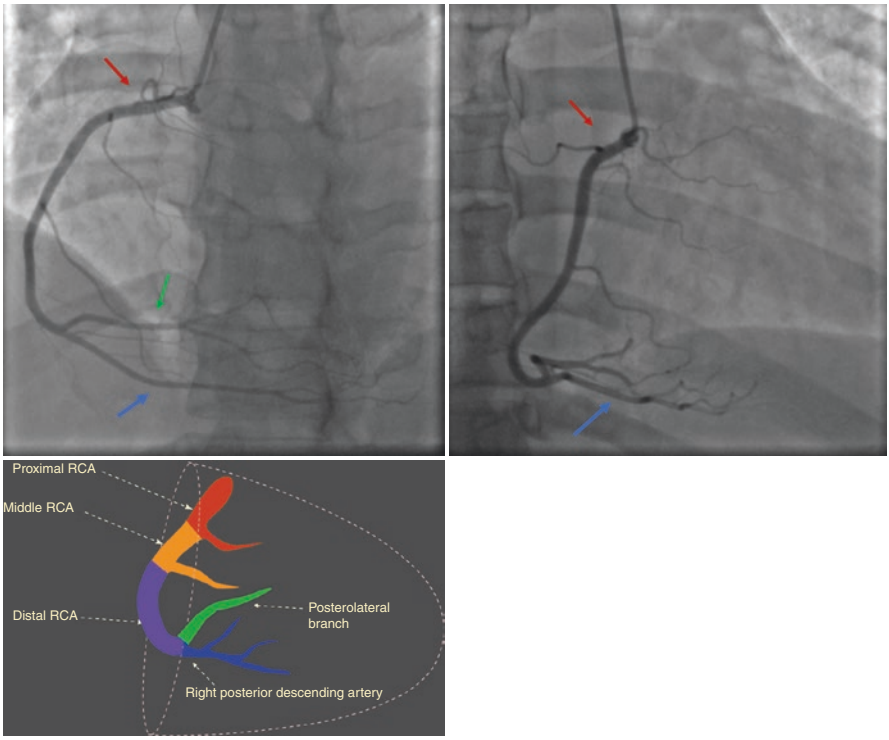

Fig. 6.14 Two images and an illustration of right coronary artery in the LAO and RAO views (red arrow "segment," proximal RCA; orange, middle RCA; purple, distal RCA; blue, right posterior descending artery; green, posterolateral branch)

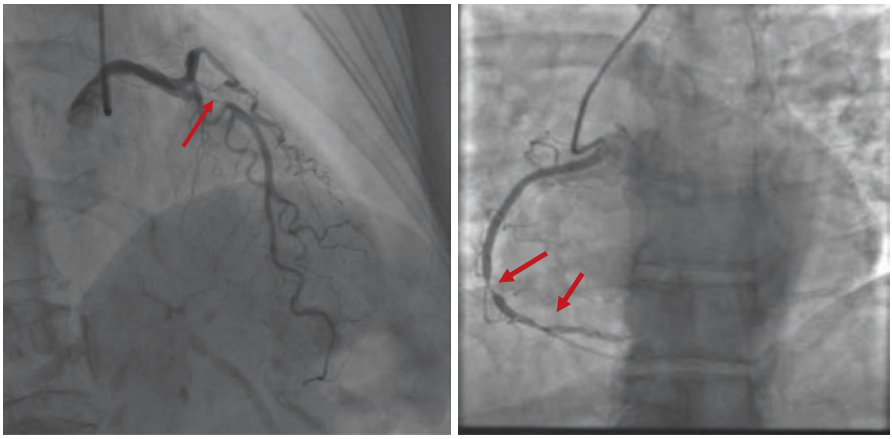

Fig. 6.15 Two examples of severe stenosis of the left anterior descending artery and right coronary artery (red arrow)

Coronary atherosclerotic lesions can be eccentric, and sometimes they are not well seen, or they appear borderline in severity by coronary angiography. To better assess the severity of these lesions, other diagnostic studies can be used during cardiac catheterization. Fractional flow reserve (FFR) assesses the coronary physiology, and intravascular ultrasound (IVUS) defines the burden of coronary vascular plaque.

For measurement of FFR, a wire with a pressure sensor at its distal portion is introduced into the coronary artery. After crossing the area of concern, the wire measures the pressure distal to the lesion. FFR is a ratio of the mean distal coronary pressure and the mean pressure in the aorta; two measurements are made, first at baseline (or the resting condition) and next after induction of maximal hyperemia (with either intracoronary or intravenous adenosine). A trans-lesional FFR value of ≤0.8 at baseline or after hyperemia correlates with a hemodynamically significant lesion and is considered abnormal. Several studies have validated the FFR to guide coronary revascularization (Fig. 6.16).

In IVUS, a miniaturized ultrasound probe is introduced inside the coronary artery. The probe produces ultrasound images of the vessel wall (intima, media, and adventitia). It accurately assesses the atherosclerotic plaque burden, plaque composition, degree of calcification, luminal area, and degree of stenosis (Fig. 6.17). A minimal luminal area of ≤5.9 mm^2 for the left main artery and ≤4.0 mm^2 for the

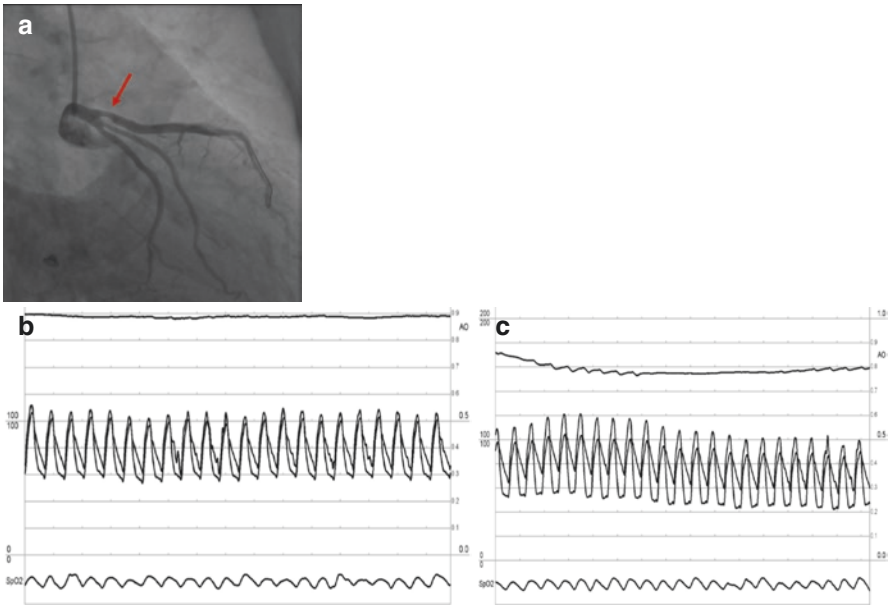

Fig. 6.16 (**a**) Coronary angiography of an intermediate lesion at the ostium of the left anterior descending artery (red arrow). (**b**) The FFR across the ostium of the left anterior descending at rest was 0.89. (**c**) After administration of intracoronary adenosine, the FFR drops to 0.72, which is evidence of hemodynamically significant lesion

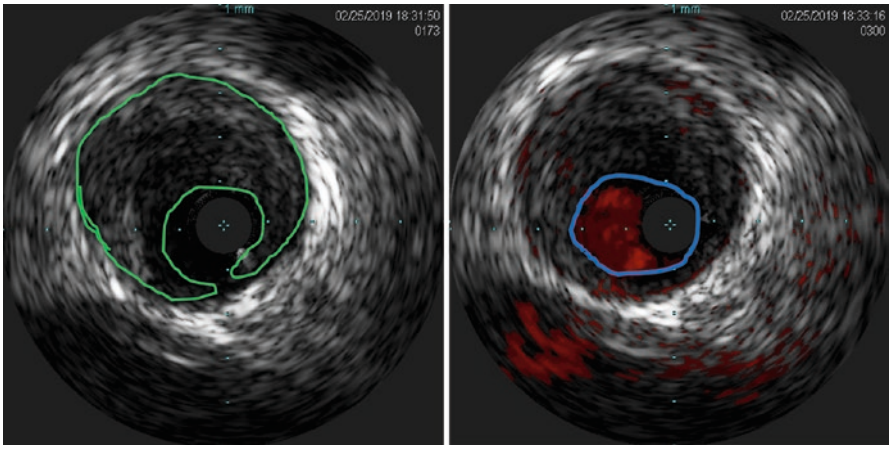

Fig. 6.17 IVUS of the same left anterior descending artery lesion as in Fig. 6.15, revealing extensive plaque burden (area inside the green line) and significant luminal narrowing (area inside the blue line)

proximal segment of the other vessels are evidence of hemodynamically significant disease.

For patients who need valve surgery, right heart hemodynamics often are measured during cardiac catheterization. Severe pulmonary hypertension and RV dysfunction portend high mortality. Right heart catheterization data are critical in preoperative decision-making and in optimizing postoperative management.

Further Readings

Kern MJ. The cardiac catheterization handbook. 6th ed. Philadelphia: Elsevier; 2016.
Moscucci M. Grossman & Baim's cardiac catheterization, angiography, and intervention. 8th ed. Philadelphia: LWW; 2014.
Zipes DP. Braunwald's heart disease. 11th ed. Philadelphia: Elsevier; 2018.

Medically Optimizing the Cardiac Surgical Patient

7

Afaf Anter and Zoheb Fazal

As to diseases, make a habit of two things — to help, or at least, to do no harm.

—Hippocrates

Introduction

Medical optimization of patients undergoing cardiac operations and high-risk vascular procedures is crucial in preventing postoperative complications and obtaining positive outcomes. Sound perioperative medicine practice is vital in minimizing such complications. Perioperative medicine also provides an opportunity for multidisciplinary collaboration among surgeons, anesthesia providers, and internists to provide high-quality patient care.

This chapter highlights the important facets of perioperative medicine and the management of common medical conditions in patients undergoing cardiac surgery. The main objective of perioperative care is the medical optimization of patients. Patients with many complex chronic conditions cannot be cured. Therefore, the goal of internists is only to stabilize such conditions before the operation. Failure to do so can result in higher rates of complications intraoperatively and postoperatively. The timing of surgery, kind of surgery, and the urgency of surgery also dictate such factors.

Medical optimization includes the identification of complex medical conditions, taking preventive measures to reduce complications, e.g., cessation of smoking, and managing the conditions in the perioperative period. The importance of communication with patients, their families, and other consultants involved in the care of the

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patient cannot be overstated. In the next few sections, we will highlight the components of medical optimization.

Management of Comorbid Medical Conditions

Hypertension

According to guidelines of the American College of Cardiology and the American Heart Association, published in September 2018, hypertension is defined as blood pressure greater than 130/80 mm Hg. This value is lower than the previously defined value, by the Eighth Joint National Committee, of blood pressure greater than or equal to 140/90 mm Hg. Hypertension is the second leading cause of death in the United States after smoking. Long-standing untreated hypertension is associated with cardiovascular disease, stroke, heart failure, chronic kidney disease, and retinopathy. In a case-control study evaluating cardiovascular-related mortality after elective surgery, isolated hypertension was associated with a 40% rate. Therefore, recognition and prompt management of hypertension in the perioperative setting is imperative.

When hypertension is diagnosed during the preoperative phase, evaluation for end-organ damage is essential. Although blood pressure can be labile in the perioperative setting, it is important to differentiate hypertensive emergency from hypertensive urgency. Hypertensive emergency is defined as systolic blood pressure greater than 180 mm Hg and diastolic greater than 120 mm Hg with the presence of acute end-organ damage. Hypertensive urgency, on the other hand, is elevated blood pressure without evidence of end-organ damage.

- *Management of hypertensive emergency* requires intensive therapy with intravenous medication aimed at a gradual reduction in blood pressure. The goal is that blood pressure decreases by 10–20% in the first hour, with an additional 5–15% reduction in the next 23 hours. There are, however, few exceptions, including acute ischemic or hemorrhagic stroke and aortic dissection where management will differ. Precautions should be taken to avoid excessive reduction in blood pressure as it can result in adverse outcomes including acute kidney injury and myocardial ischemia.
- *Hypertensive urgency* is generally treated with oral antihypertensives in addition to control of factors that provokes dramatic increase of blood pressure such as pain, anxiety, excess salt load (through the administration of intravenous fluids), and constipation.
- *Management of chronic hypertension* in the perioperative setting usually consists of continuing antihypertensives, but with exceptions. Several studies have shown a higher incidence of intraoperative hypotension with angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers; therefore, it is reasonable to withhold these medications the evening before or the morning of surgery. Diuretics are also generally withheld, given their impact on volume status and electrolyte balance. Beta blockers and clonidine generally are continued in the

perioperative setting because of the risk of rebound hypertension and tachycardia if they are stopped.

Ischemic Heart Disease

Ischemic heart disease is the leading cause of postoperative cardiovascular mortality. For patients undergoing elective cardiac surgery other than CABG, it is important that they be evaluated for ischemic heart disease preoperatively, especially if they have risk factors for atherosclerotic cardiovascular disease (old age, hypertension, diabetes, hyperlipidemia, prior stroke, smoking, positive family history, or peripheral arterial disease). A baseline electrocardiogram must be obtained; the presence of new ischemic changes, such as pathologic Q waves or ST/T abnormalities, should prompt further evaluation for ischemic cardiac disease, as with stress test or cardiac catheterization. The presence of active anginal symptoms also requires further evaluation.

- *Management of patients with stable ischemic heart disease.* Therapy for these patients has two goals:
  - Improvement in symptoms and quality of life
  - Improved prognosis

Medical management aims at mitigation of risk factors, and management of hypertension is important, as mentioned previously. In patients with known ischemic heart disease, continuation of beta blockers and antiplatelet drugs in the perioperative setting is imperative and has been shown to reduce cardiovascular mortality. Lipid-lowering therapy, as with statins, has also been beneficial, even in patients with normal serum lipid values. The importance of lifestyle modification, including smoking cessation, diet, and exercise, cannot be overstated.

- *Management of stable patients post myocardial infarction with planned CABG* will often result in postponement of surgery for 3–7 days.
- *Management of patients with acute coronary syndrome.* Acute coronary syndrome is a spectrum of clinical presentations for acute myocardial ischemia. It includes ST-elevation myocardial infarction, non-ST-elevation myocardial infarction, and unstable angina. The diagnosis is based on electrocardiographic findings and troponin release. Besides medical therapy, reperfusion and revascularization is the goal and there are two principal options for patients with coronary artery disease: percutaneous coronary intervention and CABG.

Congestive Heart Failure

Congestive heart failure is a clinical syndrome resulting from structural and functional impairment of ventricular filling or ejection of blood. As the incidence of chronic medical conditions, including hypertension and diabetes, is increasing, the

incidence of heart failure also is increasing. Preoperative assessment for patients with known or suspect heart failure includes an electrocardiogram, a chest radiograph (to assess for the presence of pulmonary edema), and an echocardiogram if the ejection fraction is unknown or to assess for underlying valvular abnormalities.

Elective surgery for patients with new onset or acute decompensated heart failure must always be postponed because of the associated high postoperative mortality. Medical optimization for patients with a history of congestive heart failure is focused on optimizing volume status and nutrition, correcting anemia, and mitigating risk factors such as diabetes, hypertension, and smoking (to be discussed in other sections).

Optimizing patients' volume status is key in management of heart failure; patients must be maintained in euvolemic state before operation. Euvolemia may be achieved through goal-directed therapy with beta blockers, angiotensin-converting enzyme inhibitors/angiotensin II receptor blockers, diuretics, and, possibly, mineralocorticoid antagonists, especially in patients who have reduced ejection fraction. For patients in acute decompensated heart failure with reduced ejection fraction, it is recommended that elective surgery be postponed for 3 months, when possible.

Malnutrition is present in up to 50% of patients with heart failure because of their high catabolic state and liver dysfunction resulting from congestive hepatopathy. Preoperative hypoalbuminemia, low prealbumin values, and abnormal body mass index are independent risk factors for complications. A low preoperative prealbumin value may be a risk factor for prolonged intubation, prolonged hospital course, infection, and mortality. Therefore, if severe malnutrition is present, consultation with a nutritionist is important, and supplemental nutrition may be needed. When possible, adequate time should be allowed for optimization of nutritional status, particularly if the operation is elective.

Lastly, proper perioperative management of medications is essential in patients with congestive heart failure. Beta blockers should be continued, especially in patients who have taken them before the currently planned operation, and they should not be initiated within a day of surgery. Angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers should generally be held a day before surgery and resumed as soon as possible after if the hemodynamic status permits. Similarly, diuretics and mineralocorticoid antagonists should be held the morning of surgery.

Anemia

Anemia is defined as hemoglobin levels <12 g/dL in women and <13 g/dL in men. Preoperative anemia is associated with impaired oxygen delivery to tissues and is an independent risk factor for postoperative complications. Preoperative anemia is an independent risk factor for morbidity and mortality in cardiothoracic surgery. In three large observational studies of patients undergoing CABG, preoperative hemoglobin less than 11 g/dL was associated with an increased incidence of postoperative heart failure, acute kidney injury, ischemic event, stroke, and cardiovascular

mortality. Cardiac surgery patients may have major perioperative blood loss and need for transfusions, so preoperative anemia screening and optimization of hemoglobin concentration is essential. Management of anemia starts with identifying the cause, then taking corrective measures, such as timely stoppage of anticoagulant medications and mitigation of the adverse effects of the anemia. In patients with preoperative iron deficiency anemia, preoperative IV iron supplementation (3 weeks or more) can be considered. In patients with severe anemia, high risk for postoperative anemia, Jehovah's Witnesses, and patients undergoing autologous donation, erythropoietin supplementation can be considered. However, the perioperative use of erythropoietin or iron supplementation for correction of anemia is controversial.

Atrial Fibrillation

Atrial fibrillation is the most common cardiac arrhythmia and is characterized by absence of distinct P waves and of irregular R-R interval on electrocardiogram. Perioperative management of atrial fibrillation includes rate and rhythm control and anticoagulation.

Rate control According to the American College of Cardiology/American Heart Association, a preoperative heart rate higher than 100 bpm is a contraindication to an elective operation. Therefore, maintaining a heart rate of less than 100 bpm should be a goal in patients with chronic atrial fibrillation; this rate is achieved mainly through the use of AV nodal blockers such as beta blockers and calcium channel blockers. If already prescribed, these medications should be continued in the perioperative period.

Management of rhythm Patients with paroxysmal atrial fibrillation may have either atrial fibrillation or sinus rhythm when they present for surgery. Correction of clinically stable atrial fibrillation is not necessary before operation, but symptomatic patients younger than age 65 are best managed with rhythm control. Rhythm management strategies include direct current cardioversion, which terminates atrial fibrillation and is superior to pharmacological cardioversion. Antiarrhythmia drugs should be continued in the perioperative period to maintain sinus rhythm. Since the American College of Cardiology/American Heart Association recommends surgical occlusion of the left atrial appendage in patients with atrial fibrillation undergoing cardiac surgery, it is advisable to proceed with the cardiac surgery. Surgical Maze procedure can be added with no additional risk to the patient.

Management of anticoagulation Stroke is a devastating complication of atrial fibrillation. Patients who have this arrhythmia are often treated with antiplatelets or anticoagulants to mitigate the risk of stroke. CHA_2DS_2Vasc score is a clinically validated tool to estimate stroke risk. Generally, warfarin should be discontinued at least 5 days before elective surgery in patients with CHA_2DS_2Vasc score <5. High-risk patients, including those with CHA_2DS_2Vasc score >5, mechanical valve, or

prior stroke, should have bridging with intravenous heparin or low-molecular-weight heparin. For patients undergoing CABG who are taking aspirin, the medication is generally continued in the perioperative setting.

COPD

COPD is a progressive lung disease characterized by fixed or irreversible airflow limitation associated with chronic inflammatory response in the airways and lungs. COPD is a risk factor for postoperative pulmonary complications, including pneumonia, atelectasis, and respiratory failure. Smoking is one of the biggest risk factors for COPD. Hallmark clinical features of COPD include chronic progressive dyspnea, chronic cough and sputum production, and recurrent lower respiratory tract symptoms. The diagnosis is mainly made through spirometry testing, with a post-bronchodilator FEV_1/FVC ratio of <0.7. Treatment includes the use of short- and long-acting bronchodilators (beta-2 agonists or anticholinergics), inhaled glucocorticoids, oral bronchodilators (theophylline), and/or phosphodiesterase-4 inhibitors, e.g., roflumilast. Systemic steroids and/or antibiotics are used for treatment of acute exacerbations.

In the preoperative assessment of patients with COPD, it is important to determine the severity of the disease, which can be achieved with spirometry. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) classifies the severity of disease based on post-bronchodilator FEV_1 as:

- Mild – GOLD 1: FEV1 > 80%
- Moderate – GOLD 2: 50% < FEV1 < 80%
- Severe – GOLD 3: 30% < FEV1 < 50%
- Very severe – GOLD 4: FEV1 < 30%

Another important consideration in the preoperative assessment of COPD is determination of the degree of hypoxemia and hypercapnia. Although knowing the baseline arterial blood gas value will not change management, it may assist in the development of a perioperative management plan, especially the anesthesia to be used.

The most important feature in medical optimization of COPD patients is smoking cessation. In addition, patients receiving chronic maintenance medical therapy for COPD should continue their medications, with the exception of theophylline, in the perioperative period; theophylline has a narrow therapeutic index and high risk for cardiac arrhythmias. Preoperative education of patients about self-administered incentive spirometry for training their inspiratory muscles may decrease the incidence of postoperative pulmonary complications. Elective operations should be postponed for at least 6 weeks for patients with acute exacerbation of COPD being treated with systemic corticosteroids.

Postoperative management for patients with COPD includes the use of scheduled inhaled albuterol or ipratropium; aggressive pulmonary toilet, including the

use of incentive spirometry; and minimal use of opioids to reduce the risk of respiratory depression and hypercarbia. For patients unable to tolerate incentive spirometry, noninvasive positive-pressure ventilation may be used to keep the lungs expanded.

Asthma

Asthma is a chronic inflammatory disease characterized by variable airflow obstruction and bronchial hyperresponsiveness. It is classified into intermittent or persistent (mild, moderate, severe) based on symptoms, spirometry values, and the frequency of acute exacerbations. Pharmacotherapy includes short- and long-acting inhaled bronchodilators, inhaled and oral corticosteroids, leukotriene modifiers, and the anti-IgE monoclonal antibody omalizumab.

As in COPD, the mainstay for perioperative management of patients with asthma includes smoking cessation and treatment of symptoms. Well-controlled asthma does not increase the risk of postoperative pulmonary complications, but elective operations should be postponed for patients with acute exacerbations. Acute bronchospasm is a risk in the perioperative period; therefore, scheduled treatment with shorting inhaled beta-2 agonists or anticholinergics should be used. Also, aggressive postoperative pulmonary toilet will minimize risk of respiratory decompensation, as mentioned for COPD.

Obstructive Sleep Apnea

Obstructive sleep apnea (OSA) is defined as cessation of breathing due to upper airway obstruction lasting more than 10 seconds. It is characterized by fatigue, excessive daytime sleeping, and poor concentration. Risk factors for OSA include obesity, advanced age, craniofacial abnormalities that obstruct the upper airways, alcohol consumption, and smoking. The condition is diagnosed with polysomnography testing. Treatment includes positive airway pressure; lifestyle modification, including weight reduction; alcohol and smoking cessation; and surgery.

OSA may be associated with complications, including hypertension, congestive heart failure, stroke, pulmonary hypertension, and diabetes. Thus, patients with untreated OSA are at high risk for perioperative cardiovascular and pulmonary complications.

During perioperative evaluation of patients with known OSA, it is crucial to determine the severity of OSA and patients' compliance with treatment. Severity is classified according to the apnea-hypopnea index, which is determined with polysomnography testing. It is reasonable for patients with mild to moderate disease who are compliant with positive airway pressure treatment to proceed with surgery. Positive airway pressure therapy should continue perioperatively. For patients with severe OSA and who are noncompliant with therapy or have uncontrolled comorbidities, elective surgery should be delayed until they are medically optimized.

Diabetes Mellitus

Diabetes mellitus is a metabolic disorder that is characterized by hyperglycemia resulting from impaired insulin secretion, decreased glucose utilization because of insulin resistance, or increased hepatic glucose production. Diabetes is most commonly classified into type 1 and type 2. Type 1 diabetes results from autoimmune destruction of pancreatic islet cells, resulting in insulin deficiency. Type 2 is due to variable insulin resistance, impaired insulin secretion, or increased hepatic glucose production. Poorly controlled diabetes is a risk factor for morbidity and mortality after cardiac surgery; complications include deep sternal wound infections and prolonged hospital stay.

During preoperative evaluation, patients with significant risk factors should be screened for diabetes. Fasting glucose >126 mg/dl or random blood glucose >200 mg/dl is strongly suggestive of diabetes. Values in this range should prompt retesting, with measurement of hemoglobin A1c also. For patients with known diabetes, it is important to determine long-term appropriate glycemic control. Patients with well-controlled diabetes with hemoglobin A1c <7% may proceed with surgery. Poorly controlled diabetes should be managed with combination of long-acting and short-acting subcutaneous insulin or, for inpatients awaiting surgery, insulin infusion per protocol. The goal of either regimen is blood glucose <180 mg/dl.

Thyrotoxicosis and Hyperthyroidism

Thyrotoxicosis refers to clinical manifestations resulting from elevated thyroid hormone values or action in peripheral tissues. It is most commonly due to an autoimmune condition such as Graves' disease, subacute thyroiditis, lymphocytic thyroiditis, toxic adenoma, or toxic multinodular goiter. Thyrotoxicosis is also precipitated by medications (amiodarone, lithium, interferon alpha, or tyrosine kinase inhibitors) or by contrast dye.

Screening for thyroid disorders is generally not recommended unless the patient is symptomatic. Measurement of thyroid-stimulating hormone is used for initial screening; if it is found abnormal, T4 and total T3 should be measured to test for subclinical thyroid disorders.

For patients undergoing non-emergent cardiac surgery, it is recommended that the operation be delayed until a euthyroid state is achieved. The management of hyperthyroidism includes administration of antithyroid drugs, radioactive iodine therapy, and surgery.

Hypothyroidism

Hypothyroidism results from inadequate production of thyroid hormone by the thyroid gland or decreased utilization of the hormone by peripheral tissues. Primary hypothyroidism is due to a disorder of the thyroid gland, whereas secondary

hypothyroidism is due to disruption in the hypothalamic-pituitary axis. The most common cause of hypothyroidism in the United States is Hashimoto thyroiditis, which is caused by autoimmune destruction of the thyroid gland. Certain medications may also affect thyroid hormone production, e.g., amiodarone, which may also cause hyperthyroidism.

Thyroid replacement therapy is the mainstay in treatment of hypothyroidism. It is safe and well tolerated. In perioperative settings, thyroid replacement therapy should be continued. For patients with moderate to severe disease, elective or nonurgent surgery should be delayed until a euthyroid state is achieved. Endocrinology consultation should be obtained for administration of intravenous thyroid replacement in patients with severe hypothyroidism who require emergency surgery.

Bleeding Disorders

Bleeding disorders include a spectrum of conditions arising from quantitative or qualitative platelet dysfunction and acquired or inherited factor deficiencies and/or inhibitors. Detection of these conditions during preoperative evaluation may be difficult, as routine preoperative testing is not enough to diagnose a bleeding disorder. Screening is recommended for patients with personal or family history of prolonged nonsurgical bleeding or a family history of bleeding disorder. As cardiac operations may pose high risk for bleeding, careful consideration and testing should be performed in patients with a suspected bleeding disorder, and consultation with a hematologist is recommended.

Acquired platelet dysfunction can occur with hepatic or renal impairment, hematological malignancies, or antiplatelet medications (aspirin or clopidogrel). For patients with mild platelet disorders undergoing cardiac surgery, perioperative treatment with antifibrinolytics, such as tranexamic acid or desmopressin, may be needed. For patients with moderate to severe platelet dysfunction, delay of operation and discontinuation of antiplatelet medications may be needed, if feasible; otherwise, transfusion of platelets may be considered.

Renal Insufficiency

Patients with renal disease are at increased risk for acute kidney injury during cardiac surgery. This occurs in up to 1–2% of patients, which may be the result of intraoperative hemodynamic changes, use of nephrotoxic medications, or poor oral intake perioperatively. Perioperative evaluation of patients with chronic kidney disease is focused on management of modifiable factors, particularly avoidance of nephrotoxic agents; optimizing volume status; and avoiding hypotension. In patients with end-stage renal disease who require dialysis, attention should be given to hyperkalemia, metabolic acidosis, volume status, and bleeding diathesis due to acquired platelet dysfunction, as mentioned above.

Tobacco Use

Tobacco use is associated with increased risk of postoperative complications, and cessation of smoking 3–4 weeks before surgery can improve outcomes. Postoperative risks associated with tobacco use include cardiovascular and cerebrovascular complications, pneumonia and respiratory failure requiring prolonged ventilation, and poor wound healing. Guidelines recommend use of behavioral counseling and pharmacological therapy to assist with smoking cessation, and nicotine replacement therapy is commonly used in the perioperative setting. The combination of behavioral counseling and nicotine replacement therapy may have a 50–70% cessation rate.

Perioperative medication management is discussed in Chap. 5.

Further Readings

2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery, 2014

Aronson S, Boisvert D, Lapp W. Isolated systolic hypertension is associated with adverse outcomes from coronary artery bypass grafting surgery. Anesth Analg. 2002;94(5):1079.

Cardiology Consult Manual: Hanna Z. Mieszczanska, 2018.

Jameson JL, Anthony AS. Harrison's principles of internal medicine 20e. New York: McGraw-Hill Education; 2018.

Jackson MB, et al. The perioperative medicine consult handbook. Cham: Springer International Publishing; 2015.

Kulier A, Levin J, Moser R, et al. Impact of preoperative anemia on outcome in patients undergoing coronary artery bypass graft surgery. Circulation. 2007;116:471–9.

Medically Challenging Patients Undergoing Cardiothoracic Surgery: Neal H. Cohen, MD MPH, 2009.

Pichette M, Liszkowski M, Ducharme A. Preoperative optimization of the heart failure patient undergoing cardiac surgery. Can J Cardiol. 2017;33(1):72–9.

Preanesthetic consultation for cardiac surgery in adults: Atilio Barbeito, MD, MPH, Aug 28, 2018.

Preoperative medical evaluation of the healthy adult patient, Gerald W Smetana, MD, 2019.

Part III

Room Preparation and Patient Monitoring

8 Cardiac Operating Room Setup and Preparation of the Patient for Surgery

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Prior proper planning and preparation prevent piss poor performance.

—James Baker

Preoperative Planning

The current practice at most institutions for elective cardiac surgical patients is to evaluate the patient before the day of surgery in the preoperative anesthesia clinic. The anesthesia provider who will be involved in the case interacts with the patient a day before surgery if the patient is in the hospital or just prior to the surgery for a same-day admission patient. Most patients will have had a general medical examination, left and right cardiac catheterization, echocardiogram, and carotid duplex evaluation before the day of surgery. The results of these tests should be readily available.

Other diagnostic tests (electrocardiogram [ECG], chest radiograph, and recent routine laboratory test results) also should be available and reviewed. If required, pulmonary functions tests and other studies should be ordered. Preparation for the scheduled procedure starts the day before surgery. The anesthesia provider must know the type of surgical procedure to be performed so that the anesthetic management and room setup can be arranged; for example, if the procedure is a minimally invasive one, additional equipment and setup will be needed.

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To build a sound anesthetic plan tailored to the patient's needs, the anesthesia team must know and discuss all the patient's available preoperative information, including the severity of valve dysfunction, coronary artery anatomy, myocardial function, airway anatomy, relevant anesthetic history, and the critical implications of these factors.

Emergency cardiac surgery presents special challenges. Because of the operative urgency, relevant medical and anesthetic history, cardiac catheterization results, echocardiogram reports, and laboratory test results may not be available, and the patient may be unable to provide the necessary information.

The main types of cardiac surgery can be separated into two categories: epicardial operations, that is, operations on the surface of the myocardium, such as coronary artery bypass grafting, and intracardiac operations, such as valvular operations. Some cardiac operations can be performed off bypass ("off-pump"), while most are performed with cardiopulmonary bypass (CPB; "on-pump"). The anesthesia team should be prepared for the need to institute emergent, or "crash," bypass at any moment.

Cardiac Operating Room Setup

Anesthesia technicians usually stock supplies and help set up intravenous lines and transducers for invasive monitoring in cardiac rooms. The anesthesia provider assigned to the case is responsible for making sure that all necessary supplies are available in the operating room.

Operating Room Setup

Following the same setup routine each day in the operating room increases efficiency and creates a streamlined work environment. This routine helps reduce errors and reaction time during the perioperative phase. Many institutions use electronic medical records; thus, after arrival to the operating room, the anesthesia provider should ensure that the electronic medical recording system is in working order. Many anesthesia providers use acronyms as memory tools to help them safely and efficiently set up the operating room. Two of the most common acronyms are SOAP-TIM (suction, oxygen, airway, pharmacy, table, IVs, medications) and MS-MAIDS-T (machine, suction, monitor, airway, IVs, drugs, special equipment, and transesophageal echocardiography [TEE]). These mnemonics help ensure that all essential items for delivery and life support services are prepared and ready at a moment's notice.

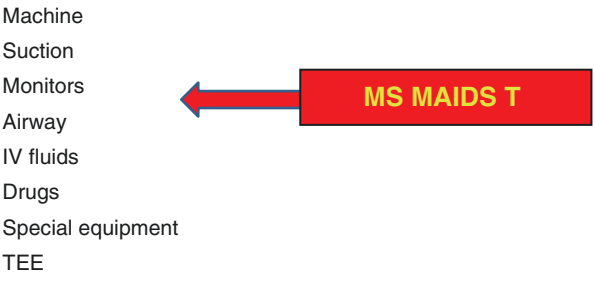

Fig. 8.1 Anesthesia machine check is a vital part of the OR setup. (**a**) *Proper monitor configuration,* (**b**) *Check of volatile agent dispensers*, *and* (**c**) *CO2 absorbent*

Machine Check

Each anesthesia machine or workstation must be checked thoroughly *daily* to ensure foolproof machine function. First, the anesthesia machine is turned off and on, and the machine check proceeds (most are automated with on-screen instructions). The anesthesia provider must make sure that the machine passes its intrinsic self-check; verify adequate pipeline and backup tank pressures; check that the vaporizers are adequately filled; confirm that the circuit is pressurized, as a check for any leaks; and make sure the CO_2 absorbent is not exhausted (Fig. 8.1).

Suction

A fully functioning suction setup is crucial for all cases. Suctioning can clear secretions to help visualization of the airway. Sutioning also functions as a rescue to evacuate the airway of debris, including vomitus and help deflate the lung in minimally invasive or robotic procedures that require single-lung ventilation. The anesthesia provider should make sure that the vacuum tubing is connected to the vacuum port on the suction canister and the Yankauer suction tip is connected through disposable connection tubing to the patient port on the canister. Next, the anesthesia provider should make sure that the on/off valve is turned on, that all unused ports of the suction canister are capped, and that the Yankauer suction tip is functional (Fig. 8.2).

Monitors

The anesthesia provider should make sure that the monitor display is appropriately configured with the elements needed for monitoring cardiac surgical patients. The screen usually is preprogrammed with options appropriate for the type of anesthesia used, for example, general, pediatric, perfusion (for the CPB portion of the case), or cardiac. The preprogrammed cardiac option should have the necessary hemodynamic parameters for cardiac surgery cases (Fig. 8.3). Also, the anesthesia provider

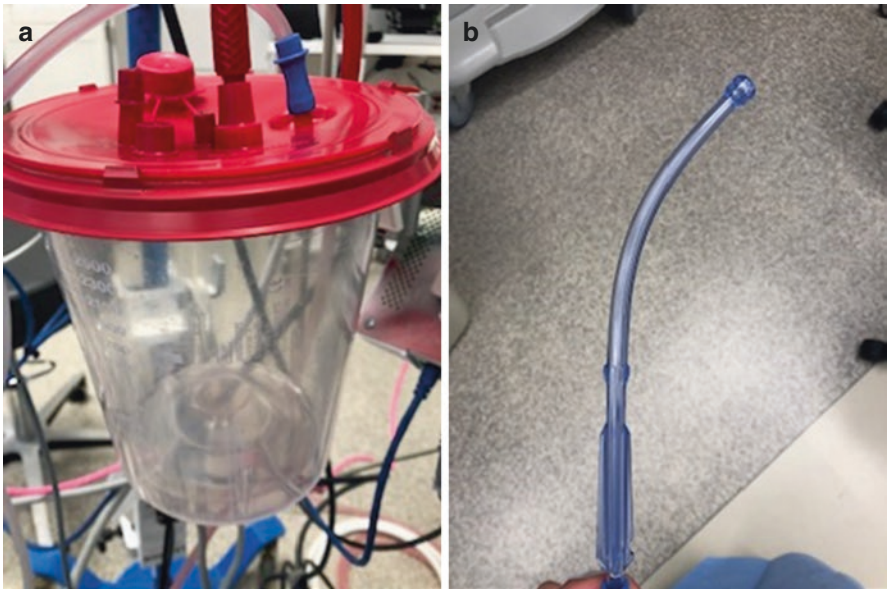

Fig. 8.2 Suction should be ready and functional; *suction canister should be sealed and have ample volume for adequate suction, b. Yankauer—used for clearing debris from patients' oropharynx*

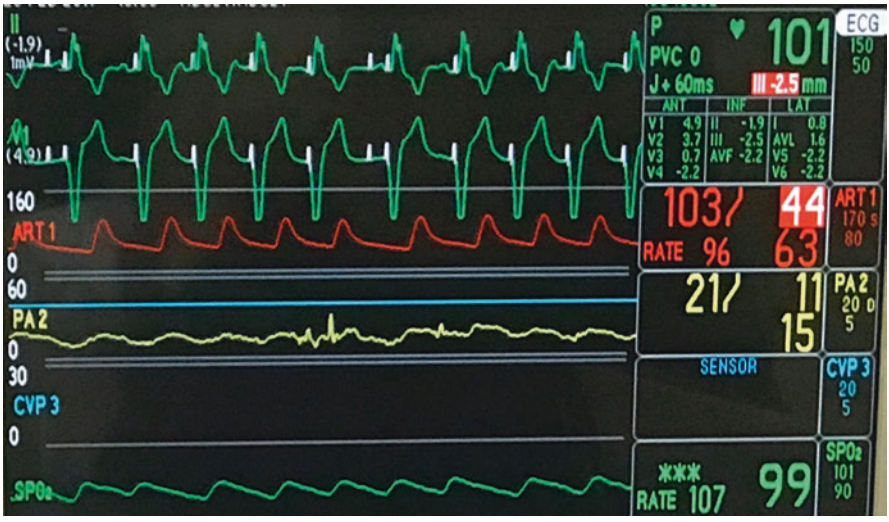

Fig. 8.3 Monitor display should be appropriately configured; *ensure waveforms for EKG, SpO2, temperature, end tidal CO2, NIBP, A-line, CVP +/− PA catheter are all present*

should make sure that the mirroring function is operational, so that other operating room screens can display the patient's vital signs. Please refer to Chaps. 4 and 9 for more information about monitoring and recognizing arrhythmias.

Cardiac cases require all standard American Society of Anesthesiologists monitors (pulse oximetry, capnography, temperature, ECG and noninvasive blood pressure monitoring) plus several additional monitors.

Additional Monitors for Cardiac Cases

- Additional ECG leads

A five-lead ECG is mandatory to increase the detection of myocardial ischemia. Additional ECG leads may be connected to the patient for a defibrillator and the TEE machine. The ECG signal may be "slaved off" from the primary ECG monitor.

- Pressure transducers

It is advisable to have a transducer manifold capable of holding three to six transducers. The transducer manifold should be attached to the operating room table so that it is maintained in a fixed position relative to the heart as the table is moved up and down (Fig. 8.4). At a minimum, an arterial line pressure transducer is required.

Fig. 8.4 Transducers on the manifold are primed and zeroed

If central venous pressure (CVP) and/or pulmonary artery (PA) pressure are monitored, two more pressure transducers are required; these can be attached to a 500 mL or 1000 mL saline bag via single or split tubing. The transducers are then attached to their respective pressure cables. It is advisable that the pressure cables be labeled for ease of identification throughout the case. The manometer tubing should be primed, and the transducers zeroed. The dial on the pressure bags can be turned off to avoid dripping of fluid until the patient is in the room and attached to the monitors.

- Invasive blood pressure (A-line)

An arterial line is used in every case. It is essential that blood pressure be monitored beat-to-beat during cardiac cases; further, with non-pulsatile flow during CPB, the arterial line is the only means of monitoring mean arterial pressure.

- Central line

The CVP/Central line is vital during open-heart surgery, as it allows for rapid resuscitation and additional monitoring. The decision of whether to place this line pre-induction or post-induction depends on each patient and the anesthesiologist. Placing the line before induction assures reliable venous access. The type of central line used may be institutionally dependent; however, commonly, a two-lumen 9 French introducer is used (Fig. 8.5). This introducer has one lumen for use as an infusion and/or CVP line and another for use as a bolus/blood-product line. Usually, it has also a central introducer hub to allow for insertion of a PA catheter if needed.

Monitoring CVP values and trends is a useful, albeit nonspecific, means of gauging preload.

For setup, the anesthesia provider needs to prepare:

(a) Central venous catheter kit
(b) Flushes

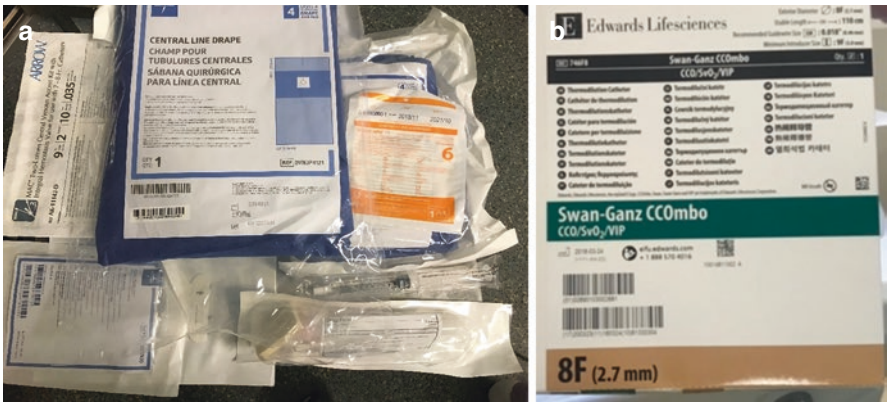

Fig. 8.5 (**a**) An example of a 9 French two-lumen central venous catheter kit. (**b**). CCO/MvO_2 Swan–Ganz/pulmonary artery catheter kit. (*CCO* continuous cardiac output, MvO_2 mixed venous oxygen saturation)

(c) Sterile gloves, gowns, towels, and whole-body drape
(d) Line dressing. An antibiotic-impregnated dressing is suggested
(e) Mayo stand or an operating room table
(f) Ultrasound machine with sterile ultrasound probe cover
(g) Swan Ganz catheter if required for the operation

Not all cardiac cases require a PA catheter; however, a PA catheter is the only way to directly and continually monitor PA pressure, and it can be a helpful tool in determining left ventricular filling. There are many types of Swan Ganz catheters: regular PA catheters, pacing PA catheters (useful in patients undergoing minimally invasive valve surgery with conduction problems), continuous cardiac output PA catheters, and mixed venous oxygen saturation catheters. Often, a combination of these catheters is incorporated into a single catheter.

- Central Nervous System monitoring
  Cardiac surgery patients are at increased risk of stroke, postoperative delirium, and postoperative cognitive dysfunction. Processed electroencephalographic monitoring, such as the bispectral index and cerebral oximetry, are monitoring modalities that help regulate the depth of anesthesia and the adequacy of blood flow to the brain, respectively. Both monitors are placed on the patient's forehead.
  – The bispectral index
    The bispectral index is used to assess the depth of anesthesia. It converts EEG data into a numerical scale (0 = electrical silence to 100 = full wakefulness), which allows for easier interpretation by anesthesia providers, especially during CPB, when the perfusionist may be the principal provider of anesthetics via the CPB circuit.
  – Cerebral oximetry
    Cerebral oximetry is a noninvasive monitor that uses infrared light and the optical properties of hemoglobin, which permit early recognition of hemoglobin desaturation in superficial cortical layers; hemoglobin desaturation may be a surrogate measure of cerebral perfusion (Fig. 8.6).
- Point-of-care glucose monitoring

For reasons described elsewhere, it is essential to track a cardiac patient's blood glucose levels (Fig. 8.7). However, with regards to preparedness, it is recommended

Fig. 8.6 An example of a cerebral oximetry monitor

Fig. 8.7 Point of care (POC) glucose monitoring

that a point-of-care glucose monitor be available in the room. These monitors have quality-control routines, which usually are run daily. Blood samples can be obtained from the central line or A-line, thus obviating the need for lancets, alcohol swabs, or 2 × 2 gauze sponges.

Airway

As with every operation, one of the primary responsibilities of the anesthesia provider in cardiac operations is managing the airway. Endotracheal tubes (ETTs) and laryngeal mask airways should be readily available. If a problematic airway is suspected, proper intubation equipment, that is, a video laryngoscope or a fiberoptic bronchoscope, should be in the room. The significance of this precaution cannot be overstated, as induction of anesthesia can lead to loss of airway patency (Fig. 8.8).

Cardiac patients have limited reserves and may be sensitive to apnea. They require adequate oxygenation (to help avoid ischemia) and may have associated respiratory disease. Therefore, backup plans for airway management, such as laryngeal mask airways and video-assisted laryngoscopy, must be available and ready to use at a moment's notice. Various airway assist devices can be deployed—for example, nasal or oral airways—to help overcome airway obstruction. It is important that these airway adjuncts be immediately available in order to minimize nonventilated time. In some instances, such as minimally invasive valve operations, coronary artery bypass graft, or robotic cardiac operations, lung isolation is required; this is best accomplished by the use of double-lumen tubes or a bronchial blocker.

In *any* case, it is essential that the room be stocked with these vital airway supplies:

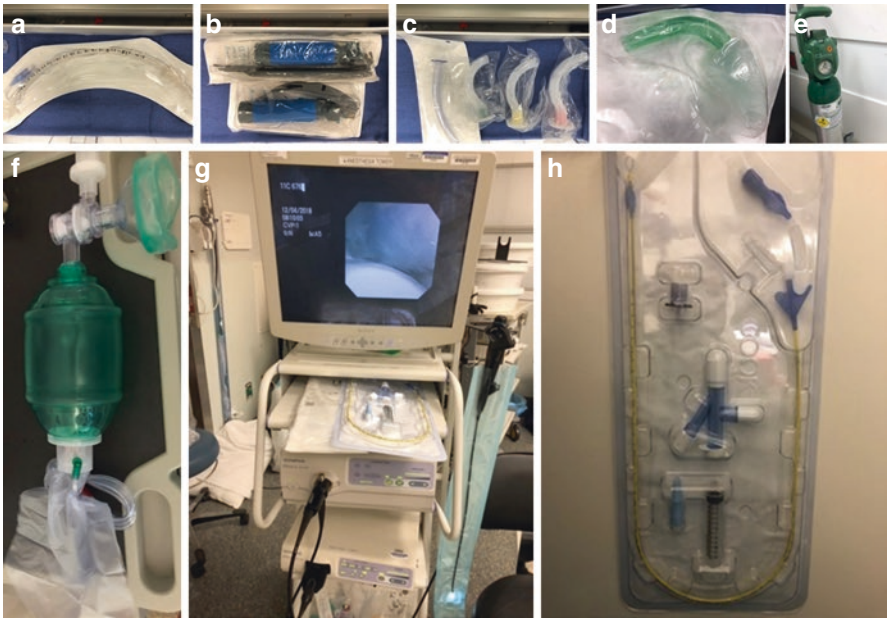

Fig. 8.8 (**a**–**h**) Airway equipment. (**a**) Endotracheal tube (ETT) with balloon cuff attached. (**b**) Laryngoscope blades. (**c**) Adjunct airway equipment—pictured is a nasopharyngeal airway on the far left, accompanied by various sizes of oral airways. (**d**) Laryngeal mask airway (LMA). (**e**) oxygen tank. (**f**) Bag mask valve (BMV). (**g**) Fiberoptic bronchoscope (FOB). (**h**) Bronchial blocker

(i) Styletted ETT (usually size 8.0 for female patients, 8.5 for male patients) with a syringe
(ii) Laryngoscope handle with functioning Macintosh No. 3 or 4 blades or Miller blades
(iii) Airway adjuncts (multiple sizes of oral airways), tongue blades, and mask strap
(iv) Silk tape to secure ETT
(v) Tape or protectors for the eyes
(vi) Orogastric tube with lubricant
(vii) Circuit mask
(viii) Ambu bag
(ix) Oxygen tank
(x) If bronchial blocker/double-lumen tube will be used, spray the endobronchial blocker and fiberoptic scope with lubricant or silicone spray. The lubricant can be applied by using gauze. It is often helpful to lubricate the tip of the double-lumen tube for easier insertion through the larynx

IV Fluids

Intravenous (IV) fluids are drugs. The anesthesia provider must be judicious in the amount of IV fluid delivered to the patient to avoid the development of a markedly positive fluid balance, which may affect ventricular preload and myocardial contractility. Two fluid lines should be prepared for central-line infusions. For volume resuscitation, a balanced crystalloid solution usually is primed in a fluid line, with a large-caliber tubing running through a warming device. Another fluid line is primed to be a fluid carrier line, with multiple stopcocks or other attachments for the delivery of medication drips. There are two main types of fluids: colloids and crystalloids. In addition to these, other volume expanders—such as packed red blood cells, which have the added benefit of built-in oxygen-carrying capacity—should be available. It is essential to recognize that the CPB circuit is a generator of extracellular volume expansion; the expansion can affect patient's fluid balance, hematocrit and the rheology of the blood, as well as the intraoperative anesthetic management—namely dilution of plasma concentrations of drugs, which may result in the need to re-dose medications. Recall that most total body water, about 65%, is intracellular, while the remainder is extracellular fluid that is distributed to the interstitial space and plasma. Of the IV fluids available, balanced crystalloid solutions that contain magnesium and potassium, such as Plasma-Lyte solution, have several benefits, including maintenance of renal function, avoidance of hyperchloremic metabolic acidosis, and perhaps reduced incidence of postoperative atrial fibrillation. Crystalloid IV fluids can generate volume expansion of 20% of the administered fluid, and colloids generate even more expansion. In the post-CPB state, both crystalloids and colloids lose their oncotic volume-expanding effects and are siphoned off into the interstitial space. As a result, CPB patients are prone to developing interstitial edema postoperatively.

The main colloid used in the anesthesia management of cardiac surgical patients is albumin (human-derived), which is available in 5% and 25% concentrations. The main crystalloids are normal saline and lactated Ringer's solution. When considering crystalloid solutions, it is essential to try to maintain physiologic tonicity. This requirement has significant implications, as normal saline is *hyper*tonic with relation to plasma; hypertonicity has been associated with renal vasoconstriction (in animal studies), as well as hyperchloremic acidosis when administered in large quantities. Lactated Ringer's solution is mildly hypotonic relative to plasma.

At present, the literature does not allow one to make a final determination as to whether crystalloid or colloid substitution should be preferred for cardiac surgery; both colloids and crystalloids are reasonable to use, with isotonic solutions being favored.

In preparing the operating room for IV fluid administration, a fluid warmer should be ready for use in most situations. This is *especially* true for redo and off-pump cases.

The goals and parameters of IVF management include maintenance of the following:

- Mixed venous oxygen saturation (SvO_2) >65%
- Mean arterial pressure (MAP) >65 mmHg
- Central venous pressure (CVP) 8–12 mmHg
- Cardiac index (CI) >2 L/min/m^2
- Pulmonary artery occlusion pressure (PAOP) 12–15 mmHg
- Diuresis >0.5 mL/kg/h

Drugs

Perhaps the most time-intensive portion of the cardiac operating room setup is the preparation of the drugs that are needed for these complicated cases. The medications include those for induction, pressors, muscle relaxants, antihypertensives, heparin, protamine, antifibrinolytics, and, potentially, inotropes. For a more detailed discussion of drug pharmacology, please refer to Chap. 3.

Standard Anesthesia Medications

Induction and rescue medications should be pre-drawn and appropriately labeled.

- Induction medications:
  - Propofol, etomidate, or midazolam can be used to induce anesthesia in cardiac surgery patients. Care should be taken to choose the agent and dose that will provide the greatest hemodynamic stability. When it is essential to maintain sympathetic drive and tachycardia is not deleterious, such as in patients with cardiac tamponade, ketamine can be used as well.
  - Opioids, typically fentanyl or sufentanil, are titrated to provide a balanced and hemodynamically stable induction.
- Analgesia:
  - Usually accomplished with fentanyl or sufentanil.
    Adequate analgesia blunts the patient's sympathetic response to pain. If left unchecked, the response may put undue stress on the cardiovascular system and precipitate myocardial ischemia.
- Muscle relaxation:
  - Non-depolarizing neuromuscular blockers, such as rocuronium, vecuronium, and cisatracurium may be used. Pancuronium had been used often in the past, when cardiac operations were longer, and because of its mild sympathomimetic properties, which counterbalance the bradycardia produced by opioids and induction agents. However, for fast track and rapid extubation after surgery, residual neuromuscular weakness after operation is undesirable, so pancuronium is used less often.
  - Succinylcholine has advantages when rapid securing of the airway is needed. In the era of sugammadex, rapid relaxation can also be achieved with rocuronium, especially if it is administered in a moderately higher dose than

its ED95 intubation dose. The choice of paralytic agent may be an institutional or individual preference if the patient does not have significant comorbidities; otherwise, the choice may be tailored based on the presence of renal or hepatobiliary disease.

Syringes for Bolus Doses

Cardiac surgical patients may decompensate at any time. Heparin and other emergency drugs should be drawn up and ready for use in the event of a "crash" on CPB (Fig. 8.9).

Table 8.1 lists common cardiac medications that should be prepared, with suggested syringe sizes and concentrations.

Drips

Most institutions have a dedicated "Heart Bag" or a cardiac tray that is prepared by the pharmacy. It includes pre-made drips, which may include regular insulin,

Fig. 8.9 Syringes with medications drawn for bolus doses; ostensibly separated by drug class, induction, maintenance, vasopressors, and vasodilators should be easily identifiable and separated to help prevent mistaken administration

Table 8.1 Common medications used in the cardiac surgery operating room

Drug	Syringe	Concentration
Propofol	20 mL	10 mg/mL
Etomidate	10 mL	2 mg/mL
Aminocaproic acid (on-pump-only cases)	60 mL × 2	10 g/mL
Heparin	30 mL	1000 units/mL
Rocuronium	10 mL	10 mg/mL
Fentanyl	20 mL	50 μg/mL
Midazolam	10 mL	1 mg/mL
Succinylcholine	5 mL	20 mg/mL
Nitroglycerin	20 mL × 2	40–50 μg/mL
Phenylephrine	20 mL × 2	100 μg/mL
Epinephrine	20 mL	4 μg/mL
Norepinephrine	20 mL × 2	4–16 μg/mL
Atropine	5 mL	0.1 mg/mL
Calcium chloride	10 mL	100 mg/mL
Glycopyrrolate	3 mL	0.2 mg/mL
Lidocaine	5 mL	20 mg/mL

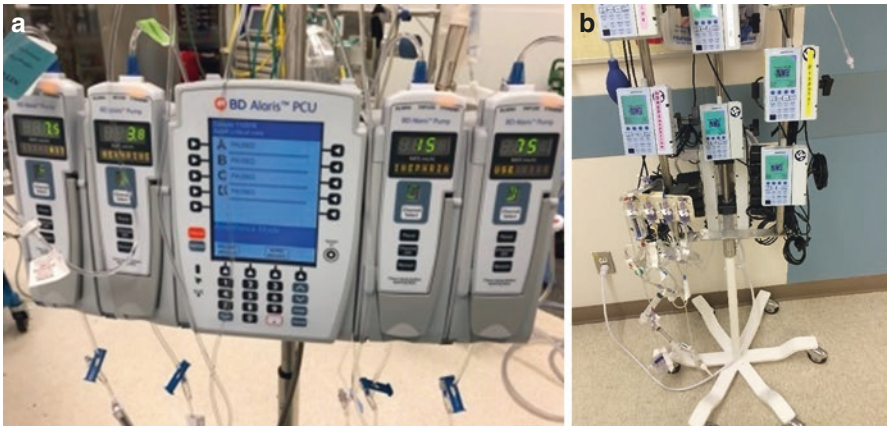

Fig. 8.10 (**a**) Alaris programmable pumps with drips primed. (**b**) Baxter programmable pumps on IV pole each labeled for cardiac drug infusion

milrinone, epinephrine, norepinephrine, phenylephrine, dopamine, and nitroglycerin. It is advisable to prime nitroglycerin and a pressor, such as norepinephrine, and have them ready for use on an infusion pump (Fig. 8.10). Concentrations of drugs used may vary among institutions. Table 8.2 list common cardiac drips.

A good practice for safe medication delivery is to label the ends of all continuous infusion lines with large readable print and color-coded stickers that correctly identify the medication.

Common Cardiac Drips

Table 8.2 Common cardiac drips*

Drug (Starting infusion rate)	Concentration	Dilution
Aminocaproic Acid Load: 5–10 g then (1.5 g/h)	20 g/250 mL	80 mg/mL
AMIODARONE Load: 150 mg over 10 min, then (1 mg/min for first 6 h)	450 mg/250 mL	1.8 mg/mL
Dexmedetomidine Load: 1 mcg/kg over 20 minutes then (0.2–0.7 mcg/kg/h)		
Diltiazem (2.5–15 mg/h)	125 mg/125 mL	1 mg/mL
Dobutamine (2.5–20 mcg/kg/min)	500 mg/250 mL	2 mg/mL
Dopamine (0.5–20 mcg/kg/min)	400 mg/250 mL	1.6 mg/mL
Epinephrine (0.01–0.05 mcg/kg/min)	2 mg/250 mL	8 mcg/mL
Esmolol Load: 150 mcg/kg over 30 seconds then (10–300 mcg/kg/min)	2000 mg/100 mL	20 mg/mL
Fenoldopam (0.1–0.16 mcg/kg/min)	10 mg/250 mL	40 mcg/mL
Milrinone Load: 50 mcg/kg then (0.5 mcg/kg/min)	20 mg/100 mL	200 mcg/mL
Isoproterenol Load: 1–4 mcg then (0.01 mcg/kg/min)	4 mg/250 mL	16 mcg/mL
Lidocaine Load: 100 mg then (1 mg/min)	2 g/250 mL	8 mg/mL
Nicardipine (2.5–15 mg/h)	40 mg/200 mL	200 mcg/mL
Nitroglycerin (1 mcg/kg/min)	100 mg/250 mL	400 mcg/mL
Nitroprusside (0.1–2 mcg/kg/min)	50 mg/250 mL	200 mcg/mL
Norepinephrine (0.1 mcg/kg/min)	4 mg/250 mL	16 mcg/mL
Phenylephrine (50–150 mcg/min)	100 mg/250 mL	400 mcg/mL
Regular insulin (4–10 U/h)	100 units/100 mL	1 unit/mL
Rocuronium Load 0.6–1.2 mg/kg then (8–12 mcg/kg/min)	1250 mg/250 mL	5 mg/mL
Vasopressin (0.02–0.04 units/min)	20 units/100 mL	0.2 unit/mL

Antibiotic Prophylaxis

Antibiotics are given prophylactically before all cardiac surgical operations to prevent surgical site infection or endocarditis. Treatment is initiated intravenously within the hour preceding surgical incision; a good time to begin infusing the

antibiotic is during surgical skin preparation. According to the Society of Thoracic Surgeons guidelines, the principal recommended prophylactic antibiotic for adult cardiac surgery is a first-generation cephalosporin, usually cefazolin. 2 g of cefazolin is a reasonable preoperative prophylactic dose for a patient weighing more than 60 kg, with a second dose of 1 g administered after 3–4 hours.

In patients who are allergic to penicillin or cephalosporins, vancomycin is recommended as the primary alternative. Vancomycin is given in a dose of 1 to 1.5 g or a weight-adjusted dose of 15 mg/kg. It is recommended that vancomycin be given intravenously over 1 hour and that the infusion is concluded within 1 hour before surgical incision. In patients at high risk for staphylococcal infection, vancomycin is added to cefazolin.

Special Equipment

- Body-warming devices: There are many varieties of body-warming devices. These can be subdivided into active or passive devices, based on whether they generate heat or are merely reflective. Active warming systems include forced-air warmers, resistive warming blankets, water-filled mattresses, and circulating water garments. An active warming system is mandatory during open-heart surgery in order to reverse hypothermia after CPB and maintain normothermia. The anesthesia provider should make sure that the controller device is plugged to a power source, and the blanket or pad is on the bed.
- Pacemaker: A functional pacer box is always required in the operating room. It should be tested for proper functioning, and fresh batteries should be available in the event of a depleted battery.
- Defibrillator: A defibrillator must be available during open-heart surgery (Fig. 8.11). Adhesive defibrillator pads can be used for defibrillation or

Fig. 8.11 Transcutaneous pacing/defibrillator device; it is essential that the functionality of the device is confirmed prior to commencing the procedure

transcutaneous pacing and should be applied to the patient in minimally invasive cases where sternotomy is to be avoided, in redo sternotomy cases, or if a malignant arrhythmia can be anticipated prior to sternotomy. This is best accomplished prior to surgical skin preparation and draping. One electrode is placed on the lateral side of the lower chest close to the cardiac apex, and the other over the right scapula.

Regardless of whether adhesive pads or traditional paddles are used, the anesthesia provider should verify that the defibrillator is in working order and all necessary supplies are available. During the sternotomy portion of a heart case, the surgeon typically uses internal paddles.

- Operating room table: The anesthesia provider should make sure that the table is plugged to a power source, and the table control is functioning well. Also, two clean arm boards should be available for use before the arms are tucked.
- Transesophageal echocardiogram (TEE): TEE is now the standard of care for cardiac surgeries. It provides superior visualization of the heart, valves, and major vessels branching from the heart. It also allows for real-time observation of cardiac function during heart surgeries without interfering with the surgical field. It is useful in the heart room as a monitoring and diagnostic tool. Part of the cardiac OR preparation is to set up the TEE machine. Please refer to Chap. 12 for further information on TEE.

  Setup TEE machine:

  (i) Plug in the electrical cord to a power source and turn the machine on.
  (ii) Connect a clean TEE probe to the machine and lock it into the receptacle.
  (iii) Type in the patient's demographics.
  (iv) Enter the name of the anesthesiologist performing the TEE exam and the name of the responsible surgeon.
  (v) Enter the planned *procedure*.
  (vi) A screenshot of the demographics dialog screen can be acquired as a still image for easy review later.
  (vii) Verify that ultrasonography gel is available.
  (viii) Verify that a TEE mouth guard/soft mouth block is available.

Patient Preparation

(a) Immediate Preoperative Assessment

After setting up the OR, the anesthesia provider will see the patient in the preoperative holding area. This may be the first time the provider who will perform the anesthetic encounters the patient face to face. By this time, the patient should be dressed in a patient gown and have undergone a chlorhexidine bath. After introductions and a brief explanation of the anesthesia provider's role, a focused preoperative assessment is carried out by a combination of record review, patient and/or family interview, physical examination, and communication with other cardiac surgery team members. Next, the anesthesia provider

should make sure that a blood product transfusion consent and an anesthesia consent have been obtained, and if not, obtain such consent(s) from the patient or his/her healthcare power of attorney. The anesthesia provider should verify that the surgical consent matches the patient's understanding of the surgery being performed. Informed consent is a crucial part of the preoperative interview and must be obtained for all patients prior to administering any sedation. The anesthesia provider begins by describing the planned anesthesia technique, including procedure descriptions in layman's terms. Then, the anesthesia provider discusses the risks in appropriate detail without any minimization, since cardiac surgery is associated with serious and frequent complications including stroke, myocardial infarction, death, acute kidney injury and transfusion of blood products. A realistic description of the risks involved is tailored to the patient; some patients require more details of potential risk than others. While it is primarily the responsibility of the surgical team to advise the patient on the type of prosthetic valve to be used (in cases involving valve surgery), it is prudent for the anesthesia provider to confirm this information with the patient and surgeon before sedation, lest there is a misunderstanding. Similarly, anesthesia providers should inform themselves about the possibility of radial artery harvesting. Should the radial artery be required as a bypass conduit (usually the left radial artery), the anesthesia provider should not access that vessel under any circumstances.

Important considerations for the cardiac surgery patients on the day of surgery:

- Change in physical condition since the most recent visit to the preoperative clinic
- Current dyspnea or chest pain
- Swallowing problems, esophageal disease, or prior upper gastrointestinal surgery, since TEE is standard of care
- Prior difficulty with anesthesia by the patient or family members
- Previous exposure to heparin or a history of heparin-induced thrombocytopenia
- Blood dyscrasias or abnormal bleeding tendencies not related to anticoagulation, antiplatelet therapies, or direct thrombin inhibitors
- For redo cardiac surgery cases: additional peripheral IV access is advisable due to the increased risk of bleeding
- Check for availability of cross-matched blood with the blood bank
- Patients on beta blockers should receive their daily beta-blocker dose with a sip of water if hemodynamically feasible
- A documented negative pregnancy test in women of child-bearing age who have not undergone surgical sterilization

(b) Transportation to the operating room

Once the above assessments have been performed, the patient can be transferred to the operating room. If the patient has an intra-aortic balloon pump or a ventricular assist device, is intubated, or is on multiple vasoactive drips, he/she may have to be evaluated in the unit where they have been admitted.

Transporting such patients to the operating room may be challenging and may require the help of additional personnel. Patients who have an in-dwelling intra-aortic balloon pump can be transported with the help of a perfusionist directly to the operating room. Critically ill cardiac patients usually cannot be separated from their cardiovascular infusions and need to be transported with those infusions running. Emergency drugs must be available during transportation.

(c) Preparing the patient for induction
An arterial and central line should be established next. This can be achieved in the holding area or in the operating room. For most cardiac surgical cases, it is a matter of patient safety to establish an arterial line prior to induction.

If the invasive lines are placed prior to induction, they should be placed with the use of local anesthetic, blood pressure recording, EKG and pulse oximetry, light sedation, supplemental oxygen, and full sterile barrier precautions in the case of central line placement. Ultrasound-guided techniques are the method of choice as they are deemed safer and faster compared to surface landmarks only. In stable and asymptomatic patients, the central line can also be placed after the induction of anesthesia.

Once the patient has been transferred to the OR, the monitors and defibrillator pads (if using) are applied, and all IV access is secured. A "time-out" pre-induction verification is performed. The patient is reidentified, surgery and surgical site are verified, patient allergies are checked, the risk of blood loss is estimated, and the availability of cross-matched blood is confirmed. Any concerns for airway or adequacy of IV access should be addressed at this point. The OR staff must verify that no essential surgical equipment is missing. Once no outstanding concerns are present, the patient is ready for anesthesia induction.

Further Reading

1. Engelman R. (2007-reaffirmed 2011) The Society of Thoracic Surgeons Practice Guideline Series: Antibiotic Prophylaxis in Cardiac Surgery, Part II: Antibiotic Choice. Ann Thorac Surg. 2007;83:1569–76.
2. Gravlee GP, Shaw AD. A Hensley's practical approach to cardiothoracic anesthesia. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2019.
3. Kaplan J. Kaplan's cardiac anesthesia for cardiac and none cardiac surgery. Philadelphia: Saunders; 2017.
4. Shaw A. Fluid Management in Cardiac Surgery. Colloid or Crystalloid? Anesthesiology Clin. 2013;31(2013):269–80.

9 Intraoperative Hemodynamic Monitoring for the Cardiac Surgery Patient

Christopher Potestio and Xiaolu Xu

Doubt must be no more than vigilance, otherwise it can become dangerous.

—Georg C. Lichtenberg

Introduction

During cardiac surgery, drastic hemodynamic changes may occur at any given time prior to initiation of cardiopulmonary bypass (CPB) and during the separation from CPB. A number of factors can cause these changes, including the patient's baseline cardiopulmonary function at the time of surgery, the effect of the anesthetics on the patient, blood loss, and the physiologic responses to CPB. Effective hemodynamic monitoring is the cornerstone in the management of patients undergoing cardiac surgery to help reduce the risk of complications and improve outcomes. These monitors should ideally be easy to use and accessible, operator independent, with rapid response times, and facilitate the anesthesia provider with goal-directed therapies. In addition, these monitors should give accurate, reproducible, and interpretable data.

This chapter focuses on hemodynamic monitoring in addition to the standard monitors recommended by the American Society of Anesthesiologists for all other patients receiving anesthesia (pulse oximetry, noninvasive blood pressure measurement, electrocardiogram, temperature, and end tidal CO_2).

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Meaning of Hemodynamic Monitoring

Hemodynamic monitoring is the measurement and interpretation of blood flow dynamics to evaluate cardio-circulatory performance. Since monitoring is not therapy, the anesthesia provider must know how to interpret the hemodynamic monitoring data to be able use it as a guide for therapeutic decision-making. It is based on both noninvasive and invasive measurements of features of cardiovascular physiology, specifically effective circulating volume (preload), regulated arterial vascular tone (afterload), and adequate intrinsic cardiac function (cardiac output/stroke volume). Hemodynamic monitoring can be achieved in two different ways by measuring either static or dynamic variables. These variables are used as a surrogate of tissue perfusion. The intermittent hemodynamic parameters focus mainly on static measurements such as the following:

- Arterial pressure and mean arterial pressure
- Central venous pressure
- Pulmonary artery wedge pressure
- Heart rate
- Urine output

The dynamic variables focus on recognizing signs of tissue hypoxia, although difficult to obtain, are more reflective of the actual perfusion and filling status of the patient such as the following:

- Continuous invasive arterial pressure monitoring
- Continuous cardiac output
- Systolic pressure variation
- Stroke volume variation monitoring

Goals of Hemodynamic Management

The goals of hemodynamic management are to optimize the balance between tissue oxygen supply and oxygen demand. This is achieved by maximizing oxygen delivery while ensuring appropriate fluid management, so avoiding hyper- and hypovolemia. The whole mark of hemodynamic management is by achieving predetermined hemodynamic targets by obtaining hemodynamic measurements and adjusting therapeutic management according to these specific targets to intervene and adjust therapeutic management for the patient. Adequacy of tissue and vital organs perfusion since organ blood perfusion cannot be directly measured.

The Pathophysiology of Inadequate Cardio-circulatory Performance

To understand fully the hemodynamic changes that might occur during cardiac surgery it is worth reviewing the pathophysiology principles and the causes of inadequate cardio-circulatory performance. They are classified into four general categorizes: hypovolemic, cardiogenic, obstructive, or distributive.

A simplified approach to why patients might have inadequate cardio-circulatory performance includes:

- Decreased preload (decreased intravascular volume), which can be due to hypovolemia, mechanical obstruction, vasoplegia, or a combination of these causes.
- Myocardial dysfunction, which can be due to cardiomyopathy, myocardial infarction, vasoplegia, or a combination of these causes.
- Decreased afterload, which can be due to myocardial dysfunction, vasoplegia, or a combination of both.

Treatment of Hemodynamic Failure

There are three specific therapies that can be used in treatment of hemodynamic failure:

- Fluid therapy to avoid fluid deficit and optimize circulating volume. Monitoring is carried out by static indices of preload CVP and pulmonary capillary wedge pressure or by dynamic indices of preload stroke volume variation (SVV) and pulse pressure variation (PPV). The dynamic indices have a higher accuracy when compared to the static indices. A close correlation exists between perioperative complications and the amount of fluid administrated to the patients. In one side hypovolemia, is associated with low cardiac output and tissue hypoperfusion that might lead to organ failure and adverse outcome. On the other hand, fluid overload and hypervolemia are associated with high risk of tissue and lung edema, hemodilution, coagulopathy, congestive heart failure, and organ dysfunction as well as adverse outcome. So, the goal is to achieve normovolemia in the perioperative period.
- Inotropic support when the cardiac output is inadequate after optimizing the volume. Monitoring is done by measuring flows, for example, cardiac output.
- Vasopressor or vasodilator therapy to regulate arterial pressure and vascular tone optimization. Monitoring is carried out by measuring tissue oxygenation, for example, mixed venous saturation.

Assumptions during Hemodynamic Static Monitoring

Pressure reflects volume so using pressure measurement as a surrogate to volume assessment, for example:

- Right atrial pressure or CVP during diastole is equal to right ventricular end diastolic pressure. This reflects right ventricular end-diastolic volume, which is an estimate of right ventricular preload.
- Pulmonary capillary wedge pressure or left atrial pressure during diastole is equal to left ventricular end-diastolic pressure reflecting left ventricular in diastolic volume, which is an estimate of left ventricular preload.

Limitations to Monitoring Preload by Static Indices

Conditions that may alter the reliability of pressure as an estimate of adequate circulating volumes include:

- Abnormal pressure and volume relationship, for example, poor compliance of the right ventricle or left ventricle.
- Increased intrathoracic pressure, such as mechanically ventilated patients on positive end expiratory pressure PEEP as well as increase in intra-abdominal pressure and how those may affect flow through the thoracic cavity.
- Valvular heart disease, such as mitral stenosis.

Limitations to Monitoring Preload by Dynamic Indices

These are some limitations to monitoring preload by SVV/PPV:

- Only for patients on mechanical ventilation (TV more than or equal 8 ml/kg).
- Arrhythmia can dramatically affect SVV/PPV.
- High-level of PEEP can increase SVV/PPV.
- Changes in lung or chest compliance such as after sternotomy and open chest will affect readings.
- Right ventricular dysfunction may affect readings.

Fundamentals of Invasive Pressure Monitoring

Components of the Pressure Monitoring System

Overview

This pressure monitoring system is achieved by placing a hollow catheter inside the lumen of a vessel of the cardiovascular system and connecting it to an electronic

transducer through a fluid-filled, low-compliant tubing system that allows for accurate pressure measurements and displaying it on a screen.

Electronic Transducer
Changes in the intracardiac chamber or intravascular lumen pressure are transmitted via the column of fluid in the low-compliant pressure tubing to the transducer diaphragm where the mechanical vibration is converted to an electrical signal by the transducer

Fast Flush Device
It allows to manually flush the tubing system. It is located on the transducer

Pressure Tubing
Sterile Fluid-Filled Low Compliant High-Pressure Tubing Connects the Catheter to the Transducer.

I.V. Fluid
A bag of normal saline placed in an inflated pressure bag will allow continuous constant flow of flush fluids. This will prevent blood from backing in the tubes.

Flush Tubing
Intravenous infusion sets connect the IV fluid bag to the transducer.

Transducer Cable
It connects electronic transducer to the pressure monitor.

Pressure Monitor
It allows the conversion of the electrical signal created by the transducer and then amplify it into a pressure waveform with numeric value displayed in real time on the screen.

Three-Way Stopcock or Closed Blood Sampling System (Vamp)
Allows for blood sampling and zeroing.

Leveling and Pressure Transducer Alignment
Accomplished by positioning the transducer at the level of the patient's right atrium or the phlebostatic axis. The phlebostatic axis is located at the junction of the fourth intercostal space with the midaxillary line (Fig. 9.1). This will eliminate the effect of the hydrostatic pressure produced by the fluid in the pressure tubing. If the transducer is positioned below the phlebostatic axis, the weight of the fluid in the tubing will exert added pressure on the transducer's diaphragm, causing abnormally elevated pressure reading. On the other hand, if the transducer is positioned above the phlebostatic axis, the fluid in the tubing will exert lower pressure on the transducer's diaphragm, causing abnormally lower pressure reading. For every 10 cm, the transducer above the phlebostatic axis is equal to a 7.5 mmHg decrease in pressure reading and vice versa.

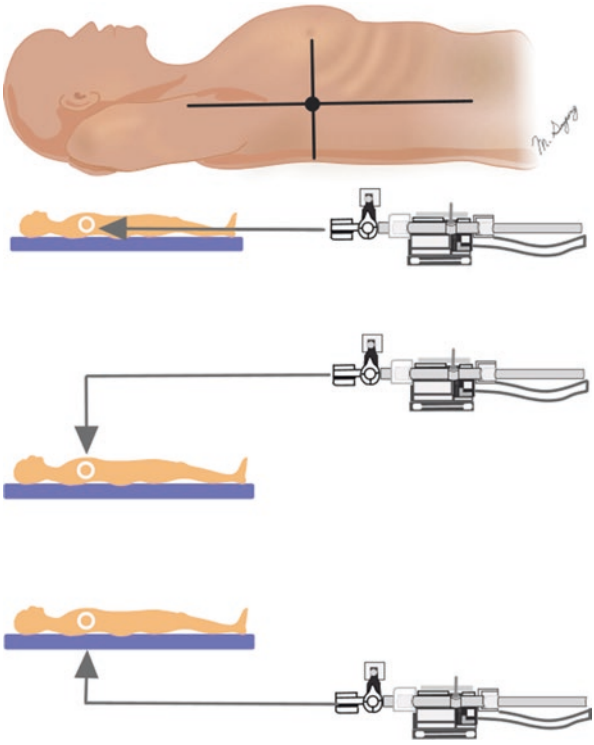

Fig. 9.1 Illustration of monitoring system transducer levelling

Zeroing

After levelling the pressure monitoring system, the system is opened to the air to establish the atmospheric pressure as zero, thus allowing the monitoring system to begin measuring at 0 mmHg.

Resonance and Damping

Damping is an effect that reduces the size of the pressure waveform. Increased damping falsely underestimates the systolic pressure and falsely overestimates the diastolic pressure. Resonance occurs when the frequency of the force contributing to waveform is close to the transducer natural frequency. Thus, the monitoring system will oscillate with larger waveform amplitude. Underdamping falsely overestimates the systolic pressure and falsely underestimates the diastolic pressure. However, in both cases, the mean arterial pressure is not altered.

Square Wave Test or Fast-Flush Test

This simple test is performed by fast flushing for 1–2 s and observing the shape of the square waveform formed on the screen as noted in Fig. 9.2.

- Optimally damped square wave will have a perfectly vertical upstroke with a straight downstroke with one to two oscillations after the downstroke.

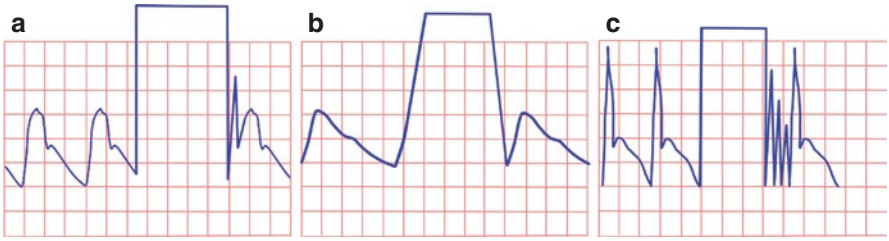

Fig. 9.2 (**a**) Optimally damped square wave; (**b**) Overdamped square wave; (**c**) Underdamped or hyperresonance square wave

- Overdamped square wave will have slurred upstroke and downstroke with no or little oscillation after the downstroke. Corrections can be done by making sure to eliminate air bubbles and blood clots from the pressure tubing.
- Underdamped or hyperresonance square wave will have multiple oscillations after the downstroke. Corrections can be done by incorporating a commercially available damping device (Resonance Overshoot Eliminator or R.O.S.E) that can be used in pressure monitoring tubing systems and also by temporarily injecting a tiny air bubble in the tubing. However this might be a dangerous practice if the anesthesia provider forgets and flush the air into the patient. Restricting the pressure tubing length to 4 ft can correct the problem as well.

Assessing Afterload

Afterload refers to the pressure that the heart must work against to pump blood. Afterload depends on several variables: blood volume, vascular resistance, ventricular geometry, and blood viscosity "hemoglobin concentration." Arterial blood pressure is the value most often used as a surrogate measurement for afterload.

Invasive Blood Pressure Monitoring

Noninvasive blood pressure (NIBP) monitoring can provide accurate readings in most situations. In cardiac surgery, the potential for acute and drastic hemodynamic changes requires arterial blood pressure monitoring in almost every case because of the need of "beat-to-beat" blood pressure readings. Arterial blood pressure monitoring may also circumvent some of the problems associated with the use of NIBP cuffs such as inaccurate readings due to cuff size mismatch or the surgical staff compressing the cuff against the edge of the table. Also, during CPB, invasive arterial blood pressure is mandatory because of loss of pulsatile flow while on the bypass machine.

Arterial Line Cannulation

An arterial line not only provides "beat-to-beat" blood pressure monitoring, it also allows for frequent arterial lab draws and assessment of volume status. Selection of a suitable site and good insertion technique are necessary to obtain a high rate of success with lower incidence of complications. Like with most medical procedures, there are some potential complications, including ischemia, pseudoaneurysm, arteriovenous fistula, thrombosis, peripheral neuropathy, infection, and bleeding.

Radial Artery Cannulation

The most common place to insert an arterial blood pressure monitor is in the radial artery, usually inserted into patient's nondominant hand unless contraindications dictate a specific site for placing such as inadequate collateral blood flow to the hand or infection at the site of cannulation. Also, certain cardiac surgeries require a specific extremity, for example:

1. Surgery on the aortic arch or thoracic aorta may require blood pressure monitoring on both upper extremities. Arch surgery may render invasive blood pressure monitoring from the left radial artery impossible, and a right radial artery will be required in such cases. In aortic arch disease, discordant blood pressure readings on the right and left upper extremity may be a sign of aneurysm rupture or worsening dissection. Even in elective cases on the aortic arch, measurement of bilateral upper extremity blood pressure may alert the surgeon and anesthesia provider to changes in flow to the great vessels that may compromise cerebral perfusion.
2. The radial artery is planned to be harvested and used as a conduit for coronary artery bypass grafting surgery.

Technique of Cannulation

The procedure is accomplished using a variety of methods:

1. Blind thread-off technique, where a catheter and needle system are placed in the artery and then the catheter is slid off (Fig. 9.3).
2. Doppler auditory assistance, where audible blood flow provides either using doppler probe to locate the artery and then use thread-off technique as discussed above or needle–catheter–doppler system such as SmartNeedle® Vascular Access System where the needle is attached to an integrated doppler monitor that allows continuous auditory feedback.
3. Ultrasound guidance, where the ultrasound imaging is used to guide catheter insertion, especially after a failed attempt https://youtu.be/YOxyssqqYNE.
4. In Seldinger technique, two varieties of techniques can be used:
   (a) Through-and-through method, in which the needle is deliberately allowed to puncture the anterior and posterior wall of the artery and go through and through. Then the needle is removed from the catheter and the catheter is

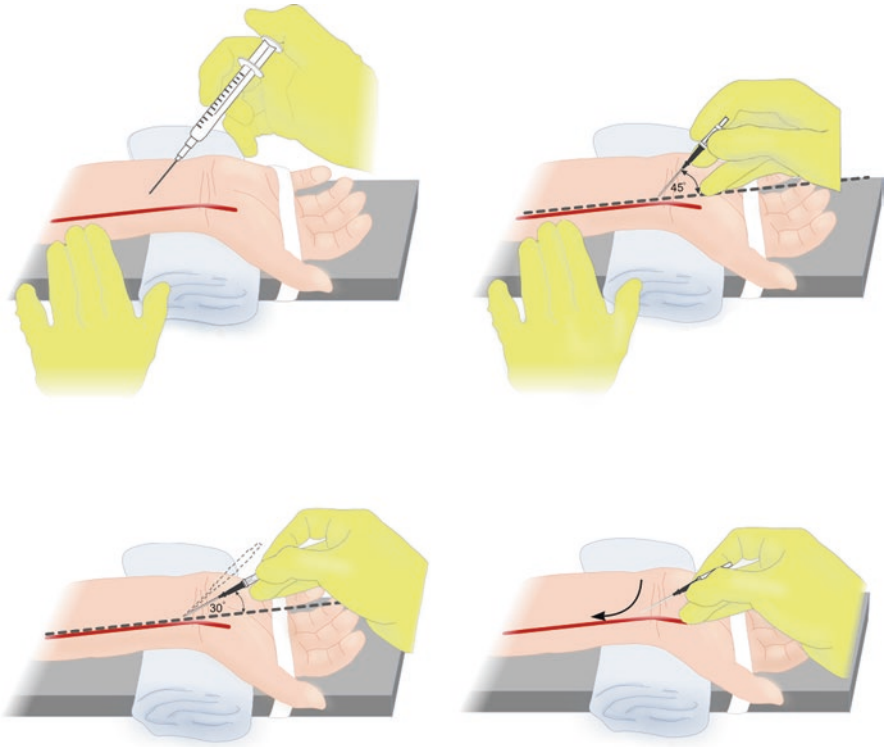

Fig. 9.3 Illustration of a line-blind thread-off technique

slowly pulled back until its tip lies completely in the lumen of the artery with obvious spirting of pulsatile arterial blood coming out. At this point, a guidewire is inserted through the catheter and the catheter is advanced back over the wire using the Seldinger technique.

(b) All-in-one access catheter-over-wire technique in which an arterial catheter system has a catheter with an integral spring wire guide. It is an integrated needle–catheter–wire system.

5. Catheter over the needle percutaneous technique or with the aid of ultrasound to cannulate the radial, most common site, brachial, axillary, or femoral artery (Fig. 9.4).

Setup

The necessary materials should be readily available, which includes chlorhexidine antiseptic skin prep, sterile towels, a syringe with lidocaine 1% local anesthesia with a 25 G needle attached, either catheter over-the- needle catheter "Angiocath IV catheter" or with integrated needle–catheter–wire system, separate guide wire, gauze, sterile dressing, sterile gloves, counterpressure bag, IV fluid solution bag, and an arm board. Commercial prepackaged tray is also available in some institutions (Fig. 9.5).

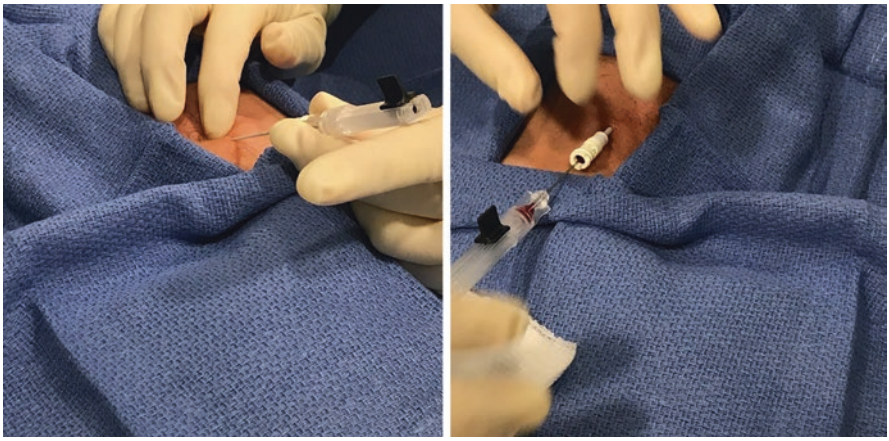

Fig. 9.4 Illustration of all-in-one access catheter-over-wire technique

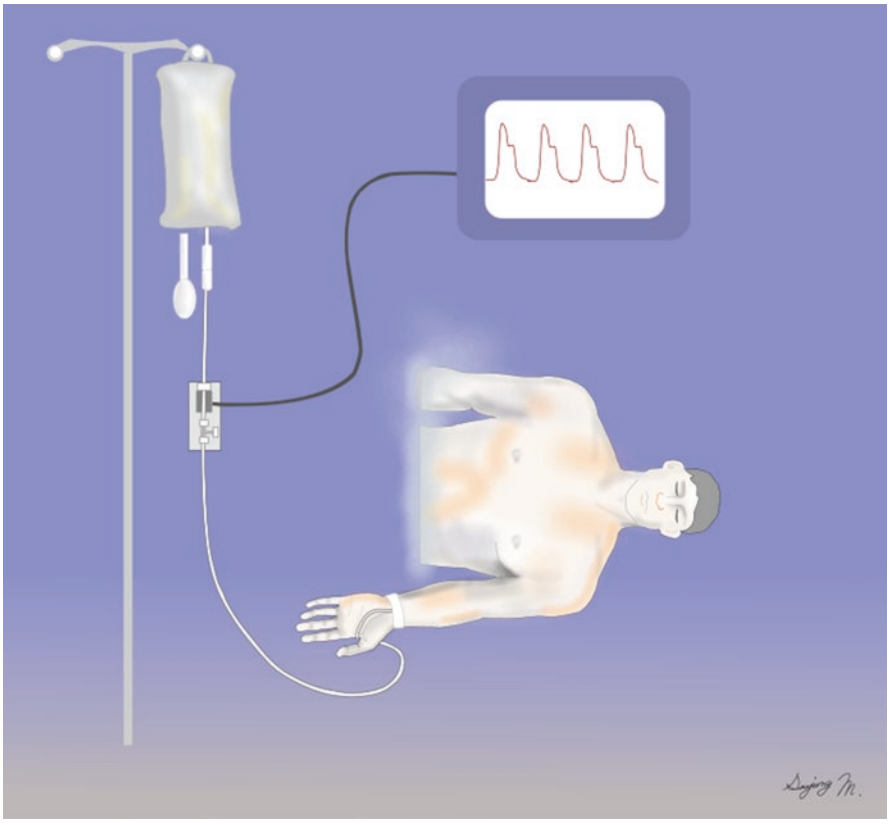

Fig. 9.5 Illustration of complete a line transducer system

The arterial line transducer system is attached to the monitoring cable and the saline-filled connecting tubing is flushed and cleared from air before the insertion of the catheter.

Patient Preparation and Procedure Technique

The fully extended patient's arm is placed on a flat surface with the elbow supported. The wrist is immobilized and dorsiflexed using a soft roll of gauze. This makes the radial artery more superficial to aid in cannulation. The course of the radial artery is mapped via palpation, doppler, or ultrasound. The area is prepared with an antiseptic solution and covered by sterile drape and then the skin is infiltrated with local anesthetic. If catheter-over-wire is used, the guide wire must be checked to make sure that it advances freely through the needle. At 45-degree angle, the needle is advanced toward the radial artery pulsation and then it is punctured. After obtaining flash of blood, the needle is slightly advanced further followed by dropping the angle to allow the catheter to be parallel to the vessel and permit smooth advancement. After the catheter is slid in using one of the techniques discussed above, the catheter is fastened to the pressure IV tubing and arterial waveform is check for. https://www.youtube.com/watch?v=3z9vHu4r6HE

If the blood flow is very sluggish, it is better to leave this catheter in situ and attempt another insertion proximally as removal will lead to hematoma or arterial spasm.

Alternate Sites

There are other sites of arterial cannulation available as well, though they are less commonly used, including femoral, brachial, axillary, and dorsalis pedis arteries.

Femoral Artery

The patient is positioned supine with the thigh slightly abducted and externally rotated for easy palpation of the artery against the femoral head. The patient is prepped, the skin is anesthetized, and then the artery is located via palpation, doppler, or ultrasound. Subsequently the needle is inserted as described above using the radial artery Seldinger technique (Fig. 9.6).

Brachial Artery

If other arteries are not available, the brachial artery can be used instead. The brachial artery is located just medial to the biceps muscle tendon in the elbow crease.

Axillary Artery

The axillary artery can be cannulated if other arteries are not available. The axillary artery is accessed very high in the axilla. A longer catheter is required.

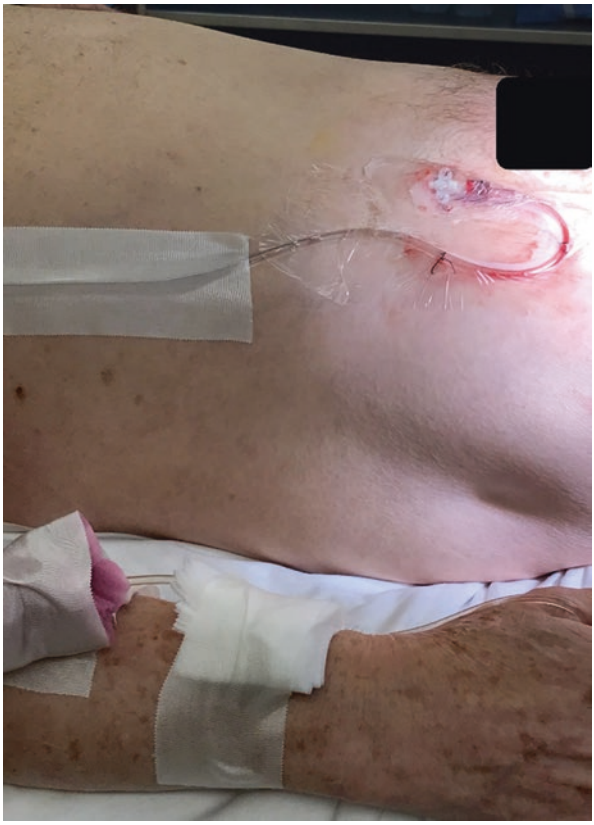

Fig. 9.6 Image of right femoral artery

Arterial Line Waveform Analysis

- The arterial waveform is formed when the blood is ejected during systole from the left ventricle through the open aortic valve into the aorta. This marks the systolic upstroke limb (anacrotic limb). After the systolic peak pressure is generated during systole, systolic wave declines as the ventricular contraction is about to end.
- The dicrotic notch reflects closure of the aortic valve and the end of systole.
- The smooth progressive run-off of blood into the peripheral circulation after the aortic valve closes marks the diastolic downstroke limb (dicrotic limb).
- The arterial pressure waveform has different shapes and values depending on which artery is monitored, for example systolic pressure measurement can increase by as much as 15 mmHg the farther the monitoring site is from the aorta.
- The morphology of the arterial waveform can provide useful information (Fig. 9.7).
- The difference between the systolic pressure and the diastolic pressure is known as pulse pressure.

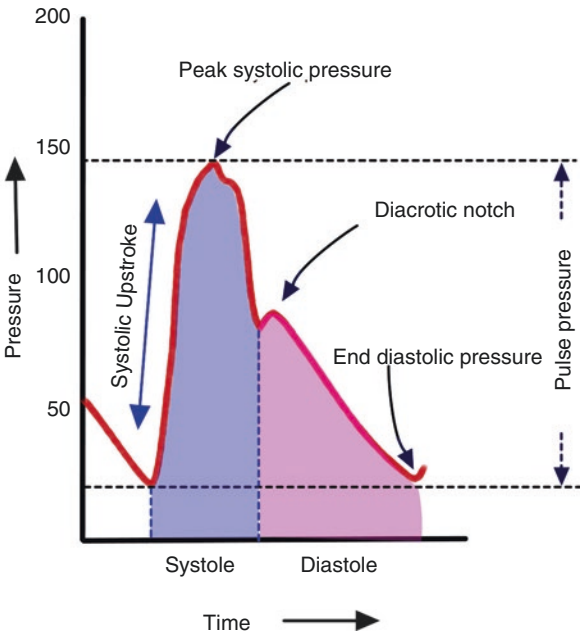

Fig. 9.7 Illustration of components of the arterial pressure waveform

- Mean arterial pressure equals systole + (2 × diastole) / 3.
- A widened pulse pressure can be indicative of distributive shock or aortic regurgitation.
- Narrow pulse pressure may be a sign of cardiogenic shock or obstructive shock (cardiac tamponade, tension pneumothorax).
- The variation of pulse pressure during positive pressure ventilation may be indicative of the intravascular volume status of the patient and ventricular filling.
- Common abnormal arterial waveforms are shown in Fig. 9.8.

Systemic Vascular Resistance Calculations

Systemic vascular resistance (SVR) can be calculated to estimate afterload by subtracting central venous pressure

from the mean arterial pressure, divided by cardiac output and then multiplied by 80 as per the following formula:

$$\mathrm{SVR} = \frac{(\mathrm{MAP} - \mathrm{CVP}) * 80}{CO} \left[\mathrm{dynes \cdot sec \cdot cm} - 5\right]$$

where MAP = mean arterial pressure, CVP = central venous pressure, CO = cardiac output.

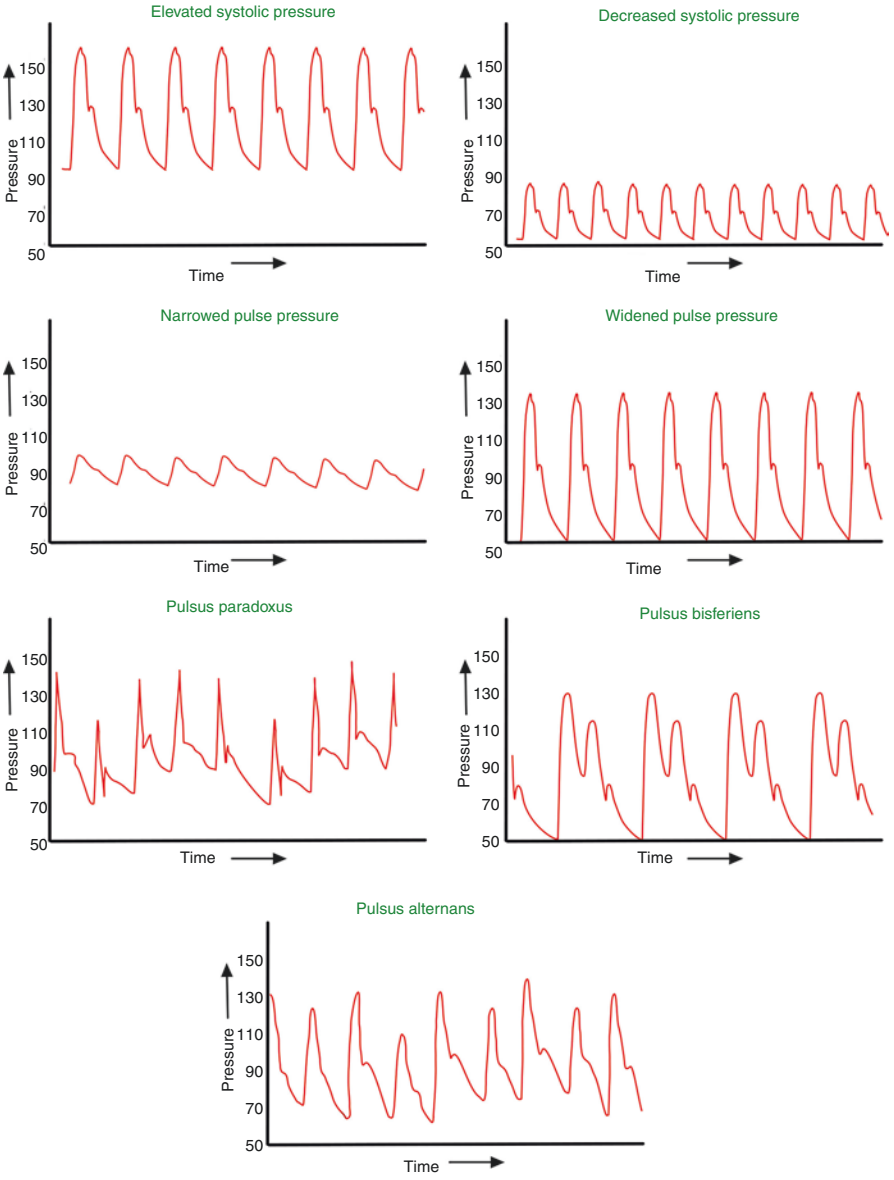

Fig. 9.8 Illustration of abnormal arterial pressure waveforms

Assessing Preload

Assessing Right Ventricular Function – "Filling Pressures"

Central Venous Catheterization

Central venous catheterization is an essential technique in the management of cardiac surgery patients. The right internal jugular vein is the most common site used

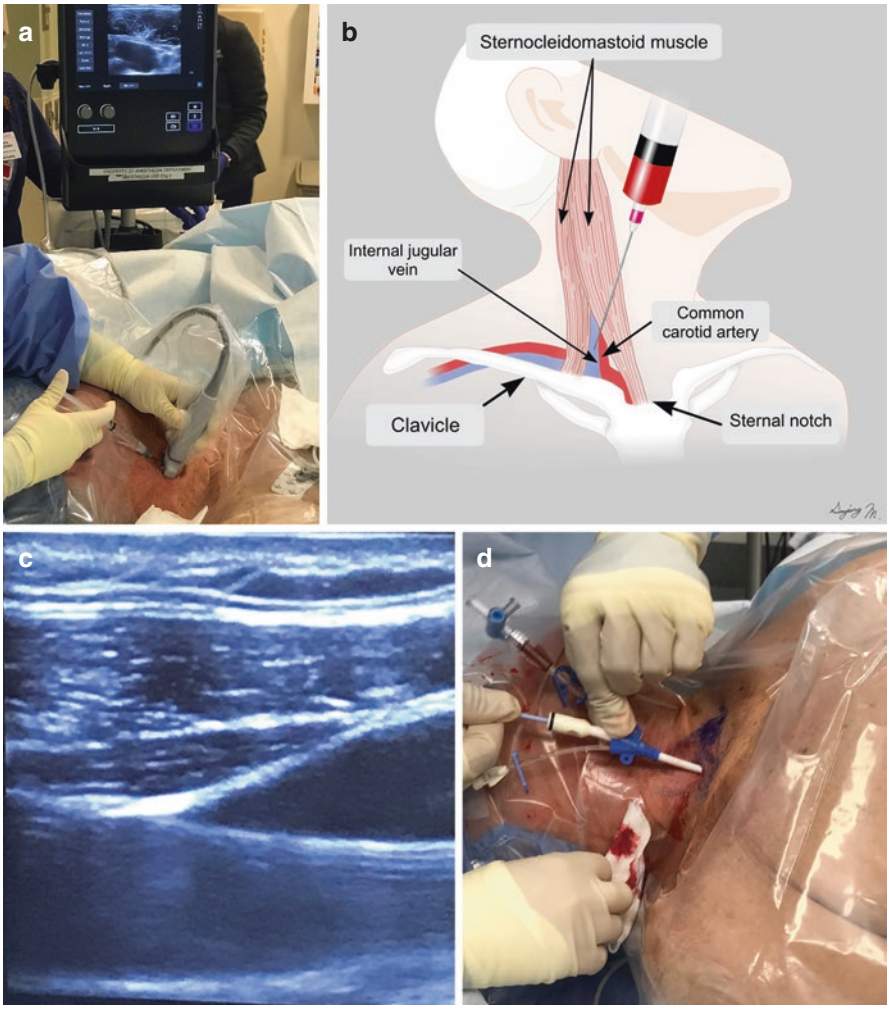

Fig. 9.9 Right internal jugular vein cannulation (**a**) ultrasound guided localization of the vein and local anesthesia skin infiltration (**b**)– anatomy of the vein in the neck and the direction of the needle (**c**) visualization of the wire in the vein (arrow) (**d**) threading the catheter over the dilator

because it is fairly straight line and the left subclavian is the second; however, other alternate sites include subclavian, external jugular, and femoral veins.

Technique of Insertion

The approach for vein location most commonly used and is recommended is by ultrasound guide (Fig. 9.9). However, the landmark approach for vein location is explained as well in case where ultrasound is not feasible. Anatomically the vein is located in the triangle between the medial and lateral heads of the sternocleidomastoid muscle and lateral to the carotid artery. After the vein is located the steps are the same for both approaches.

Steps in an awake patient

1. The necessary materials, such as central line kit, sterile gloves, full body drape, and mask, should be readily available.
2. The patient is placed in Trendelenburg position with the neck turned to the left side.
3. If necessary for patient comfort, sedation can be given and supplemental oxygen should be prescribed.
4. Local anesthesia is infiltrated in the skin after locating the vein at the site of the needle insertion.
5. A finder 22 gauge is inserted under suction at the apex of the triangle aiming at the ipsilateral nipple.
6. A large needle 18 G or a catheter is inserted using the same angle and depth.
7. A wire is threaded through the needle.
8. The catheter is passed over the wire.
9. The wire is removed.
10. The scalpel tip is used to make a small nick in the skin against the wire.
11. Threading the catheter over the wire.
12. Aspirate all ports and flush.
13. Suture in place.
14. Apply sterile dressing.

Central Venous Pressure (CVP)

CVP is measured at the junction of the superior vena cava and right atrium. CVP measurements yield a classic waveform that can change drastically in different disease states (Fig. 9.10). All hemodynamic waveforms should be measured relative to the ECG. Since electrical activity runs faster than the physical transduction of a pressure signal, there will be a little delay between ECG and CVP. CVP is measured at end expiration. Normal CVP values are 0–5 mmHg in spontaneously breathing patients, and 5–10 mmHg in patients receiving positive pressure ventilation (PPV). The indication of measuring central venous pressure or right atrial pressure is to estimate right ventricular end-diastolic pressure that reflects right ventricular

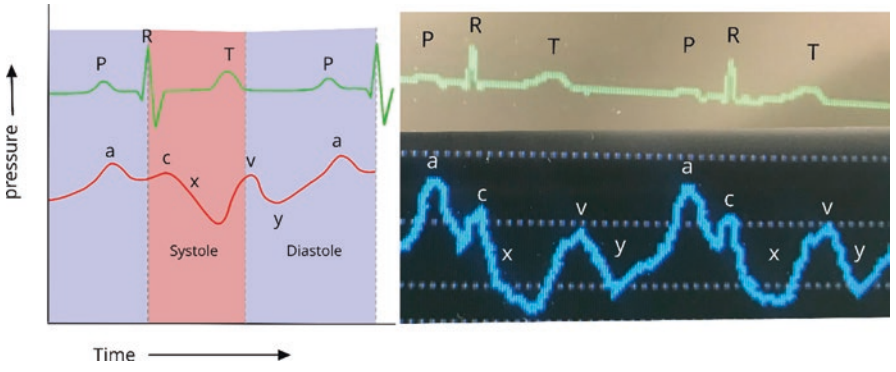

Fig. 9.10 Illustration showing components of the central venous pressure waveform

end-diastolic volume and provides an accurate assessment of preload. CVP is calculated by adding the peak of the A wave to the bottom of the X descend, and the mean of the two numbers is equal to the central venous pressure.

CVP Waveform Analysis

The CVP waveform has three peaks and two descends (Fig. 9.10).

A wave: An A wave reflects right atrial contraction and correlates with the P wave activity on the ECG. There will be no A wave with atrial fibrillation.

C wave: A C wave which correlates with the isovolumic ventricular contraction, forcing the cusp of the tricuspid valve to bulge into the right atrium correlates with the end of the QRS complex on the ECG.

X descent: As the right ventricular contracts with downward movement of the tricuspid valve, a drop-in pressure in the right atrium occurs as the tricuspid valve is pulled away from the atrium. Also, this is associated with atrial diastolic relaxation, which adds to drop-in pressure in the right atrium. This occurs before the T wave on the ECG.

V wave: The V wave reflects the passive filling of the right atrium during late ventricular systole, where blood bump into the tricuspid valve causes a back-pressure wave. It occurs after the T wave on the ECG.

Y descent: This is a drop-in pressure in the right atrium when the tricuspid valve opens and the right atrium empties in early diastole. It occurs before the P wave of the ECG.

Although CVP can be a useful indicator of intravascular volume and cardiac contractility, it alone may not distinguish between changes in volume or changes in contractility. CVP can be influenced by many other factors, for example intrathoracic pressure and venous tone.

CVP has been considered a marker for volume status, although when taken in isolation, its relationship to volume status is dubious at best due to the long list of variables affecting CVP.

CVP Respiratory Variation

CVP fluctuates with respirations and that has direct relevance as to whether a patient is breathing spontaneously or is on positive pressure ventilation with a mechanical ventilator and those waveforms will be opposite each other. The central venous pressure is measured at end expiration when the pressure differential between atmospheric and pulmonary has equalized.

Abnormal CVP Waveform

1. There will be absence of a wave with atrial fibrillation.
2. Prominent A-wave: Increased resistance to blood flow from atrium to ventricle or anything that causes backup in pressure distal to the tricuspid valve, and thus a wave dominant may occur in decreased myocardial compliance from RV ischemia, tricuspid stenosis, or pulmonary hypertension.
3. Cannon A-wave: Atria contracting simultaneously with the ventricles with AV dissociation, as may occur in heart block or junctional rhythm.
4. Regurgitant CV waves: C wave fuses with the V wave, causing large positive wave, as it occurs in tricuspid regurgitation.

CVP is measured by placing a central venous catheterization. Most patients who undergo cardiac surgery receive central venous catheterization because of frequent need for volume resuscitation and vasopressor use in addition to cardiac monitoring via the catheter itself. There are no absolute contraindications to the placement of a central venous catheter, although many situations arise when the risks of central venous catheterization outweigh the benefits – these include vessel thrombosis, SVC syndrome, severe coagulopathy, or inability to tolerate positioning for central line placement due to, for example, hypoxemia and high oxygen requirements.

Assessing Left Ventricular Function – "Filling Pressures"

Pulmonary Artery Catheter

Despite the ongoing lack of consensus surrounding the routine use of the pulmonary artery catheter (PAC), it can still provide diagnostic and monitoring valuable information in high-risk cardiac surgery patients, for example, patients with impaired right or left ventricular systolic functions. The PAC is a hollow long multi-orifice tube that is fluid-filled and connected to a transducer. Usually it can be inserted via a large bore central venous introducer (Fig. 9.11).

- The syringe plunger is limited to a maximum volume of 1.5 ml and can be locked in the closed position, preventing accidental wedging.

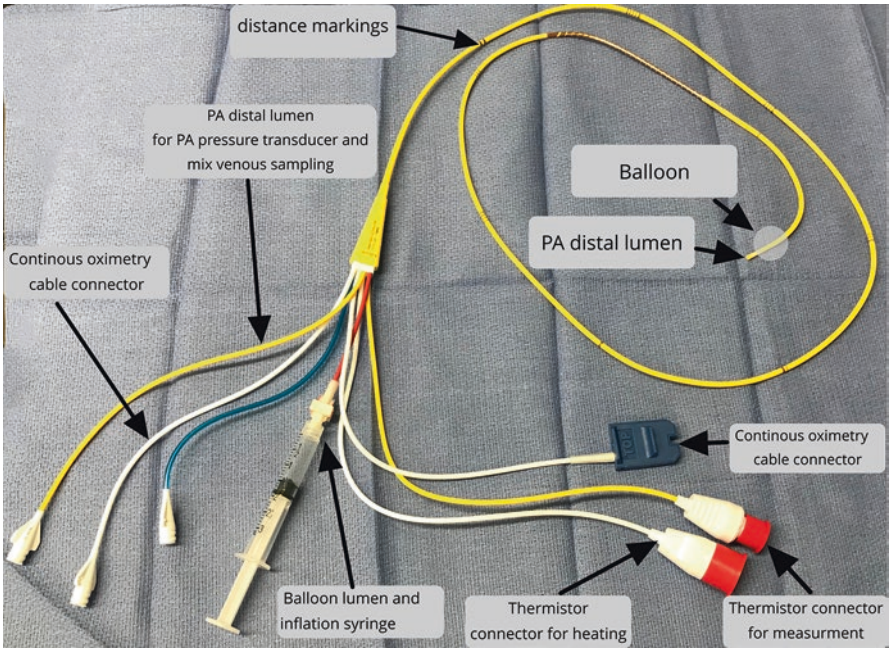

Fig. 9.11 Image of pulmonary artery catheter

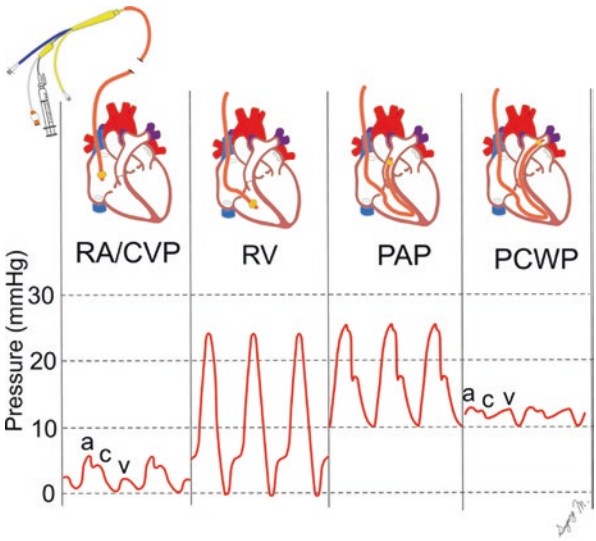

Fig. 9.12 Illustration of waveforms during insertion of a pulmonary artery catheter

- 10 cm interval markings on the catheter.
- Distance to RA: 10–15 cm from the subclavian vein, 15–20 cm from the jugular vein, 30–40 cm from the femoral vein. RV is another 10 cm, and the pulmonary artery is another 10 cm after that, and the wedge position a further 10 cm.
- Since the PAC traverses the right side of the heart, placement often causes extrasystoles and may cause a transient and sometimes permanent right bundle branch block. This can lead to complete heart block if the patient has preexisting left bundle branch block.
- Traversing the different chambers will produce different waveforms (Fig. 9.12).

There are various types of pulmonary artery catheters. Some PACs have a pacing port, that are usually used in minimally invasive AVR surgery. Others have continuous cardiac output and mixed venous oximetry.

The information measured directly from the pulmonary artery catheter includes:

- Central venous pressure
- Pulmonary artery pressure
- Wedge pressure
- Cardiac output
- Mix venous oxygen saturation

When PAC is placed in the appropriate position, the proximal orifice will be located 30 cm from the tip of the PAC and is aligned with the right atrium–SVC junction to provide a CVP measurement. The distal port of the PAC will provide a measurement of the pulmonary artery pressure. The PAC tip is equipped with a balloon. If the balloon is wedged into a small branch of the pulmonary artery, it will create a static column of fluid that directs back to the left atrium and allows to

indirectly measure the left atrial pressure (PAOP), also known as "wedge pressure." This is considered to represent left atrial and left ventricular end-diastolic filling pressures and is used to assess left ventricular filling. The PAC can also be used to measure core temperature.

In addition, this information can be calculated from direct pressure measurements:

- Stroke volume/stroke volume index based on cardiac output and heart rate
- Cardiac index based on a measure cardiac output relative to body mass
- Systemic vascular resistance based on flow and pressure differentials
- Pulmonary vascular resistance
- Oxygen delivery

The Normal Pulmonary Artery Pressure Waveform

- It is pulsatile and is measured with the balloon down and the catheter tip is in the distal pulmonary artery. The characteristic waveform has a systolic upstroke and peak systolic pressure. The dicrotic notch is reflective of the closure of the pulmonic valve. The normal range of systolic pressures between 15 and 30 mmHg, though there's beat-to-beat variability as well as respiratory variability. Diastolic pressures are between 10 and 15 mmHg. The mean pulmonary artery pressure is the pressure that is followed, which ranges between 10 to 20 mmHg in the normal range.
- Mean pulmonary artery pressure is equal to one-third systolic pressure + two-third diastolic pressure.
- It is measured relative to the ECG, where the QRS should line up with the beginning of the systolic stroke of the pulmonary artery pressure waveform.

The Pulmonary Capillary Waveform

It is very similar to the central venous pressure waveform but reflective of little more delay in time and a little more impact of respiratory variability. The balloon is inflated on the tip of the catheter, creating a static column of fluid back to the left atrium. The pulmonary capillary wedge pressure is measured at end expiration. Change of the waveform morphology is reflective of the underlying physiology. The V wave reflects an increase in pressure during atrial filling, and it could be due to a number of things. "V wave more than 50% greater in height than the A wave" probably increased venous return to the left atrium:

- Retrograde flow from mitral regurgitation
- Decreased atrial compliance and a stiff wall
- Increased flow into the right atrium
- Interpretation of the pulmonary capillary wedge pressure

Estimating Cardiac Output

Venous Oxygen Saturation

Venous oxygen saturation (S_VO_2) can be measured at any point in the venous system but is traditionally measured in the proximal pulmonary artery – this is referred to as mixed venous oxygen saturation (M_VO_2). When compared to arterial oxygen saturation (SaO_2), M_VO_2 can give an estimated amount of oxygen extraction: oxygen extraction fraction = (SaO_2 – M_VO_2)/SaO_2. The normal value for M_VO_2 is 65%–75.

Fick Method of Assessing Cardiac Output

In addition to the estimation of oxygen extraction, M_VO_2 can be used to calculate cardiac output in patients where other methods of cardiac output measurement are unavailable (e.g., patients with left ventricular assist device in whom a PAC is contraindicated or absent). The Fick principle states that "the total uptake of (or release of) a substance by the peripheral tissues is equal to the product of the blood flow to the peripheral tissues and the arterial–venous concentration difference of the substance." Cardiac output can be calculated from the difference in oxygen content in blood before it enters and after it leaves the lungs according to the following equation (Fick equation):

$$CO = \frac{VO_2}{\left(CaO_2 - CvO_2\right) \times 10},$$

where VO_2 = O_2 consumption, CaO_2 = arterial oxygen content, CvO_2 = venous oxygen content, VO_2 = 125 × body surface area (BSA, estimated), CaO_2 = 1.36 × Hgb × SaO_2, CvO_2 = 1.36 × Hgb × MvO_2, SaO_2 = arterial oxygen saturation, MvO_2 = mixed venous oxygen saturation (from proximal pulmonary artery), Hgb = hemoglobin [mg/dL].

Thermodilution Method of Assessing Cardiac Output

In addition to pressure measurement and SvO_2 sampling, the PAC can be used to measure cardiac output via the thermodilution (TD) method. The TD method is a variation of the indicator dilution technique. It utilizes the change in blood temperature caused either by injection of cold saline (manual technique) or the warming up of the blood by a coil on the surface of the PAC (continuous technique).

The manual technique is performed as follows. Ten milliliters of cold saline is injected into a proximal port of the PAC. This saline injection will enter the circulation around the RA–SVC junction, cooling down the blood. RV contraction will propagate the cooler blood past the distal portion of the PAC. A temperature sensor

on the tip of the PAC measures the change in temperature, generating a thermodilution (temperature over time) curve. The area under the curve is displayed on the monitor and is inversely proportional to the CO. Most monitors will calculate the cardiac output automatically.

Multiple sources of error in TD CO measurement exist and the anesthesia provider should be aware of them. Right to left shunts will cause the injectate to bypass the thermistor and result in an overestimation of CO. Left to right shunts, conversely, will lead to an underestimation of CO due to excessive dilution of the injectate. Both tricuspid valve and pulmonic valve regurgitation lead to unreliable measurements due to prolonged decay of the temperature gradient over time, leading to underestimation of the CO. This effect will be more pronounced as the degree of regurgitation increases.

Complications of the use of the PAC include PA rupture, arrhythmia, pulmonary embolism, line sepsis, or misinterpretation of CO values, leading to inappropriate therapies. Therefore, less invasive and more dynamic variables monitoring those estimates CO have gained favor.

Dynamic Assessment of Intravascular Volume

Various new minimally invasive techniques and devices are available for monitoring dynamic variables and the anesthesia provider needs to know about the advantages, the technical principles, and the limitations of these devices in order to have an adequate knowledge for clinical decision-making.

There are four methods to assess dynamic variables using minimally invasive technology:

- The pulse wave analysis
- Doppler technology
- Applied Fick's principle
- Thoracic bioimpedance/bioreactance

The Pulse Wave Analysis:

These devices calculate cardiac output and stroke volume from continuous pressure waveform analysis (via arterial line or finger probe). Each device has its patented unique algorithm based on the same principle which calculates the stroke volume by integrating the area under the curve of only the systolic phase of the arterial waveform.

Limitations

- Optimal arterial waveform contour is required for accurate measurement. So, if there is damping or hyperresonance, it will be inaccurate.

- Severe atherosclerosis and the location of the arterial insertion may affect the readings. For example, in patients with subclavian artery stenosis use the opposite side.
- Inaccurate reading occurs in severe arrhythmias or with the use of intra-aortic balloon pump.
- Open chest condition that changes the intrathoracic hemodynamics significantly.

Pulse wave analysis monitoring systems can be divided into two main groups:

1. The autocalibrated devices the uncalibrated devices that rely on arithmetical modelling, which include:
   - FloTrac/Vigileo
   - LiDCO rapid
   - PulsioFlex
   - Nexfin
   - Radical 7 esCCO/Mostcare

   These devices have the following characteristics:
   Easy to use, less invasive, and self-calibrated using patient demographics
2. The calibrated devices need external calibration, which Include:
   - EV 1000/Volume View (Edwards Lifesciences, USA) uses transpulmonary thermodilution method
   - LiDCO plus (LiDCOplus, LiDCO, UK) uses lithium dilution method
   - PiCCO plus (Pulsion Medical Systems, Munich, Germany) uses transpulmonary thermodilution method suitable in pediatric patients.

The use of uncalibrated continuous pulse contour cardiac output device should not be recommenced after weaning from bypass.

Doppler Technology

Dynamic index of cardiac output and fluid responsiveness is by Doppler technology devices, which include:

- Esophageal Doppler
- TEE/TTE echocardiography
- Ultrasonic Cardiac Output Monitor (USCOM)

Esophageal Doppler

The device measures the blood flow in the descending aorta using a flexible ultrasonic transducer with 4–5 MHz of frequency and calculate the cardiac output by multiplying the cross-sectional area of the aorta by the blood flow velocity.

Advantages:

- Simple to use
- Reliable, semi-invasive does not require access to circulation

Disadvantage:

- Limited to measure flow only in the descending aorta
- Need good probe location to acquire strong signal
- Operator dependent

The Partial Co2 Re-breathing System

The NICO system (Novametrix Medical Systems, Wallingford, USA) applies the Fick's principle to carbon dioxide (Co2) instead of oxygen to measure cardiac output in an intubated and mechanically ventilated patients, through a specific disposable re-breathing loop.

Advantages:

- Easy to use
- No need for arterial line
- Provides cardiac output measurements every 3 min.

Disadvantages:

- Affected by changes in the dead space, ventilation perfusion matching, and low minute ventilation
- Does not provide information on fluid responsiveness

Thoracic Bioimpedance/Bioreactants

Thoracic bioimpedance estimates cardiac output and other hemodynamic variables such as stroke volume and cardiac index by placing sensors on the chest wall and detects electrical changes induced by cyclic changes in blood flow in the thorax. The higher the fluid content, the lower the resistance.

Thoracic bioreactant is a modification of the thoracic bioimpedance that analyzes and tracks the phase of the electrical currents – "phase shifts" – when an oscillating current is traversing the chest. An example of thoracic bioreactant technology is noninvasive hemodynamic and cardiac output monitor (NICOM system Cheetah Medical, Portland, OR)

Advantages:

- The cardiac output is estimated using electrical skin electrodes or electrodes mounted on an endotracheal tube.

Disadvantages:

- Prone to electrical and motion artifact, arrhythmia, heart and lung pathologies.

Further Reading

American Society of Anesthesiologists Task Force on Pulmonary Artery Catheterization. Practice guidelines for pulmonary artery catheterization: an updated report by the American Society of Anesthesiologists Task Force on Pulmonary Artery Catheterization. Anesthesiology. 2003;99(4):988–1014.

DiNardo JA, Zvara DA. Anesthesia for cardiac surgery. 3rd ed. Malden: Blackwell; 2008.

Fick A. Uber die messung des Blutquantums in den Hertzvent rikeln. Sitzber Physik Med Ges Wurzburg. 1870;36

Gravlee GP, Martin DE. Hensley's practical approach to cardiothoracic anesthesia sixth edition. Philadelphia: Lippincott, Williams & Wilkins; 2018.

Gudmundsson P, Rydberg E, Winter R, Willenheimer R. Visually estimated left ventricular ejection fraction by echocardiography is closely correlated with formal quantitative methods. Int J Cardiol. 2005;101(2):209–12.

Marik PE, Baram M, Vahid B. Does central venous pressure predict fluid responsiveness?: A systematic review of the literature and the tale of seven mares. Chest. 2008;134(1):172–8.

Practice guidelines for central venous access: a report by the American Society of Anesthesiologists Task Force on Central Venous Access. Anesthesiology. 2012;116(3):539–73. https://doi.org/10.1097/ALN.0b013e31823c9569.

10 The Fundamentals of Transthoracic Echocardiography (TTE) and the Focused Assessment with Sonography in Trauma (FAST) Examination

Enrique J. Pantin and Denes Papp

Introduction

One of the many monitoring modalities used by cardiac anesthesiologists to help manage patients is echocardiography. Echocardiography, and ultrasound in general, has emerged as a key diagnostic tool in many clinical settings. Additionally, new cardiorespiratory symptomatology, change in effort level, tolerance, history of cardiac dysfunction, or valvular pathology without additional data, a newly detected heart murmur, hypotension of unknown origin, trauma assessment, and cardiac arrest management have emerged as primary indications for the use of ultrasound in the perioperative setting.

Although ultrasonography (US) is a powerful perioperative diagnostic tool, there are certain caveats associated with its proper use:

1. Two-dimensional ultrasound (the main US modality used) provides better imaging from structures that are perpendicular to the ultrasound beam. Structures that are parallel to the ultrasound beam are not imaged well, and often appear to be missing a segment ("echo dropout").
2. It is important to understand the basic physics of ultrasonography. All Doppler studies, namely continuous wave Doppler, pulse wave Doppler and color flow Doppler (CWD, PWD, and CFD respectively) provide more accurate velocity information when the Doppler cursor is parallel to blood flow. Oblique angles

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of interrogation lead to underestimation of velocities and have consequences on diagnostic interpretation.

3. When we perform an ultrasound study, we are not seeing the heart, or liver, or kidney, etc., but sound waves reflected from such structures that are reconstructed by a computer. Artifacts can easily be generated that can be misinterpreted as real structures, and lead to improper diagnostic decisions. Therefore, the basic types of US artifact need to be understood by the clinician.
4. It is sometimes difficult to obtain clear and well-defined images of certain anatomical targets. There are a variety of reasons for this which include poor windows for interrogation, air-filled structures aligned with the interrogation axis, reflections from anatomically adjacent structures, etc. If you do not see a structure, you cannot comment about it, and you should report it as such. For example, it is appropriate and proper to say, "I can't comment on the ascending aorta to rule out a dissection." It is important to not "overinterpret" unclear nondiagnostic images.
5. It is critically important to consider and interrogate objects in their entirety and to appreciate the three-dimensional nature of their anatomy. For example, when assessing ventricular function, make sure you see the ventricles from many angles before you report on their function. It is possible that a ventricle that appears to have normal contractile function in the base may have poor contractility in the mid- or apical portions. If you considered only the base, you may erroneously assess ventricular function as normal, when in fact, it is severely compromised.
6. Optimize the image by utilizing adequate ultrasound gel which facilitates the transmission of ultrasound waves.
7. Move your probe slowly, deliberately, and systematically to ensure that you are viewing all of the anatomy within the interrogation window. Even small changes of the angle of insonation may lead to missed or revealed structures or portions thereof.
8. It is important to understand and mentally visualize the anatomy of the target structure to ensure complete examination, and to facilitate the detection of anatomical abnormalities.
9. Properly document your ultrasound examination. Recording still and video images of your examination will facilitate subsequent examination by others and will serve as proper documentation.
10. Do not hesitate to seek the guidance of those with more experience in diagnostic imaging. Ultrasound is an extremely important diagnostic tool, but an improperly conducted or interpreted examination can have significant implications on diagnostic decision-making and patient management.

In order to simplify our objective, we present a basic but systematic approach to ultrasound examination. This approach will facilitate the understanding of the basic concepts and how to apply them to facilitate diagnostic decision-making and management in a fashion that improves patient outcome. We particularly value the educational videos as they serve as a compendium of instructional images and videos on a wide variety of clinical conditions.

The main conditions a novice practitioner in the field should be able to detect and evaluate include moderate to severe ventricular dysfunction, ventricular dilation, pulmonary embolism, severe hypovolemia, major valve pathology (stenosis and/or insufficiency), aortic aneurysm, aortic dissection, pericardial and pleural effusion, cardiac tamponade, pneumothorax, pulmonary edema, intra-abdominal fluid collection, and fluid or thrombus behind the bladder. The anatomical appearance of these pathophysiological states exceeds the scope of this chapter, which is intended as a guide to the performance of the examination itself.

In the companion chapter "Common Pathophysiologic findings on TTE and FAST Examination" we will discuss some common pathologies, and pertinent anatomy.

Transthoracic Echocardiography (TTE)

TTE provides a more rapid, noninvasive method to examine and provide data on cardiac anatomy and function. TTE helps in the assessment, diagnosis, and management of many conditions at the bedside, particularly in the setting of Emergency Medicine, Trauma, Critical Care, and perioperative care.

The use of TTE in acute care settings sometimes differs from that in nonacute care settings. TTE is employed in a "focused" fashion, frequently with a view to ruling out significant gross pathology, as opposed to detecting diagnostic subtleties. Another difference is that in acute care settings, the ultrasound survey is often done simultaneously with other diagnostic or therapeutic interventions. Additionally, TTE in acute care settings is used in a less comfortable environment, with space and operator position constraints. Additionally, TTE examinations are frequently performed in the supine position because of the needs of others that are simultaneously providing patient care, despite the fact that optimal imaging of cardiac structures may require positioning in the left lateral decubitus position or at least a left lateral tilt of the chest.

In cardiac surgery, surface ultrasonography can be done to obtain preprocedural information that would allow intra- and postprocedural comparisons. An example would be the case of transcatheter aortic valve replacement (TAVR) done in a sedated patient without endotracheal intubation when transesophageal echocardiography (TEE) imaging is less feasible. Another reason to emphasize the use of TTE for cardiac surgery is its academic value, as most centers do TEE in all cardiac patients. When this is done, centers which perform a TTE in the preoperative setting have the opportunity to compare its findings with the intraoperative TEE, which is a great learning opportunity.

TTE Equipment

For every ultrasonic examination, there are four main components; the ultrasound machine, the ultrasound probe(s), the patient, and the operator. The operator must not only understand the equipment, controls, limitations, and capabilities but have a good understanding of human anatomy. The sensitivity and the specificity of the

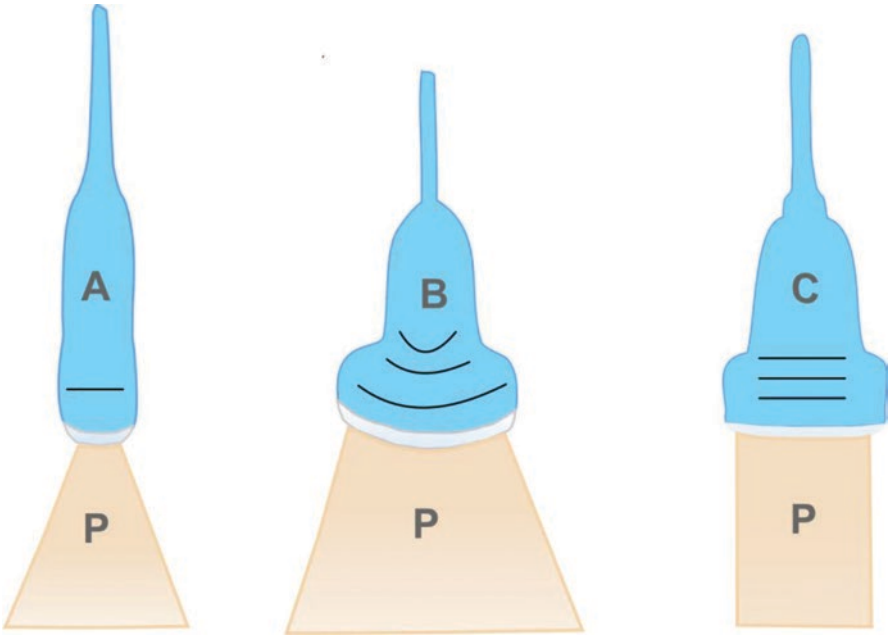

Fig. 10.1 Three types of ultrasound probes**;** (**a**), a sector array; (**b**), a curvilinear array; and (**c**), a linear array probe. The transducers are shown in blue, with their ultrasound element areas shown in white. The respective ultrasound imaging planes (**P**) are depicted in yellow. The maximum depth of signal of each probe is not shown, but in general sector and curvilinear array probes (**a** and **b**) can penetrate up to 30 cm, whereas linear array transducers (**c**) can penetrate up to 9 cm

examination is correlated with the quality of the ultrasound machine and probe, and importantly with the operator training, knowledge, and experience.

There are three ultrasound probes that are best suited for a surface examination (Fig. 10.1). Most modern probes are multiwavelength probes, meaning that they can work in a range of ultrasound frequencies that can be selected as needed. In addition, multiwavelength probes have the range displayed on the probe or on the ultrasound machine display once the probe is active. The higher frequency probes (5 MHz or more) are designed for more anatomically superficial examinations and provide higher resolution images with lower penetration, whereas lower frequency probes (4 MHz or less) are designed for deeper structures, but will output a lower resolution and thus a coarser image. Unfortunately, probe frequency and signal penetration are inversely related. The other element in probe selection is the probe area that will be in contact with the patient's surface, or "footprint." Probes are often designed with anatomical considerations in mind in order to allow a proper imaging window. Depending on the manufacturer, there are minor differences in ultrasound probe design and frequency used to examine particular areas. Often probes have their type and frequency range written on them, such as a "sector, 5 to 1 MHz" probe will be denoted as "S5–1," and a "linear 12 to 3 MHz as "L12-3," a "curvilinear 5 to 1 MHz" noted as "C5–1," and so on.

Most probes have a probe position indicator on the side that is used to facilitate correct probe orientation in relation to the image displayed in the screen.

A TTE probe that can be used for most adult diagnostic examinations is the "5–1 MHz sector array transducer". This probe is ideal for heart, and pleural cavity examinations as its footprint is a relatively small square of approximately 2 cm that can "fit" in between the intercostal spaces. Its lower frequency allows for a deeper ultrasound wave penetration (25–30 cm). This probe can be used to examine the abdominal cavity as well but will show a smaller field of view (less area displayed on the screen at the same time) due to its small footprint and a maximum examining angle of no more than 90 degrees. It is less optimal for lung examination for pneumothorax or pulmonary edema due to its lower frequency, making the near field image appear grainier, but it still can do the job. For lung examination, vascular access, and nerve blocks a "12–3 MHz linear array transducer" is ideal due to its high frequency and large footprint of around 4 cm, but its utility for cardiac and abdominal examination is marginal. For the abdominal cavity, a "5–1 MHz curved array transducer" allows maximal abdominal cavity signal penetration, and its larger angle of examination (110 degrees) and footprint (6 cm) allow a larger field of view resulting in a larger anatomical area on the screen in each probe position. This probe is also ideal for obese patients. A "9–2 MHz curved array transducer" would work well for lung and abdominal examinations due to its higher frequency, large footprint (5.5 cm) and the added benefit of a large field of view (100 degrees) in a single image. In summary, the only probe that can accomplish all tasks required for TTE and FAST exam is the adult TTE probe.

Ultrasound Imaging Modalities

Modern ultrasonography uses several diagnostic modalities. In this chapter, we are only going to refer to two-dimensional (2D) imaging, color flow Doppler (CFD), and continuous wave Doppler (CWD).

Two-dimensional images are created when the ultrasound probe sends ultrasonic waves through human tissues and registers the reflected waves from underlying structures, and the image is reconstructed by a computer. CFD analysis can be done when a segment of the 2D image is selected by activating controls in the machine, and then very small subsegments are individually analyzed using the Doppler principle. These subsegments are tagged with color by the computer depending on the direction and speed of movement of the blood within them. This creates a color pattern depending on whether the blood is moving toward or away from the ultrasound probe. Thus, flow moving towards the ultrasound probe will be always RED color, while flow moving away from the ultrasound probe will be always BLUE color. A helpful mnemonic is the famous yellow cartoon character BART (Blue Away, Red Towards). This modality is ideal for evaluating abnormal heart communications and valvular regurgitation. CWD also depends on the use of the Doppler principle: Objects (in this case red cells) moving towards the ultrasound probe reflect sound waves that are more compressed than they were insonated with, thus increasing the frequency – positive Doppler shift – whereas red cells moving away from the ultrasound probe reflect sound waves that are more apart, causing a drop in the receiving frequency or a negative Doppler shift. By convention, blood moving toward the probe is registered above the tracing baseline, whereas blood moving away is below it.

Insonation Windows and Imaging Planes

There are several ultrasound interrogation windows that can be utilized to obtain views of the cardiopulmonary and abdominal areas. In the postoperative setting, as a result of thoracic or abdominal dressings, several alternative windows can be used to provide some or all of the information sought (1). It is important to note that often an adequate image cannot be obtained from a particular ultrasonic window to make any assessment or diagnosis. However, one must always remember that the information can be obtained from other windows, often nonstandard windows, and that even incomplete information obtained may be of significant diagnostic utility.

The thoracic windows (areas where the ultrasound probe should be placed to see specific anatomical and pathological information) allow one to examine the heart, lungs, pleural spaces, and thoracic aorta. These windows are the following: parasternal long- and short-axis views, apical 5, 4, and 3 views, the right subcostal, and subxiphoid views (Figs. 10.2 and 10.3).

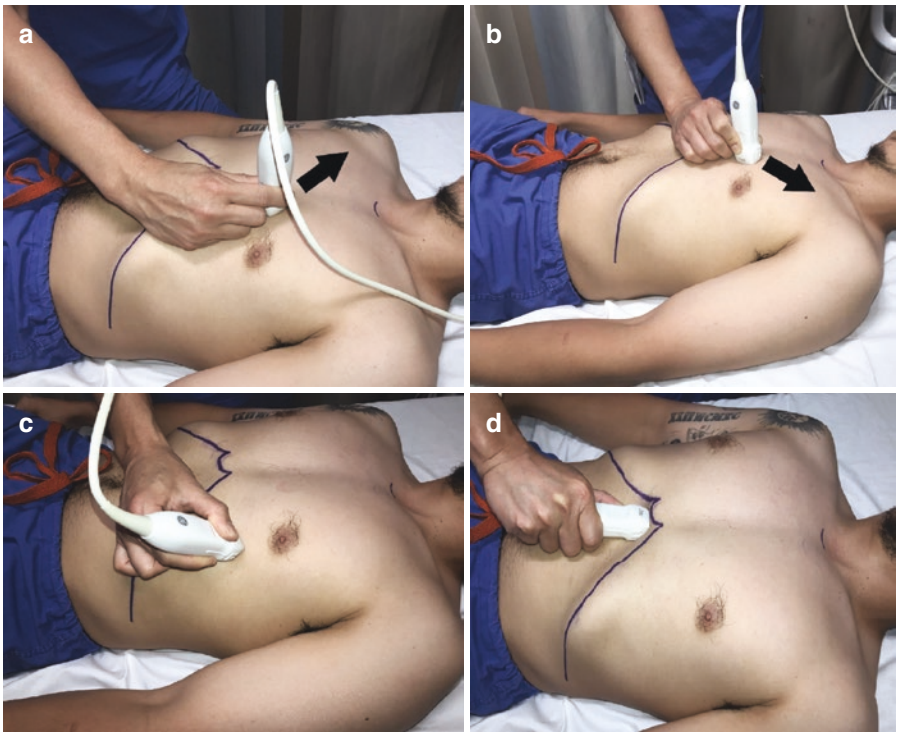

Fig. 10.2 Position of the patient and anatomical windows to show the areas where the ultrasound probe should be placed to obtain the following windows: Parasternal long-axis views, parasternal short-axis views, position, apical position, the right subcostal and subxiphoid views, position, abdominal aorta long-axis view position, the bladder view, the right lateral and posterior pleural cavity, hemidiaphragm, liver, and right kidney, positions, left lateral and posterior pleural cavity, left hemidiaphragm, spleen, and left kidney position

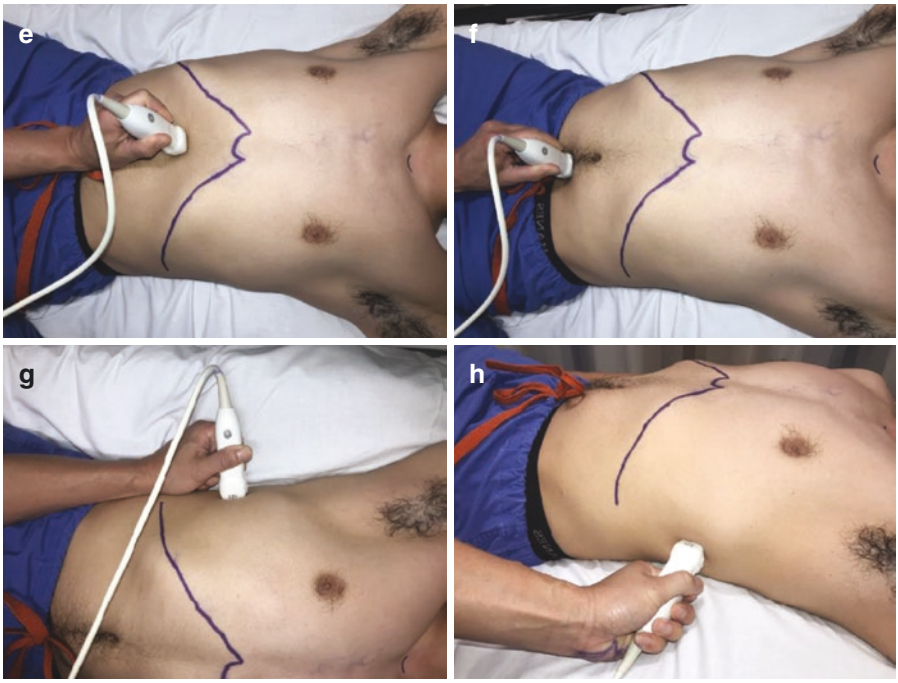

Fig. 10.2 (continued)

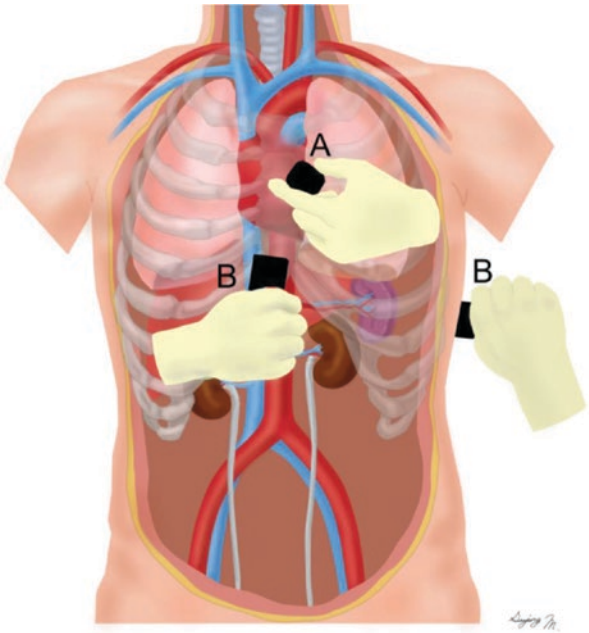

Fig. 10.3 Common ways to hold the TTE probe**.** The probe can be held as it if were a large pencil, between the first and second or second and third digits (**a**), or in one's fist like a tennis racket (**b**)

Parasternal long axis:

1. Third–fourth intercostal space left from the sternum.
2. The marker is aimed toward the right shoulder.
3. Two-scan depth.
   (i) Cardiac between 12 and 16 cm
   (ii) Effusion between 20 and 24 cm

Parasternal short axis:

1. Rotate transducer 90° from parasternal long axis to the left shoulder.
2. Tilt transducer down the left flank to start from the base of the heart.
3. Depth between 12 and 16 cm.

Apical 4 Chamber:

1. Transducer is placed on the apex (usually fifth intercostal space).
2. Tilt the transducer along the axis of the heart.
3. The marker aiming toward the patient's axilla.
4. Depth between 14 and 18 cm.

Subcoastal:

1. Transducer is placed under the xiphoid.
2. Tilt transducer along the axis of the heart.
3. Depth between 12 and 16 cm.

All of these windows have "in-between" windows as well. For example, moving the probe from the xyphoid window in the midline toward the bladder, the abdominal aorta, and vena cava can be examined. The abdominal examination targets can be separated into the four anatomical quadrants, more precisely focusing on specific organs and structures that allow a simpler identification of pathology, if there is any. When we use solid organ assessment as part of the examination, the process is simplified as these organs provide great contrast background to define pathological states, like fluid or clotted blood around or in-between them. Any organ that contains air (stomach and intestine) or subcutaneous emphysema will block the ultrasonic window. We can try to avoid stomach or intestinal air by "pushing" the gas away by exerting additional probe pressure, or by moving the probe slowly away and changing the angle to aim toward the area of interest while trying to

avoid intracavitary air. The right upper quadrant examination should include views of the right hemidiaphragm, liver, right kidney, and IVC; the left upper quadrant examination should include views of the left hemidiaphragm, spleen, left kidney, and aorta. In the upper abdominal midline area, we should evaluate the abdominal aorta and IVC, from top to bottom, as well as in the suprapubic area of the bladder area. Even if the operator does not know the names or "precise" locations of the potential anatomical windows used in ultrasonography, his or her understanding of anatomy should be sufficient to allow selection of potential windows based on human anatomy.

In the discussion below, we do not wish to leave the reader with the impression that all images can be obtained with ease and without a learning curve. In fact, it is often difficult to obtain satisfactory images that match an idealized standard. The practitioner does need to practice the acquisition of the images with deliberate repetition before he or she can expect to be capable of performing the exam on most patients.

Examination Windows

1. Parasternal long-axis views:

Obtained by placing the probe vertically, oriented with its imaging long axis pointing to the right shoulder in the area between the third and fourth or fourth and fifth intercostal spaces, just lateral to the left sternal border (Fig. 10.4).

Sometimes, because the lung is interposed in this area, no useful image can be obtained. In this case, the probe can be moved lower to seek another window, but then a true parasternal long view will not be obtained.

Alternate planes from this view are obtained by tilting the probe slightly to the right; this will demonstrate the lateral aspect of the left atrium (LA), mitral valve (MV), left ventricle (LV), the main pulmonary artery (PA), and pulmonic valve (PV).

Tilting to the left will show the medial aspects of the LA, MV, and LV, and views of right heart in long axis with the inferior vena cava (IVC), right atrium, tricuspid valve, and right ventricle (Fig. 10.5).

This window is also useful for evaluating pericardial effusions that are large enough to affect the base of the heart, left pleural effusions, the function of the right ventricular outflow track (RVOT), and base and mid portions of the left ventricle (LV), the aortic valve (AV), the aortic root (AOR), the left atrium (LA), the mitral valve (MV), a portion of the descending thoracic aorta (DTA), and part of the left pleural cavity (LPC).

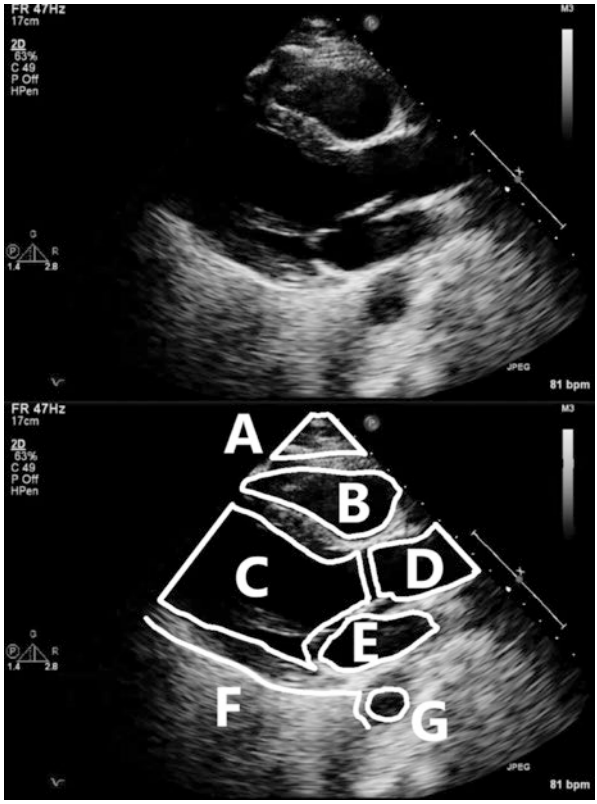

Fig. 10.4 Parasternal long-axis view of the left heart. The top is the unlabeled image, bottom is with labels; (**a**): anterior chest wall; (**b**): right ventricular outflow tract; (**c**): long-axis left ventricle, with anteroseptal wall on top and inferolateral wall on bottom; (**d**): aortic root, with AV between "(**c**)" and "(**d**)"; (**e**): left atrium, with mitral valve between "(**c**)" and "(**e**)"; (**f**): line delineating pericardium and left pleura; (**g**): descending thoracic aorta in short axis (Video 10.1)

2. Parasternal short-axis views:

From the parasternal long-axis view, the probe is rotated 90 degrees approximately until a short-axis view of the base or midportion of the LV develops (Fig. 10.6).

Alternate planes from this view are obtained by tilting the probe caudally thus aiming the US beam cephalad where the MV, and base of the RV is seen, and as we continue to tilt caudally the RA, the RV inflow–outflow view, main PA, the AV in short axis, and posterior to it the LA can be seen (Fig. 10.7).

By tilting the probe cephalad, the base of the RV and LV with the TV and MV can be seen. As we continue to tilt cephalad, the midportion of the RV and LV with the papillary muscles are visualized, and finally, the apical segments of the ventricles emerge.

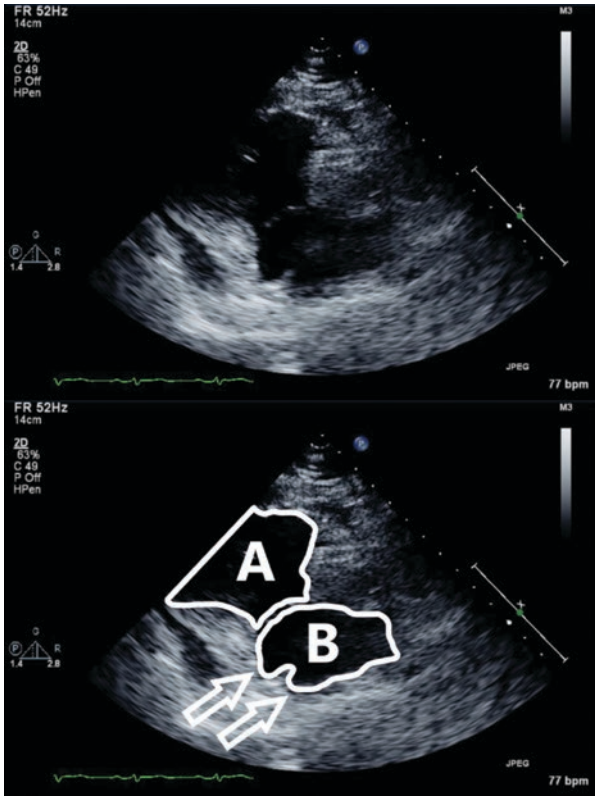

Fig. 10.5 Parasternal long-axis view of the right heart. Top: unlabeled image. Bottom: with labels. To obtain these views, the prove is tilted to the left; (**a**): right ventricle; (**b**): right atrium. Area between "**a**" and "**b**": tricuspid valve; top arrow depicts the opening of the coronary sinus, and bottom arrow indicates the inferior vena cava as it joins the right atrium (Video 10.2)

This view allows assessment of the TV, PV, AV, ventricular function, wall motion abnormalities, cavity dilation, hypovolemia, loading conditions, pericardial, and left pleural effusions.

3. Apical chamber views:

Place the probe on the area of the maximal heart impulse, usually in the fifth intercostal space and mid-clavicular line (note that the probe may need to be oriented more laterally and lower in very large hearts), aiming it toward the right shoulder and rotating it until a "4-chamber view" (4Ch) is obtained (Fig. 10.8).

By convention, a true 4Ch view is one where the four chambers of the heart are visible, and no portion of the LVOT or AV appears in the image. When the latter structures do appear, the convention is to refer to that view as the "5-chamber view."

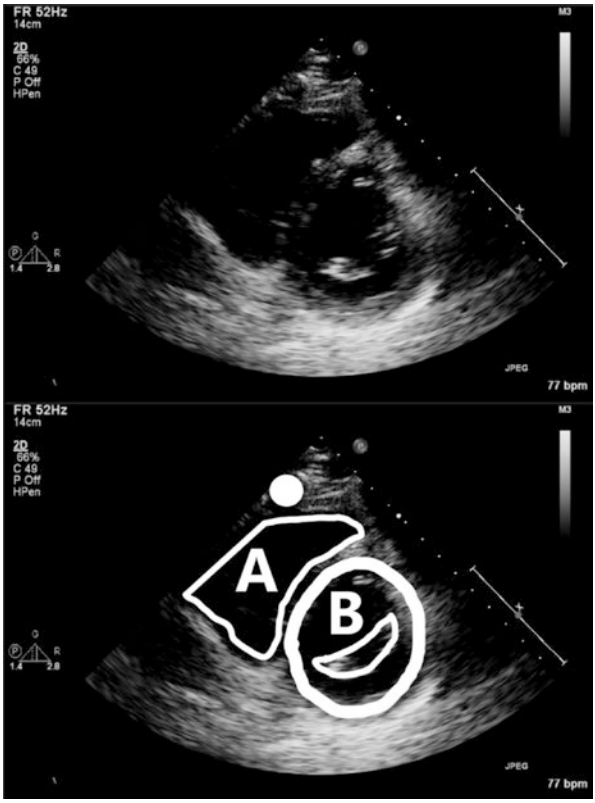

Fig. 10.6 Parasternal short-axis view**.** Short-axis view of the base of the right (**a**) and left ventricle (**b**). The area between "**a**" and "**b**" is the interventricular septum. The white dot represents a rib cartilage that only allows some ultrasound waves to pass through it and thus the area imaged distal to is blurred. Note the quarter moon shape inside the left ventricle, this is the mitral valve with its anterior leaflet on top and posterior leaflet below (Video 10.3)

From the apical 4Ch view, alternate planes are obtained by tilting the probe caudally, thus aiming the US beam cephalad. This is where the 5-chamber view will be seen, as the anteriorly located LVOT and AV come into the imaging plane (Fig. 10.9). Using CWD, this view will enable you to measure velocities across the AV as the ultrasound beam and the AV flow are well aligned (Fig. 10.10), and will make it easy to detect abnormally high velocities such as the ones seen with aortic stenosis.

The apical views allow assessment of the ventricular function and size, atrial sizes, TV, MV, AV, and pericardial effusions.

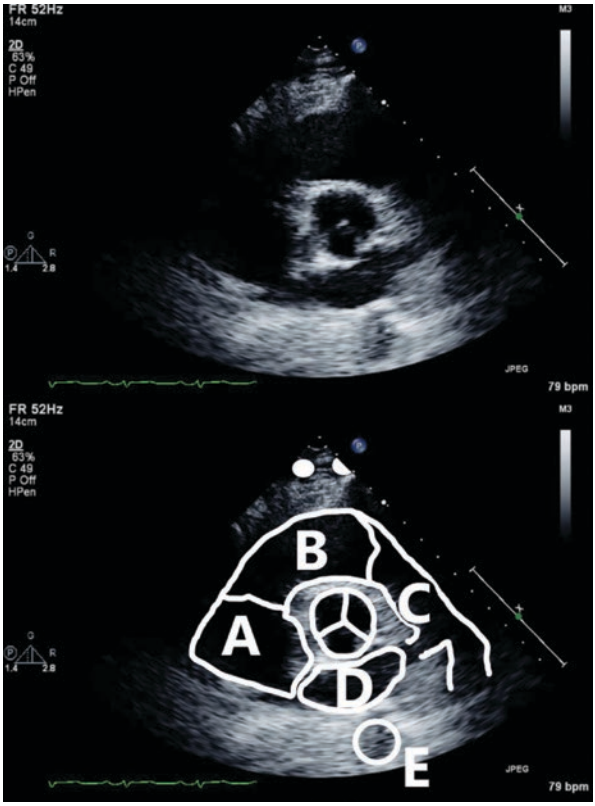

Fig. 10.7 Parasternal short-axis view. The probe has been tilted caudally from the left ventricular short-axis view, making the ultrasound plane scan cephalad. (**a**): right atrium; (**b**): right ventricle; (**c**): pulmonary artery and its bifurcation; (**d**): left atrium; (**e**): descending thoracic aorta in short axis. The tricuspid valve is located between "**a**" and "**b.**" The pulmonic valve is located between "**b**" and "**c.**" The interatrial septum is located between "**a**" and "**d.**" The aortic valve in short axis is located in the middle of the figure. The three cusps are noted. The cusp closest to the interatrial septum is the noncoronary cusp, the cusp near the pulmonary artery is the left coronary cusp, and the cusp near the right ventricle – usually anterior in the image – is the right coronary cusp. Note the two white dots at top of the image represent two ribs, and the black area beyond is the shadowing generated due to the ultrasound beam's inability to penetrate through the ribs (Video 10.4)

4. Subxiphoid views:

This view is often referred as the "subcostal view." The probe is placed just below the xiphoid or right costal margin and is held like a tennis racket, to facilitate pressing down if necessary. The probe should be positioned parallel to the skin to allow imaging below the sternum and ribs. It should be aimed initially toward the right shoulder, and the IVC should be visualized and traced toward the RA. This is done as imaging planes are obtained by rotating the probe to the patients' neck and then left shoulder as necessary until a subcostal view is obtained (Fig. 10.11).

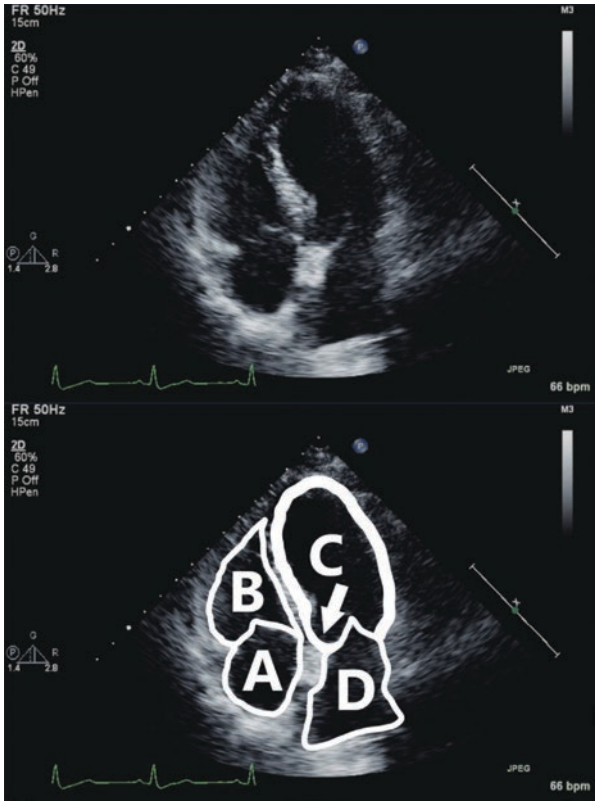

Fig. 10.8 Apical four chamber view. This image is between a "4-chamber view" and a "5-chamber view" and was obtained tilting the probe slightly caudad to have the ultrasound beam aim more anteriorly and allowing the left ventricular outflow tract to come into view. (**a**): right atrium; (**b**): right ventricle; (**c**): left ventricle; **ARROW**: left ventricular outflow tract; (**d**): left atrium. Tricuspid valve between "(**a**)" and "(**b**),", mitral valve between "(**d**)" and "(**c**)," interatrial septum between "(**a**)" and "(**d**),**"** and interventricular septum between "(**b**)" and "(**c**)" (Video 10.5)

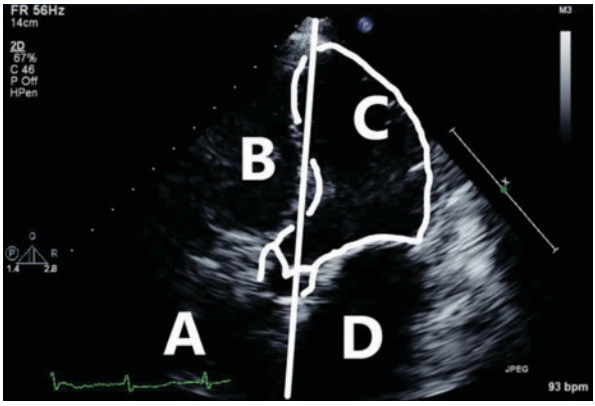

Fig. 10.9 Apical 5-chamber view. (**a**): right atrium; (**b**): right ventricle; (**c**): left ventricle; (**d**): left atrium. The straight **white line** represents the continuous wave Doppler (CWD) cursor positioned through the left ventricular outflow tract (LVOT) and the aortic valve (AV). The CWD ultrasound beam will measure blood flow velocity across the entire path of the beam, and thus the highest velocity will be generated by the smallest area (narrowest orifice) through which the stroke volume has to travel. In a normal heart this area is the AV, with a velocity that ranges between 1 and 1.4 m/sec. Note the aortic valve and mitral valve are closed in this image, representing ventricular isovolumetric contraction

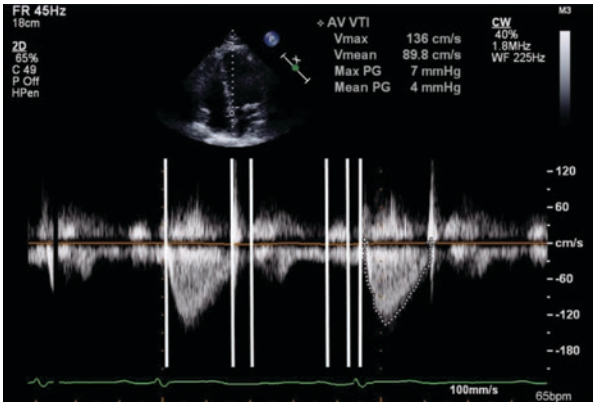

Fig. 10.10 Continuous wave Doppler (CWD)**.** The tracing has been obtained with the cursor positioned through the left ventricular outflow tract. For velocities to be properly measured, the CWD cursor should be as parallel to the blood flow as possible (the same is true about pulse wave Doppler – PWD). The CWD tracing has a baseline that represents zero velocity and a velocity scale (cm/sec) on its left or right side that in reality has no limit, but our bodies cannot generate velocities above 6–7 m/sec. The baseline or horizontal axis is a time plot in seconds, usually set up to run at 100 mm/second – four times the standard EKG speed. The baseline can be moved up or down to allow more screen space for velocities that move away (below the baseline) or towards the ultrasound probe (above the baseline). In this case a systolic velocity of 1.36 m/sec is detected (see the EKG tracing to help with systolic and diastolic timing of events). Using the modified Bernoulli equation ($P = 4 \times V^2$, where P is pressure in mmHg and V is velocity in cm/second) to estimate the pressure gradient generated through the narrowest area (in this case the aortic valve), a peak gradient of 7 mmHg is recorded. Note the **white lines** placed by us on the image; they separate all of the events that occur in a cardiac cycle, starting in this example with systole: v**entricular ejection, i**sovolumetric ventricular relaxation, passive ventricular filling, atrial contraction ("atrial kick"), and isovolumetric contraction

It is very useful to try to "use" the liver as a window to see the heart because often, at the epigastrium or left subcostal area, air from the stomach or colon can block the view (if the probe is not directly placed under the rib/xiphoid area).

This view allows a general assessment of the ventricular function and size, atrial sizes, interatrial septum, TV, MV, AV, and pericardial effusion.

5. Right flank views:

For all flank views where ribs are present, it is important to remember that they create complete shadowing, and thus a "black curtain" is present behind them where the US cannot penetrate. This black area can easily be confused with the appearance of a pleural effusion. We image the flank areas with the probe oriented in a coronal (frontal) plane; this will allow viewing multiple structures in the same screen, where they can be used as anatomical landmarks. The probe is placed in

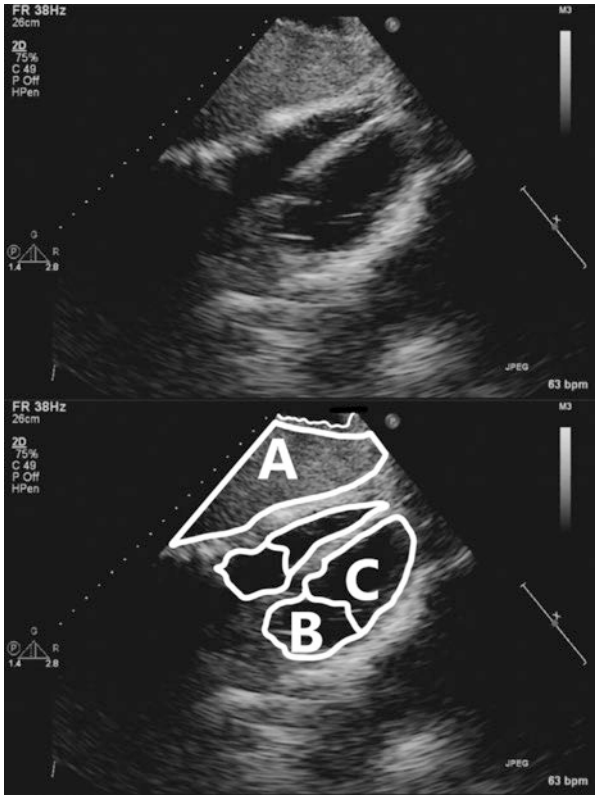

Fig. 10.11 Subxiphoid views**.** With the probe placed just below the xiphoid appendage, the abdominal wall is seen, then the liver "(**a**)," and through it the right heart on top and then the left heart bellow. (**b**): left atrium, below the right atrium; (**c**): left ventricle, below the right ventricle (Video 10.6)

the fourth or fifth intercostal space between mid- and posterior axillary lines. The probe is then to be slowly slid inferiorly to evaluate, from top to bottom, the right lung and pleural cavity, hemidiaphragm, liver, right kidney, and all spaces between these structures (Figs. 10.12 and 10.13). Once the liver has been identified by tilting the probe anteriorly (tilting the US plane posteriorly), often the IVC and then with added tilting the abdominal aorta (AO) in the far field can be seen in their long axis. The IVC and AO can be seen as well by scanning the liver anteriorly in the sagittal plane, with the IVC to the right of the patient's spine (Fig. 10.14) and the AO to the left (Fig. 10.15).

These views allow the assessment of the right lung, right pleural and abdominal effusions, and IVC.

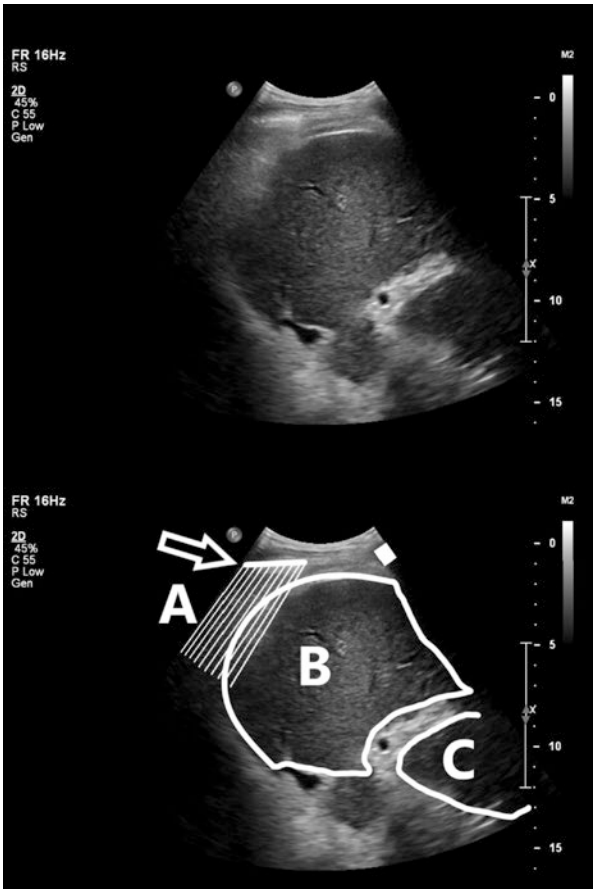

Fig. 10.12 Right flank view with lower lung, diaphragm, liver, and kidney views. Placing the probe between mid- and posterior axillary line in a coronal (frontal) plane, we can see to the left of the image an arrow representing the lung and below it the artifact (**a**) generated by the lung. **b**: liver. Note the convex subdiaphragmatic liver surface. (**c**): the right kidney upper pole. The space between the inferior surface of the liver and the kidney is called the hepatorenal recess or Morrison's pouch. To the right of the image the small white square represents a rib and below it is a black shadow generated by lack of ultrasound penetration. Note how the shadow generated from the lung is different than the one generated by bone. The lung shadow generated by the lower portion of the lung will move with respiration

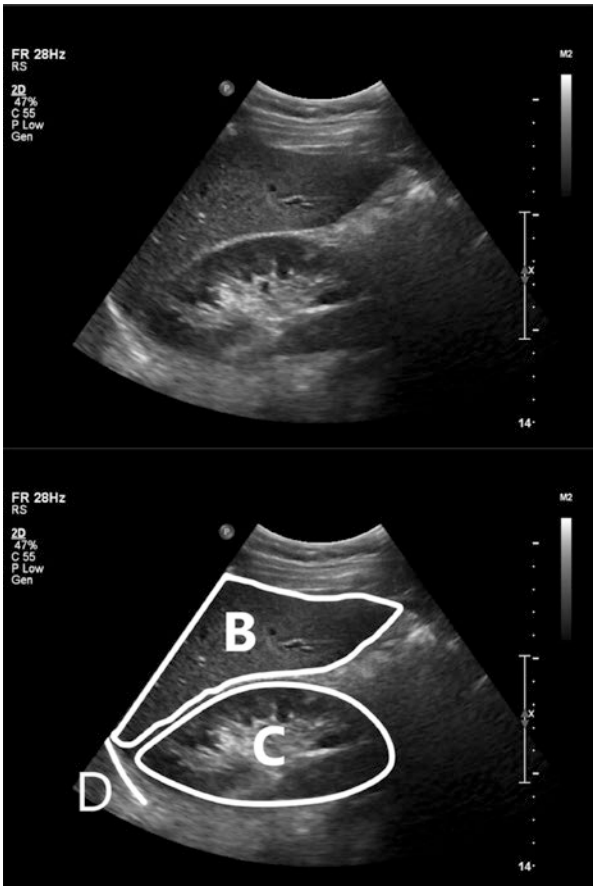

Fig. 10.13 Right flank view. The probe has been moved inferiorly from Fig. 10.12, with the posterior diaphragm (**d**), liver (**b**), and kidney (**c**) coming into view

6. Left flank views

The probe is placed initially in a coronal (frontal) plane at the fourth or fifth intercostal space close to the posterior axillary line, as the spleen lies posteriorly in the abdominal cavity. The probe is then slowly advanced inferiorly to evaluate, from top to bottom, left lung and pleural cavity, hemidiaphragm, spleen, left kidney, and all spaces between these structures (Fig. 10.16). Often the descending thoracic–upper abdominal AO can be seen as well.

This view allows assessment of the left lung, left pleural and abdominal effusions, and AO.

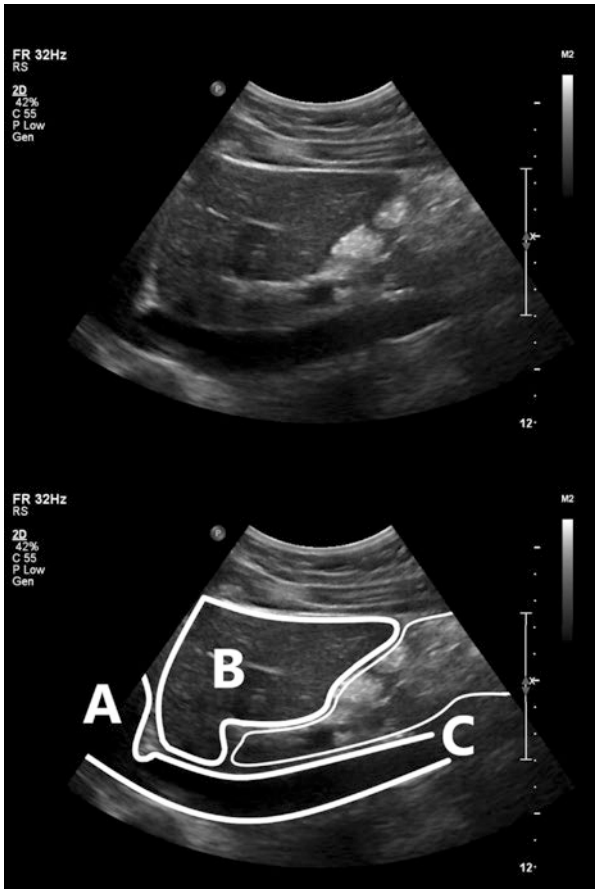

Fig. 10.14 Right upper quadrant view through the liver with a focus on the inferior vena cava (IVC) in long axis (**c**). (**a**): right atrium – IVC. (**b**): liver. The area traced by the thin white line corresponds to intestinal content

7. Abdominal aorta and caval views:

With the probe placed in the subxiphoid area and the US beam oriented left–right to generate a transverse plane view, and with the probe indicator to the right, the AO will be seen to the right on the screen, and the IVC to the left (Fig. 10.17). Both vessels will be in short axis. A caudad tilt of the probe will allow an added view of the proximal AO. The probe should be slid distally and vertically toward the pubis while following the AO until its bifurcation, somewhere near the upper umbilical area. If air is encountered while scanning, pressure can be applied to the probe. Asking the patient to exhale can facilitate the examination. If measurements of the aorta are taken, they are done from outer wall to outer wall.

This view allows assessment of abdominal aortic aneurysms and dissection, and flow status into the celiac trunk, superior mesenteric artery, and common iliac arteries. If difficulty is encountered visualizing flow in the iliac arteries, a superficial scan of the proximal femoral arteries in the groin area can be done.

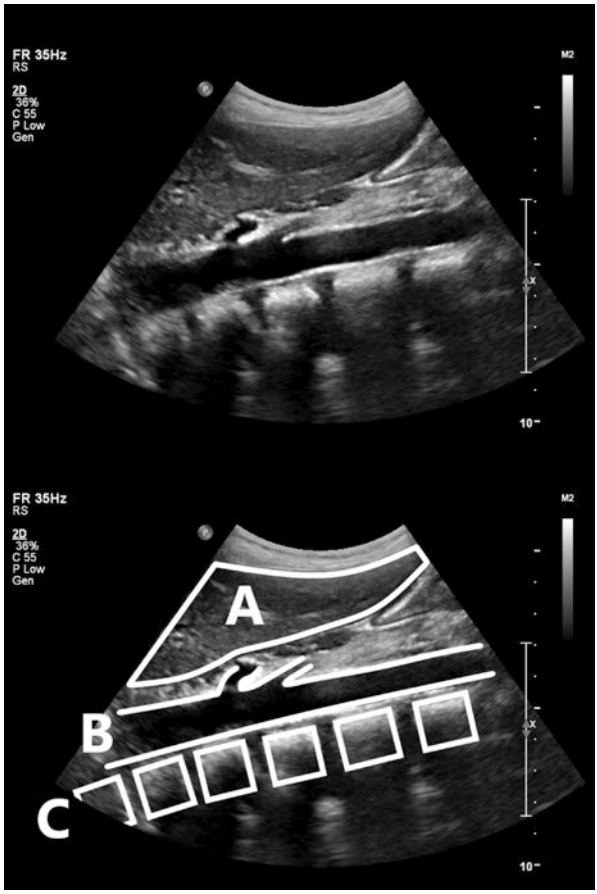

Fig. 10.15 Right upper quadrant view through the liver (**a**) with a focus on the abdominal aorta in long axis (**b**). From left to right, the celiac trunk and the superior mesenteric artery takeoff are visible. This aortic view is obtained by tilting the probe to the right when the image in Fig. 14 is obtained. This will allow the ultrasound beam to move to the left. The probe can also be slid to the left of the patient to obtain this view. The aorta and the IVC are very close to each other, and only minimal motion is necessary. (**c**): vertebral bodies

8. Bladder views

The probe is placed in a transverse plane, immediately above the pubic symphysis and tilted cephalad, then caudad. Unless the patient has just voided, has end-stage renal disease, or has an indwelling bladder catheter, there will be urine in the bladder. With the probe tilted cephalad, the prostate or uterus are easily visualized behind the bladder (Fig. 10.18).

This view allows assessment of lower abdomen and pelvic fluid collections.

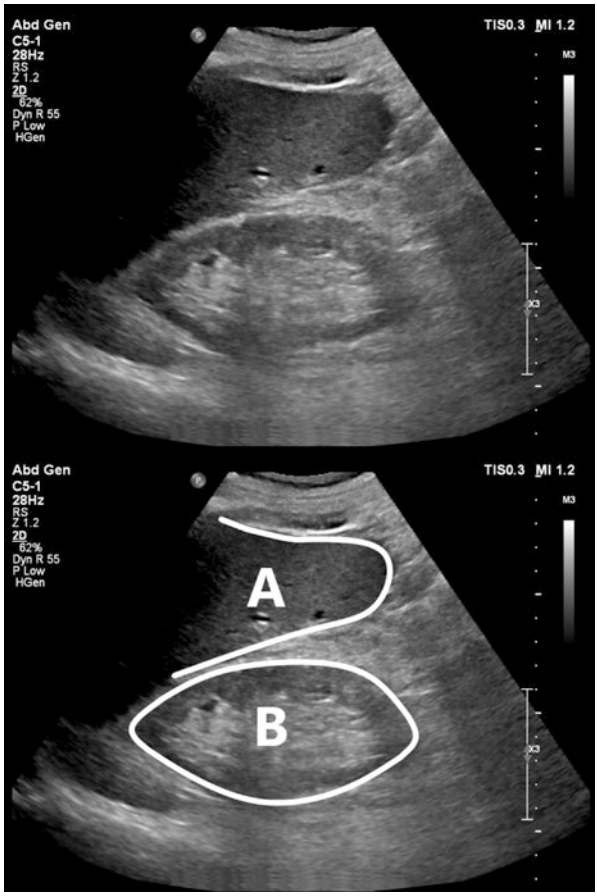

Fig. 10.16 Left flank view**.** The spleen is usually more posterior than we think thus the probe should be positioned at the posterior axillary line at the start of your exam. (**a**): spleen; (**b**): left kidney. The space between "**a**" and "**b**" is called the splenorenal recess or Koller's pouch

9. Left and right anterior chest views:

The probe is placed in a sagittal plane in the midaxillary line in order to scan several intercostal spaces cephalad to caudad, starting at the second intercostal space. Each space scanned should allow view of a full intercostal area with the rib above and below in short axis (Fig. 10.19), and this should be done for the left and right lung fields.

This view allows assessment of the left and right lungs. Some pathologies that can be visualized in this view include lung atelectasis, pneumothorax, and occasionally a large pleural effusion.

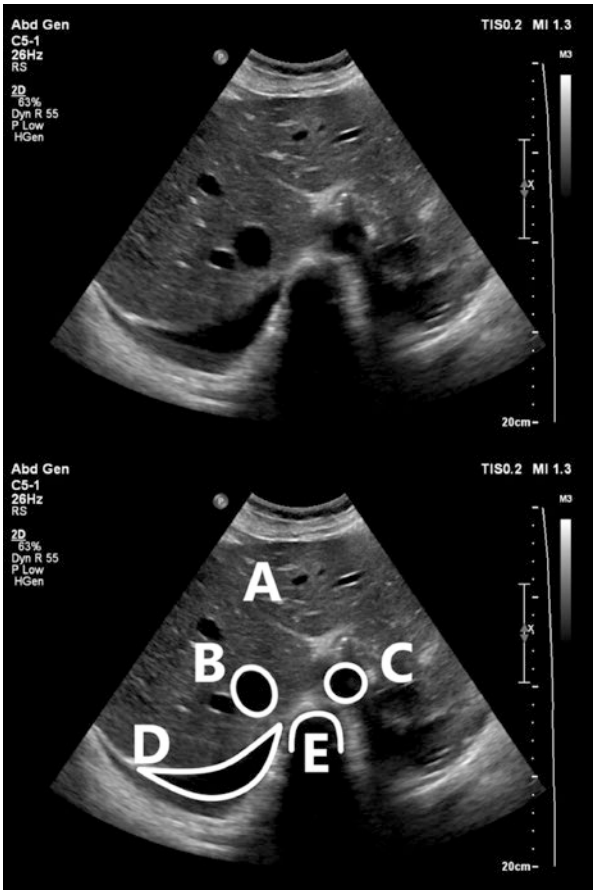

Fig. 10.17 Abdominal aorta and caval view**.** The probe is placed in the subxiphoid area in a transverse plane. The liver (**a**) occupies most of the image. The inferior vena cava (**b**) is seen to the left of the spine (**e**) on screen, and the abdominal aorta (**c**) is seen to the right of the spine. Note that the most inferior aspect of the right pleural cavity can be seen in this view demonstrating a right pleural effusion (**d**)

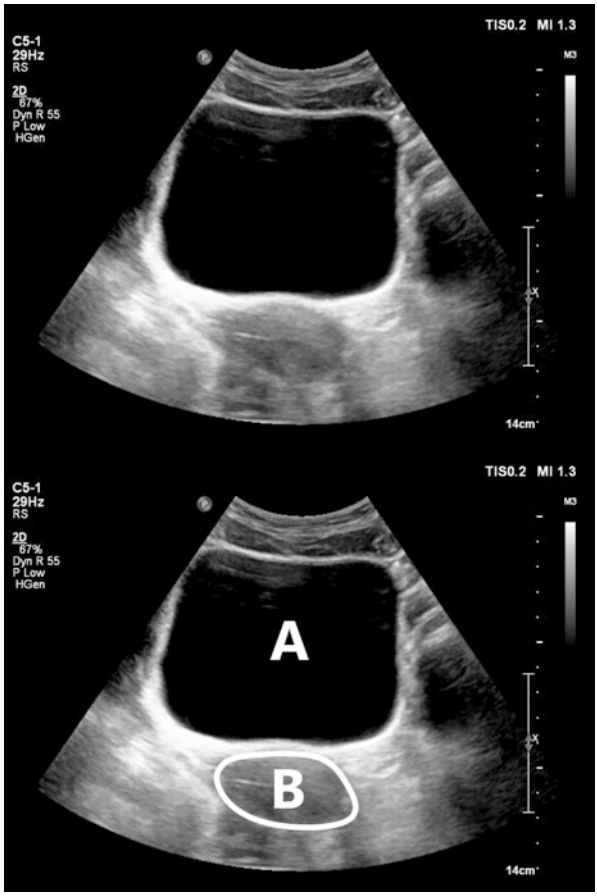

Fig. 10.18 Bladder view. The probe is placed initially in a transverse plane, just above the pubic symphysis. The bladder (**a**) and the uterus (**b**) are easily seen. The rectouterine excavation (pouch of Douglas) in women or the rectovesical excavation (pouch of Proust) in men should be explored for fluid collections

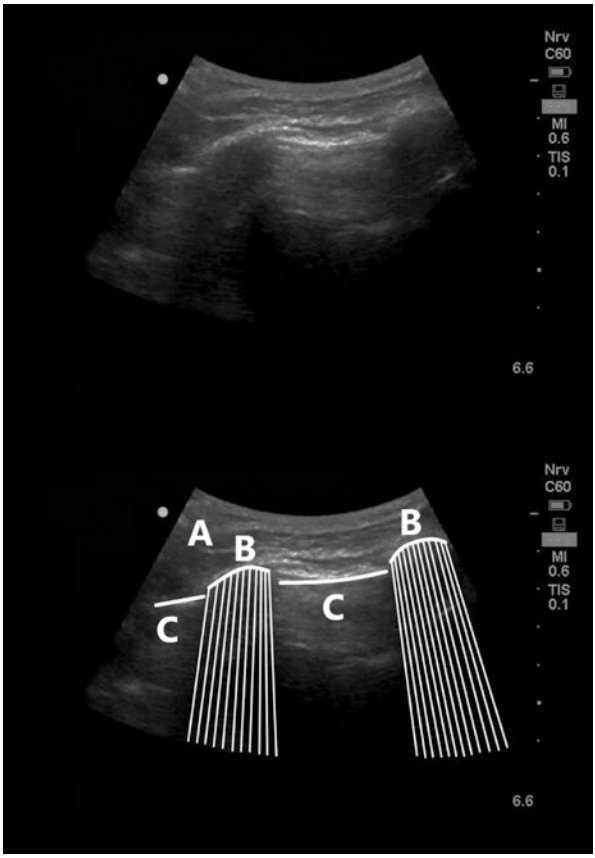

Fig. 10.19 Left and right anterior chest view. With the probe placed in a sagittal plane in the midaxillary line the chest wall (**a**) is scanned. Each space scanned should allow view of a full intercostal area with the rib above (**b**) and below (**b**) in short axis. Note the shadowing generated below the ribs, and it is thus impossible to see beyond them. The pleural line (**c**) seen sliding under the chest wall demonstrating there is lung present below the chest wall, and therefore there is no pneumothorax or pleural effusion in the area examined (Video 10.7)

Bibliography

1. Jensen MB, Sloth E, Larsen KM, Schidt MB. Transthoracic Echocardiography for cardiopulmonary monitoring in the intensive care unit. Eur J Anaesthesiol. 2004;21:700–7.

Common Pathophysiologic Findings on TTE and FAST Examination

11

Enrique J. Pantin and Denes Papp

You cannon open a book without learning something

Confucius

Brief History of Ultrasonography

The history of the discovery and use of ultrasound is a long and interesting one. The use of ultrasound in medicine began in the late 1930s, when its potential in aiding medical diagnosis was realized. In 1942, the Austrian physician Dr. Karl Theodore Dussik published the first paper in medical ultrasonography titled: "On the possibility of using ultrasound waves as a diagnostic aid". The cooperation between Dr. Inge Edler, considered the "Father of Echocardiography," who was in charge of the Cardiology Department at the University Hospital, Lund, Sweden, and the Physicist Carl Hellmuth Hertz who worked at the nuclear physics department of the University of Lund made the original description of "M-mode" imaging possible in 1953. By early 1955 Dr. Edler was so confident in the technique that he relied solely on this modality of ultrasound for the diagnosis of mitral stenosis, a diagnosis that until then required cardiac catheterization. The diagnostic utility of ultrasound was elucidated by Dr. Ian Donald, Dr. John McVicar, and Tom Brown in a manuscript titled "Investigation of Abdominal Masses by Pulsed Ultrasound," published in *the Lancet* in 1958. The evolution and introduction into other fields of medicine was progressive in the 1960s. Transesophageal echocardiography began to be used by anesthesiologists in the early 1990s. In 1999,

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literature began to be published recommending the routine use of ultrasound for regional nerve blocks and central vascular access. Currently, many medical specialties utilize diagnostic ultrasound imaging.

Introduction

To learn ultrasonography, we must apply the same principles used to lean a new language. The only way to learn and perfect this new language is to practice as often as possible, to take every opportunity to examine a patient and to use local resources (teachers, echo laboratory, etc.) to perfect this new language.

Ultrasound has become an extension of our senses, as the utility of ultrasonography as an integral part of the patient's "physical" examination has no parallel due to its accuracy and speed of data acquisition. There are many acute and chronic pathophysiologic conditions that we can accurately diagnose and thus change the management of the patient.

In the following sections we describe common pathologies in the thorax and abdomen. The imaging sequence order is for organizational purposes only and should be changed depending on the clinical scenario. While obtaining views, it is advisable to use enough ultrasound gel, and to move the probe slowly, especially as you look for additional planes within a window. When doing so, one should rest one's hand on the patient's chest to avoid inadvertent probe movement, and also remember that oftentimes the image will appear to move away or disappear and then reappear due to respiratory motion. In this case, it is best to be patient and wait until the area of interest "comes back" into the screen. Once you obtain a view, it is important to remember that by slightly and slowly tilting the probe left or right, cephalad or caudad, or by rotating left or right, many imaging planes can be generated, and that when we tilt the ultrasound probe the beam crosses areas opposed to the probe movement. The ultrasound probe is commonly held in two different ways (with variations) depending on the window examined. Some images require the probe to sit as flat as possible against the patient's surface. It can be held as it if were a large pencil between the first and second or second and third digits, or in the palm of the hand like the grip of a tennis racket.

Basic Pathophysiologic Conditions

The conditions presented in this chapter can coexist. Thus, we should always try to perform an exam as completely as possible when conditions permit. It is important to realize that we are not only looking for pathologies, but confirmation of normal anatomy is equally informative.

Supplementary videos are available, and should be viewed to obtain the full learning benefit of this chapter. Certain aspects of the pathology such as ventricular hypokinesis can only be appreciated on moving images.

Some of the most common and important conditions to evaluate are severe ventricular dysfunction and dilation, severe mitral and aortic valve insufficiency and stenosis, pericardial effusion, pleural effusion, pneumothorax, aortic aneurysm and dissection, pulmonary embolism, and intra-abdominal fluid. Below are examples of some of these important pathologies.

Severely decreased ventricular function shown in [Fig. 11.1]:

Severely decreased ventricular function [shown in Fig. 11.2]:

Severely decreased ventricular function [shown in Fig. 11.3]:

Moderate aortic regurgitation [shown in Fig. 11.4]:

Severe aortic stenosis [shown in Figs. 11.5, 11.6, and 11.7]:

Severe tricuspid insufficiency [shown in Fig. 11.8]:

Pericardial effusion, moderate [shown in Figs. 11.9 and 11.10]:

Pericardial effusion, large [shown in Fig. 11.11]:

Pleural effusion, left, large [shown in Fig. 11.12]:

Pleural effusion, left, large [shown in Fig. 11.13]:

Acute pulmonary embolism [shown in Figs. 11.14, 11.15, and 11.16]:

Figures 11.14, 11.15, and 11.16 Elderly patient who had acute severe hypotension and desaturation in recovery room post total hip replacement. No thrombus seen, but signs of severe right heart strain were present. Diagnosis of pulmonary embolism was confirmed by computed tomography.

Right pleural effusion, large [shown in Fig. 11.17]:

Right pleural effusion and lung atelectasis [shown in Fig. 11.18]

Right pleural effusion and intra-abdominal fluid collection [shown in Figs. 11.19 and 11.20]:

Intra-abdominal fluid collection in left abdomen shown in [Figs. 11.21 and 11.22]:

Intra-abdominal fluid collection in left lower quadrant:

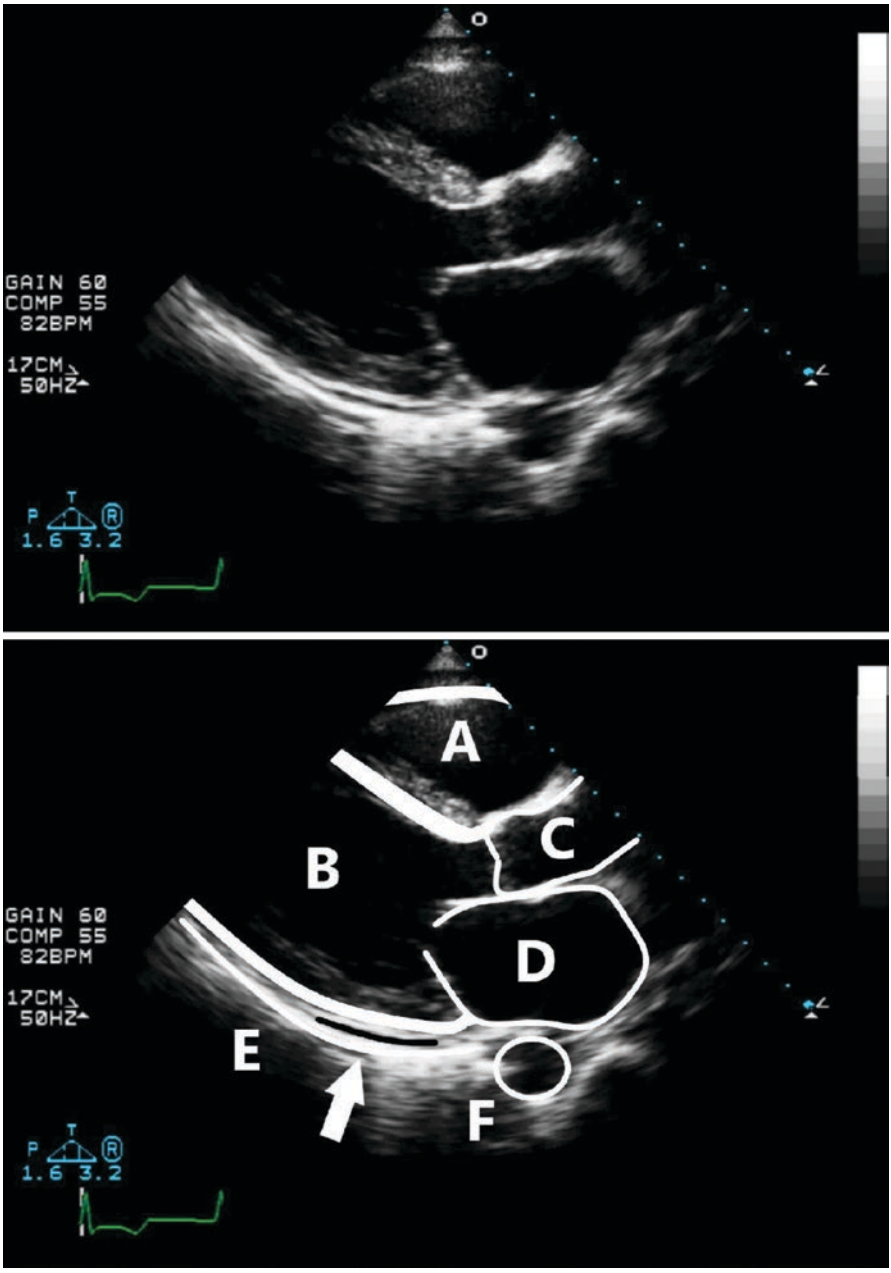

Fig. 11.1 Parasternal long-axis view of the left ventricle. An indication of impaired right ventricular function as right ventricular outflow tract (**A**) is not contracting. Poor left ventricular function and dilated left ventricle (**B**), atrium (**D**), and mitral annulus (between "**B**" and "**D**"). Mitral valve leaflets barely coapting, but structurally normal. Normal aortic valve (between "**B**" and "**C**") and aortic root (**C**). (**E**): left pleural lining. *ARROW*: minimal posterior pericardial effusion. (**F**): normal appearing descending thoracic aorta (Video 11.1)

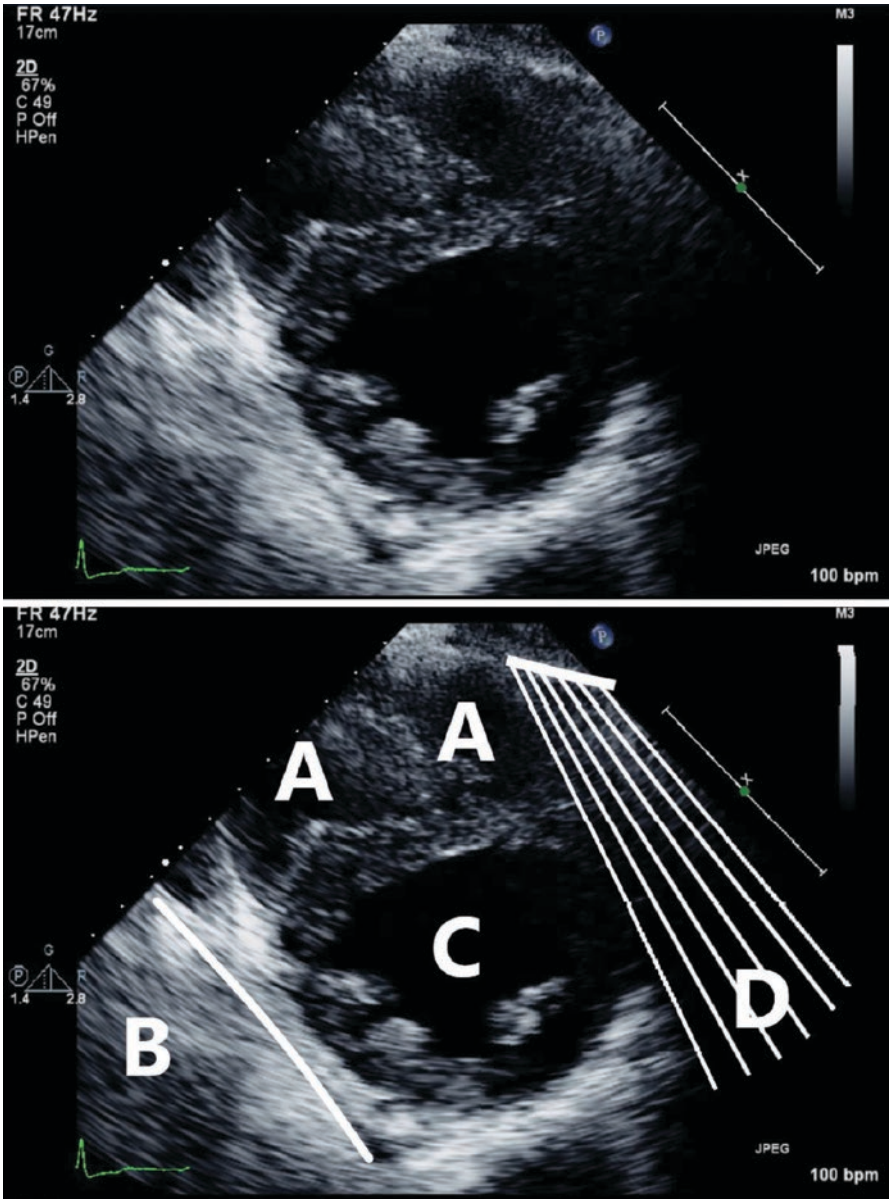

Fig. 11.2 Zoomed view of the parasternal short-axis view of both ventricles. The right ventricle (**A**) appears dilated, note right ventricular moderator band between both the right ventricle and the left pleural lining (**B**). Midportion of the left ventricle (**C**) with papillary muscles seen. Moderately to severely decreased biventricular ventricular function. Flattened interventricular septum (area between **A** and **C**) caused by high right ventricular pressure, and shadow generated by chest wall rib (**D**). Arrythmia noted with ventricular ectopy in EKG translated by irregular heart beat on the 2D video (Video 11.2).

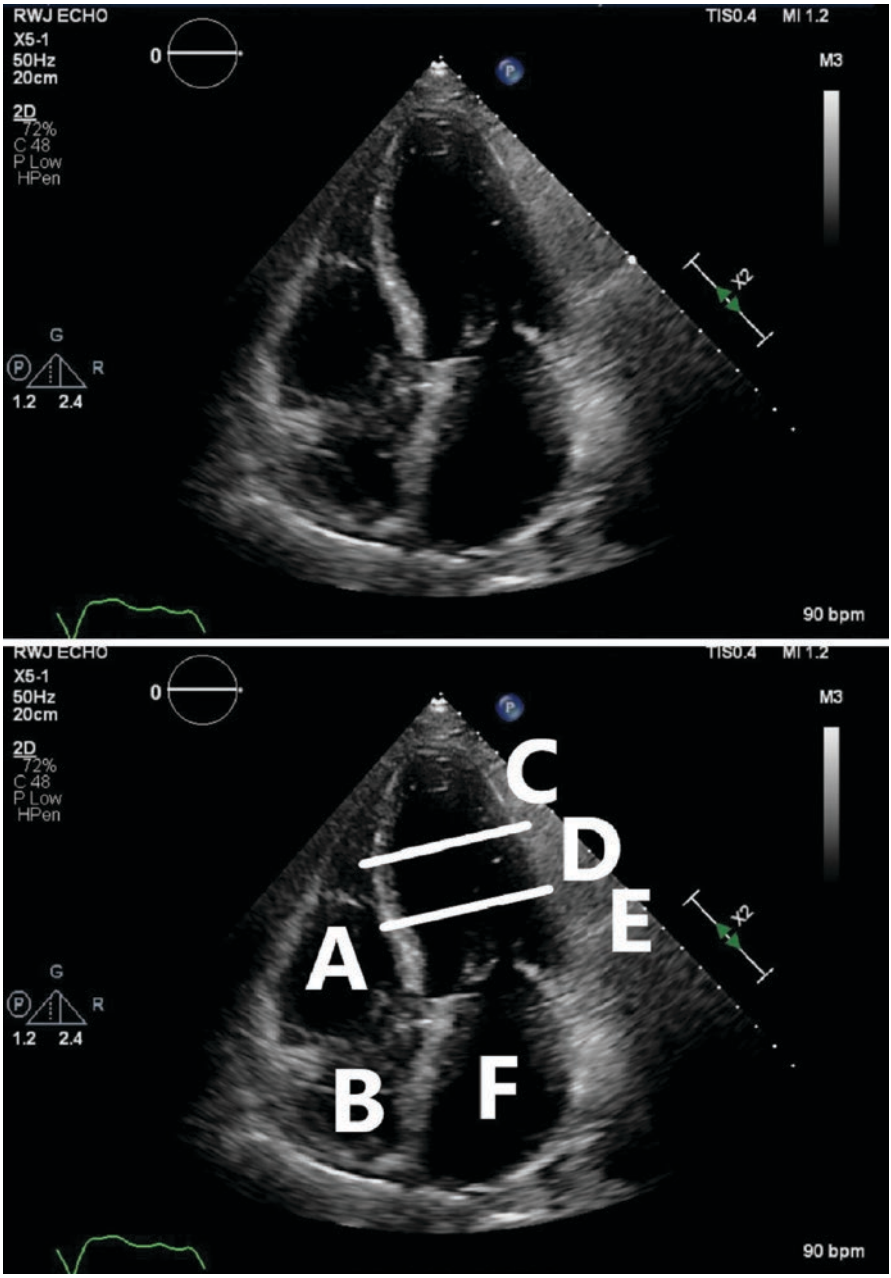

Fig. 11.3 Apical four chamber view. Hyperdynamic contractility of the proximal right ventricle (**A**) free wall, with poor contractility of mid and distal right ventricle free wall. In this view the lateral and septal walls of the left ventricle are seen. The left ventricle is divided in three segments, apical (**C**), mid (**D**), and basal (**E**) segments. Severely decreased left ventricular function, with only the left basal lateral contracting normally in this view. (**B**): right atrium, (**F**): dilated left atrium (Video 11.3)

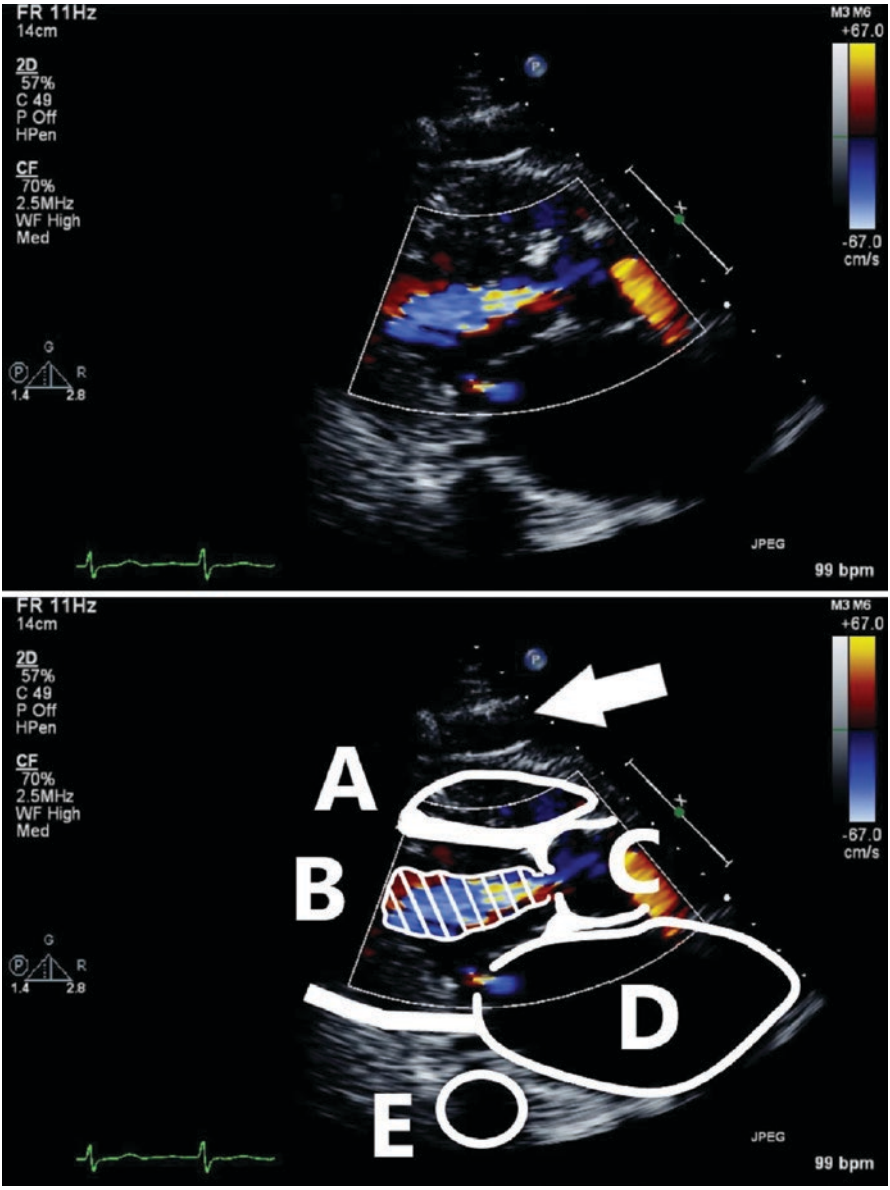

Fig. 11.4 Parasternal long-axis view of the left ventricle. Moderate aortic valve regurgitation (highlighted color Doppler tracing between **B** and **C**), and mild mitral valve regurgitation (small color jet between **B** and **D**). (**A**): right ventricular outflow tract. (**B**): left ventricle. Normal right ventricular outflow and basal left ventricular contractility. (**C**): aortic root. (**D**): dilated left atrium. (**E**): descending thoracic aorta. *Arrow*: dark area in chest wall area that could represent a pericardial effusion, but most likely is related to poor visualization due to an inadequate gain setting. On the video loop the aortic and mitral valve are opening normally (Video 11.4)

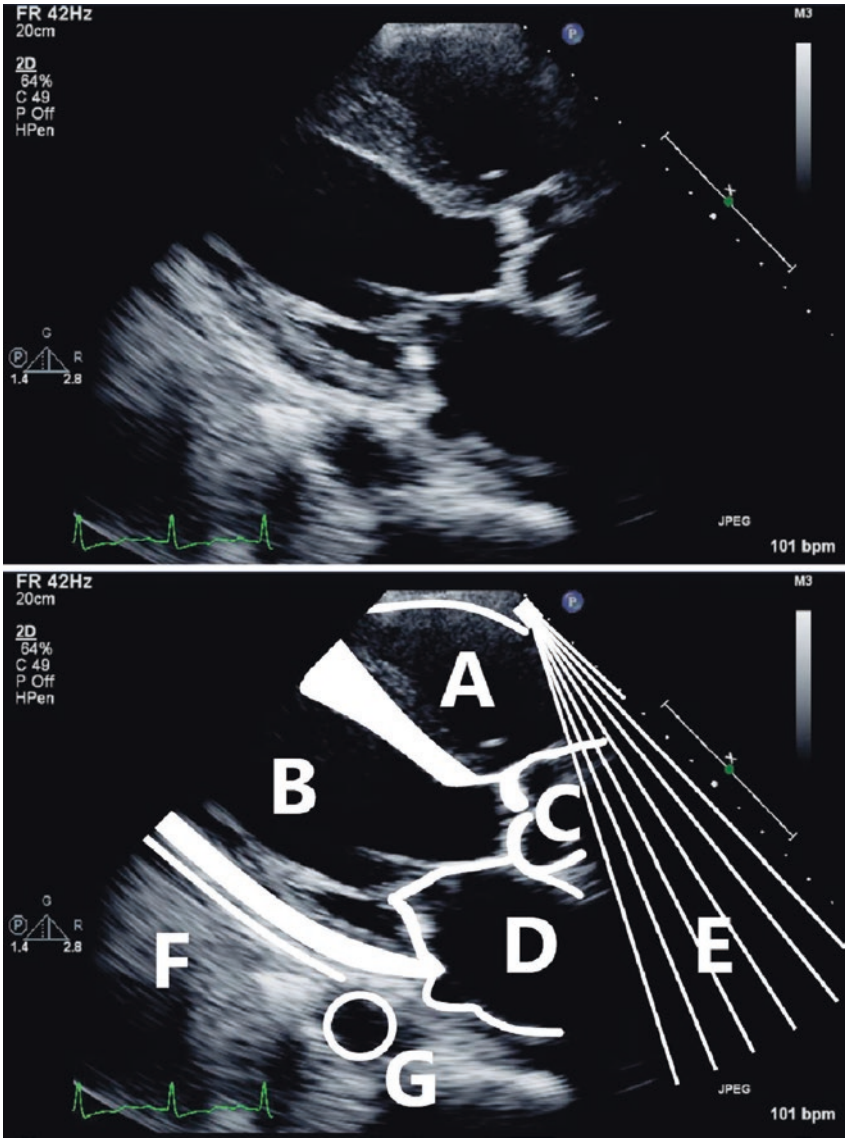

Fig. 11.5 Parasternal long-axis view, the right ventricular outflow tract (**A**) and the left ventricle (**B**) have moderately decreased function. There is shadowing (**E**) caused by a chest wall rib that only allows the aortic valve and sinus of Valsalva to be seen. The aortic valve (**C**) leaflets appear very thickened and are barely moving, which is indicative of aortic stenosis. The mitral valve (between **B** and **D**) appears thickened, especially the posterior leaflet. The mitral opening as seen in the video appears to be sufficient to rule out severe mitral stenosis. The left atrium (**D**) is enlarged. The left pleura (**F**) is noted, and the descending thoracic aorta (**G**) is of normal size. The aorta diameter is largest at the sinus of Valsalva, narrowing at the level of the sinotubular junction and subsequently tapering down distally. Aortic size increases with age and male gender. Roughly maximum diameter of the aorta at the level of the sinuses of Valsalva is ≤37 mm; at the sinotubular junction ≤36 mm; descending thoracic aorta ≤25 mm, and upper abdominal aorta ≤20 mm [1] (Video 11.5)

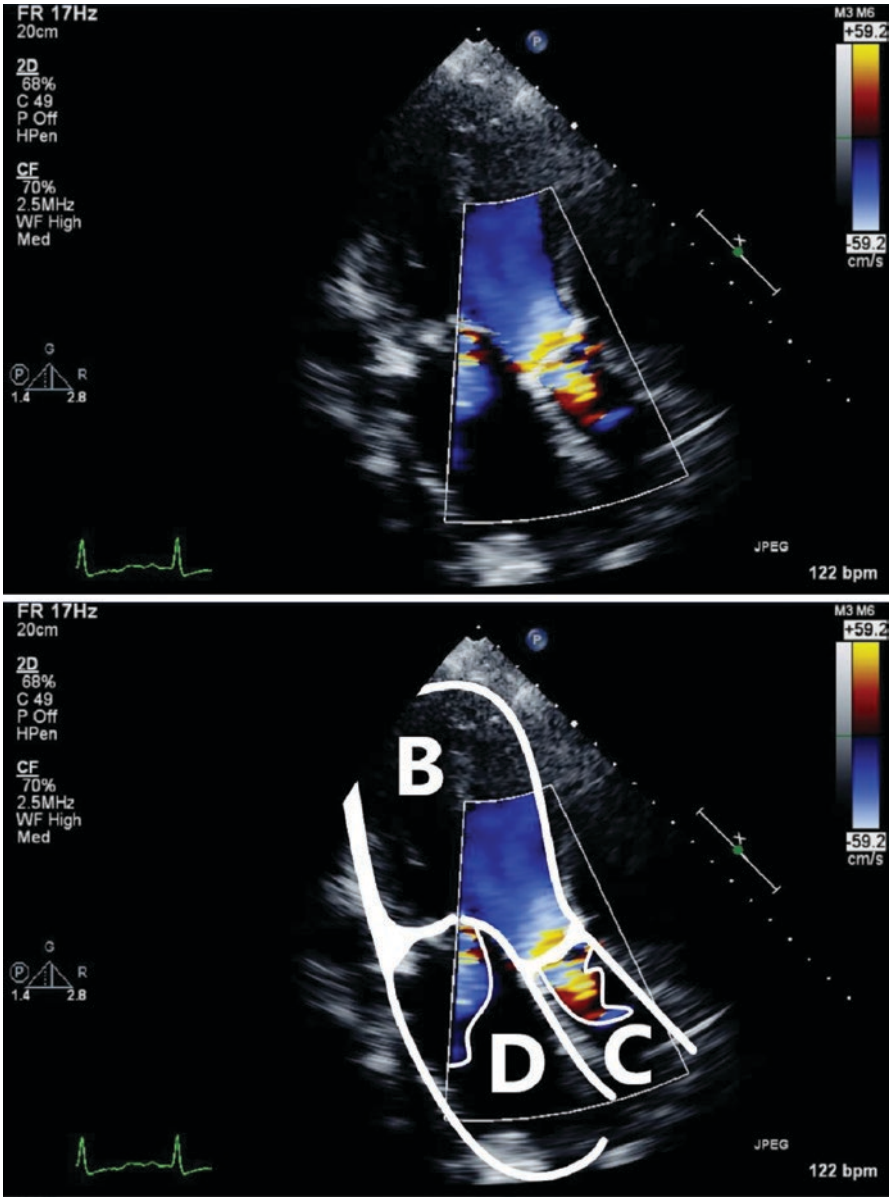

Fig. 11.6 Apical window showing the three-chamber view (left atrium, ventricle, and ventricular outflow tract). The left ventricle (**B**) is poorly visualized, but exhibits decreased function. Very thickened and immobile aortic valve seen (between **B** and **C**). (**C**): ascending aorta seen. Color flow Doppler window covering part of the mitral valve and the whole aortic valve. Note the mitral regurgitation jet propagates deep into the left atrium (**D**), and is therefore at least of moderate severity. Aortic insufficiency jet noted in the LVOT in diastole and mosaic pattern in the aortic root in systole is suggestive of turbulent flow as seen in significant aortic stenosis (Video 11.5)

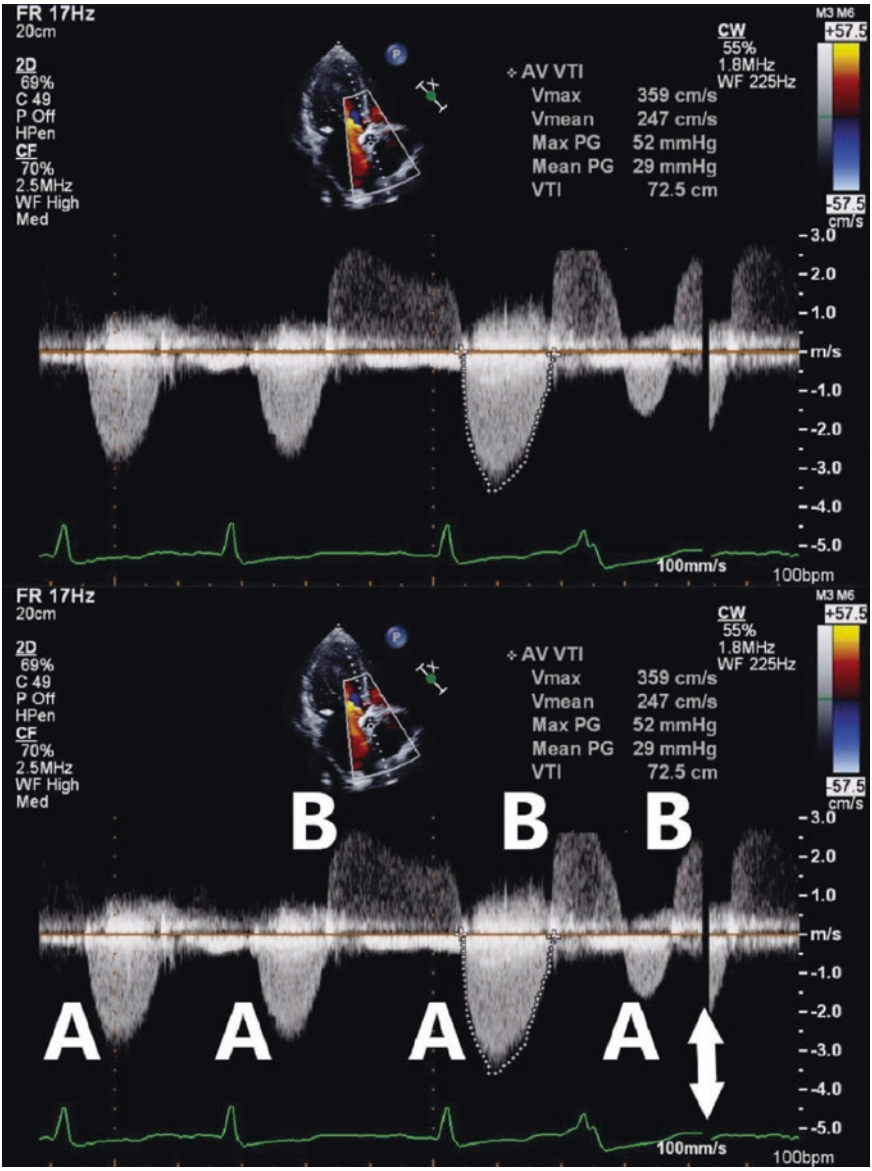

Fig. 11.7 Apical window showing the three-chamber view, with a continuous wave Doppler cursor placed through the aortic valve. Note a small 2D image on top depicting the position of the CWD cursor in the 2D image. The Doppler systolic tracing is recorded below the baseline, representing flow through the path of the whole CWD cursor. The highest velocity (**A**) in systole occurs when blood flows through the narrowest structure, in this case the aortic valve, as this was the narrowest structure seen in the 2D image. Note that the four systolic waves noted (**A**) are of different area and velocity due to arrhythmia (AF and PVC) generating different stroke volumes. In diastole, blood is flowing back through the aortic valve due to aortic insufficiency and a regurgitant velocity tracing (**B**) is recorded above the baseline. The double arrow demonstrates the sweep cursor, as the image is generated from left to right on the screen at 100 mm/sec (Video 11.5)

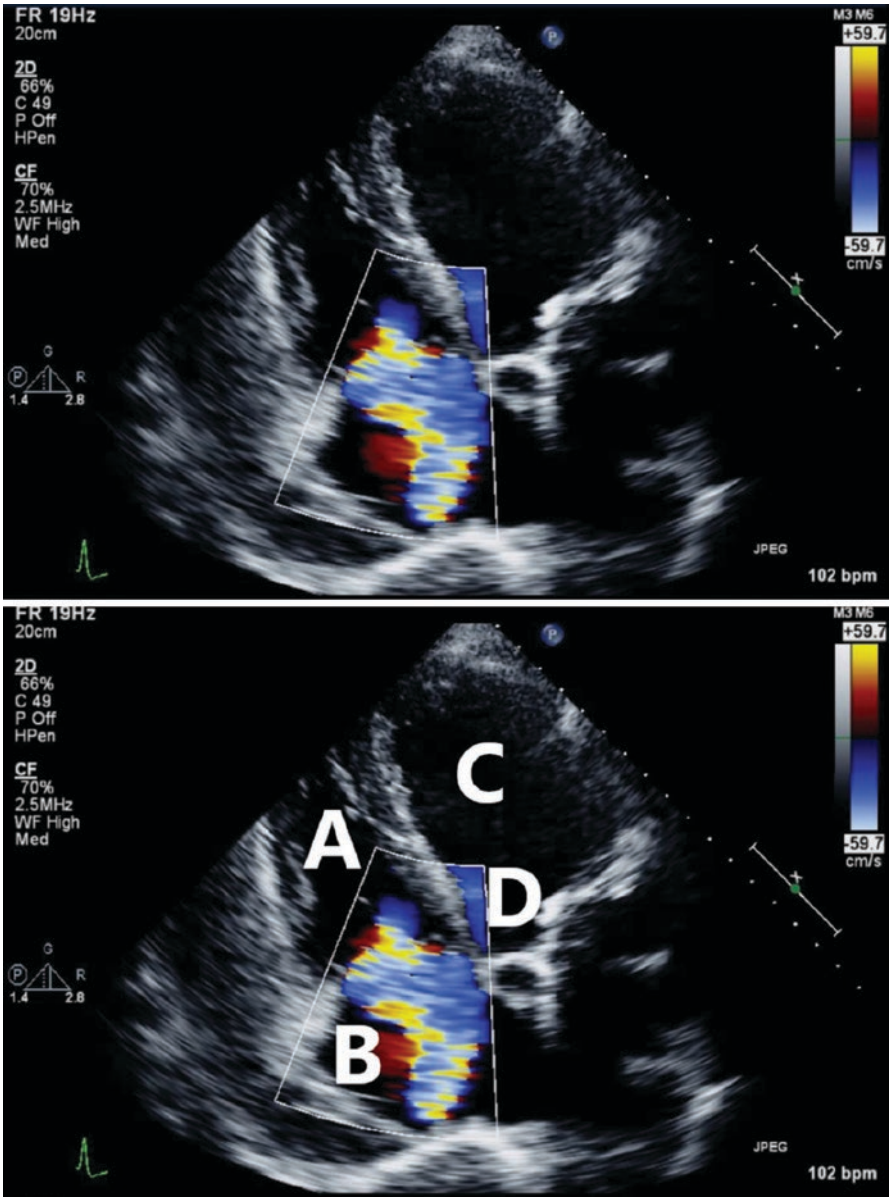

Fig. 11.8 . Apical window showing a four-chamber view, where the mitral valve and left atrium are not seen. Right ventricle (**A**) and atrium (**B**) appear dilated, with normal ventricular contractility. The color flow Doppler cursor is placed over the tricuspid valve, with severe regurgitant flow demonstrated. The left ventricle (**C**) has decreased contractility. (**D**): left ventricular outflow tract (Video 11.6)

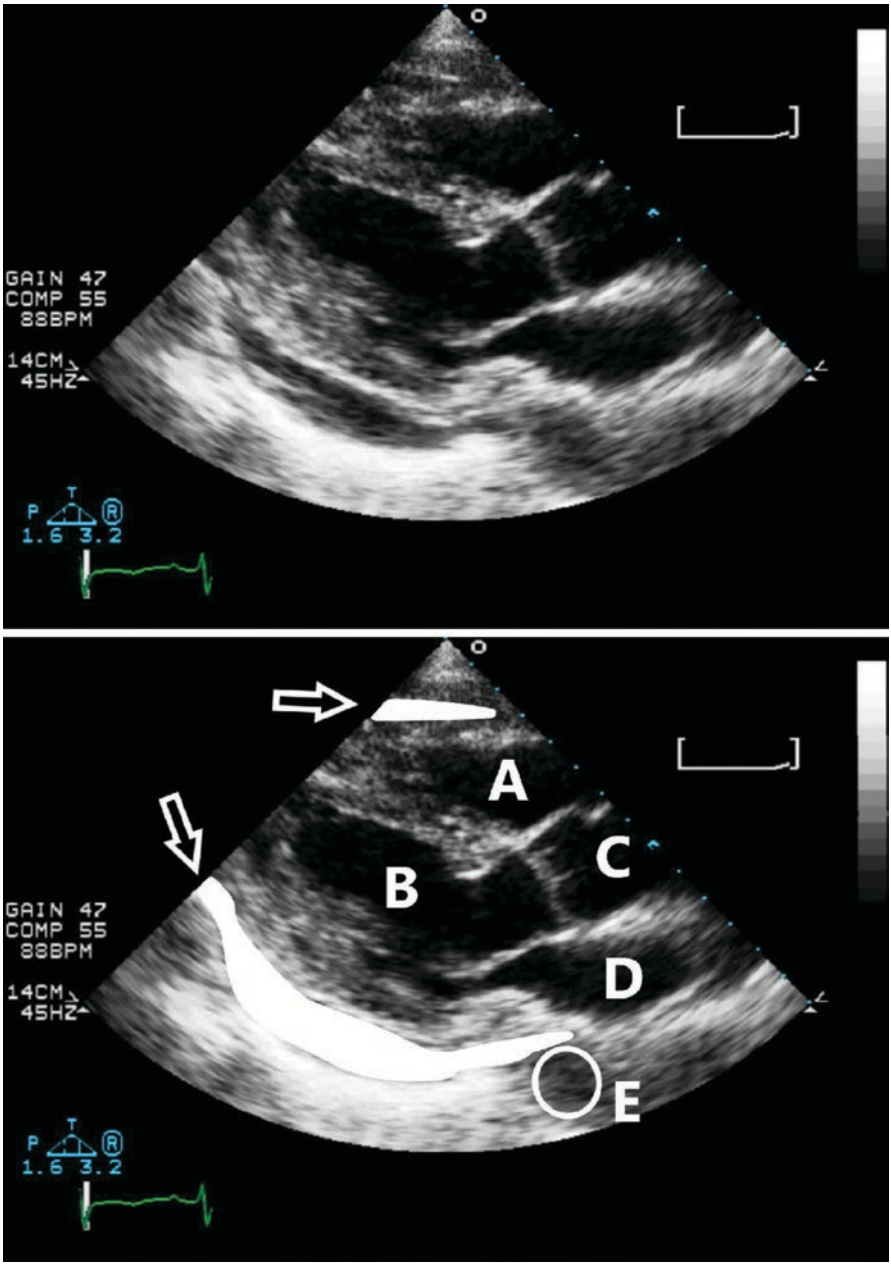

Fig. 11.9 Parasternal long-axis view of the left ventricle. Pericardial effusion noted between the chest wall and right ventricular outflow tract, and under the left ventricle inferior wall (*arrows*). Normal aortic, mitral valves and ventricular function. (**A**): right ventricular outflow tract. (**B**): left ventricle. (**C**): aortic root. Aortic valve between (**B**) and (**C**). (**D**): left atrium. Mitral valve between (**B**) and (**D**). Inferior pericardial collections (bottom arrow) can be differentiated form left pleural effusions because they lie anterior to the descending thoracic aorta (**E**) (Video 11.7)

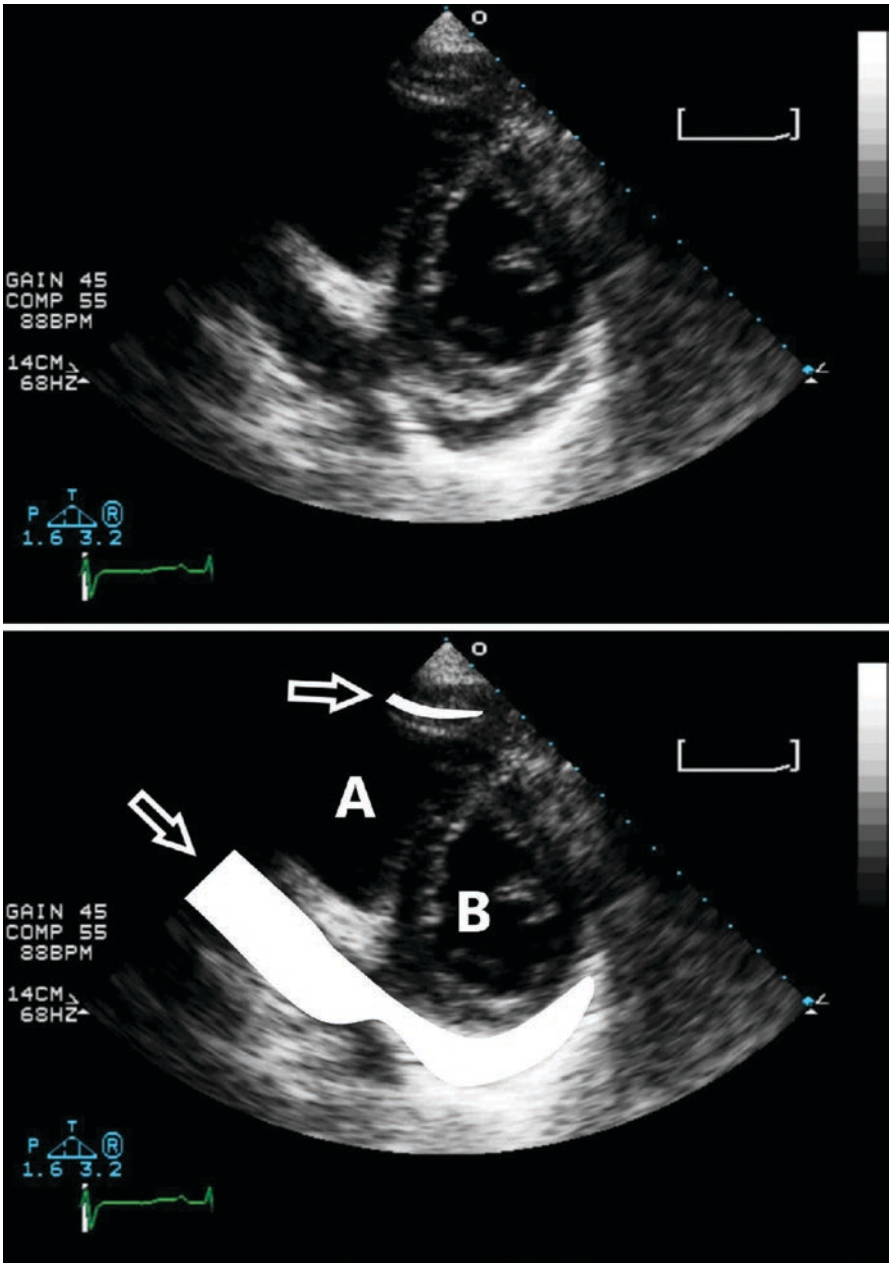

Fig. 11.10 Parasternal short-axis view of the left ventricle. Small pericardial effusion (*superior arrow*) noted between the chest wall and right ventricle (**A**), and large effusion, posterior to the heart (*inferior arrow*). (**B**): mid-segment of the left ventricle (Video 11.7)

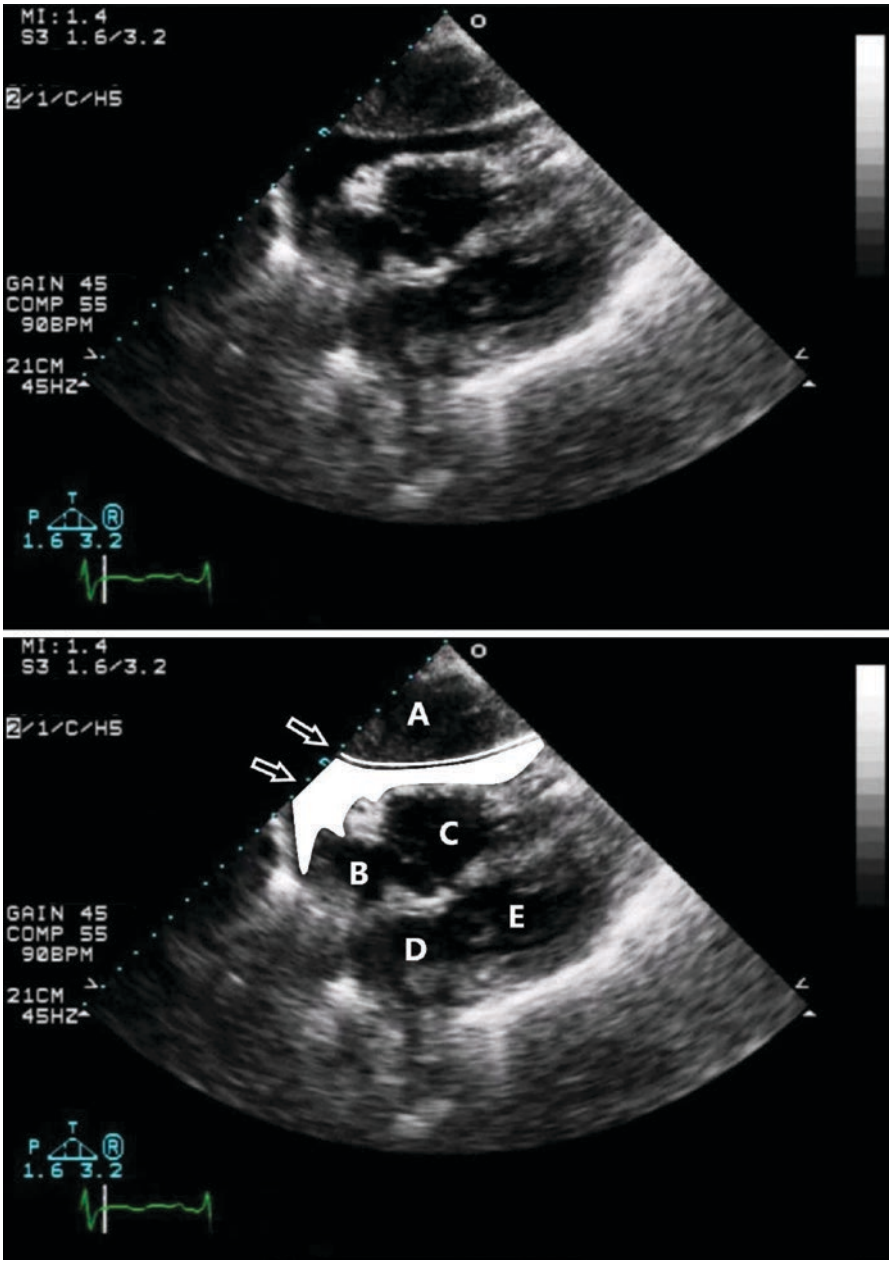

Fig. 11.11 Subxiphoid four chamber view of the heart. The liver (**A**) and diaphragm (*top arrow*) are used as a window to evaluate the heart. Large pericardial effusion (*bottom arrow*) seen in front of the right atrium (**B**) and ventricle (**C**). Right atrial collapse, and partial systolic collapse of the right ventricle (tamponade) noted. (**D**): left atrium. (**E**): left ventricle (Video 11.8)

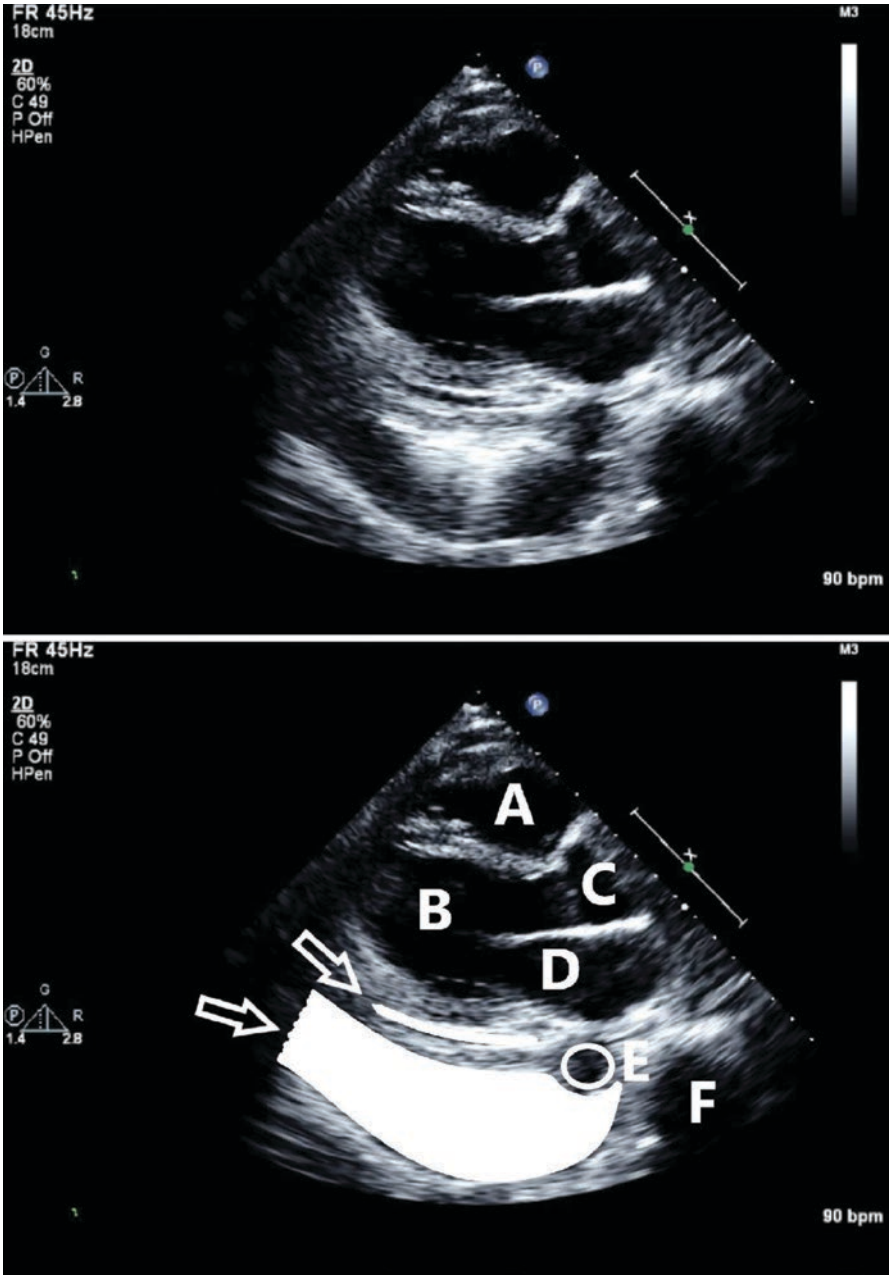

Fig. 11.12 Parasternal long-axis view of the left ventricle. The depth has been set at 18 cm to allow visualization of the left pleural space. Normal function and structure of the right ventricular outflow tract (**A**), left ventricle (**B**), aortic (**C**) and mitral (**D**) valves. Small pericardial effusion (*top arrow*) below the inferior wall of the left ventricle. Large left pleural effusion (*bottom arrow*). Note the pleural effusion extends under the descending thoracic aorta (**E**). (**F**): vertebral body shadow (Video 11.9)

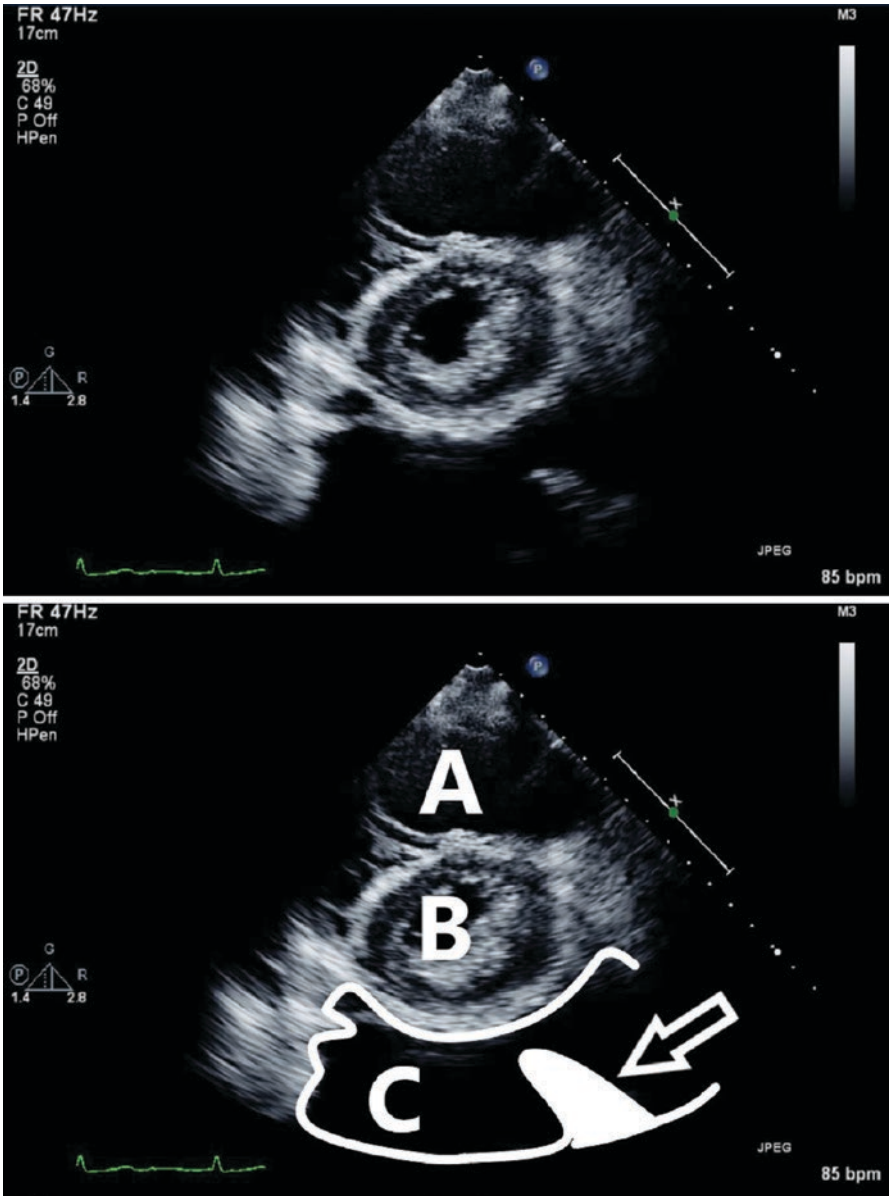

Fig. 11.13 Parasternal short-axis view of the left ventricle. The depth has been set at 17 cm to allow visualization of the left pleural space. Normal function and structure of the right ventricle (**A**) but appears dilated. The left ventricle (**B**) seen in short axis with flattened septum in diastole is indicative of high right ventricular pressure. Large left pleural effusion (**C**). Note within the left pleural effusion an atelectatic segment of the left lung (*arrow*). Collapsed lung and clotted blood have similar density as liver (Video 11.10)

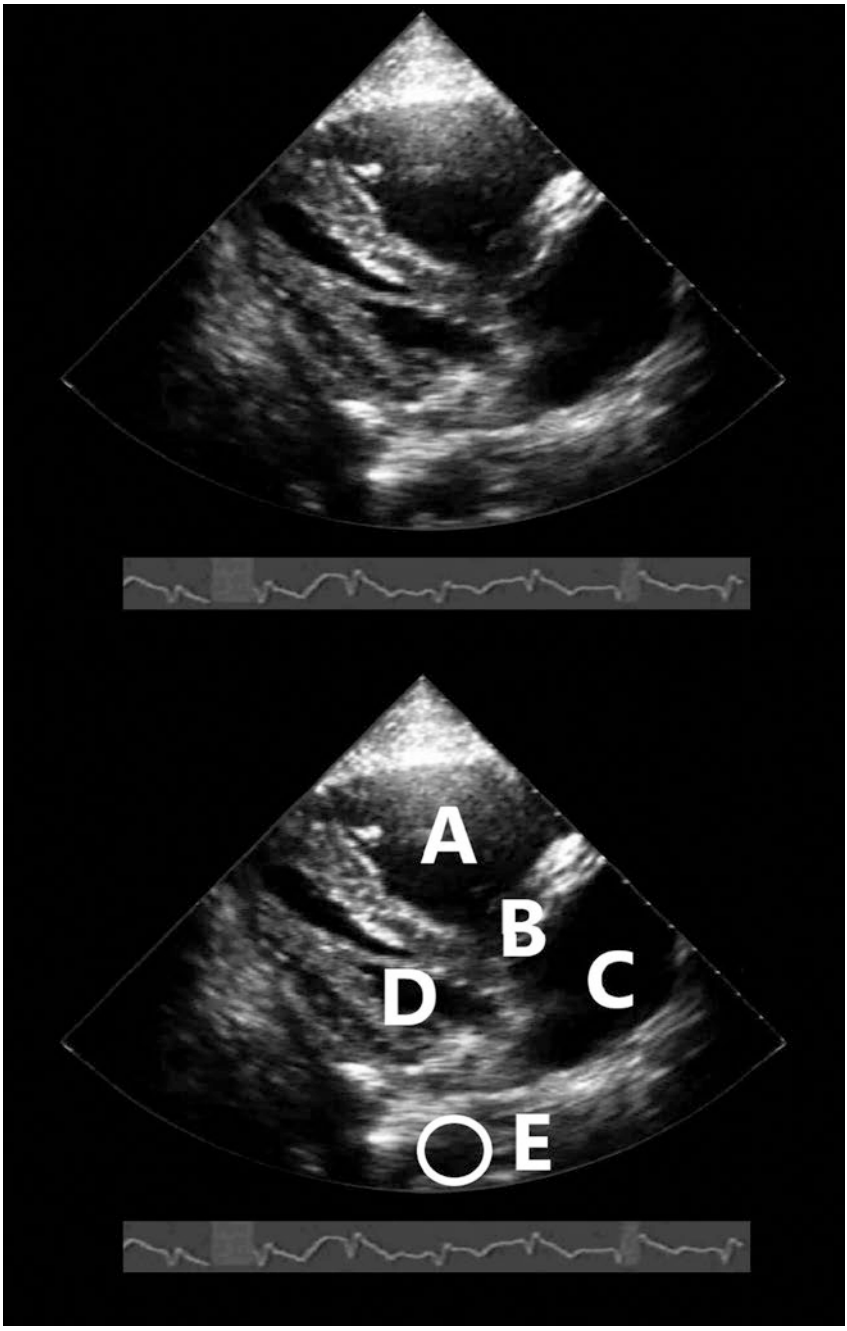

Fig. 11.14 Parasternal long-axis view of the right ventricle. Severely dilated and hypokinetic right ventricle (**A**). (**B**): tricuspid valve. Severely dilated right atrium (**C**). Hyperdynamic, and relatively empty left ventricle (**D**). (**E**): descending thoracic aorta (Video 11.11)

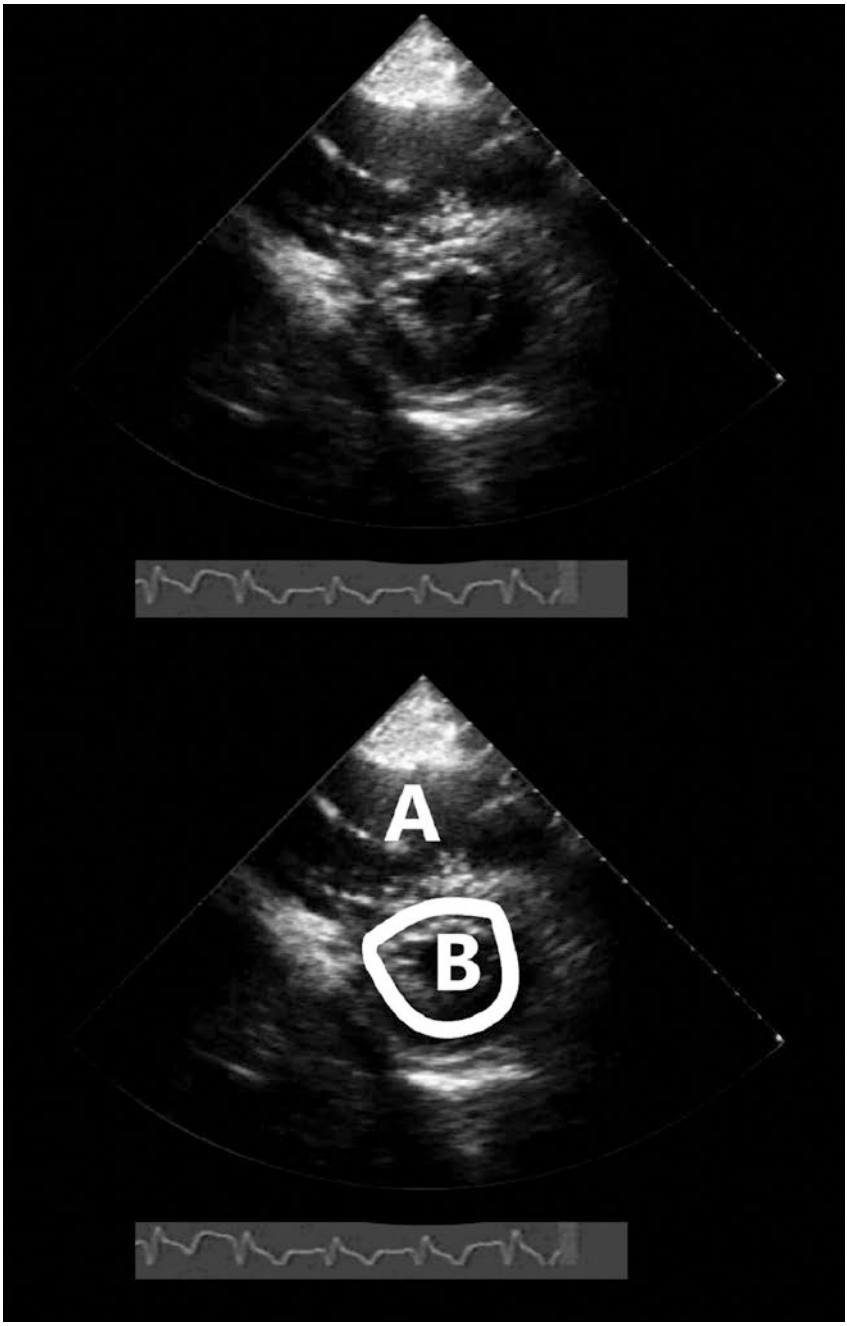

Fig. 11.15 Parasternal short-axis view of the ventricles. Severely dilated and hypokinetic right ventricle (**A**). Hyperdynamic and relatively empty left ventricle (**B**). Flattened interventricular septum ("D"-shaped left ventricle) throughout the cardiac cycle, indicative of severely increased right ventricular pressures (Video 11.11)

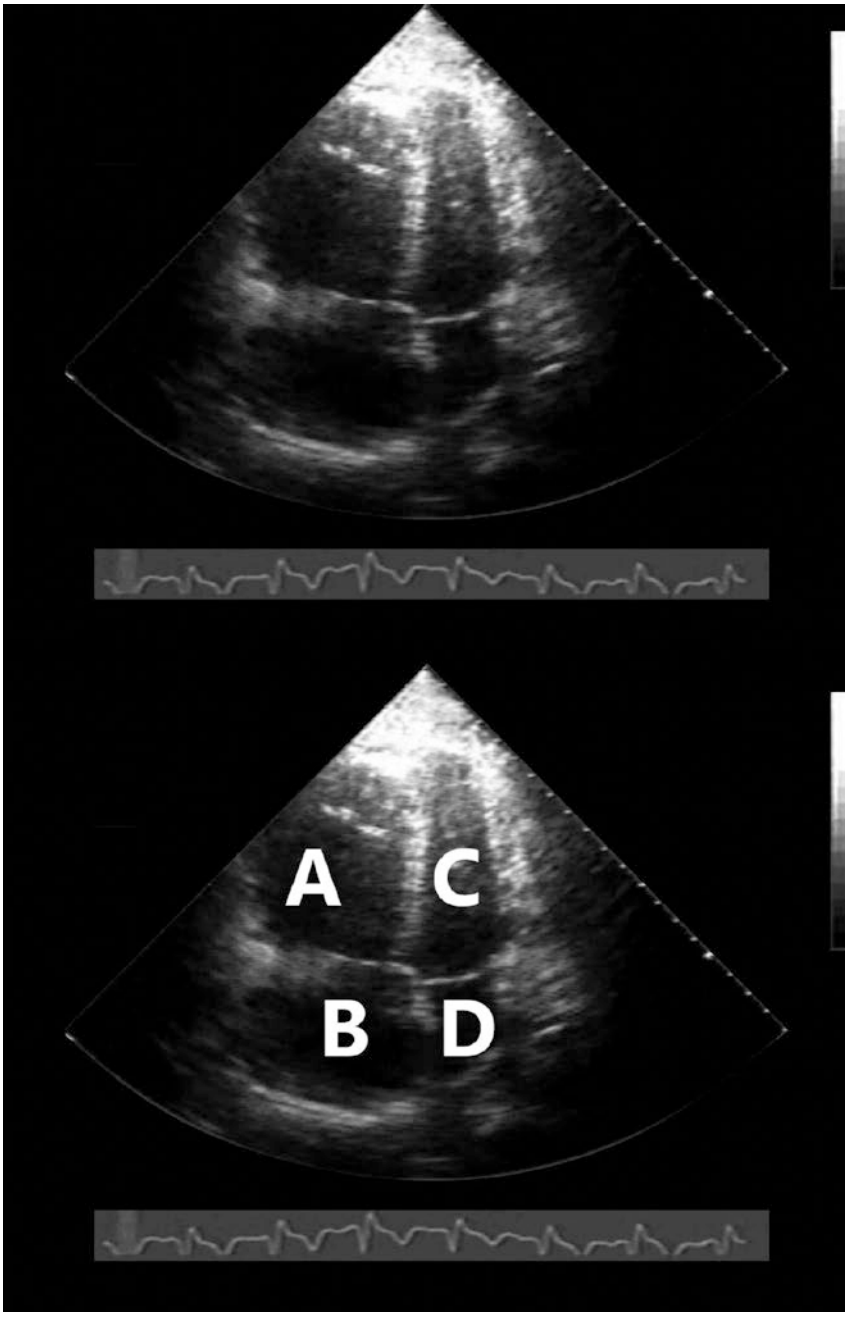

Fig. 11.16 Apical four-chamber view. Severely dilated and hypokinetic right ventricle (**A**). Dilated right atrium (**B**). Hyperdynamic and relatively empty left ventricle (**C**). (**D**): left atrium (Video 11.11)

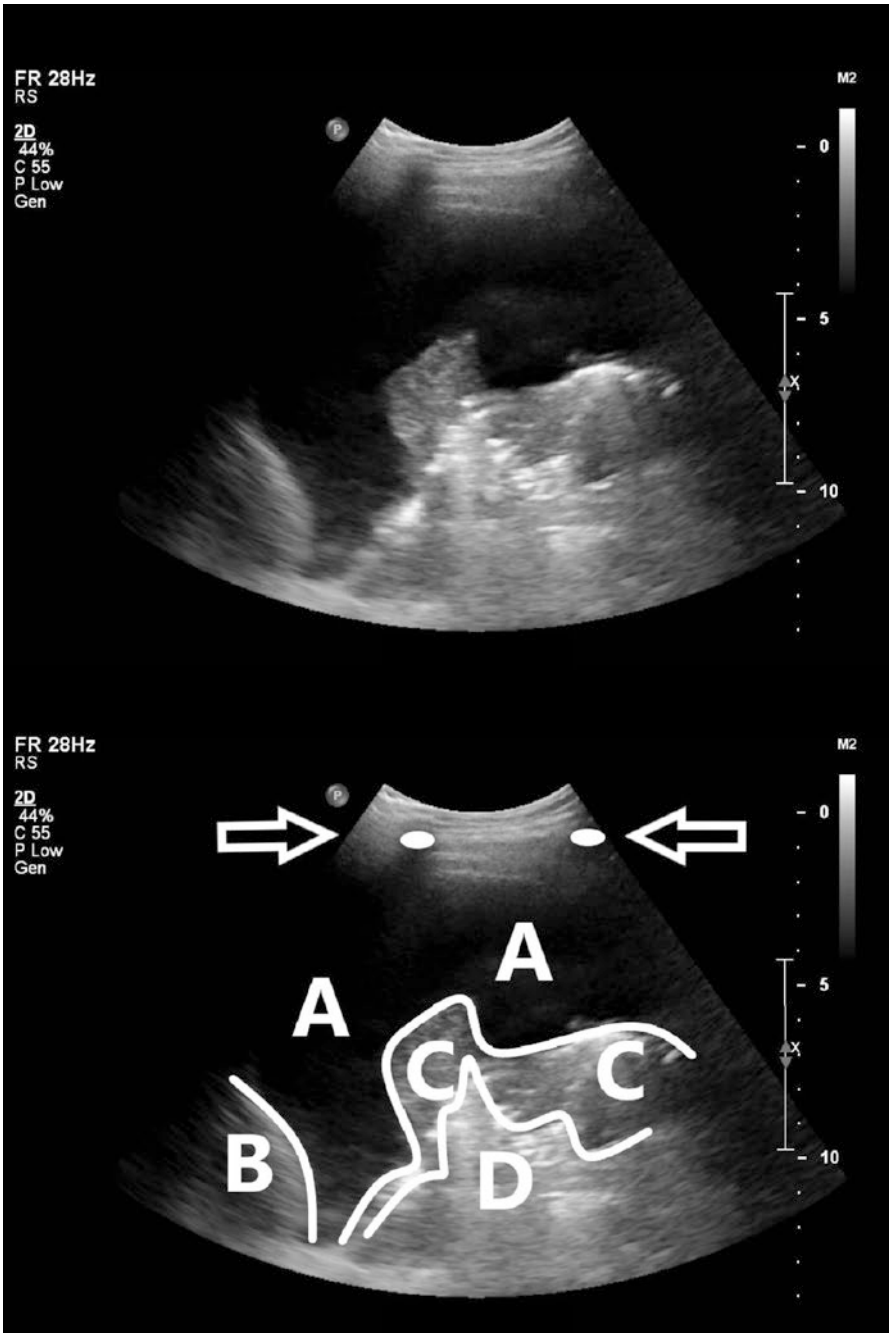

Fig. 11.17 Ribs (*arrows*) generating shadowing. (**A**): large pleural effusion. (**B**): liver and diaphragm. (**C**): lung collapse with minimal air trapping. (**D**): lung collapse but with some air inside. When the lung is totally collapsed (**C**), it has similar ultrasound characteristics as the liver or spleen

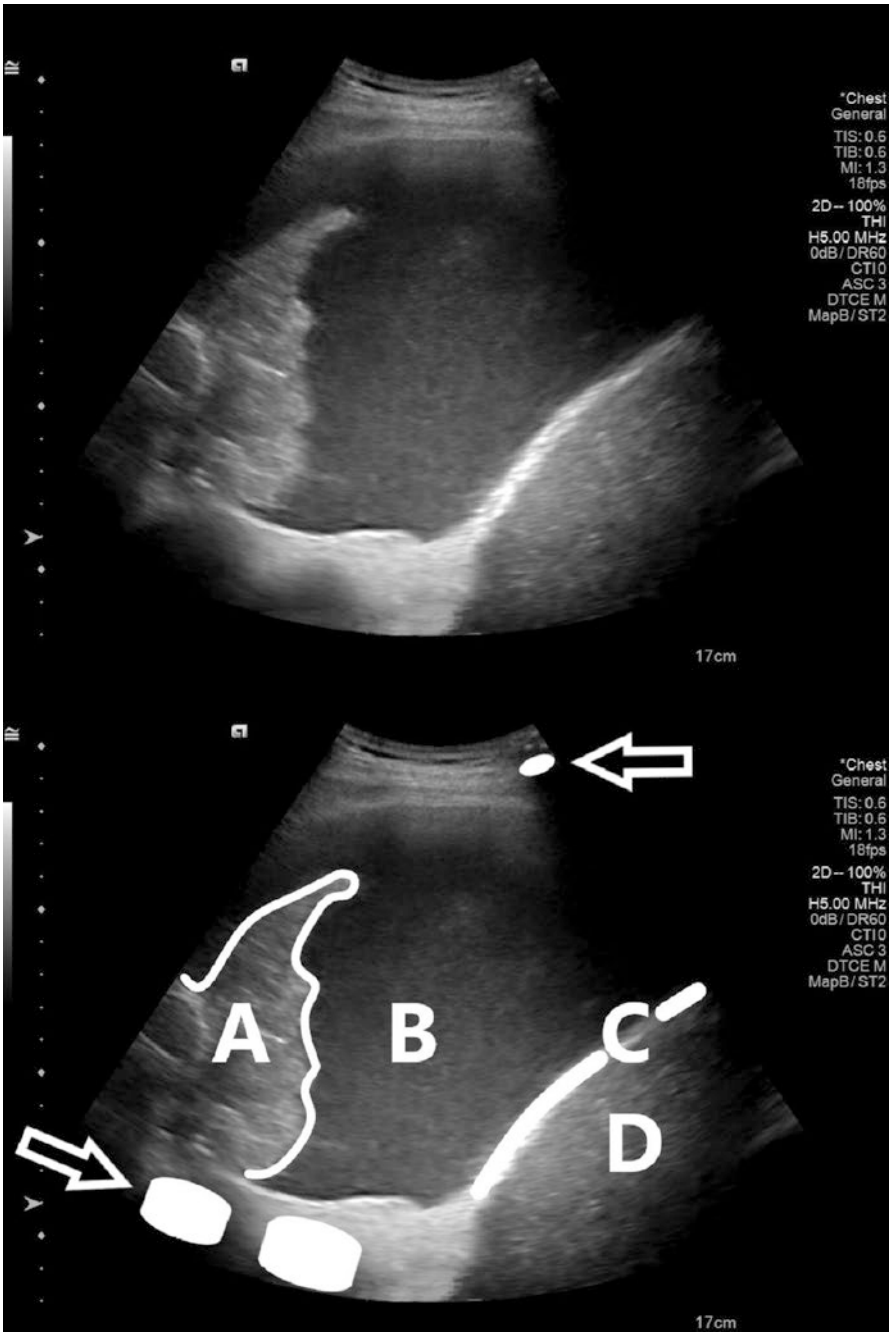

Fig. 11.18 Rib (*arrow*) generating posterior shadowing. (**A**): lung collapse with minimal air trapping. (**B**): large pleural effusion. (**C**): diaphragm. (**D**): liver. *Bottom arrow*: vertebral body shadows

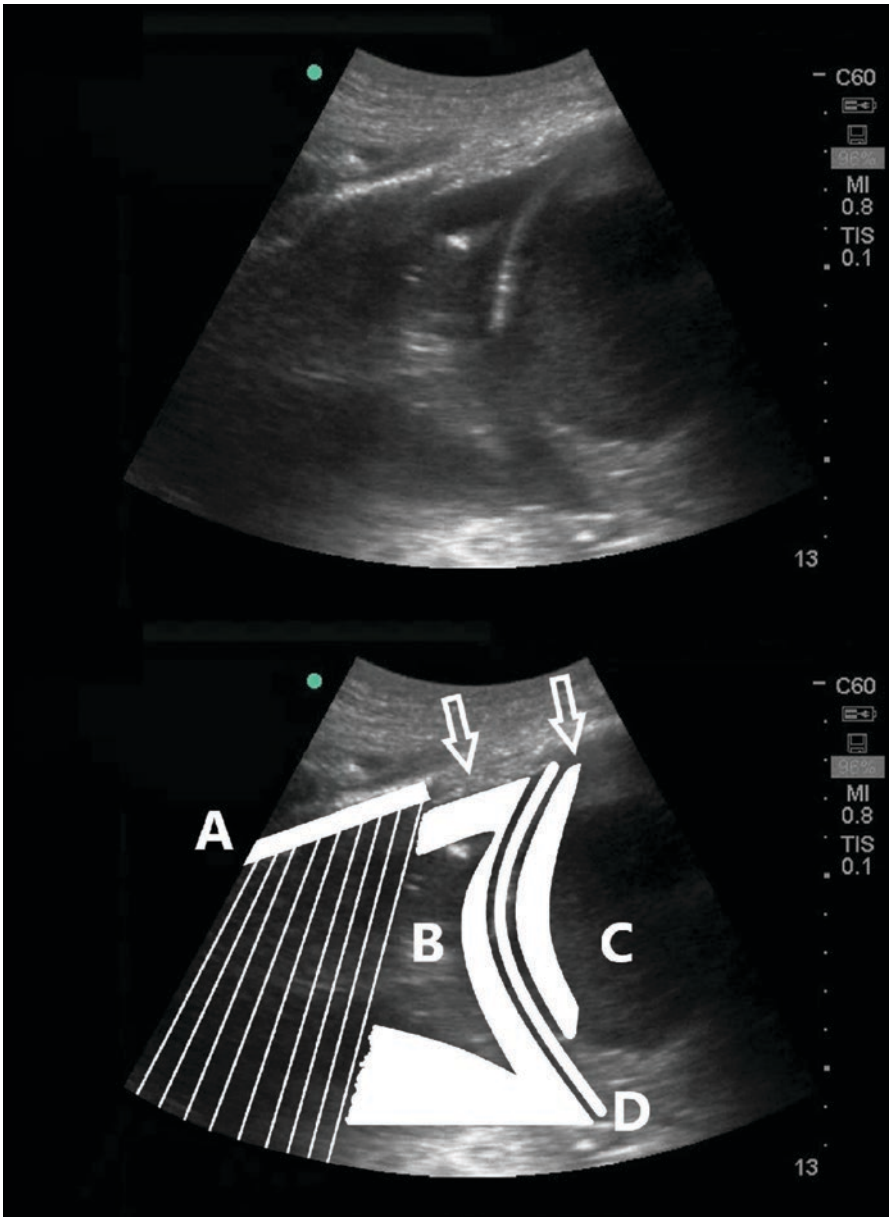

Fig. 11.19 Right pleural effusion and infra-diaphragmatic fluid collection. (**A**): rib with shadowing that obscures the right lung. With the patient taking a deep breath, the atelectatic (**B**) lung appears below the rib shadowing. With patient exhaling the lung retracts under the rib-shadowed area and only the pleural effusion (*top left arrow*) is seen. (**C**): liver. (**D**): diaphragm. *Right-sided arrow*: intra-abdominal fluid seen bellow the diaphragm and above the liver (Video 11.12)

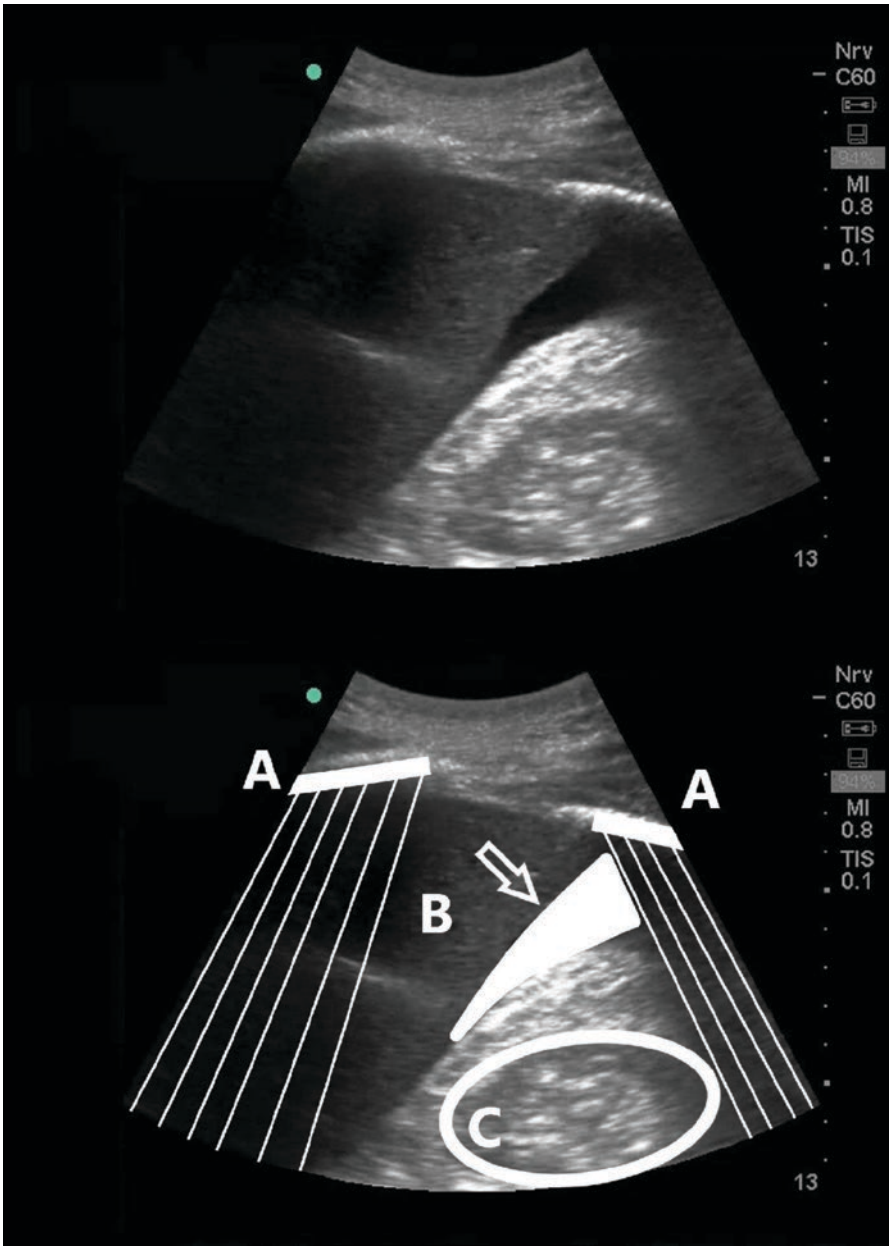

Fig. 11.20 Intra-abdominal fluid collection. (**A**): ribs with shadowing that obscures the cephalad portion of the liver (**B**), intra-abdominal fluid collection and lower pole of the liver. (**C**): right kidney. *Arrow*: fluid between the liver and kidney (Morrison's pouch) (Video 11.12)

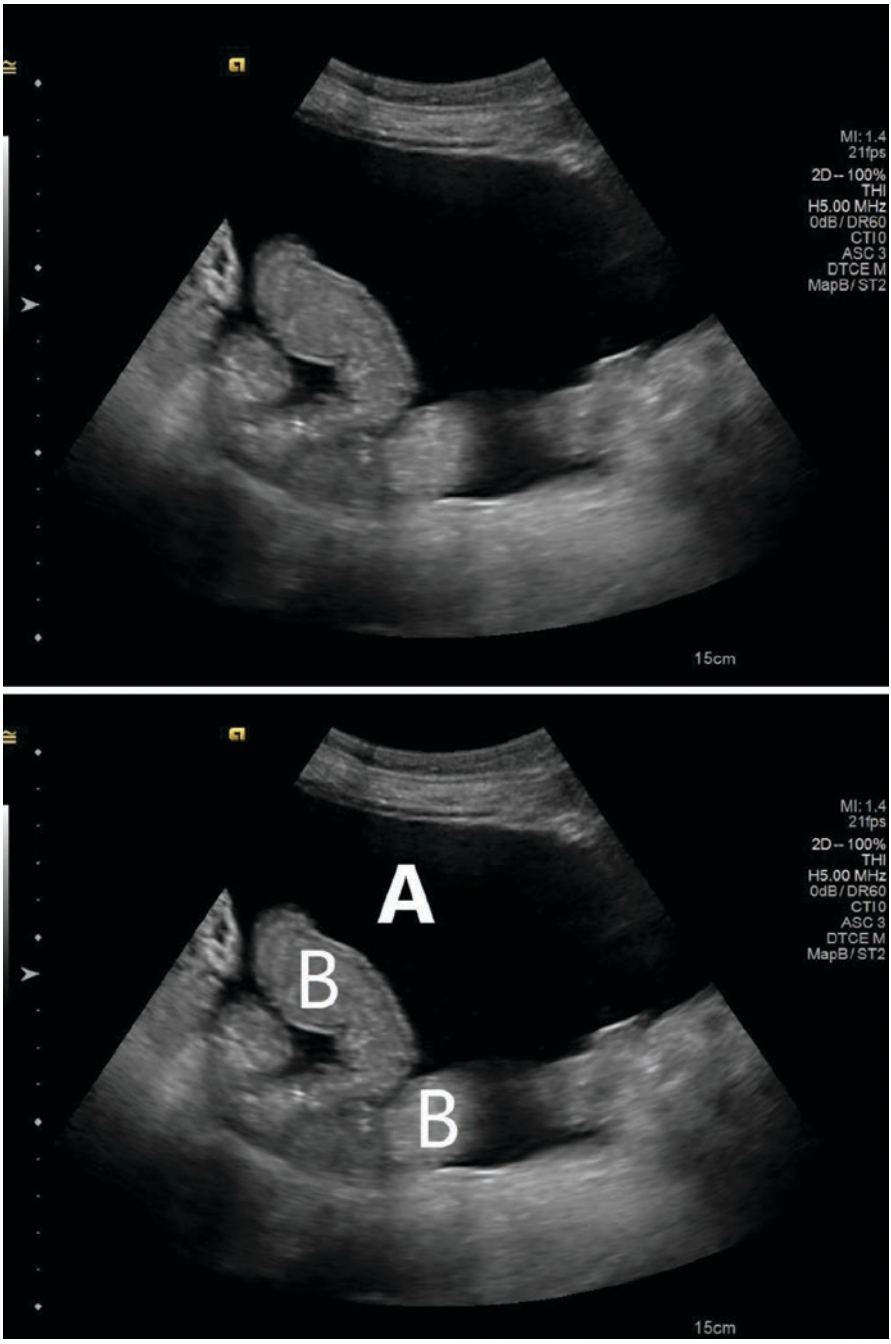

Fig. 11.21 Patient with chronic ascites. Very large intra-abdominal fluid collection (**A**) with "floating" bowel loops (**B**) visible

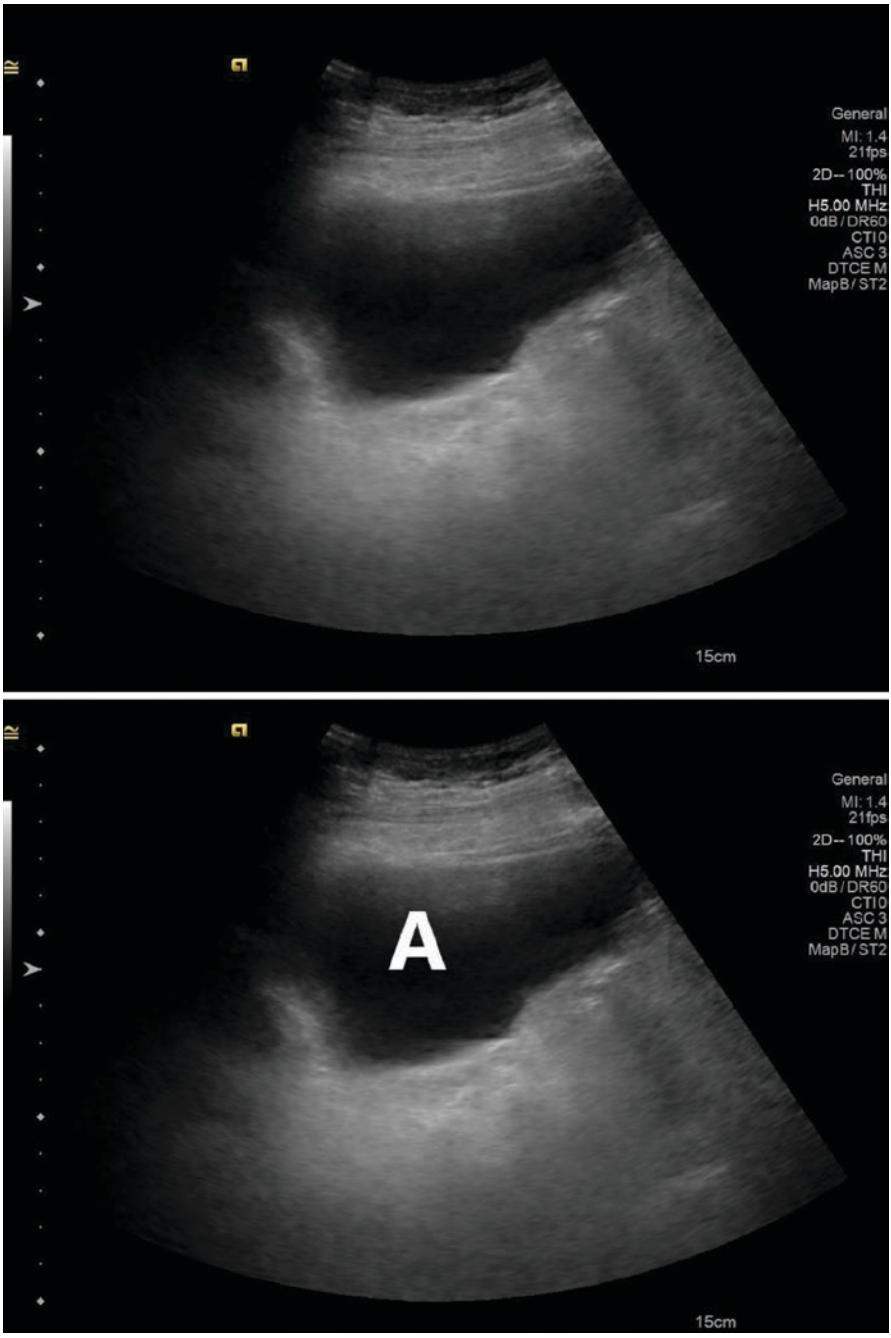

Fig. 11.22 Fluid collection (**A**) with no other structures definable

Internet Resources

American Society of Echocardiography Guidelines

Main Guideline Search Page:

- https://www.asecho.org/guidelines-search/
- Recommendations for Noninvasive Evaluation of Native Valvular Regurgitation.
- https://www.asecho.org/wp-content/uploads/2017/04/2017VavularRegurgitationGuideline.pdf
- Recommendations on the Echocardiographic Assessment of Aortic Valve Stenosis.
- https://www.asecho.org/wp-content/uploads/2017/04/2017ValveStenosisGuideline.pdf
- Echocardiographic Assessment of Valve Stenosis: EAE/ASE Recommendations for Clinical Practice.
- https://www.asecho.org/wp-content/uploads/2014/05/2009_Echo-Assessment-of-Valve-Stenosis.pdf

Educational videos:

- YouTube: search for generic and specific search terms such as: TTE, echocardiography, FAST, ventricular function by echocardiography, aortic dissection ultrasound, lung ultrasound, pneumothorax on ultrasound, etc.
- Resources and Tutorials on Emergency Medicine Ultrasound.
- http://www.emergencyultrasoundteaching.com/narrated_lectures.html

Bibliography

1. Multimodal Imaging of Diseases of the Thoracic Aorta in Adults. https://www.asecho.org/wp-content/uploads/2015/01/2015_Thoracic-Aorta.pdf

The Fundamentals of Transesophageal Echocardiography

12

Brett A. Waldman, Priscilla J. Peters, Julie Wise, Samir Patel, and Sandeep Krishnan

Nothing in all the world is more dangerous than sincere ignorance and conscientious stupidity.

—Martin Luther King, Jr.

Key Points

Intraoperative TEE	Transducer knobology
Advantages and pitfalls of TEE	TEE machine knobology
Complications and contraindications to TEE	Tomographic views
TEE probe insertion	

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Introduction

Transesophageal echocardiography plays a crucial role in patients undergoing cardiac surgery and transcatheter procedures. Intraoperative TEE can be used for diagnostic purposes as well as for hemodynamic monitoring. TEE can be used to identify global or regional wall motion abnormalities of the myocardium, valvular dysfunction, and to assess and calculate hemodynamic parameters including cardiac output, preload and left ventricular filling, and ejection fraction (EF). During cardiac surgery, TEE is often used to guide the surgical intervention. For example, during mitral valve repair, TEE is used for initial assessment of the valvular dysfunction and for close inspection of the valve repair after cardiopulmonary bypass. TEE can also be used to diagnose regional or global wall motion abnormalities upon separation from bypass after coronary artery bypass grafting (CABG) and other cardiac operations. Routine use of TEE during CABG has been shown to have a significant impact on management and outcomes. TEE can even be used during noncardiac cases or in the ICU to guide management of critically ill patients. In a study by Hofer et al., which evaluated patients after vascular, visceral, and chest surgery, TEE was reported to be frequently involved with changes in drug (47%) and fluid therapy (24%). For these reasons TEE has become an integral part of the vast majority of open cardiac surgical procedures, as well some noncardiac procedures. Table 12.1 describes the indications for intraoperative TEE.

Advantages and Pitfalls of TEE

TEE offers superior image clarity and resolution to transthoracic echocardiogram. This improvement in image quality is the result of a shorter distance between the transducer and the heart (in the esophagus versus on the chest), higher-frequency transducers, and elimination of intruding bone and tissue in the chest. For this reason, TEE has become the diagnostic test of choice in the management of many

Table 12.1 Indications for intraoperative transesophageal echocardiography (TEE)

Operation/procedure	Examples and purpose
All open-heart operations	Valvular replacement or repair Aortic dissection repair Congenital heart disease HOCM
CABG	Assess LV volume Ventricular function
Transcatheter procedures	TAVR Mitral clip placement, Septal ablation for HOCM, ASD/PFO closure, Placement of LAA occlusion devices
Intracardiac device implants	Ventricular assist device Cannula placement

HOCM hypertrophic obstructive cardiomyopathy, *CABG* coronary artery bypass graft, *TAVR* transcatheter aortic valve replacement, *ASD* atrial septal defect, *PFO* patent foramen ovale, *LAA* left atrial appendage

cardiovascular conditions including valvular disease, congenital heart disease, and cardiac sources of emboli. TEE is used almost universally during cardiac surgery, as TEE can obtain real-time imaging of cardiac structures during the entire procedure without interrupting the surgery or contaminating the sterile field.

It should be noted that a TEE does not replace a transthoracic echo exam, and, in some instances, a transthoracic echocardiogram may provide better image quality than TEE, particularly when evaluating anterior cardiac structures. Those structures would fall into the far field of the echo beam on TEE exam. In addition, a transthoracic study offers more acoustic windows, which is especially important when measuring Doppler velocities. If the Doppler beam is not aligned parallel to the flow of blood, this may result in an incorrect calculation of velocities.

Complications and Contraindications to TEE

TEE has a very low incidence of complications with a reported mortality of less than 1 in 10,000. Injury to lips (13%), hoarseness (12%), and dysphagia (1.8%) are among the most common complications. Major bleeding is rare and occurs in less than 0.01% of patients. Esophageal perforation has been reported in association with TEE probe placement or manipulation. Such perforations may remain undetected for a period of time after the procedure, leading to mediastinitis or other deleterious effects. These potential risks should be taken into consideration when deciding if the clinical information to be gained from TEE outweighs them. Extra caution is advised in patients who have had any recent type of bariatric or esophageal surgery. Patients who have had bariatric surgery may be best served by not having transgastric images. Regarding absolute contraindications to TEE.

Five absolute contraindications to transesophageal echocardiography:

- Esophageal tumor.
- Esophageal perforation.
- Esophageal stricture.
- Active upper GI bleed.
- Perforated viscus.

Probe Insertion

The patient is induced and intubated. The gastric contents are then suctioned to improve image quality since the air insufflated during ventilation is a poor conductor of US. The TEE probe is inserted into the patient's pharynx and a bite block is inserted into the mouth to protect the TEE probe. The probe is inserted through the bite block using ultrasound gel into the patient's oropharynx with the imaging surface of the transducer facing the patient's tongue and the probe tip slightly anteflexed. The probe is then guided into the esophagus using a blind technique of lifting the patient's lower jaw while gently advancing. If difficulty with passing the probe into the esophagus is encountered, direct visualization with laryngoscopy or glidescope can be attempted.

A bimanual anterior jaw thrust or flexing the head by an assistant may also be tried. Caution is necessary when advancing the probe to avoid displacement of the endotracheal tube, as well as to avoid harm to the posterior pharynx or esophagus. The distance from the incisors to the mid-esophagus is typically about 30 cm.

Clinical Pearls

- Prior to probe insertion, the machine is prepared for operation (Fig. 12.1). Start by attaching the echo probe to the machine and entering the patient's information into the system.
- Next, make sure the probe is in the unlocked position for insertion.
- Have a bite block on hand, which can be placed over the TEE probe or directly in the patient's mouth prior to insertion.
- Insert OG tube and suction air introduced to the stomach during hand ventilation.
- Generously lubricate the tip and distal length of the probe.
- The examiner may place the controls and proximal portion of the probe around his or her neck (Fig. 12.2) or hang the probe from a hook or stylet near to the patient.
- The tip should then be inserted into the mouth and slight anteflexion is often helpful to navigate the curve of the tongue (Fig. 12.2).
- The lower jaw should then be lifted gently to align the oropharynx with the esophagus and allow for ease of probe placement.
- Force should not be used during placement. If resistance is met, the operator should try a different approach, such as placement guided by a laryngoscope (direct or video).

Transducer Knobology

Modern transesophageal probes consist of a matrix of piezoelectric crystals mounted on the tip of the probe. The matrix can produce a two- or three-dimensional image depending on which crystals are activated at any given time. There are five ways of moving and manipulating the probe either electronically or mechanically. Refer to Table 12.2 and Images 3–8 for complete terminology of transducer motions (Figs. 12.3, 12.4, 12.5, 12.6, and 12.7).

Fig. 12.1 Pictures of Left: Plug in the power cord Middle: Attach the echo probe to the machine Right: Press the power button to turn the machine on

Fig. 12.2 Pictures of Left. Proper method of holding of the probe to free the left hand for opening the mouth and lifting the jaw Right: Blind intubation of the esophagus with the TEE probe

Fig. 12.3 Picture of the probe tip in neutral position

Table 12.2 Transducer motions

Motion	Definition
1. Repositioning	Advancing the TEE probe down or withdrawing the probe up in the esophagus
2. Rotation	Rotating the transducer imaging plane from 0° to 180°
3. Turning	Rotation of the entire probe toward the patient right or left
4. Angulation ("big control wheel")	Flexing the tip of the probe which has the imaging transducer, so the image plane is directed anteriorly (anteflex) or posteriorly (retroflex)
5. Tilt ("small control wheel")	Flexing the tip of the probe to create lateral motion of the transducer tip to right or left of the patient

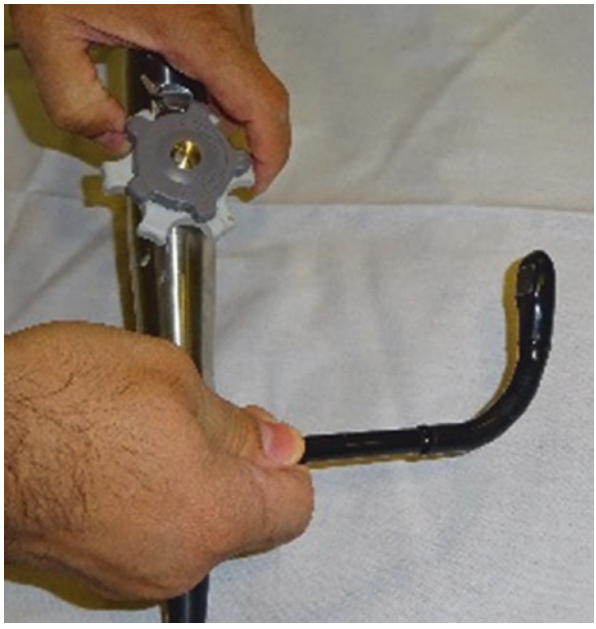


Fig. 12.4 Picture of probe tip anteflexion by turning the big wheel clockwise

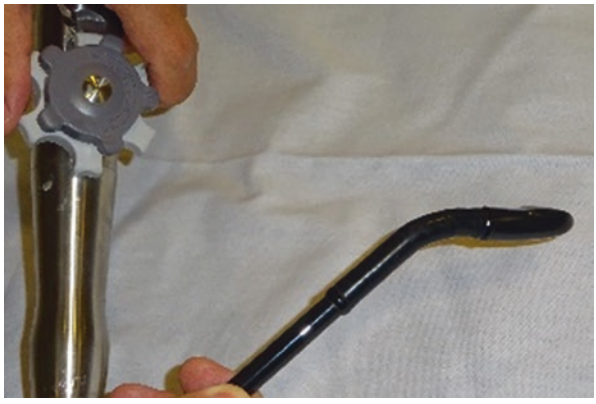


Fig. 12.5 Picture of probe tip retroflexion by turning the big control wheel counterclockwise

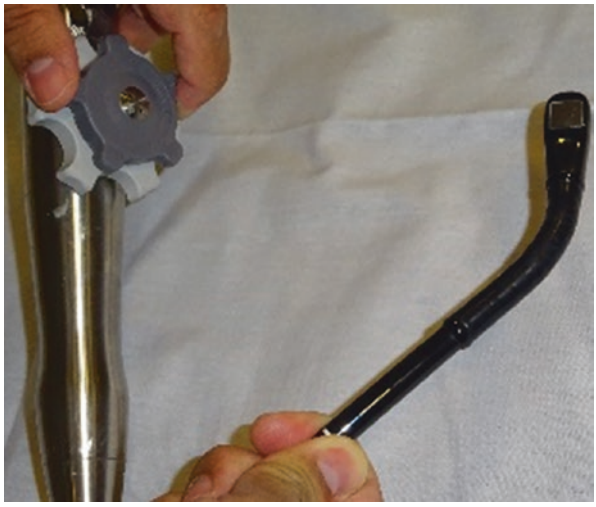


Fig. 12.6 Picture of tilting of the probe tip to the left of the patient by turning the small control wheel clockwise

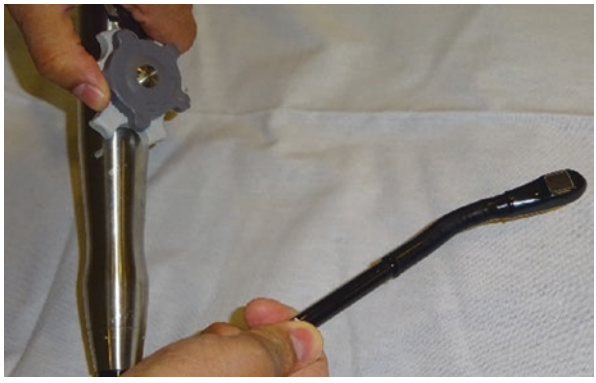


Fig. 12.7 Picture of tilting the probe tip to the right of the patient by turning the small control wheel counterclockwise

Omniplane: By electronically controlling the active crystals, the two-dimensional imaging place can be rotated from 0 to 180 degrees around the long axis of the probe using rotation buttons. It enables us to view an object at various angles by rotating around that object. On the echo probe handle there are two small buttons: the top button (closest to the end of the handle) will decrease the angle; the bottom button (closest to the patient) will increase the angle. Alternately, there is a knob on the machine that will increase or decrease the angle. The probe is capable of rotating from 0 to 180 degrees Refer to Fig. 12.8.

TEE Machine Knobology

The many knobs on a TEE machine are used to acquire and optimize images for evaluation. Refer to Table 12.3 and (Fig. 12.9) for commonly used knobs on the TEE machine and their functions.

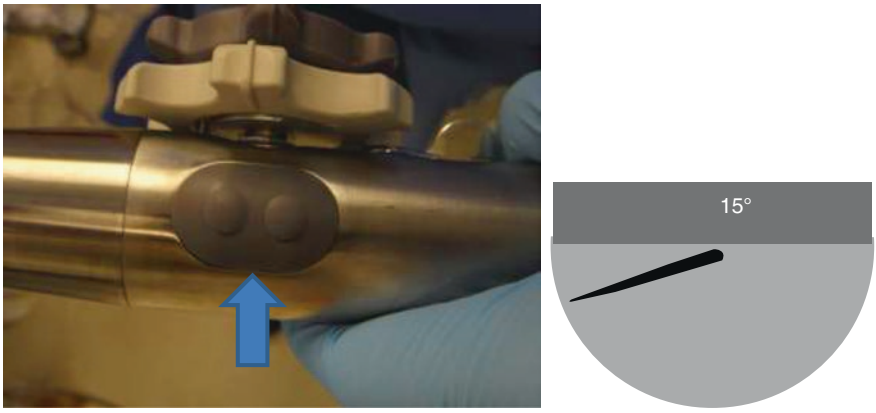


Fig. 12.8 Picture of Left: Omniplane rotation buttons—a function that changes the angle of the tip of the echo probe and can be rotated from 0 to 180 degrees around the long axis of object Right: Illustration of omniplane indicator on the screen

Table 12.3 Commonly used knobs/buttons for image acquisition or optimization

Knob/Button	Function
Patient data	Allows user to enter or review patient information (name, medical record number, etc.)
Gain	Adjusts amplification of returning sound waves. *Increasing gain will result in increased detail and often a lighter image. *Too much gain causes distortion of the image.
Depth	Adjusts the depth of the image seen on TEE *Increasing the depth requires the machine to evaluate portions of the image at a greater distance, thereby reducing the resolution and frame rate.
Caliper	Allows the operator to measure the distance between two points
iSCAN	Automated image optimization process available to users through controls (gain, time-gain compensation, and compression)
Acquire	Saves still images and cine loops to the study "Acquire" can be set to save cine loops of various duration based on a chosen number of cardiac cycles or units of time.
Review	Allows user to review images or acquired cine clips
Freeze	Allows images to be frozen and evaluated *Users can also use the trackball to move forward and backward within the saved cine clip.
Time-gain compensation	Compensates for ultrasound attenuation as ultrasound waves travel through the tissue

Acquiring high-quality real-time TEE imaging depends on three variables: operator experience, patient anatomy, and the TEE machine. An in-depth knowledge of cardiac anatomy, ability to manipulate the probe, and an understanding of TEE knobology are prerequisites for successful and correct interpretation of the images acquired.

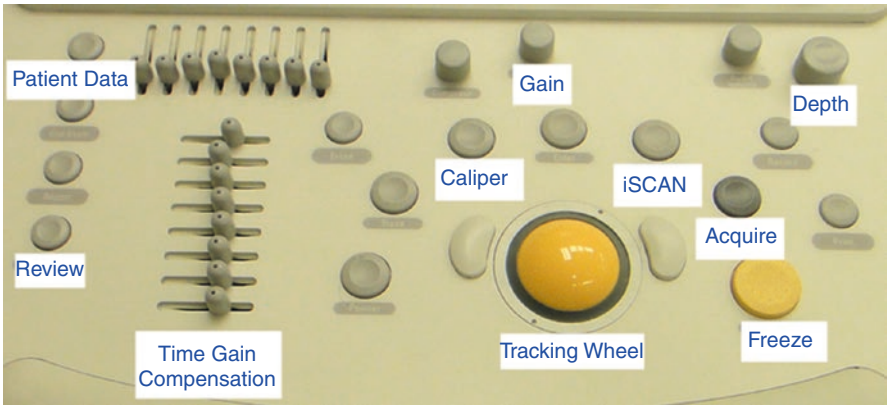


Fig. 12.9 Picture of commonly used knobs/buttons for image optimization

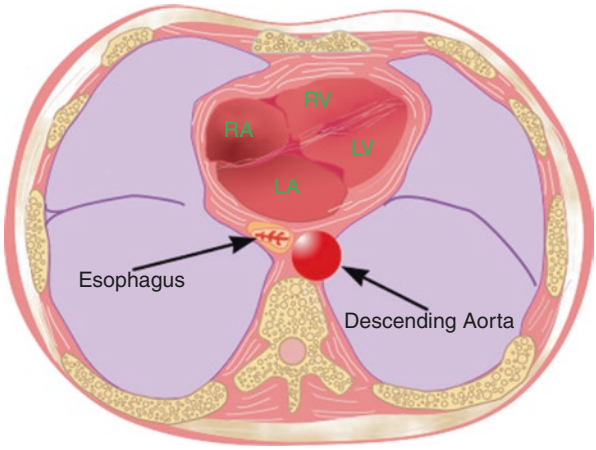


Fig. 12.10 Illustration showing the relation of the esophagus to the posterior wall of the left atrium

Sonoanatomy

To understand displayed images acquired during TEE exam, it is important to appreciate the path and the relationship of the esophagus, where the probe lies, to the other chest contents. The esophagus has three parts: cervical, thoracic, and abdominal. The thoracic part lies between the vertebral column posteriorly and the trachea anteriorly. Then it passes behind the aortic arch, right pulmonary artery, and left bronchus. Further down it passes behind the left atrium (LA). The proximity of the esophagus to the posterior wall of the LA allows the LA to be the main window for image acquisition of the heart (Fig. 12.10).

The four echo windows for image acquisition and the 20 views are summarized in Table 12.4 and shown in (Fig. 12.11).

The TEE probe has 10 cm depth markers which guide manipulation.

Table 12.4 The four echo windows for image acquisition and the 20 views

1.	Upper Esophageal (UE) Window	2 standard views	Distance of the probe tip from teeth is 20–25 cm
2.	Mid-esophageal (ME) window	12 standard views	Distance of the probe tip from teeth is 30–40 cm
3.	Transgastric (TG) window	5 standard views	Distance of the probe tip from teeth is 40–45 cm
4.	Deep transgastric (DTG) window	1 standard view	Distance of the probe tip from teeth is 45–50 cm

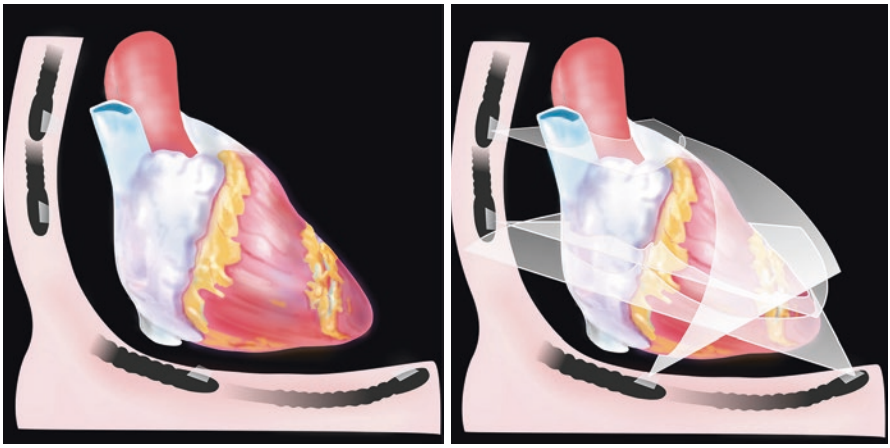


Fig. 12.11 Illustration of the four echo windows for image acquisition Left: Without scan beam Right: With scan beam

Tomographic Views

ASE/SCA guidelines for the standard TEE views that included 20 views was published in 1999. Two more recent intraoperative TEE examination publications were published in 2013 by ASE/SCA: a basic perioperative consensus statement that included the 20 views and a comprehensive TEE guideline that included 28 views. This chapter focuses on the basic 20 views.

The operator should first focus on the primary clinical reason for the TEE exam. In most cases a complete TEE exam can also be performed as this only requires a few additional minutes. A complete TEE examination includes visualization of the following:

- All four cardiac chambers.
- All four cardiac valves.
- The great arteries (aorta, pulmonary artery).
- Atrial septum and LA appendage.
- Pulmonary veins, SVC, IVC.

When performing a TEE, it is important to approach obtaining the 20 basic views systematically. The operator can choose the order they prefer but by repeating that order during each exam, it is less likely that a view is missed, or an important finding overlooked. Basically, choose your order and stick to it.

There are 20 basic TEE views. Multiple online interactive tutorials and TEE educational web sites exist. The interested reader is encouraged to seek them out and use them as needed.

Mid-Esophageal Views (ME)

In the mid-esophagus, the transducer will be just behind the posterior wall of the left atrium. This proximity will allow us to acquire most views. 12 out of the 20 standard views will be acquired in that position (Fig. 12.12).

ME Four-Chamber View

The mid-esophageal 4-chamber (4Ch) view is considered the "home base" of standard esophageal views. It is often the first image that is acquired and serves as a reference point for the operator to orient themselves before proceeding with the primary objective of the exam. Adjust the sector depth to about 14–16 cm and advance the probe to 30–40 cm or until all four chambers of the heart come into view. This view is best imaged at 0°–15° with angulation toward the left ventricular apex. Slight retroflexion is often required to avoid apical foreshortening. The 4Ch view is useful for evaluation of biventricular systolic function, notably the

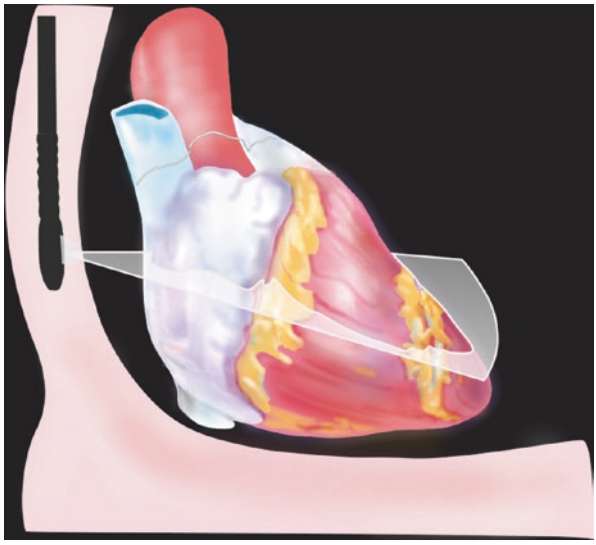


Fig. 12.12 Illustration of mid-esophageal (ME) transducer position within the esophagus

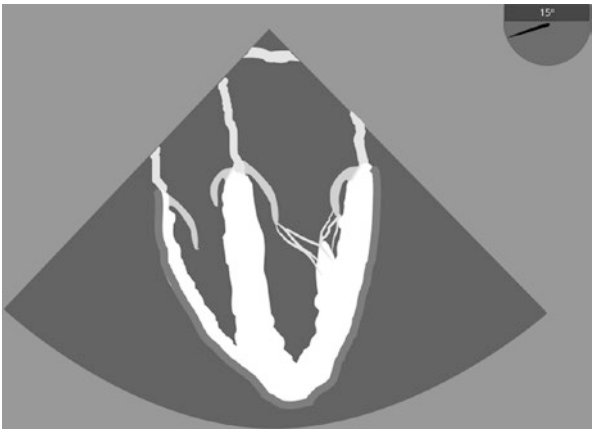


Fig. 12.13 Illustration of TEE ME four-chamber view

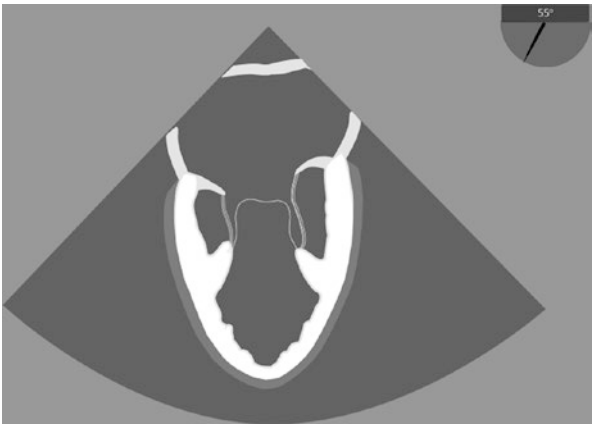


Fig. 12.14 Illustration of TEE ME mitral commissural view

inferoseptal and lateral LV segments. The middle scallops of the mitral valve (A2, P2) and the septal and anterior leaflets of the tricuspid valve are also best imaged in this view (Fig. 12.13).

ME Mitral Commissural

From the four-chamber view, place the mitral valve in the center of the screen and rotate the omniplane angle to 45–60° to obtain the mid-commissural view. Here three scallops can be seen. The middle scallop of the anterior mitral leaflet (A2) fills most of the central annulus area while smaller portions of the posterior mitral leaflet (P3, P1) are seen medially and laterally (Fig. 12.14).

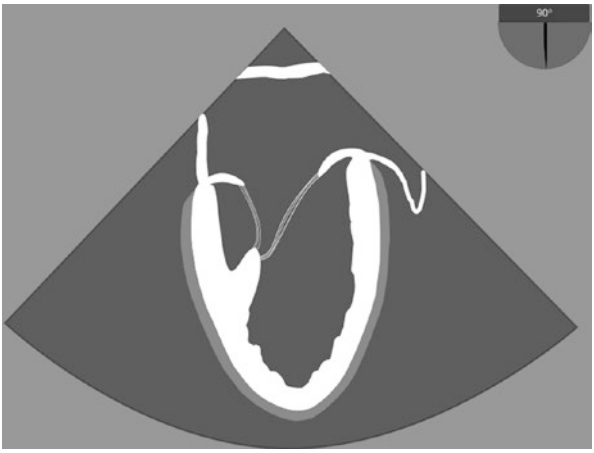


Fig. 12.15 Illustration of TEE ME two-chamber view

ME Two-Chamber View

From the commissural view rotate the omniplane angle to 90° in order to gain the two-chamber view. Here the left atrium and left ventricles can be seen as well as the mitral valve. The anterior and inferior LV segments are imaged in this view (Fig. 12.15). The LAA is also often brought into view. The operator can anteflex the probe in order to optimize the view of the LAA. Keep the LAA centered in the image and use the Zoom feature. Also, The LAA should be imaged in at least two orthogonal views by using biplane mode to rule out thrombus. With slight withdrawal of the TEE probe the left superior pulmonary vein can be imaged.

ME Long Axis View

The mid-esophageal long-axis view is obtained from two-chamber view by keeping the mitral valve in the center and further rotation of the image plane to 120°–140°. In this view the left ventricular outflow tract (LVOT), right and non-coronary leaflets of the aortic valve, proximal ascending aorta, and mitral leaflets (A2, P2 scallops) are visualized. The anteroseptal and posterior segments of the left ventricle are also seen in this view in addition to a portion of the RVOT anterior to the aortic valve. The operator may need to advance or withdraw the probe slightly to optimize the view (Fig. 12.16)

ME Aortic Valve LAX

From the LAX of the heart view, decrease the sector depth and withdraw the probe slightly to focus in on the aortic root. In this view one can evaluate the LVOT, aortic valve, aortic root, and ascending aorta. The LV is often cut in a way that only a

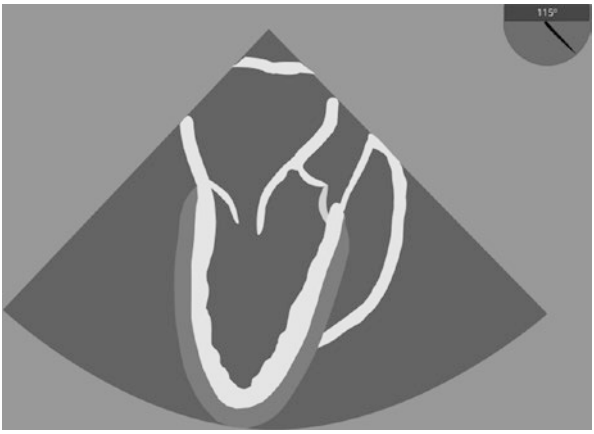


Fig. 12.16 Illustration of TEE ME LAX view

Fig. 12.17 Illustration of TEE ME AV LAX view

portion is seen. Sometimes slight anteflexion can optimize the structures in this view. The operator can also decrease the depth to focus in better on the LVOT, aortic valve, and ascending aorta (Fig. 12.17).

ME Ascending Aorta LAX

ME Ascending Aorta LAX: From the AV LAX view, withdraw the probe slowly and decrease the angle to 90–120° until the ascending aorta can be seen in long axis. This is a good view for evaluation of the ascending aorta for plaque and calcification in the location where the surgeon may be placing a cannula and cross clamp. The PA can also be seen in short axis here (Fig. 12.18).

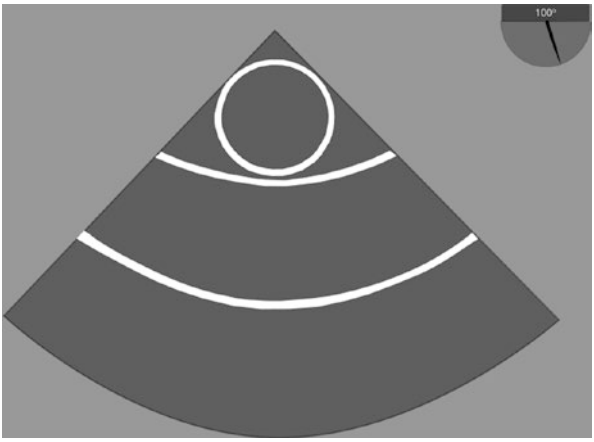


Fig. 12.18 Illustration of TEE ME ascending aorta LAX

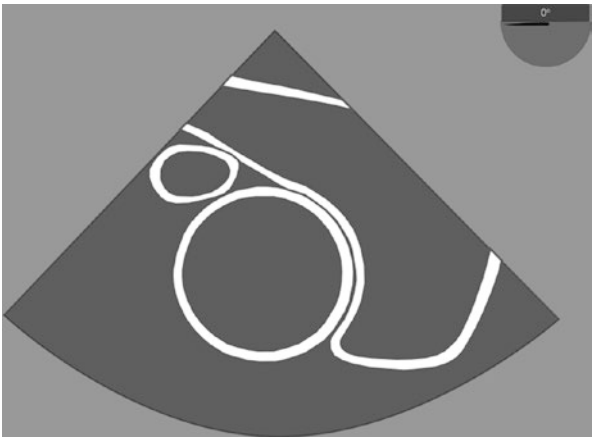


Fig. 12.19 Illustration of TEE ME ascending aorta SAX

ME Ascending Aorta SAX

From the ascending aorta LAX view, reduce the angle of the probe to 0° (should be between 0–15°) and the aortic valve will be seen in short axis. Anteflexion of the probe should bring the main PA and right PA into view as well as the SVC. This is also known as the great vessel view (Fig. 12.19).

ME AV SAX

To obtain a short axis view from the ascending aorta SAX view, advance the probe until the AV becomes apparent. Then, the image plane is rotated between 30° and 45° until the AV looks like a "Mercedes Benz" sign (if the valve is trileaflet). Decrease the depth to focus in on the aortic valve (Fig. 12.20).

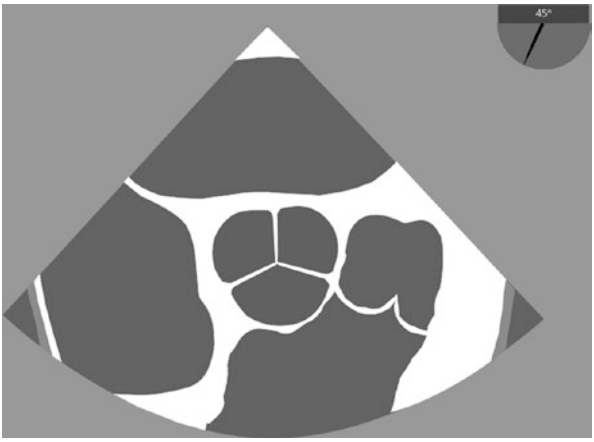


Fig. 12.20 Illustration of TEE ME AV SAX view

Fig. 12.21 Illustration of TEE RV inflow-outflow

ME RV Inflow-Outflow

From the AV SAX view, advance the probe slowly and open the angle to 50–60°. Increase the sector depth to 10–12 cm. In this view the AV is still visible although all three leaflets may not be visualized. The Interatrial septum can be evaluated here, and color flow Doppler can be used to assess for PFO. Put color on and use the scale knob to decrease the scale to around −20 to +20 in order to look for PFO. In addition, this view is ideal for evaluating the RA, RV, and TV. The RVOT is usually well visualized here and the pulmonic valve can be evaluated as well (Fig. 12.21).

ME Bicaval View

From the RV inflow-outflow rotate the angle to 90–100° and turning the probe towards the patient's right side until both atria are in view with the LA at the top of the screen and the RA below it. In this view, the right atrium (RA) and the atrial septum are the primary focus with the inferior vena cava entering the RA from the left side of the screen and the superior vena cava from the right (Fig. 12.22)

Descending Aorta SAX

From the bicaval view, rotate the angle back to 0°. Turn the probe to the left until the descending aorta can be seen (it should look circular). Decrease the depth between 5–8 cm to more closely evaluate the aorta. Advance and withdraw the probe to examine the length of the descending thoracic aorta (Fig. 12.23).

Fig. 12.22 Illustration of TEE ME bicaval view

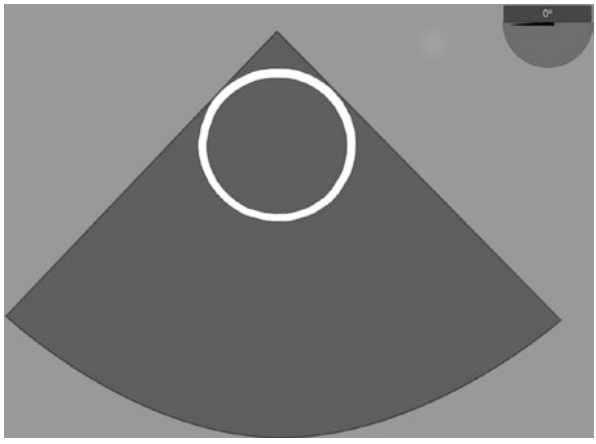


Fig. 12.23 Illustration of TEE ME LAX view

Descending Aorta LAX

From the descending aorta SAX view, rotate the angle out to 90°maintaining the same depth. In this view, the walls of the aorta can be seen in parallel to each other. Again, advance and withdraw the probe to examine the length of the aorta (Fig. 12.24).

Transgastric Views (TG)

With the transducer in the stomach, 5 out of the 20 standard views will be acquired in that position (Fig. 12.25).

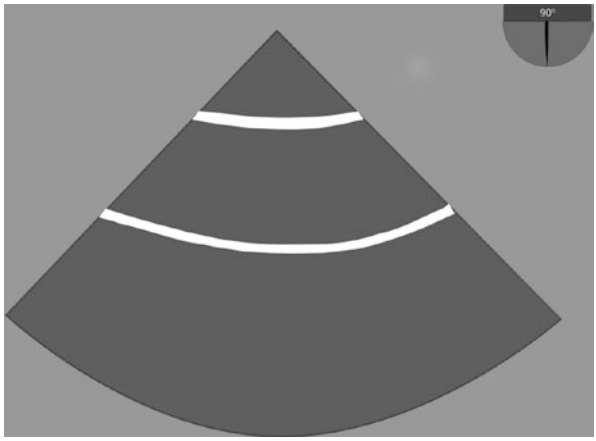


Fig. 12.24 Illustration of TEE ME LAX view

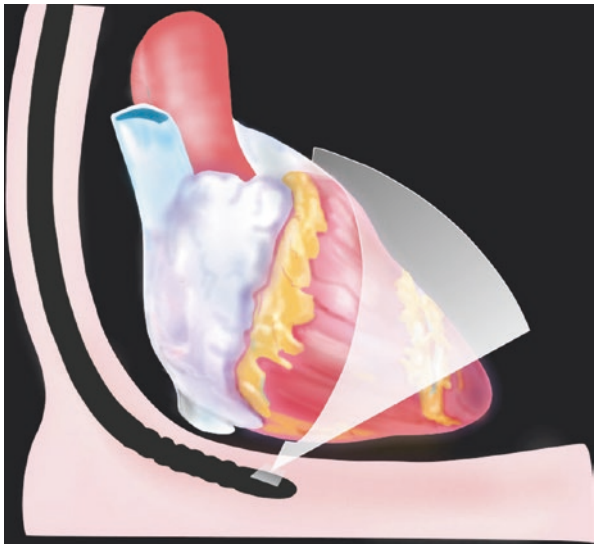


Fig. 12.25 Illustration of transgastric (TG) transducer position within the stomach

Transgastric Mid-Papillary SAX

From the mid-esophageal view, the transducer is slowly advanced past the gastro-esophageal junction into the stomach. With the image plane at 0° and anteflexion applied to the big wheel, a short-axis view of the left ventricle at the papillary level is obtained. This is the optimal view for evaluation of global LV systolic function, regional wall motion, and LV wall thickness (Fig. 12.26).

TG Basal SAX View

From the mid-pap view, anteflex the probe until the mitral valve apparatus comes into view. Alternately, the operator can withdraw the probe until the mitral valve is brought into view. This is the basal segment of the LV (Fig. 12.27).

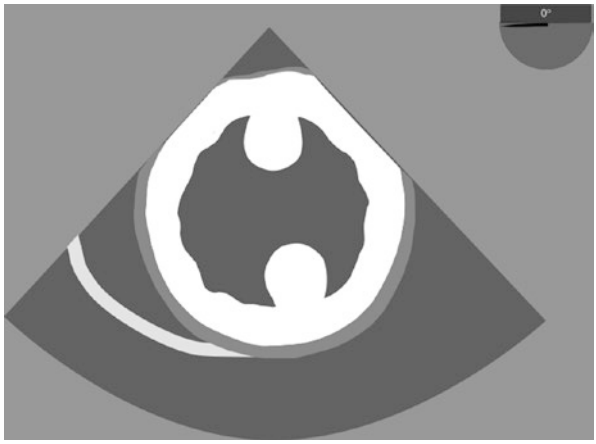


Fig. 12.26 Illustration of TEE TG mid-papillary SAX

Fig. 12.27 Illustration of TEE TG basal SAX view

TG Two-Chamber View

From the TG mid-papillary SAX, rotate the angle out to 90–110°. If the probe was anteflexed in the mid-pap view, maintain the anteflexion while rotating out to the two-chamber view. Here the LA, LAA, basal LV, and mitral valve should be visible (Fig. 12.28).

TG LAX View

From the TG two-chamber view, rotate to 120–140°. The operator may need to turn the probe slightly to the right in order to optimize the view. This view provides excellent parallel alignment of the aortic valve and the left ventricular outflow tract for Doppler evaluation (Fig. 12.29).

Fig. 12.28 Illustration of TEE TG two-chamber view

Fig. 12.29 Illustration of TEE TG LAX view

TG RV Inflow-Outflow View

From the TG LAX view, decrease the angle to between 90–110° and turn the probe to the right. The right atrium and right ventricle can be seen in this view as well as the TV. This is a difficult view to acquire. Anteflexion and omniplane to 0–20° may be needed to optimize the view (Fig. 12.30).

Deep Transgastric View (DTG)

With the transducer in deep the stomach, the only view out of the 20 standard views will be acquired in that position (Fig. 12.31).

Fig. 12.30 Illustration of TEE TG RV inflow view

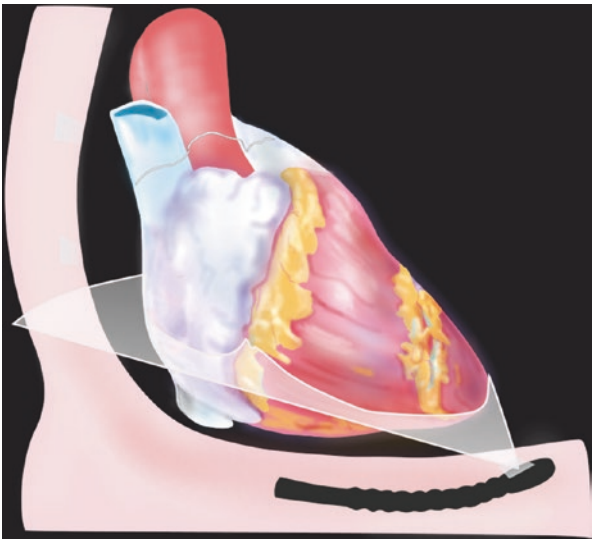


Fig. 12.31 Illustration of deep transgastric (DTG) transducer position within the stomach

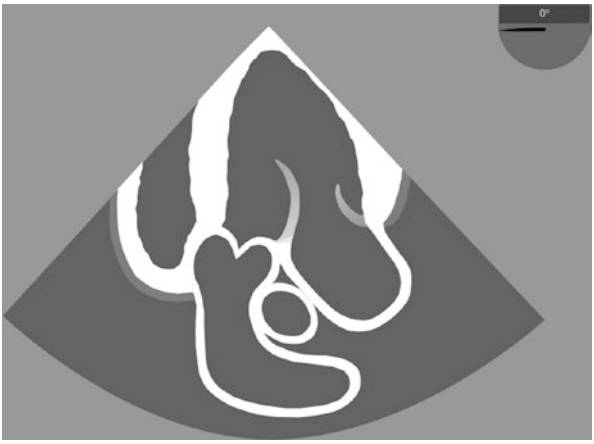


Fig. 12.32 Illustration of TEE deep TG LAX view

From the TG RV inflow view, decrease the angle to between 0–20° and release any flexion on the probe then advance further into the stomach until slight resistance is felt. Next, anteflex the probe ("big wheel") and withdraw the probe slowly until the heart comes into view. Once in view, make very gentle movements advancing or withdrawing the probe and then turn it slightly left with left flexion ("small wheel") until the aortic valve leaflets can be seen opening and closing. Increase the sector depth to about 16–18 cm. Because of the optimal parallel alignment of the Doppler beam with transaortic flow and the LVOT, therefore, this view is useful for the evaluation of aortic valve gradients in aortic stenosis (Fig. 12.32).

Upper Esophageal (UE)

With the transducer withdrawn in upper esophagus, two views out of the 20 standard views showing the great vessels will be acquired in that position (Fig. 12.33).

UE Aortic Arch LAX

From the descending aorta SAX view, at 0–10°, withdraw the probe until the aorta becomes oval in shape rather than round and drop-out occurs due to the overlying trachea. The depth should be between 5 and 8 cm. At this point, very slight movements should be made to optimize the view. Turning the probe slightly to the left with minor anteflexion often brings the best image of the arch into view. (Fig. 12.34).

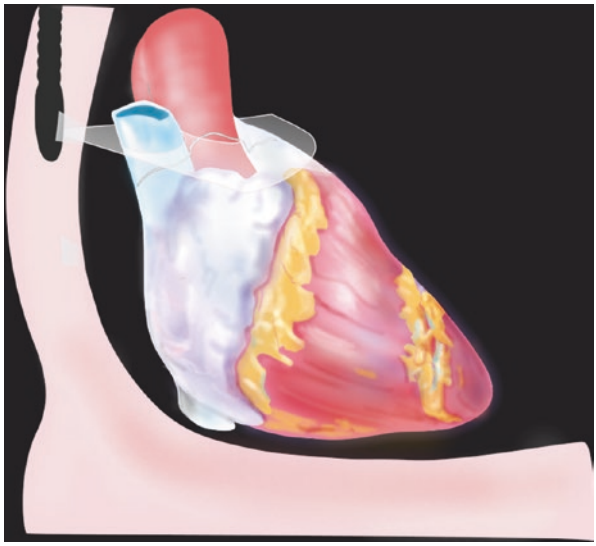


Fig. 12.33 Illustration of upper esophageal (UE) transducer position within the esophagus

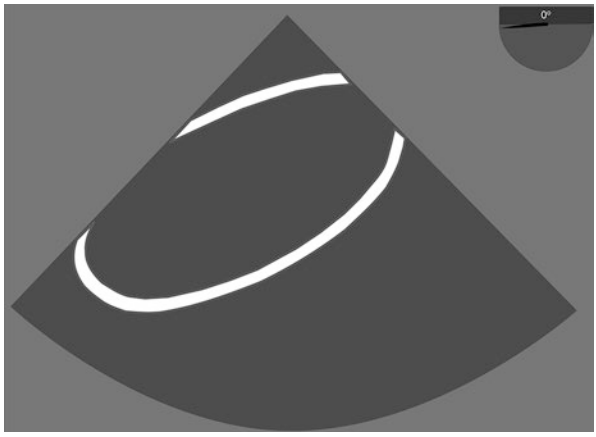


Fig. 12.34 Illustration of UE aortic arch LAX

UE Aortic Arch SAX

From the UE aortic arch LAX, open the angle to 60–90 degrees. Here, the aortic arch can be seen in short axis and will appear circular in shape. The left subclavian artery may be seen by counterclockwise rotation in this view. The pulmonary artery and pulmonic valve are visible here and will appear on the left of the screen with the PA at the top extending into the pulmonic valve, which will appear toward the bottom of the screen (Fig. 12.35).

Below is a summary and systematic approach for image acquisition and assessment of the 20 standard TEE views (Table 12.5).

Key Points

- TEE is a safe, low-risk procedure used for most cardiac surgeries. Major bleeding is rare and occurs in less than 0.01% of patients.
- There are 20 standard TEE views obtained from the esophageal and gastric levels.
- Hemodynamic changes during anesthesia may falsely underestimate severity of valvular stenosis or regurgitation.

Fig. 12.35 Illustration of UE aortic arch SAX

Table 12.5 Summary and systematic approach for image acquisition and assessment of the 20 standard TEE views

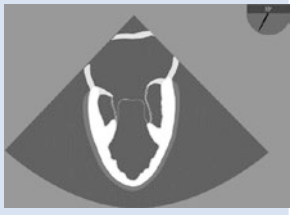
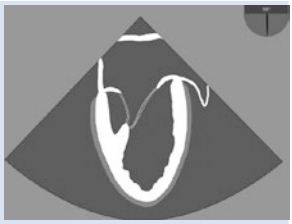
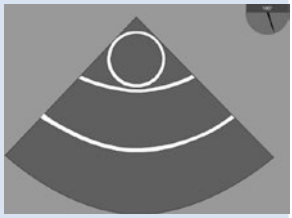
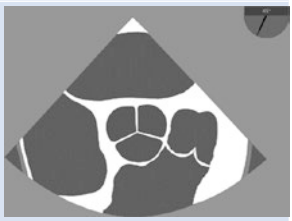
How to acquire	What to see	Image illustration
1. *ME 4C view* (0–20°) Insert probe to 30–35 cm with sector Depth 14–16 cm Retroflex the probe tip to avoid foreshortening Adjust the depth to ensure to view entire heart	Image all four chambers Apply color doppler to evaluate MVR and TVR Evaluate lateral and septal LV wall motion and free wall of the RV Evaluate chambers size	
2. *ME mitral commissural view* (45–65°) Probe distance and sector depth as before From the previous image, keep the MV in the center in the 4 CV view Open angle to 45–65° Retroflex slightly to see LV apex	Seagull view Evaluate mitral valve scallops Apply color Doppler to evaluate MVR MV annulus measure	

Table 12.5 (continued)

How to acquire	What to see	Image illustration
3. *ME 2C view* (80–100°) Probe distance and sector depth as before From the previous image, keep the MV in the center in mitral commissural view Open angle to 80–100°	Image LA, LV, and LAA Evaluate anterior and inferior LV wall motion Apply color Doppler to evaluate MVR Rule out LAA thrombus	
4. *ME LAX view* (120°–140°) Probe distance and sector depth as before From the previous image, open angle to 120–140° Retroflex slightly to see LV apex	Image LA, LV, LVOT, AV, and MV Apply color Doppler to evaluate MVR and AVR Evaluate anteroseptal and inferolateral LV wall motion	
5. *ME AV LAX* (120–140°) From the previous image, withdraw the probe slightly and anteflex Decrease the sector depth decrease depth to focus on aortic valve	Image LVOT, aortic root, ascending aorta, AV, and MV Evaluate MV and AV diseases Apply color Doppler to evaluate MVR and AVR	
6. *ME ascending aorta LAX view (*90°–120°) Probe is in mid-esophagus with 35–40 cm distance and sector depth 8–10 cm From the previous image, withdraw the probe and decrease the angle slightly	Image mid-ascending aorta and Rt. PA Evaluate presence of pulmonary embolus Select cannula and cross clamp site	
7. *ME ascending aorta SAX view (*0°–15°) From the previous image, reduce the angle to 0–15° and anteflex Adjust sector depth to 10-12 cm	Image mid-ascending aorta, main PA and bifurcation Apply color Doppler to evaluate presence of PDA Evaluate presence of pulmonary embolus	
8. *ME AV SAX view* (35°–55°) From the previous image, advance and anteflex Decrease sector depth to 8–10 cm	Image leaflets of the valve "Mercedes Benz" sign Check presence of AS and perform planimetry Apply color Doppler to evaluate AI and PI	

(continued)

Table 12.5 (continued)

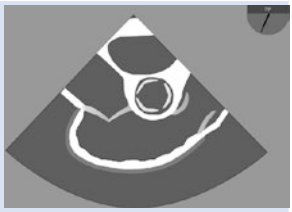
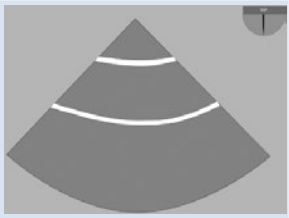
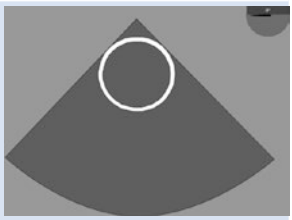
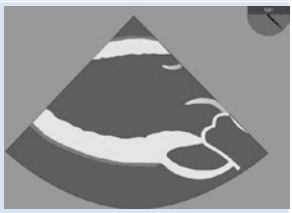
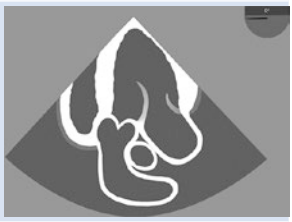
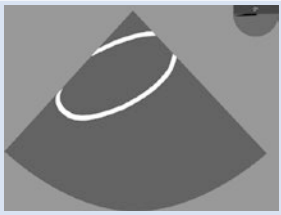
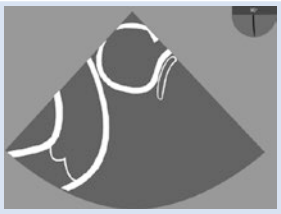
How to acquire	What to see	Image illustration
9. *ME RV inflow-outflow view* (50°–60°) From the previous image, open angle to 50°–60° Maintain sector depth to 8–10 cm	Image RA, RV, TV, LA, PV, RVOT, and AV Evaluate interatrial septum	
10. *ME bicaval view* (90–100°) From the previous image, open angle to 90–100°	Image RA, LA, and the SVC and IVC Evaluate interatrial septum for a PFO or ASD	
11. *ME descending aorta LAX* (90–100°) From the previous image, turn the probe to the left and open angle to 90–100° Decrease sector depth to 6–8 cm	Image the descending aorta Evaluate atheroma disease Assess severity of AI by Doppler wave Guide IABP	
12. *ME descending aorta SAX* From the previous image, decrease angle to 0–10° Maintain sector depth to 6–8 cm Slight anteflex	Image the descending aorta Evaluate atheroma disease Evaluate presence of pleural effusion	
13. *TG mid-SAX view* (0–20°) From the previous image, release any flexion and push the probe in the stomach 35–40 cm with a sector depth of 12–4 cm Anteflex as needed Adjust gain to see endocardial borders	Image the LV in cross section Assess wall motion abnormality for mid-LV Assess interventricular septum shape	
14. *TG basal SAX view* (0°) From the previous image, maintain sector depth to 12–14 cm Slightly withdraw and anteflex	Image the LV and mitral valve enface Assess basal segment of the LV Apply color Doppler to evaluate origin of MR	

Table 12.5 (continued)

How to acquire	What to see	Image illustration
15. *TG 2C view* (90°) From the previous image, open angle to 90–100° Maintain slight anteflexion and same sector depth Keep the anterior LV wall horizontal	Image the LV and mitral valve apparatus Assess LV anterior and inferior wall motion Assess LAA (useful if you can't get it in any other plane)	
16. *TG LAX view* (120–140°) From the previous image, open the angle to 120–140° cm with Turn the probe slightly to the right	Image the LV, LVOT, RV, and mitral valve Measure gradient across LVOT and AV by spectral Doppler	
17. *TG RV inflow view* (90°) From the previous image, decrease the angle to 90° Turn the probe to the right Anteflex	Image the right atrium and right ventricle Assess TV	
18. *Deep TG LAX TG 5C view* (0°–15°) From the previous image, release any flexion then advance the probe deep in the stomach Turn the probe to the left side Anteflex and left flex the tip	Image the LV, LVOT, RV, AV, and mitral valve. Apical 5 chamber view Measure gradient across LVOT and AV by spectral Doppler Check for AV paravalvular leak	
19. *UE aortic arch LAX* (0°) From the DA SAX view, at 0–10°, withdraw the probe until it becomes oval Decrease the depth to 5–8 cm Turn the probe slightly to the left with minor anteflexion	Image the aortic arch Innominate vein	
20. *UE aortic arch SAX* (60–90°) From the previous image, open the angle to 60–90° Maintain same sector depth	Image the aortic arch, left subclavian artery, innominate vein, and PV Assess presence of PDA	

Further Reading

1. Couture P, et al. Impact of routine use of intraoperative transesophageal echocardiography during cardiac surgery. Can J Anesth. 2000;47:20–6.
2. Eltzschig HK, et al. Impact of intraoperative transesophageal echocardiography on surgical decisions in 12,666 patients undergoing cardiac surgery. Ann Thorac Surg. 2008;85(3):852–3.
3. Hahn RT, et al. Guidelines for performing a comprehensive transesophageal echocardiographic examination: recommendations from the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists. J Am Soc Echocardiogr. 2013;26:921–64.
4. Hofer CK, et al. Therapeutic impact of intraoperative transesophageal echocardiography during non-cardiac surgery. Anaesthesia. 2004;59:3–9.
5. http://pie.med.utoronto.ca/TEE/index.htm
6. Otto MC. Textbook of clinical echocardiography. 6th ed:: Elsevier; 2019.
7. Reeves ST, Finley AC, Skubas NJ, et al. Basic perioperative transesophageal echocardiography examination: a consensus statement of the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists. J Am Soc Echocardiogr. 2013;26(5):443–56. https://doi.org/10.1016/j.echo.2013.02.015
8. Vegas A. Normal tee views. In: Perioperative two-dimensional transesophageal echocardiography. New York, NY: Springer; 2012.

Part IV

Cardiopulmonary Bypass

Fundamentals of Cardiopulmonary Bypass Machine Equipment and Technique

13

Michael Hancock and Enrique J. Pantin

The greatest problem in communication is the illusion that it has been accomplished

—Daniel W. Davenport

Introduction

The advances in the field of cardiac surgery have been achievable because of the development and technological innovation of cardiopulmonary bypass (CPB). After years of experiments, in 1953 Dr. John Gibbon created a cardiopulmonary bypass (CPB) machine and executed the first successful intracardiac repair using an artificial heart and lung extracorporeal blood circuit. Over the last 65 years, the heart-lung machine has evolved into a safe, efficient, and reliable extracorporeal system, through the continuous improvements in techniques, methods, and materials. The primary goal and function of the CPB machine is to temporarily replace the patient's cardiopulmonary system and perfuse the other vital organs with oxygenated blood while the heart is arrested (Fig. 13.1).

Electronic Supplementary Material The online version of this chapter (https://doi.org/10.1007/978-3-030-51755-7_13) contains supplementary material, which is available to authorized users.

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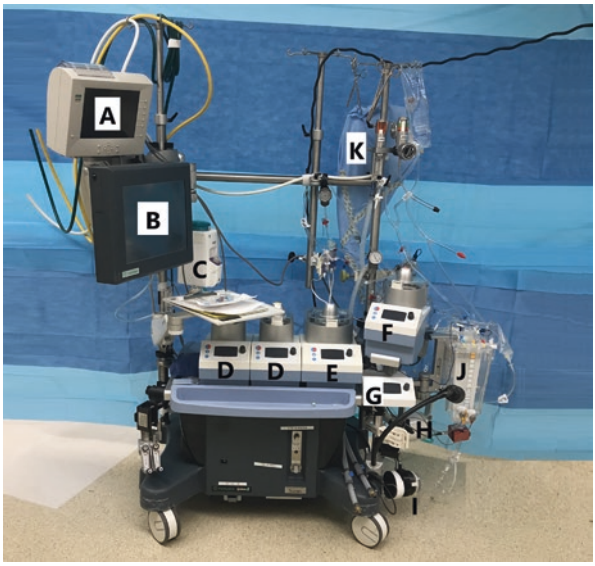


Fig. 13.1 Cardiopulmonary bypass machine. A: gas analyzer console. B: main console. C: anesthetic vaporizer. D: roller pumps used as secondary suctions. E: roller pump, used as main suction pump. F: roller pump used to deliver cardioplegia. G: console of centrifugal pump. H: oxygenator. I: centrifugal pump. J: venous reservoir, also called cardiotomy reservoir. K: sterile tubbing package that contains the arterial, venous, cardioplegia, and suction lines all connected in a closed-loop circuit that allows the pump to circulate the pump prime and test the integrity and functionality of the circuit before connecting the patient to the machine. Once the surgical team receives sterilely all the tubbing contained in this package the arterial/venous lines will be clamped and then divided in preparation for each of them to be connected to the arterial cannula and venous cannula

Cardiopulmonary Bypass Components and the Perfusion Circuit

The essential functions of the CPB machine are to provide

1. Oxygenation
2. Ventilation and elimination of carbon dioxide
3. Circulation of blood
4. Temperature control (cooling/rewarming) of blood

The Basic Extracorporeal Circuit Components

The standard CPB machine has five necessary components, which comprise two principal functional units: the arterial pump (artificial heart) and the blood oxygenator (artificial lung), with interconnected series of cannulas and tubes.

The five components are the following (Fig. 13.2):

1. Venous reservoir
2. Main pump
3. Oxygenator

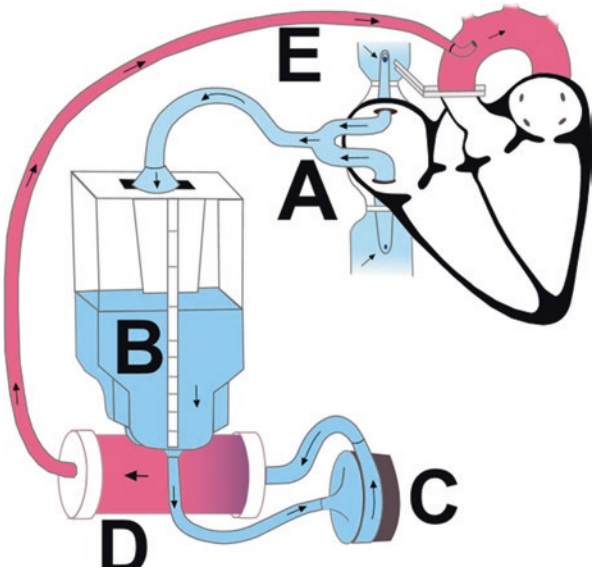


Fig. 13.2 Diagram of the main components of the extracorporeal circuit. A: venous drainage cannula. B: cardiotomy reservoir. C: blood pump "pump head." D membrane oxygenators with heat exchanger. E: arterial bloodline

4. Heat exchanger
5. Tubing, connectors, and cannulas

The complete CPB machine for clinical use includes many other accessory components, such as various types of filters and additional pump heads for suction, venting (draining) of the heart, and delivery of cardioplegic drugs.

The CPB console is a platform that accommodates the various components that interface with the main and accessory components. The console also has control and monitoring panels, safety sensors with alarms, vacuum sources, central microprocessor, and electrical power.

Current-generation CPB circuits have commercially available packages that consist of disposable tubing, a hard-shell venous reservoir, a membrane oxygenator with integrated heat exchanger, connectors, an arterial filter (some systems no longer use arterial filters since the cardiotomy reservoir filter provides high-quality filtering), and a disposable cardioplegia delivery portion, which connects to form an extracorporeal circuit system that supports the function of patients' vital organs.

The Perfusion Circuit Components

The individual components and their role in the CPB circuit are described next.

1. Cannulas

During the operation, the surgeon inserts several cannulas:

- Venous cannulas
- Arterial cannulas
- Cardioplegia-administration cannulas
- Vent/suction cannulas

Venous Cannulas

The purpose of venous cannulas is to drain as much of the patient's venous return as possible, sending the deoxygenated blood to the CPB collection reservoir. A few types of venous cannulas are available, and the choices among them are governed by the type of surgical procedure to be performed, surgeon's preference, patient's body size, and required flow rate.

Types of Venous Cannulas

The most common types of venous cannulas used are:

- Cavo-atrial dual stage or multistage cannula. Each "stage" is a group of holes in the cannula. These cannulas are inserted into the right atrial appendage, with the tip lying in the inferior vena cava, thus capturing the upper- and lower-body venous return. The cavo-atrial dual stage is the most common kind of cannulation for aortic valve and coronary artery bypass graft surgery (Fig. 13.3).
- Bicaval cannulas are two separate single-stage cannulas. They are inserted in each cava directly (plastic or metal tip cannula) or indirectly via the right atrium, forcing all the venous return into the pump circuit. Bicaval cannulas are used when total CPB is needed, as with surgery on right heart structures, e.g., tricuspid valve or right atrial masses. If mitral valve surgery via the trans-atrial septal approach is performed, bicaval cannulas prevent air from entering the pump circuit and minimize the amount of blood in the operative field (Fig. 13.3).

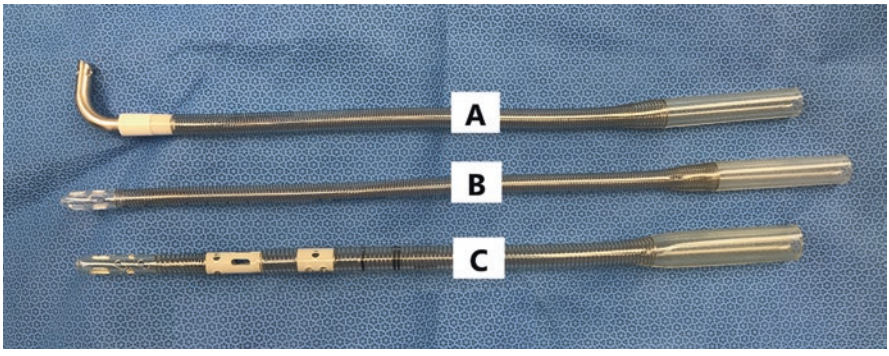


Fig. 13.3 Venous cannulas used to cannulate the heart directly. There are several types. For central cannulation for procedures requiring bicaval cannulation, a cannula is placed in each cava. These can be metal tip (**a**), or single stage (**b**). Commonly a dual- or triple-stage cannula (**c**) is inserted into the right atrial appendage with the tip lying in the IVC for procedures that do not require work to be done in the atrial septum, the right atrium, or ventricle.

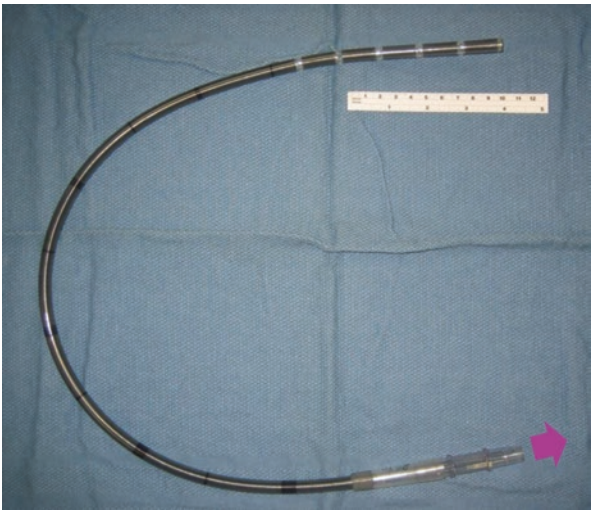


Fig. 13.4 Venous cannulas used to cannulate the heart through the femoral vein.

- Femoral vein cannulas are employed for minimally invasive procedures. A long catheter is advanced under transesophageal guidance and positioned with the tip at the junction of superior vena cava and right atrium. Drainage holes in the catheter capture blood from the upper and lower body (Fig. 13.4).

Types of Venous Drainage

The amount of venous return captured depends on cannula size and position, the patient's available blood volume, and the overall negative pressure pulling blood into the perfusion circuit.

The two most common types of drainage are:

1. *Gravity*

The blood reservoir is positioned lower than the patient's body to allow gravity to assist in draining the venous blood by siphon effect. If the venous tubing line becomes filled with air, blood drainage will be interrupted.

2. *Vacuum-assisted drainage*

Vacuum-assisted venous drainage can be used to increase the negative pressure, thus drawing more blood into the circuit. This kind of drainage is achieved by attaching the venous line to the venous reservoir, to which negative pressure (typically −20 to −50 mm Hg) is employed. A phenomenon called atrial chatter can occur when the negative pressure is too high, thus causing the atrial walls to collapse around the cannula. This will limit venous drainage and reduce pump flow. Reducing vacuum pressure will diminish the negative pressure, and, by partially occluding the venous line or adding volume to the system, the chatter will be eliminated.

Effect of Persistent Left Superior Vena Cava (LSVC) on Venous Drainage

Persistent LSVC is a remnant of fetal development, in which vessels of the left head and arm most commonly drain via an LSVC into the distal coronary sinus. This congenital anomaly is usually suspected when a large coronary sinus is visualized on TEE. It can be confirmed by injecting agitated saline into a left arm vein and seeing the bubble contrast appears initially in the distal coronary sinus and then drains through it, back into the right atrium. In a normal right superior vena cava, the bubble contrast appears first in the right atrium on TEE. Problems that can arise from this anomaly include incomplete venous drainage of the right side of the heart and an inability to use retrograde cardioplegia in surgery because the coronary sinus receives blood not only from the heart but from systemic venous drainage; thus it is impossible to pressurize the coronary sinus during retrograde administration of cardioplegia. If the left innominate vein is present, temporarily occluding the LSVC will suffice, but if the left innominate vein is absent, a third venous cannula in the LSVC is needed.

Arterial Cannulas

Arterial cannulas made of various materials are available, and the choice of which to use depends on the site of insertion and purpose of the procedure (Fig. 13.5). Other factors that determine the choice of the cannula are the size, wall thickness, presence of calcification, and presence of mobile atheroma in the aorta, and the patient's size and perfusion requirements.

Types of Arterial Cannulas

These are the most common arterial cannulas:

1. *Soft diffusion cannulas* with multiple side holes and angled tips that decrease the velocity of the jet (to avoid jets that can cause arterial dissection or dislodge atheroma)

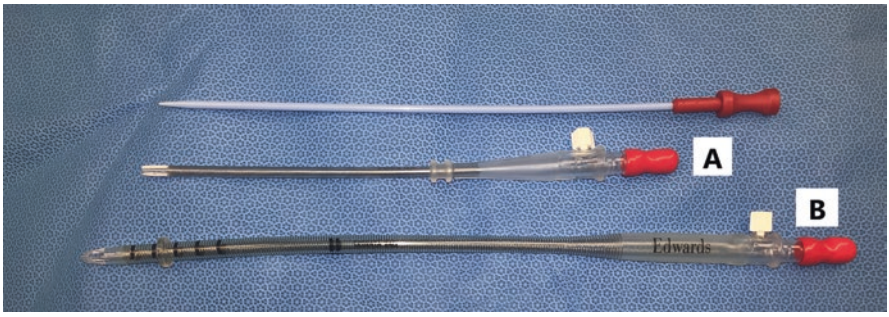


Fig. 13.5 Arterial cannulas. There are several types. Showen below the commonly used type for femoral cannulation with its obturator (**a**), and the one used for direct aortic cannulation (**b**)

2. *Oblique metal-tipped cannulas or hard plastic-tipped cannulas* with or without a small disc, which can fix the cannula to the aorta
3. *Flexible cannulas or wire-enforced cannulas* to prevent kinking when placed outside the surgical field
4. *Cannulas with Luer ports* for removal of air or for pressure monitoring
5. *Cannulas with an intra-aortic filter* to catch embolic materials

Cannulation Site Options

- Ascending aorta:
  This is the most common approach. The aortic arterial cannula is carefully placed in the distal ascending aorta. Surgeons have relied on palpation to select sites for cannulation and cross-clamping; however, epi-aortic ultrasonography provides an effective way to detect atherosclerotic plaques. Purse-string sutures are used to secure the cannula in place and prevent bleeding. The tip of the arterial cannula is the point of highest resistance within the perfusion circuit. Once the arterial cannula is inserted, the surgeon will back bleed the cannula, then make an air-free connection to the circuit's arterial line. The perfusionist will test the flow and pressure, making sure cannulation into a false lumen of the aortic wall did not occur.
- Femoral artery
  This is the second most common site. Femoral arterial cannulation can be used for minimally invasive operations, treatment of aneurysms of the ascending aorta, and redo surgery. In the femoral approach, the cannula is inserted into the artery percutaneously or by cutdown, guided (ideally) by TEE. TEE allows visualization of the thoracic or abdominal aorta to assure that the introducer wire is placed intraluminally. The tip of femoral arterial cannulas resides in the iliac artery and provides a route for retrograde perfusion.
- Innominate artery
  Use of this site eliminates the requirement for a second incision, as is required with a subclavian approach.
- Axillary/subclavian artery
  This site is preferred in some cases of type A aortic dissection. Commonly, the surgeon will sew an 8-mm tube graft to the artery and connect the arterial cannula to it to avoid occluding the artery distally, which could occur if the cannula were inserted directly.
- Left ventricular apex
  With the use of this site, which is rarely used, the left ventricular apex is cannulated, and the cannula is passed through the aortic valve into the aortic root.

Cardioplegia-Administration Cannulas

To both arrest and protect the heart, cardioplegia solution is delivered directly to the myocardium through its vasculature after cross-clamp application.

Types of Cardioplegia-Administration Cannulas:

A specific cannula type must be selected, depending on the operation to be performed.

These are the most common cardioplegia cannulas (Fig. 13.6):

1. Aortic root cannulas
   These cannulas feature a small-gauge tip attached to clear tubing with or without separate vent lines, and they usually have a stainless-steel introducer needle. An additional feature might include an aortic-root pressure monitoring apparatus.
2. Handheld ostial cannulas
   These cannulas allow infusion directly into the coronary arteries.

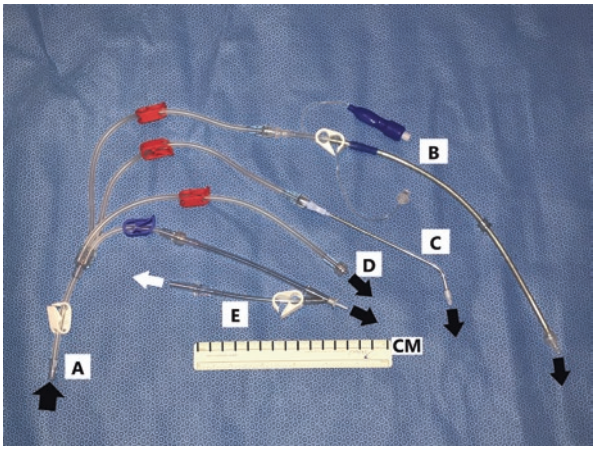


Fig. 13.6 Cardioplegia cannulas. There are several options for cardioplegia administration. (**a**): manifold that once connected to the cardioplegia line, managed by perfusion, will allow cardioplegia delivery through different routes depending on surgical requirements and which clamp is open. The surgeon and perfusionist work closely during cardioplegia administration to avoid confusion. (**b**): coronary sinus cannula for retrograde cardioplegia administration. Note that this cannula has an inflatable balloon and a pressure measuring port that is transduced during the cardioplegia administration to avoid excessive coronary sinus pressure and reduce the risk of coronary sinus rupture. (**c**): metal canula for direct coronary ostium cardioplegia administration. (**d**): extra port that can be used to deliver cardioplegia to a vein graft. (**e**): aortic root cannula used to deliver cardioplegia or drain the aortic root (by connecting white arrow port to CPB machine suction pump) depending on what the surgeon requires. This is controlled by opening or closing corresponding clamps while working with perfusion. (*CM*: centimeters)

3. Balloon-tipped retrograde cannulas
   There are many types of coronary sinus cannulas, including self-inflating balloon cannulas that auto-inflate with the administration of cardioplegia. These catheters either have a rigid, preshaped stylet with a directing handle or a flexible stylet.

Routes of Injecting Cardioplegia Solutions

- *Antegrade cardioplegia delivery*
  This kind of delivery is accomplished by placing a cannula just distal to the aortic valve and proximal to the aortic root, leaving enough room for placement of a cross-clamp. Once the cross clamp is applied, the cardioplegia solution is administered into the aortic root at a flow rate high enough to close the aortic valve leaflets. This manner of administration forces the cardioplegic solution directly into the coronary arteries, where it travels the normal pathway of coronary arterial circulation. If the distribution of the solution is satisfactory, the heart will soon have diastolic arrest, as evident from asystole on the ECG monitor.
- *Direct delivery of cardioplegia into the coronary ostium*
  With this route, used in aortic valve replacements where the aorta is open, maintenance doses of cardioplegia are administered through handheld ostial cannulas.
- *Retrograde cardioplegia delivery*
  Administration of cardioplegia agents through this route is accomplished by inserting a cannula retrograde into the coronary sinus and delivering the cardioplegia solution backward through the coronary sinus into the coronary venous tributaries. Retrograde delivery is used in operations where antegrade infusion is not possible, such as in coronary artery bypass graft operations when there is severe narrowing or blockage of coronary arteries with poorly developed collaterals. This route of delivery is often used also in aortic valve surgery, in minimally invasive aortic or mitral valve surgery, or in cases of moderate to severe aortic valvular insufficiency. Retrograde cardioplegia might deliver more uniform cardiac arrest and even cooling. A percutaneously inserted coronary sinus catheter also can be used for cardioplegia delivery. This catheter is inserted through an introducer placed in the internal jugular vein by the anesthesiologist using TEE guidance or fluoroscopy.
- Direct vein graft delivery of cardioplegia
  During coronary artery bypass graft operations, cardioplegia solution can be delivered into the proximal end of the vein graft once the distal anastomosis is complete. This timing allows for the cardioplegia agent to be delivered to a previously ischemic area of the myocardium; for determining how well blood flows through the new vein graft attached to a coronary artery; and for assessing the quality of the new anastomosis by pressurizing the graft and looking for leaks that will require correction during CPB.

Types of Cardioplegia Solutions

There are many types of cardioplegia solutions. These are discussed in Chap. 14.

Left Ventricular Vent/Suction Cannulas

During CPB, intracardiac blood that is not captured by the venous cannula must be removed to prevent distention of the heart and rapid myocardial rewarming, as well as provide a dry surgical field (Fig. 13.7).

- Dual-functioning aortic root vent/antegrade cardioplegia cannula
  This cannula is inserted just distal to the aortic valve in most CPB cases, with sharing of the same catheter as the antegrade cardioplegia cannula. After antegrade cardioplegia is terminated, suction is applied through tubing connected to one of the CPB pumps to remove any blood that may have accumulated in the aortic root during the operation.
- Right superior pulmonary vein vent cannula
  The LV vent catheter is inserted into the right superior pulmonary vein and advanced into the left ventricle across the mitral valve, where it resides and continually pulls blood into the perfusion circuit. There are a variety of right superior pulmonary vein vent cannulas: straight, preformed, or malleable PVC tubes with depth markings. The catheters have a flexible or stiff guidewire introducer for easy insertion and placement.
- LV apex vent cannula
  This cannula is inserted through the LV apex via a stab wound and secured with a purse-string suture.
- Pulmonary trunk cannula
  This drainage cannula, called a "pulmonary artery vent," has the shape of a Swan-Ganz catheter. The anesthesia provider places it through a large introducer

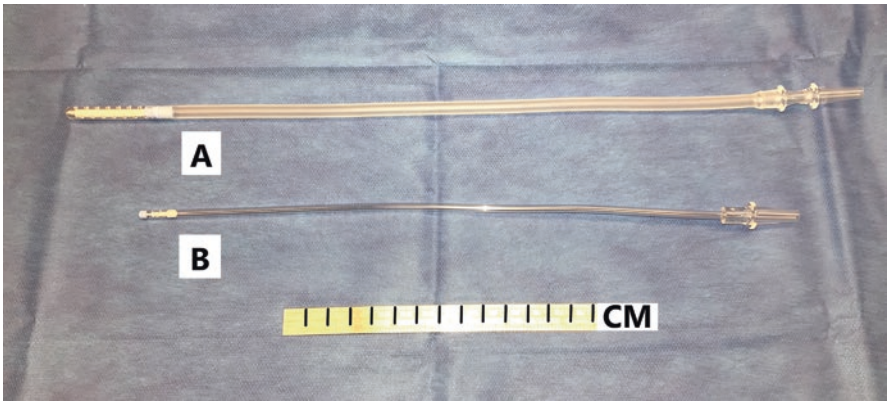


Fig. 13.7 Two types of "vents" or suction cannulas are shown. These are commonly relatively soft and multiorificed in their distal portion to maximize their draining capabilities

inserted into the internal jugular vein and floats it into the main pulmonary artery, guided by invasive pressure monitoring and TEE.

The causes of blood accumulation to the heart on bypass are:

1. Bronchial venous return blood emptying into the left atrium via the pulmonary veins rather than into the right atrium from the azygos veins
2. Thebesian veins, which are tiny valveless veins in the walls of the heart, draining directly into the chambers of the heart
3. Aortic regurgitation from the aortic root to the LV because the aortic clamp is not entirely clamped
4. Presence of patent ductus arteriosus, LSVC, atrial septal defect, or ventricular septal defect

1. BLOOD PUMPS and Components of the Heart Lung Machine

Two basic types of pumps in the circuit are roller pumps and centrifugal pumps.

(a) The roller pump (Fig. 13.8) is a positive-displacement pump that usually uses two rotating arms to compress the tubing in a counterclockwise fashion, propelling the blood forward. One of the roller pumps usually is reserved for the pump

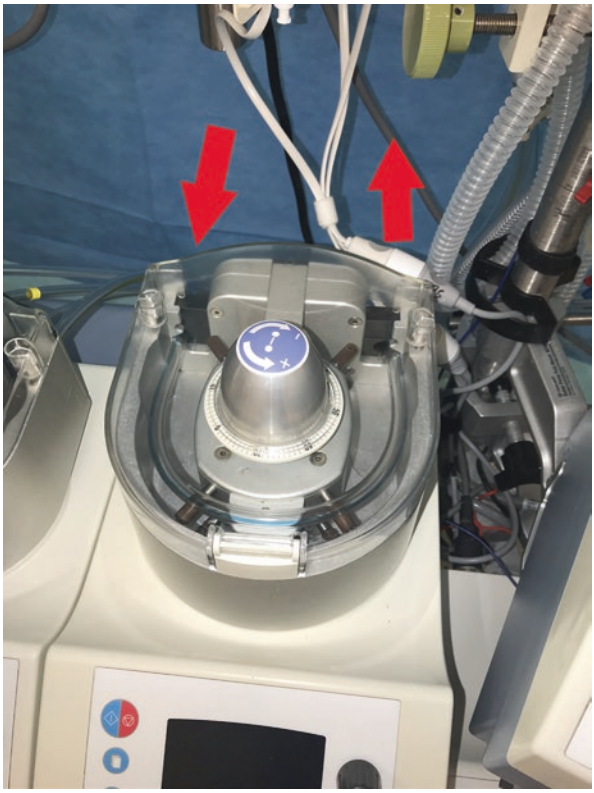


Fig. 13.8 Roller pump. Note arrows depicting direction of blood flow

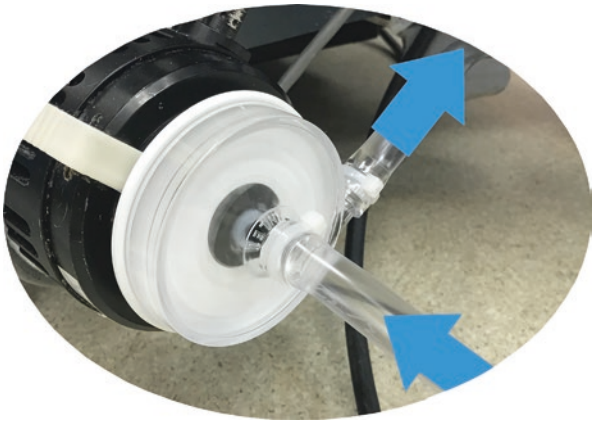


Fig. 13.9 Centrifugal pump. Note arrows depicting direction of blood flow

sucker, which is a handheld suction catheter used to suck blood that has accumulated in the surgical field back to the pump.

(b) The centrifugal pump (Fig. 13.9) uses a magnet to rotate internal fans at a given RPM to propel blood forward. Centrifugal pumps produce a nonpulsatile pump flow, which is measured in liters per minute. The nonocclusive centrifugal pump is afterload-dependent; as such, it must overcome the patient's systemic vascular resistance and resistance within the circuit to maintain forward flow. It does not pump air.

2. Cardiotomy Reservoir
   The cardiotomy reservoir (Fig. 13.10) is both the collection chamber and filter for blood drained from the patient. The blood is first filtered in order to trap any debris or particulate matter from it. The hard-shell cardiotomy reservoir used in most cases is termed an "open system," as it is open to air, allowing air entering the circuit to rise and escape into the atmosphere. A substance called Antifoam A®, placed in the collection chamber, prevents foaming in the reservoir due to the blood–air interface. The typical reservoir used for operations on adults can hold up to 5 liters of blood.
3. Oxygenator
   The oxygenator serves as the artificial lung of the perfusion circuit (Fig. 13.11). There are two types of oxygenators: bubble and membrane. Bubble oxygenators have a direct blood–gas interface. All oxygenators currently in use are membrane oxygenators. The oxygenator consists of a network of polypropylene hollow fibers that permit gases to diffuse between the gas and blood compartments. A mixture of oxygen and room air is delivered into the middle of these fibers (with the blood passing by on the outside), which remove CO_2 and add O_2. The color of the dark venous blood entering the oxygenator changes to the bright red of oxygenated blood on exiting. This color change should occur as soon as CPB is initiated, confirming that the patient's respiration is being supported. Most oxygenators contain an integrated arterial filter with a purge line, which is the last means of filtering the blood before it enters the patient's systemic arterial circulation.

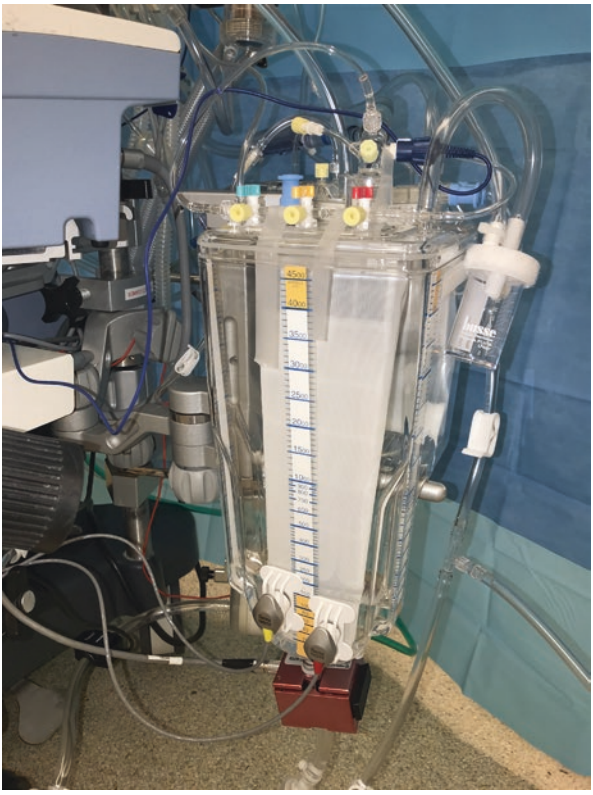


Fig. 13.10 Cardiotomy reservoir. Blood enters on the top and is suctioned from the bottom by a roller pump or, more commonly, by a centrifugal pump that pushes it through the oxygenator and into the patient

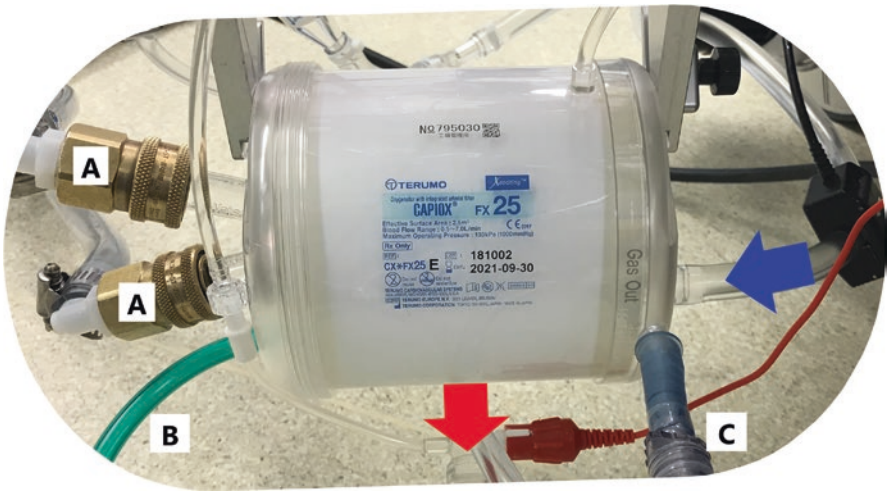


Fig. 13.11 Oxygenator / heat exchanger. BLUE ARROW: venous blood being pumped into the oxygenator. RED ARROW: blood exiting the oxygenator after gas exchange has occurred. (**a**): connectors carry and take water used for heat exchange. (**b**): green tube delivers breathing gases and inhaled anesthetics. (**c**): waste gases outlet connected to scavenger

4. Heat Exchanger
   The heat exchanger can either heat or cool the blood throughout the operation by circulating water at a set temperature, which changes temperature, by conduction, to regulate the patient's temperature. Heat exchangers are always incorporated as an integral part of the disposable CPB oxygenator. Cooling the blood during most of the bypass operation reduces the patient's metabolic demands; for every 1° C drop in temperature, metabolic demand is reduced by 7%. Mild hypothermia of 32–34° C is routinely used for cardiac surgery. During active rewarming, gradients greater than 10°C can lead to gas bubbles released into the blood and should be avoided. For this reason, the heat exchanger is located proximal to the oxygenator; overheating the arterial blood to more than 37°C can cause brain damage.
5. Arterial Filter
   An important safety feature in every CPB circuit is the arterial filter, which is the last means of filtering the blood before it enters the patient's systemic arterial circulation.
6. Air/Oxygen Blender

Gas blenders are used to accurately control and monitor the gas flow to the oxygenator, as well as adjust the proportion of oxygen from the oxygen/medical air mixture which is used to supply the oxygenator, and required for the blood oxygenation.

An oxygen analyzer should be placed in the gas supply line to measure the $Fi0_2$ of the gas mixture. Sweep gas flow refers to the gas flow passing through the membrane oxygenator, which functions to eliminate CO_2, while simultaneously oxygenating the blood passing along the membrane, according to partial pressure gradients of given gases (in this case CO_2 and O_2). The higher the sweep gas flow, the more CO_2 is blown off by the oxygenator, thus reducing the pCO_2 of the arterial blood gas. Waste gas from the oxygenator is scavenged to an outlet connected to wall vacuum. Oxygenators are constructed with multiple gas exhaust holes to avoid pressure buildup. Serial arterial blood gas measurements, generally performed every half hour, are used to adjust the $Fi0_2$, the flow rate of the sweeping gas, and electrolyte and hematocrit balances.

7. Bubble Sensor
   The bubble sensor is a safety device that is routinely placed before the arterial pump; it detects air emboli and triggers an audible alarm.
8. Low-Level Sensor
   The low-level sensor on the CPB reservoir is used to signal alert when the cardiotomy reservoir volume falls below critical level, usually between 300–400 ml.
9. Pressure Transducers
   Current-generation CPB machines have electronic alarms to prevent overpressurization of the circuit components. One transducer is on the arterial line, the other on the cardioplegia circuit. These transducers are often servo-regulated to shut off the pumps if pressures exceed a set limit.
10. Flow Probe
    An ultrasonic flow probe is necessary to determine flow from a centrifugal pump. The flow probe is routinely placed on the arterial line exiting the oxygenator to monitor the flow.

11. Vaporizer
    Often the CPB machine has an anesthetic-agent vaporizer added to the gas circuit. Thus, it can deliver inhaled anesthetic (commonly isoflurane or sevoflurane) to keep the patient anesthetized during the CPB. Another means of maintaining the patient "asleep" during the CPB is the administration of intravenous anesthetics, commonly propofol, through an infusion pump connected to the CPB machine or given by the anesthesiologist. The anesthesia team must maintain satisfactory concentrations of other anesthetic agents, such as muscle relaxants and opioids, throughout the operation.
12. Hemoconcentrator Filter
    A hemoconcentrator filter can be added to any perfusion circuit. It removes plasma fluid and solute. Using a hemoconcentrator is often termed "ultrafiltration" because it filters out plasma-free water and other solutes. It is commonly used to reduce fluid overload and increase hemoglobin concentration. Another frequent use of the hemoconcentrator is removal of potassium ions in cases of hyperkalemia from cardioplegia administration. This practice is called zero balance ultrafiltration and is achieved by adding normal saline to the reservoir and practice continual ultrafiltration. The hemoconcentrator also filters heparin and bicarbonate, thus attention is required to maintain adequate levels of these substances.

Patient Monitoring

Besides managing the CPB, the perfusionist must monitor the patient's hemodynamics and react accordingly under the direction of the cardiac surgeon and anesthesiologist.

The adequacy of perfusion is assessed by observing blood pressure, blood saturation, urine output, and blood gas values. Once the patient is on CPB, mean arterial pressure is kept at 60–70 mm Hg and is treated with vasopressors or increasing pump flow if hypotension occurs. Mixed venous saturation is expected to be in the 75–85% range during mild hypothermia. Cerebral oximetry can also be monitored to trend head oxygen saturation throughout the case, with audible alarms in place for deviations from baseline. Acceptable urine output of patients on CPB is 1–3 ml/kg/hr. and represents adequate tissue perfusion in the absence of renal dysfunction. Arterial blood gases are measured every 30 minutes to ensure the patient is stable from the respiratory and metabolic standpoint. TEE is a vital monitor during most of the CPB. It is used to guide the placement of cannulas, catheters, and vents as well as to monitor ventricular distention and causes of poor perfusion. TEE is also used to assess the severity of atherosclerosis, myocardial contractility, and the sufficiency of de-airing at the conclusion of CPB.

Extracorporeal Circuit Setup

The first step for the perfusionist in setting up and assembling the extracorporeal circuit is communicating with the surgeon and the anesthesia provider about the planned operation and its execution. The perfusionist should consider the patient's size and condition before assembly of the CPB machine since these factors will determine the choice of equipment needed for the individual patient. Next, the perfusionist chooses the appropriate bypass circuit and opens the disposable items,

placing them together under aseptic conditions to assemble a functioning CPB circuit, and performs circuit debubbling by priming the extracorporeal circuit.

Priming

Crystalloid Priming

Prime is a solution that fills the CPB circuit and its components. The circuit requires 1.5 to 2.0 L of a balanced electrolyte solution, such as lactated Ringer's, Plasma-Lyte A®, or Normosol R®, to exclude air from the circuit and test the integrity of the circuit and many of its sensors. The perfusionist can add medications to the pump as needed: colloid to increase osmotic pressure; packed red blood cells to treat anemia below a hematocrit of 20%; mannitol to stimulate osmotic diuresis; heparin to make certain that the ACT value is appropriate; and corticosteroids. With the initiation of CPB, the prime solution added to the patient's blood will lead to hemodilution; a hematocrit of 21–25% is not uncommon.

Autologous Priming

- Retrograde autologous priming. This kind of priming is used to replace up to 1 L of crystalloid from the CPB circuit to reduce the prime volume, thereby decreasing the extent of hemodilution. The perfusionist will allow the patient's blood to drain from the arterial line to fill the CPB circuit. The anesthesia provider usually gives boluses of vasopressors to maintain hemodynamic stability during the retrograde autologous priming.
- Antegrade autologous priming. This kind of priming refers to displacing some of the crystalloid priming solution into an IV bag connected to the circuit, while the venous blood drains from the venous cannula into the pump.
- For patients with poor kidney function, priming may be used less often or avoided altogether, in order to prevent the development of hypovolemia. The use of pressor agents to compensate for the developed hypovolemia might worsen kidney function postoperatively.

The Conduct of CPB

Anticoagulation

The conduct of CPB starts with systemic anticoagulation. Proper anticoagulation is essential to prevent thrombosis in the pump-oxygenator system. Heparin is given through a central line after drawing blood and prior to cannulation at a dose of 300–400 units/kg to ensure a functioning line. An ACT is conducted 3 minutes after administration of heparin to ensure that the patient is adequately anticoagulated. A value of more than 300 seconds is required prior to cannulation, and an ACT value of greater than 400 seconds is adequate for the institution of CPB; however, an ACT of greater than 480 is essential for hypothermic CPB. The pump prime contains 3 units of

heparin per milliliter of prime. Additional heparin is given to maintain the ACT greater than 480 seconds during hypothermic CPB. The ACT is verified every 30 minutes. Heparin resistance occurs when patients are deficient in antithrombin III as a result of prior heparin treatment. Giving higher doses of heparin can correct the heparin resistance. However, in refractory heparin resistance, 1000 units of antithrombin III concentrate, may be administered. Another option is to give fresh-frozen plasma, 2 to 4 units.

In patients with established or suspected heparin-induced thrombocytopenia, alternative anticoagulants such as direct antithrombin (bivalirudin) or argatroban can be used in CPB, however post-CPB bleeding is a risk with these agents. A recently reported anticoagulation strategy is to use Cangrelor, an intravenous antiplatelet agent in combination with heparin for optimum anticoagulation during CPB. Cangrelor is discussed in the pharmacology chapter.

Protocol for bivalirudin:

- 50 mg bivalirudin in the prime
- loading dose 1 mg/kg plus 2.5 mg/kg/h continuous infusion
- at initiation of CPB, continuous infusion of bivalirudin at 2.5 mg/kg/h directly into venous line
- avoid stagnation of blood
- at least 2.5 × baseline ACT
- No hemofiltration during CPB

Dividing the Lines

The perfusionist will hand out the sterile arterial and venous lines to the operating table. The lines arrive as one unit to allow for recirculation of the prime and de-airing. After the pre-bypass checklist is completed, the lines are clamped and divided at the operating table; one end will be connected to the arterial cannula, the other to the venous drainage cannula. The lines are usually color coded (red = arterial; blue = venous) to reduce the chance of error

DRUGS GIVEN ON CPB:

- Heparin
- Anesthetic agents
- Vasopressors
- Sodium Bicarbonate
- Mannitol
- Magnesium / Calcium
- Albumin

Cannulation

The surgeon will insert numerous cannulas throughout the operation, as discussed above. After the arterial and venous cannulas are inserted (Fig. 13.12) and connected to the circuit, CPB can begin.

Commencing CPB

The perfusionist slowly decreases the venous occlusion, allowing venous blood to drain from the patient into the venous reservoir while increasing the RPM of the main pump to allow arterial flow to the patient. After full flow is achieved, the anesthesia provider is notified to stop the ventilator and confirm that the adjustable pressure-limiting (APL) valve of the anesthesia machine is completely open, to prevent lung inflation.

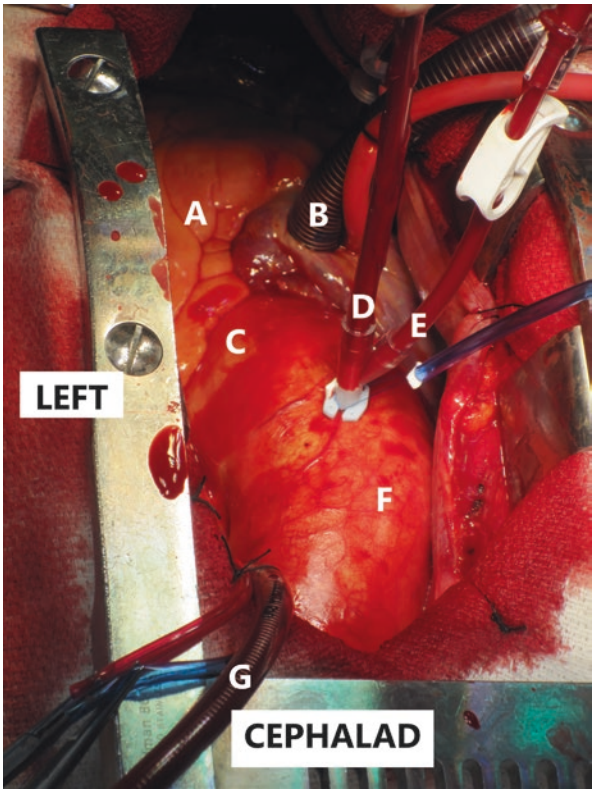


Fig. 13.12 Median sternotomy heart cannulation. (**a**): right ventricle. (**b**): triple-stage venous drainage cannula placed through the right atrial appendage. (**c**): aortic root. (**d**): aortic root cannula, this port is used to give root cardioplegia. (**e**): aortic root cannula, this port is used to "vent" (drain) the aortic root. (**f**): ascending aorta. (**g**): arterial cannula

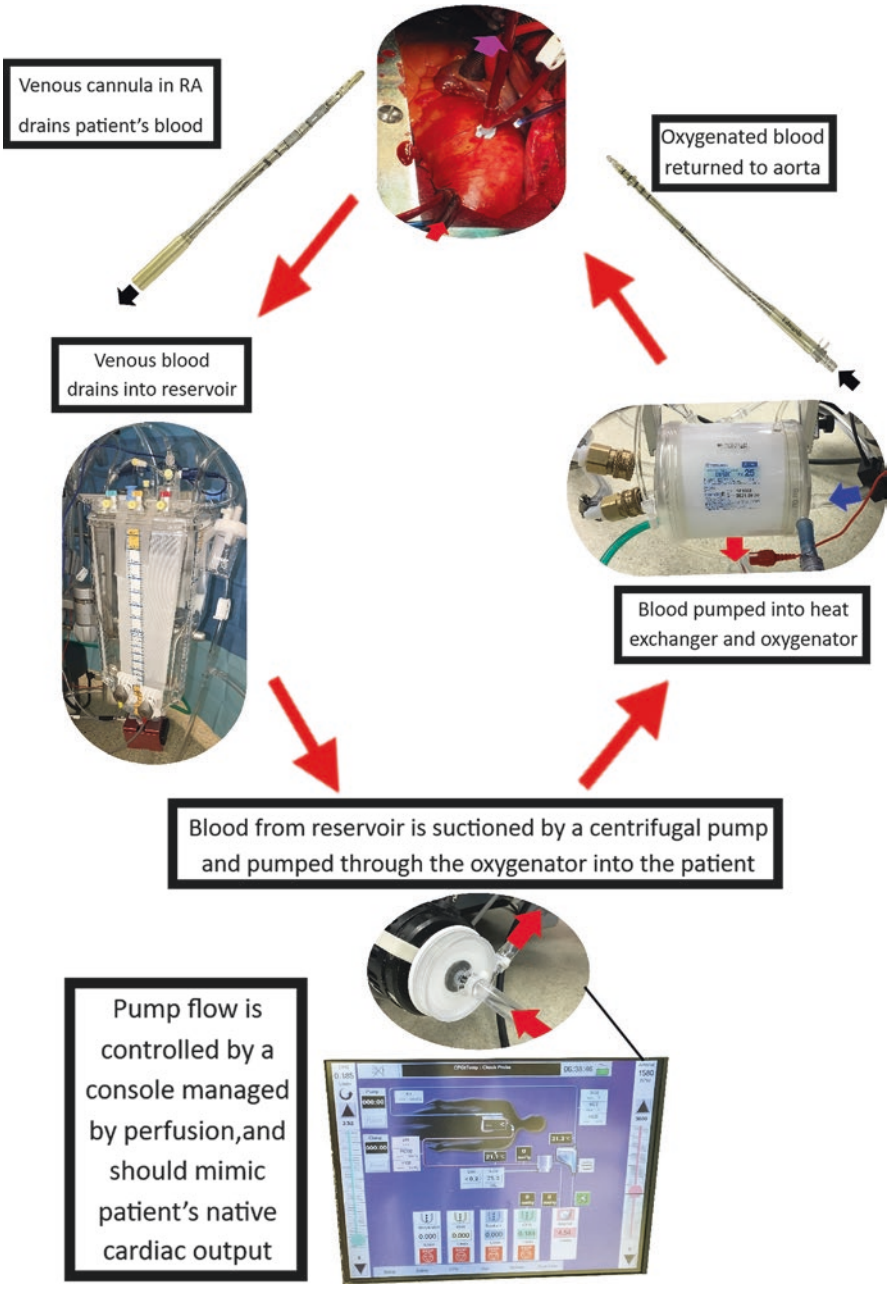
Arresting the Heart

After the cross clamp is applied to the ascending aorta (proximal to the arterial cannula but distal to the aortic root cannula), thus separating the systemic arterial circulation from the coronary circulation, cardioplegia agents can be delivered selectively to the intended myocardial tissue. Arresting the heart allows the surgeon to operate on a motionless organ, making operation technically easier as well as protecting the myocardium during the cross-clamping period of the operation. Depending on the type of cardioplegia solution used and the length of the operation, cardioplegia solution is often re-dosed.

Weaning from bypass will be discussed in another chapter.

The Cardiopulmonary Bypass Machine Circuit in Brief

The pathway that blood follows through the body in CPB starts at the venous cannula, where the deoxygenated blood is diverted from the right atrium and drained through the venous tubing into the cardiotomy reservoir. The blood is then filtered through a ~100-micron filter to ensure that no particulate matter or emboli flow to the patient. The open cardiotomy system allows for any entrapped air to escape to the atmosphere rather than continuing through the circuit. The filtered blood collects in the reservoir and is pulled from its lowest port into the centrifugal pump head, which is the main pump and the driving force of the CPB circuit. The perfusionist controls the pump flow rate and regulates revolutions per minute (RPM), to mimic the patient's native cardiac output. The blood is then propelled into the heat exchanger, where it is cooled or warmed, depending on the stage of the procedure. The blood is then directed into the oxygenator, which acts as the artificial lung for the circuit. There, CO_2 is swept out of the blood and O_2 is delivered. The pO_2 of the blood after oxygenation is usually >300 mm Hg. The oxygenated blood is passed through an arterial filter to prevent emboli from traveling further downstream. Once the oxygenated blood leaves the oxygenator, it is delivered to the patient's aorta via the arterial cannula.


Venous cannula in RA drains patient's blood
Oxygenated blood returned to aorta
Venous blood drains into reservoir
Blood pumped into heat exchanger and oxygenator
Blood from reservoir is suctioned by a centrifugal pump and pumped through the oxygenator into the patient
Pump flow is controlled by a console managed by perfusion,and should mimic patient's native cardiac output

Chronology of Events Leading to CPB

GREEN TEXT = Anesthesia Tips and clinical Pearls and Reminders!!!

1- Make surgical incision for exposure
- Sternotomy (Open Cases)
- Right Anterior Thoracotomy (Mini AVR)
- Right Thoracotomy (Mini MVR)
- Left Thoracotomy (MIDCAB)

2- Give Heparin (before cannulation)
- 300 units/kg bolus

3- Check ACT in 3 minutes
- Must be > 400 seconds before cannulation
- Must be > 480 seconds before CPB

Arterial Cannulation - means to returnoxygenated blood to the patient after the heart

Cannulation Sites:
- Ascending Aorta
- Femoral Artery
- Subclavian Artery

Retrograde Autologous Priming (RAP)

- Once arterial cannulation is complete:
- Drain blood from the patient backwards through the arterial cannula to displace the pump circuit of crystalloid
  - Reduces the effect of hemodilution due to priming the CPB circuit with Plasmalyte
- Anesthesia must give a bolus of Phenylephrine to get the SBP ~120 to allow the perfusionist to remove blood from the patient

Venous Cannulation - a means of draining the patient's systemic venous blood to the CPB Machine

Cannulation Sites:
- Right Atrium
- SVC/IVC
- Femoral Vein

Antegrade Autologous Priming (AAP)-

- Once venous cannulation is complete:
- Allow patient's blood to displace the crystalloid in the venous line reducing hemodilution
  - Need another bump in pressure to tolerate

Cardioplegia Delivery Setup:

- Antegrade delivery (into the Coronary Ostia via the Aortic Root)
  - ✓ Uses a Y cannula to act as a cardioplegia delivery cannula and an aortic root vent to drain excess blood from the heart, preventing distention
- Retrograde delivery (via the Coronary Sinus)
  - ✓ Delivers cardioplegia backwards through the cardiac venous system
  - ✓ Used in some AI, valve replacement, and CABG cases

TEE View of the Coronary Sinus

- Surgeon will ask echocardiographer for a view of the coronary sinus on the TEE to check if the catheter is in the proper position and to check for LV distention when the LV is not visible (such as in cases of minimally invasive surgery)

Initiation of Cardiopulmonary Bypass: perfusion will open both venous and arterial lines to drain the systemic venous return into the cardiotomy reservoir and start perfusing the patient

- Decompresses the heart -seen by flat arterial waveform tracing
- Increases pump flow to take over by providing cardiac output and systemic, perfusion –When perfusion states ***"Full Flow"*** it means they are providing all of the cardiac output

"Lungs off"

- Shut the Ventilator OFF once the CPB is at "Full Flow" because no blood is going to the lungs and CPB becomes the artificial lung

Further Reading

Kyriakos Anastasiadis. Principles of miniaturized ExtraCorporeal circulation from science and technology to clinical practice. 2013.
Gravlee GP. Cardiopulmonary bypass and mechanical support: principles and practice fourth edition. 2016.
Ghosh S. Cardiopulmonary bypass. 2nd ed; 2016.
Hessel EA II. What's new in cardiopulmonary bypass. J Cardiothoracic Vascul Anesthes. 2019.
Iaizzo PA. Handbook of cardiac anatomy, physiology, and devices. 2015.
Mongero LB. On bypass: advanced perfusion techniques. 2010.

Myocardial Preservation During Cardiopulmonary Bypass

14

Scott R. Coleman and Michael S. Green

Guard your heart, mind and time.

—Andrena Sawyer

Introduction

Myocardial injury occurs in almost all patients undergoing cardiac surgery with cardiopulmonary bypass (CPB). Minimizing the injury and limiting damage to reversible defects are the primary goals of the cardiac surgery team. "Stunned" myocardium can recover, but tissue necrosis cannot. The myocardium may be injured at any time during the perioperative period, but inadequate preservation during CPB is the most common cause.

Strategies to Protect the Heart for the Cardiac Surgery Patient

The perioperative period of cardiac operations has four phases: preoperative, prebypass, bypass, and pre-separation of bypass. The ideal strategy to protect the patient's heart during the perioperative period depends on the cardiac pathophysiology and the type of operation performed.

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Preoperative Period

Optimization of myocardial oxygen supply/demand is as important preoperatively as it is intraoperatively. Measures to optimize oxygen supply may include the use of intravenous nitrates to improve coronary blood flow, anticoagulants to diminish thromboembolic risk, and intra-aortic balloon pump if medical therapy is not successful.

Pre-bypass Period

During this period, the anesthesia provider should take measures to increase myocardial blood supply and decrease demand. Even allowing the patient to move to the operating table can increase demand and trigger myocardial ischemia. Measures that can be taken to shield against ischemia and protect the myocardium include preemptively avoiding pain, tachycardia, and severe hypertension, with increased afterload and wall tension during the periods of increased cardiac demand (incision, sternotomy, and aortic manipulation).

Bypass Period

This is the period when the major injury to the myocardium occurs. The current gold standard for myocardial protection during the bypass period is the use of cardioplegia. This chapter will cover all aspects of that period.

Pre-separation of Bypass Period

Steps that can be taken to prevent myocardial damage from occurring during the period of rewarming and weaning before separation from bypass will be discussed later in the chapter.

Pathophysiology of Injury during CPB

To optimize operating conditions during cardiac surgery, blood flow to coronary arteries is markedly reduced. Cross-clamping of the aorta interrupts blood flow to the coronaries entirely, resulting in a drastic decline in oxygen supply. A mismatch of oxygen supply and demand leads to cardiomyocyte ischemia, high-energy phosphate substrates are exhausted, intracellular calcium levels rise, and increased anaerobic metabolism leads to acidosis. Although a safe duration of cross-clamping has not been defined, the risk of ischemic injury clearly increases with longer durations of clamping. Releasing the cross-clamp also has a risk: reperfusion injury is a primary concern. In reperfusion, the release of free radicals and radical oxygen species creates oxidative stress for cardiomyocytes, and cellular edema and excessive intracellular calcium also contribute to injury. Embolism of fat, air, or thrombi into coronary arteries may occur after releasing the cross-clamp.

Risk Factors for Injury

There are a few patient-specific risk factors that correlate with the potential for myocardial injury: poor left ventricular function, left ventricular hypertrophy, and diffuse coronary artery disease.

Strategies to Minimize Injury and Limit Damage to the Myocardium

Cardioplegia

The primary goals of cardioplegia are reduction in cardiac energy usage and preservation of high-energy substrates. Cardioplegia is administered after aortic cross-clamping. The constituents of cardioplegia solutions vary among institutions, but the primary element is high dose of potassium. Potassium-rich solutions induce membrane hyperpolarization, which causes flaccid diastolic arrest of the heart. Pharmacological arrest of both electrical and mechanical function of the heart is the most impactful method to decrease oxygen consumption, which can be reduced by as much as 90%.

Delivery of Cardioplegia

Cardioplegia can be administered in antegrade or retrograde fashion. Using a combination of both routes provides the most complete myocardial protection. Cardioplegia delivery techniques are discussed in Chap. 13.

Depending upon the duration of CPB, cardioplegia often needs to be re-dosed. Over time, rewarming and washout of the cardioplegia occur. Re-dosing helps maintain an isoelectric state and removal of metabolites that may inhibit anaerobic metabolism. Cardioplegia can be given via continuous infusion, but more commonly additional doses are administered intermittently (about every 30 minutes).

Constitution of Cardioplegia

As mentioned above, the optimal composition of cardioplegia has not been determined and varies among institutions; numerous ingredients have been tried. Potassium is a universal ingredient, but the doses vary, from 10 to 40 mEq/L. A common approach uses higher doses (up to 40 mEq/L) at the start of cardioplegia and smaller doses (10 mEq/L) for subsequent doses. This prevents dangerous hyperkalemia from developing during weaning from bypass. Sodium is often included at a dose of less than 140 mEq/L. This low dose is chosen because ischemia causes blood sodium concentrations to rise. Magnesium can be added to help antagonize the high amounts of calcium that are expected. Common energy sources that may be added are glucose, glutamate, and aspartate. The addition of mannitol may help to scavenge free radicals and reduce cell edema. The buffers bicarbonate or tromethamine can be used to neutralize the acid that is produced during CPB.

Cardioplegia can be administered in a crystalloid-based solution, a blood-based solution, or a combination of the two. Available data do not show a superiority of

either delivery method. However, the most common technique in the United States is a combination of crystalloid and blood.

1. *Cold crystalloid cardioplegic solutions*:
   Crystalloid cardioplegia broadly consists of two kinds of solutions, which are designated intracellular and extracellular, based on their concentrations of sodium and calcium ions. Intracellular solutions contain no or low concentrations of sodium and calcium, while extracellular solutions contain higher concentrations of sodium, calcium, and magnesium. Custodiol (also HTK or Brettschneider's) solution is an example of an intracellular solution, in which the sodium concentration is 15 mEq/L. St. Thomas' II solution (Plegisol Pfizer, Inc.) is an example of an extracellular solution, in which the sodium concentration is 110 mEq/L.
2. *Blood cardioplegia*:
   Blood cardioplegia can be either warm or cold. Cold blood cardioplegia uses blood hyperkalemic solution: Autologous blood from the extracorporeal circuit is diluted with crystalloid solution. Buckberg solution is mixed with crystalloid at a blood to crystalloid ratio of 4 to 1, whereas del Nido solution (Baxter Healthcare) (Fig. 14.1) has crystalloid to blood ratio 4 to 1, where a dose of 20 ml/kg will provide myocardial protection for 90 min.

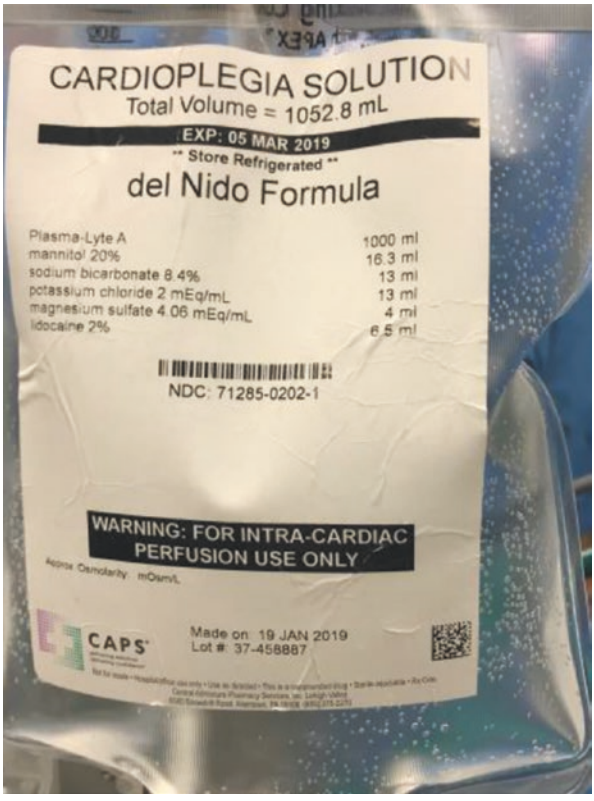

Fig. 14.1 Image of del Nido crystalloid cardioplegia solution bag (Baxter Healthcare)

Prevention of Ventricular Tachyarrhythmias

Heart rate is the most important determinant of cardiac oxygen consumption. Therefore, tachyarrhythmias are a potential source of significant oxygen demand. Ventricular tachyarrhythmias frequently occur during cardiac surgery and should be recognized and treated promptly. Effective use of cardioplegia helps prevent malignant arrhythmias. When such arrhythmias occur, defibrillation should be immediately applied, and anti-arrhythmic medications, such as lidocaine and amiodarone, can be used.

Cardiac Decompression and Left Ventricular Venting

During CPB, blood can enter the left ventricle via shunts, aortic insufficiency, Thebesian veins, or bronchial circulation. Cardiac distention and increased temperature occur if blood from these sources is not removed. As distention of the ventricle occurs, the chamber enlarges and myocardial wall tension increases. Increased wall tension raises oxygen requirements for the tissues and limits subendocardial blood flow. To prevent these untoward events, blood is vented into the reservoir of the CPB system. Additional benefits of venting are de-airing of the ventricle and reduction in left atrial and pulmonary vein pressures.

Hypothermia

Hypothermia protects the heart and brain during periods of decreased perfusion. A decline in core body temperature of 1 degree Celsius reduces the cardiac metabolic rate by about 8%. As the metabolic rate decreases, oxygen consumption declines and substrates for energy are better preserved. Hypothermia can be classified as mild (32–35 °C), moderate (28–32 °C), and deep (< 28 °C). The protective benefits of mild and moderate hypothermia are similar to deep hypothermia. However, deep hypothermia has associated increased risk of arrhythmias, coagulopathy, infection, and heart failure. Although hypothermia has traditionally been used to preserve cardiac and brain function, studies comparing hypothermia and normothermia during cardiac surgery have found no difference in mortality or morbidity; in fact, lower rates of transfusion of blood products have been reported with normothermia. When hypothermia is used, mild and moderate cooling is used more often than is deep cooling. Deep hypothermia is used primarily for cerebral protection in cases of circulatory arrest. Hypothermia can be either active or passive. Active cooling is achieved with the CPB machine. Passive cooling can be achieved with cold cardioplegia or instillation of ice slush directly in the thoracic cavity.

Shivering

One of the consequences of hypothermia is shivering. Shivering generates a large metabolic requirement and a mismatch of oxygen supply and demand in the heart, so it should be prevented. Preventing shivering is especially important for patients

undergoing coronary artery surgery. The use of non-depolarizing muscle relaxants during surgery can prevent shivering; recently, use of dexamethasone also has been suggested.

Surface Myocardial Cooling

Topical cooling by bathing the heart with ice slush has been used as an adjunct for myocardial protection. Although the cardioprotective benefit of this technique is questionable and possibly ineffective, many surgeons still use it, even though it may be associated with an increased incidence of phrenic nerve cold injury that leads to postoperative pulmonary complications and diaphragmatic paralysis.

Rewarming and Weaning from CPB

The process of weaning from CPB can also be optimized to preserve cardiac tissue and improve patient outcomes. Recommended measures are as follows:

- Residual hyperkalemia from cardioplegia should be corrected.
- Warm cardioplegia or the Hotshot can be used near the end of CPB to rewarm the patient, washout metabolites of anaerobic metabolism, and provide additional high-energy substrates.
- Calcium is administered routinely following removal of the cross-clamp. Calcium directly antagonizes the myocardial effects of hyperkalemia without lowering serum potassium concentrations. However, administration should be judicious as detrimental intracellular levels of calcium commonly occur with ischemia.
- The heart should first be allowed to contract while still empty. This facilitates recovery of stunned myocardium.
- Unnecessary or excessive use of inotropes should also be avoided as they will increase myocardial oxygen demand.

Other Strategies for Cardiac Protection Without Cardioplegia

- Intermittent cross-clamp fibrillation on bypass:
  - Cardioplegia is not given, and moderate hypothermia is achieved. Fibrillation is induced by DC current, and the aorta is cross-clamped as soon as the heart is fibrillated.
- Ischemic precondition
  - It provides protection immediately following ischemia and lasts for 12 hours.
- Deep Hypothermic Circulatory Arrest (DHCA)
  - This can be performed if the aorta cannot be clamped.
- Beating heart on bypass
  - The aorta is not clamped, the coronary arteries are perfused, no cardioplegia is given, and the heart is empty and not arrested.

Failure to Chemically Arrest the Heart

Common problems causing the heart's electrical activity to persist during CPB are as follows:

- There is inadequate delivery of cardioplegia agents.
  1. Proximal right coronary artery lesion prevents flow to the sinoatrial node.
  2. Aortic insufficiency will lead to cardioplegia filling and distending LV while decreasing the flow to the coronary arteries.
  3. The cross-clamp is not completely occlusive, and the cardioplegic solution gets diluted with aortic blood.
- Defective cardioplegia solution or the plegia is not cold.
- Inappropriate washout caused by inadequate venous drainage of the heart.
  1. Venous airlock.
  2. Kinked or clamped venous line.
  3. Obstruction to the flow by tight caval snares.

Signs of Poor Myocardial Preservation

Myocardial preservation cannot be evaluated during CPB with electrocardiography or transesophageal echocardiography, as the heart is arrested. Therefore, assessment of myocardial function starts when cardiac activity resumes. Indications of poor myocardial preservation include reduced cardiac output, diminished left ventricular function, and arrhythmias.

Further Reading

1. Barry AE, Chaney MA, London MJ. Anesthetic management during cardiopulmonary bypass: a systematic review. Anesth Analg. 2015;120(4):749–69.
2. Barsh P. Clinical anesthesia, chapter 39. 8th ed; 2017.
3. Butterworth J. Morgan and Mikhail's clinical anesthesiology. Chapter 22. 6th ed; 2018.
4. Eugene A, Hessel II. What's new in cardiopulmonary bypass. J Cardiothorac Vasc Anesth. 2019;
5. Gravlee GP. Cardiopulmonary bypass and mechanical support: principles and practice. 4th ed; 2016.
6. Miller R. Miller's anesthesia. Chapter 67. 8th ed; 2015.

Blood Conservation in Cardiac Surgery

15

Scott Oldebeken and Anna E. Jankowska

All bleeding stops eventually.

—from "The House of God" by Samuel Shem

Key Points

- Complications associated with blood product transfusions
- Risk factors for blood transfusions in cardiac surgery
- Preoperative strategies to decrease blood product transfusion need
- Intraoperative strategies to minimize blood transfusions
- Postoperative strategies to decrease blood product use in cardiac surgery

Introduction

Blood product transfusions are an important perioperative tool to prevent anemia-related tissue hypoxia as well as to correct coagulopathy and prevent further bleeding. However, despite reduction in the direct harm associated with transfusions (infection, leukoreduction), there is growing evidence concerning the indirect effects associated with higher morbidity and mortality. Cardiac surgery disproportionately consumes more blood products than any other field of medicine, accounting for 20% of total US blood transfusions (Fig. 15.1).

Approximately half of all cardiac surgeries result in transfusion. Furthermore, the observation of wide discrepancies in transfusion rates among cardiac surgical centers has highlighted the need for uniform modern blood conservation guidelines.

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Fig. 15.1 Image of an example of cardiac surgery that consumed significant amount of blood products

Table 15.1 Infection risk associated with blood transfusion

Estimated risk of infection by direct transmission from blood transfusion	
HIV	1 in 1.8 million
Hepatitis B virus	1 in 220,000
Hepatitis C virus	1 in 1.6 million
HTLV-1	1 in 640,000
West Nile virus	1 in >1 million

Implementation of blood conservation strategies has been shown to decrease transfusion rates, improve outcomes, and reduce costs.

Complications

Infection

The risk of direct infection from blood transfusion has been dramatically reduced since the 1980s due to advances in donor screening and pre-transfusion blood testing. However, public stigma of transfusion-related infection often lags behind these advances in transfusion safety. It is therefore helpful to inform, and therefore comfort patients with up-to-date infection risk (see Table 15.1). While donor screening and blood testing have led to large reductions in infection transmission from donor to recipient, microbial contamination during the donation and storage process represents a more durable challenge. Transfusion-associated sepsis (TAS) accounts for approximately 10% of allogeneic blood transfusion (ABT)-related deaths in the United States. Of these, platelets account for 70% of TAS-related deaths, largely in part due to the relatively high storage temperature of 22 degrees Celsius, and product exposure to more venipunctures in the setting of pooled donor platelets.

TRALI

Transfusion-related acute lung injury (TRALI) is the leading cause of ABT-related death. The clinical signs and symptoms mimic adult respiratory distress syndrome (ARDS), but TRALI occurs within 6 hours of ABT. The pulmonary infiltrates seen in TRALI are caused by increased vascular permeability and occur in the absence of left atrial hypertension. Fresh frozen plasma (FFP) is the most frequently implicated component. The case-fatality ratio in TRALI is 5–10%. Treatment is largely supportive with 70% of cases requiring mechanical ventilation and rarely the use of veno-venous extracorporeal membrane oxygenation (VV-ECMO).

TACO

Transfusion-associated cardiac overload (TACO) presents with clinical features similar to TRALI but differs in the mechanism and treatment. In TACO, pulmonary infiltrates develop due to increased hydrostatic pressure secondary to volume overload. Physical exam, echocardiography, BNP serum levels, and response to diuretics are often helpful in differentiating TACO from TRALI.

Transfusion-Related Immunomodulation (TRIM)

ABT suppresses recipient cell-mediated immunity and has been associated with a dose-dependent increase in the risk of postoperative infection. While the exact mechanism is unclear, it is commonly hypothesized that donor WBCs present in ABT are the cause of TRIM. This hypothesis has been tested numerous times in studies comparing leukoreduced blood products to WBC-containing products, with mixed results in mortality and infection risk. There is proven benefit to leukoreduction in decreasing the incidence of non-hemolytic febrile transfusion reactions and CMV transmission. As blood banks trend towards using universally leukoreduced blood products, it has become much harder to conduct appropriately powered clinical trials comparing leukoreduced blood products to leukocyte-containing products.

Predictors of Perioperative Bleeding or Transfusion

There are many factors that can predict the risk of perioperative bleeding necessitating transfusion in cardiac surgery: age >75, female gender, low body surface area (BSA), preoperative anemia or thrombocytopenia, anticoagulation, antiplatelet medications, coagulopathy, Cr >1.3, combined CABG and valve surgery, emergency surgery, preoperative shock state or use of intra-aortic balloon pump, and prolonged duration of cardiopulmonary bypass (CPB).

Preoperative Strategies

Preoperative Anemia

Perioperative blood conservation starts with preoperative anemia screening and treatment when the operative timeline allows. Preoperative anemia correction and medical optimization is discussed in Chap. 7.

Autologous Donation

Preoperative autologous blood donation decreases ABT but increases total transfusion rate (including autologous donation). The challenge of this technique is allowing the patient enough time for hematopoietic recovery without losing donated blood quality through aging. While autologous donation has been shown to have a low cost-effectiveness in the general population, it is especially helpful in the non-anemic patient who has a rare blood type, difficult crossmatch, Jehovah's Witness, or in times of blood shortage. Contraindications for autologous donation include unstable angina and aortic stenosis, which affects a significant proportion of cardiac surgery patients.

Intraoperative Strategies

Acute Normovolemic Hemodilution (ANH)

ANH is the collection of autologous whole blood after induction of general anesthesia, with concurrent IV hydration to maintain isovolemia. As a result, autologous units are sequentially more dilute due to the ongoing hemodilution. ANH reduces hematocrit so that less RBC mass is lost during surgical bleeding. Autologous blood is reinfused, as needed, in reverse order of collection. This allows for the most concentrated unit to be transfused during the period of least blood loss (Fig. 15.2). ANH is a low-cost procedure that is relatively simple to perform and can easily be combined with other techniques like preoperative autologous donation and cell salvage. ANH has been shown to decrease exposure to ABT, and is especially suited for patients with high preoperative hemoglobin levels who are expected to experience significant blood loss.

Antifibrinolytics

Antifibrinolytic agents have been shown to decrease blood loss and transfusion rates in cardiac surgery. The commonly used agents are tranexamic acid (TXA) and aminocaproic acid (ACA). Aprotinin, another slightly more hemostatic antifibrinolytic, lost FDA approval, in 2008, after a single trial demonstrated increased mortality with its use. These agents are discussed in detail in the pharmacological blood conservation section of Chap. 3. The Society of Thoracic Surgeons (STS) and the

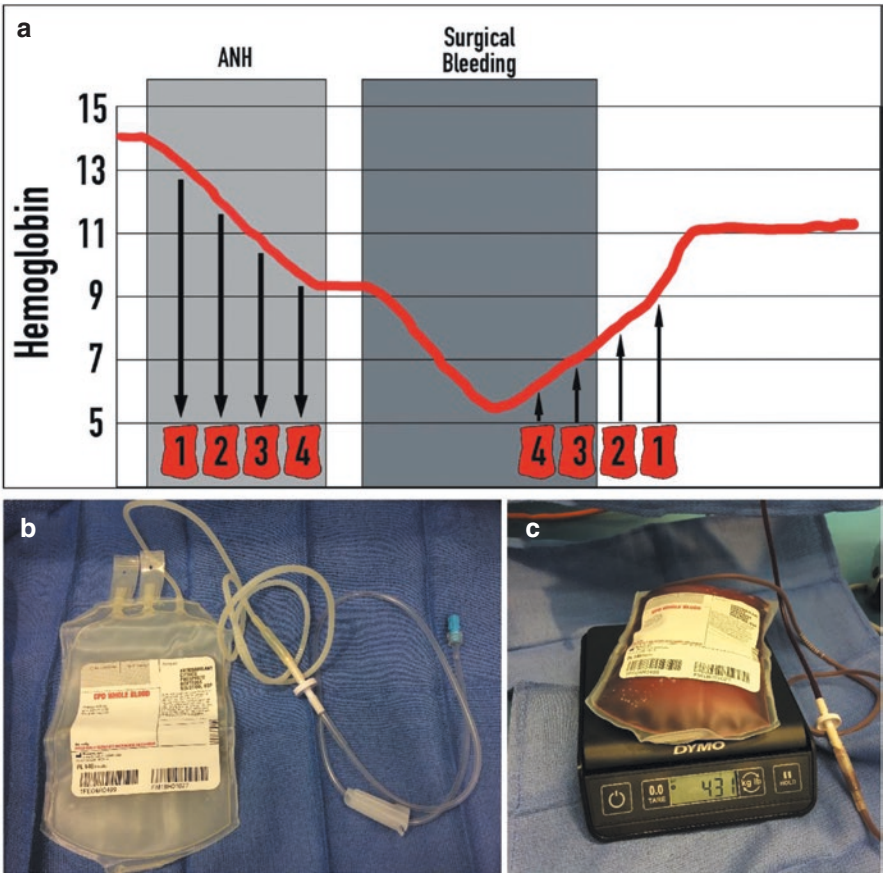

Fig. 15.2 Acute normovolemic hemodilution. (**a**) Phases of ANH during surgery. (**b**) ANH citrate collection bag/tubing. (**c**) Whole blood collection during ANH

Society of Cardiovascular Anesthesiologists (SCA) recommend the routine use of antifibrinolytics during cardiac surgery.

Surgical Factors

There are many surgical factors that influence the quantity of blood loss and ABT in cardiac surgery. The avoidance of cardiopulmonary bypass-related hemodilution and coagulopathy with surgical techniques such as off-pump CABG has been shown to reduce perioperative bleeding and ABT. Additionally, the blood loss associated with median sternotomy can be reduced with minimally invasive (MI) surgical approaches, namely, parasternal or mini-thoracotomy approaches for CABG and robotic mitral valve surgery.

Cardiopulmonary Bypass (CPB) Factors

The CPB machine ushered in the era of modern cardiac surgery, but with this essential tool come the undesired side effects of hemodilution and platelet dysfunction. Over time, these problems have been reduced, but not eliminated, by various optimizations of the CPB machine.

Retrograde autologous priming (RAP) involves replacing a portion of the CPB crystalloid-based priming volume with 400–800 mL of the patient's own blood. This reduces the CPB-related hemodilution, improves end-organ perfusion while on CPB, and reduces ABT during cardiac surgery. RAP occurs just prior to initiating full CPB and typically takes 2–5 min. Vasopressors are often administered, during RAP, to maintain adequate mean arterial pressures (MAPs).

In addition to RAP, other features of the CPB machine have been optimized to aid in blood conservation. Centrifugal CPB pumps have largely replaced standard roller pumps due to their decreased shear force exerted on RBCs and subsequent hemolysis, as well as a reduction in particulate embolization from the breakdown of the circuit tubing. Drug-coated CPB circuits have shown modest benefits in reducing allogeneic RBC transfusion through decreased systemic anticoagulation requirements (heparin-coated) and postoperative bleeding (phosphorylcholine-coated). CPB circuit miniaturization can reduce priming volumes by as much as 1 L, resulting in less hemodilution and reduced rate of ABT.

Many of the physiologic and pharmacologic changes induced for CPB, namely, hypothermia and heparinization, need to be reversed after separating from bypass in order to promote hemostasis. Hypothermia can produce a profound coagulopathy functionally equivalent to clotting factor-deficient states, despite normal factor levels. It is therefore important to achieve normothermia during the post-CPB phase of cardiac surgery in order to optimize hemostasis and prevent unnecessary ABT. Likewise, the heparinization required for CPB is reversed with protamine after separation from CPB. Protamine, when given alone or in disproportion to heparin levels, acts as an anticoagulant so it is important to avoid overdosing. There is some suggestion in the literature that protamine dosing to serum heparin level is more appropriate than blindly dosing to a fixed 1:1 ratio of the original dose of heparin, as some heparin is metabolized and/or lost to the CPB circuit, resulting in protamine overdose, preventable coagulopathy, and bleeding.

Cell Salvage

Cell saver is a cell salvage technique involving the collection, washing, and centrifugation of recovered blood. Autologous cell saver transfusion can reduce ABT, but the majority of the clotting factors and platelets are lost during centrifugation. The resultant RBC predominant autotransfusion can predispose to and perpetuate coagulopathic bleeding in some patients. Despite this pitfall, the net benefit of reduced ABT exposure was confirmed in a recent meta-analysis leading the STS/SCA to make cell salvage use a Class 1A recommendation during cardiac surgery.

Ultrafiltration is a newer form of cell salvage in which salvaged blood is exposed to a polycarbonate superadsorber capable of withdrawing plasma but holds the unique advantage of preserving *all* cell species. Once hemoconcentrated, the salvaged blood is transfused back to the patient as needed.

Point of Care Testing

CPB confers a number of unique challenges related to blood conservation. Rapid heparinization, under- or over-reversal with protamine, hemodilution, prolonged contact with CPB circuit tubing, shear stress of CPB pump, reduced coagulation factors, hyperfibrinolysis, and platelet dysfunction and consumption all contribute to CPB-related coagulopathy and anemia, increasing the use of allogeneic blood products.

Current pre- and postoperative practice involves using laboratory-based assays (platelet count, fibrinogen, Prothrombin Time (PT)/ International Normalized Ratio (INR), Partial Thromboplastin Time (PTT)) to characterize and treat coagulopathic bleeding in the nonoperative setting. However, standard laboratory-based assay turnaround is often too slow to guide intraoperative blood management. While the experienced anesthesiologist and surgeon may be able to identify the signs of microvascular bleeding in the surgical field and initiate early empiric treatment, subjective assessment will often lead to over- or undertreatment of coagulopathic bleeding. Point of care (POC) testing offers a quicker turnaround leading to more timely targeted therapies. POC glucose, hemoglobin, activated clotting time (ACT), and blood gas analysis are routinely used in cardiac surgery, but the introduction of POC viscoelastic testing (thromboelastography (TEG) or rotational thromboelastometry (ROTEM)) offers rapid assessment of clotting time, thrombocytopenia, hypofibrinogenemia, and fibrinolysis. During critical portions of cardiac surgery, this quick evaluation can guide the appropriate intervention in the form of transfusion, pharmacotherapy, or surgical revision. POC viscoelastic testing has been shown to reduce the exposure to allogeneic blood products, postoperative blood loss, and even ICU length of stay.

Restrictive Transfusion Strategy

Previously, little high-quality evidence existed to guide intraoperative transfusion strategy, resulting in a wide variability in rate of ABT among cardiac surgery centers. Accumulating evidence has challenged the prevailing view that any perioperative anemia is associated with increased morbidity by demonstrating the dose-dependent risk of ABT.

Restrictive RBC transfusion strategy (Hgb <7.5 g/dL intra- and postoperatively) has recently been shown to be non-inferior compared to liberal transfusion strategy (<9.5 g/dL intraoperatively and 8.5 g/dL postoperatively), with respect to mortality at 28 days and 6 months, as well as major comorbidities (MI, stroke, and renal failure requiring dialysis). Rate of transfusion decreased approximately 20% when employing the restrictive transfusion thresholds. It is important to note that the

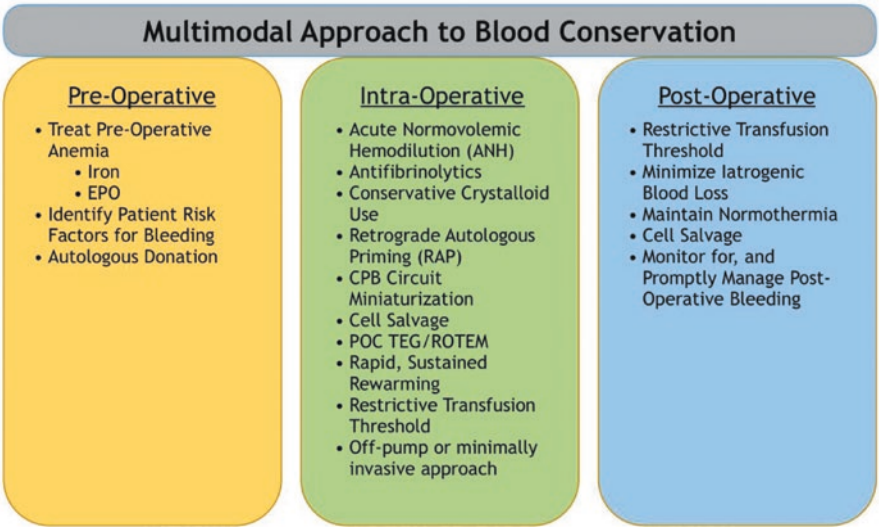

Fig. 15.3 Summary of multimodal blood conservation strategy

safety of permissive anemia has not been validated in patients with acute coronary syndrome (ACS), a significant subpopulation in cardiac surgery.

Figure 15.3 summarizes multimodal blood conservation strategy.

Further Readings

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.

Blaudszun G, Butchart A, Klein AA. Blood conservation in cardiac surgery. Transfus Med. 2018;28(2):168–80.

Carless PA, Henry DA, Moxey AJ, et al. Cell salvage for minimising perioperative allogeneic blood transfusion. Cochrane Database Syst Rev. 2006;(4):CD001888.

Deppe AC, Weber C, Zimmermann J, et al. Point-of-care thromboelastography/thromboelastometry-based coagulation management in cardiac surgery: a meta-analysis of 8332 patients. J Surg Res. 2016;203(2):424–33.

Ferraris VA, Brown JR, Despotis GJ, et al. 2011 update to the Society of Thoracic Surgeons and the Society of Cardiovascular Anesthesiologists blood conservation clinical practice guidelines. Ann Thorac Surg. 2011;91(3):944–82.

Friedman T, Javidroozi M, Lobel G, et al. Complications of allogeneic blood product administration, with emphasis on transfusion-related acute lung injury and transfusion-associated circulatory overload. Adv Anesth. 2017;35(1):159–73.

Krieger KH, Isom OW. Blood conservation in cardiac surgery. New York: Springer; 1998.

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.

Salis S, Mazzanti VV, Merli G, et al. Cardiopulmonary bypass duration is an independent predictor of morbidity and mortality after cardiac surgery. J Cardiothorac Vasc Anesth. 2008;22(6):814–22.

Sun P, Ji B, Sun Y, et al. Effects of retrograde autologous priming on blood transfusion and clinical outcomes in adults: a meta-analysis. Perfusion. 2013;28(3):238–43.

Anesthetic Management During Cardiopulmonary Bypass

16

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Introduction

In 1952, John Gibbon performed the first cardiac operation (repair of atrial septal defect) using cardiopulmonary bypass (CPB). Present-day cardiac surgery requires careful collaboration between the cardiac surgeon, cardiac anesthesia provider, and perfusionist. The mechanistic components of CPB are discussed in Chap. 13, "Fundamentals of Cardiopulmonary Bypass Machine Equipment and Technique." The events that happen during the bypass period will predict how smooth weaning from bypass will be. Paying attention to the surgical technique and monitoring the function of multiple systems will allow the anesthesia provider to detect problems that might arise and can lead to devastating outcomes. This chapter focuses on the pathophysiologic effects of CPB, the role of the anesthesia provider during the period of cardiopulmonary bypass, and the methods of providing and maintaining anesthesia during CPB, while avoiding complications.

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Pathophysiologic Effects of CPB

CPB cause a systemic inflammatory response in cardiac surgery patients. The pathophysiology of this inflammatory response is often triggered by the physical contact of the blood with the CPB circuit; endotoxemia after restoration of perfusion, which activates key components of the inflammatory response; and end-organ injury due to ischemia/reperfusion. Restoration of perfusion after release of the aortic cross-clamp can cause an inflammatory response because of activation of the complement and cytokine cascades, release of nitric oxide, activation of the coagulation/fibrinolysis cascade, and activation of the endothelium. Aortic cross-clamping and unclamping with the associated ischemia and reperfusion injury also can affect organs, including the brain, heart, lungs, liver, and kidneys.

The magnitude and duration of the inflammatory response is dependent on myriad factors, including the patient's preoperative cardiac condition and hemodynamic stability; type and duration of surgery; preoperative organ function; use of blood products (red blood cells, platelets, fresh frozen plasma, and cryoprecipitate); and the presence of diabetes mellitus. Techniques to minimize the inflammatory response and its consequences include preoperative optimization (especially for elective surgery); minimizing CPB time; use of less traumatic surgical techniques; decreasing CPB suction vacuum pressure; minimal use of blood products; maintenance of hemodynamic stability; adequate anesthesia; proper control of electrolyte balances and blood glucose; and optimizing fluid administration.

Maintenance of General Anesthesia

Although inhalation agents, provided by the perfusionist, are the preferred agents for maintaining anesthesia in CPB, the anesthesia provider is responsible for monitoring the depth of anesthesia and the adequacy of muscle relaxation. It is important to note, however, that CPB can affect the plasma concentration of the anesthetics used. The effectiveness of intravenous anesthetics can be altered by hemodilution and plasma protein binding.

In addition to the difficulty in dosing medication and volatile anesthetics during CPB because of the effects mentioned above, monitoring the depth of anesthesia also can be difficult. Tools are available for monitoring the depth of anesthesia, including a bispectral index monitor, but studies on the benefits of these devices have yielded equivocal results. Managing anesthesia by use of a balanced technique that includes opioids, benzodiazepines, volatile anesthetics, and muscle relaxants has reduced the incidence of awareness during CPB to <3%. Ensuring adequate analgesia, amnesia, and muscle relaxation on bypass is achieved by appropriate dosing of opioids, benzodiazepines, volatile anesthetics, and muscle relaxants—often in an empiric fashion. Supplemental doses usually are given with the initiation of CPB to compensate for the decrease in the level of the drug blood concentration due to acute hemodilution as well as with rewarming to make up for the increased enzymatic degradation of the drug by the liver.

Mean Arterial Pressure and Tissue Perfusion

A mean arterial pressure (MAP) of ≥65 mm Hg usually is adequate to maintain satisfactory tissue perfusion during CPB. A higher MAP may be needed in elderly, hypertensive patients, and those with a history of cerebrovascular disease. During bypass, patients may require mild support to attain the target MAP. Patients in cardiogenic shock or septic shock from endocarditis may require substantial vasopressor support with agents that increase systemic vascular resistance, such as norepinephrine or phenylephrine. Also, vasopressin can be useful in maintaining blood pressure in conditions such as vasoplegic syndrome, which is increasingly recognized with the continuation of preoperative angiotensin-converting enzyme inhibitors. On the other hand, MAP above the upper limit of cerebral autoregulation (>90 mm Hg) during CPB may require treatment, which usually consists of first increasing the depth of anesthesia and administering intravenous analgesics. If required, a vasodilator can be added.

Monitoring Adequate Tissue Perfusion

Assessment of end-organ function is used to ensure adequate tissue perfusion. Monitoring the trend in end-organ function is more important than the absolute number. The patient's underlying clinical condition and the complexity of the surgical procedure should be considered when interpreting the data. Several measurements for monitoring tissue perfusion are used: in-line venous saturation, blood gases, urine output, near-infrared spectroscopy (to measure local oxygen consumption), and blood lactate concentrations. Suggested values to ensure adequate tissue perfusion with CPB are pump flow 2.2–2.5 L/min/m, MBP 60–65 (70–80 for patients with chronic renal insufficiency, cerebral atherosclerosis, or peripheral vascular disease), hematocrit above 22%, maintaining cerebral oximetry more than 75% of pre-induction values, and mixed venous oxygen above 75%.

Temperature Monitoring and Management During Cardiopulmonary Bypass

The plan for management of patients' temperature during CPB should be communicated between the surgeon, the anesthesia provider, and the perfusionist. Whether normothermia or hypothermia is preferable has been debated extensively. Supporters of hypothermia point to the decrease in the cerebral metabolic rate of oxygen and the neuroprotection that even a small degree of hypothermia provides. Hypothermia during CPB has also been purported to protect the vulnerable myocardium, by a mechanism like that of neuroprotection. Many sites have been used for monitoring patients' temperature during CPB (pulmonary artery catheter, nasopharynx, tympanic membrane, rectum, bladder), but the recommended site is the oxygenator arterial outlet because it is a surrogate for cerebral temperature. During the cooling

phase, body-warming devices (forced-air warming blankets, HotDog warming device, and intravenous fluid warmer) are turned off. During rewarming, the temperature gradient between the oxygenator arterial outlet and the venous inlet should not exceed 10 °C to prevent the formation of bubbles and gaseous emboli. Also, faster rewarming rates have been associated with increased amounts of glial fibrillary acidic protein (a brain injury biomarker) and an increased rate of stroke.

Maintenance of Adequate Anticoagulation

Maintenance of anticoagulation is essential during CPB. Use of unfractionated heparin has been the mainstay of anticoagulation during CPB for more than 40 years. Activated clotting time (ACT), measured every 30 minutes, is used to determine the adequacy of heparinization, which should be above 480 seconds. Typically, heparin doses for CPB are 250–400 units/kg to achieve ACT values >480 seconds; supplemental heparin may be given to achieve this target value. Hemodilution, protamine overdose, and hypothermia will prolong the ACT. The Hepcon HMS Plus® (Medtronic, Minneapolis, MN) can measure heparin blood concentrations and is used to calculate the dose during CPB. Alternatives to heparin in patients with heparin-induced thrombocytopenia ("direct thrombin inhibitors") are discussed in Chap. 3. Ecarin clotting time can be used to monitor anticoagulation produced by direct thrombin inhibitors. In addition, medications such as aminocaproic acid and tranexamic acid may be used to prevent acute fibrinolysis after CPB. Aminocaproic acid dose has typically been 5–10 g loading dose over the first hour and 1 g per hour thereafter for the duration of the case.

Glucose Management

Hyperglycemia is common during and after CPB. Causes of this increase in blood glucose are multifactorial and include surgical stress, hypothermia and hyperoxia, heparin administration, decreased insulin secretion due to non-pulsatile flow to the pancreas during CPB, and the use of glucogenic catecholamines. Significant research has been conducted on the effects of hyperglycemia in patients undergoing CPB. Hyperglycemia has been proven to be an independent predictor of morbidity and mortality for both diabetic and nondiabetic patients. Increased blood sugar concentrations have also been associated with increased incidence of myocardial infarction, infection, and stroke.

Although there has been debate over the last decade about the desirable blood glucose target range (tight vs. lenient control), most surgical and critical-care organizations advise blood glucose values of ≤180 mg/dL, thus avoiding the risks of hypoglycemia. Intravenous regular insulin is the preferred agent for treating high blood glucose values. Intravenous bolus and continuous infusion dosing can be used for rapid dose titration. As patients recover from surgery, transition to subcutaneous insulin injections may be appropriate.

Electrolyte Management During CPB

Correction of acid/base and electrolyte disturbances are part of standard patient care throughout cardiac surgical cases, more so with CPB.

Potassium values often increase during CPB as a result of the administration of hyperkalemic cardioplegia solutions. Although the safety and utility of hyperkalemic cardioplegia solutions has been debated for decades, they are still used in most cardiac surgery cases because of the rapid electrochemical arrest they can induce. However, research has shown that hyperkalemic cardioplegia solutions and potassium depolarization are linked to arrhythmias and conduction issues: coronary vasospasm; myocardial stunning, leading to low output syndrome; increased platelet aggregation; and transmembrane ionic imbalance.

It is critical to correct patients' high potassium concentrations before separation from CPB because of the potential for adverse effects as mentioned above. Typically, potassium values are brought to <5.5–6 mEq/L before separation from CPB. This goal can be accomplished in many ways, including watchful waiting to allow renal excretion; insulin administration to drive potassium intracellularly; administration of sodium bicarbonate, which provides intracellular binding sites for potassium; and "zero-balance ultrafiltration" (discussed in Chap. 13), which lowers the potassium concentration without increasing the volume of blood in the CPB circuit.

Hypocalcemia also is a common electrolyte abnormality in CPB. Decreased total serum calcium value likely are due to hemodilution. However, administration of albumin, or citrate-containing blood products can raise total serum calcium value. Severe hypocalcemia can be treated before separation from CPB, but research has concluded that empiric calcium administration can worsen reperfusion injury after CPB.

Significant acidosis present before separation from CPB should trigger an aggressive search for the cause and a correction, as acidosis can cause myocardial dysfunction, interfere with inotropic action, and increase pulmonary vascular resistance.

Monitoring of Hemoglobin/Hematocrit During CPB

In the early history of cardiac surgery, anemia from hemodilution (due to the administration of crystalloid or colloid infusions and circuit priming) triggered the need for blood transfusion in almost all open-heart operations. However, recent research has changed transfusion practice significantly, as it was found that transfusion leads to increased time to extubation, increased length of stay in the intensive care unit, and increased morbidity and mortality. Multiple perioperative techniques have been used to lower the risk of transfusion, as discussed in Chap. 15.

The decision to transfuse a patient is a potentially life-altering decision, and a protocol should be developed with the surgical team to reduce the need for transfusion. Hemoglobin concentration is usually monitored throughout CPB on blood samples sent to the main laboratory or by using point-of-care devices such as i-STAT® (Abbott Point of Care, Princeton, NJ), GEM 4000® (Instrumentation

Laboratory, Bedford, MA), and HemoCue® (Hemocue, Brea, CA). Current strategies for transfusion usually do not give allogenic red blood cells to patients at a hemoglobin >7 g/dL unless signs of decreased oxygen carrying capacity and anemia-induced tissue hypoxia are present.

Use of Transesophageal Echocardiography During CPB

Transesophageal echocardiography (TEE) can be a useful tool in surgical interventions, including verifying the position of arterial, venous, coronary sinus cannulas; vents; or intra-aortic balloon pumps. With weaning from CPB, TEE can help in assessing ventricular filling and function, checking for valvular dysfunction, and detecting blood or fluid in the pleurae.

Organ Dysfunction During CPB

CPB can affect multiple organ systems. Some of these effects may be present acutely during CPB, and interventions can be taken to lessen their harm. CPB mainly affects the pulmonary, cardiovascular, neurologic, renal, and hematologic systems.

Lungs

Acute lung injury is a common pulmonary complication after cardiac surgery. The risk and severity of the injury have been consistently linked to the duration of CPB. The presence of early pulmonary dysfunction increases overall morbidity and mortality; renal, neurologic, and infectious complications; and duration of mechanical ventilation and intensive care unit stays. Reducing the systemic inflammatory response by decreasing the time on CPB has been hypothesized to reduce the frequency of acute lung injury and pulmonary complications.

Myocardium

The predominant cause of injury to the heart during CPB is inadequate myocardial protection. The three phases of myocardial protection are pre-arrest preparation, arrest, and reperfusion as discussed in Chap. 14. During pre-arrest preparation, care should be taken to ensure adequate coronary perfusion and to decrease myocardial oxygen demand. The anesthesia provider needs to do little to protect the myocardium during the arrest portion of the procedure, but TEE performed upon initiation of CPB can reveal left ventricular overdistention, which can negatively affect myocardial protection during the arrest period and may lead to increased cell death and poor functional recovery during the reperfusion phase. The reperfusion phase after

release of the aortic cross-clamp causes injury as the heart is reperfused with immunologically primed (from contact with the CPB circuit), fully anticoagulated, and highly oxygenated blood, which may lead to increased incidence of arrhythmia, myocardial stunning, low cardiac output, and perioperative myocardial infarction.

Brain

The potentially negative neurologic effects of CPB are stroke, encephalopathy, and neurocognitive impairment. Causes of these complications may be embolization, hypoperfusion, and inflammation. Embolic events are usually caused by surgical manipulation, but hypoperfusion may be a cause and should be prevented by the anesthesia provider in close communication with the perfusionist. Research has shown that cerebral autoregulation is maintained during hypothermic CPB and that the major effectors of cerebral blood flow during CPB are temperature, anesthetic depth, cerebral metabolic rate, and $PaCO_2$. CPB flow has little to no effect on cerebral blood flow. Finally, systemic inflammation from CPB is thought to alter the endothelium in cardiac surgery patients.

Cerebral oximetry with near-infrared spectroscopy is a commonly used modality to monitor cerebral oxygen saturation. Although studies have reported that monitoring of cerebral oximetry is useful for avoiding neurologic complications, specific thresholds for intervention have not been defined. Nonetheless, cerebral oximetry is often used to follow major bilateral changes and significant unilateral changes in cerebral oxygen saturation saturation (see above "Monitoring Adequate Tissue Perfusion" section). Measurement of jugular bulb oxygen saturation and electroencephalographic monitoring have been used less frequently to monitor global cerebral oxygen saturation.

Kidneys

Renal protection during CPB is a significant issue for cardiac surgery patients, as about 3% of patients undergoing cardiac surgery require temporary or long-term renal replacement therapy postoperatively. The mainstays of renal protection are avoiding hypotension; ensuring oxygen delivery to the kidney; preventing and/or treating low cardiac output states; and avoiding fluid overload. Hypotension can be prevented by giving crystalloid infusions and using vasopressors as needed, or in combination with the fluids, to maintain normal MAPs. Although no target MAP has been shown to reduce the incidence of acute kidney injury after CPB, pressures >60 mm Hg are usually thought to ensure adequate renal perfusion. Ensuring oxygen delivery to the kidneys is accomplished by minimizing hemodilution during CPB and transfusing red blood cells as needed. Research has shown that avoiding hematocrit levels <24% reduces the incidence of acute kidney injury. Low cardiac output states can contribute to decreased blood flow to the kidneys and hypotension as well as to decreased oxygen delivery to the renal tissue, thus worsening kidney injury. Fluid overload can raise central venous pressure and lead to renal congestion and reduced glomerular filtration.

Hematologic

Hematologic consequences of CPB include problems caused by prolonged contact of blood with the CPB circuit, high doses of heparin, and hypothermia. Contact of a patient's blood with the CPB circuit activates the intrinsic and extrinsic coagulation cascades. High doses of heparin cause increased intraoperative bleeding and platelet activation. Platelet activation is also increased in hypothermia. These consequences of CPB can cause coagulopathy when weaning, which may require the anesthesia provider to administer blood products. Common methods to quickly evaluate coagulation status are ACT measurements and thromboelastography, which assesses coagulation factor function, platelet function, clot strength, and fibrinolysis.

Further Reading

Barry AE, Chaney MA, London MJ. Anesthetic management during cardiopulmonary bypass: a systematic review. Anesth Analg. 2015;120(4):749–69.

Hessel, E. What's New in Cardiopulmonary Bypass. J Cardiothorac Vasc Anesth, 2019 Aug; 33(8): 2296–2326.

Gravlee G, Glenn P, Davis R, et al, Cardiopulmonary Bypass and Mechanical Support: Principles and Practice, 4th Edition, Philadelphia, Lippincott, Williams, & Wilkins, 2015.

Laffey JG, Boylan JF, Cheng DC. The systemic inflammatory response to cardiac surgery: implications for the anesthesiologist. Anesthesiology. 2002;97(1):215–52.

Shore-Lesserson L, Baker R, Ferraris V, et al. The Society of Thoracic Surgeons, the Society of Cardiovascular Anesthesiologists, and the American Society of ExtraCorporeal Technology: clinical practice guidelines—anticoagulation during cardiopulmonary bypass. Ann Thorac Surg. 2018;105(2):650–62.

Weaning from Cardiopulmonary Bypass and Management of Difficulties

17

Alexander Kahan

A good hockey player plays where the puck is. A great hockey player plays where the puck is going to be.

—Wayne Gretzky

Introduction

One of the most tense and hemodynamically labile moments during any on-pump procedure occurs while attempting to separate from the cardiopulmonary bypass (CPB) machine. It is imperative that the anesthesiologist, perfusionist, and surgeon work in concert in order to have a successful transition back to the patient's native circulation.

Weaning Process Steps

When weaning the patient from CPB, the perfusionist begins to reduce arterial flow while sequentially clamping the venous return (Fig. 17.1). Successful transition from bypass necessitates careful preparation; omission of any vital step can lead to catastrophe.

Hemodynamic instability following separation from CPB can result from many causes, including systemic vasodilation, inadequate surgical repair, left ventricular dysfunction, right heart failure, pulmonary hypertension, metabolic disturbances, and alterations in hemostasis. Many anesthesia providers use acronyms as memory

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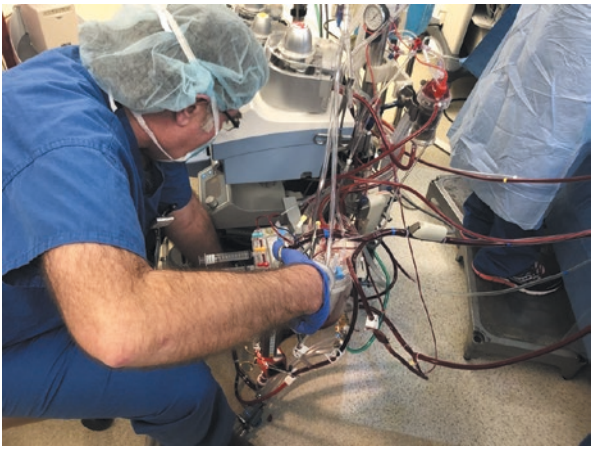

Fig. 17.1 Perfusionist clamping venous return

Table 17.1 The OK TO PROCEED mnemonic details a systematic, checklist syle approach to safely wean from CPB

O – Oxygen and ventilation
K – K^+ (potassium) and other electrolytes
T – Temperature
O – Output
P – Procedural outcome
R – RBCs
O – Optimizing inotropy
C – Coagulation status
E – Entrained air
E – Electrical activity
D – Distention

tools to help achieve safe and efficient weaning from CPB. Three of the most common acronyms are ABCDEFG (Airway, Beating heart, Crit, Drugs, Electrolytes, Fahrenheit, Gradient) and WAAARRRRMM (Warm, Anesthesia, Adjuvant drugs, Air, Rhythm, Rate, Resistance, Respiration, Metabolism, Monitoring). Here we introduce a third, the OK TO PROCEED mnemonic (Table 17.1) and explain each step in detail.

Oxygen and Ventilation

Weaning from bypass requires the re-establishment of flow through the native cardiopulmonary circulation. As the perfusionist begins to reduce flow into the venous reservoir of the machine, blood is diverted to the heart and lungs. With less reliance on extracorporeal oxygenation, the anesthesia provider must to enable the necessary alarms, adjust monitors, and ensure adequate ventilation and oxygenation to minimize the introduction of shunt. A gentle recruitment maneuver is often employed at the initiation of mechanical ventilation to recruit atelectatic lung while avoiding disruption of fragile surgical grafts. So, it is advisable to ask the surgeon to protect the grafts, especially the mammary, during this maneuver.

It is also important to reinstitute the delivery of any volatile anesthetic that the anesthesiologist wishes to provide. The act of warming a patient and the transition of anesthetic delivery from the CPB circuit back to the ventilator during weaning contribute to an increased risk of intraoperative awareness during this period. Therefore, it is essential to minimize gaps in anesthetic delivery, or augment the anesthetic with an amnestic agent such as midazolam.

K^+ and Other Electrolytes

Electrolyte abnormalities are commonly encountered during CPB. The physiologic stress response, coupled with the administration of exogenous catecholamines, potassium solutions, sodium bicarbonate, and insulin may adversely affect serum pH and electrolyte concentrations. With the aid of frequent blood gas analyses, the anesthesia provider and perfusionist can act to achieve homeostasis until a suitable heart rhythm is attained. Multiple sources support the routine use of calcium and magnesium administration to counter the hyperkalemia that may be present before termination of CPB, however there is a also a concern regarding calcium-mediated reperfusion injury.

Temperature

The temperatures most often maintained during CPB range from mild to deep hypothermia. In some cases, the surgeon may rely on hypothermia to keep the heart in a fibrillatory or arrested state. Achieving and maintaining normal cardiac conduction, optimal hemostatic and bacteriostatic conditions, and electrolyte homeostasis all depend on the restoration of normothermia. This is facilitated through the administration of warmed fluids and blood, use of forced air warming devices, and warming of the operating room ambient temperature. Care should be taken to not warm a patient above 38 °C or faster than 0.5 °C/minute, as extreme changes in temperature have been shown to worsen neurologic outcomes.

Cardiac Output

Underfilling of the heart, which can be appreciated by direct observation of the right ventricle and through transesophageal echocardiogram analysis of the left ventricle, is a common cause of hypotension occurring immediately after separation from bypass. To optimize preload during this period, the perfusionist can rapidly transfuse volume that persists in the CPB reservoir through the arterial cannula in an increment of 100 cc at a time.

Cardiac output normally is maintained by heart rate and stroke volume, and it is therefore dependent on myocardial contractility. Regional myocardial dysfunction can be identified and swiftly diagnosed by echocardiography, so that the causes of

dysfunction can be managed appropriately. When regional dysfunction after bypass matches the pre-bypass dysfunction, bypass graft dysfunction/occlusion, or myocardial stunning can be at fault. Coronary flow probes (Transonic®, Ithaca NY) can be used to analyze and surgically correct the former, whereas time and minor inotropic support can help manage myocardial stunning.

After surgical correction of functional pop-off valves (such as correction of a regurgitant mitral valve), the left ventricle must work harder to pump against an increased afterload. Healthy ventricles often can accommodate this change with little to no inotropic support, but underlying ventricular dysfunction occasionally becomes uncovered after surgical repair and must be aggressively treated pharmacologically or, rarely, with mechanical support devices such as with Impella pumps (Abiomed®, Danvers MA) or intra-aortic balloon pumps to reduce afterload.

Procedural Outcome

Transesophageal echocardiogram examination should be employed routinely prior to weaning from bypass to confirm proper heart and valvular function, the absence of intracardiac shunts, the absence of leak or a residual stump following appendage ligation, and the absence of an iatrogenic aortic dissection. Causes of improper valvular function, such as the presence of paravalvular leaks, the rocking of prosthetics, dynamic or fixed valvular stenoses, or improper leaflet movement, should be looked for.

Intracardiac shunts can be identified through color Doppler analysis and through comparison of pulmonary and systemic flow (Qp:Qs) following liberation from bypass. Leaks can be identified via color doppler and notice should be taken to rule out any expanding posterior pericardial effusions. Lastly, the aorta should be analyzed for dissection flaps at and distal to the cannula insertion site.

RBCs

Routine blood gas analysis is invaluable for monitoring hematocrit and electrolytes concentrations throughout the CPB period. Blood can be lost to the surgical field, and hematocrit may become diluted in a pump that was not primed with autologous blood. Our institution attempts to maintain a minimum hemoglobin concentration of 7–7.5 g/dL upon separation of CPB to ensure adequate oxygen delivery to potentially at-risk myocardium. Note that there may be large amounts of autologous cell-salvage blood available after the patient is weaned from CPB, and caution should be taken in rapidly transfusing a struggling right ventricle.

Optimizing Inotropy

Predicting ventricular dysfunction and the need for inotrope support after CPB has been extensively studied (Table 17.2).

Table 17.2 Risk factors for needing post-bypass ionotropic support

1. Advanced age
2. Female
3. Left ventricular ejection fraction <35%
4. Reoperation
5. Aortic cross-clamp time >90 minutes
6. Preoperative beta-blockers
7. Congestive heart failure
8. Mitral regurgitation
9. Chronic kidney disease

Closed-loop communication with the perfusionist is paramount during the CPB period. If the perfusionist reports increasing vasopressor requirements, the patient may be experiencing vasoplegia. This condition may occur as an overexpression of inflammatory mediators, deficient release of vasopressin and angiotensin II, and subsequent endothelial dysfunction. Increased vasopressor requirements during and after separation from CPB likely will be needed.

Similarly, patients who had severely depressed cardiac function preoperatively, may have a greater need for inotropic support and/or the need for a ventricular assist device (such as an Impella®) to enable termination of CPB support.

Coagulation Status

Patients' preoperative coagulation status and recent antiplatelet therapies can be predictors for post-CPB bleeding; thus, they should be discussed during the time-out in the operating room before the start of the case. Often, the perfusionist will run a thromboelastogram once warming has been initiated and will obtain a platelet count so that post-bypass transfusion needs can be anticipated.

When CPB support has been terminated and the venous cannula clamped, the surgeon will request the administration of protamine, a cationic protein that binds heparin to form a stable salt. Protamine is typically given at a ratio of 10 mg protamine/1000 units of heparin that was given before going on CPB. Note: Administration of protamine before clamping the venous cannula can clot the entirety of the blood in the CPB machine reservoir, rendering it useless and effectively exsanguinating the patient. It is extremely important that the start of protamine administration be communicated to both the surgeon and the perfusionist. At one third of the administered dose, the perfusionist should be notified so all field suckers can be turned off to avoid clotting in the machine.

Protamine is administered slowly, occasionally through micro-drip tubing. Please refer to Chap. 3 for more information about protamine and protamine reactions.

Entrained Air

Before the patient is separated from CPB, the anesthesia provider must report if intracardiac air is present. Air bubbles tend to mobilize with initiation of ventilation and can most often present on the anterior surface of the left atrium, in the apex of

the left ventricle, along the anteroseptal area of the interventricular septum, or in the right pulmonary veins. Maintaining partial bypass while allowing the heart to eject can help evacuate air out to the bypass vents. Alternatively, the surgeon may manually agitate the heart so entrapped air will quickly dislodge, or may elect to place a small-gauge needle in the apex of the left ventricle or the aortic root to vent the air (Fig. 17.2).

Occasionally, air may present itself after the administration of protamine and after CPB pump suckers and vents have been removed. The right coronary artery is the most susceptible to air embolization given its relatively anterior positioning while the patient is lying in the supine position. Inferior-lead ECG changes often reflect the resulting hemodynamic instability. The anesthesia provider should be

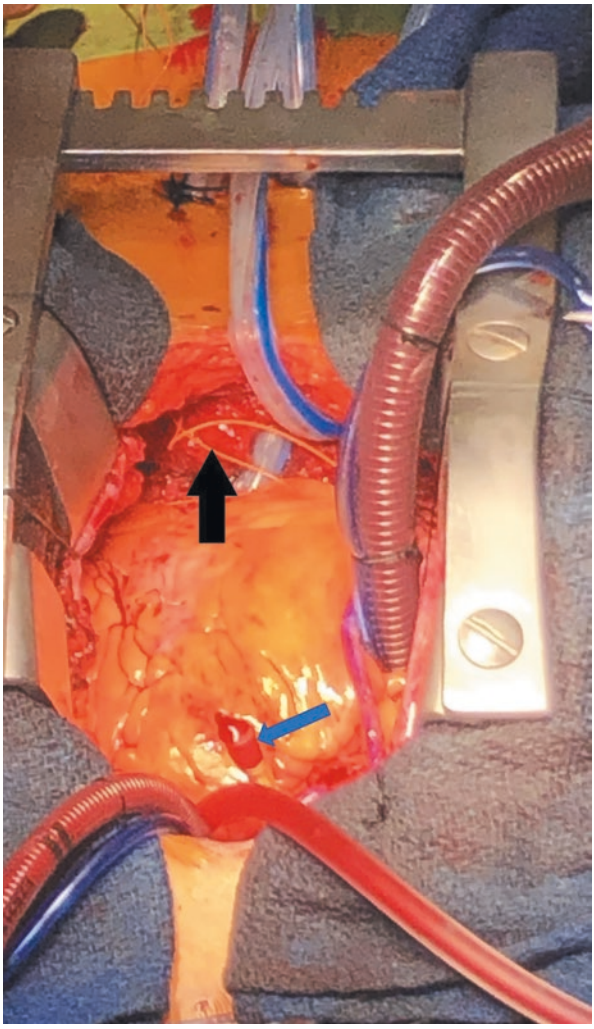

Fig. 17.2 The black arrow indicates epicardial leads placed for treatment of persistent junctional rhythm while weaning from bypass was being attempted. The blue arrow indicates an angiocath that had been placed in the aortic root after intracardiac air was noted, but after the root vent had been removed

prepared to recognize this complication and aggressively provide hemodynamic support. Raising the mean arterial pressure and coronary perfusion pressure might help to disintegrate the air bubble or push it further downstream.

Electrical Activity (Rate, Rhythm, Pacer)

After cross-clamp removal and cardioplegia dissolution, the ECG may reveal the return of electrical activity. Ventricular fibrillation and ventricular tachycardia, if present, should be treated with defibrillation or antiarrhythmic drugs such as lidocaine or amiodarone. If the dysrhythmia persists, an underlying cause, such as graft embolism/thrombosis or electrolyte abnormality, should be swiftly sought and addressed. Allowing the heart to remain in fibrillation or tachycardia exponentially increases metabolic demand on the heart.

Occasionally, there may be complete heart block, bradycardia, or junctional dysrhythmias during weaning. If pharmacologic treatment for these proves unsuccessful, temporary epicardial pacemaker leads placed by the surgeon will provide an adequate rate and rhythm (Fig. 17.2).

Distention

Ventricular dysfunction remains the most common cause of failure upon attempting to wean from bypass. As volume is returned to the patient, the heart's intrinsic inotropic forces are needed to move the blood; failure to do so is soon recognized by the presence of ventricular dilatation. Right ventricular failure and dilatation can be easily diagnosed when a median sternotomy is used for surgical access because the right ventricle can be directly observed enlarging with the infusion of volume. Left ventricular failure can be more insidious because its diagnosis relies heavily on clinical and echocardiographic findings. Inotropic augmentation can improve contractility in both right ventricular and left ventricular failure, and vasodilators, such as inhaled nitric oxide or iloprost, can help decrease pulmonary hypertension and right ventricular strain. Other measures to improve right ventricle dysfunction and to lessen distention include faster atrioventricular pacing to decrease diastolic time and hence filing, raising the head of the bed, slowing rapid infusion through the central line, ask the surgeon to open the purse-string at the venous cannula site in the right atrium to drain some blood out, and finally reheparinize and go on CPB again until the team figures out the next plan of action.

Conclusion

Maintaining a methodical, stepwise approach to weaning from cardiopulmonary bypass is invaluable for minimizing hemodynamic instability during this dangerous period. Clear, closed-loop communication between all members of the anesthesia,

perfusion, and surgical teams optimizes patient safety. Working within this framework enables cardiac anesthesia trainees to logically approach the complex physiology of cardiac surgical patients and improve their outcomes.

Further Reading

1. Wahr A, Prager L, Abernathy A, Martinez C, Salas C, Seifert D, et al. Patient safety in the cardiac operating room: human factors and teamwork: a scientific statement from the American Heart Association. Circulation. 2013;128(10):1139–69. https://doi.org/10.1161/CIR.0b013e3182a38efa.
2. Lewis KP, Canelli RJ, Ortega R. OK to proceed? What every health care provider should know about patient safety. Boston: Boston Medical Center; 2018. p. 351. ISBN 978 0 692 18660 2.
3. Licker M, Diaper J, Cartier V, Ellenberger C, Cikirikcioglu M, Kalangos A, et al. Clinical review: management of weaning from cardiopulmonary bypass after cardiac surgery. Ann Card Anaesth. 2012;15(3):206–23. https://doi.org/10.4103/0971-9784.97977.
4. Barry E, Chaney A, London J. Anesthetic management during cardiopulmonary bypass: a systematic review. Anesth Analg. 2015;120(4):749–69. https://doi.org/10.1213/ANE.0000000000000612.
5. Lombard W, Grichnik P. Update on management strategies for separation from cardiopulmonary bypass. Curr Opin Anaesthesiol. 2011;24(1):49–57. https://doi.org/10.1097/ACO.0b013e328342064a.
6. Rao V, Ivanov J, Weisel R, Ikonomidis J, Christakis G, David T. Predictors of low cardiac output syndrome after coronary artery bypass. J Thorac Cardiovasc Surg. 1996;112(1):38–51. https://doi.org/10.1016/S0022-5223(96)70176-9.
7. Mazer C, Whitlock R, Fergusson D, Hall J, Belley-Cote E, Connolly K, 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.
8. Michael G Fitzsimons, MD, Jonathan B Mark, MD, Nancy A Nussmeier, MD, FAHA. Weaning from cardiopulmonary bypass. UpToDate.com.

Part V

Anesthesia for Cardiac Procedures

18 Anesthetic Management for Conventional Myocardial Revascularization

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In theory there is no difference between theory and practice. In practice there is.

—Yogi Berra

Key Points

- Induction of anesthesia
- Positioning considerations for CABG
- Sternotomy and harvesting conduit
- Cannulation
- Initiating cardiopulmonary bypass
- Aortic cross clamp, cardiac arrest, anastomoses
- Weaning from cardiopulmonary bypass
- Case completion

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Introduction

Coronary artery bypass grafting (CABG) is one of the most common operations performed throughout the world. The use of a heart lung machine (cardiopulmonary bypass – CPB) is a prerequisite to cardiac surgical training and successful practice. Advantages associated with the use of cardiopulmonary bypass during CABG include a quiet, bloodless field, as well as optimal access to the coronary arteries. The risks associated with CPB are relatively low. Most relate to the contact of blood with the plastic components of the heart lung machine. Complications may also arise from surgical manipulation of the cardiac structures.

Important anesthetic goals for patients undergoing CABG utilizing CPB include the management of myocardial oxygen supply and demand to avoid ischemia; ensuring adequate anticoagulation before the initiation of CPB and restoration of normal coagulation after CPB; monitoring for complications during institution of and separation from CPB; and management of the hemodynamic state during surgical manipulation and following coronary revascularization.

Induction of Anesthesia

Prior to the induction of anesthesia, supplemental oxygen should be provided to maximize oxygen supply to the myocardium. In patients with significant coronary artery disease, defibrillator pads are usually applied prior to induction. Because of the large sterile surgical field, all EKG leads are placed on the patient's sides, flank, or shoulders. The leads may also be covered with waterproof tape to prevent antiseptic skin preparation solutions from interfering with the lead function.

An arterial line is usually placed prior to induction. If the radial artery will be harvested for use as a surgical conduit, the arterial line should be placed on the contralateral side. If the radial artery will not be harvested, consider preferentially placing an arterial line on the side opposite the surgeon. This may avoid erroneous blood pressure readings resulting from a surgeon leaning against the monitored arterial system. The presence of an arterio-venous fistula precludes placement of an arterial line in the same extremity.

Prior to induction, baseline laboratory tests should be drawn including an arterial blood gas, an activated clotting time (ACT), a blood glucose, and possibly other coagulation studies depending on the institutional protocol. Large-bore central venous catheters can be placed either before or after the induction of anesthesia.

The goal for anesthetic induction is to maximize oxygen supply and minimize oxygen demand through the tight control of heart rate and blood pressure. The specific choice of anxiolytic, opioid, hypnotic, and muscle relaxant is less critical than their careful titration to effect. Anesthetic dosing should be sufficient to control the sympathetic response to noxious stimulation but should also avoid excessive depth of anesthesia and resulting hypotension. Alpha-1 (phenylephrine, norepinephrine) and beta-1 (ephedrine, epinephrine) agonists should be available in both bolus and infusion forms in order to immediately correct any hemodynamic compromise.

Patient Positioning

Supine positioning is used for CABG and the surgical field includes the anterior surface of the chest, abdomen, groin as well as both legs prepared in a circumferential fashion. The surgical field must provide access to both groins if needed for line placement or CPB cannulation, as well as both lower extremities in case additional conduit (vein) is needed (Fig. 18.1).

Arms are tucked at the patient's side and are generally inaccessible during standard sternotomy. When positioning the arms, all pressure points (IV hubs, stopcocks, bed rails etc.) must be carefully padded to avoid soft tissue or nerve injuries (Fig. 18.2). Once positioning has been completed it is important to confirm adequate functioning of all peripheral intravenous lines, hemodynamic monitors, and the presence of radial pulses.

A "bump" (shoulder roll) is placed under the patient's shoulders, separating the plane of the mandible from the anterior chest wall to ease the process of sternotomy. Placement of the shoulder roll may leave the patient's head and neck in a vulnerable position, particularly in patients with limited cervical spine mobility. The head must be well supported, and the cervical spine not hyperextended as this can lead to postoperative pain or neurological injury.

A transesophageal echo (TEE) probe may be placed after the induction of anesthesia provided that no contraindications exist. Placement of the probe prior to surgical draping allows for easier placement and the use of a laryngoscope should any difficulties be encountered (Fig. 18.3).

If the patient is lightly anesthetized during positioning, the heart rate and blood pressure may increase in response. Conversely, if the patient is deeply anesthetized, blood pressure support may be required to maintain sufficient perfusion to the myocardium.

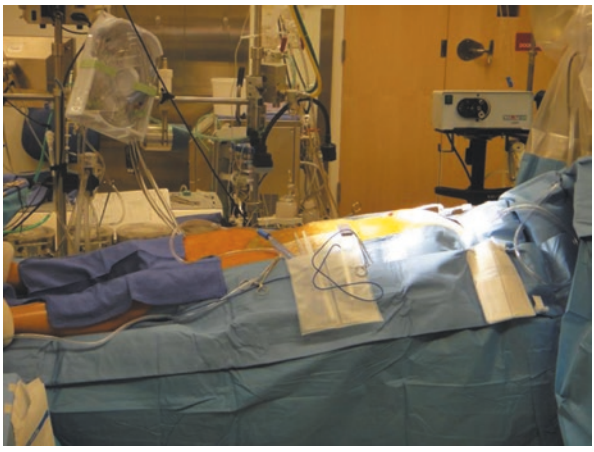

Fig. 18.1 A picture of patient positioned and ready for surgery

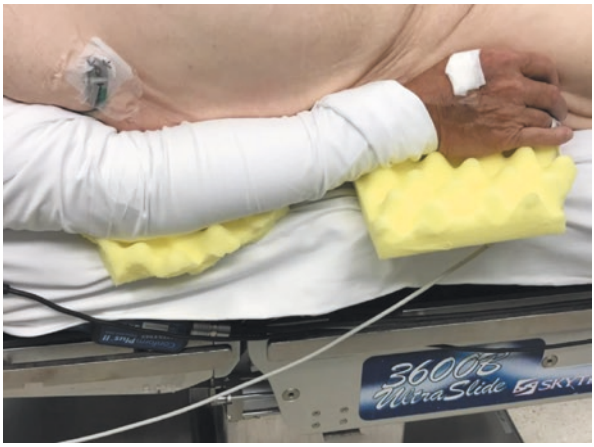

Fig. 18.2 A picture of a patient with all pressure points carefully padded

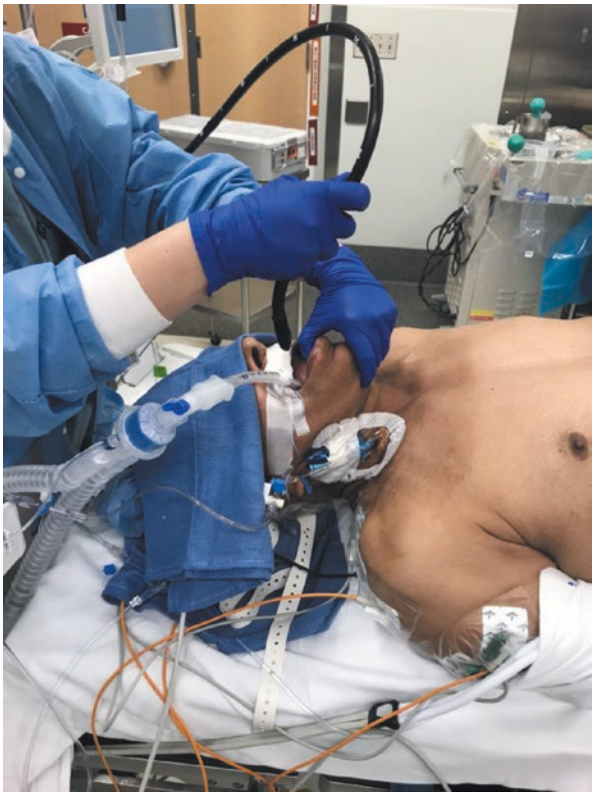

Fig. 18.3 A picture of TEE probe insertion

Sternotomy and Conduit Harvest

A midline sternotomy is performed extending a skin incision from the sternal notch to the xiphoid process (Fig. 18.4). Soft tissue is dissected down to the sternum which is then opened with a sternal saw. The stimulation associated with skin incision and sternotomy is significant. If untreated, patients will become hypertensive and tachycardic during this period. Preemptive increases in volatile anesthetic or supplemental opioids just prior to incision will help control the hemodynamic response. If the patient's hemodynamic status does not allow for additional anesthetic agents, preparations should be made to treat increases in heart rate and / or blood pressure. Control of blood pressure during this time assists with hemostasis and minimizing blood loss from the marrow of the divided sternum. In redo-sternotomy procedures, blood should be readily available in the operating room. Finally, to avoid injury to the anterior aspects of each lung the anesthesia ventilator must be turned off and the adjustable pressure limiting valve must be open during sternotomy.

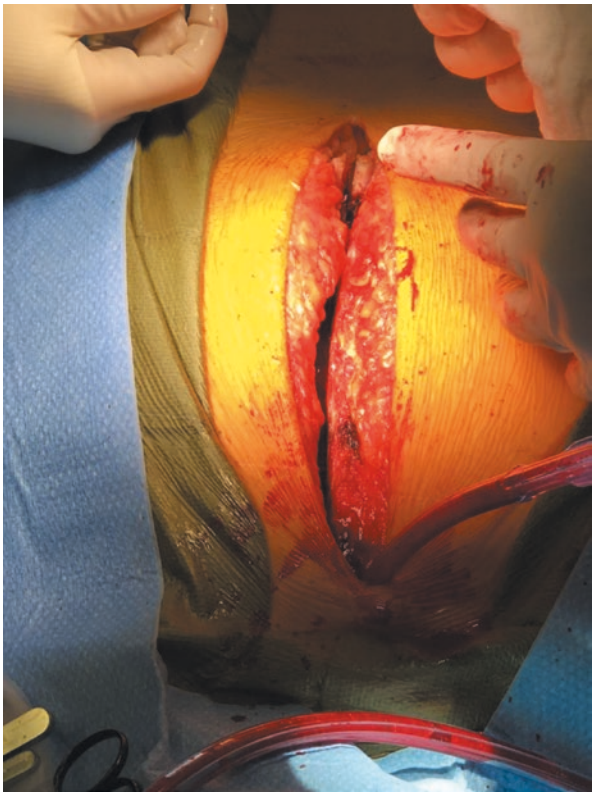

Fig. 18.4 A picture of a midline sternotomy

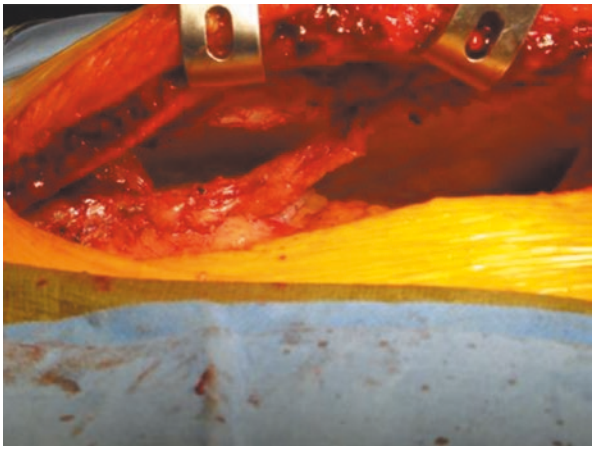

Fig. 18.5 A picture of left internal mammary artery harvest

The most common conduit for CABG is the left internal mammary artery (LIMA). It is prepared as a pedicle graft, preserving its proximal attachment to the left subclavian artery (Fig. 18.5).

Similarly, a right internal mammary artery harvest can be accomplished through the same incision, with similar technique. Entering the pleural space during a mammary artery harvest is common and sometimes intentional. It will allow drainage of any effusions, confirmation of bilateral lung expansion, and the ability to position the heart without restriction.

Simultaneous to the mammary artery harvest, the greater saphenous vein may be obtained from the leg. The saphenous vein is the second most common conduit for coronary artery surgery. This is performed via an open surgical dissection or endoscopic approach. Endoscopic vein harvest commonly requires insufflation of the subcutaneous fat with carbon dioxide (CO_2). This CO_2 may be rapidly absorbed resulting in a respiratory acidosis. When using endoscopic techniques this phenomenon should be anticipated and the minute ventilation should be adjusted according to end-tidal CO_2 levels.

Radial artery harvest can also occur concomitantly via an open or endoscopic approach. When choosing to harvest radial arteries, preoperative Allen's testing should be documented to ensure ulnar flow to the palmar arch.

While the bypass conduits are being harvested, there is a significant decrease in the intensity of surgical stimulation. As a result, the patient may become hypotensive and require blood pressure support and a reduction in anesthetic depth. Adequate neuromuscular blockade should be maintained during sternotomy and LIMA harvest to allow for the chest wall to be elevated.

Cannulation

Once adequate bypass conduit is obtained, the pericardium is opened and secured to the wound edges. Purse string sutures are placed on the ascending aorta and the right atrium. The aortic purse strings are placed in an area of the ascending aorta that is free of atheroma to prevent embolization during cannulation. Intravenous heparin (300 units/kg) is administered at least 3 minutes prior to aortic cannulation to permit adequate circulation time. An ACT greater than 480 seconds is required prior to initiating CPB, although the precise target ACT varies by institution. After heparin has circulated, the arterial cannula is placed in the ascending aorta through the center of the purse string stiches (Fig. 18.6).

Systolic blood pressure should be reduced immediately prior to and during arterial cannulation to decrease blood loss and limit the risk of arterial injury. Systolic pressures below 100 mmHg are optimal and can normally be achieved via short-acting vasodilators (NTG, nitroprusside). Aortic pressures are compared to radial arterial measurements following cannulation to ensure proper placement of the cannula in the aortic lumen. Aortic dissection during cannulation may be recognized by high cannula pressures, a purple discoloration of the ascending aorta, and/or the presence of a dissection flap on TEE examination.

Following successful aortic cannula placement, a venous cannula is placed in the right atrium. Multiple fenestrations in this cannula allow it to drain both the inferior vena cava as well as the right atrium (superior vena cava) simultaneously (Fig. 18.7).

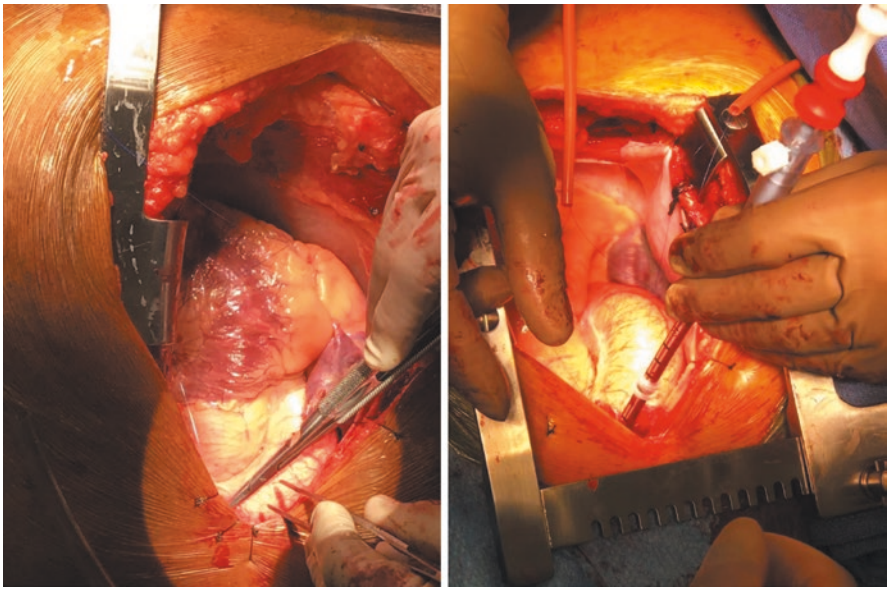

Fig. 18.6 Pictures of aortic cannulation. Left: Prior to cannulation; suturing purse string. Right: Inserting aortic cannula

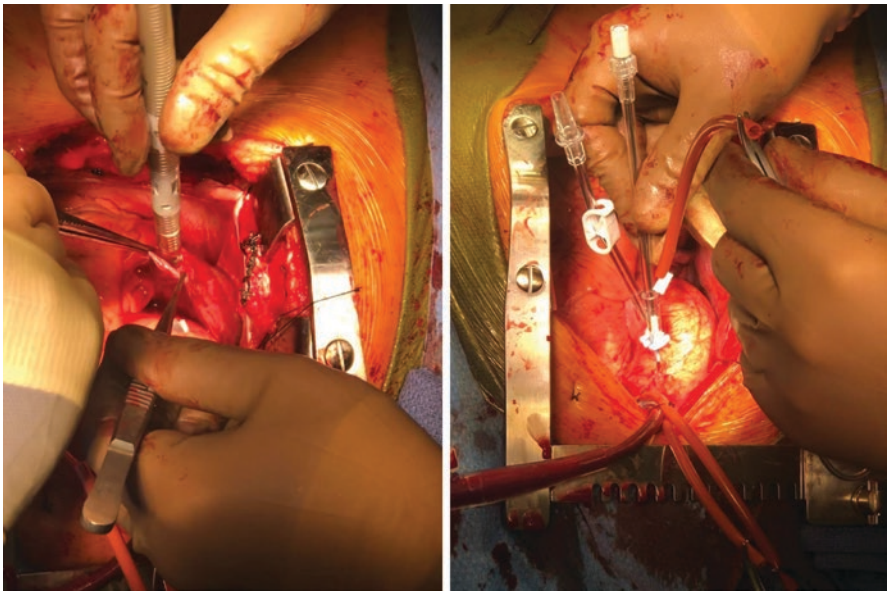

Fig. 18.7 Pictures of venous cannulation. Left: Inserting two-stage venous cannula in right atrium. Right: Inserting antegrade cardioplegia catheter

Proper positioning of the venous cannula is essential to allow adequate venous drainage to the heart-lung machine.

Initiation of Cardiopulmonary Bypass

Immediately prior to initiating CPB, consideration should be given to removing excess crystalloid from the heart lung machine. In order to minimize hemodilution the technique of retrograde autologous priming (RAP) may be used. This maneuver allows the patient's native arterial pressure to drive blood flow backwards through the arterial side of the CPB circuit. The perfusionist simultaneously drains the priming crystalloid solution from the CPB circuit. This results in a state of relative hypovolemia and vasoconstrictors may be needed to avoid hypotension until CPB is initiated. Close communication between the anesthesiologist, perfusionist, and surgeon is required when employing RAP.

CPB is then initiated (Fig. 18.8) by allowing venous drainage from the right atrium to the venous reservoir. If clinically appropriate, the initial venous drainage can also replace the remainder of the excess crystalloid in the circuit. Once venous drainage is confirmed, forward aortic flow is assessed. Finally, once the perfusionist has achieved the targeted CPB flow and pulmonary circulation has ceased, the ventilator may be turned off. Initiation of CPB is a critical time in the operation requiring the focused attention of all team members and continuous closed-loop

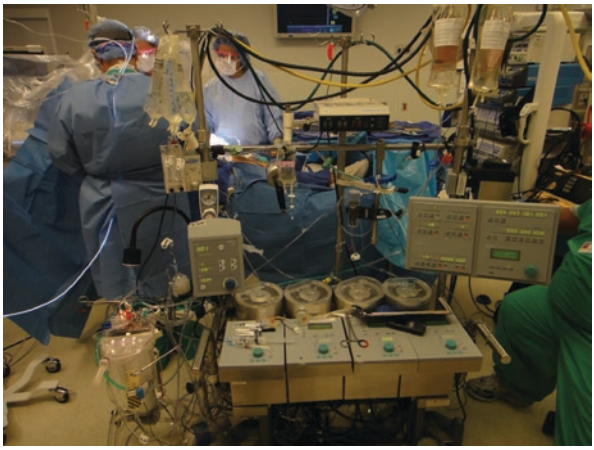

Fig. 18.8 A picture of readiness to initiate CPB

communication. If problems which limit CPB flow are not rapidly recognized, multisystem organ damage may occur.

Aortic Cross Clamp, Cardiac Arrest, and Anastomosis

Cardiac arrest is implemented once the CPB circuit is providing total circulatory support. Cardiac arrest allows the required coronary artery anastomoses to be created on a motionless and bloodless surgical field. The steps in achieving cardiac arrest include: placement of an aortic clamp proximal to the aortic perfusion cannula (Fig. 18.9), venting any remaining blood in the isolated heart, administering solution (cardioplegia) to stop the electrical activity of the myocardial cells, and cooling the myocardium.

When selecting the location for the aortic clamp, areas of palpable atheroma are avoided. In patients with known or suspected disease of the ascending aorta, an epiaortic ultrasound examination may be used. The clamp is placed just proximal to the aortic cannula thereby preventing oxygenated blood from the heart lung machine from reaching the coronary ostia. Cold cardioplegic solution, high in potassium, is administered into the aortic root via a small cannula and is directed into the coronary arteries. The local hyperkalemia produces arrest of myocardial electrical activity, while cooling of the myocardium reduces any residual basal cellular oxygen requirements. If concerns exist that severe coronary artery disease may prevent delivery of cardioplegia to all areas of the myocardium, a cannula may be placed in the coronary sinus and cardioplegia can be delivered in a retrograde fashion to the heart's venous system.

The heart remains arrested until removal of the aortic clamp. The surgeon may choose to complete all of the proximal and distal anastomoses prior to allowing myocardial reperfusion or may remove the aortic clamp after the distal coronary graft sites are finished. In the latter case, proximal aortic anastomoses will be

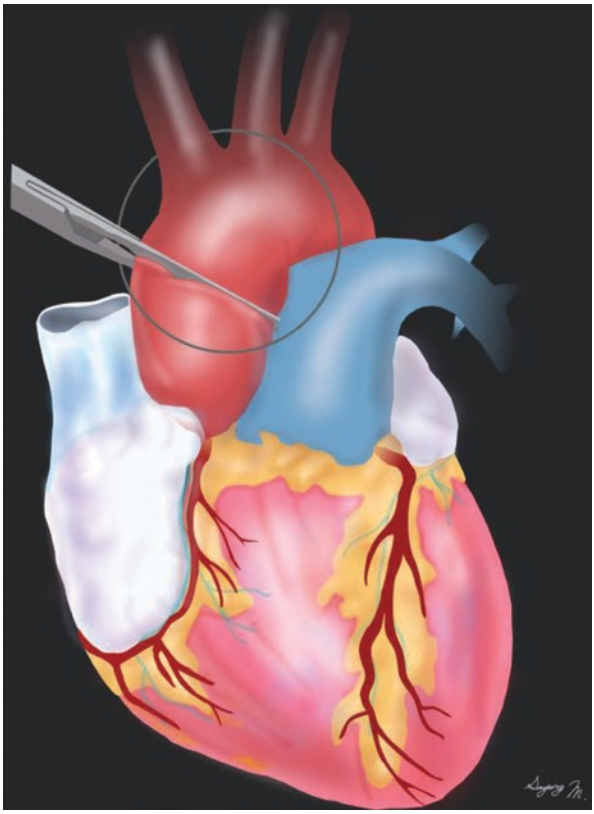

Fig. 18.9 Illustration of aortic cross clamp prior arresting the heart

completed with the aid of a partial aortic clamp or other commercial device to allow isolation of a limited segment of the aortic wall. Once coronary flow is reestablished, the effects of the cardioplegia solution dissipate rapidly and normal patterns of electrical activity return to the heart (Fig. 18.10).

During the period that the patient is supported with CPB, continuous monitoring of mean arterial pressure, temperature, urine output, and blood sugar should be maintained. Neuromuscular blockade may need redosing and should be guided by train of four monitoring. While on CPB, an anesthetized state is maintained via volatile anesthetic vapor administered through the CPB circuit oxygenator. However, consideration should be given to administering supplemental intravenous agents (e.g. midazolam, fentanyl) to minimize the risk of recall during CPB in patients at high risk of intraoperative awareness (e.g. young age, prior episodes of intra-operative recall). The period of rewarming at the end of CPB is historically associated with the greatest incidence of awareness under anesthesia.

Weaning from Bypass

During separation from CPB, the patient's heart begins to take over its function of circulating blood to the patient's body from the bypass circuit. As with the initiation of CPB, this period requires the focused attention of all team members and

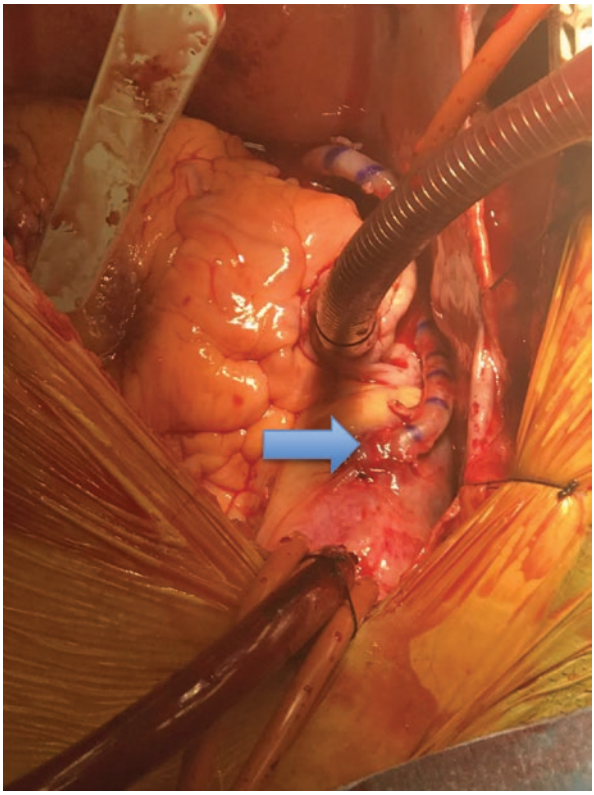

Fig. 18.10 A picture of preparing to come off CPB. Arrow: proximal anastomosis of the new bypass graft

continuous closed-loop communication. A deliberate standardized stepwise approach is essential to avoid complications. More detailed discussion about weaning from CPB can be found in Chap. 17.

While preparing for CPB separation, metabolic abnormalities should be corrected. Significant metabolic acidosis may not be easily compensated for with hyperventilation after resumption of mechanical ventilation and treatment with sodium bicarbonate while on CPB should be considered. Severe anemia (Hg < 7 gm/dL) will impair oxygen delivery to the myocardium even following revascularization and transfusion should be considered. Hypocalcemia will impair myocardial contractility and systemic vascular resistance and calcium replacement immediately prior to terminating CPB is routine.

Lung recruitment begins with hand ventilation to expand the lungs and minimize atelectasis. This occurs while the surgeon ensures that lung hyperinflation does not disturb the new bypass grafts, in particular any IMA grafts as they course from the chest wall to their coronary target.

Epicardial pacing wires are placed on the right ventricle and, in select patients, the right atrium. The pacing leads should be connected to an external pacemaker and tested to ensure capture. If the patient has an acceptable rhythm and rate, the pacemaker may be set to pace at a back-up rate in case of acute bradycardia. Anastomotic sites are checked for obvious bleeding. The TEE should be examined to gauge global cardiac function, identify any wall motion abnormalities, and assess for valvular abnormalities not present pre-CPB.

The act of weaning from CPB begins by slowly obstructing drainage to the venous reservoir thereby diverting blood to the ventricles and allowing cardiac ejection. As venous return is further restricted the flow rate through the aortic cannula decreases as well. When the patient is stable, all CPB flow is stopped. Once hemodynamic stability (e.g. cardiac output, arterial pressure), oxygenation, and ventilation (ABG if necessary) are optimized, the venous and arterial cannulas are removed. Any blood remaining in the CPB machine is then returned to the patient.

During and immediately following separation from CPB, blood pressure management is critically important. High blood pressure can lead to bleeding and cause damage to the new anastomoses. Hypotension may lead to ischemia.

Once separation from CPB is achieved, protamine is administered to reverse the effect of heparin. Once the total dose of protamine has been given, an ACT should be drawn in combination with an ABG as well as any other coagulation studies dictated by institutional protocol.

Case Completion

After protamine administration, surgical closure begins. Chest tubes are placed in the mediastinum and pleural space to monitor postoperative bleeding. The sternum is closed with wires. At each stage of the post-CPB period (i.e. after separation from CPB, protamine administration, and sternal closure) it is important to reassess the hemodynamic state and perform a TEE examination. New regional wall motion abnormalities or severe global myocardial dysfunction may occur as a result of focal or global ischemia and may prompt immediate surgical reexploration.

After wound closure the patient is transferred to the ICU for postop care, most often intubated and sedated. During movement to the ICU the patient must be continuously monitored with EKG, pulse oximetry, and arterial pressure measurement. Emergency airway equipment and medications to allow resuscitation should be accessible to the anesthesiologist at all times during transport from the OR to the ICU. The external pacemaker should also accompany the patient.

In most institutions the goal of postoperative care is to allow extubation following a brief period (1–6 hours) of observation to rule out early postoperative complications (e.g. bleeding, myocardial dysfunction, arrhythmias). This requires an anesthetic plan which permits a smooth transition from surgical anesthesia to postoperative analgesia and sedation. Sympathetic discharge and agitation from under-sedation can be as deleterious as over-sedation and hypotension. Appropriate intraoperative titration of anesthetic agents and judicious use of sedative infusions (propofol, dexmedetomidine) are essential. Stages of the procedure with anesthetic considerations are listed in Table 18.1

Clinical Pearls

- Effective communication between anesthesiologists, surgeons, and perfusionists is critical for successful patient outcomes.
- In order to minimize ischemia to the myocardium, oxygen supply must be maximized and oxygen demand must be minimized.

- Myocardial oxygen supply is primarily determined by coronary blood flow (e.g. mean arterial pressure, time in diastole) and oxygen carrying capacity of blood (e.g. hemoglobin concentration and oxygen saturation).
- Myocardia oxygen demand is determined by heart rate, contractility, and wall tension.
- Periods of high stress (e.g. intubation, sternotomy, transport to ICU) should be anticipated and may require a deepening of the anesthetic state to minimize the sympathetic response (e.g. increase heart rate and therefore oxygen demand) to stress.
- Periods of low surgical stimulation (sterile preparation and draping, conduit harvest) may require hemodynamic support to maintain perfusion of the coronary arteries.

Table 18.1 Stages of the procedure with anesthetic considerations

Stage of care	What is happening to the patient	Anesthetic considerations
Preinduction and induction of anesthesia	Line placement, intubation	Maximize oxygen supply Maintain MAP Slow HR Oxygenation of blood Minimize oxygen demand Slow HR Avoid hypertension
Patient positioning	Low levels of stimulation	Blood pressure support may be required Maintain adequate anesthetic depth to avoid increase in HR or MAP
Skin Incision/ sternotomy	Intense surgical stimulation	Preemptively deepen the anesthetic (opioids, volatile agent) prior to incision
Conduit harvest	Low levels of stimulation	Blood pressure support may be required
Cannulation	Fluctuations in blood pressure or arrhythmias during cardiac manipulation	Observe surgical field closely to identify possible causes of hypotension Maintain systolic BP below 100 mm Hg for aortic cannulation
Cardiopulmonary bypass	The perfusionist is controlling hemodynamics	The perfusionist may ask for a vasoactive infusion to help control blood pressure
Weaning from bypass	Cardiac function is assessed Cardiac output is transferred from the CPB machine back to the patient Protamine is administered	Correct anemia Correct hypocalcemia Correct acidosis Check pacemaker and pace if needed to maintain cardiac output Be prepared to manage a protamine reaction
Case completion	Chest tubes are placed Chest is closed	Repeat TEE examination to assess cardiac function Anticipate decrease in preload after chest closure as intrathoracic pressure increases
ICU transport	Patient begins to emerge from anesthesia	Changes in blood pressure and heart rate should be anticipated and managed

Further Reading

Ardehali A, Portis TA. Myocardia oxygen supply and demand. Chest. 1990;98(3):699–705.
Kaplan J. Cardiac anesthesia for cardiac and non-cardiac surgery. Philadelphia: Elsevier; 2016.
Shroyer AL, Hattler B, Wagner TH, Collins JF, Balts JH, Quin JA, Almassi GH, Kozora E, Bakaeen F, Cleveland JC Jr, Bishawi M, Grover FL. Five-year outcomes after on-pump and off-pump coronary-artery bypass. NEJM. 2017;377(7):623–32.

19 Anesthetic Management and Surgical Considerations for the Patient Undergoing Off-Pump Coronary Artery Bypass Grafting

Kinjal Patel, Jia Weng, Katherine McMackin, Ronak Desai, Richard Highbloom, and Keyur Trivedi

To be or not to be (on cardiopulmonary bypass)?

—William Shakespeare (with liberties…)

Key Points

- Perioperative monitoring
- Induction and maintenance
- Management during grafting
- Case completion

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Introduction

Coronary artery bypass grafting (CABG) was first developed in the 1950s and matured as a routine surgical technique in the 1960s. During its infancy, most coronary bypass surgeries were performed on a beating heart. However, with the development of cardiopulmonary bypass (CPB) and cardioplegia in the 1970s, on-pump CABG became the gold standard. Beginning in the 1990s there was a renewed interest in off-pump CABG (OPCAB) due to the perceived benefits that might be gained by avoiding CPB.

The OPCAB technique avoids interaction of blood elements with the foreign surfaces of the heart–lung machine which can trigger an overexpression of inflammatory mediators. This approach also avoids the requirement for cannulation of the aorta. Aortotomy and placement of a large bore cannula risks injury to the vessel or embolic stroke in patients with severe aortic atheromatous disease.

Studies have demonstrated that OPCAB is associated with a reduction in postoperative pathophysiology including systemic inflammatory response, coagulopathy associated with platelet dysfunction, and organ dysfunction (myocardial, cerebral, renal). OPCAB may also be associated with a decrease in costs during the initial hospitalization. Disadvantages of OPCAB include a postoperative prothrombotic state that may lead to graft thrombosis, the potential for incomplete revascularization due to limited surgical access to regions of the heart, and decreased patency rate for venous bypass grafts.

Indications

OPCAB can be performed safely in a wide range of patients. Common indications for OPCAB include: poor LV function, preoperative renal or liver dysfunction, severe calcification or atheromatous disease of the aorta where aortic clamping is contraindicated, distribution of native coronary disease requiring limited bypass grafting, and obstructive coronary disease limited to vessels easily accessible on a beating heart. Surgical expertise in performing OPCAB also plays a significant role in the decision to use a beating heart technique rather than employing CPB.

Perioperative Monitoring

Please refer to Chaps. 8 “Cardiac Operating Room Setup” and 9 “Intraoperative Hemodynamic Monitoring” for more detailed discussion.

Intraoperative Monitoring

ASA standard monitoring, arterial catheterization, and central venous catheterization are routinely used during OPCAB. It should be expected that the EKG amplitude and QRS complex direction will vary greatly throughout the case due to ongoing repositioning of the heart within the chest cavity.

Near-Infrared Spectroscopy (NIRS) or Cerebral Oximetry

Global cerebral perfusion may be compromised due to extremes of positioning and hemodynamic state during OPCAB. Cerebral oximetry may help detect changes in regional blood oxygen saturation in the brain due to embolic events or decreased perfusion pressure during OPCAB.

Activated Clotting Time (ACT)

The ACT goal for OPCAB varies from institution to institution and may range from >250 seconds to >400 seconds.

Glucose

Intraoperative glucose management is discussed in Chap. 16.

Transesophageal Echocardiogram (TEE)

Cardiac function can be effectively monitored during OPCAB using TEE. New regional wall motion abnormalities are the most sensitive indicator of myocardial ischemia and may prompt immediate revision of the surgical plan when detected (Fig. 19.1). Imaging may be limited at times during OPCAB due to extreme cardiac positioning and the presence of sponges or air between the heart and the probe.

Temperature

The importance of maintaining normothermia can be easily overlooked during OPCAB. The development of hypothermia during the initial phases of cardiac

Fig. 19.1 Image of anesthesia provider performing TEE exam

surgery is common due to extensive exposure and skin preparation with alcohol-based antiseptic solutions. During OPCAB in particular, the absence of a CPB heat-exchanger and limited body surface area available for forced air warming makes maintaining normothermia challenging.

The operating room should be warm prior to patient arrival. Underbody warming devices or forced air warming blankets positioned under the surgical drapes should be used. After the induction of general anesthesia, the patient should remain covered at all times except when exposure is required for procedures or the application of skin antiseptics. Intravenous fluids should be warmed. A hypothermic patient may display signs of coagulopathy, may shiver (leading to increased myocardial oxygen consumption), may be predisposed to developing ventricular arrhythmias, and may experience delayed emergence in the ICU.

Induction and Maintenance

Induction of general anesthesia for cardiac surgical patients historically used high doses of opiates and benzodiazepines. This approach was favored because it avoided the negative inotropic effects and vasodilation associated with other anesthetic agents. However, the high-dose narcotic technique has been associated with prolonged intubation postoperatively. For this reason, contemporary clinical practice utilizes limited doses of benzodiazepines and opiates in combination with shorter acting induction agents such as propofol or etomidate to facilitate induction.

Maintenance of anesthesia is achieved with volatile anesthetics such as sevoflurane or isoflurane. Muscle relaxation with an intermediate duration nondepolarizing agent facilitates endotracheal intubation and sternal displacement for internal mammary harvest. Care should be taken not to overdose muscle relaxants, particularly in the latter stages of the procedure to avoid prolonged postoperative intubation.

Patient Positioning

The patient's arms will be tucked at their side for standard sternotomy and will be generally inaccessible. It is important to check all lines and monitors, including arterial line, pulse oximeter and peripheral IVs for appropriate functionality prior to surgical draping. A bump (shoulder roll) is placed under the patient's shoulders to improve access to the chest for sternotomy. The patient's head must be carefully supported to avoid hyperextension of the cervical spine. After positioning has been completed, the anterior chest as well as both groins and legs are prepared with the application of sterile antiseptic. These areas are then isolated with surgical drapes to allow for femoral vessel access and saphenous vein harvesting during the procedure.

Hemodynamic Targets

The balance between myocardial oxygen supply and myocardial oxygen demand must be maintained during anesthesia for OPCAB. Although this same principle applies universally to all intra-operative care, the challenge may be particularly acute during OPCAB surgery. Overall objectives should include maintenance of coronary (diastolic) perfusion pressure, a low to normal heart rate, and avoidance of severe hyper- or hypovolemia. Adequate depth of anesthesia must be ensured prior to intubation, TEE placement, skin incision, and during sternotomy. Tachycardia in response to these stimulating events will increase myocardial oxygen demand and must be avoided.

Sternotomy and Conduit Harvest

After surgical draping a midline skin incision is performed extending from below the sternal notch to the xiphoid process followed by sternotomy. Hypertension should be avoided during and after sternotomy to minimize blood loss.

The most common conduit for CABG is the left internal mammary artery (Fig. 19.2). It is commonly prepared as a pedicle graft maintaining its inflow from the left subclavian artery (Figs. 19.3 and 9.4). A right internal mammary artery harvest can be accomplished through the same incision with similar technique. Entering the pleural space during the mammary artery harvest is common and sometimes intentional. An open pleural space will allow drainage of any effusions, confirmation of appropriate mechanical ventilation, and the ability to position the heart without restriction.

Concurrently with the preparation of the internal mammary artery, the greater saphenous vein may also be harvested (Fig. 19.5). This is performed either by an open surgical dissection or endoscopic approach. Endoscopic vein harvest

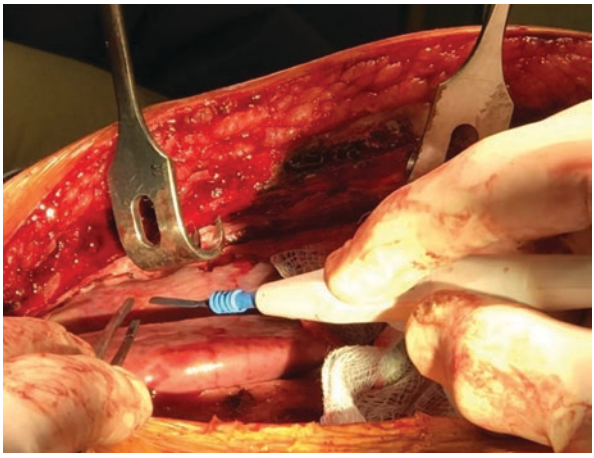

Fig. 19.2 Image of chest wall prior to left internal mammary artery harvest

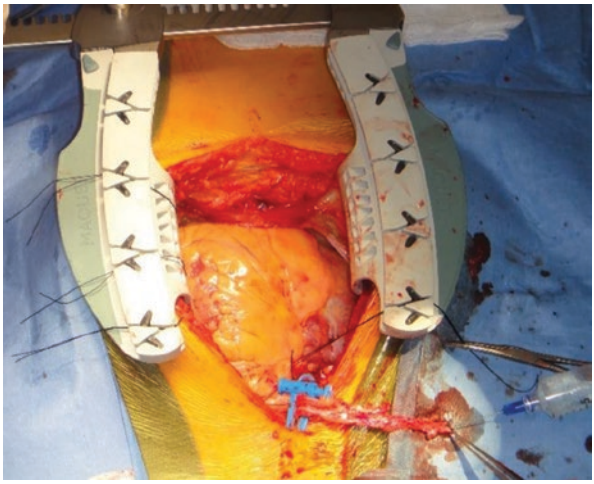

Fig. 19.3 Image: left internal mammary artery after harvest

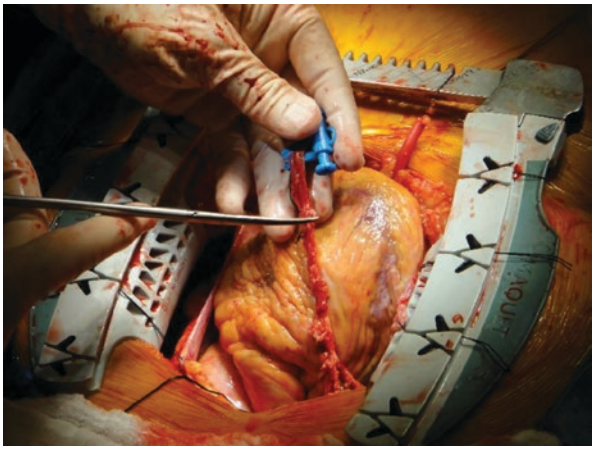

Fig. 19.4 The mammary artery is measured for appropriate length prior to anastomosis

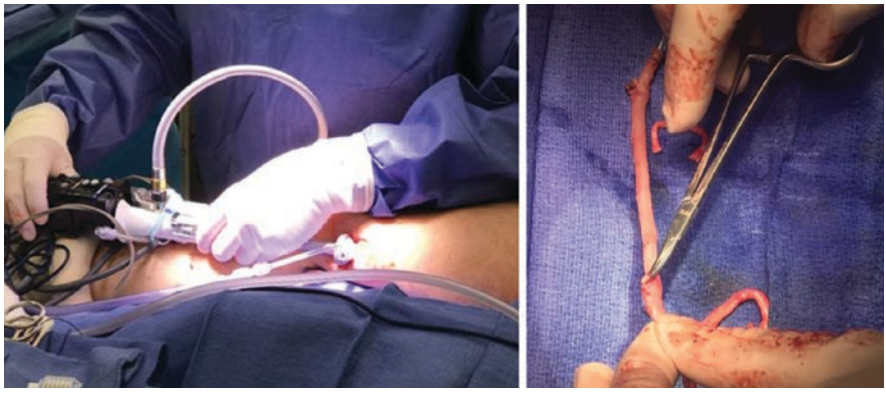

Fig. 19.5 Left: Endoscopic saphenous vein harvest. Right: Preparing the vein for grafting

commonly requires insufflation of the subcutaneous fat layer with CO2 which may lead to systemic hypercarbia. Hemodynamic parameters and $EtCO_2$ should be closely monitored. Radial artery harvest can also occur concomitantly either via an open or endoscopic approach.

Coronary Anastomoses

Once sufficient bypass conduit has been secured, the distal anastomosis sites are identified. Systemic heparinization is administered prior to beginning any vascular anastomoses. An ACT of 250 seconds or greater is sufficient to prevent intravascular thrombosis during arterial occlusion. However, establishing that the patient is not heparin resistant and that an ACT >400 seconds can be achieved quickly when needed provides reassurance should conversion to CPB be emergently required.

Distal Anastomoses

After the location and sequence of coronary anastomoses has been established, the heart is positioned to allow surgical access to the epicardial vessels (Fig. 19.6). One or more sutures are placed at the posterior pericardial reflection and are pulled taut to elevate the heart within the chest cavity.

Surgical sponges may be placed in the pericardial space to augment positioning. Finally, an apical suction device on an articulated arm attached to the sternal retractor is commonly used to improve access during grafting to the obtuse marginal or posterior descending arteries. Positioning of the heart commonly distorts the mitral and/or tricuspid annuli causing new regurgitation visible on TEE examination. Trendelenburg positioning, intravenous fluid administration, and vasopressor use are all commonly employed to augment preload and afterload and to minimize the

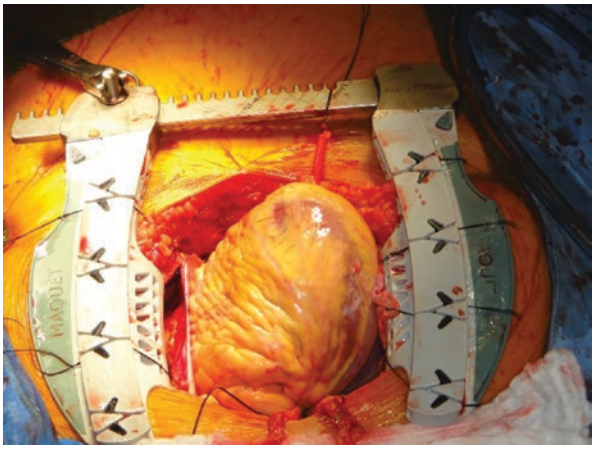

Fig. 19.6 The heart is elevated and positioned for distal anastomosis

effects of reduced stroke volume during this phase of surgery. The graft to the left anterior descending (LAD) artery is usually performed first (Fig. 19.7).

The LAD is grafted first because it can be exposed with minimal manipulation of the heart and revascularizing the LAD distribution will often be of greatest benefit to the patient. The distribution of LAD blood flow, including collateral flow to other coronary artery systems, may provide significant protection from ischemia during subsequent coronary anastomoses.

As each coronary segment targeted for revascularization is identified an epicardial stabilization device is applied to minimize cardiac wall motion in order to facilitate grafting (Fig. 19.8). These devices commonly employ suction ports on their epicardial surfaces to lift the tissues to which they are applied and minimize compression of the underlying ventricular chamber.

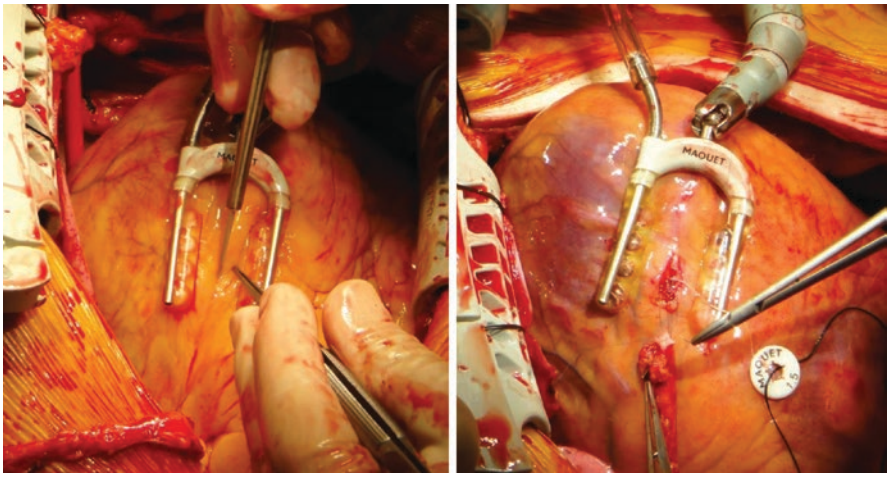

Fig. 19.7 Images: Distal anastomosis using mammary arterial graft to the left anterior descending

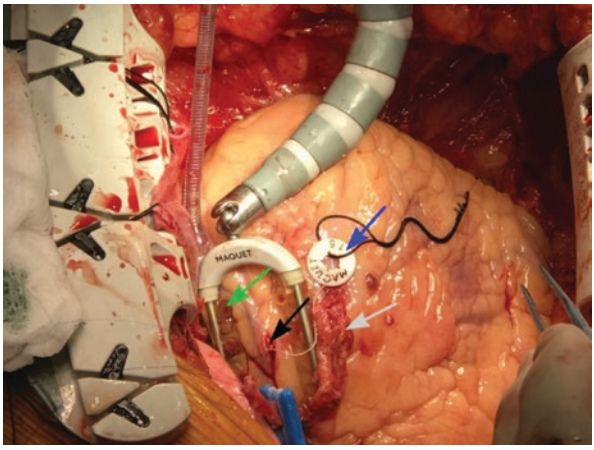

Fig. 19.8 Distal anastomosis of arterial grafts: blue arrow showing coil flow shunt pull device, white arrow showing mammary to LAD, black arrow showing suturing of a conduit to diagonal vessel with shunt inside, green arrow showing suction cups on the stabilization device

Once the surgical field is stable, the coronary artery is either occluded to prevent blood flow or is incised and an intra-coronary shunt placed to maintain blood flow during the grafting process. Vigilance during this stage is essential to ensure early detection of myocardial ischemia. If ischemia is detected by EKG or TEE, the impact on overall hemodynamics must be quickly assessed. If it is determined that adequate perfusion pressure can be maintained, short periods of ischemia during the creation of a distal anastomosis may be tolerated without permanent myocardial injury.

Proximal Anastomoses

Those conduits that will function as free grafts (saphenous vein, radial artery, free right internal mammary artery) require creation of a proximal anastomosis to the ascending aorta. Radial artery grafts often receive their arterial inflow from an end-to-side anastomosis with an internal mammary artery graft. The proximal anastomosis for a saphenous vein graft is usually located on the ascending aorta a few centimeters distal to the sino-tubular junction. Completion of a proximal anastomosis with the aorta requires temporary restriction of blood flow to the involved region of the aortic wall. This may be achieved with either a partial aortic clamp or specialized temporary occlusion devices (Fig. 19.9). A variety of devices that allow for saphenous vein to aorta anastomosis without side-clamping are available, including the HEARTSTRING system.

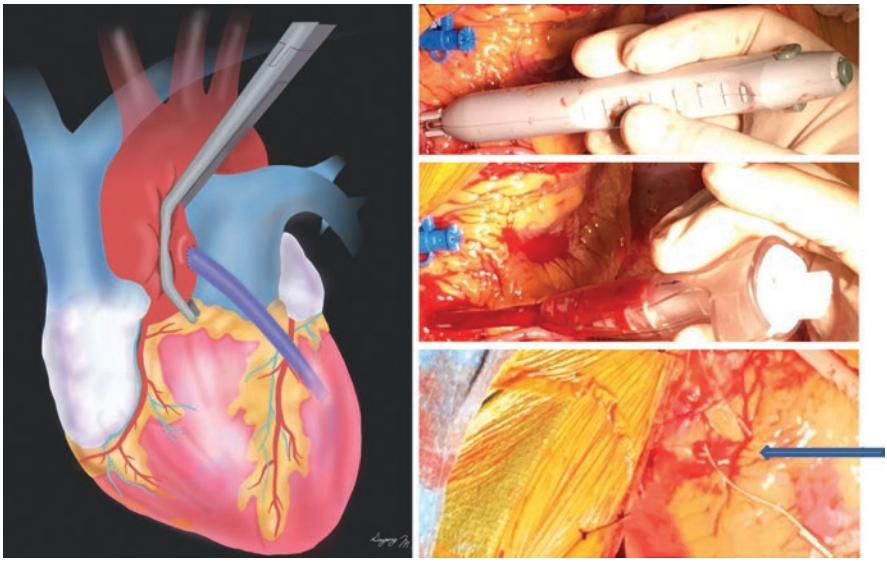

Fig. 19.9 Images of proximal anastomosis. Left: Illustration of side biting clamp. Right top: Aortic punch Right middle: Temporary partial occlusion seal device. Right bottom: Deployed seal with stem and tension spring (arrow)

Completion of the proximal anastomoses with either technique requires a reduction in systolic arterial pressure to less than 100 mmHg until the anastomosis is complete. This relative hypotension both minimizes the leakage of blood around the occluding device, allowing for a cleaner surgical field, and reduces the likelihood of injuring the aorta during the required manipulations.

Case Completion

Once all planned bypass grafts have been completed, protamine should be administered at a ratio of 1 mg per 100 units heparin. Protamine should be dosed slowly by infusion or small intermittent boluses to minimize the risk of hypotension due to histamine release, and to allow early detection of a pulmonary hypertensive or anaphylactic response. ACT measurement following protamine should confirm a return to baseline levels. TEE should be assessed to ensure that no new regional wall motion abnormalities have developed after coronary bypass grafting. Monitoring the EKG for any ST segment elevations or depressions can also help alert the team to any potential graft dysfunction.

Transition to postoperative ICU care is the final phase of anesthetic management for OPCAB. In healthy patients for whom the operative period was unremarkable, early extubation in the operating room is increasingly common. If this option is selected, the normal thresholds for extubation must be satisfied – hemodynamic stability, normothermia, adequate respiratory function, complete reversal of neuromuscular blockade, and recovery of a sufficient level of consciousness to ensure airway protection.

If the patient is to be transferred to the ICU while remaining intubated and ventilated the transition from surgical anesthesia to postoperative sedation must be carefully managed. Excessive sedation may result in hypotension and compromised flow in the newly created bypass grafts, as well as delayed emergence from anesthesia. Inadequate sedation or analgesia may lead to severe hypertension and agitation, in turn increasing the likelihood of postoperative bleeding. Overdosage of muscle relaxants can also lead to delayed postoperative extubation. Judicious titration of anesthetic agents during the final stages of the surgical procedure and a smooth transition to short-term reversable sedation (propofol, dexmedetomidine) allows for the rapid recovery and early extubation expected of OPCAB patients. Stages of the procedure with anesthetic considerations are listed in Table 19.1.

Clinical Pearls

- Off pump CABG is an alternative to CABG with cardiopulmonary bypass, especially for patients with severe comorbidities.
- Maintenance of hemodynamic stability during cardiac manipulation may be achieved via increased fluid administration or transient vasopressor use.
- Anesthetic goals should include maintaining myocardial oxygen balance prior to revascularization - decreasing myocardial oxygen consumption and/or increasing myocardial oxygen supply.

- Periods of myocardial ischemia during surgical graft anastomosis occur, and diligent monitoring for regional wall motion abnormalities is necessary.
- Temperature maintenance is critical and must be vigilantly monitored to ensure avoidance of hypothermia and its related side effects.
- Use of short-acting agents such as propofol and dexmedetomidine when transitioning from anesthesia to postoperative sedation allows for the rapid recovery expected of OPCAB patients.

Table 19.1 Stages of the procedure with anesthetic considerations

Stage of care	What is happening to the patient	Anesthetic considerations
Preinduction and induction of anesthesia	Standard ASA Monitors Arterial line – pre induction Central line ± PAC Cerebral oximetry TEE ACT monitoring Temperature management plan Intubation	Maximize oxygen supply Maintain MAP Oxygenation of blood Minimize oxygen demand Slow HR Avoid hypertension
Patient positioning	Low levels of stimulation	Blood pressure support may be required Maintain adequate anesthetic depth to avoid increase in HR or MAP
Skin incision/ sternotomy	Intense surgical stimulation	Preemptively deepen the anesthetic (opioids, volatile agent) prior to incision
Conduit harvest	Low levels of stimulation	Blood pressure support may be required
Distal graft anastomosis	Fluctuations in blood pressure or arrhythmias during cardiac manipulation Monitor for ischemia - ST depressions/elevations Regional wall motion abnormalities can be detected via TEE	Administer heparin and check ACT Observe surgical field closely to identify possible causes of hypotension Maintain systolic BP above 100 mm Hg Administering IV fluids & albumin Adjust Trendelenburg position Administering vasopressors Monitor urine output Maintain myocardial oxygen supply Avoid increase in myocardial oxygen demand Be prepared to go on bypass
Proximal graft anastomosis	Aortic puncture or aortic side-clamping Cardiac function is assessed via Cardiac Index ST depressions/elevations can be a sign of graft dysfunction or introduction of air in the grafts	Observe the heart directly to assess volume status and correct anemia Avoid hypertension during aortic puncture and aortic side clamping Global and regional wall motion abnormalities/volume status can be detectable via TEE Correct acidosis

(continued)

Table 19.1 (continued)

Stage of care	What is happening to the patient	Anesthetic considerations
Grafts are done	Deairing of grafts and removal of distal clamps Placing pacing wires if needed Cardiac function is assessed ST depressions/elevations can be a sign of graft dysfunction or air in the grafts Protamine is administered	Check pacemaker and pace if needed to maintain cardiac output Global and Regional wall motion abnormalities can be detectable via TEE Be prepared to manage a protamine reaction
Case completion	Chest tubes are placed Chest is closed	Repeat TEE examination to assess cardiac function to check global and regional wall abnormalities, and presence of blood collection in thoracic and pericardial cavities Anticipate decrease in preload after chest closure as intrathoracic pressure increases
ICU transport	Patient begins to emerge from anesthesia	Changes in blood pressure and heart rate should be anticipated and managed. Don't overdose with benzodiazepines/opiates Don't overdose with muscle relaxant Use propofol or dexmedetomidine for sedation into the postoperative period

Further Reading

Gravlee GP, Shaw AD, Bartels K. Alternative approaches to cardiothoracic surgery with and without cardiopulmonary bypass. In: Hensley's practical approach to cardiothoracic anesthesia. 6th ed. Philadelphia: Wolters Kluwer Health; 2018.

Hannan EL, Samadashvili Z, Wechsler A, Jordan D, Lahey SJ, Culliford AT, et al. The relationship between perioperative temperature and adverse outcomes after off-pump coronary artery bypass graft surgery. J Thorac Cardiovasc Surg. 2010;139:1568–75.

Hattler B, Messenger JC, Shroyer AL, Collins JF, Haugen SJ, Garcia JA, Baltz JH, Cleveland JC, Novitzky D, Grover FL. Off-pump coronary artery bypass surgery is associated with worse arterial and saphenous vein graft patency and less effective revascularization: results from the Veterans Affairs Randomized On/Off Bypass (ROOBY) trial. Circulation. 2012;125:2827–35.

Lamy A, Devereaux PJ, Prabhakaran D, Taggart DP, Hu S, Straka Z, et al; CORONARY Investigators. Five-year outcomes after off-pump or on-pump coronary-artery bypass grafting. N Engl J Med 2016;375(24):2359–2368.

Anesthesia for Endovascular Thoracic Aortic Aneurysm Repair (TEVAR)

20

Yvonne Fetterman, Karuna Puttur Rajkumar, and Y. Yuliana Salamanca-Padilla

The secret of science is to ask the right question.

—Sir Henry Tizard

Key Points

- Preoperative Evaluation
- Anesthetic Goals
- Anesthetic Choice
- Intraoperative Monitoring
- Intraoperative Management
- Postoperative Care
- Spinal Cord Ischemia
- Temperature Management
- Emergent/Urgent Repair Considerations
- Prepare for Disaster

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Introduction

Thoracic endovascular aortic aneurysm repair (TEVAR) was initially introduced as a less-invasive alternative to open repair in patients (Fig. 20.1) whose medical comorbidities rendered them poor surgical candidates. However, it has now become a popular option for many patients with thoracic aneurysms regardless of their physical status.

Thoracic endovascular aortic repair is presently the standard of care for Type B dissections due to the lower risk of procedural complications compared with open repairs. Whenever TEVAR is performed, conversion to an open or hybrid procedure may become necessary intraoperatively. This requires that the preoperative evaluation should be as stringent as that for open repair.

TEVAR utilizes a percutaneous technique or surgical cutdown for vascular access. As a result, it may be performed with local anesthetic and sedation, or with neuraxial techniques if patients are able to tolerate a supine position for an extended period. Some anesthesiologists, however, prefer general anesthesia for reasons described later in this chapter. The choice of anesthetic is best decided on a case-by-case basis after considering the case-specific surgical requirements and the patient's comorbidities. Neurologic injury due to ischemia, embolization, or vascular occlusion by the endograft is the most feared complication of TEVAR. Precise blood pressure management before, during, and after endograft deployment is critical.

Preanesthetic Evaluation

TEVAR is considered an intermediate risk procedure with up to 1% risk of a major adverse cardiac event (MACE). Most patients undergoing TEVAR will have several other major cardiovascular risk factors including hypertension, coronary artery disease, cerebral vascular disease, diabetes, smoking, or chronic obstructive pulmonary disease. Chapter 5 discusses preoperative testing for cardiac and vascular procedures in general. A thorough discussion of the planned approach with the surgeon is essential because of the unique nature of each TEVAR procedure. The information sought should include an understanding of intraoperative hemodynamic surgical requirements as well as the planned configuration of vascular access for intervention, monitoring, and volume replacement.

Preoperative Renal Function

The risk of postoperative renal dysfunction is significant after TEVAR. Baseline renal dysfunction, preoperative angiography with nephrotoxic agents, and embolic injury to the kidneys secondary to dislodgement of atherosclerotic plaque by the intraarterial cannulas and deployment system are all contributing factors. Patients should understand the risk of kidney injury, the potential consequences, and be included in perioperative decision making. Accepted protective maneuvers include volume expansion with normal saline or isotonic sodium bicarbonate, and the use of

low volume iso-osmolar contrast during the procedure. Other interventions with less supporting evidence include the administration of Vitamin C or N-acetyl cystine before contrast load. Diuretics and ACE inhibitors should be used with caution in the perioperative period.

Preoperative Neurologic Assessment

TEVAR carries a risk of neurologic injury due to spinal cord ischemia, embolic events (stroke), or stent occlusion of critical vascular structures. A comprehensive neurologic exam should be performed preoperatively and preexisting neurological deficits should be clearly documented.

Drainage of cerebral spinal fluid both intra-operatively and during the immediate postoperative period is frequently used to increase spinal cord perfusion pressure (SCPP = mean arterial pressure – cerebrospinal fluid pressure) in patients at high risk for spinal cord ischemia, or once symptoms suggestive of spinal cord ischemia are encountered (Fig. 20.1). If the use of a spinal drain is planned, patients should be counseled to stop any chronic anticoagulants prior to their surgery according to current guidelines. (See Appendix 2 – Spinal Cord Ischemia and Stroke).

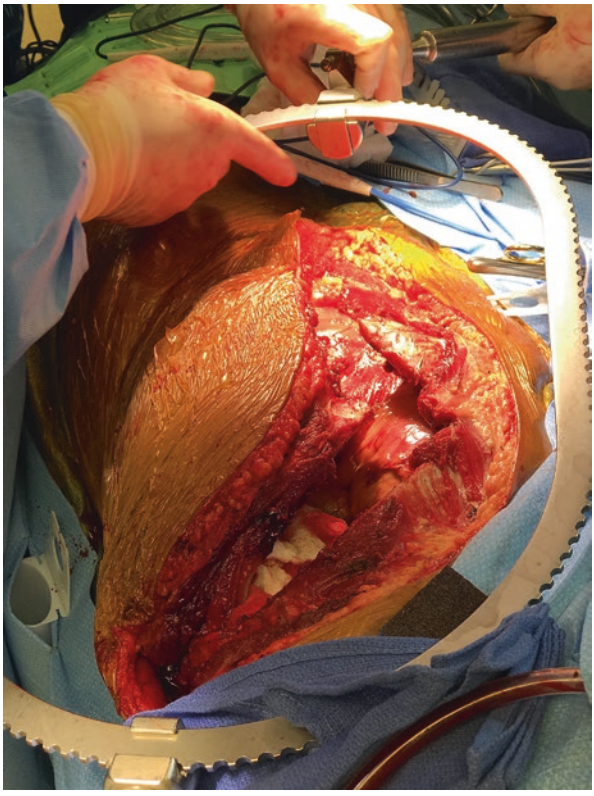


Fig. 20.1 *Image* showing how *extensive* surgical incision in open repair

Anesthetic Goals

- *Facilitating the patient's ability to lie still in the supine position for 1–3 hours* – movement during stent deployment can lead to catastrophic complications.
- *Prevention of contrast-induced nephropathy with adequate hydration* – optimize hemodynamic and intravascular volume status to minimize the risk of kidney injury
- *Anticoagulation with serial activated clotting time (ACT) monitoring* – minimize the risk of thromboembolic complications through adequate anticoagulation while instrumenting the arterial system
- *Strict blood pressure control before during and after stent-graft deployment* – critical to ensure proper placement of the stent graft
- *Temperature control* – wide exposure and minimal access to body surfaces to affect warming may lead to significant hypothermia
- *Preparation for massive transfusion protocol should a rare but significant bleeding event occur* – ensure adequate vascular access has been secured and blood products are immediately available

Anesthetic Choice

There are no randomized control trials comparing anesthetic techniques for TEVAR. Local or regional techniques may be more appropriate for procedures limited to the descending aorta. General anesthesia permits the use of TEE which allows for intraoperative assessment of cardiac function, evaluation of the aorta to determine safe landing zones for endograft positioning (although intravascular ultrasound can also be utilized for this purpose), and evaluation of potential endoleaks after graft deployment.

TEVAR requires intraoperative heparinization which, when combined with neuraxial techniques, may increase the risk for epidural hematoma or spinal hemorrhage. Use of a spinal fluid drain may be incompatible with a neuraxial anesthetic approach. Further, neuraxial blockade interferes with neuromonitoring and assessment of neurological function in the immediate postoperative period.

The overall procedural setup must be carefully considered if the planned anesthetic does not include an endotracheal tube and controlled ventilation. If a rapid conversion to general anesthesia is required intraoperatively both the position of the fluoroscopy equipment and the configuration of the sterile field may impede rapid access to the patient.

Some surgical techniques may require large doses of adenosine or rapid ventricular pacing to create stasis in the aortic arch for up to 60 seconds during endograft positioning. This may result in discomfort in an awake patient. Potential advantages of different types of anesthesia techniques are summarized in Table 20.1.

Intraoperative Monitoring

During TEVAR, there is potential for either planned or inadvertent occlusion of the left subclavian artery. Therefore, the arterial line should be placed in the right arm

Table 20.1 Summary of potential advantages of local with MAC/regional/neuraxial techniques vs. general anesthesia

Local anesthesia, MAC, regional, neuraxial	General anesthesia
Awake patient: Local anesthesia w/MAC allows early detection of intraoperative spinal cord ischemia Early detection of aneurysm rupture due to complaints of back pain Early detection of anaphylactic reactions – complaints of pruritis, tongue swelling, bronchospasm, etc.	Guarantees an immobile patient Allows for intraoperative TEE – especially helpful in repair of aortic arch Avoids risk of epidural hematoma or spinal hemorrhage due to spinal or epidural placement Secured airway prior to positioning of C-arm. C-arm can interfere with airway access.
Decreased catecholamine-induced hypertension during induction (laryngoscopy) and emergence	Allows complete interruption of respiration or during stent deployment
Decreased myocardial depressant effects of general anesthesia	Allows use of adenosine or rapid pacing if required during graft deployment
Avoidance of hemodynamic effects associated with positive pressure ventilation	Decreased bowel peristalsis allowing better intraoperative imaging
Decreased risk of pulmonary complications associated with positive pressure ventilation	Can facilitate retroperitoneal access if femoral or iliac access is not possible

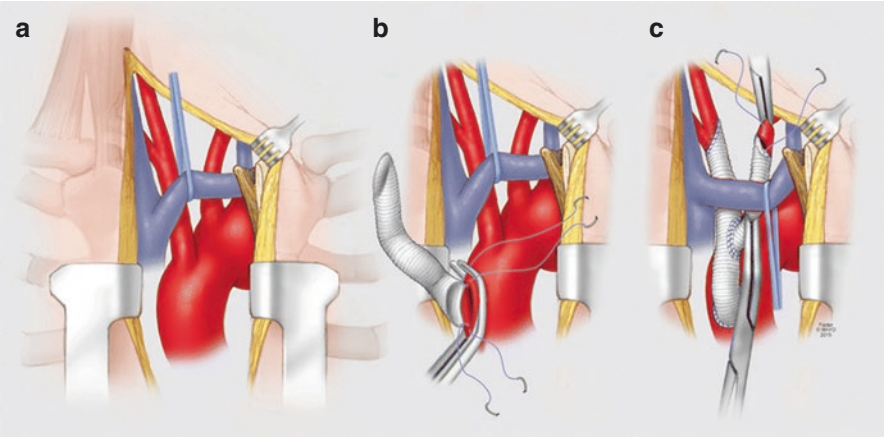


Fig. 20.2 Illustration of aortic arch debranching which may make CVC monitoring difficult or impossible. (https://link.springer.com/chapter/10.1007/978-3-319-15192-2_36)

whenever possible. Right internal jugular central venous or pulmonary artery catheter placement may not be possible when performing arch repair if the surgical field requires cutdown of the bilateral carotid arteries (Fig. 20.2). At least two large bore peripheral intravenous catheters should be placed and remain accessible. TEE may be particularly helpful in patients with coronary artery disease or those undergoing arch repair, and may allow for early detection of complications such as aortic dissection after stent deployment. If neurological monitoring with somatosensory evoked potentials (SSEPs) or motor evoked potentials (MEPs) are planned, avoid prolonged neuromuscular blockade and limit inhaled anesthetics to 0.5 MAC.

Intraoperative Management

TEVAR requires different hemodynamic goals than open repair. Relative hypotension is preferred immediately before and during stent-graft deployment. Short-acting vasodilators should be readily available during this portion of the case. Hypotension facilitates accurate positioning of the stent-graft and minimizes the potential of distal embolic phenomenon related to dislodgement of an atherosclerotic plaque or calcification. Other than during stent deployment, controlled hypertension is preferred during a totally endovascular procedure. This is because there is no risk of bleeding from a surgical anastomosis and hypertension minimizes the risk of spinal cord ischemia. If a hybrid procedure is planned, hemodynamic goals should be carefully discussed with the surgeon regarding blood pressure management during each portion of the procedure.

Immediately before stent deployment → controlled hypotension

The goal is accurate positioning of the stent-graft with low risk for movement during deployment

During stent deployment → controlled hypotension +/− cardiac arrest with adenosine or rapid ventricular pacing.

The surgeon may want the heart arrested or cardiac output minimized during stent deployment. High-dose adenosine or rapid ventricular pacing may be utilized for this purpose. This may be particularly helpful in stent-grafts which involve the aortic arch. Inadvertent coverage of the carotid arteries or left subclavian could cause catastrophic ischemic stroke or malperfusion syndrome. If either of these techniques is used to facilitate stent-graft positioning, be vigilant as arrythmia may occur following such maneuvers.

- *Adenosine Dose*: 36 mg will produce asystole for a period of 4–6 seconds. Subsequent doses of 18 mg will produce additional short periods of cardiac arrest.
- *Rapid Ventricular Pacing*: pace at 160 to 180 beats per minute via temporary transvenous pacing wire. The rapid pacing is stopped after deployment of the stent graft.

After deployment → controlled hypertension → perfuse the spinal cord

A variable number of radicular arteries that feed the spinal cord will have been occluded by stent deployment. It is imperative to maintain blood flow to the anterior spinal artery to prevent spinal cord ischemia and permanent motor deficits.

- Maintain MAP greater than 80–90 mmHg.
- Vasopressin or phenylephrine may be beneficial in patients with coronary artery disease as both will increase blood pressure without increasing heart rate.
- Drain CSF to 10–12 mmHg if there is evidence of spinal cord ischemia (evoked potentials or awake patient).

Postoperative Care

Following TEVAR the most important aspect of postoperative care is monitoring for new neurological deficits. Early detection and intervention may reduce or reverse long-term disability in certain patients, although not all. Hand-off to the ICU team

must include the mean arterial pressure goals. In addition, if a spinal drain is present a complete description of its intraoperative management should be provided and plans for postoperative care (drainage parameters, timing of removal) should be discussed.

Temperature Management

There is potential for significant unplanned hypothermia during TEVAR due to multiple factors. Large volumes of un-warmed intra-aortic saline irrigation and intravenous contrast may be administered. Depending on the procedural access points, a large proportion of the patient's body surface area may be exposed. For similar reasons there may be limited ability to provide external warming. Hypothermia increases the risk of coagulopathy leading to increased blood loss, transfusion requirements, infection, delayed emergence, and cardiac events. Careful efforts should be made to avoid hypothermia including prewarming the patient, increased ambient temperature, the use of forced air warming devices when possible, and warmed intravenous fluids.

Emergent/Urgent Repair Considerations

Emergency indications for thoracic aortic repair include ruptured degenerative aneurysm, complicated type B dissection, blunt trauma to the thorax, and relatively rarer conditions such as penetrating ulcers and intramural hematomas.

Ruptured Aneurysms

The incidence of the ruptures of aneurysms of the thoracic aorta is 5/100,000 and 30% involve the descending thoracic aorta. Open surgery with aneurysmectomy under extracorporal circulation is associated with a perioperative mortality rate approaching 45%. TEVAR provides more favorable postoperative outcomes compared to open repair, although conversion to open procedure may be required in a significant number of the cases.

Complicated Type B Dissections

Open surgical repair of acute complicated (involving end-organ ischemia) type B dissections has been associated with mortality as high as 50%. TEVAR has been applied to patients with acute complicated type B dissections with a 30-day mortality rate of just 4% and improved 5-year survival compared with open repair.

Traumatic Aortic Injury

Most patients with thoracic aortic injuries resulting from blunt trauma die before reaching the hospital. Open surgical repair among survivors is associated with a 25% mortality rate and 16% paraplegia rate. TEVAR has been applied as an

off-label treatment in this setting. The extent of graft coverage required is typically small compared with degenerative aneurysms or dissections. The Society for Vascular Surgery, citing weak evidence, suggests that TEVAR is associated with improved survival and lower complication rates in these patients.

Prepare for Disaster

Potential intraoperative disasters include rupture or dissection of the aneurysm as well as ischemic or embolic neurologic events. Preparation for these potentially catastrophic events is essential. Blood must be immediately available and vasoactive agents must be readily on hand.

Disaster Checklist

- Adequate access to blood products
- Vasopressors: epinephrine, norepinephrine, phenylephrine
- Antiarrhythmics: lidocaine, amiodarone, diltiazem
- Defibrillator pads applied
- Perfusionist aware and immediately available

Anesthetic management of TEVAR is summarized in Table 20.2.

Table 20.2 Summary of anesthesia management of TEVAR

Location of bypass	Chest via unilateral or bilateral groin access
Duration of operation	30–90 min
Anesthetic	General endotracheal anesthesia
Medications	5000+ units heparin at discretion of surgeon Notify at 3 min after administration Notify surgical team at 1 h after administration for possible re-dosing
Positioning	Supine Bilateral arm tuck
Blood loss	Variable
Case specifics	Lumbar drain for prevention of paraplegia, drain 10 cc prior to stent placement at discretion of surgeon Emergent cases do not require lumbar drain placement Arterial line preferentially in right radial, may cover left subclavian during case Patients with contrast allergy require 125 mg solumedrol prior to starting case No metal ET tube holder—interferes with imaging Breath holds required for abdominal imaging for accurate imaging of great vessels Percutaneous access requires patient to lie flat and keep access leg straight for 2–4 hours to prevent bleeding Maintain MAP 90–110 mmHg at end of case to ensure spinal perfusion May require additional bypass procedures prior to endograft placement if covering great vessels (i.e. carotid–subclavian or carotid–carotid bypass) Foley catheter for monitoring urine output
Modifications	Some patients may require arm-vein harvest, rendering that arm not useable for IV access
Postop care	Intensive care unit

Clinical Pearls

- Although rare, be prepared for rapid conversion to open procedure or massive blood loss.
- Be aware of, and plan for, potentially limited patient access during the procedure, particularly limited airway and IV access.
- Procedures involving the aortic arch have increased risk for coronary ischemia, cerebral ischemia, and ischemic syndrome from graft occlusion of the left subclavian
- Place arterial monitoring lines in the right arm as the left subclavian artery may become occluded during endograft deployment
- Before endograft deployment, controlled hypotension is instituted to decrease the risk of endograft malposition or drift during deployment. After endograft deployment, relative hypertension is preferred to maintain spinal cord perfusion pressure
- During hand off to the ICU team, be clear about hemodynamic management goals and handling of the spinal drain if present, as postoperative neurologic deficits are more common in TEVAR.

Appendix 1 – Surgical Nomenclature

Classification of Thoracoabdominal aortic aneurysm shown in (Fig. 20.3)
Extent I, distal to the left subclavian artery to above the renal arteries. *Extent II,* distal to the left subclavian artery to below the renal arteries; this is the most extensive type of aneurysm. *Extent III,* from the sixth intercostal space to below the renal arteries. *Extent IV,* from the twelfth intercostal space to the iliac bifurcation. *Extent V,* below the sixth intercostal space to just above the renal arteries[9].

Landing Zones shown in (Fig. 20.4):
Landing zones are the portions of the aorta where the proximal and distal ends of the endograft must create a seal with the aorta. These areas must be relatively free of calcification or thrombus and be at least 2 cm in length. The location of the landing zone has implications for the anesthesiologist. Embolic phenomena or coverage of a branch vessel can cause cerebral ischemia. Perioperative acute ischemic stroke is more common with TEVAR repair of the proximal aorta in landing zones 0–2. Other factors which increase risk of stroke are excessive wire and catheter manipulation in a diseased aortic arch, air embolism from deployment systems, and inadvertent coverage of arch branches.

Types of Repair shown in (Fig. 20.5)
TEVAR may involve a wide variety of purely percutaneous graft configurations, or it may involve a combination of endovascular stenting and open bypass grafting. The list below is not meant to be comprehensive. The examples provided illustrate

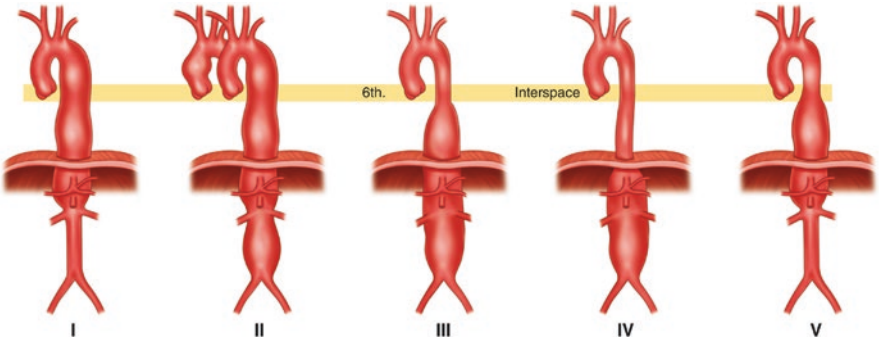


Fig. 20.3 Classification of thoracoabdominal aortic aneurysms. https://link.springer.com/chapter/10.1007/978-3-319-71300-7_17. Lee A., Dake M.D. (2018) Abdominal and Thoracic Aortic Aneurysms. In: Keefe N., Haskal Z., Park A., Angle J. (eds) IR Playbook. Springer, Cham

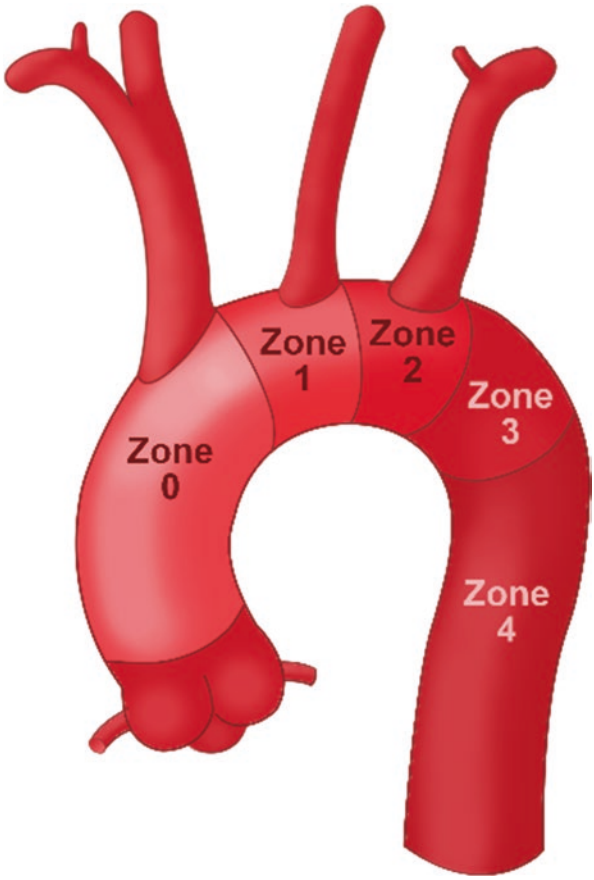


Fig. 20.4 Landing zones for TEVAR of Aortic Arch. *Drawing by Yvonne Fetterman, MD*

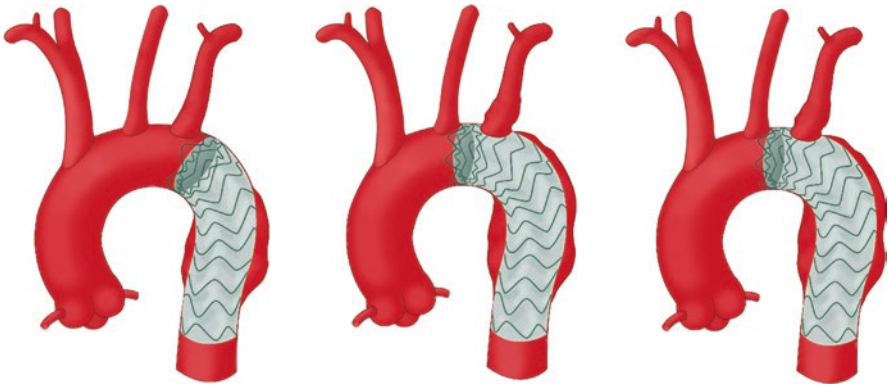


Fig. 20.5 TEVAR without SCA exclusion, TEVAR with SCA exclusion, and TEVAR with LCA exclusion with revascularization. *Drawing by Yvonne Fetterman, MD*

the fact that discussion with the surgical team is critical for successful anesthetic planning, particularly when the aortic arch branch vessels are involved. For instance, an aortic arch repair with chimney graft (see below) may require general anesthesia with TEE, while local anesthetic with sedation may be preferred for a Type B aortic dissection with landing zone distal to the left subclavian artery. TEVAR with 3 branch vessel aortic repair may require a wide surgical field including the bilateral carotid arteries and percutaneous access of bilateral groins which would preclude central venous catheter placement. Similarly, extra-thoracic debranching may also require a sterile surgical field which includes both carotid arteries if the right carotid is needed for revascularization of the branch vessels. This technique is becoming increasingly popular due to decreased morbidity compared to traditional debranching via medial sternotomy and aortic cross clamping.

TEVAR for Descending Thoracic Aorta +/— Left Subclavian Exclusion +/— Revascularization

Left subclavian artery revascularization is mandatory in patients who have undergone prior coronary artery bypass employing a left internal mammary artery graft shown in (Fig. 20.6).

If blood supply to the left subclavian is interrupted, flow to the internal mammary graft will also cease. – **BAD!**

Parallel Stent Grafts Shown in (Fig. 20.7) (Chimney/Periscope Stent Grafts)

This technique employs a smaller stent graft which runs *outside* and parallel to the larger main stent graft. The aortic end of the smaller graft protrudes into the aorta

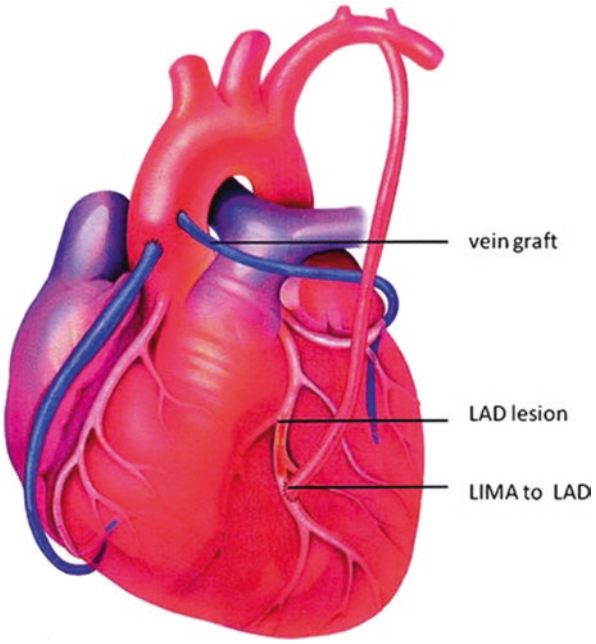


Fig. 20.6 Heart with prior CABG utilizing LIMA to left anterior descending (LAD) artery: https://link.springer.com/chapter/10.1007/978-1-4614-1475-9_22. Liao K. (2012) Surgical Treatment of Coronary Artery Disease. In: Vlodaver Z., Wilson R., Garry D. (eds) Coronary Heart Disease. Springer, Boston, MA

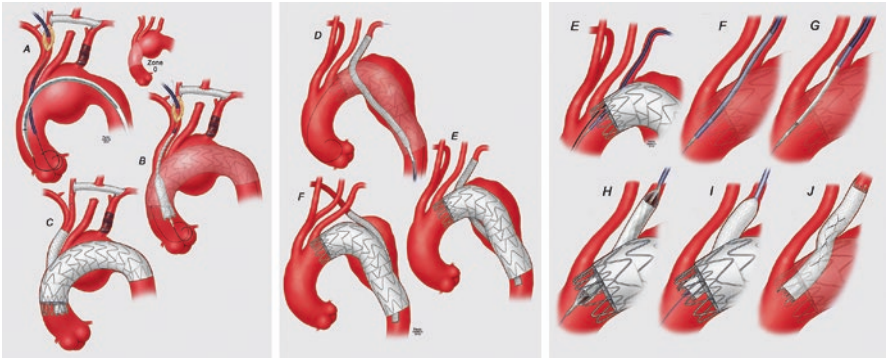


Fig. 20.7 Criado F.J. (2017) Parallel Stent Graft Techniques to Facilitate Endovascular Repair in the Aortic Arch. In: Oderich G. (eds) Endovascular Aortic Repair. Springer, Cham. https://link.springer.com/chapter/10.1007/978-3-319-15192-2_35

beyond the extent of the main graft and preserves flow into a branch vessel that would otherwise become occluded.

Branched and Fenestrated Endografts Shown in (Figs. 20.8 and 20.9)

Fenestrated grafts have openings in the main stent-graft which allow flow to branch arteries. From a catheter introduced into a branch vessel, smaller stent-grafts can be deployed so that their proximal end extends through the fenestrations. Branched grafts are manufactured with smaller diameter side-arms which branch directly off the main stent-graft.

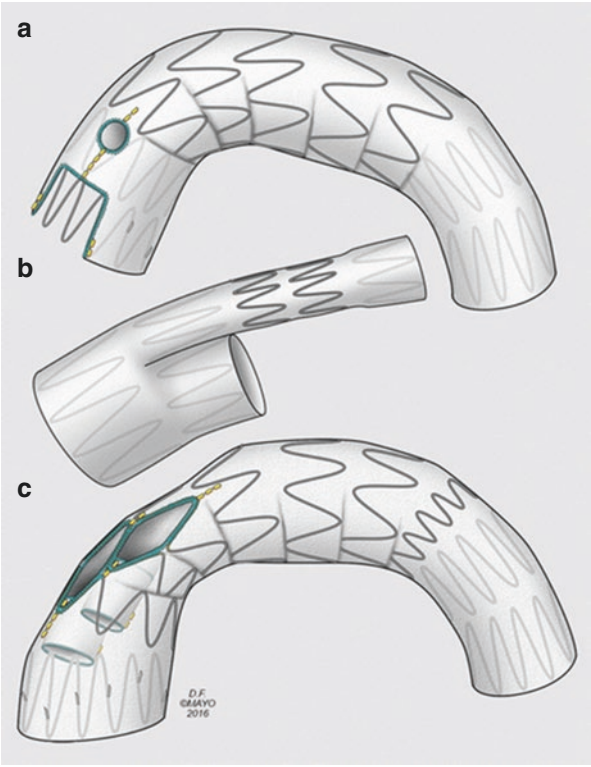


Fig. 20.8 Rana M.A., Oderich G.S. (2017) Current Device Designs to Incorporate Supra-aortic Arch Trunks for Endovascular Repair. In: Oderich G. (eds) Endovascular Aortic Repair. Springer, Cham. https://link.springer.com/chapter/10.1007/978-3-319-15192-2_32

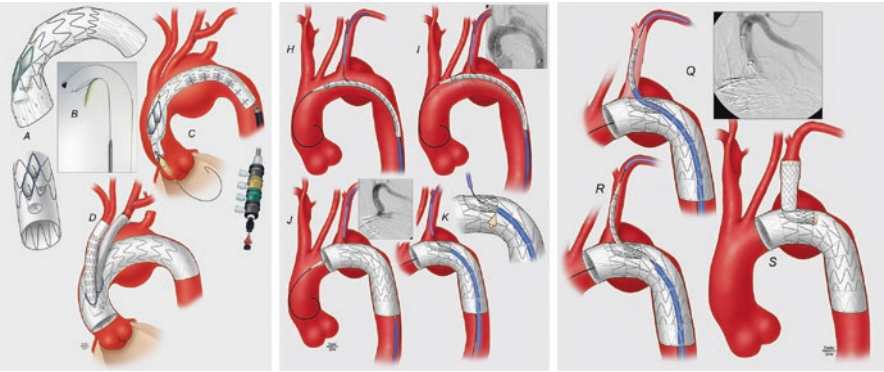


Fig. 20.9 Milne C.P.E., Haulon S., Oderich G.S. (2017) Technical Aspects and Results of Branched Endografts for Repair of Aortic Arch Aneurysms. In: Oderich G. (eds) Endovascular Aortic Repair. Springer, Cham. https://link.springer.com/chapter/10.1007/978-3-319-15192-2_33

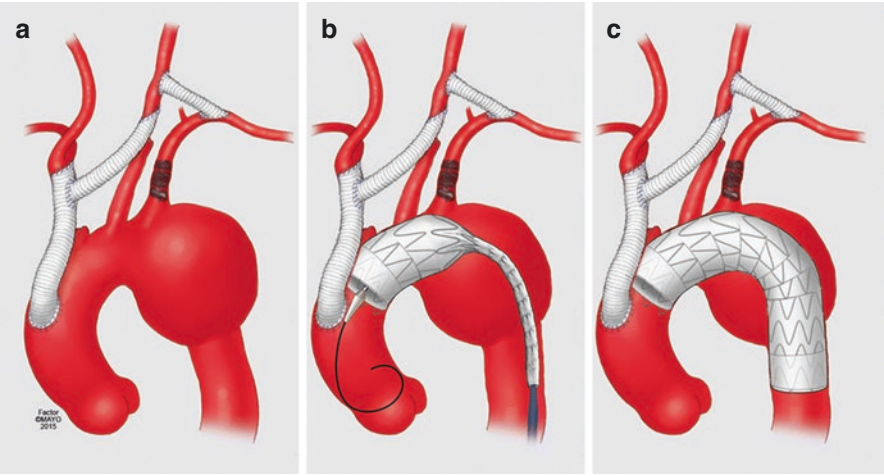


Fig. 20.10 Rana M.A., Oderich G.S., Pochettino A. (2017) Techniques and Results of Aortic Arch Hybrid Repair. In: Oderich G. (eds) Endovascular Aortic Repair. Springer, Cham. https://link.springer.com/chapter/10.1007/978-3-319-15192-2_36

Hybrid Arch Debranching Shown in (Fig. 20.10)

This technique requires median sternotomy, aortic cross clamping, and deep hypothermic circulatory arrest. The aortic branches are separated from the aortic arch and reattached at a more proximal disease-free location. The distal aortic arch is then repaired with TEVAR.

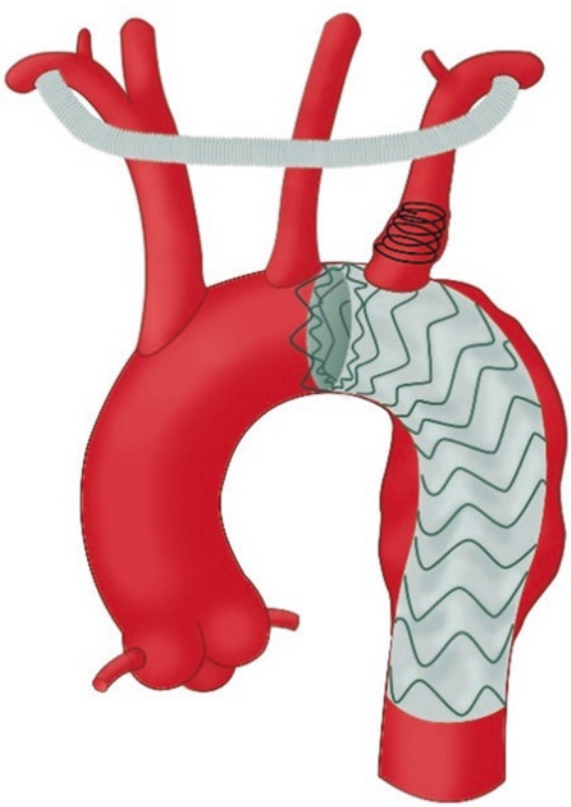


Fig. 20.11 TEVAR with Extra-thoracic debranching for planned left SCA exclusion. (*Drawing by Yvonne Fetterman, MD*)

Extra-thoracic Debranching Shown in (Fig. 20.11)

The benefit to this hybrid approach is that extra-thoracic debranching via open surgical repair does not require a sternotomy or deep hypothermic circulatory arrest.

Hybrid Arch Repair with Debranching, Elephant Trunk, and Endovascular Stent Graft Shown in (Fig. 20.12)

This surgical repair requires medial sternotomy, cardiopulmonary bypass and deep hypothermic circulatory arrest with aortic cross clamping. The proximal aorta is repaired with traditional graft. Near the midpoint of the large main graft an anastomosis is created to the aortic arch. The distal end of the main graft is then allowed to float freely within the descending aorta. In a later second stage to the procedure, TEVAR is used to repair the descending aorta with the elephant trunk graft used as a landing zone for the proximal end of the stent graft. A "frozen elephant trunk" refers to a single staged procedure where the endovascular stent graft is deployed during deep hypothermic circulatory arrest after debranching and repair of the aortic arch has been completed.

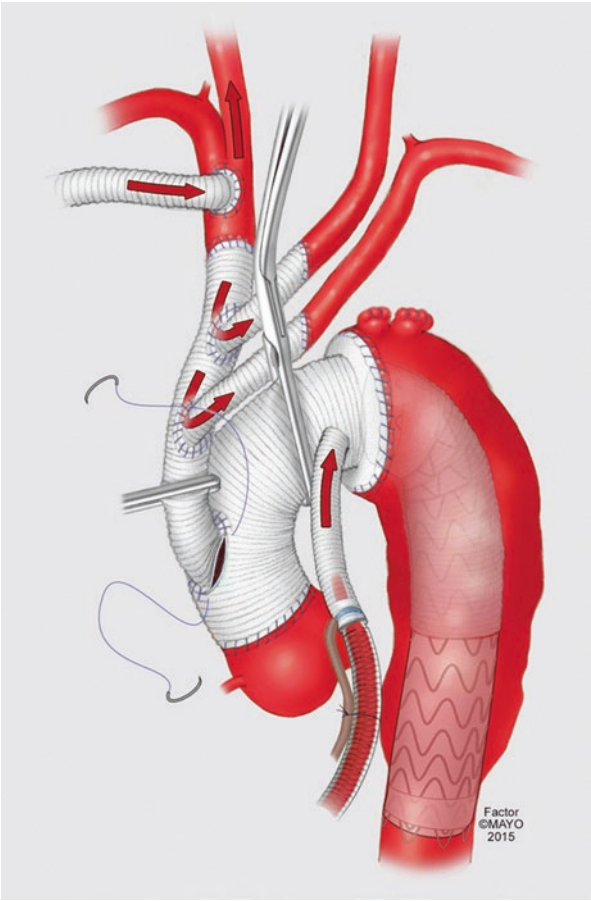
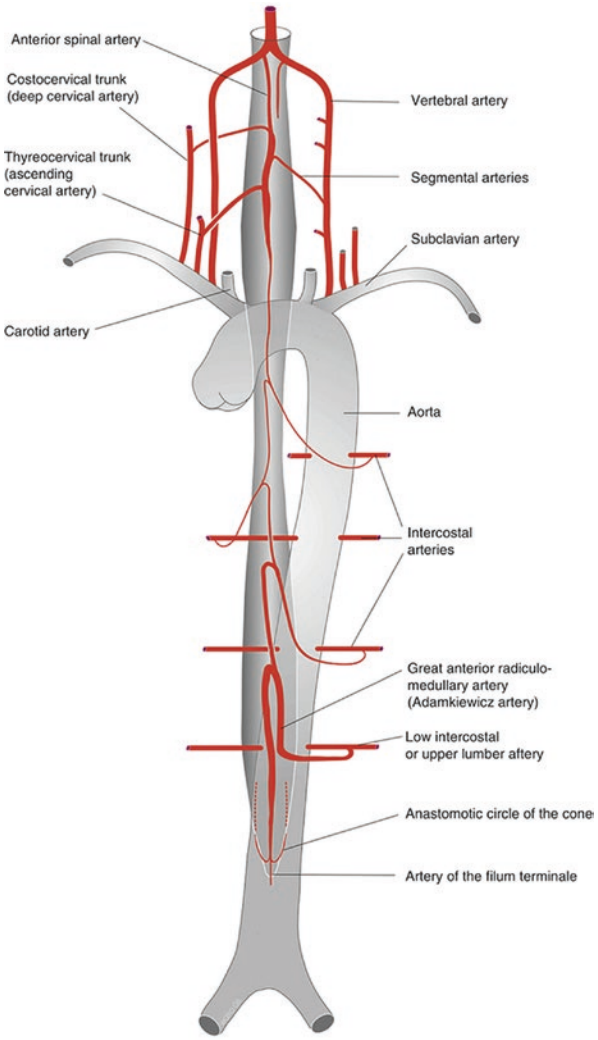


Fig 20.12 Preventza O, Coselli JS (2017) Techniques and Results of Hybrid Arch Replacement with Elephant Trunk. In: Oderich G. (eds) Endovascular Aortic Repair. Springer, Cham. https://link.springer.com/chapter/10.1007/978-3-319-15192-2_37

Appendix 2 – Spinal Cord Ischemia and Stroke

Postoperative paralysis is one of the most devastating complications of thoracoabdominal aortic aneurysm repair (TAAAR), whether an open or endovascular approach is used. While TEVAR is associated with reductions in intensive care unit (ICU) length of stay, organ dysfunction, postoperative pain, and overall cost, the incidence of spinal cord ischemia (SCI) has not been found to be lower than with an open surgical technique. Delayed paralysis with onset during the postoperative period is the most common presentation after TEVAR. It is believed to be caused by both intraoperative and postoperative spinal cord ischemia as well as reperfusion injury. Placement of the TEVAR graft results in both direct occlusion of the intercostal arteries supplying the thoracic aorta as well as embolic phenomena.



Spinal Cord Arterial Supply:

The anatomy of the blood supply to the spinal cord (Fig. 20.13) is critical to understanding strategies for spinal cord protection. Blood flow to the posterior one third of the spinal cord is more robust because it is supplied by two posterior spinal arteries that have strong anastomotic connections. In contrast, the anterior two thirds of the spinal cord is supplied by a single anterior spinal artery. The anterior spinal artery has only weak connections to the posterior spinal arteries. It is mainly dependent on segmental radicular arteries originating from the posterior branches of the intercostal and lumbar arteries for its blood supply. In adults only 6–8 dominant radicular arteries persist: two to three pairs in the cervical region, one to three pairs

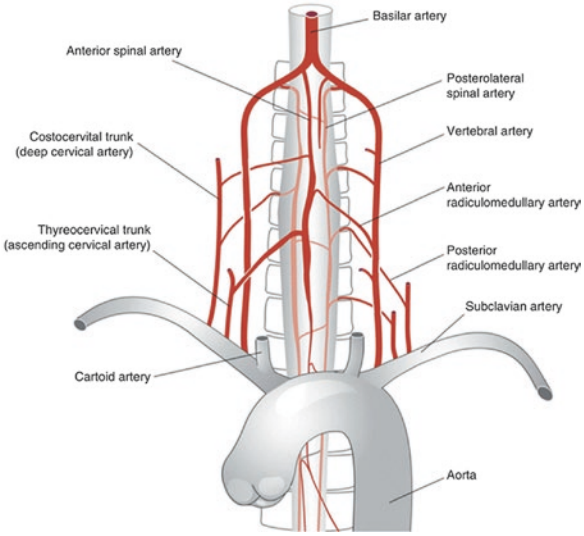


Fig. 20.13 Thron A.K. (2016) Arterial Blood Supply. In: Vascular Anatomy of the Spinal Cord. Springer, Cham. https://link.springer.com/chapter/10.1007/978-3-319-27440-9_2

in the thoracic region, and few if any in the lumbar portion of the spinal cord. Perfusion to the anterior spinal artery is most vulnerable in the lumbar region and ischemia here will predominantly affect the motor neurons of the anterior horn. The main arterial supply to the lower anterior spinal artery is the Artery of Adamkiewicz which originates anywhere from T9-L2 in 85% of patients.

Special Note about Left Subclavian Artery:
The subclavian arteries supply the vertebral arteries, which in turn feed the anterior spinal artery. Additionally, the subclavian arteries feed the arteries of the thyrocervical trunk which supply the cervical spinal cord. Occlusion of the left subclavian artery increases the risk of spinal cord ischemia and the risk of vertebrobasilar stroke or arm ischemia. Perfusion of the vertebral arteries with subclavian revascularization is the best approach to prevent ischemic events in the brainstem and the posterior cerebral circulation.

Reperfusion of the left subclavian artery is required if the patient has a dominant left vertebral artery.

Predictors of spinal cord ischemia for TEVAR:
- Patient Factors: renal failure, preexisting hypertension, COPD, age greater than 70 years old.
- Left subclavian artery exclusion without revascularization,
- Hypogastric artery exclusion,
- Coverage of greater than one vascular territory,

- Use of three or more stent grafts,
- Concomitant open abdominal aortic surgery or prior abdominal aneurysm repair,
- Prolonged procedure or urgent/emergent surgery,
- Excessive blood loss
- Perioperative hypotension with mean arterial pressure less than 70 mmHg.

Strategies to prevent spinal cord ischemia in endovascular repair:

- *Strict blood pressure control both intraoperatively and postoperatively.* Maintain mean arterial pressure (MAP) > 90 mmHg and spinal cord perfusion pressure (SCPP) > 80 mmHg.
- *Cerebrospinal fluid (CSF) drainage*. CSF pressure should be maintained <10 mmHg, draining no more than 15 mL/hour when the patient is neurologically intact. If postoperative neurological deficits occur, then drain to CSF pressure < 5 mmHg.
- *Maintain oxygen delivery*. Cardiac index >2.5 L/min/m^2, Hemoglobin >12 mg/dL.
- *Mild passive hypothermia.* Intra-procedure core temperature 32–35 °C.
- *Somatosensory Evoked Potentials (SSEPs) and Motor Evoked Potentials (MEPs).* Currently not widely used in TEVAR due to inability to differentiate severe vs. mild spinal cord ischemia.

Risk factors for stroke include:

- Patient Factors: prior transient ischemic attack, prior stroke (strong risk factor indicating presence of atheroma in aortic arch or branches), obesity, female sex, history of coronary artery disease.
- Involvement of the proximal descending aorta,
- High-grade atheroma of the aortic arch,
- Presence of an intra-aortic mural thrombus >3/4 the diameter of the normal aorta in a nondiseased portion.
- Greater intraoperative blood loss
- Longer surgery duration
- Use of a pull-through wire in patients with prior stroke

Strategies to Prevent Stroke:

- Careful preoperative planning of approach considering the burden of plaque in the aortic arch and identification of a dominant left vertebral artery.
- Controlled hypotension during stent positioning to avoid distal embolism. Controlled hypertension during the rest of the procedure to allow perfusion to the circle of Willis, particularly if the left subclavian is occluded.
- Temporary clamping or balloon occlusion of the left subclavian and common carotid arteries during passage of the stent-grafts through the aortic arch.
- Transcranial dopplers (TCDs) to monitor for emboli and Cerebra embolic protection devices (filters) have been used. TCDs detect cerebral microemboli as a high-intensity signal and are associated with cognitive impairment.

Suggested Reading

Bottiger B, Raja A. Thoracic aortic procedures – endovascular thoracic aortic repair. Clin Adv. 2019;18:2019.

Muehle A, Uzun I, Jalali Z, Khoynezhad A. Perioperative hemodynamic management and pharmacotherapeutics of patients undergoing thoracic endovascular aortic repair. Adv Vasc Med. 2014;2014:1–6, https://www.hindawi.com/journals/avm/2014/586084/.

Reidy CM. Anesthetic considerations in TEVAR and TAVI. Adv Anesth. 2012;30:47–59.

El Sayed HF, Collard CD, Awad H, Tili E, Tolpin DA, Ramadan ME. Spinal cord injury after thoracic endovascular aortic aneurysm repairLésion de la moelle épinière après réparation endovasculaire d'un anévrisme de l'aorte thoracique. Can J Anesth Can d'anesthésie. 2017;64:1218–35.

Anesthetic Management for Valvular Heart Disease

21

William Marion

The good physician treats the disease; the great physician treats the patient who has the disease

—William Osler

Key Points

- Types of valvular disease
- Severity grading
- Pathophysiologic considerations
- Fluid status
- Intraoperative hemodynamic management

Introduction

Each year there are over five million new diagnoses of valvular heart disease in America alone. Of those patients, at least 186,000 will require surgery to either repair or replace their diseased valve. Without this intervention, mortality rates may be as high as 80% in some cases. The two most common types of procedures involving valvular heart disease are aortic valve replacement and mitral valve surgery. This chapter reviews the anesthetic considerations for patients undergoing cardiac surgery for heart valve lesions with a focus on the aortic and mitral valves. We hope to give you a handle on the basics needed to safely care for patients undergoing valve repair or replacement surgery.

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A. S. Awad (ed.), *Cardiac Anesthesia*,
https://doi.org/10.1007/978-3-030-51755-7_21

Aortic Valve Disease

Aortic valve pathology that requires surgery can be broadly categorized as either stenosis or regurgitation. Stenosis is any narrowing of the valve orifice that restricts flow. Regurgitation is a leakage or reversal of blood flow from an incompetent valve during diastole. Stenotic lesions result most commonly from calcific aortic stenosis. Stenotic lesions can also result from a congenital bicuspid valve where the cusps fail to separate during development, from age related calcification or, rarely, from rheumatic disease. Regurgitant lesions represent a failure of adequate cusp apposition or damage to one or more of the cusps. This can be a normal result of aging, from endocarditis, rheumatic disease or trauma. In order to fully understand this, it's important to have a good working knowledge of the anatomy of the aortic valve apparatus which was discussed in Chap. 1.

Aortic Stenosis

Pathophysiology and Severity

A properly functioning aortic valve will have an area of 2.6 cm^2 or larger and a gradient of <5 mmHg during left ventricular systolic ejection. As the orifice area decreases, the gradient or pressure difference between the left ventricle and aorta increases. However, it is important to note that critical stenosis may have a near normal gradient and weak murmur in patients that have low flow through the stenotic lesion. As the orifice narrows and gradient increases during disease progression, anatomical and physiologic changes begin to occur prior to the development of symptoms.

First, increased intraercavitary pressure increases left ventricular wall tension which, in turn, increases myocardial oxygen demand. Next, concentric hypertrophy develops which decreases both compliance and left ventricular cavity volume leading to diastolic dysfunction. These patients tend to have the greatest degree of hypertrophy of any valvular disease. Finally, if left untreated, left ventricular dilation and failure occur. Symptoms generally tend to develop between the stages of hypertrophy and failure and are an indication of poor prognosis. They also depend on the size of valve area with normal being between 2 and 4 cm with mean gradian of 0 mmHg while severe or critical stenosis has a valve area of <1.0 cm^2 with mean gradian of >40 mmHg.

Anesthetic Management for Aortic Valve Replacement

Perioperative Monitoring

Successfully managing a patient through aortic valve surgery starts in the perioperative holding area. Please refer to Chaps. 8 "cardiac operating room setup" and 9 "Intraoperative Hemodynamic monitoring" for more detailed discussion. As

a special consideration for critical AS, it is important to note that resuscitation of the patient may be more difficult due to the stenotic valve; chest compressions may be less effective so, ensure adequate peripheral IV access so that medications and larger fluid volumes can be administered rapidly if necessary. ASA standard monitoring, arterial catheter, and central venous catheter are routinely used during the procedure with the addition of cerebral oximetry and bispectral index monitoring. A Swan Ganz catheter might also be needed depending on the patient's clinical condition.

Induction and Maintenance

Intravenous lidocaine 1 mg/kg is often an initial component of anesthetic induction. Midazolam can be used to induce anesthesia and has a favorable hemodynamic profile. Fentanyl and, less commonly, sufentanil are used to mitigate the sympathetic output associated with laryngoscopy. It is also important to take care to avoid opioid-induced chest wall rigidity with rapid injection of narcotic agents. In addition, opiates blunt the hemodynamic swings associated with skin incision that can tax an already stressed myocardium. Common hypnotics used for induction are propofol 1–2 mg/kg, etomidate 0.2–0.3 mg/kg, and ketamine 1–2 mg/kg. This may be where provider preference and type of lesion most commonly come into play. Propofol may be the choice of many providers due to individual comfort level given its frequent use as an induction agent across many types of procedures. However, of all the aforementioned agents it has the highest propensity for hypotension, making it a good choice for isolated regurgitant lesions but potentially deleterious for patients with stenotic lesions or elevated right-sided pressures. Etomidate has an excellent hemodynamic stability profile and thus may be the primary choice for patients with stenotic lesions or elevated right-sided pressures. Ketamine is an excellent choice for patients with regurgitant lesions who may have less-than-optimal ventricular function. Although it has some direct myocardial suppressant characteristics, ketamine directly stimulates the central nervous system to increase catecholamine release which supports ventricular function.

Neuromuscular blockade is also administered and requires several considerations. First, if a difficult airway is anticipated and the K+ is not elevated, succinylcholine 1.5 mg/kg should be administered and followed by a longer acting nondepolarizing neuromuscular blocker. If a difficult airway is not anticipated but the K^+ is elevated, rocuronium 1.2 mg/kg is a good first choice. If RSI is not necessary, rocuronium 0.45–0.6 mg/kg and vecuronium 0.1–0.2 mg/kg are both acceptable choices. In patients with severely impaired renal function, cis-atracurium 0.15–0.2 mg/kg can be considered.

All modern inhalational anesthetics with the exception of desflurane can be used for maintenance. Isoflurane has the dual benefit of minimal effect on the myocardium and significant suppression of cerebral metabolic oxygen requirements. Sevoflurane also decreases cerebral metabolic oxygen requirements and has a myocardial protective effect through volatile anesthetic-induced preconditioning, which is relevant here if aortic stenosis ventricles become ischemic.

Once stable induction and maintenance have been achieved, if not already in place, central venous access should be established. During this time, blood samples for baseline arterial blood gas analysis (ABG) and activated clotting time (ACT) should be drawn and run. This will help identify any hematological, metabolic, or electrolyte abnormalities that may cause instability and provide a reference range for terminating bypass.

Echocardiographic evaluation of cardiac function and anatomy should focus on the diseased valve. The degree of stenosis, the state of the left and right ventricles, the size of the aortic root annulus and the presence or absence of annular or aortic calcification should be ascertained. All TEE findings should be communicated to the surgeon. Antibiotic prophylaxis should be administered directly prior to incision except for vancomycin, the infusion of which is typically started 1 hour prior to incision. For sternotomy, ventilations need to be held with the pop-off valve fully open to allow the lungs to partially deflate. The next major step for the anesthesia provider is the administration of heparin prior to aortic cannulation. The dose range for heparin is 300–400 U/kg. Three minutes after administration, a blood sample should be drawn and both ABG and ACT should be assessed with the target ACT being in the high 400s. Once the surgeon is ready to cannulate the aorta, bed positioning and medication should be used to achieve a systolic blood pressure of no greater than 100 mmHg to help prevent excessive bleeding and aortic dissection. Once CPB has been initiated, the alarms should be muted, the ventilator turned off, fresh gas flows reduced as appropriate and the volatile agent turned off.

Hemodynamic Targets

Clinical Focus
The goals of induction and maintenance of patients with AS are to maintain adequate preload, keep the heart rate in the lower range of normal and maintain normal SVR.

Steps of Surgical Technique for Conventional Approach

The surgeon opens the chest

1. The chest is opened via median sternotomy
2. Heparin is administered and target ACT is reached.
3. Cannulation is accomplished with the standard technique using a two-stage venous canula (Fig. 21.1)
4. CPB is initiated and the patient is cooled to mild hypothermia at 32°
5. Vent is placed and aorta cross clamped
6. The heart is arrested using cardioplegia injection

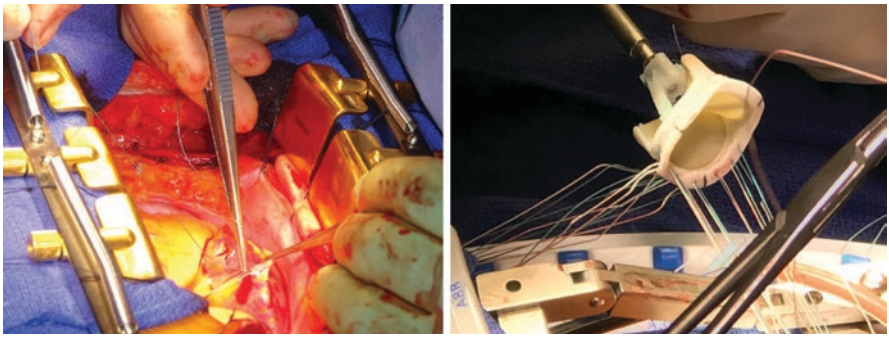

Fig. 21.1 Pictures of AVR surgery. Left: Prior to two-stage venous cannulation. Right: Suturing aortic valve sewing ring of the prosthetic valve to the aortic annulus

7. Aortotomy is made
8. The damaged valve is excised, and annular debridement is performed
9. The valve is sized using TEE measurements and mechanical sizers
10. Sutures are placed through the annulus of the valve to the sewing ring of the prosthetic valve (Fig. 21.1)
11. The prosthetic valve is implanted by tying all sutures
12. The aortotomy incision is sutured closed
13. Aorta is declamped
14. Deairing of the heart is accomplished with the aid of TEE
15. Pacing wires are placed
16. Calcium is administered and ventilation begins
17. Weaning from CPB

Weaning and Post-bypass Anesthetic Management

The focus of weaning from bypass is to ensure a stable transition from mechanical circulation and oxygenation to the return of physiologic heart and lung function. Please refer to Chap. 17 "Weaning from Cardiopulmonary Bypass and Management of Difficulties" for more detailed discussion.

TEE is invaluable in detecting dysfunction of the prosthetic valve and/or the LV. Proper functioning of all leaflets, especially in mechanical valves, should be ascertained. The presence or absence of central aortic regurgitation or paravalvular leak(s) should be documented. TEE is also the monitor of choice for de-airing the left side of the heart in all open cardiac surgical cases involving the left chambers. Once the hemodynamic state of the patient has been normalized, gradients across the prosthetic valve can be measured and documented.

Once the patient has been successfully weaned from bypass and is stable with acceptable ventricular function, heparin can be fully reversed. This is usually done through the administration of protamine sulfate at a ratio of 100:1 (i.e. 200 mg protamine to reverse 20,000 u heparin). However, types of protamine reactions and

management is discussed in Chap. 3 "Basic cardiovascular pharmacology." Additionally, it needs to be communicated loudly and clearly once 1/3–1/2 of the protamine has been administered so that the suction draining to the pump can be shut down to prevent clotting in the bypass machine. Three minutes after completion of protamine administration, ACT should be measured to ensure a return to baseline.

From this point on, anesthetic management is very similar to closure of many other major surgical procedures. Hemodynamic stability should be maintained using vasoactive medications while anemia, metabolic and electrolyte abnormalities should be corrected. When the wires are tightened to reapproximate the two halves of the sternum, some surgeons ask that the ventilations are held. A final echocardiographic evaluation of preload and ventricular function after chest closure is advisable.

Types of Aortic Valve Prostheses

Mechanical Prostheses

Mechanical valves are the longest lasting type of prosthetic valves, most of which are designed to last the patient's remaining lifetime. However, they are not without inherent risks or complications. Most notably, these risks include hemolysis, obstruction from thrombosis, embolic events, leaflet seizure causing stenosis or regurgitation, and prosthetic valve endocarditis. They also require lifelong anticoagulation which carries its own risks and limitations. There are three main types of mechanical valves:

1. Bileaflet
2. Ball-in-cage (no longer used due to poor functional profile and thromboembolism)
3. Tilting disc

Bioprosthetic Valves

These are made from either human, bovine, or porcine tissue, are trileaflet, and may be stented or non-stented. They more closely resemble native valves and are significantly less thrombogenic. Generally, these valves only require short-term anticoagulation or aspirin therapy. However, they are significantly less durable than mechanical valves and have a maximum lifespan of around 15 years in elderly patients. This can decrease significantly in younger patients due to immunologic responses, calcification, and wear and tear due to more active lifestyles.

There are four main types of bioprosthetic valve implants:

1. *Homograft* – This is a human cadaveric donor graft usually consisting of the valve and part of the aortic root. However, it is rarely performed due technical difficulties of the procedure and an exceedingly high failure rate of the graft.
2. *Xenograft* – This is a graft made from porcine aortic valve cusps that may be stented or stentless, with the stentless valves having a larger internal diameter and better hemodynamic profile.
3. *Bovine Pericardial graft* – As indicated, this valve has leaflets made from bovine pericardium that are secured behind a stent. This graft has less durability than a xenograft and limits to its internal diameter due to the stented structure.
4. *Ross Procedure* – A patient's diseased aortic valve is replaced with their own native pulmonic valve. The pulmonic valve is replaced with a donor valve. This procedure is uncommon due to technical difficulties.

Aortic Regurgitation

Pathophysiology and Severity

Aortic regurgitation is a disease of progressive LV volume overload determined by regurgitant area, the pressure gradient across the aortic valve and heart rate (more specifically, diastolic time). This occurs because of flow reversal across the aortic valve during diastole from either a failure of adequate cusp apposition or damage to one or more of the cusps. This can be a normal result of aging or it can occur from endocarditis, rheumatic disease or trauma. The resultant increase in wall tension during diastole leads to eccentric LV hypertrophy. With that in mind, it is also important to recognize that acute and chronic aortic regurgitation may have different ventricular effects and different presentations.

In the case of acute aortic insufficiency, most often from dissection or endocarditis, there is a rapid increase in volume overload leading to acute decompensation of LV and development of CHF. In the case of chronic aortic insufficiency and as the disease advances, progressive dilation of the LV with mitral annular dilation will lead to MR, pulmonary hypertension, and CHF.

Timing of surgical intervention depends on developing of symptoms and the size of the left ventricle as graded by TEE.

Anesthetic Management

As the regurgitant volume partially depends on regurgitant time and the gradient across the valve, anesthetic induction should be tailored with these factors in mind in order to ensure patient stability. Regurgitant time is a function of heart rate so a higher heart rate results in a decrease in regurgitant volume. However, tachycardia may result in ischemia which can cause or worsen LV dysfunction. Considering

this, it is important to find a target heart rate which decreases regurgitant time without risking ischemia. Heart rates in the high normal range should accomplish this goal, with the ideal rate being approximately 90 bpm.

With the target heart rate in mind, medications that do not cause a reflex bradycardia should be considered if support of the blood pressure is necessary. Ephedrine or very low dose epinephrine can help maintain the heart rate while providing blood pressure support. Ketamine may be an appropriate induction agent. However, it is important to keep in mind that elevating the blood pressure may increase the gradient and worsen the regurgitation. As a result, a hypertensive patient without significant coronary artery disease may benefit from induction with propofol to lower the blood pressure and improve the gradient. The resulting decrease in gradient may slightly improve LV offloading and function.

Surgical Steps

Surgical steps are as described for aortic valve replacement in AS.

Special Considerations

Significant Aortic Insufficiency

A significantly incompetent aortic valve will allow cardioplegia to enter and be diluted in the LV. Therefore, another mode of cardioplegia delivery is necessary. This can be achieved through either of two techniques. First, the coronary sinus can be cannulated in order to provide retrograde delivery of cardioplegia. Second, the coronary ostia may be individually cannulated and antegrade cardioplegia can be administered using handheld cardioplegia cannulas.

Mixed Lesions

In patients with mixed lesions, the anesthetic should be tailored with the more severe lesion in mind. Mixed AS and AR is poorly tolerated, and these patients are often ischemic with angina.

Redo Surgery

Redo aortic valve surgery (Fig. 21.2) tends to be well tolerated but comes with its own considerations. These patients are more likely to have large amounts of blood loss due collateralized vessels through adhesions and healed tissue. The need to carefully dissect that tissue results in a longer surgical time.

Patient-Prosthesis Mismatch

This occurs most commonly in patients with a small aortic root or annulus where a prosthetic valve with too small of an opening has been implanted compared to the patient's ideal native valve. This can lead to LV overload and failure and may require

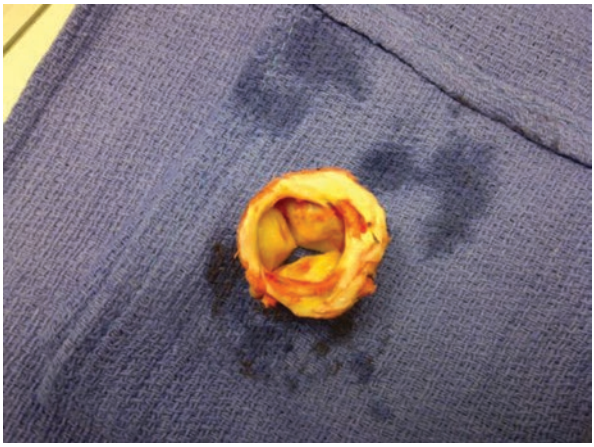

Fig. 21.2 Picture of Redo AVR surgery for prosthetic valve stenosis

immediate replacement post initial bypass. Hemodynamically, these patients behave similar to those with severe AS.

Mitral Valve Disease

As with aortic disease, mitral valve disease is broadly categorized into stenotic or regurgitant disease. Stenotic lesions can be caused by rheumatic fever or calcification as part of the aging process. As the incidence of rheumatic disease in the Western world has declined, so has the incidence of mitral stenosis (MS). More rarely, MS can be caused by autoimmune diseases including systemic lupus erythematosus or radiation exposure.

Mitral regurgitation (MR) can be subclassified into primary (degenerative), secondary (functional), and mixed. Pathophysiological changes that lead to MR are leaflet prolapse (congenital or acquired), endocarditis, rheumatic fever, damaged chordae, myocardial infarction, papillary muscle dysfunction from ischemia or an infarct, trauma or cardiomyopathy. A solid understanding of the mitral apparatus is necessary to understand these disease processes which is discussed in Chap. 1.

Mitral Stenosis

Pathophysiology

In the average adult, a normally functioning mitral valve has a maximal opening area of 4–6 cm^2, with progressive or mild stenosis having a valve area > 1.5 cm^2 and a gradient <5 mmHg. Over time MS lesion leads to an increase in the left atrial pressure. Since, the pulmonary veins are valveless, there is an early rise in pulmonary artery (PA) pressure. Over time this can lead to RV overload and failure. Potentially

complicating this is the remodeling of the PA in response to the increased pressures. This leads to hypertrophy that may cause persistent pulmonary hypertension and RV dysfunction even after the stenosis has been corrected.

As LV filling is restricted, preload depends on the length of the diastolic filling time, making these patients particularly susceptible to the negative hemodynamic effects of tachycardia. Additionally, the increased pressure in the left atrium will eventually cause dilation which can frequently lead to atrial fibrillation. Not only does this mean a loss in the volume contributed by the "atrial kick," but the rapid ventricular response severely impairs passive diastolic filling. As with other stenotic disease, the prognosis and severity of symptoms are closely related to the severity of stenosis.

Anesthetic Management for Mitral Valve Stenosis Surgery

Pre-induction Management

Successful navigation of mitral valve surgery begins by optimizing the patient status prior to induction. Pre-induction management is similar to aortic stenosis pre-induction management discussed earlier. Specific attention for MS patient's volume status is needed since hypovolemia will drop preload and compromise adequate forward flow. On the other hand, fluid overload in a patient with coexisting CHF can lead to pulmonary edema.

Induction and Maintenance

Choice of induction agents is similar to those used for aortic patients but with a few important differences. Premedication should be undertaken cautiously as lowering preload can have deleterious hemodynamic effects. This may be due to either loss of preload or the pulmonary hypertension and possible RV failure that can accompany the hypercapnia and hypoxia resulting from decreased respiratory drive. Because LV filling is significantly aided by longer diastolic times, medications that precipitate tachycardia such as ketamine and desflurane are better avoided. However, it is important to realize that enhanced stroke volume alone has limitations in maintaining cardiac output at very low rates, so significant bradycardia should also be avoided.

Another consideration during induction is the functional status of the patient's ventricles. Patients with advanced mitral stenosis and pulmonary hypertension resulting in RV dysfunction, or those with preexisting LV dysfunction, may require inotropic support during induction. The choice of agent will depend on the patient's blood pressure. Lowering afterload does little to aid in forward flow in cases of stenosis but there may be significant increases in systemic vascular resistance (SVR) due to the decrease in cardiac output. With that in mind, it is important to maintain a blood pressure in the normal range. If a patient is hypotensive or in the low-normal

range, epinephrine may be the more appropriate choice. However, it is important to monitor the heart rate as epinephrine's chronotropic effects may have a negative impact on cardiac output.

Anesthetic maintenance will continue from this point up to bypass in an almost identical manner to that of the anesthetic for aortic valve surgery.

Hemodynamic Targets

Clinical Focus
The hemodynamic goals of anesthesia maintenance in patients with mitral stenosis should include maintaining the heart rate in the low normal to slightly bradycardic range, while keeping the SVR in the normal range.

Steps of Surgical Technique for MV Replacement by Conventional Approach

1. The surgeon opens the chest via median sternotomy
2. Heparin is administered and target ACT is reached
3. Cannulation is accomplished in standard aorto-bicaval bypass with caval snares to ensure adequate venous drainage (Fig. 21.3)
4. CPB is initiated and the patient is cooled to mild hypothermia at 32°
5. Vent is placed and aorta is cross clamped
6. The heart is arrested using cardioplegia injection
7. Atriotomy is made in left atrium or the right atrium if a transeptal approach is employed
8. The damaged valve is excised, and annular debridement is performed
9. The valve is sized
10. Sutures are placed through the annulus of the valve and the sewing ring of the prosthetic valve
11. The prosthetic valve is implanted by tightening and tying of all the sutures
12. The atriotomy incision is sutured closed
13. The aortic clamp is removed
14. Deairing of the heart is accomplished with the aid of TEE
15. Pacing wires are placed
16. Calcium is administered and ventilation begins
17. The patient is weaned from CPB

Weaning and Post-bypass Anesthetic Management

Weaning from bypass and management afterwards does not differ greatly between aortic and mitral valve surgery. However, it is important to remember

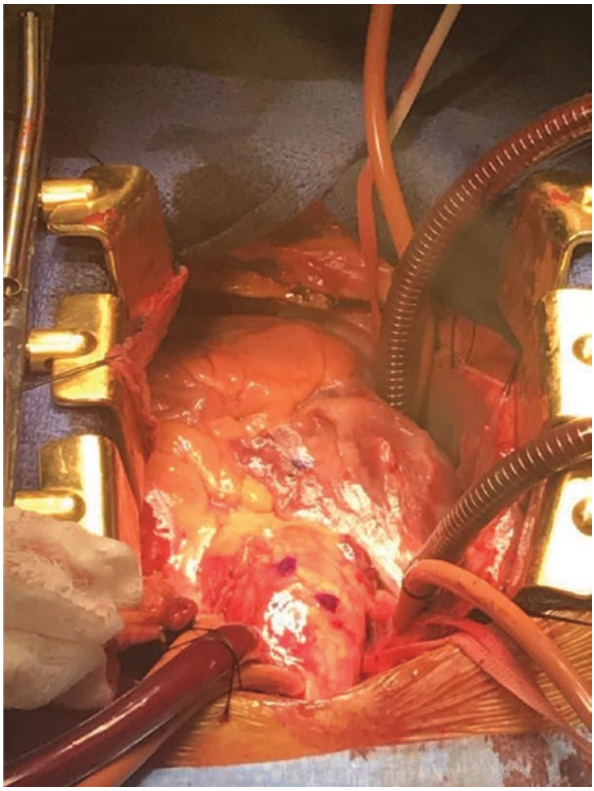

Fig. 21.3 Picture of cannulation that is accomplished by aorto-bicaval bypass with caval snares

that mitral patients are at a higher risk of preoperative pulmonary hypertension with RV dysfunction. This translates into a greater risk of post bypass RV dysfunction requiring slightly different management. Inotropic support with epinephrine or milrinone 0.375–0.75 mcg/kg/min infusion can help revive the RV. However, in patients with persistent pulmonary hypertension after mitral valve surgery, they may benefit from inhaled nitric oxide at 20 ppm or epoprostenol starting at 2 ng/kg/min.

Following MV repair or replacement, TEE plays an important role in weaning from bypass. The success of the repair or the function of the prosthetic valve should be evaluated before complete separation from bypass. Ventricular function should also be assessed and the heart de-aired. Particular attention should be paid to the territories supplied by the right coronary artery (RCA) and the circumflex artery, as flow in these areas may be compromised by air (RCA) or inadvertent iatrogenic damage while working close to the atrioventricular groove and the circumflex artery (e.g. suture).

Clinical Pearls

Acute RV failure may develop insidiously after termination of bypass. The patient may acutely decompensate with falling systemic pressures and rising pulmonary pressures. This is a life-threatening event that requires rapid intervention. There patients may benefit from a "pharmacological balloon pump":

1. Epinephrine bolus 4–8 mcg
2. Nitroglycerine bolus 50–100 mcg

Alternatively, the provider can position the patient in reverse Trendelenburg and support their blood pressure. In both interventions this can provide inotropic support to the RV while offloading it, decreasing PA pressures and restoring the balance of myocardial O2 delivery and consumption

Mitral Regurgitation

Pathophysiology

MR is a common valvular disorder that frequently requires no intervention. However, in cases of acute regurgitation or progressive chronic regurgitation, repair or replacement is necessary to preserve long-term survival.

Degenerative mitral valve disease is by far the most common group of lesions that leads to MR. Fibroelastic deficiency (FED) and Barlow's disease are the two commonest entities of that group leading to degenerative MV disease. Barlow's is due to myxoid degeneration of the mitral valve tissue and presents with large, thickened, and redundant leaflets, chordal elongation or rupture, and sometimes also dilated MV annulus. It typically affects multiple segments of the anterior and posterior MV leaflets (AMVL and PMVL). In contrast, FED often affects a single segment of the MV and is due to insufficient or inadequate connective tissue. This leads to chordal thinning and rupture with associated segmental prolapse. Patients with FED may be asymptomatic until a chordal rupture and significant MR occurs.

Various classification systems for MR exist. Carpentier's functional classification is particularly useful in combining the aspects of abnormal leaflet motion with the underlying pathology, thereby aiding in the understanding of the etiology of MR. In Carpentier's functional classification, Type I MR represents failure of sufficient coaptation with otherwise normal leaflet motion. Type II represents degenerative MR due to increased leaflet motion, with subtype IIA being FED and IIB, Barlow's disease; and Type III is MR due to restricted leaflet motion. In most cases, degenerative MV disease can be successfully repaired, obviating the need for MV replacement.

Regardless of type or etiology, mitral regurgitation is a disease of LV volume overload, as the regurgitant volume is returned to the LV during diastole. In acute MR, loss of LA compliance leads to a rapid increase in pulmonary pressures that cause pulmonary edema and potentially biventricular failure. The rapid increase in preload also increases the end diastolic pressure, which leads to ischemia and may dilate the LV or worsen systolic function. The tachycardia that results as an attempt to maintain cardiac output is usually ineffective, as it prevents complete emptying of the LV and worsens any existing ischemia. It is for those reasons that acute MR is poorly tolerated and requires urgent surgical intervention. Acute MR is typically caused by chordal or papillary muscle rupture or dysfunction from an infarction or by endocarditis with perforation.

In contrast, chronic MR is associated with left atrial pressure that progresses to left atrial enlargement and development of atrial fibrillation. Also, the LV volume increases, and this volume overload leads to LV cavity enlargement and eccentric hypertrophy. Over time, it will also cause annular dilation or worsening regurgitant flow. With this dilation of the LV also comes an inherent increase in wall tension with an accompanying imbalance of O_2 demand and delivery leading to chronic subendocardial ischemia. Ultimately, the LV function worsens to the point of decompensation. Once in a decompensated state, pulmonary pressures rise leading to RV dysfunction and the development of worsening symptoms.

Anesthetic Management for Mitral Valve Regurgitation Surgery

The monitoring modalities used for patients with mitral regurgitation are identical to those used in mitral stenosis. Patients with acute or chronic decompensated mitral regurgitation tend to have elevated pulmonary pressures with varying degrees of ventricular dysfunction. Additionally, the severity of regurgitation depends at least in part on the LA/LV and LV/aortic gradients. Keeping these facts in mind is necessary for ensuring a stable induction and maintenance of anesthesia in this patient population.

Clinical Pearls

Avoiding hypoxia and hypercapnia are primary concerns. A patient with pulmonary hypertension may not be able to compensate for the resulting pulmonary vasoconstriction and is susceptible to fulminant RV failure.

In contrast to mitral stenosis, hemodynamic goals should include maintaining a high-normal heart rate of around 90 bpm and a low or low-normal SVR. This will decrease the regurgitant time without markedly increasing O_2 demand and will help with forward flow as the decreased afterload has a positive impact on the gradients influencing the degree of regurgitation. If ventricular support is required, milrinone

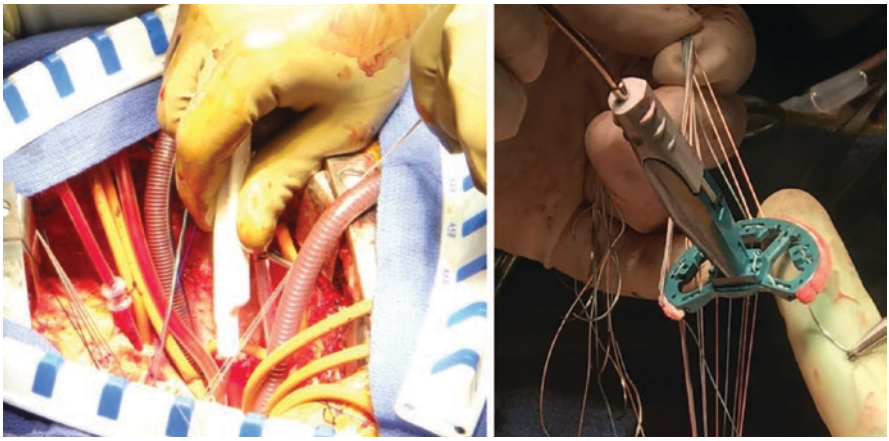

Fig. 21.4 Pictures of mitral valve repair surgery. Left: Sizing the ring. Right: Mitral ring sewing

is often the better choice given its ability to relax vascular smooth muscle. Additionally, adequate depth of anesthesia helps avoid intraoperative pulmonary hypertension. If PA pressures continue to be elevated despite adequate precautions, inhaled nitric oxide or epoprostenol are excellent options.

Steps of Surgical Technique

Surgical management of MR is either by MV repair or replacement. Exposure for MV repair or replacement is largely identical for mitral stenosis and regurgitation as described above (Fig. 21.4). Types and technique of MV repairs exceed the scope of this chapter and do not influence the anesthetic management in and of themselves. However, an echocardiographer is required to verify a successful MV repair on TEE or identify repair problems such as residual MR that necessitate further intervention.

Special Conditions for Mitral Valve Surgery

Mixed Lesions

Rheumatic disease most commonly involves MS and MR together, the more severe lesion determines hemodynamic goals; heart rate, SVR, and contractility should be optimized, agents or situations that lead to pulmonary vasoconstriction should be avoided.

Dehiscence

Patients with recurrent endocarditis are at risk for dehiscence of the prosthetic valve or annuloplasty ring due to poor quality of the anchoring tissue. This requires urgent reoperation to minimize mortality.

Redo Surgery

Reoperation has similar mortality to initial procedures but has a higher rate of prolonged intubation and transfusion. It carries an increased risk of intraoperative bleeding and prolonged surgical time.

Tricuspid Disease

Isolated tricuspid disease requiring surgical intervention is uncommon in the general population but frequently occcurs with endocarditis secondary to intravenous drug use. The pathological causes and etiology include the following:

Tricuspid Stenosis (TS)

1. Rheumatic disease which always presents in conjunction with another valvular pathology. Most commonly, it is seen with tricuspid regurgitation (TR) but may also coexist with rheumatic mitral disease.
2. Carcinoid syndrome
3. Congenital disease
4. Systemic Lupus
5. Right atrial masses

Tricuspid Regurgitation (TR)

1. Functional lesion stemming from pulmonary hypertension, RV failure or left-sided lesions that eventually impact RV afterload.
2. Infective as seen in IVDA-related endocarditis
3. Carcinoid syndrome
4. Ebstein anomaly
5. Prolapse related to connective tissue disorders, or trauma

Pathophysiology

As the largest heart valve, a normal adult tricuspid valve is tri-leaflet with an area of 7–9 cm^2 and a gradient of 1 mmHg or less.

TS

Generally, patients with TS are asymptomatic until significant valvular degeneration has occurred, and the area has decreased to approximately 1.5 cm^2. There will be an increase in RA pressure accompanied by RA dilation along with the expected loss of forward flow. Severity is assessed by either cardiac catheterization or TEE

TR

As with other regurgitant lesions, isolated TR is a state of RV volume overload that is often tolerated fairly well. The surgical approach to the exposure of the heart and cannulation for bypass are similar to TS. The decision to repair or replace the native TV depends on the severity and etiology of the regurgitation and whether or not there is concomitant TS. Repair is most frequently done through ring annuloplasty but may also include debridement or repair of the subvalvular apparatus. It should be noted that tricuspid repair carries a significantly lower mortality than tricuspid replacement.

Surgical Technique

Bicaval cannulation with caval snares is required for venous drainage as the right atrium should remain empty in order to provide an unobscured view of the valve. Once adequate exposure is gained, the decision to repair or replace the valve is made. If calcification or vegetation is not excessive, debridement and commissurotomy are effective interventions. If calcification is extensive or the valve is damaged irreparably from endocarditis, replacement is indicated. In the case of repetitive endocarditis with severe damage due to IV drug use, the valve may be excised and not replaced until a prescribed course of antibiotic therapy and abstinence from drug use is completed. From here, closure, weaning from bypass and deairing proceeds as with any other valve case. However, these patients are very likely to need RV inotropic support with either epinephrine or milrinone. If RV function is adequate but the SVR is low, norepinephrine or vasopressin are appropriate agents.

Anesthetic Management for Tricuspid Valve

In TS, ensuring adequate preload is the initial step in crafting a smooth induction and stable anesthetic. Adequate SVR should be maintained throughout, such that there is adequate venous return to maximize ventricular filling. It is also important to maintain normal sinus rhythm or a normal rate if the patient is in an uncorrectable rhythm. As RV filling is impaired, contractility may also be decreased and supporting RV function though inotropes, if necessary, will help maintain cardiac output. Additionally, it is imperative to monitor CVP and periodically inspect the patient's head for edema or venous stasis to ensure the SVC cannula is draining appropriately.

In TR, optimizing preload helps aid in forward flow as loss of venous return can significantly impair RV stroke volume. Additionally, maintaining a high-normal heart rate will help decrease regurgitant time and support forward flow without significantly increasing O_2 demand. In the case of a failing RV which frequently occurs in patients with severe TR, inotropic support with milrinone or epinephrine may be appropriate with milrinone being the better option in the face of pulmonary

hypertension. However, it should be noted that decreasing SVR will have little effect on TR or the failing RV. During CPB it is important to monitor the patient's head for signs of venous congestion to ensure that the caval cannulas are providing adequate drainage. Also, there may be new stenosis following replacement as the available valves tend to be smaller than native tricuspid valves.

Hemodynamic Targets

Clinical Focus

- Anesthetic goals prioritize maintaining preload, afterload, and contractility.
- Maintain fast-normal heart rate
- Inotropic support if necessary
- Decreasing PVR in case of TR helps, decreasing SVR does little
- Avoid hypoxia and hypercapnia

Weaning and Post-bypass Management

Weaning from bypass in this setting does not carry special risks or require special interventions in case of TS. However, in TR when weaning from bypass there is no longer a means of venting excess pressure in the RV if the pulmonary vascular resistance (PVR) is high. This makes RV failure a distinct possibility and requires vigilance and preparation. Interventions may include inotropic support of the RV or even adjuvant treatment with inhaled nitric oxide or epoprostenol. As the RV acclimates and the PVR decreases, these treatments can be weaned. The repaired or prosthetic valve and RV function should be evaluated by TEE prior to separation from bypass. If replaced, the new valve should be assessed for leaflet function, gradient, seating and the presence of perivalvular leak. In the case of left-sided interventions, TEE guidance during deairing is also necessary.

Special Considerations for Tricuspid Valve Surgery

Redo Surgery

This carries the additional time and bleeding constraints characteristic of other redo procedures. The surgeon may choose to excise the valve without replacing it.

Mixed Lesions

Tricuspid lesions almost always occur in the presence of another lesion whether TS and TR or a tricuspid lesion with disease of another valve. The focus of anesthetic management is determined by the more hemodynamically significant lesion.

Diseases of the Pulmonic Valve

Isolated pulmonic valve disease requiring surgical intervention is uncommon. The pathological causes and etiology include the following:

Pulmonic Stenosis (PS)

1. Congenital disease
2. Rheumatic disease
3. Carcinoid syndrome

Pulmonic Regurgitation (PR)

1. Primary causes
   (a) Rheumatic disease
   (b) Endocarditis
   (c) Carcinoid syndrome
   (d) Congenital disease
2. Secondary causes
   (a) Pulmonary hypertension
   (b) Dilated cardiomyopathy
   (c) Previous repair of a stenotic lesion
   (d) Functional effect of pregnancy

Pathophysiology

PS is a disease of pressure overload for which the RV has a limited capacity to compensate. As a result, RV systolic function will eventually begin to fail leading to ventricular dilation.

PR, in contrast to stenosis, is a disease of RV volume overload for which the RV can compensate over a longer period of time. However, once the compensatory mechanisms are exhausted, the RV will begin to fail and dilate. This can lead to functional TR.

Anesthetic Management for Pulmonic Disease

The monitoring modalities used are identical to any surgery for valvular heart disease.

With induction of PS patients, preoxygenation is key and drugs that cause tachycardia, hypotension, or ventricular suppression should be avoided. Additionally, optimizing preload and avoiding decreases in SVR may help by ensuring adequate

RV filling. However, lowering PVR may have little to no effect as the obstructing pathology is an outflow obstruction of the RV rather than an issue of pulmonary circulation. Finally, inotropic support with epinephrine during induction may be warranted if the RV is significantly compromised.

Since PR is a disease of RV overload, anything that increases PVR and thus the regurgitant volume should be avoided. Additionally, maintaining faster heart rates decreases regurgitant time but tachycardia can exacerbate ischemia and RV dysfunction. Oftentimes the underlying pathology involves some degree of pulmonary hypertension that creates or exacerbates the regurgitation. Considering the above factors, the choice of anesthetic agents should target forward flow through the pulmonary circulation. It may also be necessary to support the RV through induction. Epinephrine provides good inotropic support but milrinone may be better suited considering its effects on PVR.

Surgical Intervention

Approach is via median sternotomy. A longitudinal incision is made in the pulmonary trunk to expose the pulmonary valve. Commissurotomy, an incision restoring commisural separation, may be performed to repair the valve if the valve is calcific and the leaflets are fused. Otherwise, replacement of the valve must be performed.

Weaning and Post-bypass Management

Weaning and post-bypass management for PS patient is a similar process to other right-sided valve surgeries but may be slightly more difficult depending on the degree of RV dysfunction. Ventricular support might also be needed. If there is concomitant pulmonary hypertension, the RV may require lowering pulmonary pressures in addition to ventricular support.

Weaning and post-bypass management for the PR patient is like the PS patient but with special attention paid to pulmonary pressures, volume status and RV function. There may be a need for inotropic support or PVR lowering measures.

Clinical Pearls

Avoid

- Hypoxia
- Hypercapnia
- Hypotension
- Bradycardia or tachycardia

Maintain

- Normal HR
- Normal SVR

Suggested Reading

Carpentier A. Cardiac valve surgery–the "French correction". J Thoracic Cardiovasc Surg. 1983;86:323–37.

Castillo JG, Anyanwu AC, Fuster V, Adams DH. A near 100% repair rate for mitral valve prolapse is achievable in a reference center: implications for future guidelines. J Thoracic Cardiovasc Surg. 2012;144:308–12. https://doi.org/10.1016/j.jtcvs.2011.12.054.

Anesthetic Management for Thoracic Aortic Procedures

22

Antonio Chiricolo, Kim Linden, and Frank Bowen

To acquire knowledge, one must study; but to acquire wisdom, one must observe.

—Marilyn Vos Savant

Aortic Anatomy

- The aortic root is the segment between the left ventricle and the ascending aorta. It is comprised of the sub-commissural triangles, the aortic annulus, the aortic cusps, the three aortic sinuses or sinuses of Valsalva, and the sinotubular junction (Fig. 1.7). A variation in the diameter of the sinotubular junction or dilation and prolapse of the cusps can produce significant aortic insufficiency. This can occur in cases of acute aortic dissections, annuloaortic ectasia, or aneurysm of the sinus of Valsalva, all of which will require surgical interventions.
- The left and right sinuses give rise to the left main and right coronary arteries, respectively. Normally, during diastole, the free margin and part of the three cusps co-apt, creating a seal, thus preventing aortic regurgitation and ensuring adequate diastolic blood pressure (Fig. 1.8).

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- The aortic arch is defined as the segment of aorta between a line at a right angle proximal to the innominate artery (brachiocephalic artery) origin and extending to a line drawn at a right angle distal to the origin of the left subclavian artery.
- The ascending aorta includes the root and arch, while the descending aorta is that part which is distal to the left subclavian artery.
- The descending thoracic aorta begins at the T4 vertebral body and descends on the left side of the fifth through twelfth thoracic vertebral bodies (T5–T12), and enters the abdomen through the aortic hiatus at T12 (Fig. 22.1).

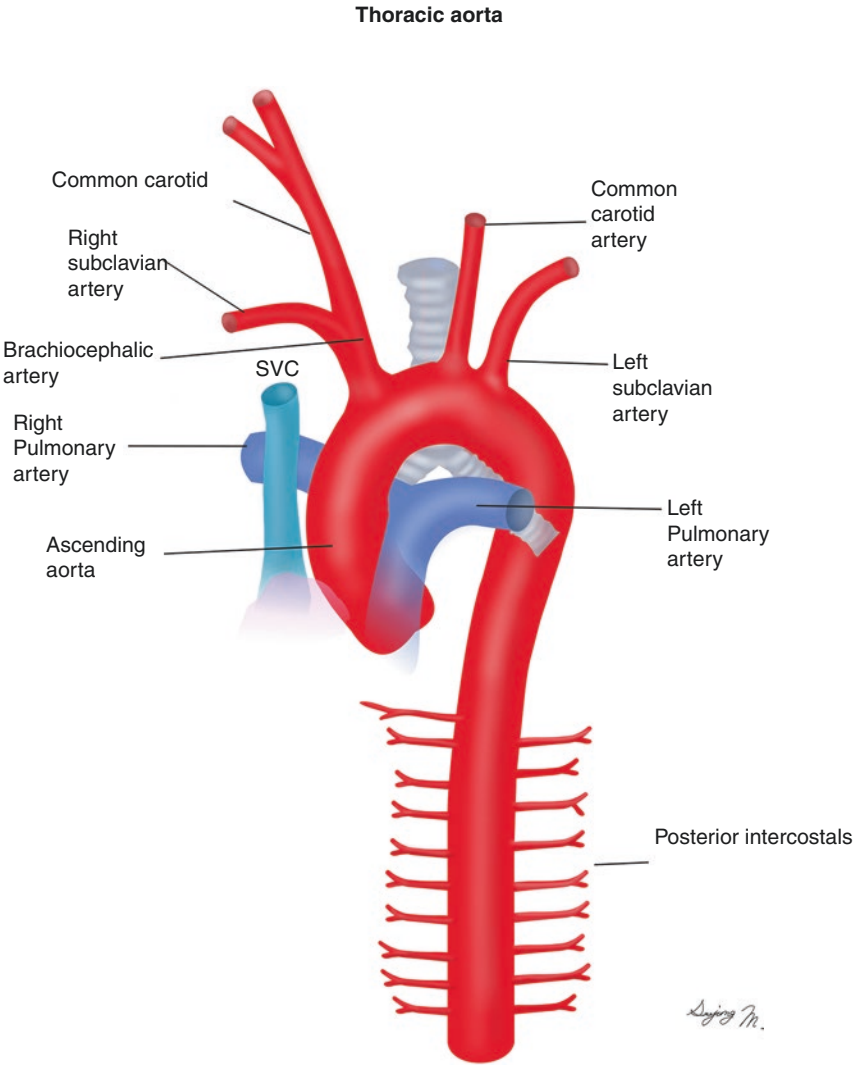

Fig. 22.1 Illustration of anatomy of the thoracoabdominal aorta

- The thoracic aorta has a number of branches. The first branches of the aorta are the coronary arteries, which originate at the sinuses of Valsalva. Three branches generally arise from the arch (listed here, from proximal to distal): the brachiocephalic trunk or innominate, left common carotid, and left subclavian arteries.
- A bovine arch is the most common variant of the aortic arch, and is defined by the brachiocephalic and the left common carotid artery sharing a common trunk.

With many possible aortic arch anatomic variations, a good understanding of the patient's anatomy is imperative for the safe management of aortic pathology. Aortic arch surgery alone is uncommon, but the aortic arch is involved in 10% of aortic aneurysm and 70% of type A dissections. Thoracoabdominal aneurysm and aortic dissections all may require surgical intervention.

Preoperative Assessment

- The preoperative assessment for the open thoracic aortic surgical candidates has tended to address the typical issues related to a standard general anesthetic for a cardiac patient, including a thorough airway assessment disscused in Chap. 5.
- Open aortic repair should be classified as a high-risk procedure. Prior to elective open repair, patients should undergo significant preoperative risk assessment and optimization.
- Patients are generally older and often have cardiac disease, and therefore should undergo cardiac evaluation with EKG and echocardiogram. Those patients exhibiting symptoms of myocardial ischemia or with impaired ventricular function (EF < 30%) should have cardiac catherization or stress test to determine the need for coronary revascularization prior to repair of the aortic pathology.
- Pulmonary function should be assessed as entry to the thoracic cavity and single lung ventilation is required. Generally, a patient with an FEV1 > 1.0 L and a PCO2 < 45 mmHg will tolerate the embarrassment of pulmonary function associated with the procedure.
- Carotid artery disease should be assessed if the patient is of advanced age, has a history of cerebrovascular accident/transient ischemic attack, or carotid bruits.
- Aortic pathology is often a result of a systemic disease and the evaluation of all organ systems should be performed.
- Patients requiring distal thoracic aortic repair should receive peripheral vascular studies. This is commonly done with contrast tomographic imaging. Also, patients should be evaluated for the risk of postoperative paraplegia, especially with large descending thoracoabdominal aneurysms.
- CSF drainage is recommended for both open and endovascular repairs when there is a high risk of developing postoperative paraplegia. Risk factors include older patients, comorbid renal dysfunction, emergency surgery, aortic rupture, acute dissection, extensive repair and long repair time, prior aortic surgery, and exclusion of the hypogastric artery.
- Patients should be counseled about the need for blood transfusions, postoperative mechanical ventilation, and invasive monitoring, including an evaluation for any contraindication to transesophageal echocardiography (TEE). Finally, all questions should be answered, and consent should be obtained when possible.

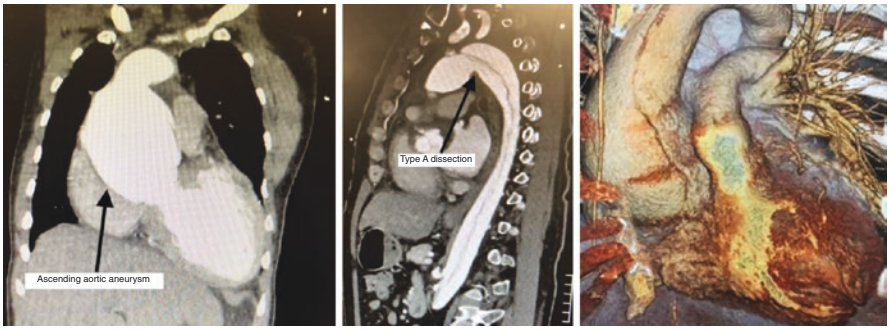

Fig. 22.2 Images of CT showing different pathology of the aorta

Aortic Imaging

Aortic imaging is imperative to define the specific anatomical pathology and plan the surgical repair. The most common diagnostic tests are computed tomography and magnetic resonance angiography. The use of intravenous contrast helps differentiate between intraluminal and extraluminal structures, which facilitates the detection and defines the extent of aortic dissection (Fig. 22.2).

Transesophageal echocardiography (TEE) is also utilized preoperatively to delineate the aortic pathology [QR code]. Furthermore, intraoperative TEE is necessary to help guide hemodynamic management of patients who undergo aortic surgery, and to differentiate between the true and false lumen of an aortic dissection. TEE also provides for real-time evaluation of myocardial regional wall motion, cardiac function, and pericardial effusion. Importantly TEE also provides a mechanism to evaluate the aortic root and valve intraoperatively to guide the surgical procedure and facilitate intraoperative management. The following Quick Response Code (QR code) will automatically open a YouTube video of TEE, as applied to surgical aortic pathology, and surgical photos of a type A dissection, when scanned from any mobile device.

Aortic Dissection

Definition

Aortic dissection is the separation of the aortic media by pulsatile blood along the internal and external elastic laminae creating a true and a false lumen.

Epidemiology

It is one of the most common pathologies involving the aorta. It is rarely associated with aneurysm. Although seen in all age groups, the majority of them present between 50 and–69 years of age and are more common in males. Two-thirds involve the ascending aorta (type A) and a third involve the descending aorta (type B). Risk factors include hypertension, connective tissue disorders, bicuspid aortic valve, aortic coarctation, atherosclerosis, cocaine use, iatrogenic injury, and pregnancy.

Pathophysiology

The process begins with an intimal tear (Fig. 22.3). Patients often present with severe and acute sharp or tearing chest or back pain. Occasionally patients can have symptoms of inadequate perfusion; including stroke, pulse deficit, visceral ischemia, renal ischemia, or paraplegia.

Classification

Dissections are classified either by the Stanford classification or the DeBakey classification. The Stanford classification has two groups. Stanford A classification includes the ascending aorta with or without other regions. Stanford B classification

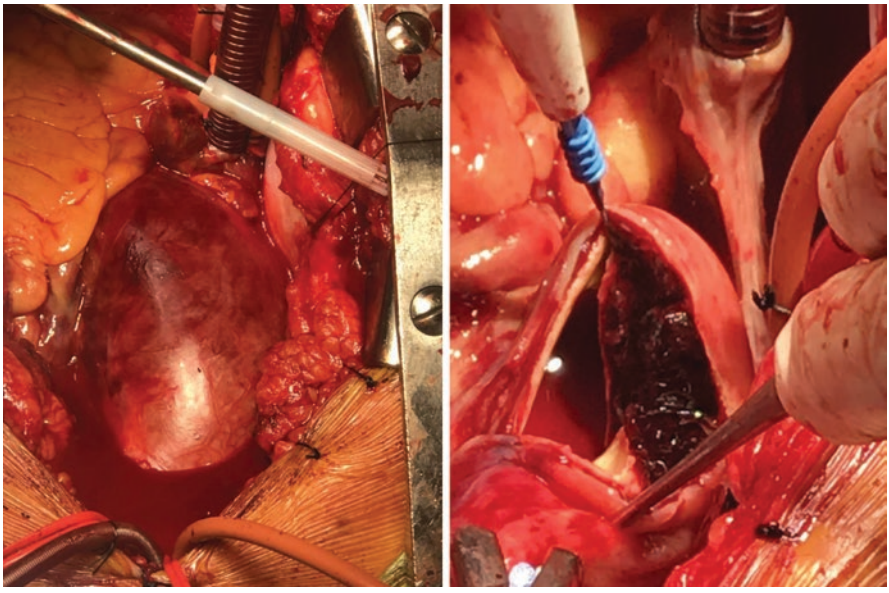

Fig. 22.3 Images of Stanford type A ascending aorta dissection

does not include the ascending aorta. The DeBakey classification has three categories. DeBakey I involves the whole aorta. DeBakey II involves the ascending aorta. DeBakey III involves the descending aorta. DeBakey I and II are Stanford classification A.

Dissection Management

Management focuses on the initial presentation.

- Hypotensive patients should be resuscitated.
- Hypertensive patients require adequate analgesia, and blood pressure management to maintain a systolic blood pressure between 110 and 120 mmHg, in an effort to reduce shear stress on the dissection. Beta- and alpha-blockers should be used as first-line agents. Nitrates and nitroprusside should not be used without prior beta-blockage to avoid tachycardia and an increase in delta pressure/delta time (dP/dt).
- Stanford A dissections
  - They require surgical management via median sternotomy. Generally, the goal is to resect the intimal tear, obliterate the entry into the false lumen proximally with adhesive reconstruction, and replace the ascending aorta and proximal arch to prevent complications that lead to death such as intrapericardial rupture, acute severe aortic insufficiency, proximal extension, or myocardial ischemia (coronary malperfusion). The main surgical approaches depend on the location of the primary intimal tear and the extent of the aortic dissection. If the primary intimal tear is in the ascending aorta "DeBakey type II dissections" then, interposition tube graft replacement of the ascending aorta is performed where the sinuses are preserved. If the primary intimal tear is in the aortic arch, then the ascending aorta and the aortic arch are replaced. The extent of the repair (hemiarch versus total arch) is dependent on extent of ascending and arch involvement. If the primary intimal tear is in the in the sinus of Valsalva or the dissection extends to the coronary ostium, aortic root replacement with or without reimplantation of the coronary arteries is performed. If the aortic valve is incompetent, aortic root replacement with resuspension of the structurally normal valve. If the aortic valve or the aortic root is abnormal, complete aortic root replacement with a composite valve graft and coronary arteries reimplantation is performed as described by Bentall and Cabrol procedures. Valve-sparing root replacement is performed if the aortic valve has strucurally normal valve leaflets as described by David reimplantation and Yacoub remodeling procedures.

The arterial cannulation line utilized for cardiopulmonary bypass should either be placed in the femoral or axillary artery. The right atrium or femoral vein maybe used for the venous drainage. The patient should be cooled, with the degree of hypothermia determined by the extent of the necessary repair, and the possible need for circulatory arrest.

- Stanford B dissections
  The management of Type B dissection continues to be debated. Overall, studies have shown that there is no major difference in early or late outcome between patients treated medically or surgically. Generally, adequate analgesia, control of hypertension, and maintenance of urine output are needed. Emergent or urgent operations are indicated for patients with evidence of rupture (hemothorax), inadequate perfusion, or persistent pain. Most groups favor an approach which addresses the idiosyncratic nature of the dissection and related complications for patients with acute type B dissections. Patients with connective tissue disorders should likely have early surgical intervention. Open or endovascular repairs may be done for patients who fail medical management.
- Intramural hematomas
  Intramural hematomas and penetrating ulcers are also forms of aortic instability. Intramural hematomas are thought to originate from rupture of the vasa vasorum within the outer third of the media resulting in accumulation of blood. A penetrating atherosclerotic ulcer forms after the rupture of an atheromatous plaque through the internal elastic lamina and allows extravasation of blood into the aortic wall. These should be treated surgically in a fashion similar to type A dissections.
- Chronic dissections
  Chronic dissections do exist, and generally lead to progressive aortic enlargement. Patients should be offered operative management when the ascending aorta is >5.5 cm (5 cm in Marfan syndrome), the rate of expansion is greater than 0.5 cm/year, or when patients become symptomatic. These patients require aggressive control of their blood pressure post operatively with beta-blockade. Currently, 80–90% of patients with type A dissection survive.

Intraoperative Anesthetic Management of an Aortic Dissection

Introduction

Acute ascending aortic dissections are a surgical emergency. These cases should preferentially be performed in a Cardiac Surgery Suite as these ORs have the equipment (bypass machine, large monitor screens, TEE machine, rapid blood transfusion capability, pharmaceutical agents needed to manipulate hemodynamic parameters, invasive monitoring equipment, etc.) and size that is requisite to safely care for these patients. Upon entering the operating theatre, we should physically move the patient over from the stretcher to the operating table on a sliding board to avoid patient exertion and hypertension, which could result in increased shear stress on the dissection.

Monitoring

- Monitoring for these patients follows American Society of Anesthesiologists (ASA) guidelines for standard monitors and additionally follows the standard protocol for performing cardiac surgery that involves cardiopulmonary bypass.

- A large bore central venous introducer with a pulmonary artery catheter is beneficial. If the case involves a redo-sternotomy, a second central intravenous line is helpful due to the high risk of significant blood loss.
- A focused transthoracic echocardiogram is frequently performed to evaluate the aortic anatomy, identify the true and false lumens, and to detect aortic insufficiency or regional wall motion abnormalities secondary to myocardial ischemia, or tamponade, when the dissection extends to include the aortic root, coronary ostia, or pericardium, respectively.
- A radial arterial catheter should be placed prior to the induction of anesthesia for invasive blood pressure monitoring. A femoral artery catheter may be used for blood pressure monitoring and may also be used to help expedite emergent femoral arterial cannulation for femoral–femoral cardiopulmonary bypass if needed, especially in patients requiring a redo-sternotomy. The surgeon may request bilateral radial arterial lines in order to detect a pulse deficit and to monitor the mean arterial pressure in the right upper extremity for cases utilizing right axillary arterial cannulation and antegrade cerebral perfusion during deep hypothermic circulatory arrest (DHCA). In this scenario, the blood pressure measured from the right radial artery provides the best estimate of cerebral perfusion. If a pulse deficit is detected, choose the artery that best represents the central aortic pressure for management.
- Near infrared spectroscopy is readily available in the operating room and should be used to assist with detecting cerebral malperfusion. In general, the anesthesia provider should try to maintain the cerebral oximetry value within 10–20% of baseline. A unilateral decrement should be communicated with the surgeon as this may help detect a misplaced arterial cannula or progression of the pathology that requires immediate surgical attention.
- Temperature monitoring should be employed with both a temperature-sensing Foley urinary catheter and a nasopharyngeal temperature probe. It should be noted that the PA catheter thermistor will not accurately reflect core body temperature if on cardiopulmonary bypass since there is no flow through the pulmonary artery during this time.
- Electroencephalography monitoring is often used during elective procedures of the ascending and aortic arch requiring DHCA. The goal is to ensure that cerebral metabolism is maximally reduced before interruption of cerebral flow in cases which involve deep hypothermic circulatory arrest. The goal is to pharmacologically induce an isoelectric state on the EEG before circulatory arrest is commenced.
- TEE should be used to verify the diagnosis, guide surgical management, anticipate hemodynamic perturbations, and to assess the surgical repair. In particular, patients suffering cardiac tamponade may experience severe and sudden hypertension causing aortic rupture, when the tamponade physiology is relieved upon opening the pericardium.

Procedure Technique and Anesthetic Management

- Surgical repair of the ascending aorta is performed on cardiopulmonary bypass (CPB) via a full sternotomy.
- The decision to use deep hypothermic circulatory arrest (DHCA) with or without antegrade or retrograde cerebral perfusion (ACP/RCP) should be discussed with the surgeon prior to the start of the procedure. DHCA is used to produce EEG silence. This generally drops the core temperature to 18 degrees Celsius, which leads to EEG silence and reduces ischemic injury. Inability to cannulate and clamp the ascending aorta is the main reason to use DHCA. This technique will have implications regarding the monitoring of pressures (cerebral and systemic) and the monitoring of temperature during deliberate hypothermia. DHCA with ACP or RCP is used to provide brain, spinal cord, kidney, and splanchnic organ protection for aortic surgery that involves the aortic arch. RCP is effective for brain cooling and can supply nutritive flow and flush out arterial emboli. RCP reduces dependence on deep hypothermic circulatory arrest and therefore the coagulopathy associated with it. It is initiated via the SVC. Tendegree Celsius blood is given at a rate < 500ml/min to keep CVP < 25mmHg. This technique is safe for 40 minutes. Most centers utilize antegrade cerebral perfusion techniques. ACP offers the most effective protection and moderate hypothermia 25 degree Celsius but can be technically difficult. Flow is initiated at a rate of 800ml/min or to keep right radial pressure of 40mmHg. In cases involving DHCA, the head is often actively cooled with ice or a special-purpose cooling helmet-type device which cools the head with cool circulating fluid. The myocardium is protected with cold blood cardioplegia and systemic hypothermia via cardiopulmonary bypass, and myocardial temperature is monitored.
- Corticosteroids are given to reduce the systemic inflammatory response generated by CPB and surgical inflammation. Corticosteroids are also commonly used for neuroprotection, although the data has not unequivocally demonstrated therapeutic utility.
- The main goal for the induction of anesthesia in cases of ascending aortic surgery is to avoid inducing hypertension. Uncontrolled systemic hypertension increases aortic wall tension and may lead to aortic rupture and death. Opioid analgesics are frequently utilized to blunt the sympathetic nervous system response to laryngoscopy and surgical simulation. The anesthesia provider should also have anti-hypertensive medications readily available, including esmolol and nicardipine. The intravenous antihypertension infusions running upon arrival may need to be discontinued or the dose reduced to avoid severe hypotension during the induction of anesthesia. This is especially true when the dissection is associated with pericardial tamponade or coronary artery compromise.
- An intraoperative blood salvage system may be used to collect autologous blood for later transfusion. The autologous blood is transfused after weaning from CPB, reversal of heparinization, and control of surgical bleeding. If the blood is harvested

after the administration of heparin, it is important to dose additional protamine for the reversal of the anticoagulant effect. Normovolemic hemodilution may also be utilized to minimize the need for allogeneic blood transfusions. A rapid infuser system should be available as blood loss can be precipitous and prodigious.

- Anticoagulation should be administered for an activated clotting time of at least 480 seconds prior to cannulation for cardiopulmonary bypass. Once CPB is established, systemic cooling to the chosen temperature is performed.
- It is important to monitor cerebral perfusion throughout the procedure, and near-infrared spectroscopy is a readily available monitoring technique to help diagnose and manage intraoperative malperfusion syndrome.
- In preparation for DHCA, the patient's head is often packed in ice while protecting the ears, eyes, and nose with gauze to prevent direct contact and thermal damage. Propofol, opioid, and non-depolarizing neuromuscular blockers are often re-dosed to further decrease cerebral metabolism and therefore provides neuroprotection.
- Hyperglycemia is common during DHCA and blood glucose should be maintained between 120 and 180 mmHg throughout the procedure.
- All infusions should be stopped during DHCA, especially epsilon-aminocaproic acid, if used as a prophylactic inhibitor of fibrinolysis.

Weaning from CPB

- To prepare for the termination of CPB, the patient should be fully rewarmed and vasoactive agents should be prepared and readily available to control hemodynamic conditions as needed. Any metabolic abnormality should be corrected, and epicardial pacemaker leads should be placed as required to maintain the desired heart rate and rhythm.
- A TEE exam is performed to assess the surgical repair and evaluate aortic valve, aortic anatomy, and myocardial function. Warming devices are employed to maintain normothermia.
- A viscoelastic test or rotational thromboelastometry is performed to determine coagulation status and guides the administration of blood products after separation from bypass and protamine administration.
- The patient is brought to the intensive care unit at the completion of the operation. They remain intubated, sedated, and monitored throughout transport.

Aortic Aneurysm

Definition

An aortic aneurysm is the localized dilation of the aorta, at least 50% greater than that of the normal adjacent aorta. Thoracic aorta aneurysms can be classified depending on the location into four anatomic categories:

- Aortic Root and ascending aortic aneurysms 50-60%.
- Aortic arch aneurysms 10%.

- Descending aortic aneurysms 30–40%.
- Thoracoabdominal aneurysms 10%.

Some aneurysms involve more than one area. A thoracoabdominal aortic aneurysm (TAAA) involves both the thoracic descending aorta and the abdominal aorta.

Pathophysiology

This is generally secondary to nonspecific medial degeneration, expansion secondary to chronic aortic dissection, or connective tissue disorders such as Marfan syndrome or Ehler–Danlos syndrome, which result in aortic dilation. Aneurysms can be also be caused by infections of the aortic wall, previous trauma, or aortitis. Generally, TAAA are found incidentally in older (>60 years of age) asymptomatic patients. Patients found to have a TAAA should undergo dedicated CT or MRI to delineate the extent of aorta involved.

Aortic Aneurysm Management

Repair is based on the extent of the aneurysm and therefore the risks associated with repair. Repair is intended to prevent fatal rupture, and should be performed when the operative morbidity and mortality is less than that of fatal rupture. Surgical and anesthetic management for aneurysms involving the aortic root, ascending aorta, and aortic arch are the same as disscused earlier in Type A dissection. For aneurysms involving the descending thoracic aorta, management is usually done by endovascular therapy Chap. 20. The current recommendation is to perform elective open repair in asymptomatic patients with TAAA >6 cm. Patients with connective tissue disorders such as Marfan should likely be repaired earlier (>5.5 cm). Repair should also be offered when rate of dilation exceeds 0.5 cm/year as this is indicative of an unstable aneurysm. Symptomatic aneurysms should also be repaired. Acute onset of symptoms is generally indicative of impending rupture or significant malperfusion.

Crawford Classification

The Crawford classification divides the repairs into four extents of repair. Extent I involve the aorta from just distal to the left subclavian artery to the suprarenal abdominal aorta. Extent II is the most extensive and involves the aorta from the left subclavian artery take off to aortoiliac bifurcation. Extent II has the highest surgical risk. Extent III involves the mid-descending thoracic aorta (below the sixth rib) to the aortoiliac bifurcation. Extent IV begins within the diaphragmatic hiatus and extents to the abdominal aorta (Fig. 20.3).

Intraoperative Anesthetic Considerations of Thoracoabdominal Aortic Aneurysm

As descending TAAs extend to the thoracoabdominal aorta, the extent of surgery increases the risks of paraplegia, renal insufficiency, and mortality. These cases may require full CPB, full CPB with DHCA, or simply left heart bypass.

Left Heart Bypass

- Left heart bypass (LHB) helps provide distal aortic perfusion and reduce ischemic conditions and is generally reserved for extensive Extent I and II TAAA repairs.
- LHB is generally performed from the left atrium to the femoral artery. During LHB, blood is shunted from the left atrium through a centrifugal pump and reinfused via the left femoral artery after the thoracic aorta is clamped for perfusion to the visceral organs and lower extremities.
- The blood in this circuit can be oxygenated, cooled, and heated. This partial bypass with the native circulatory system provides perfusion above the aortic cross-clamp and distal perfusion is provided by a left atrial shunt and the cardiopulmonary bypass machine.
- An arterial pressure monitoring line is placed in the upper and lower circulation to provide information about these two interrelated but separate systems.
- The anesthesia provider should maintain good communication with the perfusionist to adjust the pump speed and venous return for adequate unloading so that proximal and distal perfusion pressures are maintained.
- Selective visceral perfusion can also be employed with minimal added risk for extensive Extent I and II TAAA.

Position

The patient is positioned in left lateral decubitus and these cases are often performed through a left thoracotomy incision (Figs. 22.4 and 22.5).

Lung Isolation

- One-lung ventilation is required for a left thoracotomy approach and either a double lumen tube (DLT) or a single lumen tube (SLT) with bronchial blocker is utilized.
- Insertion of the DLT and TEE probe should be performed with caution and should not be forced against resistance, as it may lead to aneurysm rupture.
- Confirmation of proper placement of the DLT or BB with fiberoptic bronchoscopy is the standard of care.

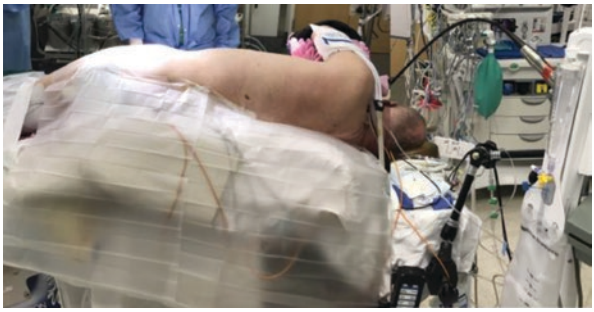

Fig. 22.4 A picture of patient positioned in the right lateral decubitus and ready for surgery

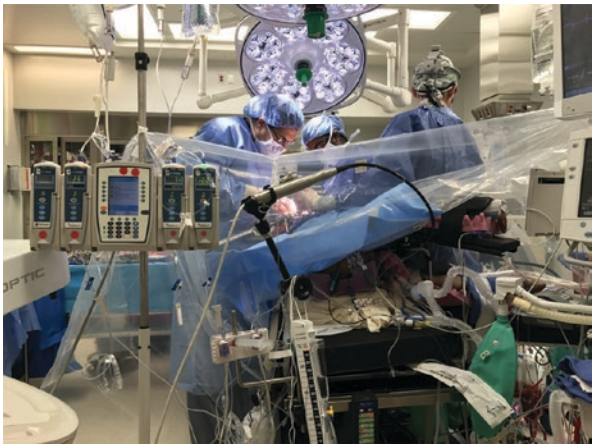

Fig. 22.5 A picture of patient positioned as surgery starts

Anticoagulation

- Anticoagulation with heparin depends on the type of bypass; approximately 1 mg/kg is used for LHB with an ACT goal of 300–400.
- Higher doses are required for full CPB.
- An antifibrinolytic medication is not used for LHB cases due to the risk of thrombosis.

Spinal Cord Perfusion

- Spinal cord perfusion is also compromised with TAAA repair secondary to sacrifice of intercostal (artery of Adamkiewicz) and lumbar arteries and are most at risk in extensive Extent II repairs.
- Ischemic complications can lead to paraplegia or paraparesis, which is a most devastating complication, with an incidence of as high as 15%. Spinal perfusion pressure (SPP) is the mean arterial pressure–spinal perfusion pressure.

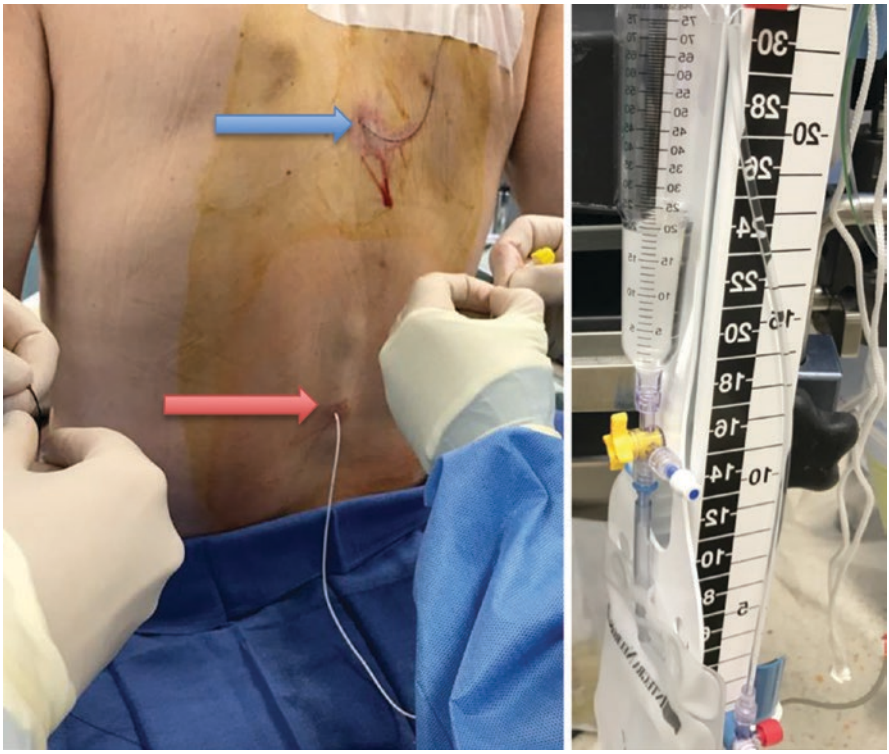

Fig. 22.6 Images of lumbar drain. Left: insertion of lumbar drain (red arrow) and thoracic epidural (blue arrow). Right: CSF collection system

- Cerebral spinal fluid (CSF) drainage has decreased the incidence of such complications by reducing the CSF pressure, and therefore improving spinal perfusion pressure (Fig. 22.6).
- CSF drainage is recommended for both open and endovascular repairs at high risk of developing paraplegia.
- Risk factors include older patients, comorbid renal dysfunction, emergency surgery, aortic rupture, acute dissection, extensive repair, long repair time, prior aortic surgery, and exclusion of the hypogastric artery.
- CSF drainage should be continued for 24–48 hours after surgery unless paraplegia or paraparesis is present, to maintain a target CSF pressure of 10–12 mmHg.
- Other techniques to reduce ischemia include sequential clamping and reimplantation of the intercostal arteries.
- Overly aggressive CSF drainage may lead to a subdural hematoma and bleeding into the CSF fluid, or frank herniation.

Renal Protection

- Renal protection should also be performed by direct perfusion of the renal arteries with cold lactated ringers or blood.

- When the ostia are accessible, a balloon-tipped catheter can be inserted and utilized to deliver a bolus of cold Lactated Ringers solution with mannitol (12.5 g/l) and methylprednisolone (125 mg/l) every 15–20 minutes, with diligent monitoring of fluid status.

Monitoring Techniques and Hemodynamic Targets

- The intraoperative anesthesia goals are similar to those of an ascending aortic dissection repair discussed above. Hemodynamic stability is the primary goal to preserve organ perfusion.
- The aim is to maintain hemodynamic stability and euvolemia without excessive volume administration.
- Maintenance of cerebral perfusion and the avoidance of emboli are important intraoperative goals if the aortic arch is involved. This is particularly important because of the comorbidities present in this patient population.
- A right radial or brachial artery catheter is needed to monitor pressures above the cross-clamp because the left subclavian artery may be surgically cross-clamped at some point during the operation. A femoral arterial catheter should be placed in the right femoral artery for blood pressure monitoring and to assess perfusion distal to the lower aortic cross-clamp. The left femoral artery is reserved for the surgical team for cases utilizing distal perfusion with LHB.
- The systolic blood pressure should be maintained at approximately 105–115 mmHg, and heart rate should be kept at 60–80 beats/min because a hyperdynamic myocardium may result in shear stress, leading to aortic rupture.
- Motor-evoked potentials (MEP) and somatosensory-evoked potentials (SSEP) are established methods of neuromonitoring geared at preventing paraplegia after thoracic aortic repair. SSEP involves monitoring the cortical electrical potentials after stimulation of distal nerves via the dorsal column. MEP involves electrical stimulation of the cranium overlying the motor cortex and recording the peripheral muscle action potential via the ventral column. Essentially, SSEP monitors the posterior or sensory pathway of the spinal cord. MEP evaluates the anterior or motor pathways, which are most prone to ischemia during aortic cross-clamping of the descending aorta. MEP and SSEP are selectively performed based on the surgeon's preference. Evoked potential monitoring requires total intravenous anesthesia (TIVA) technique, as volatile agents affect the amplitude of the recorded signals. Spinal cord perfusion is also compromised with TAAA repair secondary to sacrifice of intercostal (artery of Adamkiewicz) and lumbar arteries. The highest risk is in extensive Extent II repairs (Fig. 22.7). Ischemic complications can lead to paraplegia or paraparesis, which is the most devastating complication and occurs with a frequency as high as 15%. Spinal cord perfusion pressure is the pressure difference between mean arterial pressure and cerebrospinal fluid pressure.
- The anesthesia provider should be vigilant during the clamping and unclamping of the aorta. Unclamping syndrome may lead to severe hypotension as a result of severe hypovolemia caused by pooling of blood in the reperfused tissues,

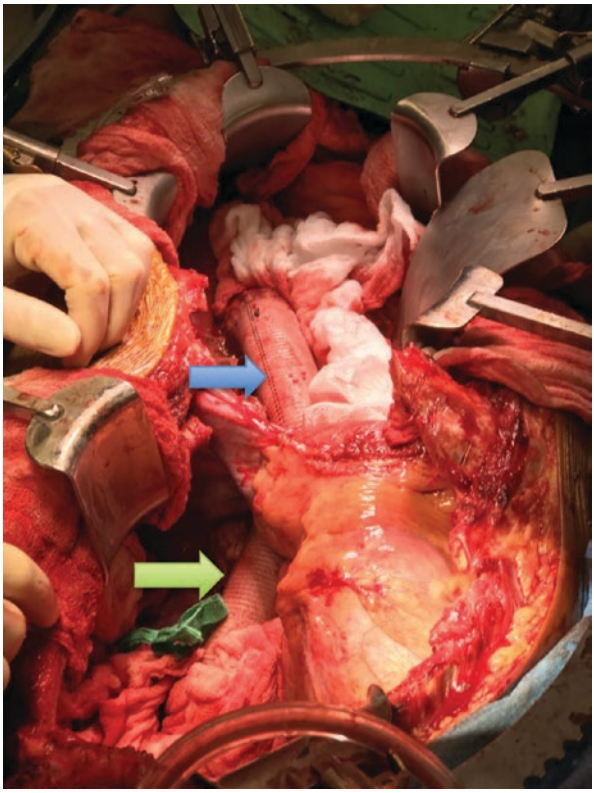

Fig. 22.7 A picture of thoracoabdominal aortic aneurysm repair with graft tube extending from the thorax (green arrow) to the abdomen (blue arrow)

vasodilatation, and myocardial depressant effects mediated by hypoxemia, and the metabolites from anaerobic glycolysis. Closely monitor and correct any acid–base disorder with serial arterial blood gas measurements.

- Fluid warmers and forced warm air blankets are necessary to prevent hypothermia as these cases often involve the transfusion of significant volume of blood products and evaporative heat loss, both of which can lead to hypothermia. Lower body warmers should not be used if the lower extremities are not being perfused during aortic cross-clamping, as it can increase the buildup of lactic acid.

Postoperative Management

- Unless it is obvious that the patient will shortly be returning to surgery for bleeding, change the DLT for a SLT. Video laryngoscopy with visualization and two soft-tip endotracheal tube exchangers is a safe and effective means for the procedure.
- The patient is brought to the intensive care unit at the completion of the operation.
- They remain intubated, sedated, and monitored throughout the transport.

- Postoperatively the patient should ideally be extubated within the first 24–36 hours with aggressive pulmonary toilet, as pulmonary complications are common.
- Initially the blood pressure should be rigorously managed, with a MAP of 80–90 mmHg for the first 24–48 hours, to maintain adequate tissue perfusion as hypotension can lower the SPP and increase the risk of paraplegia.
- Avoid hypertension which could lead to postoperative bleeding.
- Renal function should be monitored closely and addressed quickly if impaired.
- Lines and drains should be removed as soon as possible to decrease the risk of infection.
- Morality rates are dependent on the extent of repair, ranging from 3% to 16%.
- Paraplegia rates are also related to the extent of repair and range from 1% to 15%.
- Initially the blood pressure should be controlled tightly with a MAP around 80–90 mmHg for the first 24–48 hours.

Anesthetic management of open thoracoabdominal aortic aneurysm repair is summarized in Table 22.1.

Table 22.1 Summary of anesthesia management of open thoracoabdominal aortic aneurysm repair

Preoperative	Mild sedation
Location	Chest/abdomen
Duration	2–5 hours
Anesthetic	GETA Epidural for postoperative pain control
Medications	5000+ units heparin at discretion of surgeon Notify at 3 min Notify at 1 hour for possible re-dosing 25 g mannitol
Drips	Phenylephrine, nicardipine
Positioning	Right lateral decubitus (bean bag, axillary roll, ASIS at bed break, bed cannot be reversed)
Cases specifics	Two arterial line, central line Atrio-femoral bypass (left heart bypass), case done with perfusionist Lumbar drain required for prevention paraplegia Double lumen ET tube required Close discussion regarding clamping and unclamping Expected pressure increase with proximal clamp placement Expected pressure drop with distal clamp removal (reperfusion of legs) Communicate regarding lower urine output, may indicate problems with repair or need for diuretic at conclusion of case Communicate regarding BP drops, may indicate excessive retraction on IVC; perfusionist may need to adjust pump flow in distal circulation or unrecognized bleed Cell-saver required, estimated blood loss 1–2 liters Foley
Modifications	Type IV TAAA repair mirrors open AAA repair, CT surgery and atrio-femoral bypass not needed
Post-op Care	Intensive care unit

Further Reading

1. Patel PR, Augoustides JO, Pantin EN, Cheung AL. Thoracic aorta. In: Kaplan JO, Augoustides JO, Maneck GE, Maus TI, Reich DA, editors. Kaplan's cardiac anesthesia: for cardiac and noncardiac surgery. 7th ed. Philadelphia: Elsevier; 2017. p. 843–82.
2. Fedorow CA, Moon MC, Mutch WA, Grocott HP. Lumbar cerebrospinal fluid drainage for thoracoabdominal aortic surgery: rationale and practical considerations for management. Anesth Analg. 2010;111(1):46–58. https://doi.org/10.1213/ANE.0b013e3181ddddd6.

Anesthetic Management for Minimally Invasive Cardiac Surgery

23

Ahmed Zaky and Brad Meers

A little learning is a dangerous thing. Drink deep, or, taste not the Pierian spring.

—Alexander Pope

Introduction

Minimally invasive cardiac surgery (MICS) stands at the verge between the "historic" *open*-heart and the "current" *noninvasive* interventional techniques. It describes cardiac surgical procedures performed via incisions that are not full sternotomy. Outcomes based on these techniques are largely dependent on surgical experience and case volume. Current evidence demonstrates a cosmetic advantage, less hospital stay, and less arrhythmias and transfusion compared with conventional cardiac surgery. Currently, MICS has not been compared to rapidly evolving and recently validated noninvasive catheter-based techniques. The future of MICS is dependent on the scope and long-term outcomes of percutaneous catheter-based techniques. Also, adequately powered outcome studies are needed to compare MICS with its invasive and noninvasive counterparts. In this chapter we will review commonly conducted minimally invasive cardiac procedures, their surgical and anesthetic considerations, and complications.

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General Considerations for MICS

A comparison between minimally invasive cardiac surgery and conventional cardiac surgery is shown in Table 23.1.

Anesthetic Considerations Specific for MICS

(a) *Preoperative evaluation*

In addition to routine evaluation for conventional open-cardiac surgery, careful attention to the following is warranted:

1. *Preoperative chest CT*: This will provide valuable information about the amount of calcification, chest wall deformation, and the location of the great vessels in the mediastinum to facilitate surgical exposure.
2. *Preoperative pulmonary status:* In the form of pulmonary function tests as well as smoking history and any previous surgery on the lungs. These factors can predict the tolerability to one lung ventilation intraoperatively.
3. *Anatomy and patency of neck vessels*: This is relevant for cannulation of the coronary sinus via the internal jugular route.
4. *Any esophageal pathology* such as diverticulum or hiatal hernia. This may contraindicate placement of transesophageal echocardiography (TEE). TEE is invaluable in these cases.
5. *Peripheral vascular disease*: Native vascular integrity and the presence of vascular grafts need to be sought preoperatively in order to decide on cannulation side and site and cannulae size.

(b) *Monitoring*

Proceeds as in open-heart approaches including ASA monitoring and invasive monitoring in the form of arterial, central venous, pulmonary artery, and TEE. Care should be taken to free the right internal jugular site if coronary sinus cannulation is intended via the internal jugular approach for retrograde cardioplegia.

Table 23.1 Minimally invasive cardiac surgery versus conventional cardiac surgery

Point of comparison	MICS	Conventional open-heart surgery
Cosmesis	Superior	Inferior
Length of stay, ICU	Shorter	Longer
Length of stay, hospital	Shorter	Longer
Mechanical ventilation	Shorter	Longer
Return to full functionality	Shorter	Longer
Complication		
Blood transfusion	Less	More
Surgical pain	Less	More
Atrial fibrillation	Less[a]	More

MICS minimally invasive cardiac surgery, *SAVR* surgical aortic valve replacement, *ICU* intensive care unit

[a]In minimally invasive mitral valve repair

(c) *Positioning*

Careful communication with the surgeon is imperative regarding patient positioning. An anterior thoracotomy approach for minimally invasive mitral valve surgery (MIMVS) will usually require a slight left lateral tilt of the torso.

(d) *Conduct of anesthesia*

Although performing minimally invasive aortic valve replacement (MIAVR) under thoracic epidural anesthesia has been reported, the convention is to perform these cases under general endotracheal anesthesia. The use of short-acting induction agents and muscle relaxants is imperative for early extubation and fast-tracking of the patient.

(e) *Airway management*

One-lung ventilation (OLV) is often required to facilitate exposure and to check for bleeding points after separating from CPB. The right lung is usually deflated for aortic and mitral minimally invasive surgery. For CABG requiring access through the left chest, the left lung is deflated. OLV is managed in the same manner as in thoracic procedures. If a double-lumen tube is used, before the completion of the procedure, it is replaced by a single-lumen endotracheal tube using glidoscope and tube exchanger to avoid risk of lossing the airway. An alternative for a double-lumen tube is a bronchial blocker which has the advantage of not requiring an exchange of endotracheal tube at the end of the procedure. The bronchial cuff preferably deflated prior to initiation of CPB to avoid bronchial wall ischemia since bronchial blood flow during CPB is reduced and OLV should be reversed prior to weaning from CPB to prevent pulmonary edema in the deflated lung. If the surgeon requires it, it can be reinstated after separation from bypass for a short period of time. If extubation is planned in the operating theater, the double-lumen tube may be continued through the end of the procedure with both lung ventilation and without the need to exchange to a single-lumen tube.

(f) *Intraoperative transesophageal echocardiography*

In addition to conventional indications to use TEE in open-heart surgery, TEE serves as an invaluable tool in MICS to assess the following:

1. Cannulation sites: Placement of guidewires for peripheral arterial and venous cannulation as well the use of coronary sinus cannula for retrograde cardioplegia, if used
2. De-airing is a crucial step MICS
3. Assessment of perivalvular leak
4. Assessment of postprocedural complications, such as cardiac tamponade

Important TEE windows relevant to MICS:

1. Basic and comprehensive exam related to the valvular pathology
2. Mid-esophageal bicaval/modified-bicaval view to confirm venous drainage cannulation
3. Transgastric descending aortic view to detect arterial wires
4. Mid-esophageal ascending aortic long-axis view to examine the endoclamp position
5. Mid-esophageal ascending aortic short-axis view to confirm EndoVent pulmonary artery catheter

(g) *Postoperative pain relief:*

Perioperative analgesia aims effective pain control with minimal respiratory depression. Opioids should be minimized to enhance faster recovery and less side effects in the form of nausea and ileus. Oral gabapentin and Tylenol are usually given orally preoperatively. After the completion of the procedure, multilevel intercostal nerve block using long-acting local anesthetic is frequently employed. Other regional techniques in the form of high thoracic epidural, paravertebral blocks are less frequently employed.

(h) *Fast-tracking*

One of the foremost advantages of MICS is faster recovery. Institution protocols for early extubation vary across institutions; with most institutions attempt to extubate the patient in the operating room or within the first 2 hours of arrival to the intensive care unit, provided there is hemodynamic stability and no concern for bleeding. Usually, patients are discharged to the ward the following morning and discharged home 3–5 days after surgery.

Specific Procedures

Minimally Invasive Aortic Valve Replacement (MIAVR)

Patient Selection

Contraindications for MIAVR:

1. Chest wall deformities such as pectus excavatum
2. Patients with severe calcification of the aortic root
3. History of pneumonectomy
4. Previous sternotomies
5. History of chest irradiation
6. History of pericardial disease
7. Multiple valvular abnormalities requiring surgical intervention
8. Morbid obesity is a relative contraindication

Surgical Considerations

(a) *Approach:*

The most common approach is partial "J" mini sternotomy, followed by right upper mini thoracotomy, and then followed by others. Parasternal and transverse sternotomy incisions have been abandoned because of higher incidence of lung herniation requiring re-operation (Fig. 23.1). The different approaches for MIAVR are noted in Table 23.2.

Incisions are assessed based on the following criteria:

1. Ease of exposure
2. Ease of venous and aortic cannulation
3. Ease of delivering cardioplegia to achieve myocardial protection

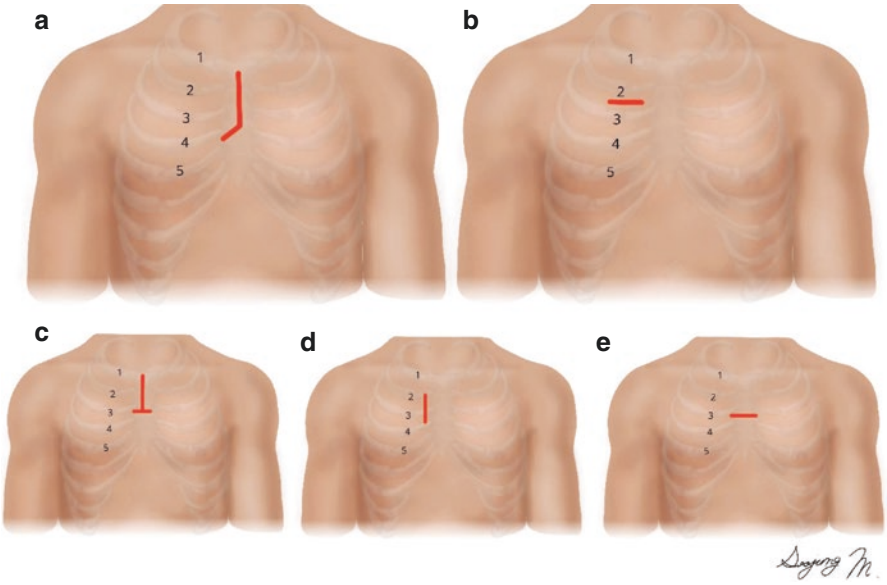

Fig. 23.1 Various minimally invasive approaches for minimally invasive access to the aortic valve. (**a**) Upper (J) mini-sternotomy. (**b**) Right anterior mini-thoracotomy. (**c**) Inverted "T" incision. (**d**) Right parasternal incision. (**e**) Transverse sternotomy

Table 23.2 Comparison of different approaches for MIAVR

Point of comparison	Mini-sternotomy	Mini-thoracotomy
Adoptability	+++	+
Exposure	Easier	Harder
Cannulation		
Aortic cannulation	Harder	Harder
Right atrial cannulation	Easier	Easier
Access to aortic root pathology	Easier	Harder
Patient selection criteria	None special	At the level of the ascending aorta, the RPA is located rightward to the sternal border Distance from ascending aorta to sternum is <10 cm α angle between the midline and inclination of the Asc Ao is >45 °
Outcomes/complications		
Afib	+++	+
Hospital length of stay	++	+

RPA right pulmonary artery, *Asc Ao* ascending aorta, *Afib* atrial fibrillation

4. Adequacy of venting of the left ventricle
5. The ease of addressing concomitant aortic root pathologies
6. Postoperative outcomes and complications

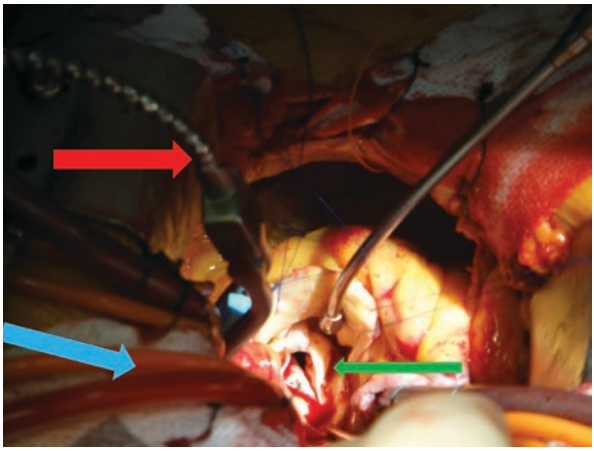

Fig. 23.2 Image of Mini AVR through right thoracotomy for stenotic bicuspid aortic valve (green arrow) with central cannulation (blue arrow) and flexible aortic clamp (red arrow)

(b) *Defibrillation pads:*

Transcutaneous pads are usually used given the small incision. Pediatric internal defibrillation paddles can be attempted in the mini-sternotomy approach.

(c) *Cannulation*:

Central aortic cannulation (Fig. 23.2) is preferred to peripheral cannulation since the latter is less physiologic and is associated with vascular injury and a higher incidence of stroke.

(d) *Left ventricular venting:*

Compared to an open approach, venting via the right superior pulmonary vein might be more challenging. An LV vent can be placed through the left atrium and the mitral valve and its position verified using TEE.

(e) *Cardioplegia:*

Antegrade cardioplegia can be employed in the usual fashion. Retrograde cardioplegia can be delivered in case of aortic regurgitation via a coronary sinus cannula inserted under fluoroscopy or under echo guidance. Figure 23.3 shows the EndoPledge® CS catheter (Edwards Lifesciences, Irvine, CA).

(f) *Placement of LV pacing wires*:

This takes place while still on cardiopulmonary bypass as compared with after coming off in the conventional surgical aortic valve replacement (SAVR). It may be advisable to check the pacing threshold to ensure the wire makes good contact with the epicardium, as it is surgically more challenging to place the wires through a small incision that is some distance away from the epicardium.

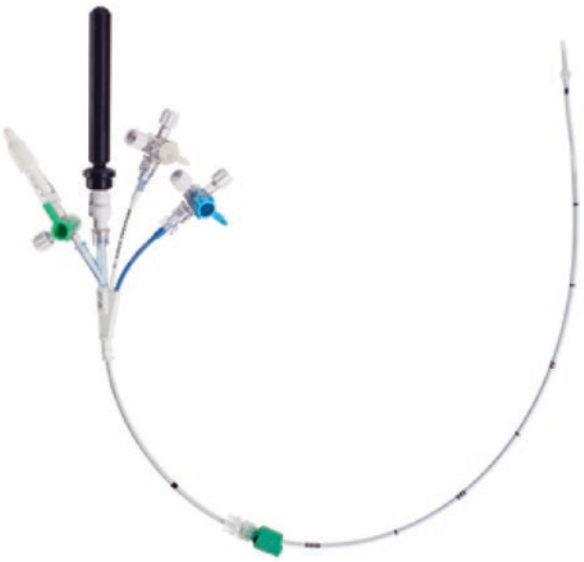

Fig. 23.3 Percutaneous coronary sinus cardioplegia catheter. (EndoPledge® CS catheter, Edwards Lifesciences, Irvine, CA)

Minimally Invasive Mitral Valve Surgery (MIMVS)

Patient Selection

Whereas most aortic valve operations can be performed through a minimally invasive approach, mitral valve surgery patients must be selected more carefully.

Contraindications for MIMVS

1. Aortic valve insufficiency greater than 1+
2. Severe mitral annular calcification
3. Significant left ventricular dysfunction or dilation
4. Severe pulmonary hypertension
5. Extensive aortoiliac atherosclerosis
6. Small femoral artery diameter (<7 mm)
7. Chest wall abnormalities, such as pectus excavatum
8. Previous left lobectomy or pneumonectomy causing a shift of the mediastinum to the left
9. Morbid obesity is a relative contraindication

Surgical Considerations

(a) *Approach:*

Right anterolateral thoracotomy is the preferred and most widely adopted approach. The right parasternal incision was abandoned because of complications in the form of chest wall instability and bradycardia as a result of transection of the sinus node artery during an altered transseptal approach (Figs. 23.4 and 23.5).

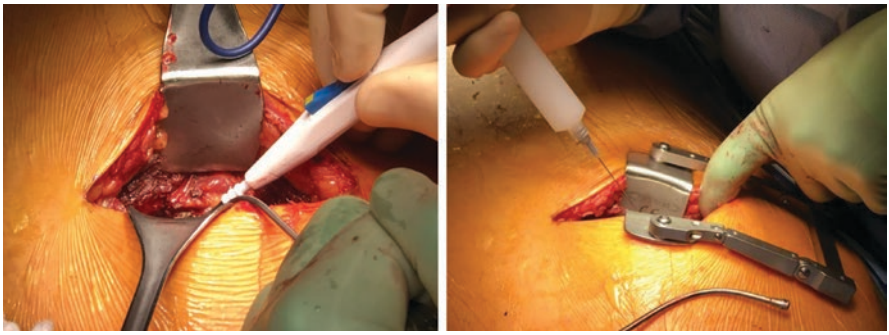

Fig. 23.4 The right anterolateral thoracotomy approach. *Left*. Opening intercostal space. *Right*. Injecting long-acting bupivacaine (EXPAREL) for postoperative analgesia

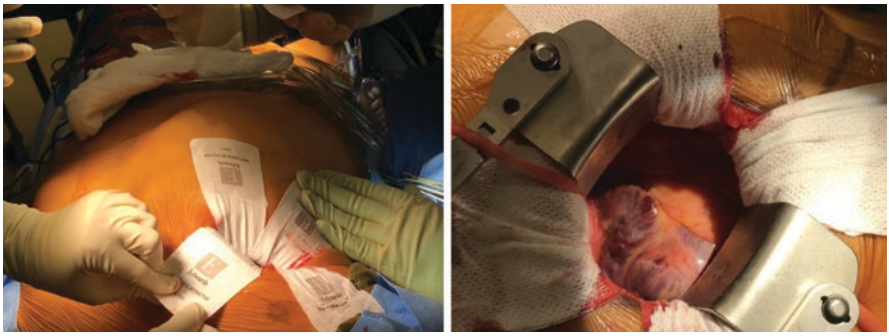

Fig. 23.5 The right anterolateral thoracotomy approach. *Left*. Soft self-retaining retractor. *Right*. Full exposure with soft and metal self-retaining retractor

(b) *Defibrillation pads:*

As a result of the small incision, there is little room for conventional or pediatric defibrillation paddles. As such the transcutaneous pads are used.

(c) *Cannulation:*

1. *Venous Cannulation:*

Venous cannulation is typically performed peripherally via the femoral vein, after a small dose of heparin (Fig. 23.6).

- Some surgeons prefer additional venous drainage via a right internal jugular vein (IJV) or SVC cannulation. The orifice of the SVC cannula must reside in the body of the right atrium for adequate drainage. The cannulation site for the IJV is typically *low* in the neck. Occasionally, some surgeons use the EndoVent pulmonary catheter (Edwards Lifesciences, Irvine, CA) to achieve pulmonary artery venting, and hence better surgical exposure. The EndoVent pulmonary catheter is similar to the conventional pulmonary artery catheter (PAC), yet is flimsier and

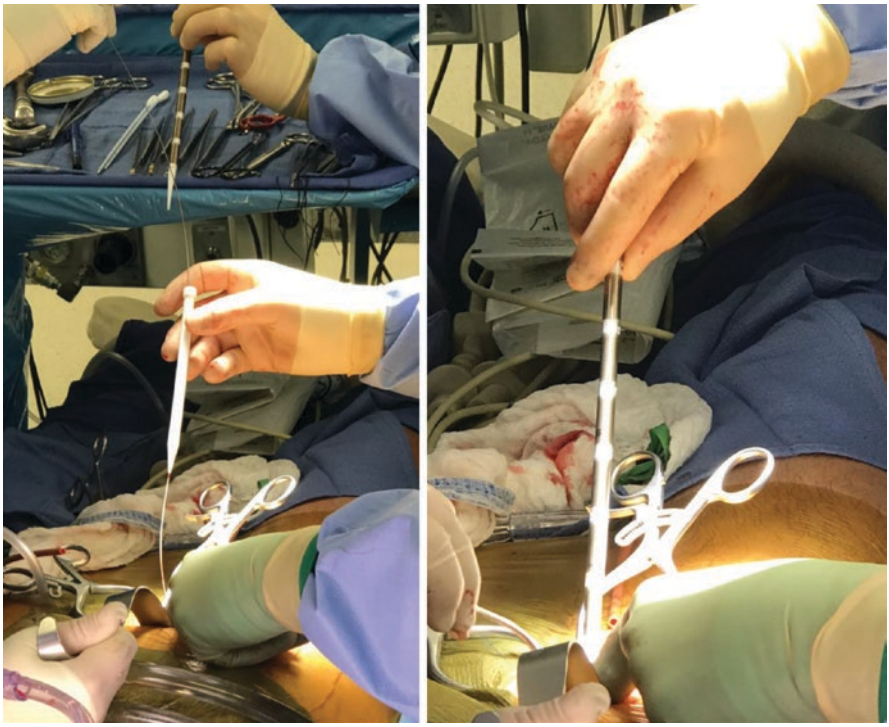

Fig. 23.6 Right groin cannulation. *Left*. Femoral vein dilator. *Right.* Femoral vein cannulation

contains multiple distal venting openings, allowing an average drainage rate of about 50 ml/min (Fig. 23.7).

- The latter strategy is often employed in MIMVS as the surgical approach to the mitral valve is via a left atriotomy as opposed to the transseptal approach used in conventional mitral surgery. If, however, there is a plan for right atriotomy (as is the case in concomitant tricuspid repair or atrial septal defect closure), a bicaval cannulation and occlusion may be necessary to prevent air entrainment from the exposed right atrium. It is important to recognize the risk of venous trauma, arrhythmias, and atrial perforation during this multisite cannulation. The propensity of cardiac tamponade must always be entertained by the anesthesiologist and sought via TEE.

2. *Arterial* Cannulation

   Arterial cannulation is achieved via the femoral artery (Fig. 23.8). A percutaneous transthoracic aortic cross clamp is placed through a separate stab incision. The location of this incision and the precise type of clamp is surgeon specific. The use of an endoclamp (Endoclamp Aprtic Catheter, Edwards Lifesciences), which is a balloon-based occlusion and cardioplegia delivery

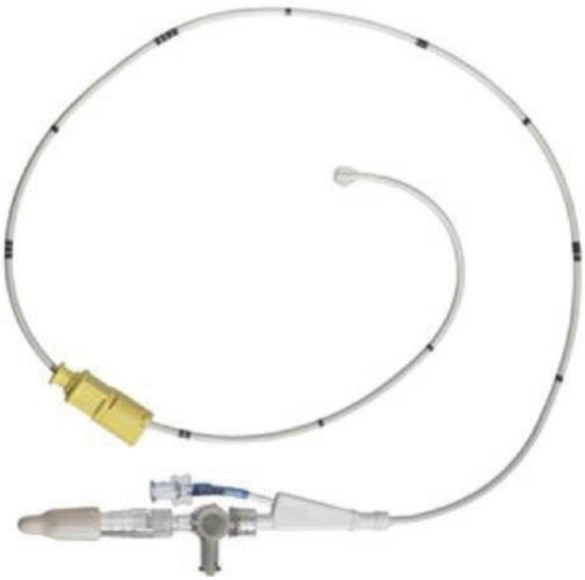

Fig. 23.7 Pulmonary artery venting catheter. (EndoVent Pulmonary Catheter, Edwards Lifesciences, Irvine, CA)

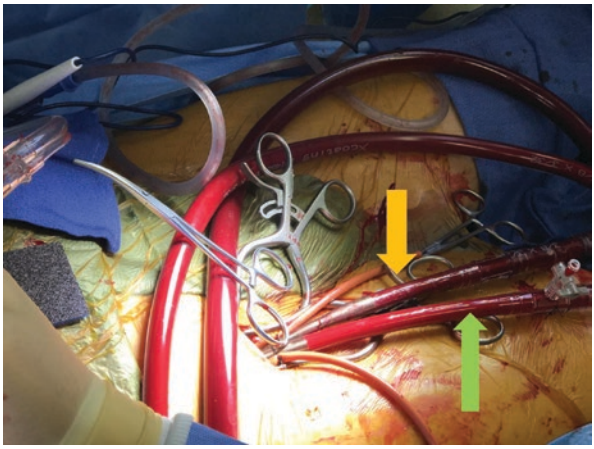

Fig. 23.8 Cannulation of the femoral vein (yellow arrow) and artery (green arrow) cannulas

system positioned in the proximal aorta, was associated with a higher incidence of stroke and procedural complications. Its use has therefore declined in modern MICS. Care should be exercised in peripheral femoral cannulation since it is more commonly associated with vascular injury and propensity for limb ischemia. Patients with peripheral vascular disease are specifically predisposed.

(d) *Cardioplegia:*

Antegrade cardioplegia can be employed in the usual fashion. Retrograde cardioplegia can be delivered in case of aortic regurgitation via a coronary sinus cannula inserted under fluoroscopy or under echo guidance.

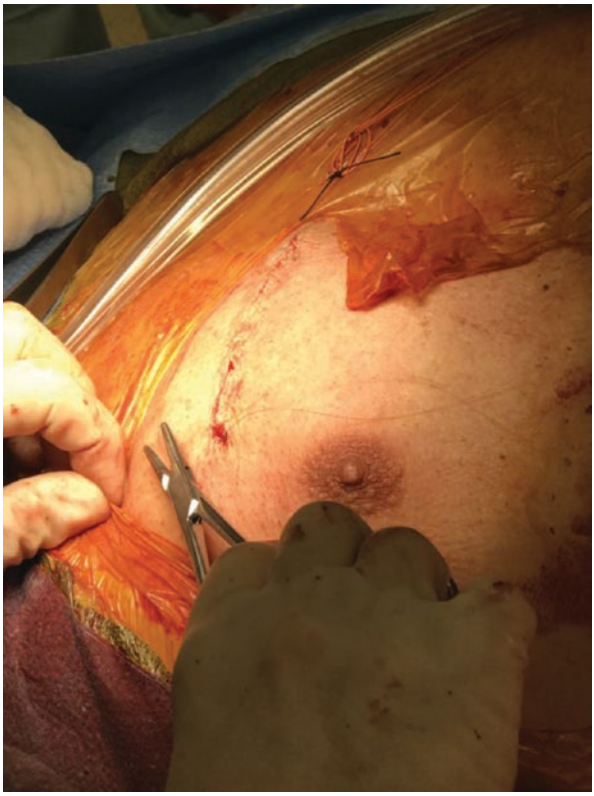

Fig. 23.9 Image of right anterolateral thoracotomy skin incision closure (notice the small incision)

(e) *Placement of LV pacing wires:*
This takes place while still on cardiopulmonary bypass as compared with after coming off in the conventional MVR.

(f) *Wound closure:*
Closure usually yield a small incision (Fig. 23.9).

Other Minimally Invasive Cardiac Surgery

Right Atrial Procedures

At times, a minimally invasive approach will be favored for closure of an atrial septal defect, removal of a right-sided cardiac tumor, or for treatment of an isolated tricuspid valve condition. We would not generally favor a minimally invasive approach for tricuspid valve replacement in which case there is a high probability of needing a permanent pacemaker. Permanent pacemakers cannot be placed across a prosthetic valve and it is desirable to have well-functioning permanent epicardial leads placed at the time of operation.

Our surgical approach is through a right anterolateral mini-thoracotomy. Since complete cardiopulmonary bypass is required for right-sided surgery, tourniquets are placed surrounding the superior and inferior vena cavae (SVC and IVC). Inferior

vena caval venous drainage is obtained in the usual manner with standard percutaneous femoral venous cannulation. Transesophageal echo guidance is used to manipulate the venous cannula into position just beneath the diaphragm. Superior vena caval drainage is obtained percutaneously through the right internal jugular vein. A 16 or 17 French cannula is placed by a cardiac anesthesiologist, often in collaboration with the surgeon. These large bore cannulas must be flushed with heparinized saline solution. Particular attention must be paid toward not introducing air during placement of the cannula. This is especially important in surgery for atrial septal defect. Caval tourniquets are secured prior to opening the right atrium and are not released until the right atrium is closed. Double access of the right internal jugular vein is possible and is often performed, where one of the cannulae is the SVC drainage cannula and the other is an 8 or 9F introducer for volume and vasoactive drip administration, and measurement of CVP.

Minimally Invasive Coronary Artery Bypass Grafting (MICAB)

Despite of its initiation in the 1990s, it has not been widely adopted because of difficult exposure of the coronaries and the technical difficulty in performing proximal anastomoses. Besides, the perception of off-pump coronary artery bypass grafting (CABG) as a "less invasive" procedure compared to CABG has reduced the popularity of this technique. As a result, there are no adequately powered comparative studies to compare this technique to conventional CABG or open off-pump CABG (OPCAB).

The procedure is performed via a left thoracotomy incision, requiring one lung ventilation. Patient selection, contraindications, and anesthetic considerations are similar to other minimally invasive procedures. The procedure may be performed off-pump. This procedure is best suited to grafting the left internal mammary artery since this requires only one distal anastomosis to be performed. The sequence of surgical events and anticoagulation follow the same sequence as OPCAB. The procedure can also be performed using the da Vinci® surgical robotic systems (Intuitive Surgical, Sunnyvale, CA).

The following are predictors of converting to open sternotomy or full CPB:

1. Heavy smoking history
2. Intolerance to OLV
3. Dilated LV and low ejection fraction
4. Multivessel disease
5. Concomitant mitral regurgitation
6. Inadequate surgical exposure

Further Reading

1. William F J, Gorav A. Surgical management of minimally invasive aortic valve operations. Semin Cardiothorac Vasc Anesth. 2012;16(1):41–51.
2. Klein P, Klop IDG, Kloppenburg GLT, van Putte BP. Planning for minimally invasive aortic valve replacement: key steps for patient assessment. Eur J Cardiothorac Surg. 2018;1(53(suppl_2)):ii3–ii8.
3. Une D, Sakaguchi T. Initiation and modification of minimally invasive coronary artery bypass grafting. Gen Thorac Cardiovasc Surg. 2019;67:349. https://doi.org/10.1007/s11748-018-1050-7.
4. Cheng DC, Martin J, Lal A, et al. Minimally invasive versus conventional open mitral valve surgery: a meta-analysis and systematic review. Innovations (Phila). 2011;6:84–103.
5. Modi P, Hassan A, Chitwood WR Jr. Minimally invasive mitral valve surgery: a systematic review and meta-analysis. Eur J Cardiothorac Surg. 2008;34:943–52.

Anesthetic Management for Heart Failure and Transplantation

24

Shonali Pawar and Eric Wilkens

If you fail to plan, you are planning to fail.

—Benjamin Franklin

Key Points

- Recipient Selection
- Left Ventricular Assist Devices
- Anesthetic Preoperative Checklist
- Line Placement and Monitoring
- Induction and Intraoperative Management
- Pulmonary Hypertension and Right Heart Failure
- Nitric Oxide Therapy

Introduction

Cardiac transplantation is widely accepted as a treatment of choice for patients with end-stage heart failure. Successful outcomes depend on many factors including proper patient selection, donor optimization, coordination and timing, optimized strategies for immunosuppression, and detailed perioperative care. In this chapter, we highlight patient selection, monitoring, and intraoperative and postoperative management of heart transplants.

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General Considerations

The most common conditions leading to transplantation are nonischemic cardiomyopathy followed by ischemic cardiomyopathy. Impaired myocardial systolic function may be associated with a higher resting heart rate, chronically increased preload, and reduced afterload as a result of pharmacological therapies. Many patients will be treated with anticoagulants to prevent intracardiac thrombus and systemic embolization. Those patients who have undergone left ventricular assist device (LVAD) placement may demonstrate better overall physical conditioning due to increased tolerance for exercise. LVAD patients may also tolerate hemodynamic changes during anesthetic induction better than those with unassisted physiology. Of significant importance is the donor and recipient matching, immunosuppression, and denervated heart physiology after transplant. Coordination of donor and recipient surgical procedures in order to minimize ischemic time is essential for graft function. Right ventricular failure is the most common complication immediately following transplantation requiring vigilance and immediate intervention.

Recipient Selection

Heart transplant recipients typically present with New York Heart Association (NYHA) Class III or IV heart failure symptoms. Some other indications for cardiac transplantation are as listed in Table 24.1.

Conversely, contraindications to heart transplantation have traditionally included the comorbidities listed in Table 24.2. More recently some centers have performed multiorgan transplants challenging the notion that these conditions prohibit consideration for cardiac transplantation.

Table 24.1 Indications for heart transplantation

Refractory cardiogenic shock requiring intra-aortic balloon pump (IABP) or left ventricular assist device (LVAD)
Cardiogenic shock requiring continuous inotropic therapy (dobutamine, milrinone etc.)
Peak VO2 less than 10 ml/kg/min
NYHA Class III or IV despite maximized medical and resynchronization therapy
Recurrent life-threatening left ventricular arrhythmias despite implantable cardiac defibrillator, ablation, and antiarrhythmic therapy
End-stage congenital HF with no evidence of pulmonary hypertension
Refractory angina without potential medical or surgical therapeutic options

ACC/AHA guidelines, Jessup et al. Circulation (2009)

Table 24.2 Contraindications to heart transplantation

Advanced irreversible renal failure
Advanced irreversible liver disease
Advanced irreversible pulmonary parenchymal disease
Advanced irreversible pulmonary artery hypertension
History of solid organ or hematologic malignancy in the last 5 years

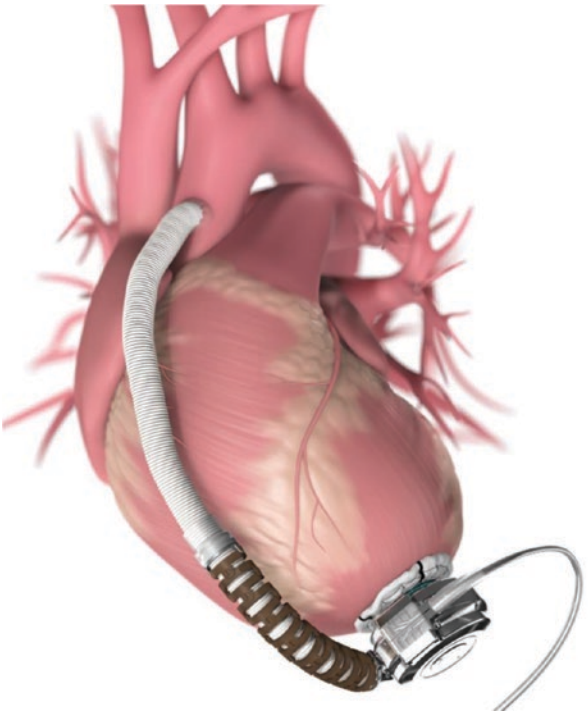

Fig. 24.1 Illustration of Heartware device – typical configuration for a continuous flow LVAD with apical positioning of the inflow device and ascending aortic outflow

Left Ventricular Assist Devices

Many patients presenting for cardiac transplant will have undergone prior LVAD insertion. It is important to understand the basic physiology of these mechanical circulatory assist devices, and to recognize the increased risk posed by the need to redo sternotomy with potential injury to cardiac structures or the LVAD device itself. Examples of LVAD devices are shown in Figs. 24.1 and 24.2.

LVAD flow is dependent on both preload and afterload. The device output can be adjusted by altering the pump speed (rpm). It is important to make any such changes in alignment with the preload delivered by the right ventricle. If an increase in

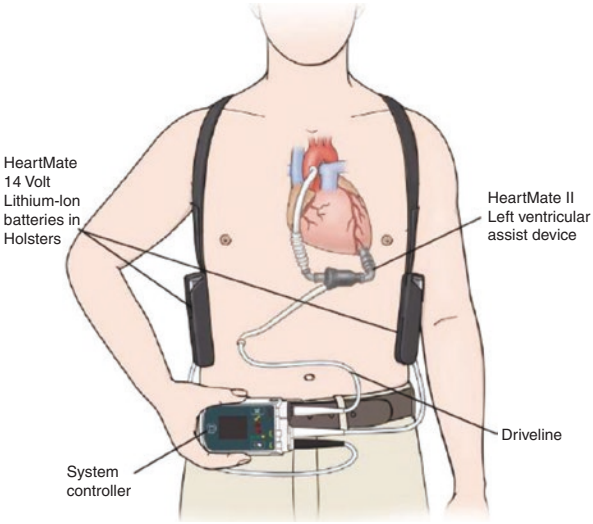

Fig. 24.2 Illustration of ambulatory LVAD Therapy – external power source and controlling unit connected through a single percutaneous line to the implanted continuous flow device

LVAD speed is not matched by adequate delivery of blood volume by the right ventricle, the left ventricle will collapse (a "suction event"), resulting in reduced device output and systemic hypotension. Intraoperative TEE can help monitor optimal LVAD flow by ensuring a neutral position of the interventricular septum and close monitoring of right ventricular function.

Anesthetic Preoperative Checklist

An organized approach to prepare for an impending cardiac transplantation procedure will minimize the risk of overlooking critical issues.

- *Communication*: Coordinate with heart transplant coordinator and surgical team information/changes regarding procedure times and patient information. Both donor and recipient teams stay in close contact to minimize ischemic times (4–6 hours).
- *Preoperative Evaluation*: History and physical examination to identify issues relevant to anesthetic and surgical care.
- *Informed Consent*: Ensure surgical and anesthesia consents have been obtained. Maintain the anonymity of donor while in depth discussion with recipient and family members.
- *Arrhythmia Management*: Note any history of malignant arrhythmias or implantation of an antiarrhythmic device. An AICD will require reprogramming to disable antiarrhythmic therapies prior to surgery.
- *Cardiac Function*: Review available electrocardiograms, chest X-rays, echocardiograms, and left and right heart catheterizations to fully understand the recipients baseline cardiac status.

- *Lab Studies*: Complete blood count (CBC), chemistry, arterial blood gases, prothrombin time (PT), partial thromboplastin time (PTT), type, and crossmatch.
- *Blood Products*: Packed red blood cells (PRBCs) must be available prior to induction for all redo sternotomy patients. Minimize recipient alloimmunization through the use of leukocyte filters and cytomegalovirus-matched donor units. If organ donor is CMV positive, patient will also receive antivirals.
- *Immunosuppressants*: Request appropriate immunosuppression regimen from the transplant coordinator (simulect or campath induction). Confirm that cyclosporine has been administered preoperatively.
- *Antibiosis*: Ensure that needed antibiotics are available. Institutional protocols may vary.
- *NPO Status*: Heart transplants are unscheduled procedures, but often involve advanced notice to the potential recipients. If the duration of preoperative fasting is questionable, consider administering a nonparticulate antacid, metoclopramide, and H2 blockers.
- *Circulatory Assist Devices*: In most centers, a perfusion team will help transport to the OR any patient supported with an LVAD or IABP device. LVAD batteries need to charge prior to transport and a device-specific control console should be available in the operating room.

Pharmacological Agents

Advanced preparation of the pharmacological agents, likely to be required during cardiac transplantation, can save time and smooth the workflow during the procedure itself. For more information, please check Chap. 3.

- *Vasopressors* – vasopressin, norepinephrine, phenylephrine should be readily available. Many cardiac transplant recipients will be chronically treated with ACE inhibitors, angiotensin receptor blockers, or other direct vasodilators. Post–cardiopulmonary bypass (CPB) vasoplegia can be a challenge in these patients.
- *Inotropes* – dobutamine, epinephrine, and milrinone should be accessible before induction.
- *Inhaled nitric oxide (iNO)* – if required by institutional protocol should be brought to the OR and connected with the anesthetic breathing circuit. The theoretical benefit of this agent in cardiac transplant is its action as a selective pulmonary vasodilator.
- *Antifibrinolytics* – aminocaproic acid or tranexamic acid should be administered prior to CPB to reduce plasminogen activation and improve postoperative clot stability.
- *Vitamin K* – once acceptance of the donor organ has been confirmed, vitamin K 10 mg IV should be administered to any patient receiving warfarin preoperatively. If the INR is >2, reversal with prothrombin complex concentrates such as Kcentra may be considered post-CPB (25 units/kg for INR 2–4, 35 units/kg for INR > 4).

- *Heparin* – 300 units/kg should be ready for immediate administration if required due to hemodynamic compromise at the time of anesthetic induction.
- *Immunosuppressants* – either simulect or campath are given following anesthetic induction and methylprednisone 1 gm IV is administered at the time of heparinization.
- *Insulin* – may be required to maintain blood glucose levels, in particular following high dose steroid administration.

Line Placement and Monitoring

Standard American Society of Anesthesiologists (ASA) monitors are used, and cerebral oximetry is particularly valuable to assist in interpreting hemodynamic changes during the induction of anesthesia. In cases where hemodynamic status limits the doses of anesthetic agents that can be administered, consideration should be given to monitoring depth of anesthetic state with a modified EEG (e.g., BIS).

All intravascular catheters should be placed in a strictly aseptic fashion using maximal barrier precautions in immunocompromised transplant recipients. Preexisting central venous or arterial lines from the intensive care unit should be removed and new catheters placed in a separate site to reduce the chances of infection. PICC lines are also usually removed once new central venous access is achieved.

Arterial line placement is challenging for many of these patients due to chronically low cardiac output, low systemic arterial blood pressures, and non-pulsatile arterial blood flow generated by axial flow LVADs. The use of ultrasound to locate the radial or brachial arteries can greatly expedite access. Cannulation of a femoral artery should be considered when upper limb vessels prove to be inaccessible.

A large bore central venous introducer should be placed under ultrasound guidance. It is important to rule out clots or strictures in the right internal jugular vein which may have formed from prior cannulation or placement of pacing devices. Left internal jugular vein cannulation is considered in some institutions to preserve the right side for future endomyocardial biopsies.

A pulmonary artery catheter should be used in most cardiac transplant recipients. Some institutions prefer to limit initial advancement of the catheter to 20 cm. The intent is to locate the catheter tip within the superior vena cava (SVC) so that it does not interfere with CPB cannulation and dissection of the native heart. It is also reasonable to advance the pulmonary artery catheter beyond the pulmonic valve to obtain baseline pressure measurements and then to withdraw it into SVC.

Induction and Intraoperative Management

Any inotropic support, IABP counterpulsation, or mechanical circulatory assistance present on the patient's arrival to the operating room should be continued uninterrupted until institution of CPB. The anesthesia team should be certain to have an

understanding of any hospital or ambulatory infusion pumps which arrive with the patient so that infusion rates can be adjusted, as necessary. Additional infusion pumps and vasoactive medications should be readily available prior to induction.

Prior to induction external defibrillator pads should be placed appropriately and proper monitor function should be confirmed. The sequence of invasive monitor placement is institution specific. However, strong consideration should be given to securing arterial pressure monitoring prior to induction of general anesthesia. Placement of central venous access after induction is reasonable in most cases.

Because of the nonelective nature of cardiac transplant procedures, rapid sequence induction may be necessary for airway protection. If a full stomach is not an overriding concern, a balanced anesthetic technique using titrated doses of midazolam, etomidate, and fentanyl followed by a volatile gas is preferred. Muscle relaxation can be facilitated with succinylcholine or rocuronium. Anesthetic induction in patients with poor ventricular function can be complicated by hemodynamic instability with cardiovascular collapse and presence of the surgical team at the time of induction is warranted.

While administering anesthetics and vasoactive medications in heart transplant patients, it is important to remain aware of the slow circulation time and onset of pharmacological effects. Maintenance of anesthesia can be achieved with a low inhaled concentration of volatile anesthetics such as isoflurane or sevoflurane supplemented with a benzodiazepine if potential awareness is a concern. Hemodynamic goals in the pre-CPB period include maintenance of normal to high preload, appropriate afterload to achieve the targeted blood pressure based on pre-induction values, and a higher heart rate to offset a compromised stroke volume.

In preparation for skin incision and sternotomy, additional fentanyl and isoflurane should be titrated to prevent hypertension and tachycardia in response to the new surgical stimulus. Muscle relaxants should be re-dosed to prevent reflexive movement during sternotomy. After sternotomy and pericardiectomy, an activated clotting time (ACT) of more than 400–450 s is achieved with 300–400 units/kg of heparin prior to aortic cannulation.

Transesophageal Echocardiography

Orthotopic heart transplant is a Society of Cardiovascular Anesthesiologists category 2 indication for intraoperative TEE. During the pre-CPB period, the TEE examination should devote special attention to the potential for intracardiac thrombus or mobile atheroma in aorta, global assessment of biventricular function to guide hemodynamic support, and examination of the tricuspid jet velocity to estimate pulmonary artery systolic pressure and identify preexisting pulmonary hypertension.

During weaning from CPB, the TEE examination is focused on assessment of cardiac chamber de-airing, integrity of surgical anastomosis (SVC, IVC, and pulmonary vein flow velocities), and identifying the cause of any hemodynamic instability such as right or left ventricular dysfunction. Should early graft dysfunction

occur, TEE can be essential in guiding placement of temporary mechanical support devices and confirming their correct function.

Surgical Techniques for Heart Transplantation

There are three distinct surgical techniques for orthotopic heart transplant: the bi-atrial approach, the bi-caval approach, and the total transplantation technique. The bi-atrial approach requires four anastomoses including left and right atrial cuffs and end-to-end pulmonary artery and aortic anastomoses.

The bi-caval approach employs five anastomoses including the left atrial cuff, individual end-to-end inferior and superior vena cava anastomoses, as well as pulmonary artery and aortic anastomoses. The popularity in contemporary practice for the bi-caval approach results from its association with preservation of a normal sinus rhythm conduction pattern and less tricuspid and mitral regurgitation.

The total transplantation technique is the most involved, requiring eight anastomoses include four pulmonary veins, inferior and superior vena cava, pulmonary artery, and aorta. This technique carries the disadvantage of longer graft ischemic times.

Weaning from CPB

CPB flow may be slowly decreased while monitoring arterial and central venous pressures and carefully observing biventricular function. The pulmonary artery catheter can be advanced after removal of SVC cannula. Once the patient has been successfully weaned from CPB and hemodynamic stability and adequate cardiac output have been confirmed, protamine is administered.

Following protamine, coagulation status can be assessed using clinical criteria or point-of-care testing. Depending on the patient's baseline coagulation status, blood volume, and duration of cardiopulmonary bypass platelets, fresh frozen plasma or cryoprecipitate may be needed. Finally, primary graft failure will manifest as low cardiac output, severe biventricular dysfunction, hypotension, and high PAP pressures. In the event of worsening heart function refractory to medical management, mechanical circulatory support may be necessary.

- *Rewarming* – Maintain volatile anesthetic administration via the CPB circuit and re-dose narcotics as necessity to maintain anesthetic state. Begin ventilation early during rewarming with tidal volumes of 6–8 ml/kg and PEEP 5–6 mmHg to prevent increase in PVR.
- *TEE* – Assist with de-airing of the pulmonary veins, LV apex, interventricular septum, left atrium, and appendage. Examine flow velocities through surgical anastomoses. Estimate ventricular and valvular functions, and rule-out intracardiac shunts.

- *Methylprednisolone* – A repeat dose is administered just prior to release of the aortic cross clamp.
- *Inotropic support* – Low-to-moderate dose epinephrine is commonly used to help overcome stunning during separation from CPB. In patients with residual pulmonary hypertension, dobutamine or milrinone in combination with iNO may be suitable adjuncts.
- *Rhythm control* – The denervated heart lacks normal autonomic control and baroreceptor mediated reflexes. Two p-waves may be apparent on the EKG if a bi-atrial anastomotic technique was used. Isoproterenol or dobutamine may be used for chronotropic support in transplanted hearts. The use of epicardial pacing is more predictable and reliable compared to isoproterenol and is the primary method of rate control immediately post-CPB (goal 90–110 eats/min).
- *Vasoplegia* – Vasopressin should be considered early if a vasoconstrictor is required as chronic preoperative vasodilator therapy may make alpha-agonists less effective. Vasopressin also lacks any effect on the pulmonary vasculature and will not exacerbate pulmonary hypertension or further impair compromised right ventricular systolic function.

Pulmonary Hypertension and Right Heart Failure

Approximately 20% of early deaths after cardiac transplantation are attributable to right ventricular failure. Early detection of right ventricular systolic dysfunction and aggressive intervention are essential if morbidity is to be minimized. The hallmarks of treatment are the same as for left ventricular systolic dysfunction – careful optimization of pre-load, afterload, contractility, and implementation of mechanical support before the hemodynamic state is critically compromised. Risk factors for right ventricular failure and treatment of right ventricular failure are noted in Tables 24.3 and 24.4 respectively.

Table 24.3 Risk factors for right ventricular failure

Hight PVR
Prolonged hypothermic cardioplegic time
Mechanical trauma during harvest
Prolonged warm ischemia during implantation
Air embolism of right coronary artery during reperfusion
Thin right ventricular free wall

Table 24.4 Treatment of right ventricular failure

Optimization of right ventricular preload
Inotropic support (milrinone, epinephrine)
Reduced afterload/pulmonary vasodilatation (nitric oxide, inhaled epoprostenol)
Improvement of coronary perfusion pressure
Avoidance of hypercarbia and acidosis
Avoidance of hypothermia, pain
Mechanical circulatory support (RVAD)

Nitric Oxide Therapy

Inhaled nitric oxide is a potent pulmonary vasodilator, which reduces pulmonary vascular resistance and augments blood flow to well-ventilated regions of the lung. It is rapidly bound by hemoglobin and thus has little or no effect on the systemic vascular resistance.

Nitric oxide may be administered via the inspiratory limb of anesthesia circuit. A monitoring line is attached 6 inches proximal to the circuit Y-piece to ensure correct concentration of the drug in the inhaled gas mixture. It is recommended to keep fresh gas flow greater than or equal to the total minute ventilation to ensure accurate dosing of the nitric oxide. The suggested starting dose to treat acute pulmonary hypertension is 80 ppm followed by a weaning protocol requiring a reduction of 20 ppm every 10 min until a dose of 20 ppm is reached.

Clinical Pearls

- Severe myocardial systolic dysfunction can result in dramatic hemodynamic instability with the induction of general anesthesia.
- A thorough understanding of the cardiovascular status is required prior to induction, and contingency plans should be clear and communicated to all team members in advance.
- Preparation for pharmacological therapy and emergent institution of mechanical circulatory support should be completed before induction of anesthesia.
- Mechanical support of left ventricular function may unmask or worsen right ventricular systolic dysfunction, especially in the setting of pulmonary hypertension.
- The post-CPB period following heart transplantation is focused on identifying early graft dysfunction, inotropic support when required, and treatment of coagulopathy.

Suggested Reading

1. Jessup M, Abraham WT, Casey DE. 2009. Focused update: failure in adults: a report of the American college of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines: developed in collaboration with the International Society for Heart and Lung Transplantation. Circulation. 2009;119(14):1977–2016.
2. Mehra MR, Canter CE, Hannan MM, Semigran MJ, Uber PA, Baran DA, Danziger-Isakov L, Kirklin JK, Kirk R, Kushwaha SS, Lund LH, Potena L, Ross HJ, Taylor DO, Verschuuren EA, Zuckermann A. International Society for Heart Lung Transplantation (ISHLT) Infectious Diseases Council, International Society for Heart Lung Transplantation (ISHLT) Pediatric Transplantation Council, International Society for Heart Lung Transplantation (ISHLT) Heart Failure and Transplantation Council. The 2016 International Society for Heart Lung Transplantation listing criteria for heart transplantation: a 10-year update. J Heart Lung Transplant. 2016;35(1):1.

3. John R, Liao K. Orthotopic heart transplantation. Oper Tech Thorac Cardiovasc Surg. 2010;15(2):138–46.
4. Ardehali A, Hughes K, Sadeghi A, Esmailian F, Marelli D, Moriguchi J, Hamilton MA, Kobashigawa J, Laks H. Inhaled nitric oxide for pulmonary hypertension after heart transplantation. Transplantation. 2001;72(4):638–41.
5. Nicoara A, Ruffin D, Cooter M, Patel CB, Thompson A, Schroder JN, Daneshmand MA, Hernandez AF, Rogers JG, Podgoreanu MV, Swaminathan M, Kretzer A, Stafford-Smith M, Milano CA, Bartz RR. Primary graft dysfunction after heart transplantation: Incidence, trends, and associated risk factors. Am J Transplant. 2018;18(6):1461–70.
6. Semigran MJ, Cockrill BA, Kacmarek R, Thompson BT, Zapol WM, Dec GW, Fifer MA. Hemodynamic effects of inhaled nitric oxide in heart failure. J Am Coll Cardiol. 1994;24(4):982–8.

Anesthetic Management for Pulmonary Thromboendarterectomy

25

Shonali Pawar, Abdalhai Alshoubi, and Edo Ginsburg

Pressure makes diamonds!

—General George S. Patton

Key Points

- Chronic thromboembolic pulmonary hypertension
- Pulmonary thromboendarterectomy
- Preoperative assessment
- Line placement and monitoring
- Induction and intraoperative management
- Transesophageal echocardiography
- Deep hypothermic circulatory arrest
- Postoperative complications

Introduction

Pulmonary thromboendarterectomy is being applied with increasing frequency to patients suffering from symptomatic chronic thromboembolic pulmonary hypertension (CTEPH). CTEPH is defined as a persistent mean pulmonary arterial pressure greater than 25 mmHg for 6 months after an inciting embolic event. CTEPH is progressive in nature and carries a poor prognosis due to the limited efficacy of

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medical therapy. Surgical intervention by pulmonary thromboendarterectomy (PTE) or lung transplantation are the only effective cures. However, the scarcity of available organs limits the viability of transplantation as a therapeutic option and the mortality rate of untreated CTEPH approaches 90% after 3 years. Therefore, PTE is becoming the therapy of choice for patients with CTEPH and a suitable distribution of pulmonary artery occlusive disease on imaging studies.

Chronic Thromboembolic Pulmonary Hypertension (CTEPH)

Patients may present initially with a wide range of symptoms including exercise intolerance, fatigue, dyspnea, or signs of right ventricular dysfunction (jugular vein distention, hepatomegaly, splenomegaly, edema, etc.). Alternatively, patients may already be under treatment for a known deep venous thrombosis or pulmonary embolism and be diagnosed with early CTEPH before symptoms develop.

Medical Management of CTEPH

Medical therapy may serve to stabilize a patient's hemodynamic state and symptomatology until surgery can be performed, recognizing surgery is the only curative route. For patients with inoperable disease, management should be targeted to reducing the clot burden and optimizing hemodynamic parameters.

Many patients will be determined to carry a hypercoagulability state and will require lifelong anticoagulation with oral anticoagulants (coumadin, dabigatran, rivaroxaban). Vascular remodeling can be achieved through the use of prostacyclin analogs (epoprostenol, treprostinil, iloprost) or endothelin antagonists (bosentan). Pulmonary vasoconstriction is reduced with phosphodiesterase inhibitors (sildenafil, tadalafil, milrinone).

In addition to the CTEPH-specific therapies above, right ventricular failure can be treated with standard therapies for heart failure, including fluid restriction, diuretics, and inotropes in advanced cases. Indications for pulmonary thromboendarterectomy (PTE) are noted in Table 25.1.

Table 25.1 Indications for pulmonary thromboendarterectomy (PTE)

Hemodynamic compromise
Respiratory dysfunction
Disease prevention (prophylaxis)

Pulmonary Thromboendarterectomy (PTE)

Surgical endarterectomy is performed via median sternotomy. A nearly bloodless field is required for the precise work of competing the endarterectomy into segmental and subsegmental vessels. The continued presence of bronchial arterial blood flow creates some filling of the pulmonary arterial vessels even during CPB and cardiac arrest. Therefore, periods of circulatory arrest are necessary to provide optimal surgical conditions.

CPB is initiated after an activated clotting time >450 s is achieved. Cooling to a core temperature (usually measured in the bladder) of 20 °C should be performed gradually (60–90 min) to allow uniform cooling of the entire body. A left ventricular vent and PA vent are placed during this period to prevent cardiac chamber distension and subendocardial ischemia (Fig. 25.1).

Once a core temperature of 20 °C is reached and an isoelectric EEG is achieved, the aorta is cross-clamped and cold cardioplegia is administered. A cooling jacket is wrapped around the heart. A topical cooling system for the head as shown in (Fig. 25.2) may be used in some centers to assist with maintaining brain temperature during circulatory arrest.

The majority of the PTE can be achieved during standard CPB. Once the bulk of the proximal disease has been resected, the surgeon will request circulatory arrest, the blood will be allowed to drain passively into the venous reservoir of the CPB circuit, and arterial inflow to the patient will cease. During this period of complete circulatory arrest, the surgeon will complete the endarterectomy of the small distal pulmonary vessels. In between the repair of each pulmonary artery bed, total circulatory arrest may be interrupted by periods of reperfusion maintaining the CPB temperature at 20 °C.

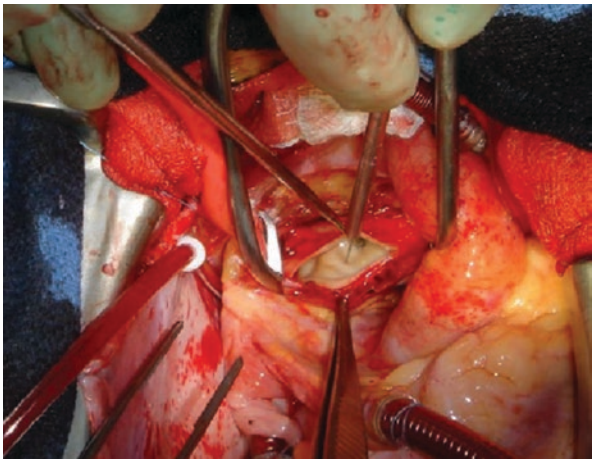

Fig. 25.1 Image of right pulmonary artery is opened after mobilization of SVC and aorta with self-retaining retractors. Swan Ganz catheter is seen in PA

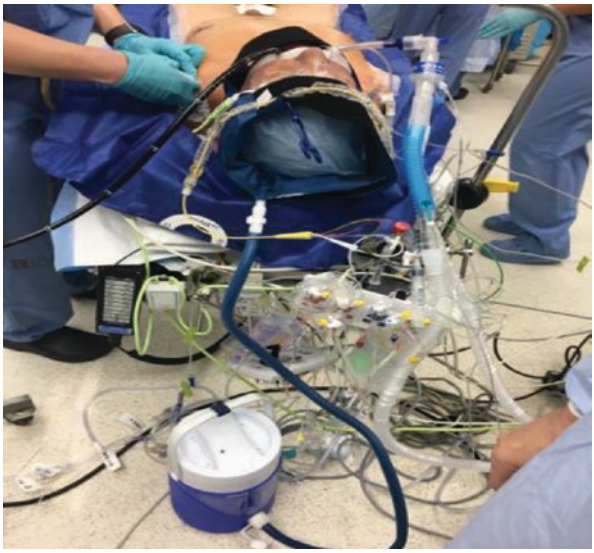

Fig. 25.2 Image of head wrap system (polar care)

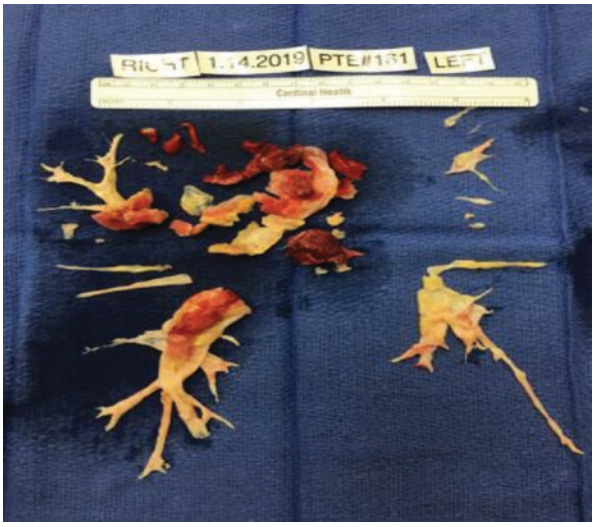

Fig. 25.3 Image of Type I disease with clot in main pulmonary arteries

Jamieson classified pulmonary thromboembolism into four types. Type I (12%) involves a major vessel clot that is visible on opening the pulmonary artery (Fig. 25.3). Type II (38%) involves main, lobar, or segmental vessels (Fig. 25.4). Type III (39%) is disease confined to segmental and subsegmental branches (Fig. 25.5). Type IV is small vessel disease and is inoperable.

Once the endarterectomy has been completed the pulmonary arteries are closed, CPB is reinitiated, and rewarming is begun.

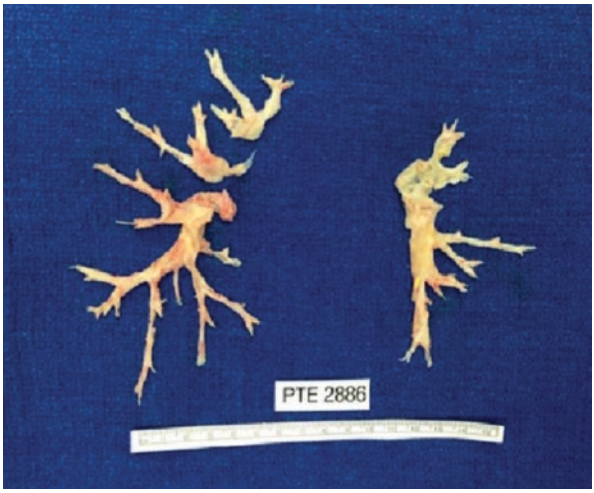

Fig. 25.4 Image of Type II disease with lobar branches are involved

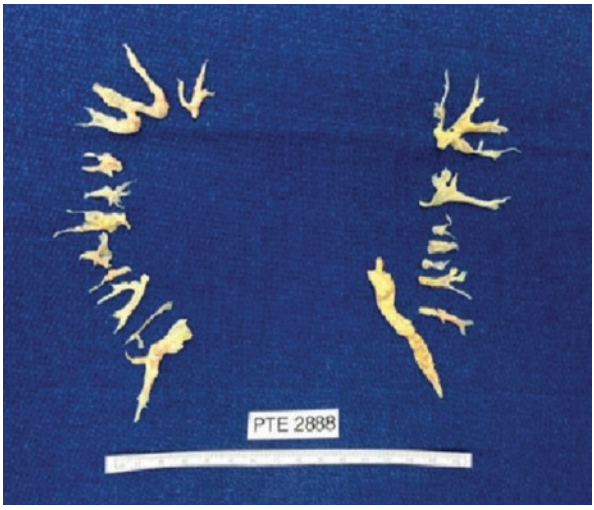

Fig. 25.5 Image of Type III disease with segmental arteries involved

Preoperative Assessment

Most patients presenting for PTE will have undergone extensive workups with complete characterization of their cardiovascular state. Some studies may have been performed at outside medical centers, but the anesthesia team should be sure to obtain all of the results available. At a minimum, it should be expected that each of these patients will have a recent transthoracic echocardiogram, right and left heart catheterization with calculation of pulmonary vascular resistance (PVR) and cardiac output, and characterization of their pulmonary arterial anatomy with chest CT and pulmonary angiogram.

All preoperative studies should be reviewed including any angiograms available to determine if clot burden is affecting the main pulmonary artery. On the right heart catheterization an increase in right ventricular end-diastolic pressures >14 mmHg, severe tricuspid regurgitation, mean PA pressure > 50 mmHg, and PVR > 600 dyne/s/cm^{-5} are signs of severe disease. In such instances, inotropic support may be initiated with low-dose epinephrine prior to induction of general anesthesia.

Disease Severity and Prognosis

A measured pre-operation PVR >1100 dyne/s/cm^2 is associated with a mortality of 41% compared with a mortality of 6% if PVR is <1100 dyne/s/cm^2. The mortality rate is 10.8% for PA systolic pressures >100 mmHg, compared to 4.2% if PA systolic pressure <100 mmHg.

Line Placement and Monitoring

Standard monitoring for PTE is similar to other open-heart procedures – ECG, non-invasive blood pressure, and pulse oximetry are all measured at baseline.

An arterial line should be placed prior to induction of general anesthesia because of the potential for cardiovascular collapse with the loss of sympathetic tone and positive pressure ventilation. Strong consideration should be given to placing a brachial arterial line due to the disparity between radial arterial pressure and central pressure measurements following deep hypothermic circulatory arrest. If questions persist regarding the accuracy of blood pressure measurements after circulatory arrest, a low threshold should be maintained for placement of a femoral arterial catheter.

A central venous introducer and pulmonary artery catheter are used for all cases and are usually placed after general endotracheal anesthesia is established. Passage of the pulmonary artery catheter through a dilated and hypokinetic right ventricle may be challenging. Repeated attempts can result in significant arrhythmias which may be poorly tolerated in these patients. If any difficulty is experienced in passing the pulmonary artery catheter, it is prudent to position it in the SVC until after sternotomy so that CPB can be quickly initiated if hemodynamic collapse should occur.

Transesophageal echocardiography is used to guide pulmonary artery catheter positioning and to monitor right ventricular function. Cerebral oximetry is helpful, particularly during the induction of anesthesia, and provides an ongoing assessment of the balance between cardiac output and cerebral oxygen demand. Electroencephalographic monitoring is employed universally to ensure that an isoelectric EEG is achieved prior to deep hypothermic circulatory arrest.

Because of the potential for wide temperature gradients during cooling and rewarming, monitoring should occur at several sites simultaneously – core temp (usually bladder), blood temperature (PA catheter), and brain temperature (nasopharyngeal).

Induction and Intraoperative Management

Sedation for PTE patients should be used cautiously and individualized carefully. Anxiety and pain can increase PVR on the one hand. On the other, excessive sedation can cause hypercarbia or hypoxia resulting in acute increases in PVR.

Many PTE patients will present with a hypertrophied and dilated right ventricle and a relatively fixed PVR. Vasodilators may demonstrate little effect on the pulmonary circulation because of the mechanical obstruction of the pulmonary arterial tree. Vasodilators may also critically reduce systemic vascular resistance compromising right ventricular coronary perfusion causing acute right ventricular ischemia and failure.

Gentle induction of general anesthesia that avoids arterial hypotension is the overriding principle in patients with severe pulmonary hypertension. For patients with severely elevated PVR or poor right ventricular systolic function a low-dose epinephrine infusion (2–4 mcg/min) is started via a peripheral IV prior to induction. The objective is to provide right ventricular support and maintenance of systemic arterial pressure. Vasopressors such as phenylephrine or vasopressin may be added as needed.

Administration of ketamine or nitrous oxide are not recommended as both are associated with an increase in pulmonary artery pressure (PAP) and pulmonary vascular resistance (PVR). Histamine releasing medications (atracurium, mivacurium) should be avoided because of their potential effect on PVR.

Frequent monitoring of arterial blood gases is recommended. Hypoxia, acidosis, and hypercarbia must be avoided because of their effects on PVR. Continuation of pharmacological pulmonary hypertension treatment through the pre-CPB period is recommended. Intraoperative strategies to avoid an increase in PVR are noted in Table 25.2, while supportive therapy for increased PVR are listed in Table 25.3.

Table 25.2 Intraoperative strategies to avoid an increase in PVR

Oxygenation with inspiratory FiO_2 0.6–1.0
Moderate hyperventilation (goal: PaCO2 30–35 mmHg)
Avoidance of metabolic acidosis (pH > 7.4)
Recruitment maneuver to avoid ventilation–perfusion mismatch
Low-tidal-volume ventilation to avoid over inflation of alveoli (goal: 6–8 mL/kg ideal body weight)

Table 25.3 Supportive therapy for increased PVR

(A) Intravenous vasodilators
Milrinone 50 μg/kg BW bolus, followed by 0.5–0.75 μg/kg BW/min continuously
Dobutamine 2–5 μg/kg BW/min continuously
Prostacyclin 4–10 ng/kg BW/min continuously
Nitroprusside 0.2–0.3 μg/kg BW/min continuously
Nitroglycerine 2–10 μg/kg BW/min continuously
(B) Inhaled vasodilators
Iloprost 5–10 μg for 10–15 min (by ultrasonic nebulizer)
Nitrous oxide 0.5–20 ppm continuously

Transesophageal Echocardiogram (TEE)

A complete TEE examination is required for patients undergoing PTE. At baseline, right and left ventricular function should be assessed, and abnormalities in chamber size and thickness documented. Any tricuspid regurgitation should be quantified, and an estimate of PA pressure should be made from the regurgitant jet velocity.

A patent foramen ovale is present in 25–35% of PTE patients. Detection and correction of this anatomical variant is important in this population. A patent foramen ovale may predispose to paradoxical embolus in the presence of severe pulmonary hypertension and elevated central venous pressures. If examination by color flow Doppler is inconclusive, contrast echocardiography should be performed (PEEP 30 cm H2O is applied for 10 s or as tolerated, then agitated blood or 5% albumin is injected into the SVC as the PEEP is released).

The TEE may also help to guide PA catheter placement or may detect right atrial or proximal PA thrombus. If visible thrombus is present in the proximal pulmonary artery, the PA catheter is advanced only into the SVC (about 20 cm) to avoid dislodging the thrombus.

Deep Hypothermic Circulatory Arrest (DHCA)

Several aspects of managing deep hypothermic circulatory arrest for PTE distinguish this technique from standard CPB.

Antifibrinolytics are not used during CPB for PTE patients because this population is inherently hypercoagulable. Just prior to complete circulatory arrest, several drugs are administered in an effort to reduce ischemia and reperfusion injuries to the central nervous system. These may vary by institution but usually include methylprednisolone (1 g), magnesium sulphate (2 g), and lidocaine (100 mg). In addition, intravenous anesthetic agents may be provided as a bolus (propofol 2.5 mg/kg) in an effort to reduce the cerebral metabolic requirement for oxygen, although the utility of this practice in the setting of an isoelectric EEG is questionable.

Criteria for initiating complete circulatory arrest include an isoelectric EEG signal and a sustained core temperature of $\leq$18 °C. During PTE the surgical resection is planned so as to limit the duration of any single episode of circulatory arrest to 20 min. Flow is then resumed for 10 min using the CPB circuit before any additional required periods of circulatory arrest.

Rewarming and Separation from CPB

The rewarming process following DHCA must be achieved gradually. The temperature of the inflow from the CPB circuit should not exceed 37.5 °C at any time. Hyperthermia following a period of ischemia may exacerbate any injury sustained to the central nervous system. Once the core temperature is greater than 32 °C, the

heart may be defibrillated if necessary. An estimate of the likely cardiovascular function post-CPB should be made once a regular rhythm has been established and ventricular ejection has begun.

If the procedure was successful in significantly reducing the PVR, immediate changes may be evident on the TEE. The right ventricular systolic function may be improved, the chamber size may be reduced, and less flattening of interventricular septum may be seen.

If residual pulmonary hypertension is evident, inotropic support with epinephrine together with milrinone and inhaled NO may be required. Right atrial pacing at 90–100 beats/min should be implemented if a relative bradycardia is present. As with any procedure using CPB, adequate de-airing of the cardiac chambers, normothermia (37 °C), and a reasonable hemoglobin (≥8 g/dL) and acid–base balance on arterial blood gas measurement are all required before terminating circulatory support.

Finally, assessing the endotracheal tube before separation from CPB for frothy sputum (reperfusion pulmonary edema) or bleeding (pulmonary hemorrhage) is prudent and may alert the anesthesia team to impending complications (see below).

Postoperative Complications

Pulmonary Hemorrhage and reperfusion pulmonary edema are two of the most serious complications that may occur in the immediate post-op period following PTE. Both can result in severely impaired ventilation and oxygenation, leading in turn to hemodynamic instability.

If bleeding into the airway is encountered, the most important immediate objective is to determine the source. If the main or segmental bronchus which is the origin of the bleeding can be identified, implementing a lung isolation strategy with a branchial blocker may preserve adequate ventilation via the uninvolved lung parenchyma. Repair of the source of the bleeding may then achieved by direct surgical exploration or embolization in the interventional radiology suite.

Reperfusion pulmonary edema is thought to occur in some patients when the distal pulmonary arterial tree is acutely exposed to a state of high flow or pressure after chronic protection by proximal thrombus. Treatment is largely supportive. Increased levels of PEEP can be effective, and FiO_2 is increased as needed to maintain arterial oxygenation. Avoidance of a high cardiac output state resulting from unnecessary inotropic infusions is appropriate. If gas exchange becomes critically impaired, a short period of support with venovenous extracorporeal membrane oxygenation (VV ECMO) may be required.

Clinical Pearls

- A detailed understanding of each patient's right ventricular function and pulmonary vascular resistance is required prior to induction of general anesthesia.

- Significant right ventricular systolic dysfunction in the presence of high PVR may require low-dose epinephrine infusion via peripheral IV at the time of anesthetic induction.
- Pre-CPB TEE examination should be performed to identify the presence of a PFO or structural tricuspid valve disease.
- Iatrogenic high cardiac output states should be avoided post-CPB to minimize the risk of reperfusion pulmonary edema.
- Nonetheless, transient inotropic support post-CPB is frequently required for patients with severe right ventricular dysfunction at baseline.

Further Reading

1. Ramakrishna G, Sprung J, Ravi BS, Chandrasekaran K, McGoon MD. Impact of pulmonary hypertension on the outcomes of noncardiac surgery: predictors of perioperative morbidity and mortality. J Am Coll Cardiol. 2005;45(10):1691–9.
2. Hartz RS, Byrne JG, Levitsky S, Park J, Rich S. Predictors of mortality in pulmonary thromboendarterectomy. Ann Thorac Surg. 1996;62:1255–9.
3. Fedullo P, Kerr KM, Kim NH, Auger WR. Chronic thromboembolic pulmonary hypertension. Am J Respir Crit Care Med. 2011;183(12):1605–13.
4. Kaw R, Pasupuleti V, Deshpande A, Hamieh T, Walker E, Minai OA. Pulmonary hypertension: an important predictor of outcomes in patients undergoing non-cardiac surgery. Respir Med. 2011;105(4):619–24.
5. Goldhaber SZ, Elliott CG. Acute pulmonary embolism: part I. Epidemiology, pathophysiology, and diagnosis. Circulation. 2003;108:2726.
6. Piazza G, Goldhaber SZ. Chronic thromboembolic pulmonary hypertension. N Engl J Med. 2011;364:351–60.

Part VI

The Complications of Cardiac Surgery

The Complications of Cardiac Surgery

26

Asma Khan and Amany Gorgy

See, and then reason and compare and control.
But see first.

—Sir William Osler

Introduction

An estimated 2 million cardiac surgeries are performed each year around the world. While working in the "heart room," along with understanding of cardiopulmonary physiology and pathophysiology, one must also have a clear understanding of complications that can occur during each step of the procedure since complications are associated with increased morbidity and mortality.

Invasive Monitoring Complications

Routine use of invasive monitoring and transesophageal echocardiogram (TEE) during cardiac surgery, although not common, is associated iatrogenic complications. Arterial monitoring can be complicated by limb ischemia and nerve injury. Insertion of central venous catheters can cause pneumothorax, jugular vein laceration arterial cannulation, blood stream infection, and air embolism. Pulmonary artery catheters can induce arrhythmias or can cause embolism, pulmonary artery rupture, and tricuspid valve injury. TEE can cause oropharyngeal injury, dental damage, and esophageal perforation. Awareness of these complications is key for prevention.

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Neurological Complications

Postoperative neurological complications can be categorized as follows:

- Type—I: Stroke, stupor, or coma; and
- Type—II: Seizure, memory, or intellectual function deficit

Overall, the incidence of stroke is 4.6%, with the greatest incidence occurring in patients who underwent valve surgery. Risk factors for perioperative stroke include atrial fibrillation, intracranial cerebral atherosclerosis, aortic atherosclerosis, and extracranial carotid disease. Incidence of stroke is higher during on pump procedures with hypothermic circulatory arrest. Preoperative atrial fibrillation is associated with high incidence of both early and late stroke. Aggressive use of anticoagulation and cardioversion has significantly reduced the incidence of late stroke related to new-onset atrial fibrillation in the postoperative period. Evidence of prophylactic left atrial appendage ligation during routine cardiac surgery in patients with AF and without AF to reduce the occurrence of early postoperative atrial fibrillation is still lacking. Intraoperative monitoring using EEG, transcranial Doppler and noninvasive near infrared spectrophotometry (NIRS) help early diagnosis of cerebral ischemia and embolic events. For patients at higher risk for aortic atherosclerosis, TEE, and epiaortic surface scanning are superior to the surgeon's manual palpation of the aorta prior to cannulation. Other sources of emboli would be left atrial or ventricular thrombus, microembolism of aggregates formed by fibrin, platelets and granulocytes, gaseous emboli (micro air bubbles), and fat aggregates.

Strokes could be also caused by intraoperative cerebral hypoperfusion especially in the watershed zones in the brain. A relatively uncommon reason for decreased cerebral perfusion would be cerebral edema and venous congestion, resulting from venous cannula malposition and SVC occlusion.

Intraoperative hyperglycemia is associated with poor neurocognitive outcome in cardiac surgery. Older age female gender and patients with lower education level and baseline cognitive scores is associated with higher incidence of postoperative delirium (POD) or postoperative cognitive dysfunction (POCD). Obviously, significant hypoglycemia may be a cause for poor neurologic outcome. Tapia's syndrome is a rare complication of cardiac surgery [1]. It presents as hoarseness of voice, difficulty in tongue movement and dysphagia. The syndrome occurs due to compression of the Vagus and Hypoglossal nerves around the first cervical vertebra from neck positioning during sternotomy, endotracheal intubation or nasogastric tube placement. Hoarseness of voice after open heart surgery could be encountered in the postoperative period due to recurrent laryngeal nerve (RLN) injury intraoperatively. The longer intra-thoracic course of the left RLN makes it more vulnerable to intraoperative injury as compared to right RLN. Direct injury to vocal cords or compression of RLN in tracheoesophageal groove by an inappropriately sized endotracheal tube cuff, sternal traction on subclavian arteries, and traction from manipulation of heart during surgery

or central venous catheterization may result in RLN injury. Phrenic nerve injury leading to diaphragmatic dysfunction can worsen pulmonary function mechanics in the postoperative period. Various insults like stretching during pericardial manipulation of the heart, pericardial ice slush use resulting in cold injury or direct injury during harvesting of internal mammary artery can cause intraoperative phrenic nerve injury. Other peripheral nerve injuries that have been reported following cardiac surgery include ulnar neuropathies and brachial plexus injury [2]. Other injuries include saphenous nerve injury during saphenous venous harvesting for CABG and peroneal nerve injuries from fibular head compression and malpositioning for several hours. Luckily, most of these injuries are self-limiting and more than half of the patients regain full sensory and motor functions within a year.

Visual Complications

Incidence of postoperative visual loss is 8.64 per 10,000 patients. Eye complications could result from relatively minor corneal abrasions to more catastrophic events like anterior ischemic optic neuropathy, retinal vascular occlusion either venous or arterial, cortical blindness, and pituitary apoplexy. Associated risk factors could be attributed to hypertension, dyslipidemia, atherosclerotic disease–affecting carotids, low hematocrit, hypotension, high dose of inotropic support, prolonged CPB, and large volume of fluid resuscitation.

Cardiovascular Complications

The majority of cardiovascular complications following cardiac surgery can be divided into a few broad categories. These categories include arrhythmias (slow and fast), ventricular dysfunction (left, right or biventricular), blood pressure dysregulation including hypotension and vasoplegia syndrome, and pericardial complications including pericardiotomy syndrome and cardiac tamponade.

Arrhythmias

Arrhythmias complicate cardiac surgery frequently. Atrial fibrillation is the most common arrhythmia in the postoperative period. The restoration of normal sinus rhythm is usually accomplished chemically. If atrial fibrillation persists for longer than 24 hours, anticoagulation is indicated initially with heparin.

Direct surgical injury or resultant edema of the conduction system may lead to atrioventricular blocks or bradyarrhythmia. Supraventricular tachyarrhythmias are less common but not rare. Ventricular tachycardia or fibrillation signify major myocardial ischemia that need to be addressed immediately by defibrillation and possibly going back on CPB to control the bleeding or revision of the grafts.

Ventricular Dysfunction

Ventricular dysfunction requiring inotropic support is not uncommon. Due to the unique anatomy of the right ventricle, it is subject to poor myocardial protection during CPB. RV failure after bypass needs immediate attention as RV dysfunction and dilation will lead to worsening of tricuspid regurgitation and bulging of the interventricular septum toward the left ventricle with resultant decreased LV compliance and preload which leads to further myocardial ischemia and worsening of the RV dysfunction. Optimization of volume status, initiation of inotropic support, and possible pulmonary vasodilators are cornerstones in breaking the vicious cycle of RV failure.

Blood Pressure Dysregulation

Hypertension

Immediate *post cardiopulmonary bypass hypertension* could be due to light anesthesia, pain, or vasoconstriction due to stress hormones. Hypertension leads to increases myocardial oxygen demand and could compromise the suture line. With the expected hemodynamic fluctuations in the immediate post CPB period, it is prudent to avoid long-acting antihypertensives.

Hypotension and Vasoplegic Syndrome

Vasoplegic syndrome is seen in the immediate postoperative period. It presents as MAP <60 mmHg, high cardiac index (>3.5 L/min/m^2), and adequate cardiac chamber–filling pressures. The exact etiology is unclear, but it has been associated with activation of inflammatory mediators (C3a, Interleukin-6, or TNF) in response to the synthetic membrane used in cardiopulmonary bypass. It could also be related to vasodilatory effect of increased levels of bradykinins or activation of nitric oxide synthase. Preoperative use of angiotensin-converting enzyme inhibitors (ACEIs), aldosterone-receptor blockers (ARBs), and calcium channel blockers are associated risk factors for development of postoperative vasoplegia. Other contributory drugs mentioned are heparin, beta-blockers, and amiodarone.

Postpericardiotomy Syndrome

Postpericardiotomy syndrome is an autoimmune phenomenon seen during the first week and up to one month after surgery. It results from formation of antibodies in patients who underwent open heart surgery. Patients present with fever, malaise, pericardial rub, and pleuritic chest pain. Patients may also develop pleural or pericardial effusion and may rarely also present with difficulty in swallowing.

Cardiac Tamponade

Cardiac tamponade following cardiac surgery requires a high level of suspicion as the symptoms and signs could be masked by other post CPB events like heart failure. Even transthoracic echocardiography might not aid the diagnosis due to surgical dressing and the altered anatomy of the pericardium after surgery. Cardiac tamponade may sometimes be a result of pulling the temporary pacing wires placed epicardially at the time of surgery.

Coronary Artery Bypass Grafting (CABG)

Postoperative Myocardial Ischemia or Early Graft Occlusion

Graft failure or spasm, incomplete revascularization, or complete occlusion of a native coronary artery could result in ischemia after surgery and could result in high (68%) postoperative in hospital mortality. Thrombosis of a graft will result in early graft occlusion in the postoperative period. Graft occlusion could also be related to technical issues related to the quality of the conduit, size, and quality of the target vessel and the flow in the graft. Hypercoagulability can also be problematic.

Low Cardiac Output Syndrome (*LCOS)*

LCOS is defined as the use of inotropic support or mechanical device (IABP) for more than 30 minutes to maintain cardiac index of 2.2 L/min/m^2 or a SBP of 90 mmHg or above. LCOS is the largest cause of mortality in CABG patients. Factors responsible for LCOS include poor ventricular contractility due to myocardial ischemia, poor myocardial protection, hypovolemia, hypoxemia, cardiac tamponade, electrolyte abnormalities, and arrhythmias.

Inotropic support and phosphodiesterase inhibitors are warranted in patients with persistent poor left ventricular function (cardiac index less than 2.0 L/min/m^2) despite optimization of heart rate, rhythm, preload, and afterload.

If pharmacotherapy fails to improve cardiac output, mechanical devices like intra-aortic balloon pump, ventricular assist device (such as the Impella pump), and extracorporeal membrane oxygenator can be used to assist weaning from bypass.

Valvular Surgery

Mitral Valve Surgery

Systolic anterior motion of the mitral valve following repair is always a challenging management dilemma. Sometimes, a period of reperfusion and the restoration of

Table 26.1 Other surgery specific complications

Type of surgery	Surgery-specific complications
CABG	Postoperative MI, graft occlusion, low cardiac output syndrome, saphenous vein donor site infections
Mitral valve replacement surgery	Atrial or ventricular rupture, injury to circumflex artery, paravalvular leak
Mitral valve repair surgery	Residual prolapse, restricted valve opening, SAM, injury to circumflex artery, cusp perforation, aorto-ventricular fistula
Tricuspid valve surgery	Heart block
Aortic root repair	Pseudoaneurysms, graft occlusion, endoleaks, coronary occlusion

sinus rhythm is sufficient to eliminate the problem, but at times it may be necessary to re-repair or replace the valve.

Injury to the circumflex artery might happen due to its proximity to the annulus or the presence of an aberrant right coronary artery. Direct passage of annuloplasty sutures through the vessel, plication of valve, or oversized ring could result in distortion of circumflex artery. Intraoperatively, injury of the circumflex artery could result in difficulty in weaning from CPB. A lateral wall motion abnormality seen on TEE should arouse suspicion of a circumflex coronary artery injury.

A dreaded complication of mitral valve surgery is A–V groove disruption. This is more commonly seen in patients with severe mitral annular calcification in which the calcium was debrided too aggressively or in whom excessive lifting of the heart was performed (Table 26.1).

Aortic Root Surgery/Aortic Dissection Repair

Residual prolapse after corrective surgery is encountered more commonly in chronically dilated aortic root with stretched aortic cusps. It could result from the valve corrective procedure itself or was preexisting and was under-corrected or not recognized during surgery. Other complications include, rarely, restricted valve opening resulting in increased pressure gradient across the valve, perforation of cusps or commissural fenestration, and aortic cusp retraction due to sutures applied during left main coronary artery reimplantation resulting in severe aortic insufficiency. Subcommissural annuloplasty can lead to fistula formation at the level of the anterior commissure between the aorta and the right ventricle. Aortic root repairs could be complicated by pseudoaneurysms.

Tricuspid Valve Surgery

Although all valve surgeries may be complicated by the need for permanent pacemaker placement in the postoperative period, tricuspid valve surgery presents the greatest risk since the A–V node sits very close to the anteroseptal commissural area. Whereas tricuspid valve repairs with an annuloplasty ring typically avoid

this area, this is not the case with tricuspid valve replacement. Permanent epicardial pacemaker leads should be placed in all patients undergoing tricuspid valve replacement since a transvenous system is not advisable. A transvenous lead would have to cross the prosthetic valve and this would adversely affect its durability.

Pulmonary Complications

Median sternotomy and thoracotomy result in altered pulmonary function in the postoperative period most often due to limitation by pain. Decreased FVC, FEV1 (>50%), peak expiratory flow rate, and maximum voluntary ventilation result in atelectasis, impaired oxygenation, and increased incidence of postoperative pneumonia. Other pulmonary complications include pulmonary edema, pneumothorax, and diaphragmatic dysfunction due to phrenic nerve injury. Acute lung injury (ALI) is a common pulmonary complication following cardiac surgery. ALI is discussed more in Chap. 16, under Transfusion-Related Acute Lung Injury (TRALI) and pulmonary embolism. Postoperative pneumonia typically seen with active smokers may be lethal.

Gastrointestinal Complications

Gastrointestinal complications are not infrequent following cardiac surgery. These include mesenteric ischemia, upper or lower GI bleed, acute pancreatitis, acute cholecystitis, paralytic ileus, and liver failure. Increased risks of GI complications are associated with preexisting COPD, steroid use, low cardiac output state, peripheral vascular disease, chronic renal insufficiency, peptic ulcer disease, prolonged CPB time, blood transfusion, and smoking. Mortality associated with these perioperative GI complications range from 11% to 67%. Hence, making early diagnosis and treatment of these complications essential.

Renal Complications

Acute tubular necrosis has been attributed as the most common cause of acute kidney injury (AKI) after cardiac surgery. Thirty-day mortality in patients with perioperative AKI requiring dialysis is estimated to be 64% as compared to 4% in patients without renal failure.

Female gender, preexisting diabetes, peripheral vascular disease, COPD, renal insufficiency, congestive heart failure, and left ventricular ejection fraction <35% have been attributed as risk factors for development of perioperative renal failure. Intraoperative hypothermia, renal artery vasoconstriction, atheroembolism, and non-pulsatile blood flow on bypass have been identified as intraoperative risk factors for development of perioperative renal failure. Use

of synthetic colloids may also be associated with worsening of renal function in perioperative period. Renal protective strategies include off pump surgery, short cross-clamp/bypass time, and hemodilution. *N*-acetylcysteine, low-dose dopamine, and fenoldopam have limited evidence for use agents to limit renal dysfunction.

Hematological Dysfunction

Cardiopulmonary bypass and its sequelae such as hypothermia and hemodilution result in coagulopathy and thrombocytopenia. Inadequate reversal of heparinization can also be a factor. Significant anemia or bleeding require transfusion of blood products that can lead to acute transfusion reaction, fluid overload, TRALI, and transfusion-related infection. PRBC transfusion has been correlated with graft failure in patients undergoing CABG. More detailed discussion about transfusion of blood products can be found in Chap. 15.

Infectious Complications

Incidence of infection following cardiac surgery is less than 5%. Surgical site infections and sternal wound infections, respiratory tract infection, catheter-related bloodstream infection (CRBSI) are the most common sites. Risk factors for infection include diabetes, end stage renal disease and smoking. In modern times, risk factor modification, adherence to a prophylactic antibiotic protocol, and strict blood glucose control in the perioperative period can reduce the incidence postoperative infections. More detailed discussion about prophylactic antibiotic can be found in Chap. 8.

Complications Related to Cannulation During Cardiac Surgery

Although not related to the anesthesia management, complications related to surgical arterial and/or venous cannulation may have a dramatic impact on the procedure being performed and its outcome. Aortic cannulation should be performed with the blood pressure reduced so that aortic wall tension is diminished. Aortic dissection, the most serious complication of cannulation, requires the OR team to react quickly in order to establish true lumen blood flow and avoid malperfusion syndrome. The surgeon must also be vigilant in eliminating any air in the circuit. Femoral arterial cannulation may result in limb ischemia.

In certain situations, particularly related to congenital defects (e.g., autism spectrum disorder), air entering the right side of the heart during venous cannulation may lead to air embolism and stroke.

Lastly, cannulation of a calcified aorta may be lethal if plaque is dislodged into the circulation. Similarly applying an aortic cross clamp to a calcified aorta carries equal

risk. Palpation alone is not sensitive enough to identify proper areas for cannulation and cross-clamping. Intraoperative epiaortic has been shown to be a valuable tool in cardiac surgery for the proper management of a calcified aorta.

Complications Related to CPB

Cardiopulmonary bypass may lead to a systemic inflammatory response, multiple organ injury as well as coagulopathy. More detailed discussion of these mechanisms can be found in Chap. 16.

Further Reading

1. Alston RP, Myles PS, Ranucci M. In: Oxford Textbook of Cardiothoracic Anaesthesia. Oxford, United Kingdom: Oxford University Press; 2015.
2. Kaplan JA, editor. Kaplan's cardiac anesthesia 7th Edition: in cardiac and noncardiac surgery. Philadelphia: Elsevier; 2017.

Part VII

Anesthetic Management for Cath Lab Procedures

Anesthesia for Cardioversion and Cardiac Ablation Procedures

27

John E. Safaryn, Aaron Rasmussen, and Grace Dippo

The heart has its reasons which reason knows not.

—Blaise Pascal

Introduction

Anesthesia for electrophysiology (EP) procedures has many complex challenges for the anesthesia provider. The number of EP cases requiring an anesthesia provider is increasing, and this trend is likely to continue. Over 15 million patients in the United States are living with a cardiac arrhythmia. Experts predict that the prevalence of atrial fibrillation will increase threefold over the next 30 years, an exponential growth pattern that will require more EP procedures. Anesthesia providers need to be familiar with the procedures and anesthetic requirements of the EP laboratory. Historically, EP suites were used mostly for diagnostic procedures, but now, device implantations, cardiac arrhythmia ablations, cardioversions, and more invasive cases are being performed at an increasing rate.

This chapter is designed to provide the basic knowledge to understand the workings of the EP suite and successfully construct a safe and effective anesthetic strategy for patients with severe, complex cardiovascular problems.

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The EP Suite

The EP suite has several features specific to the location and orientation of the room. Most EP suites lie outside the main operating room, thus leading to less availability of help when needed and less reliable delivery of backup supplies in a timely fashion. A review of the Closed Claims Database showed that in locations remote from the main operating room, there were more respiratory events, and the proportion of those events leading to death was doubled than procedures in the main OR. Backup plans and rapid diagnosis of adverse events are vital to supplying safe anesthesia.

A typical EP suite contains very large fluoroscopes, magnetic mapping machines, and monitors that obstruct access to the patient once the procedure has started. The EP table does not allow for elevating patients' heads, so they must be able to tolerate lying supine. Extension sets for intravenous infusions, oxygen masks, monitors, suction lines, and anesthesia circuits are needed, and they further increase the distance of the anesthesia provider from the patient (Figs. 27.1 and 27.2). For these reasons, making sure the patient is stable and positioned properly before the start of the case is especially important in the EP procedure room. Furthermore, effective communication between the anesthesia provider and other staff members is often challenging, as they may be in a separate control room or using headphones.

The risk of radiation exposure is high when working inside the EP suite. The use of lead aprons, thyroid shields, eye wear, and pull-down protective shields is critical for provider safety. Standing at least 6 feet away from the machine and the patient during the procedure will help personnel decrease the amount of primary radiation exposure.

Fig. 27.1 Image of EP room. Procedure rooms vary widely in layout. This room poses problems for the anesthesia team because the X-ray machine is placed near the patient's head, away from the oxygen and suction lines, and there is no readily available anesthesia machine

Fig. 27.2 Image of EP room. The location and configuration of monitors, supplies, and equipment are often different from that in the standard operating rooms. Familiarization with the room and equipment ahead of time will prevent confusion when emergencies arise

Monitoring

Standard monitors are required for EP procedures, based on the American Society of Anesthesiologists standards and practice parameters. All patients should be monitored with a 5-lead ECG, pulse oximeter, noninvasive blood pressure cuff, and temperature probe. Recording of end tidal CO_2 is standard for general anesthesia cases and should be used for all deep-sedation and monitored anesthesia cases. Invasive arterial catheters should be placed in hemodynamically unstable patients and in those undergoing procedures that are at risk for inducing hemodynamic changes. Measurements of arterial blood gas analysis may be useful in complex ablations when case duration can be several hours, and large amounts of saline irrigation can cause volume overload. Esophageal temperature monitoring is useful in preventing thermal injury during pulmonary vein isolation procedures for atrial fibrillation ablation. Serial measurements of activated clotting times are needed for monitoring patients who will receive high doses of heparin for procedures requiring anticoagulation, with target times of more than 300 s.

Cardiac Pre-assessment

A thorough history and physical examination are required for patients undergoing cardioversion or ablation procedures, especially because they have complicated medical problems. Cardiovascular conditions such as hypertension, arrhythmias, coronary artery disease, ischemia, past myocardial infarctions, and congestive heart failure should be medically optimized before the start of the procedure. The American College of Cardiology/American Hospital Association guidelines classify patients' status into three categories: active cardiac conditions, comorbid disease, and functional status. Active cardiac conditions include unstable angina, decompensated heart failure, significant arrhythmias, and severe valvular disease. Depending on the urgency of the case, these patients typically need intensive cardiac workups with ECG, echocardiogram, exercise and/or chemical stress tests, perfusion studies, coronary catheterizations, and EP mapping procedures. Preoperative assessment and functional status assessment is discussed in detail in Chap. 5.

EP Procedures

Device Implantation

Implantation of cardiac devices is a surgical procedure that is growing rapidly in parallel with our aging patient population. There are several types of devices, including:

- *Permanent pacemakers* are placed to maintain an adequate heart rate and rhythm by assisting the heart's conduction system. They are placed for treatment of certain types of bradyarrhythmias, high-grade atrioventricular (AV) conduction blocks, certain bifascicular blocks, and various cardiomyopathies. Bradyarrhythmias include sick sinus syndrome and tachycardia–bradycardia syndrome. High-grade AV blocks that require pacing include second-degree Mobitz type II block and third-degree (complete) heart block.
- *Cardiac resynchronization therapy (CRT)* devices are indicated for treatment of delayed ventricular contraction due to left bundle branch block. It is indicated for patients in congestive heart failure with an ejection fraction of less than 35% and a QRS complex wider than 120 ms. Synchronization of the left ventricle alleviates left ventricular outflow tract obstruction in patients with hypertrophic obstructive cardiomyopathy.
- *Implantable cardioverter-defibrillators (ICDs)* are placed for control of rapid defibrillation when a malignant rhythm, such as ventricular tachycardia or ventricular fibrillation, is detected. Historically, the indication for ICD placement was secondary prevention of sudden cardiac death in patients with a hemodynamically significant ventricular tachycardia and/or ventricular fibrillation. More

recent indications include primary prevention, that is, before the first malignant arrhythmia episode; conditions associated with sudden cardiac death, for example, prolonged QT syndrome; Brugada syndrome (right bundle branch block with ST elevations in leads V1 to V3); arrhythmogenic right ventricular dysplasia; hypertrophic cardiomyopathy; and diseases that increase the risk of malignant dysrhythmia (cardiac sarcoidosis, amyloidosis, and others). In addition, data from the second Multicenter Automatic Defibrillator Intervention Trial (MADIT-II) suggests that patients who have an ejection fraction less than 30% after a myocardial infarction should have an ICD implanted.

There are many device combinations: a multitude of device producers, settings, and functions; and rapidly emerging technological advancements that are beyond the scope of this chapter.

(a) Airway and Anesthesia Management

The types of anesthesia used in EP procedures vary from light sedation to general anesthesia with endotracheal intubation. Many factors concerning anesthesia must be considered before undertaking the procedure. Although deep sedation can be achieved with propofol infusion, its cardiovascular depressant effects can be especially pronounced in patients with already compromised cardiac function, who may suffer profound hypotension. In patients with airway concerns, ketamine and dexmedetomidine can be effective choices to preserve cardiac function and maintain spontaneous ventilation during deep sedation. For patients who are unable to lie supine because of shortness of breath, general anesthesia with endotracheal intubation is the preferred anesthetic method. Several factors must be considered when using general anesthesia in patients with poor cardiac function: an arterial catheter (placed before induction) will allow hemodynamic monitoring and more rapid response to an adverse hemodynamic event; if propofol is used for induction, phenylephrine, norepinephrine, and/or epinephrine should be immediately on hand for treatment of decreased cardiac output and hypotension; etomidate is an alternative induction agent to propofol that causes less cardiovascular depression; and inhalation anesthetics can affect the heart's conduction system besides having cardiac depressant effects. The effects of inhalant anesthetic agents can be lessened by giving supplemental opioids.

(b) Special Considerations

The extraction of devices is a subset of procedures that impose concerns that are different from those of implantations. Depending on the length of time the lead has been in place, methods of extraction will influence the anesthetic plan. For example, if the leads have been in place for over a year, extraction with use of a laser is usually indicated. Please refer to Chap. 31 for more information.

(c) Complications

Complications can occur secondary to the surgery, the anesthesia, or the device. During surgery, intravascular and intra-cardiac placement of leads can cause hematoma, dissection, and perforation leading to pericardial tamponade. Accessing the subclavian vessels can lead to pneumothorax and/or direct trauma to the lung. Leads can be a nidus for infection; can accumulate thrombus, fracture, or dislodge; and can malfunction and require extraction. The subcutaneous pocket where the device is implanted is susceptible to infection, hematoma, and erosion.

Catheter Ablation

Catheter ablation procedures (Fig. 27.3) performed in the EP suite vary according to the type of arrhythmia being treated. Arrhythmias that can be successfully ablated are

- Paroxysmal supraventricular tachycardia consists of AV nodal reentrant tachycardia, atrial tachycardia, and atrial reciprocating tachycardia. These tachyarrhythmias have narrow QRS complexes and are usually not life-threatening, but they are likely the result of other adverse cardiac conditions
- Atrial flutter
- Atrial fibrillation
- Premature ventricular contractions
- Ventricular tachycardia

A thorough history and physical examination should be completed to understand the patient's condition. These arrhythmias can be diagnosed by ECG or Holter monitoring and are usually first treated medically. Indications for ablation are failure of medical management, patients' preference of ablation over medications; patients'

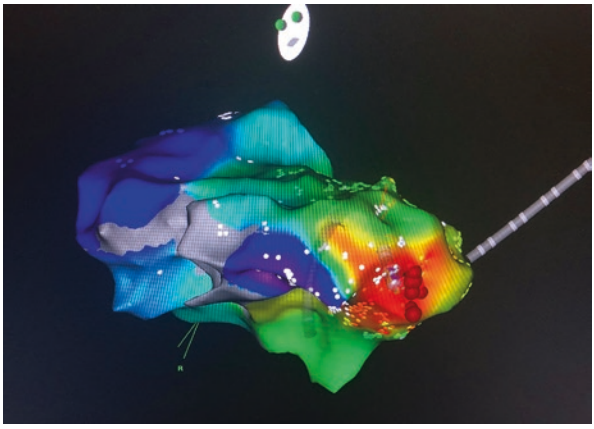

Fig. 27.3 Image of CARTO map showing focal ablation of atrial tachycardia in the left atrium (red spheres). The RF ablation catheter can be faintly seen curving to the left of the ablation area

poor tolerance of the arrhythmia, and persistence of serious symptoms. Ablation of these rhythms has a success rate of about 90%.

The Ablation Technique

Ablation procedures use various types of energy to remove diseased cardiac conductive tissue. Radiofrequency energy is the most commonly used and utilizes heat to ablate the tissue. Because of the high temperature used, cold saline is infused around the catheter tip to prevent damage to surrounding healthy cardiac tissue. In long ablation procedures, patients commonly receive more than 3 L of saline; thus, careful monitoring and fluid management is needed to avoid inducing decompensated heart failure. Another potential complication of radiofrequency ablation is thermal injury to the esophagus because of its proximity to the pulmonary vein (Fig. 27.4). Thus, intraoperative esophageal temperature must be carefully monitored; if the temperature begins to rise too quickly, the proceduralist should be warned and the ablation paused. Less common methods of ablation than radiofrequency are cryoablation, microwave, and laser ablation. Cryoablation uses a subzero coolant at the catheter tip to freeze the aberrant conducting tissue, so it does not cause burn injury. However, recurrence rates are higher with cryoablation than with radiofrequency, so it is used less often. Laser and microwave ablation are alternative methods to radiofrequency ablation that are being developed, but they are not routinely used.

The ablation techniques and procedure steps used depend on the type of arrhythmia being treated, so the anesthetic management should be tailored to the procedure.

(a) Airway and Anesthetic Management

Anesthetic and airway management are based on the patient's cardiovascular status and comorbidities and the type of ablation procedure. The advantages of general anesthesia include the elimination of patient movement, permissibility of

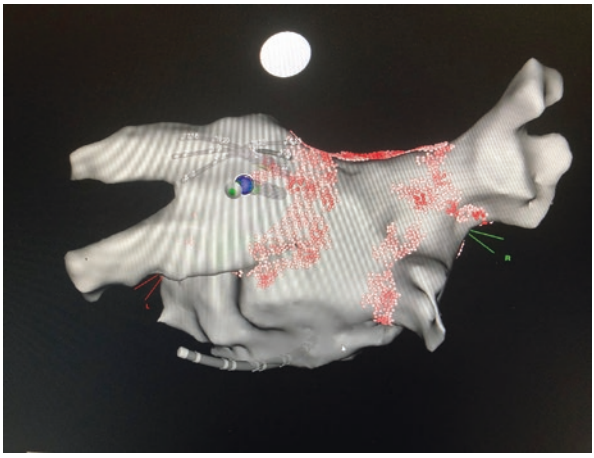

Fig. 27.4 Image of CARTO map posterior view of the left atrium showing the left and right pulmonary veins. The pink dots are the ablation points ringing the pulmonary veins. The RF ablation catheter can be seen behind the blue dot near the left upper pulmonary vein with the 5-rayed mapping catheter just above it

transesophageal echocardiographic monitoring, ability to stop respiratory motion when mapping of the myocardium is underway, patient comfort during lengthy procedures, and a favorable success rate. Also, studies have shown that general anesthesia improves catheter stability, thus decreasing procedure time. Induction agents such as propofol, etomidate, and ketamine are effective and should be considered on a patient-to-patient basis. Whether inhalational anesthesia or total intravenous anesthesia is better for maintenance of general anesthesia is controversial.

(b) Special Considerations

Long-acting neuromuscular blockers should be avoided if the proceduralist will be monitoring the phrenic nerve during the ablation.

During mapping, the proceduralist may need to induce an arrhythmia (with adenosine, isoproterenol, or dobutamine or by rapid pacing) to help locate the diseased myocardium.

(c) Complications

Three serious complications that may be encountered with ablation are cardiac tamponade, stroke, and atrial-esophageal fistula. The frequency of occurrence of these and rarer complications are listed in the following table (Table 27.1).

Cardioversion

Cardioversions in the EP laboratory are often short procedures, but they present risks and challenges for the anesthesia provider like those of implantations. Cardioversion is the brief delivery of synchronized electrical direct current (DC) across the patient chest. This DC current will allow a brief depolarization of myocardial cells, therefore enabling the sinus atrial node to resume normal pacing activity and to improve hemodynamics. Anesthetic considerations depend on the patients' comorbidities, functional status, and hemodynamic stability and on the type of dysrhythmia to be cardioverted. In cardioversion, a one-Joule-per-kilogram biphasic electrical shock, synchronized with the R wave of the QRS complex, is delivered across the chest. Current biphasic defibrillators reverse polarity and electrical

Table 27.1 The frequency of occurrence of complications encountered with ablation

Complications	Incidence
Tamponade	1.5 %
Stroke	0–7%
Atrial-esophageal fistula	0.1–0.25%
Esophageal injury	0–17%
Vascular injury	0–13%
Phrenic nerve injury	1–6.5%
Pulmonary vein stenosis	1.3–3.4%
Pericardial effusion	2.9%
Vagal nerve injury	1%

current flow after a few seconds to create a biphasic waveform as opposed to monophasic waveform defibrillators, thus allowing to use lower energy and generate more efficient myocardial cells depolarization.

Cardioversion procedures that are performed electively include conversion of atrial fibrillation and atrial flutter to normal sinus rhythm. Patients with these arrhythmias who are hemodynamically stable require a 3- to 4-week course of anticoagulants before electrical cardioversion to try to eliminate atrial thrombi and reduce the risk of embolic stroke. If the procedure is urgently needed, an echocardiogram should be performed before the cardioversion to check for left atrial thrombi.

(a) Airway and Anesthetic Management

Assessment for possible difficulties with ventilation is key for successful airway management in these procedures. Endotracheal intubation is used rarely, but when it is used, deep sedation and, sometimes, general anesthesia is required; accordingly, positive-pressure ventilation and suction equipment should be on hand throughout the procedure. If the patient has not fasted in accordance with guidelines, the airway should be secured by rapid-sequence induction of anesthesia and endotracheal intubation.

Total intravenous MAC anesthesia is the preferred anesthetic method for cardioversion, but no single agent is appropriate for all patients; anesthetic management is determined by individual hemodynamics. Etomidate, propofol, benzodiazepines, and fentanyl have specific indications for use, and a 2015 Cochrane Database Review concluded there was insufficient evidence to support the use of one agent over another.

(b) Special Considerations

Implantable devices, such as pacemakers or AICDs, should be interrogated before and after the procedure, as the electrical shock can cause lead malfunction and/or loss of capture.

Transient ST-segment elevation may appear on the ECG after cardioversion and can be a normal variant. However, if the ECG change persists for longer than 2 min, myocardial injury may have occurred, either from the electrical shock or from the patient's underlying heart disease, and evaluation is indicated. Soft bite block is routinely placed between the patient's molars after inducing of anesthesia to prevent the teeth from biting on the tongue during delivery of electrical shock.

(c) Complications

Anesthetic complications of cardioversion include airway compromise leading to hypoxia, hypercarbia, and respiratory acidosis. Hemodynamic instability due to anesthetic medications can lead to myocardial ischemia and injury to other end organs. Stroke is a risk of cardioversion, which explains the development of guidelines for performing thrombus assessment and anticoagulation before performing the procedure. If a thrombus is present in the left atrial appendage, cardioversion is contraindicated. Also, electrical shock can produce transient sinus arrest,

bradycardia, or other dysrhythmias that can lead to hemodynamic instability. Unsuccessful cardioversion is another potential complication.

Postanesthesia Care Unit

Patients who have received general anesthesia or monitored-anesthesia care should be monitored in the postanesthesia care until they are fully recovered from the anesthetic. If local anesthesia or mild/moderate sedation was used and there no complications, the patient may be fast-tracked to phase two recovery and bypass the unit.

Anesthesia for Watchman Procedure

28

Sagar S. Mungekar and Brian Raffel

I shall the effect of this good lesson keep, as watchman to my heart.

—Hamlet, 1.3.45–46

Key Points

- Introduction
- Considerations for pharmacologic anticoagulation
- Alternatives to anticoagulation
- Structure and function of the LAA
- LAA occlusion and patient considerations
- Preoperative assessment
- Preoperative preparation
- Procedural considerations
- Future directions

Introduction

Atrial fibrillation (AF) is characterized by an irregularly irregular rhythm, meaning that the time between each heart beat is variable and without any discernable pattern. In AF, P waves are not regularly seen on the EKG, indicating disorganized left atrial activity. Instead of action potentials initiating at the sinoatrial node, rapid, irregular electrical activity originates near the pulmonary veins and triggers AF. This arrhythmia may be paroxysmal (terminating within seven days without any intervention) or persistent (lasting longer than 7 days). Persistent AF can result in

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structural and electrical remodeling of the atrium. Fibrosis, inflammation, and hormonal effects from the sympathetic and parasympathetic nervous systems also contribute. When valvular abnormalities, such as mitral or aortic regurgitation increase left atrial pressure and volume, the resulting AF is called valvular. Conversely, nonvalvular AF is a term used to describe the arrhythmia when other pathogenic features such as systemic hypertension are responsible.

The absence of organized atrial contraction in late diastole deprives the ventricle of the preload blood volume referred to as the "atrial kick" and may therefore compromise cardiac output. AF also causes a low-flow state in the atrium; this state is somewhat ameliorated in valvular AF because the regurgitant jet promotes higher blood velocities in the atrium, but in nonvalvular AF, stasis of blood flow in the LAA may lead to thrombus formation.

Considerations for Pharmacologic Anticoagulation

Thromboembolism is a serious complication of AF. Emboli from a cardiac source may reach the brain and cause an ischemic stroke. Given the morbidity and mortality of cerebrovascular accidents and the likelihood of their recurrence with AF, conventional prophylaxis has been systemic anticoagulation with oral therapy. Warfarin was the only option until it was partially replaced by non-vitamin K antagonist oral anticoagulants. Due to the association of AF with advanced age and the increased incidence of falls in elderly persons the benefit of systemic anticoagulation must be carefully weighed against the risk of bleeding.

Alternatives to Anticoagulation

A number of nonpharmacological methods can combat LAA thrombus formation. If already undergoing open-heart surgery, a patient may have the LAA surgically closed, ablated, clipped, or stapled. Alternatively, percutaneous endocardial LAA occlusion is possible. The Watchman® (Boston Scientific, Natick, MA, USA) is the only device approved by the US Food and Drug Administration for this purpose. Other devices, such as the Amplatzer™ Amulet™ (St. Jude Medical–Abbott, Abbott Park, IL, USA) are in development, but not yet approved. These occlusion or exclusion devices work on a simple premise: by excluding the LAA from the systemic circulation, any thrombus within it is unlikely to embolize, and thus the risks of stroke and bleeding associated with therapeutic anticoagulation are ameliorated. The Watchman® device is delivered via a trans-septal approach. When deployed at the ostium of the LAA, it automatically expands, driving its barbs into the endocardium.

Structure and Function of the Left Atrial Appendage

The LAA is a trabeculated pouch with a few pectinate muscles across it. It is long and hooked, but the shape can vary. Three main shapes have been described: "chicken wing" LAA, "cauliflower" LAA, and "windsock" LAA. It is believed that the LAA, with its high compliance, functions as a reservoir that can expand to offload high pressure from the LA. Also, the LAA responds to atrial stretch by secreting atrial natriuretic peptide, which assists in fluid balance. Occlusion rather than excision of the LAA can preserve some of these properties.

LAA Occlusion and Patient Selection

Clinical trials have demonstrated the efficacy of the Watchman® device in stroke reduction for patients with contraindications to new oral anticoagulants NOACs. Patients must meet certain criteria to be considered for Watchman® device implantation. They must have nonvalvular AF. Fit the scoring system that predicts the risk of bleeding. The patient should also be able to take warfarin for the short term, but not the long term. Finally, the patient must have an appropriate reason to seek a nonpharmacological alternative to warfarin. Such reasons include a history of or high risk for falls; poor compliance with or efficacy of warfarin; and renal failure. After the patient's medical history and risks of stroke and anticoagulation are evaluated, the patient and another physician (who is not the interventional cardiologist who would implant the device) must engage in shared decision-making regarding the therapeutic plan.

Preoperative Assessment

By the time the anesthesiologist sees the patient for the Watchman® procedure, much of the evaluation has been completed, but review of the history and decision tree that brought the patient to this point is prudent. Please refer to Chap. 5 for more information about preoperative assessments.

Evaluation of AF includes the type of AF (paroxysmal versus persistent), associated symptoms, appearance and rate on EKG, and presence of valvular abnormalities. In preparation for Watchman® implantation, patients undergo computed tomography scans for LAA size and position and a transesophageal echocardiogram (TEE) to evaluate for presence of a thrombus. The anesthesia provider may use these studies for additional information, such as evaluation of ventricular function and other valvular or structural cardiac abnormalities. Knowledge of systolic and diastolic function along with valvular insufficiency and/or stenosis helps determine

hemodynamic goals and which monitors will be needed for perioperative management.

An association exists between obstructive sleep apnea (OSA) and AF. Patients should be screened for OSA using the STOPBANG questionnaire (see Chap. 5). Knowledge of OSA severity can assist with intra- and postoperative management. Untreated or severe OSA may be associated with right ventricular hypertrophy or failure. Postoperatively, patients with OSA are at increased risk for hypoventilation and hypoxia, especially if residual neuromuscular blockade is present or longer-acting opioids are used.

Since intraoperative blood pressure and heart rate swings are possible, knowledge of the patient's medical history can help narrow the differential diagnoses, which includes medication effects or procedural bleeding. Knowing whether the patient is on antiarrhythmic, rate-control, or anticoagulation medications, and their last doses, is critical. Because potentially nephrotoxic intravenous (IV) contrast agents are used during the procedure, preoperative blood urea nitrogen and creatinine serum levels can help differentiate among pre-, intra-, and post-renal causes of intra- and postoperative acute kidney injury and guide fluid management. Since the procedure entails a risk of bleeding, a preoperative serum hematocrit will help determine acceptable blood loss. Also, a blood type-and-screen with cross-matching should be obtained. Informed consent for anesthesia care should include a discussion about the possible need for invasive monitoring and blood product transfusion.

Clinical Pearls

- Review available imaging, such as computed tomography scans and preoperative echocardiograms to better appreciate heart function and valvular abnormalities.
- Evaluate for common comorbidities such as hypertension, OSA, obesity, and heart failure.
- Confirm that recent laboratory tests include BUN, Cr, Hct, electrolytes, and blood type-and-screen/cross.

Anesthetic Techniques

The Watchman® implantation procedure requires real-time TEE guidance. Because of this, general endotracheal anesthesia is usually the preferred modality of anesthesia. There are reports of Watchman® implantations being performed with an intracardiac echo catheter and sedation without formally securing the airway. However, since patients must lie flat for the duration of the procedure and remain immobile

during trans-septal puncture and Watchman® deployment, we perform all procedures with general endotracheal anesthesia.

Intravenous Access

Peripheral IV access should be obtained before start of the procedure. Since the potential for bleeding and cardiac tamponade exist, the caliber of the catheter should be large enough to allow easy transfusion of blood and other volume expanders. The patient's arms will be tucked; hence there is little opportunity for ad hoc line placement. The anesthesia provider should pay close attention to the location of the catheter and the path of the intravenous tubing so that the line remains unencumbered when the patient is in the final arms-tucked position. In some cases, due to the patient's condition or anxiety, a smaller IV catheter may be used for anesthesia induction, and a larger one placed post-induction. Alternatively, an access line can be shared (described later).

Anxiety Management

As with most procedures, these patients have pre-procedural anxiety. Often, verbal reassurance will suffice to allay the anxiety, but if it does not, IV anxiolytics can be administered, with caveats: Depending on the institution, patients may have to transfer themselves from a wheelchair to the articulating table by way of a step stool and remain sitting without back support while various monitors are applied to their skin, and sedation may introduce risk for these activities; benzodiazepines may alter balance and lead to a fall, which could be catastrophic in an anticoagulated patient. If preoperative IV sedatives are administered, the anesthesia provider should share this information during the hand-off from the preoperative preparation area to the hybrid procedural room so that all team members can take appropriate precautions for patient safety.

Hybrid Rooms

Hybrid rooms are procedure rooms that also have an imaging device, such as a C-arm, which makes them appropriate since multiple modalities (fluoroscopy and echocardiography) are required for Watchman® implantation. All team members who will be in the room should use appropriate personal protective equipment, which includes lead aprons with thyroid shields at a minimum, sometimes accompanied with radiation protection eyeglasses and skull caps. Acrylic shields and lead

aprons connected to booms may also be used. Since team members may face different directions with respect to the radiation source during the procedure, they should make sure that they are appropriately protected from radiation; wrap-around lead aprons may be required. At least three different teams are involved in these procedures. The echocardiographer (often a cardiologist, but in some institutions, an anesthesiologist) and technician will have a TEE machine located close to the patient's head. The anesthesiologist will also have an anesthesia machine at the head. The proceduralist will often positioned be to the patient's right side, opposite a large overhead screen system. If display monitors are shared among teams, all members should ensure that they have a continuous view of the relevant information. Certain images such as vital signs and sonograms will have to be mirrored electronically to different screens.

Monitoring

Standard American Society of Anesthesiologists monitors should be placed before induction of anesthesia in the hybrid suite. Placement of a pre-induction arterial line should be considered whenever hemodynamic instability is predicted to occur during induction, for example, in patients who have heart failure with a reduced ejection fraction or valvular disease, because a close balance of loading conditions is required.

A Foley catheter is necessary for longer procedures since fluids are infused by the anesthesia provider and the proceduralist through the access catheters. A temperature-sensing bladder catheter is useful since an esophageal temperature probe may interfere with the TEE probe. If the institutional practice is to omit indwelling urinary catheters for shorter procedures, the parties should agree on when one should be inserted if the procedure is unexpectedly long. Condom (Texas) catheters are not useful while the patient is under general anesthesia since they only measure overflow incontinence.

Placement of the monitors on the patient requires forethought. Because the procedure carries a risk of arrhythmias, defibrillator pads should be applied, which may more easily be accomplished with the patient in the sitting position. The defibrillation pads should be placed in a posterior–lateral configuration and out of the way of the fluoroscopy path. Each of the three teams may require a set of EKG leads, so the remaining available body surface area will be shared. Care should be taken to minimize reaching or stepping over the monitor cables and airway circuit during the procedure. Also, the cables and tubes should have enough slack and length to accommodate the fore and aft movements of the articulating table; this issue will require a discussion of where each team member will stand and the positions and limitations of the TEE machine, anesthesia machine, C-arm, and articulating table.

Defining Team Role

During or before the institutional time-out procedure, the teams should determine who is responsible for each sequential task. At some institutions, the proceduralist

will place a femoral arterial line for access and invasive blood pressure monitoring. At other institutions, the anesthesia provider will place a catheter in the radial artery for blood pressure monitoring. If need for central venous access is anticipated for infusion of vasopressors, the proceduralist may leave a side-port of the femoral venous access sheath available, which should be separate from any lines through which heparin is infused for intraoperative anticoagulation. If such access is not available, the anesthesiologist may have to place an internal jugular central line, but this is not routine. At some institutions, the proceduralist will purposely upsize the venous introducer catheter to serve as a conduit for rapid infusion of fluids or blood in emergent circumstances. At other institutions, it is incumbent on the anesthesiologist to obtain adequate vascular access; this issue is best discussed prior to patient positioning.

During the procedure, medications will be administered for anesthesia and the procedure. Prior agreement on who will administer heparin and protamine, and intraoperative closed-loop communication, will help avoid confusion. Finally, the parties should determine preoperatively how emergency situations will be addressed should they arise: Who will manage the defibrillator if a shockable arrhythmia develops; how will the C-arm be moved to provide access to the chest for compressions or subxiphoid pericardial drainage access; and will a surgeon be available if needed?

Induction of Anesthesia

Induction of anesthesia should be tailored to each individual patient based on their medical history and comorbidities. Lidocaine and a short-acting opioid, such as fentanyl, provide partial sympatholysis; a titrated dose of propofol provides hypnosis; and rocuronium or succinylcholine provide paralysis for ease of tracheal intubation and for procedural immobility. Cefazolin or another appropriate antibiotic provide prophylaxis for surgical site infections.

Maintenance of Anesthesia

For maintenance of anesthesia, all the volatile inhaled anesthetics are appropriate. There is no specific contraindication to the use of nitrous oxide, but some rooms in the catheterization laboratory suites may not have pipeline-supplied nitrous oxide. If a cylinder supply is used, the anesthesiologist should calculate the amount of nitrous oxide available based on fresh gas flows and cylinder weight. Using total IV anesthesia is also an option, especially if the patient will not tolerate higher doses of volatile agents. Nondepolarizing muscular blockade is used to preclude patient movement with catheter manipulation, but it should be administered in a manner that facilitates complete intraoperative reversal.

Emergence

After completion of the procedure and reversal of anticoagulants and muscle relaxants, extubation can be accomplished expeditiously in the operating room. A selective muscle relaxant binding agent such as sugammadex can be used at the conclusion of the procedure to avoid prolonged intubation when needed.

Clinical Pearls

- When setting up the room, think about where everyone is going to stand and whether cables are out of the way.
- Find appropriate lead aprons and shields prior to the start of the procedure.
- Ask ahead of time whether the access catheters can be used for invasive blood pressure monitoring and infusion of medications. Otherwise, the anesthesiologist should plan to obtain arterial and venous access elsewhere.
- The patient's arms will be tucked, resulting in very little access to the patient once the procedure starts.

Procedural Considerations

The Watchman® implant is a self-inflating device designed to seal off the LAA (Fig. 28.1). The device is inserted into the LAA through a catheter-based delivery system and by puncturing the atrial septum between the right and left atria.

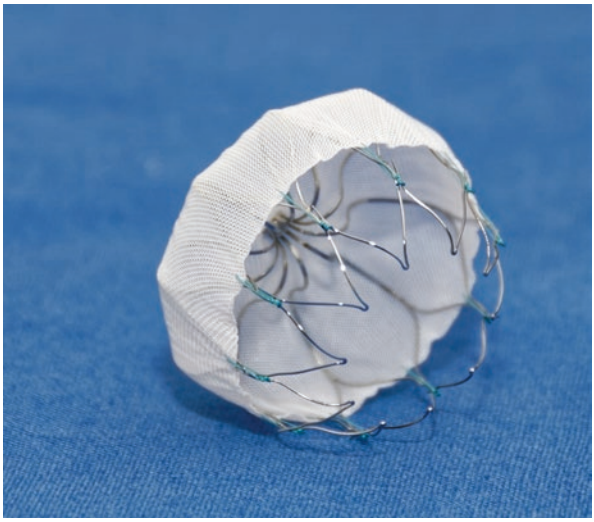

Fig. 28.1 Image of the Watchman®device

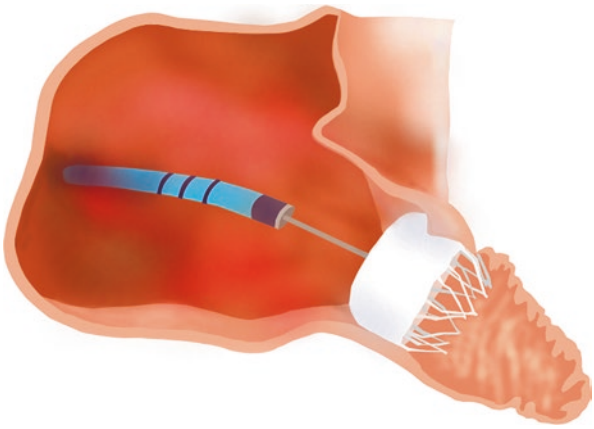

Fig. 28.2 Illustration of Watchman® device deployment

Procedural Technique

The steps of the procedure are briefly summarized:

1. Standard American Society of Anesthesiologists monitors are placed on the patient and IV access obtained.
2. Arterial line – pre-induction if needed.
3. General anesthesia induced and patient intubated.
4. TEE to measure the LAA ostium and LAA length in 0–135 deg sweep from top of the mitral valve annulus to a point 2 cm from tip of the limbus and determine device size (five sizes are available 21, 24, 27, 30, and 33 mm). The device size is selected with 20% more than the maximum measured LAA diameter to provide good seal and stabilization.
5. Implant performed with fluoroscopy and TEE.
6. Patient's position optimized.
7. Femoral access obtained.
8. Heparin administered (100 unit/kg dose for activated clotting time above 250 seconds).
9. Trans-septal puncture with echocardiographic guidance.
10. Device positioned near the orifice of the LAA.
11. Device anchored.
12. Device deployed (Fig. 28.2).
13. Size match confirmed and seal assessed with doppler echocardiography.
14. Protamine may be given to neutralize heparin if elected by the cardiologist.

Management of Complications

Pericardial tamponade and major bleeding are two of the more severe complications of Watchman® implantation; both are associated with a decrease in blood pressure, variation in stroke volume with respiration, tachycardia, and decreased end-tidal

CO_2. Close inspection of the arterial line and central venous pressure tracings can provide clues for differentiating one of these complications from the other. Violation of the myocardium with a guidewire causes extravasation of blood into the pericardial space. Although this injury may occur often during the procedure, it is more likely to occur after trans-septal puncture if the proceduralist overshoots the LAA and pierces its softer, thinner tissue. TEE is useful for determining if a pericardial effusion and consequent tamponade are present.

Vascular Injury

Gaining vascular access and deployment of the Watchman® device may be associated with inadvertent vascular injury.

Pericardial Effusion

If a pericardial effusion occurs, each team will have responsibilities for managing it: The echocardiographer can visualize the extent to which chamber collapse is occurring; the anesthesia provider can supply preload, increase the heart rate, and administer vasopressors; and the proceduralist can perform a pericardiocentesis. In rare cases, if the patient's habitus is not amenable to pericardiocentesis, a sub-xiphoid surgical approach may be necessary to accomplish drainage and decompression.

Bleeding

Bleeding may occur either intra- or postoperatively, often from attempts at femoral vascular access or ineffective closure. It is important to avoid patient movement at the site of vascular access to minimize the chance of vascular closure disruption. Occult bleeding also is possible, as with continued bleeding from a hematoma in the patient's leg, groin, or retroperitoneal space. After pericardial tamponade has been ruled out, bleeding at other sites is the next serious complication that should be explored for persistent hypotension. Signs of hypovolemia include an arterial pressure tracing with exaggerated pulse pressure variation, small end-diastolic ventricular volume on TEE, and a drop-in hematocrit. Although blood transfusion may be the first task, definitive treatment involves repair of the vascular laceration, especially when the patient is anticoagulated.

Arrhythmias

A common but often inconsequential complication during this procedure is premature ventricular contractions and other short, often self-terminating, arrhythmias due to mechanical stimulation by the various guidewires. Though these episodes

may be associated with lower stroke volumes, hypotension occurs only if they are prolonged. Constant communication with the proceduralist is important, especially if vasopressors are administered, since they may mask more ominous reasons for hypotension, such as an increasing pericardial effusion.

Air Embolism

Another common complication is air embolism. Virtually every flush of a catheter results in some entrapped air entering the circulation. TEE is a very sensitive monitor for detecting intra-cardiac air. Small amounts of air are inconsequential and are occasionally introduced deliberately for a "bubble study" to evaluate for atrial septal defects or a patent foramen ovale. Larger air emboli may enter if lines are improperly flushed or if intrathoracic pressure becomes suddenly negative, as can occur if the patient initiates inspiratory effort. Sequalae can include embolism to coronary or cerebral vessels, which requires supportive therapy. If the volume is large enough to cause an air-lock, the air should be aspirated via a catheter.

Postoperative Care

When patients are brought to the postoperative recovery area, a structured transition of care through a formal hand-off mechanism is imperative. Structured transition facilitates communication about comorbidities and special considerations that pertain to the procedure and intraoperative course. It is especially important to avoid hypoventilation and hypoxia when the patient suffers from other cardiac or pulmonary conditions, such as heart failure, fluid overload, OSA, and the presence of residual narcotic, volatile agent, or muscle relaxant.

Postoperative complications associated with the device itself are rare, but it may embolize if it is deployed incorrectly or not well seated, and a para-device leak may render the device a failure, requiring the patient to continue oral anticoagulants. Infections are rare. Postoperative follow-up TEEs are required to evaluate for these events.

Clinical Pearls

- Always consider pericardial effusion and tamponade as a reason for hypotension.
- Real-time TEE used for guidance also is valuable for directing hemodynamic goals. Low end-diastolic diameters may indicate hypovolemia.
- Bleeding may begin postoperatively due to ineffective closure of venous access and can be precipitated by patient movement and bending at the closure site.
- Poor postoperative oxygen saturation can have many causes including OSA, residual neuromuscular blockade, opioid-induced hypoventilation, and fluid overload.

Further Reading

1. Boston Scientific. WATCHMAN® Left Atrial Appendage Closure Device. Patient Information Guide. https://www.bostonscientific.com/content/gwc/en-US/products/laac-system.html
2. Hussain Z, et al. Anesthetic Management of Patients Undergoing Percutaneous Endocardial and Epicardial Left Atrial Appendage Occlusion. Seminars in Cardiothoracic and Vascular Anesthesia: The Journal of Perioperative Medicine (SCVA). 21(4):291–301. https://doi.org/10.1177/1089253217714581.
3. Tzikas A. Textbook of catheter-based cardiovascular interventions a knowledge-based approach. In: Left atrial appendage occlusion: state of the art: Springer International Publishing AG; 2018.

29 Anesthetic Management of Transcatheter Aortic Valve Replacement

Ahmed Zaky and Ludmil Mitrev

Many of life's failures are people who did not realize how close they were to success when they gave up.

—Thomas Edison

Introduction

Transcatheter aortic valve replacement (TAVR) was indicated initially for patients with aortic stenosis who met criteria for replacement of the aortic valve but had a prohibitive risk for surgical aortic valve replacement (SAVR). Recently, the indications for TAVR were expanded to include patients with intermediate surgical risk. In 2019, two trials found that TAVR was noninferior to SAVR in low-risk patients. The Partner 3 trial demonstrated that death, stroke, and rehospitalization at 1 year were significantly lower with TAVR than with SAVR in this patient population, and there was no significant difference in the rate of major vascular complications, new permanent pacemaker insertions, or moderate-to-severe paravalvular regurgitation between TAVR and SAVR. The Evolut trial found that TAVR was noninferior to SAVR in low-risk patients in mortality rate or disabling stroke at 2 years. These results paved the way for TAVR to become a valid treatment option for severe aortic stenosis, even though long-term outcomes data are still lacking.

Electronic Supplementary Material The online version of this chapter (https://doi.org/10.1007/978-3-030-51755-7_29) contains supplementary material, which is available to authorized users.

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Despite being less invasive than SAVR, TAVR poses its own challenges that require the anesthesia team to have adequate training, skills, and vigilance. The following is an updated and concise review of anesthetic management of patients undergoing TAVR.

Patient Selection, Risk Stratification, and the Decision to Pursue TAVR

Team Approach

Establishing a patient's candidacy for TAVR is an interdisciplinary *cardiac* team decision. The cardiac team is formed of cardiologists, interventional cardiologists, cardiac surgeons, radiologists, device specialists, vascular surgeons and cardiac anesthesiologists. For consideration of candidacy, patients are seen in dedicated valve clinics and are discussed at TAVR conferences to assess the risk and determine the approach, valve type, anesthetic management, and back up plans for TAVR.

Risk Assessment

Risk assessment proceeds through the following cascade: (Table 29.1):

- *Step 1. Determining the risk for SAVR (Table 29.2 TAVR vs. SAVR)*

Determining the risk for SAVR is based on either the European System for Cardiac Operative Risk Evaluation (EURO SCORE) or the Society of Thoracic Surgeons Predicted Risk Score of Mortality (STS-PROM). Common scoring systems for cardiac surgery are discussed in Chap. 5. A caveat: these scores are NOT designed for TAVR risk assessment and tend to overestimate the risk of TAVR in the elderly.

Table 29.1 Risk Assessment for TAVR

Risk assessment tool	Low[a]	Intermediate[b]	High[b]	Prohibitive[b]
STS-PROM	<4%	4%–8%	>8%	Risk of death >50% at I year
Frailty	None	1	2 or more	As above
Major organ system compromise	None	1	2	≥3
Procedural risk	None	Possible	Possible	Severe existing

STS Society of Thoracic Surgeons, *PROM* predicted risk of mortality. [a]low risk: all four conditions should be met. [b]intermediate, high or prohibitive risk: at least one of the four conditions should be met

Table 29.2 TAVR vs. SAVR in terms of outcomes

Outcome	SAVR	TAVR
Stroke	++++	++
Acute kidney injury	++++	++
Permanent pacemaker implantation	+	++++
A-fib	+++	+
Aortic annular rupture and coronary obstruction	+	++
Vascular injury	+	++++

SAVR surgical aortic valve replacement, *TAVR* transcatheter aortic valve replacement, *A-fib* atrial fibrillation, +++++: more occurrence, +: less occurrence

- *Step 2. Determining frailty in elderly patients*

This determination assesses the patient's physiologic reserve and functional capacity. It is based on a composite of serum albumin levels, hand grip strength, gait speed on a 15-ft walk, and degree of independence in daily life activities.

- *Step 3. Determining major organ system compromise and likelihood of improvement after TAVR*

The goal of this step is to estimate of the quality of life the patient may have after TAVR. Patients with advanced disease are unlikely to benefit from TAVR (see "survival with benefit," below).

- *Step 4. Procedure-specific candidacy*

This step assesses the feasibility of doing TAVR in an individual patient. Patient factors such as valve status, vascular anatomy, and chest-wall deformation are evaluated.

Absolute contraindications for TAVR

1. Life expectancy < I year even after successful TAVR.
2. Expected benefit after TAVR, yet with a less than a 25% chance of survival.

A survival-with-benefit is defined as an improvement of at least 1 point in New York Heart Association (NYHA) classification, improvement in symptoms of angina, improvement in quality of life, or longer life expectancy.

Multimodality Imaging

Several imaging modalities are used in the pre-procedural evaluation of patients to assess the following:

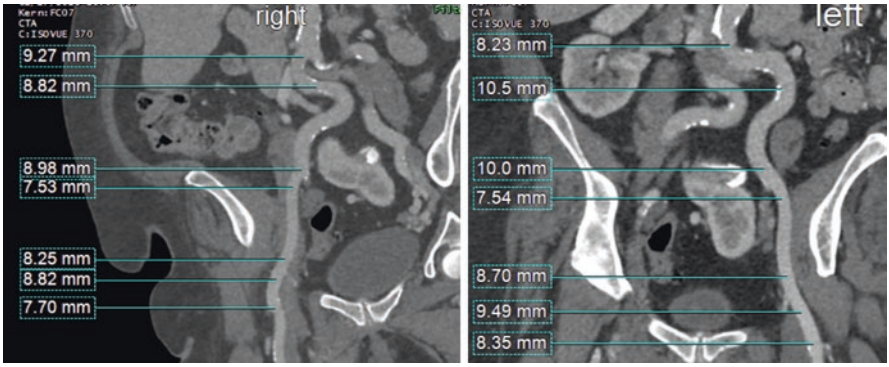

Fig. 29.1 CT angiography of access vessels (femoral, iliac and aortic) to assess vascular anatomy and morphology

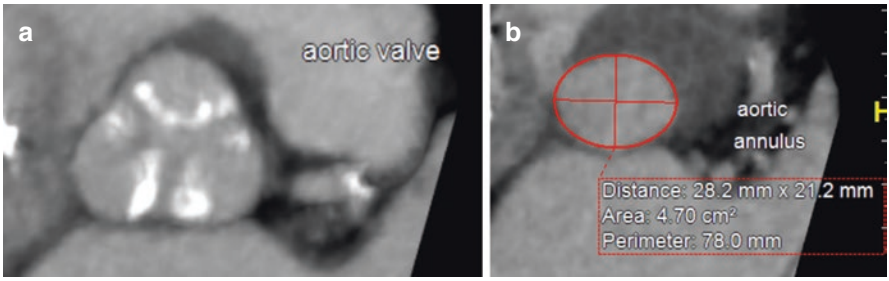

Fig. 29.2 CT imaging of aortic valve leaflets anatomy and calcifications (**a**)**,** and aortic annulus shape and size, (**b**)

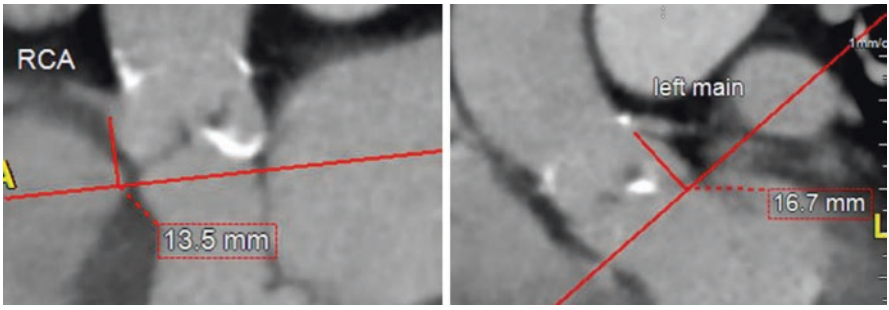

Fig. 29.3 CT imaging of the left and right coronary heights

(a) Diameter and morphology of access vessels (CT angiography, conventional angiography). Determination of vascular anatomy, plaques, and calcifications (Fig. 29.1).
(b) Valve morphology and accessibility (multi-slice CT scan). Calcium scores (Fig. 29.2a).

(c) Aortic annulus and root anatomy (CT, echocardiography). three-dimensional sizing, coronary heights, diameters of the left ventricular outflow tract, annulus, and root (Figs. 29.2b and 29.3).
(d) Cardiac structural and functional assessment (echocardiography). Severity of aortic stenosis and examination for, other valvular abnormalities.
(e) Coronary artery status (right and left cardiac angiography). Concomitant coronary artery disease, presence of pulmonary hypertension.

Choice of Anesthesia

Anesthetic Techniques (Table 29.3)

Largely determined by the interventional approach:

1. General endotracheal anesthesia (GETA).
2. Monitored anesthesia care (MAC).
3. Local anesthesia.
4. Regional (bilateral ilioinguinal and iliohypogastric nerve blocks).

Factors Determining Anesthetic Choice

1. Implantation approach.
   (a) GETA can be used in all approaches.
   (b) MAC is most commonly used with the transfemoral approach, less commonly with the subclavian approach.
2. Patient cooperation.
   Uncooperative patients, those with cognitive abnormalities and those with language barriers may require GETA.
3. The need for transesophageal echocardiography (TEE).

Table 29.3 GETA vs MAC for TAVR

Parameter	GETA	MAC
Airway control	More secure	Less secure
Hemodynamics	Less stable	More stable
PVL	Less	More
Bradycardia	Less	More
Hospital stay	+/− longer	Shorter
Ease of TEE use	Easier	Harder

GETA general endotracheal anesthesia, *MAC* monitored anesthesia care, +/−: debatable, *TEE* transesophageal echo

While TEE can be conducted under MAC, it usually requires GETA. TEE is superior to transthoracic echocardiography (TTE) for diagnosing paravalvular leaks and evaluating patients who have poor acoustic windows, such as obese patients.

4. Patient airway.
   A difficult airway favors the use of GETA, and a preexisting tracheostomy favors connection to mechanical ventilation and GETA.
5. Patient positioning.
6. Severe musculoskeletal disease precluding adequate positioning may require GETA.

Preoperative Considerations

Hybrid Room Setup

Compared to conventional surgical operating rooms, hybrid rooms (Fig. 29.4) have additional personnel and equipment such as fluoroscopy, back up heart-lung machine, setting for TAVR equipment, anesthesia machine, and echocardiography machine. The hybrid OR bed is narrower than that in the conventional OR and has different bed controls, which are operated by the interventional team. The anesthesia team must be aware of these factors before the procedure.

Fig. 29.4 A depiction of a hybrid operating room setup for the TAVR procedure

Radiation Exposure Preparedness

Lead aprons must be available, given the team members' exposure to radiation throughout the procedure. Some institutions monitor the level of workers' radiation exposure annually via dosimeters attached to the lead aprons. Thyroid lead shields should also be available.

Patient-Related Issues

Preoperative laboratory test results, vital signs, and radiological and echocardiographic reports should be reviewed. The anesthesia technique to be used should be chosen at the preoperative interdisciplinary TAVR conference. Patient's nil per os (NPO) status should be confirmed. Beta blockers, statins, and antiplatelet medications should be continued through the day of the procedure. The patient's electrocardiogram should be examined carefully for evidence of preexisting cardiac conduction abnormalities, such as type II heart block, which may increase the need for placement of a permanent pacemaker. If placement of a pacemaker is anticipated, an internal jugular vein transducer for transvenous pacing can be placed. Alternatively, the pacemaker can be implanted at the conclusion of the TAVR. In those cases, the subclavian area should be surgically prepared at the start of the case, with skin cleansing and sterile draping; the anesthesia provider should not attach anesthesia equipment to that area. A temporary pacemaker and defibrillation pads should be available before the patient arrives to the OR. Four units of packed red blood cells should be cross-matched and ready for emergent transfusion in the event of catastrophic bleeding.

Medication Dispensing

A conventional cardiac OR setting is prepared including inotrope and pressor infusions.

Intraoperative Considerations

Monitoring

Standard American Society of Anesthesiologists monitors, arterial line, and defibrillation pads are placed routinely. Central venous access may be obtained for administering vasoactive agents, although in many patients, large-bore peripheral intravenous access is adequate. A pulmonary artery catheter is not routinely placed.

Patient Positioning

The patient is placed supine with both arms tucked at the sides. Care should be taken to avoid kinks in the intravenous tubing.

Anesthetic Techniques

MAC

For MAC, dexmedetomidine, propofol, or a combination of sedative agents can be used. Dexmedetomidine can be started by administering a bolus of 1–6 μ/kg for 10 minutes followed by an infusion of 0.2–0.5 μ/kg/hr. A nasal cannula and an end-tidal carbon dioxide catheter are attached to the patient to assess the adequacy of spontaneous ventilation. Narcotics are usually not required. In patients who are prone to airway obstruction, a BiPAP mask and machine can be used. High-flow nasal cannula or high-flow therapy can be used in patients with pulmonary hypertension and chronic obstructive pulmonary disease for supplementation with heated humidified oxygen (>20 L/min); this technique reduces the patient's labor of breathing and alleviates respiratory distress.

GETA

The key for using GETA is the use of short-acting anesthetic agents and muscle relaxants, with the plan for endotracheal extubation in the OR.

Induction

Induction has the same hemodynamic goals as in any critical AV stenosis i.e., augmentation of the preload and afterload, and maintenance of a regular rhythm without tachycardia.

Maintenance

Age-adjusted minimum alveolar concentration (MAC) of fast-acting inhalation agents, such as sevoflurane is used. Propofol infusion can also be used as an alternative to the inhalation agents.

TEE Placement

A major advantage of using GETA is the ability to conduct TEE examination without patient discomfort. GETA enjoys all the advantages of TEE compared with TTE used in MAC in the form of:

(a) Adequate acoustic windows.
(b) Guidance of valve deployment.
(c) Real-time confirmation of successful valve deployment.
(d) Immediate diagnosis and quantification of paravalvular leaks.
(e) Immediate diagnosis of complications: pericardial tamponade, ventricular rupture, and valve malposition.

Emergence

In the OR, the timing of extubation is determined according to the adequacy of the patient's ventilation and oxygenation, temperature, and hemodynamic stability, with reversal of muscle relaxation.

TAVR Procedure

The reader is encouraged to watch the following videos on the links below to learn more about the procedure:

https://www.youtube.com/watch?v=JrrPwAs2pmM

https://www.youtube.com/watch?v=jJm7zBcN6-M

Operative Technique—Simplified

The steps of the TAVR procedure are briefly summarized:

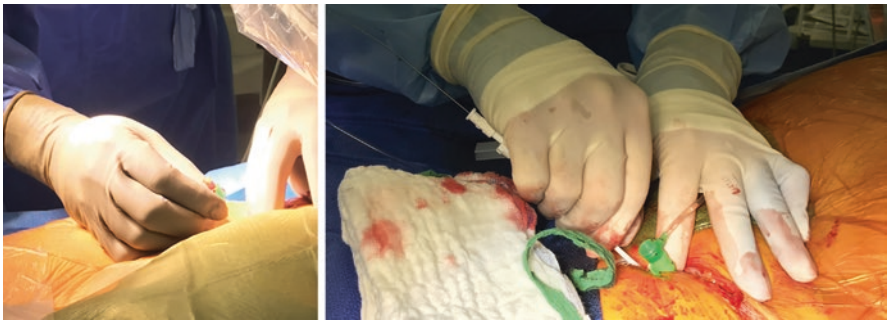

Fig. 29.5 Images of the interventionalist achieving venous, *left*, and arterial access, *right*

Fig. 29.6 Interventionalist using Perclose ProGlide® arterial closure system

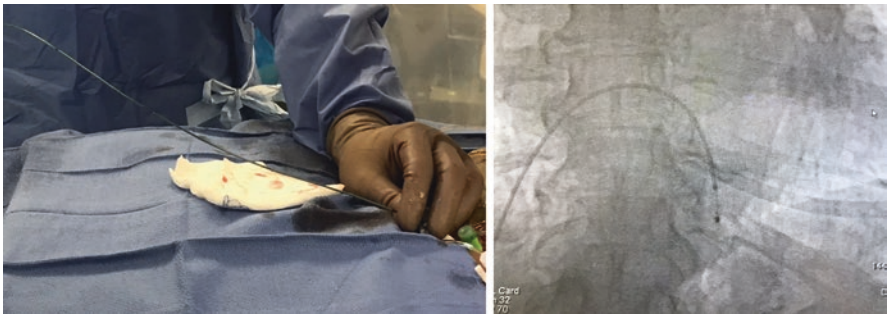

Fig. 29.7 Interventionalist introducing pacing wires, *left*, with confirmed position in RV apex by fluoroscopy, *right*

1. The interventionalist provides fluoroscopic/ultrasound-guided venous and arterial access -- usually one arterial access site for the valve deployment device and another arterial access for injection of contrast media (Fig. 29.5).
2. Two Perclose ProGlide® arterial closure systems for use at the access site through which the valve will be delivered (Fig. 29.6).
3. A pacing wire is introduced into the right ventricle via the femoral vein or internal jugular vein. The wire position is confirmed by fluoroscopy and the pacer is tested for capture (Fig. 29.7).
4. A pigtail catheter is advanced to the noncoronary cusp of the aortic valve. The catheter is used for injecting contrast material to help in positioning and deploying the prosthetic valve.
5. The stenotic aortic valve is crossed with a straight-tip wire. A pigtail catheter is then advanced into the left ventricle, and an extra stiff wire is positioned in the ventricle.
6. Unfractionated heparin (100 U/Kg) is administered to achieve anticoagulation.
7. After 3 minutes, the activated clotting time is checked, with a goal >270 seconds.
8. If balloon valvuloplasty is performed, rapid pacing is needed (Fig. 29.8).
9. The transcatheter valve is advanced across the aortic valve. Valve position is confirmed with the aid of angiography and fluoroscopy. If a Sapien valve is used, rapid ventricular pacing is needed, but pacing is not required with the CoreValve.
10. The TAVR valve is deployed (Fig. 29.9).
11. The valve position is confirmed, and complications are assessed with the aid of angiography and fluoroscopy, or TTE/TEE if needed.
12. Closure.
13. Reversal of anticoagulation.

Approaches

Transfemoral approach.

Fig. 29.8 A screen image of operating room monitor showing RVP and hemodynamic

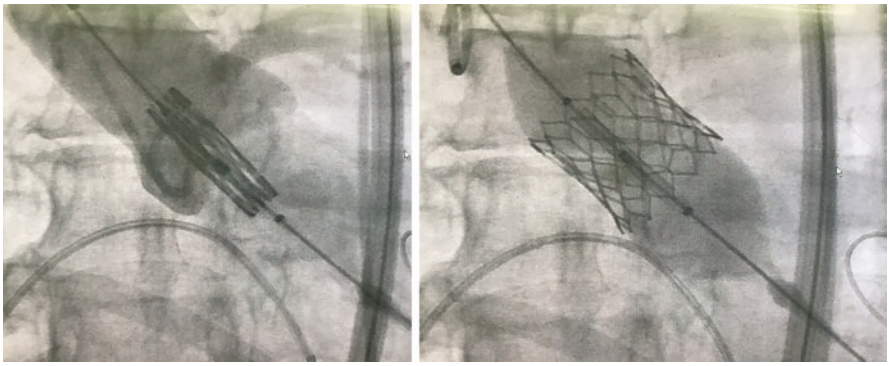

Fig. 29.9 Fluoroscopic images of valvuloplasty, *left*, and deploying a Sapien valve with balloon inflation, *right*

The transfemoral approach is the most commonly used approach unless there are iliofemoral vascular anatomical abnormalities or severe calcification and plaques.

Other approaches.

Transapical: via a left anterolateral thoracotomy primarily for SAPIEN valve.

Transaortic: via right anterior thoracotomy or mini-sternotomy. Can be used for SAPIEN and CoreValve prostheses.

Subclavian: via a cut down at the deltopectoral groove. It is used primarily for CoreValve.

Transcarotid: least commonly used approach. Used mainly for the CoreValve. Carotid perfusion is maintained via a femoro-carotid bypass shunt. The carotid puncture site is subsequently closed with a pericardial patch.

Transcaval: via the femoral vein to the inferior vena cava then to the aorta via cavoaortic fistula that is later closed with an Amplatzer device.

Types of Prosthetic Valves

The FDA-approved bio-prostheses used in TAVR are the SAPIEN and the CoreValve (Table 29.4, Figs. 29.10 and 29.11).

Rapid Ventricular Pacing (RVP)

A right ventricular wire is introduced via the femoral vein or internal jugular vein to rapidly pace the heart. RVP is used to create a still heart with temporary cessation of ventricular output, which allows for retrograde balloon valvuloplasty and valve deployment. Prior to RVP, the pacer settings should be at VOO. A test for ventricular capture should be performed, and the rate setting should be 180–220 bpm. Before RVP is instituted, the systolic blood pressure should be supported pharmacologically and adequate intravascular volume achieved, as inadequate volume can result in severe hypotension.

Table 29.4 TAVR valves approved for use in the US

Valve	Edwards SAPIEN	Medtronic COREVALVE
Design and delivery	Balloon expandable, trileaflet, bovine pericardial tissue leaflets attached to a cobalt chromium frame	Self-expandable, nitinol frame, delivered by a catheter on a guidewire
Approach	Femoral, apical, axillary, aortic	Femoral, axillary, subclavian
Sizes (mm)	20, 23, 26, 29	23, 26, 29, 31
Types available in US	Edwards SAPIEN XT Edwards SAPIEN 3	COREVALVE EVOLUTE R
Outcomes	The rates of pacemaker implantation, embolization, and reimplantation are lower than the COREVALVE	More pacemaker implantation Higher rate of embolization Higher incidence for reimplantation

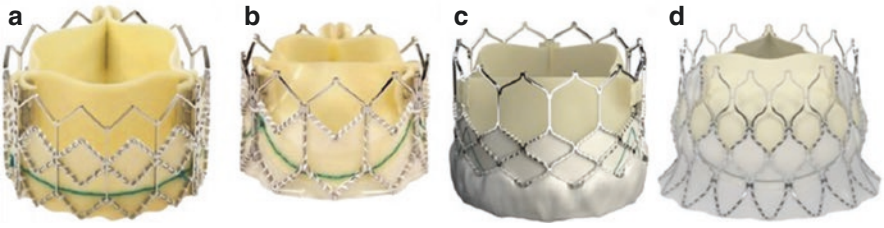

Fig. 29.10 Different models of Sapien valve (A); Sapien XT valve (B); Sapien 3 valve (C), Centera valve (Edwards Lifesciences) (D)

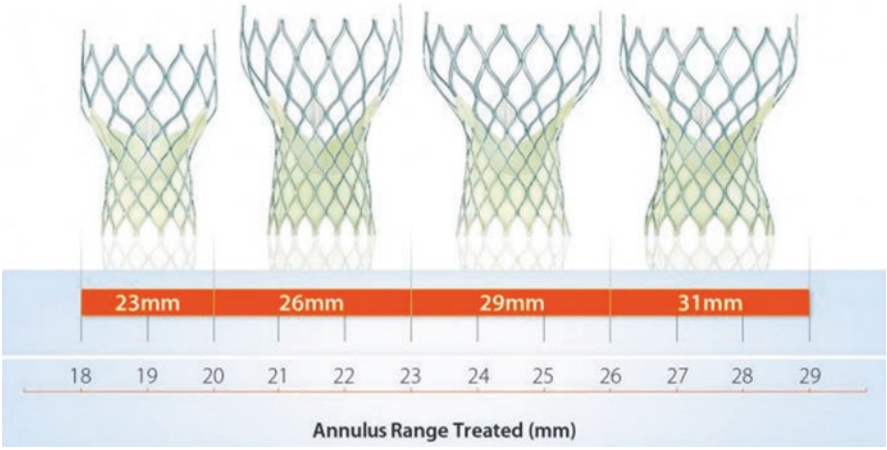

Fig. 29.11 Models of Medtronic CORE-VALVE; sizes and corresponding annular diameters

Anticoagulation

Unless contraindicated, heparin is given at a dose of 100 units/kg to achieve and maintain an activated clotting time of 250–300 seconds.

Balloon Valvuloplasty and Valve Deployment

After RVP is achieved and spontaneous (if possible) or mechanical ventilation (in case of GETA) is ceased, balloon valvuloplasty is performed. The valvuloplasty dilates the native aortic annulus to accommodate the prosthesis. After confirmation of the balloon position by fluoroscopy, RVP is terminated, and mechanical ventilation is resumed. Small boluses of a vasopressor may be needed to maintain blood pressure. If asystole develops, ventricular pacing is resumed at a regular rate, and ventricular function is assessed by echocardiography. The implantation team should be notified of persistent hemodynamic instability or new-onset EKG changes. After successful balloon valvuloplasty, RVP and ventilation hold are repeated for valve deployment.

Post-Deployment and Assessment of the New Valve Function

Successful placement of the valve usually results in a pronounced increase in systolic blood pressure, which can be treated with a bolus of nitroglycerin. It is important to check for the following after valve deployment:

1. Valve position.
2. Valve insufficiency.
3. Presence of and quantification of paravalvular regurgitation.
4. Leaflet mobility.
5. Mitral regurgitation.
6. Valve gradients.

Reversal of Anticoagulation

After confirmation of valve position and withdrawal of the deployment devices, heparin is reversed by the administration of protamine to restore activated clotting time to baseline values.

Intraoperative (Early) Complications

Predictors of death that may occur during and immediately after TAVR are listed in Table 29.5.

Early and late complications after TAVR are listed in Table 29.6; two complications are discussed here:

- Pericardial effusion may develop from rupture of the aortic annulus or left ventricle. The effusion may be self-contained and require only observation or percutaneous drainage. However, it can rapidly progress to tamponade and shock, necessitating emergent sternotomy. In that circumstance, it may be necessary to provide life support with chest compressions until the chest can be opened. Video 29.1 shows a developing pericardial effusion in the transgastric short axis view on TEE. Video 29.2 demonstrates the source of the effusion: a perforation of the left ventricle, which was easily identified after sternotomy.

Video 29.1  Pericardial effusion which occurred after deployment of a balloon expandable TAVR valve. A transgastric view on TEE is shown.

Table 29.5 Predictors of death after TAVR

Day 1 -day 30
1. Need to convert to cardiopulmonary bypass
2. Prior balloon valvuloplasty
3. Low ejection fraction (<30%)
4. Cardiac tamponade
After day 30
1. PVR >2+
2. Prior stroke
3. Chronic kidney disease

PVR paravalvular regurgitation

Table 29.6 Complications after TAVR

Immediate	Long term
Cardiac tamponade	Complete heart block
Paravalvular regurgitation	Acute kidney injury
Myocardial ischemia	Bleeding secondary to anticoagulation
Vascular access bleeding	Vascular injury
Ventricular rupture	Stroke
Valve migration	Subclinical leaflet thrombosis
Coronary obstruction with MI	

Video 29.2  Same patient as in Video 29.1. The chest has been opened. A perforation of the left ventricle, likely due to calcium in the aortic annulus and root, is present, with active bleeding clearly visible.

Videos 29.3  and 29.4  show paravalvular leaks (PVL) after Sapien valve implantation. Video 29.3 demonstrates two discreet PVLs in the deep transgastric aortic valve long axis view on TEE, as well as in 3D with color flow Doppler rendering of the PVL. Video 29.4 is that of different patient and shows a PVL in the mid-esophageal AV long- and short- axis views. The cardiac anesthesiologist should be familiar with the appearance of PVLs on TEE and should communicate this information to the proceduralist if the anesthesiologist is also the one performing the echocardiography during the procedure.

- Valve migration is a rare complication of TAVR. Figure 29.12 depicts a CoreValve that spontaneously migrated into the ascending aorta several minutes after deployment. Although the CoreValve is partially recapturable, once it is fully deployed, it cannot be returned into its delivery sheath. In this case, a second overlapping valve was deployed, which anchored the initial valve in place. The anesthesiologist must remain informed at all times about such intraprocedural events. This information will allow him/her to predict and treat complications that may arise from nonstandard approaches, e.g., vessel obstruction or valve malfunction. The anesthesiologist providing services for TAVR should be comfortable in monitoring the case not only from a hemodynamic perspective and through echocardiography but also by following the procedural fluoroscopy imaging.

Postoperative Care

After the TAVR, most patients are cared for in the cardiac intensive care unit for one night before being transferred to the floor. However, at the discretion of the multidisciplinary team, low-risk patients can be observed in a lower-acuity setting. All patients are monitored for TAVR complications, as outlined in Table 29.6, although immediate complications usually become evident in the OR. A TTE is performed prior to patient's discharge from the hospital.

Despite lack of evidence of efficacy, most patients are placed on dual antiplatelet therapy, in the form of aspirin 75–100 mg daily for life and clopidogrel 75 mg/day for 6 months. Recent evidence suggests that single antiplatelet therapy (aspirin) adequately reduces the risk of bleeding, while preventing thrombosis.

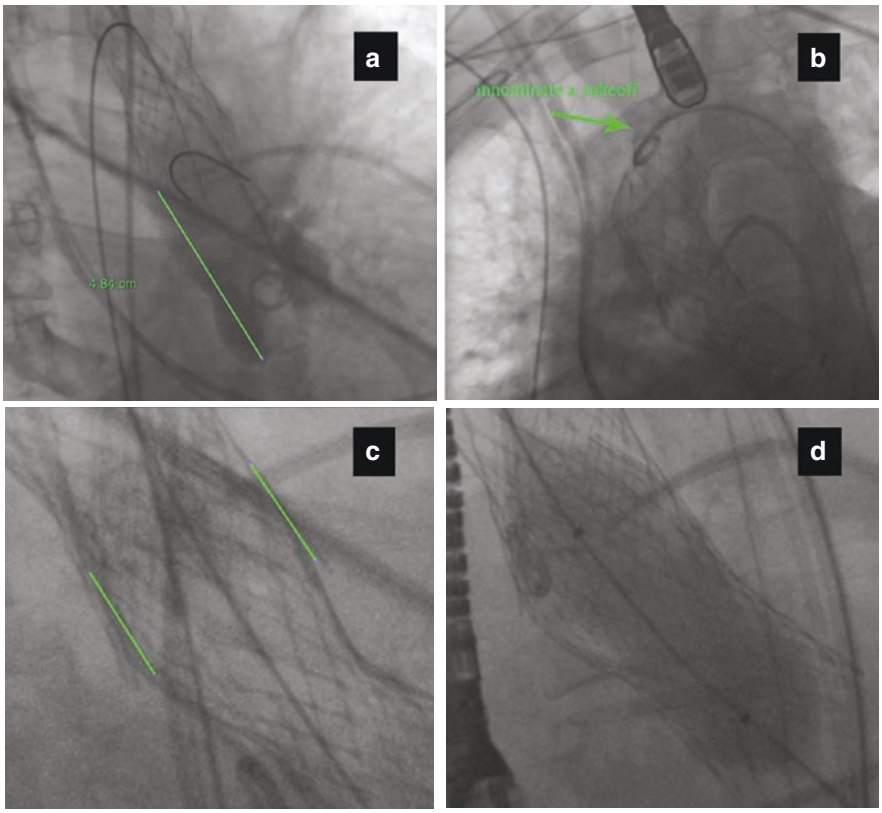

Fig. 29.12 Spontaneous displacement of COREVALVE treated with placement of a second COREVALVE. The initial valve was displaced 4.8cm proximal to the aortic valve annulus (a), with its proximal tip reaching the takeoff of the innominate artery (b). The second valve is shown in panel c overlapping the first (green lines). Panel d shows an inflated balloon used to post-dilate the second valve.

Further Reading

Transcatheter Aortic-Valve Replacement with a Balloon-Expandable Valve in Low-Risk Patients. N Engl J Med. 2019;380(18):1695–705.

Transcatheter Aortic-Valve Replacement with a Self-Expanding Valve in Low-Risk Patients. N Engl J Med. 2019;380(18):1706–15.

Franco A, Gerli C, Ruggeri L, Monaco F. Anesthetic management of transcatheter aortic valve implantation. Ann Card Anaesth [serial online] 2012 [cited 2018 Mar 7]; 15:54–63. Available from: http://www.annals.in/text.asp?2012/15/1/54/91484

Guarracino F, Baldassarri R. The anesthetic management of transcatheter aortic valve implantation. Seminars in Cardiothoracic and Vascular Anesthesia. 2016. 20(2), 141–146. Retrieved March 5, 2018, from: http://journals.sagepub.com/doi/abs/10.1177/1089253215606220

Muntazar, Solina, Awad. Transcatheter aortic valve replacement chapter. In: "Everything you need to know. Out of the operating room and minimally invasive cardiothoracic procedures" banks, Zaky, editors. Nova Science Publishers: Inc. p. 2018.

Anesthetic Considerations for Transcatheter Mitral Valve Repair with the MitraClip Device

30

Matthew B. Barajas and Isaac Y. Wu

Any sufficiently advanced technology is equivalent to magic.

—Arthur C. Clarke

Key Points
- Indications
- MitraClip System Components
- Anesthetic Management
- Procedural Steps
- Procedural Complications

Introduction

The MitraClip® (Abbott Vascular, Santa Clara, California) is a transcatheter mitral valve repair device that is used to treat mitral regurgitation (MR). The MitraClip produces an edge-to-edge leaflet repair mimicking the surgical repair known as the Alfieri stitch or Bow-Tie repair. In the surgical repair the free edges of corresponding anterior and posterior mitral valve (MV) leaflet segments are sutured together to create a double-orifice MV. The MitraClip utilizes two sets of arms and graspers to capture the MV in a similar fashion to the suture-based repair (Fig. 30.1).

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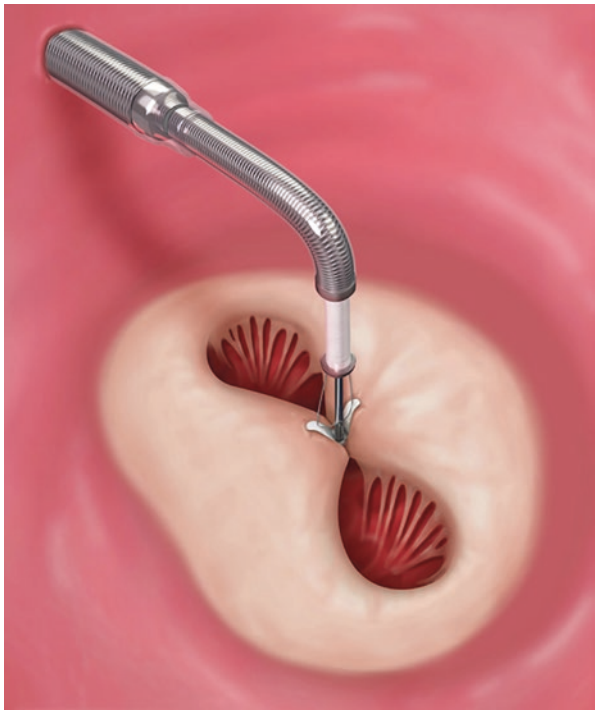

Fig. 30.1 Transcatheter Mitral Valve Repair with the MitraClip Device as viewed from the left atrium

Indications

The MitraClip is used to treat both degenerative MR (DMR) and functional MR (FMR). In 2011, the Endovascular Valve Edge-to-Edge Repair Study (EVEREST) II showed that the MitraClip procedure significantly reduced MR severity, although not as effectively as conventional surgery. Despite this, the MitraClip was associated with a similar improvement in functional outcomes and increased safety when compared to surgical therapy. This study was done in a population with predominantly DMR. Based in part upon this trial, the United States Food and Drug Administration (USFDA) approved the MitraClip procedure for commercial use in patients with DMR. In 2018, the Cardiovascular Outcomes Assessment of the MitraClip Percutaneous Therapy for Heart Failure Patients with Functional Mitral Regurgitation (COAPT) trial demonstrated that MitraClip therapy in conjunction with guideline-directed medical therapy (GDMT) reduced rehospitalization for heart failure and mortality when compared to GDMT alone. This study was done in heart failure patients with 3–4+ FMR. Subsequently, the USFDA expanded the approved use of the device to include patients with FMR. The MitraClip is currently indicated for patients who meet the following criteria:

- DMR ≥3+: symptomatic, prohibitive risk for traditional open surgical approach, existing comorbidities do not preclude the expected benefit from MR reduction.
- FMR ≥3+: symptomatic despite maximally tolerated GDMT, left ventricular ejection fraction ≥20% and ≤50%, left ventricular end systolic dimension ≤70 mm.

MitraClip use is not recommended in patients with intracardiac, inferior vena cava or femoral vein thrombus, rheumatic MV disease, active MV endocarditis, or in patients who cannot tolerate procedural anticoagulation or post-procedural antiplatelet therapy. A multidisciplinary heart valve team that includes a cardiac surgeon and cardiologist experienced in managing mitral valve disease should evaluate all prospective MitraClip patients. Additionally, a cardiac anesthesiologist or dedicated echocardiographer with expertise in MitraClip imaging should provide input on the suitability of mitral valve anatomy for clip placement. Finally, a heart failure cardiologist should evaluate all patients with functional MR who are being evaluated for MitraClip therapy.

MitraClip System Components

The MitraClip System is comprised of the Steerable Guide Catheter (SGC) and the Clip Delivery System (CDS) (Fig. 30.2). The SGC has a dilator that is used to advance it into the femoral vein, up the IVC, through the right atrium and into the

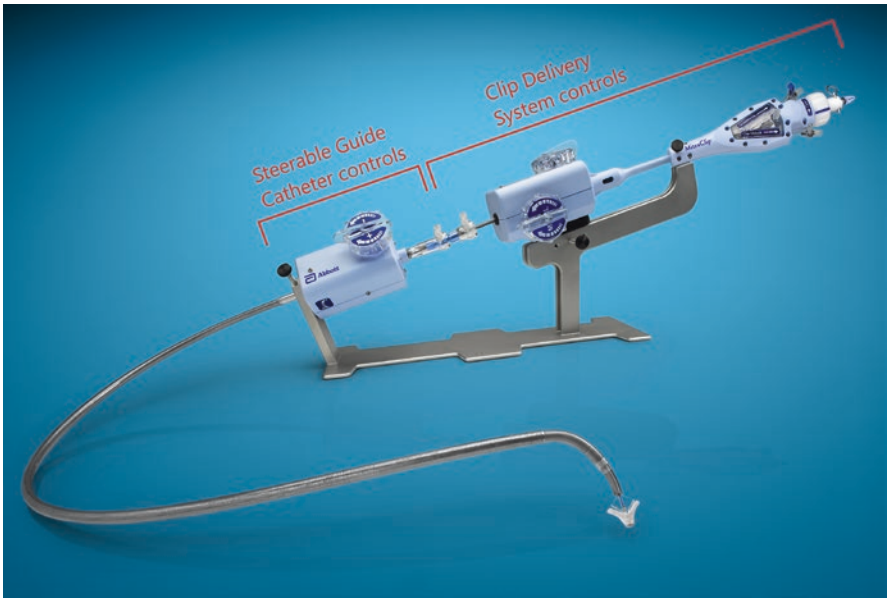

Fig. 30.2 The MitraClip System. The clip delivery system passes through the steerable guide catheter. The clip itself is attached to the distal end of the clip delivery system. (Reprinted with permission from Abbott Vascular)

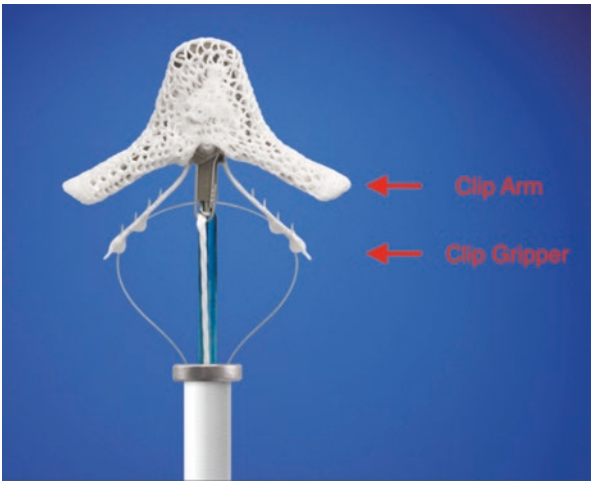

Fig. 30.3 MitraClip showing the clip arms and grippers, between which the mitral valve leaflets are captured. (Reprinted with permission from Abbott Vascular)

left atrium (LA) through the transseptal puncture (TSP) site. Once in the LA, the SGC is used to help position the CDS in the proper location and orientation above the MV. The CDS consists of three parts: a delivery catheter, steerable sleeve, and the MitraClip itself. The CDS enters the LA through the SGC. The CDS is also used to make adjustments in position and orientation relative to the MV. The MitraClip has grippers and arms (Fig. 30.3). The anterior MV leaflet is captured between one arm and gripper and the posterior MV leaflet is captured between the remaining arm and gripper.

Anesthetic Management

The MitraClip procedure is performed in high-risk patients with significant MR. Given the high-risk nature of these patients, a careful anesthetic plan is formulated with the patient's cardiac disease in mind. While severe MR will influence the induction strategy, it may not be the only pertinent pathology to consider and the anesthetic plan should be tailored to each patient. Medication choices should allow for extubation at the conclusion of the procedure. A pre-induction radial arterial line and defibrillator pads are commonly placed. General anesthesia with endotracheal tube placement is used to facilitate transesophageal echocardiographic (TEE) guidance for the procedure. Central venous access may be helpful in maintaining hemodynamic stability throughout the case.

The MitraClip procedure is performed in the catheterization laboratory or hybrid operating room, in a location that is capable of handling cardiopulmonary bypass and conversion to open heart surgery, if needed. The catheterization laboratory has the benefit of being a more familiar procedural environment for the interventionalists. On the other hand, critical personnel and resources, such as additional anesthesiologists, perfusionists, and cardiopulmonary bypass equipment, are typically more readily available in the hybrid operating room.

Due to their high-risk profile, patients have traditionally been monitored in the intensive care unit following MitraClip implantation. However, there are no standardized guidelines, and post-procedural recovery strategies vary between institutions. At some institutions select patients who are deemed to be more stable are monitored in the catheterization laboratory recovery unit or post-anesthesia care unit following the procedure.

Procedural Steps

Successful MitraClip implantation requires active communication between the interventional cardiologist, echocardiographer, and anesthesiologist. At many centers the anesthesiologist also performs the TEE guidance. Thus, it is important to know the procedural steps. The MitraClip procedure can be broken down into six main steps. While the specifics of each step may vary from institution to institution, the overarching goals should remain the same.

Step 1 – Transvenous, Transseptal Access into the LA

The first step is to access the LA via the interatrial septum (IAS). The femoral vein is accessed and a transseptal puncture (TSP) is made from the right atrium through the fossa ovalis into the LA. A well-placed TSP is critical because it gives the interventional cardiologist the necessary space to safely and effectively maneuver the CDS within the LA. During this step, heparin is administered to ensure adequate anticoagulation status throughout the procedure. An activated clotting time >250 seconds is maintained to prevent thrombus formation. In order to minimize the risk associated with TSP, a small dose of heparin (~2000–3000 units) may be administered before TSP and full heparinization can be obtained after LA access has been obtained without complication.

Step 2 – Advancing the MitraClip System into the LA

Once the LA has been accessed without complication, the larger SGC (24 Fr) is advanced through the femoral vein into the LA. The CDS is then inserted into the LA through the SGC.

Step 3 – Positioning of the MitraClip in the LA

Positioning and orientation of the clip are optimized in the LA before advancing into the LV due to the risk of subvalvular apparatus injury associated with clip manipulation in the LV. While proper positioning is dependent on the specific MV anatomy, in most cases the clip is positioned above the origin of the regurgitant jet.

The clip is then aligned so that the arms are perpendicular to the line of coaptation of the mitral valve leaflets.

Step 4 – MitraClip Leaflet Grasping

Once in the proper position and orientation, the clip, with arms extended, is advanced across the MV into the LV. Position and orientation are reconfirmed once in the LV and the clip is retracted until the anterior and posterior MV leaflets fall into the clip arms. The grippers are lowered, and the clip is partially closed to grasp the leaflets. If grasping of the MV leaflets is difficult, certain maneuvers, such as breath holds, slowing the heart rate with adenosine, or rapid pacing, can be utilized to alter MV leaflet positioning and facilitate grasping. TEE imaging is used to verify successful grasping of both MV leaflets and confirm stability of the MitraClip.

Step 5 –MitraClip Deployment

If the MR reduction is satisfactory on TEE assessment without resultant mitral stenosis (MS), the clip is released. Alternatively, the clip can be opened and repositioned if it is thought that a greater reduction in MR can be achieved. Assuming an adequate MV area, additional clips can also be placed if the first clip does not achieve an adequate reduction in MR. During this step, the anesthesiologist should bring the patient's vital signs as close as possible to baseline (awake) values. This allows for assessment of the relevant amount of residual MR or the presence of any mitral stenosis (MS) before the decision to deploy the clip is made.

Step 6 – Withdrawal of the MitraClip System

Upon completion of the procedure, the CDS and SGC are removed and hemostasis of the femoral vein is achieved. After removal of the delivery system, a residual ASD will remain. This ASD is not routinely closed. Protamine administration may be considered for heparin reversal to facilitate hemostasis.

Procedural Complications

It is critical to be able to anticipate and be prepared for the potential hemodynamic changes and complications associated with procedural maneuvers. As such, it is important for the anesthesiologist to know the potential complications and when to expect them.

Table 30.1 summarizes potential intraprocedural complications:

For many of these complications, the cardiac anesthesiologist is responsible for resuscitating the patient with fluid and blood products and supporting the patient's

Table 30.1 MitraClip® procedural complications

Complications	Commonly associated procedural steps	Prevention/treatment
Vascular injury	Steerable guide catheter insertion and removal	Surgical repair; manual pressure; supportive and resuscitative measures
Aortic injury/atrial injury/pericardial effusion/ tamponade	Transseptal puncture, steerable guide catheter and clip delivery system manipulation	TEE-guided transseptal puncture and catheter manipulations; pericardiocentesis; surgical intervention; supportive and resuscitative measures
Clot formation	Entire procedure	Heparinization with goal ACT >250 seconds
Air embolism	Entire procedure	De-airing of catheters and sheaths; supportive measures
Clip entanglement in subvalvular apparatus (SVA)/injury to SVA	Clip advancement into the LV and leaflet grasping	Inversion of clip arms and retraction of clip into the left atrium; supportive measures; surgical intervention
Partial or complete clip detachment	After leaflet grasping and clip deployment	TEE evaluation of adequate leaflet capture and clip stability before deployment; additional clip placement; device retrieval
Mitral stenosis	After leaflet grasping and clip deployment	TEE evaluation of mitral stenosis before clip deployment
Persistent atrial septal defect	Following clip delivery system and steerable guide catheter removal	Usually does not require intervention; ASD closure device can be placed if needed
Bleeding	Entire procedure due to both procedural vascular access and prolonged, frequent TEE manipulation	Reversal of heparin; manual pressure/ surgical repair; supportive and resuscitative measures; possible ENT/GI consult and extended intubation for GI bleeding

hemodynamics with vasopressors and inotropes as needed. It is important for the anesthesiologist to notify the rest of the team if the patient's vital signs change abruptly or unexpectedly. Effective communication between the cardiac anesthesiologist, interventionalist, and echocardiographer is paramount in the prevention and treatment of procedural complications.

Echocardiographic Guidance

Imaging guidance for the MitraClip procedure is an institution-specific role, with institutions relying on cardiac anesthesiologists and or cardiologists for this purpose. Figure 30.4 shows a color Doppler jet consistent with severe MR before MitraClip implantation. Figure 30.5 shows a reduction in regurgitant jet area following clip placement. Finally, Fig. 30.6 shows the typical post-clip double orifice MV viewed from the left atrium. A complete discussion of TEE guidance for the MitraClip procedure is out of the scope of this chapter. However, it is important to know that TEE imaging plays a critical role in the safety and effectiveness of the MitraClip procedure.

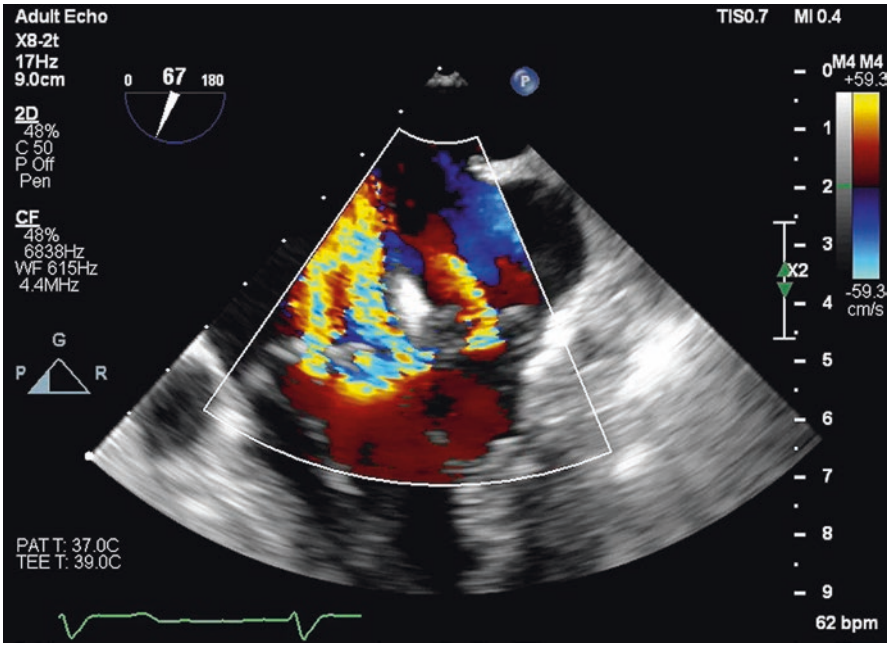

Fig. 30.4 Midesophageal mitral commissural view showing severe mitral regurgitation before clip implantation. The MitraClip is visualized just lateral to the main regurgitant jet

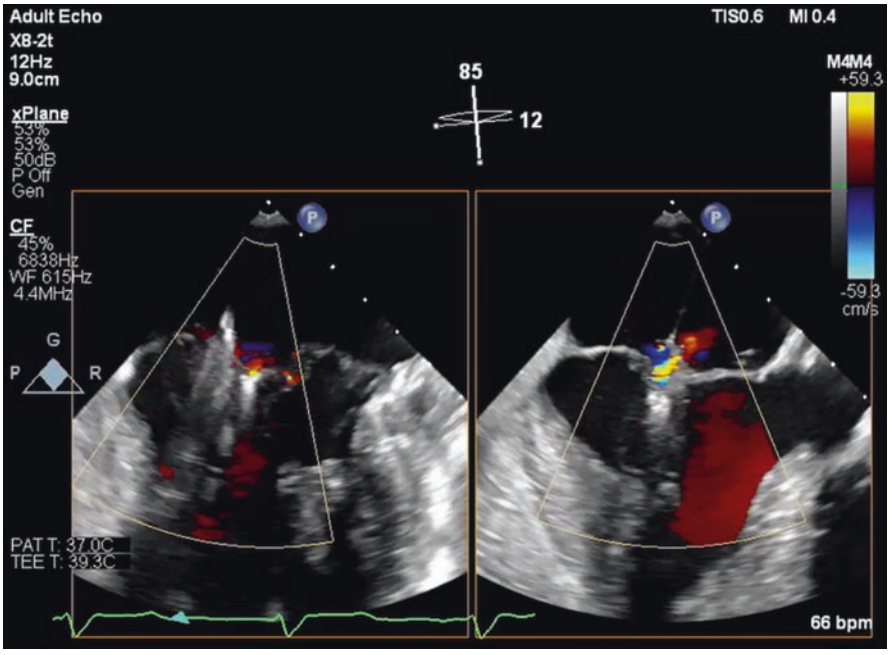

Fig. 30.5 Biplane imaging of the midesophageal mitral commissural (left panel) and long-axis (right panel) views showing reduction in the severity of mitral regurgitation after deployment of one clip and placement of a second clip

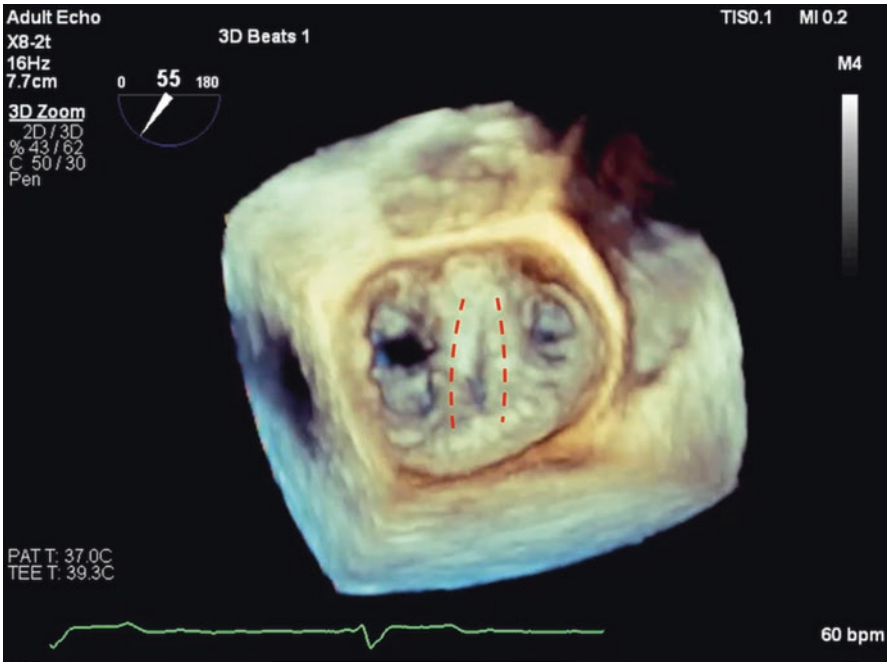

Fig. 30.6 3D en-face view of the mitral valve showing a larger lateral orifice and smaller medial orifice. A tissue bridge (dotted line) is seen in the A2-P2 region (initial clip) and a second tissue bridge (dotted line) is seen just medial to that (second clip)

As an example, mitral stenosis is a complication that can be prevented with careful echocardiographic guidance. A pre-procedural mitral valve area (MVA) ≥ 4 cm^2 is optimal for MitraClip placement, but expected post-implantation changes should also be considered. In a study by Herrmann et al., the MVA decreased by approximately 40% from baseline and the mean transmitral PG increased from 1.7 ± 0.9 mmHg to 4.1 ± 2.2 mmHg ($p < 0.05$) following MitraClip® implantation. Paranskaya et al. similarly showed a 42% reduction in valve area with one clip and a 48% reduction with multiple clips. Neuss et al. reported that a mean transmitral PG >4.4 mmHg immediately following clip placement is associated with significantly worse long-term outcomes. As such, determining MVA and transmitral PG by intraprocedural echocardiography provides critical information to guide patient management.

Clinical Pearls

- Tailor your anesthetic plan to each patient's pathology. Pre-induction arterial line placement with adequate venous access is typical for this procedure. Additionally, post-procedural extubation is commonly performed.
- Vascular injury is the most common procedural complication. Be sure that blood products are available.
- Effective communication is critical for successful MitraClip implantation. Know the steps of the procedure and potential procedural complications. Speak up if you notice any unexpected changes in the patient's vital signs.

Further Reading

1. Alkhouli M, Rihal CS, Holmes DR. Transseptal techniques for emerging structural heart interventions. J Am Coll Cardiol Intv. 2016;9:2465–80. https://doi.org/10.1016/j.jcin.2016.10.035.
2. Feldman T, Mehta A, Guerrero M, Levisay JP, Salinger MH. MitraClip therapy for mitral regurgitation: secondary mitral regurgitation. Interventional Cardiol Clin. 2016;5:83–9.
3. Feldman T, et al. Percutaneous repair or surgery for mitral regurgitation. N Engl J Med. 2011;364:1395–406. https://doi.org/10.1056/NEJMoa1009355.
4. Guarracino F, et al. Transesophageal echocardiography during MitraClip procedure. Anesth Analg. 2014;118:1188–96.
5. Stone GW, et al. Transcatheter mitral-valve repair in patients with heart failure. N Engl J Med. 2018;379:2307–18. https://doi.org/10.1056/NEJMoa1806640.

Anesthesia for Laser Lead Extraction

31

John Andriulli and Alexander Kahan

When we all think alike, then no one is thinking.

—Walter Lippmann

Introduction

In the realm of the ever-expanding cardiac implantable electrical device (CIED) market, device wires and leads are some of the most innocuous elements. The wires and leads are commonly placed under a local anesthetic with or without sedation, and, if fibrosis has not set in, they can be removed quickly.

There are 3.3 million cardiac rhythm devices in place in the United States, with 450,000 new devices implanted yearly, which equates to about 2.6 million new leads. Over time, many of these leads become defective, fractured, or infected. Accordingly, the incidence of laser-lead extraction (LLE) has dramatically increased.

Indications for Lead Extraction

With the growing number of implants and an increasing patient life expectancy, the indications for lead removal continues to increase.

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Common indications recommended by the Heart Rhythm Society for lead removal:

- Device recall.
- Lead malfunction/fracture.
- Infection.
- Thrombosis and venous occlusion.
- Chronic pain.
- Need for device upgrade.
- Redundant hardware/leads.
- Removal of magnetic resonance imaging noncompatible device.

Historic Extraction Technique

Prior to modern extraction tools were developed, leads were removed by tying weights to the ends and suspending the weights over a pulley system with the patient on bed rest in the hopes that over time these leads would come free and dislodge. At present, extraction is more sophisticated.

Preoperative Evaluation and Risk Stratification

The initial indication for placement of a CIED should be assessed and associated comorbidities evaluated; patients with a CIED may have a history of ischemic heart disease, heart failure, heart or valvular surgery, diabetes, and chronic renal failure. Patient factors and lead dwell-time can influence scar binding of the lead as well. The greatest risk factors associated with complications and long-term mortality with CIEDs extractions are female sex (4.5 x increased mortality) and extraction for infection (up to a 30 x increase in 30-day mortality). Patients who have had their leads implanted at a younger age tend to have more proliferative scarring than those who receive implants after the sixth decade of life. Also, patients who are on dialysis for end-stage renal disease may have significant calcific adhesions of intravascular leads, which adds complexity to their removal. Patients who have received long-term steroids or immunosuppressive drugs may have less binding or adhesions, making lead removal easier.

The type of lead also can influence the ease or difficulty of extraction. Implantable cardioverter defibrillator (ICD) leads differ from pacing leads; defibrillator leads have internal cables and external shock coils. Given their larger surface area and presence of an external coil, ICD leads tend to develop more intravascular adhesions, which may make extraction more difficult. The presence of a superior vena cava (SVC) coil can lead to potential difficulties and complications during removal.

Pacing leads are less complex than ICD leads. The determining factor influencing the difficulty of extraction is whether the inner coils are coaxial or coradial. Coradial leads tend to stretch when force is applied during extraction, and tensile strength may be less than that of coaxial leads. These features predispose coradial leads to pull apart or break off, leading to incomplete extraction of the leads. Incomplete extraction is readily identifiable on transesophageal echocardiography (TEE).

In preoperative evaluation of the patient, the baseline functional status, cardiac function, EKG, complete blood count, coagulation panel, type and cross match and metabolic panel should be assessed, and a physical examination focused on signs of heart failure should be performed. An echocardiogram and anterior-posterior and lateral chest radiograph may reveal the lead and its location and any deformity, thrombosis or vegetation. The presence of baseline pericardial fluid should be noted, as increasing amounts developing intraoperatively are a stark indication of a major complication.

If the lead is being removed because of infection, bacteremia may be present. Evidence supports that treatment for occult gram-positive bacteremia with antibiotics alone without removal of the device and leads has a 66% one-year mortality. It has recently been shown that capping rather than extracting abandoned leads may increase long-term infection risk.

The patient's most recently administered antibiotics should be reviewed to avoid inappropriate perioperative administration. While septic shock should be managed medically prior to proceeding with lead extraction, bacteremia can result in a systemic inflammatory response syndrome or sepsis and shock intraoperatively.

For patients who are dependent on the pacemaker lead that is to be removed, a temporary pacemaker is usually placed. In our institution, the electrophysiologist prefers to use the femoral vein for this approach. Any central vein can be used for this purpose, but consulting with the electrophysiologist, surgeon, and perfusionist should precede placement. The lead extraction process could dislodge a temporary pacemaker placed through the SVC and placing the temporary pacemaker through the same subclavian vein as the indwelling CIED may be technically challenging. Additionally, the electrophysiologist may opt to reserve the right femoral vein for the placement of a temporary Bridge Occlusion Balloon® (Phillips), although both devices can be placed through the same site concurrently.

Preprocedural Set-up

Laser lead extractions (LLE) are performed by a multidisciplinary team that includes an electrophysiologist, a cardiac anesthesiologist, a cardiothoracic surgeon, and a perfusionist. A primed cardiopulmonary bypass pump is on standby. As such, the extractions are done in an operating room or a hybrid room, complete with well-trained support staff, including cardiothoracic nurses and equipment capable of accommodating a crash onto cardiopulmonary bypass and open-chest conversion.

With these precautions, the procedure carries a major adverse event risk of only 1–1.5% and a 0.3–0.5% risk of death, which are lower rates than those with routine atrial fibrillation ablation. However, given the potential catastrophe of an SVC tear, the patient is prepped as a possible crash open-chest case with a cardiac surgeon on standby.

Anesthesia and Intraoperative Monitoring

When the patient enters the room, a standard institutionally defined "time-out" and discussion should be conducted with all involved parties. Care should be taken to confirm the presence of all vital personnel and equipment, the availability of blood,

and the plan for bailout in the event of a catastrophic vascular or cardiac injury. If the patient has never had open-heart surgery, the on-call surgeon may opt for doing a median sternotomy, whereas if the case is a redo chest, the surgeon may prefer a thoracotomy approach, should cardiac surgery become necessary.

If the patient has an AICD, the device should be interrogated, and the patient should be questioned for a history of possible device firing and precipitating events. While disabling such a device necessitates placement of external defibrillation pads as backup, all patients undergoing LLE regardless of device type should have pads placed in preparation of a cardiac arrest or malignant arrythmia precipitated by lead manipulation.

In addition to standard American Society of Anesthesiologists monitors, a radial arterial line can be placed before induction for use if needed for hemodynamic management, or a femoral arterial line can be placed after induction by the electrophysiologist for use in hemodynamic management and for providing a quick percutaneous bypass cannulation site. Heparin availability should be confirmed should the need for CPB arise.

The anesthetic management of LLE patients is tailored to each patient's risk factors. General endotracheal anesthesia is the preferred anesthetic, and it is induced with consideration of the patient's underlying pathology; this approach ensures a motionless patient with optimized surgical conditions. After the induction of general endotracheal anesthesia, the electrophysiologist places a large-bore central line, most commonly in the femoral vein, for resuscitative purposes. In the event of injury to the subclavian vein or SVC, having lower body large-bore access avoids the potential for extravasation with an upper-body central line. A Foley catheter and a TEE probe should be placed for careful monitoring of fluids and facilitating the swift diagnosis of hemodynamic instability.

Imaging

Intraoperatively, various imaging modalities are used to guide the extraction procedure, decrease procedure time, and improve outcomes, while providing a tailored strategy for each instrument to be used.

- Ultrasound: The electrophysiologist will often place a temporary pacemaker and/or rescue balloon introducer in the right femoral vein under ultrasound guidance. The introducer is often of a 9–12-F bore, which may suffice for large-bore lower body intravenous access.
- TEE is performed after induction of anesthesia to determine the heart's baseline functional status, presence, location, and size of vegetations, presence and severity of tricuspid regurgitation, and the presence of preexisting pericardial effusion and intracardiac shunts. Care should be taken to address intracardiac shunts prior to lead manipulation to avoid precipitating a paradoxical embolization. Also, real-time three-dimensional TEE can help in visualizing lead alignment and assessment of binding sites to help in choosing the optimal extraction strategy (Fig. 31.1).

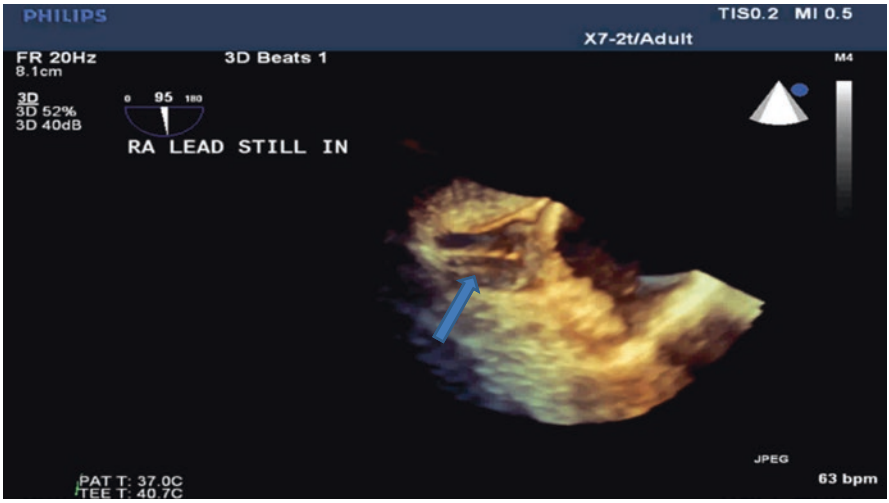

Fig. 31.1 Images of 3D TEE showing lead (blue arrow) in the superior vena cava for assessment of binding sites

- Fluoroscopy: fluoroscopy creates live images that help guide manipulation of the instruments during the extraction. Also, fluoroscopy helps in identifying intracardiac binding sites.

Procedure Techniques

Leads can be extracted by several techniques. Currently, removal is achieved by either explantation or extraction. Explantation of a lead is the simple removal with manual traction without the use of any powered or mechanical tools or lead-locking stylet; any use of these tools is defined as extraction. The goal of lead extraction is to safely and successfully remove the target lead or leads.

Stepwise Approach

1. The case progresses with dissection of the device pocket and identification and disconnection of the lead to be removed (Fig. 31.2).
2. Simple traction through the insertion vein is applied.
3. If simple traction is not successful, traction with lead-locking stylet (Liberator Universal Locking Stylet®, Cook Medical, Bloomington, IN) is used. In this technique, a wire is placed in the lumen of the lead so that traction can be applied.
4. If the lead-locking stylet technique is not adequate, a circumferential mechanical sheath that can cut through crystalized calcium or a laser sheath is placed around the lead, and laser energy is used to lyse encapsulating fibrosis and scarring. Before employing the mechanical or laser sheath and applying traction on the lead, the availability of 4 units of packed red blood cells in the room should be verified. The case usually proceeds uneventfully from this point forth (Fig. 31.3).

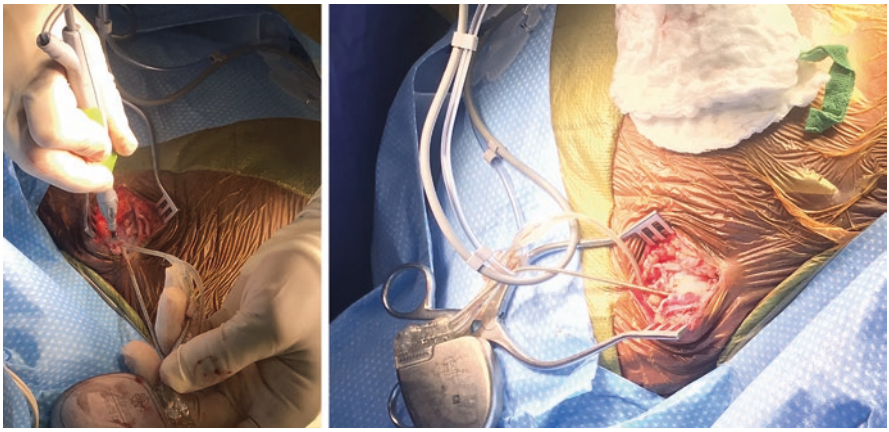

Fig. 31.2 Images of Left: pocket dissection. Right: exposure of targeted lead for extraction

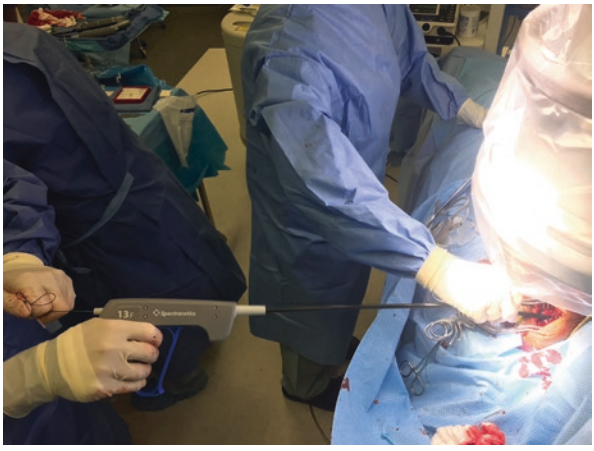

Fig. 31.3 Image of mechanical extraction tool (TightRail™)

During surgical manipulation, the TEE probe may interfere with fluoroscopic images and may need to be withdrawn into the upper esophagus. Intracardiac echocardiography may be the preferred modality of echocardiographic guidance for those who are comfortable with the technique.

Adverse Events

Sudden hemodynamic instability may occur. It is most often caused by transient arrhythmia or inversion of the right ventricle from manipulation, but a major vascular hemorrhage, as from the superior vena cava or innominate vein, should be considered. Also, cardiac injury, such as lacerations of the right atrium or perforation of the right ventricle and resultant tamponade and hemorrhagic shock may occur.

Patients who have a CIED often are quite sick, and hemodynamic instability in them can be due also to underlying depressed heart function, sepsis, thromboembolism, or dislodgement of a temporary pacemaker. Treatment of any instability should be communicated with the electrophysiologist, and care should be taken that more dire complications are not being masked by transient treatment.

Other complications of lead extraction include damage to the tricuspid valve and worsening tricuspid regurgitation, which can result from instrumentation and the extraction itself. Stroke can occur from paradoxical embolism resulting from dislodgement of vegetations from the tricuspid valve. A large vegetations dislodgement can cause acute pulmonary thromboembolism if large enough, will lead to ventilation-perfusion mismatch and hypoxemia with acute right ventricular compromise.

Rescue Protocol

Advances in technology, mainly the use of a bridge occlusion balloon, have resulted in a remarkable reduction in intraoperative death from SVC tears. The balloon can be inflated in the SVC to tamponade bleeding from injury and stabilize the hemodynamics while the surgical repair is completed. Initial data indicates that survival with an appropriately applied balloon can approach 90% as compared to 50% without balloon use (Fig. 31.4).

Should a major vascular or cardiac injury occur with lead extraction, the standby cardiac surgeon has 5–10 minutes to open the chest and control the hemorrhage before most of the intravascular volume is lost and cardiac arrest ensues. In the case of a redo chest, the surgeon may proceed with a median sternotomy or a thoracotomy. Additional assistants may be required for resuscitative efforts and possible TEE guidance of a rescue balloon.

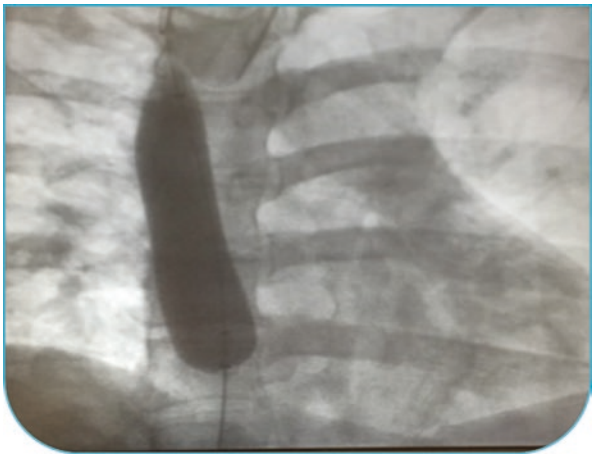

Fig. 31.4 Image of fluoroscopy showing Bridge Balloon inflated in the SVC

Postoperative Management

After uncomplicated extraction, the balloon introducer is removed, and hemostasis is achieved. If the patient remains pacemaker-dependent and did not have permanent leads replaced or reconnected for pacing support (most likely due to systemic infection), a temporary pacing lead is usually placed via the internal jugular vein prior to removing the temporary femoral pacing wire.

The patient is most often extubated in the operating room or post-anesthesia care unit. Pain is typically minimal and well controlled with a combination of local anesthetic infiltrated along the surgical pocket and non-opioid analgesics such as acetaminophen. Once criteria are met for discharge from the unit, the patient can be sent to a monitored telemetry floor or a critical-care bed if deemed necessary.

If the procedure required emergent opening of the chest or crashing onto cardiopulmonary bypass, the patient is kept intubated and discharged to the cardiothoracic intensive care unit, often with chest tubes in place. If there is no evidence of additional bleeding, tamponade, or major thromboembolic insult and the patient meets all necessary criteria, they can be safely extubated and discharged from the intensive care unit to a step-down unit.

Further Reading

1. Bhatia M, Safavi-Naeini P, Razavi M, Collard C, Tolpin D, Anton J. Anesthetic Management of Laser Lead Extraction for cardiovascular implantable electronic devices. Semin Cardiothorac Vasc Anesth. 2017;21(4):302–11. https://doi.org/10.1177/1089253217728581.
2. Fischer M, Harvey R, Boyle N, Ho J. Anesthesia considerations for Lead extraction. Cardiac Electrophysiol Clin. 2018;10(4):615–24. https://doi.org/10.1016/j.ccep.2018.04.003.
3. Kusumoto F, Schoenfeld M, Wilkoff B, Berul C, Birgersdotter-Green U, Carrillo R, Wazni O. 2017 HRS expert consensus statement on cardiovascular implantable electronic device lead management and extraction. Heart Rhythm. 2017;14(12):e503–51. https://doi.org/10.1016/j.hrthm.2017.09.001.
4. Swanton J, Keane J, Vlahakes C, Streckenbach C. Intraoperative transesophageal echocardiography in the early detection of acute tamponade after laser extraction of a defibrillator Lead. Anesth Analg. 2003;97(3):654–6. https://doi.org/10.1213/01.ANE.0000074234.13373.E7.

Part VIII

Cardiac Room Devices

Understanding Devices in the Cardiac Operating Room

32

Ahmed S. Awad, Matthew Ortman, Muhammed Muntazar, and Michael Rosenbloom

Seek simplicity, and distrust it.

—Alfred North Whitehead

Temporary Epicardial Pacemaker Therapy

Introduction

Postoperative conduction abnormalities that occur after cardiac surgery are a significant cause of mortality and morbidity. Temporary epicardial cardiac pacing provides an external artificial conduction system in which a battery-powered device (impulse generator box) delivers an electrical impulse to the heart and initiates myocardial contraction. The cardiac surgery anesthesia provider must thoroughly understand the intraoperative management of temporary epicardial pacemaker systems. Temporary pacing during cardiac surgery is commonly used for diagnosis, therapeutic intervention, and postoperative prophylaxis. After separation from cardiopulmonary bypass, some patients require a brief period of pacing until the heart has

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recovered from the consequences of cooling, cardioplegia, or heart mugging during complex surgical procedures. Although most patients require no pacing, some never recover their normal conduction system and require a permanent pacemaker. Most surgeons routinely place temporary epicardial wires, in which atrial wires are inserted into the right atrial wall and the ventricular wire is inserted into the surface of the right ventricular wall.

Modern pacemakers are sophisticated and easily programmable. They can pace and sense in one or two chambers and adjust their discharge rate by tracking intrinsic atrial and ventricular activity.

Rhythm disturbance occurring immediately after separation of cardiopulmonary bypass can be attributed to one or more factors: electrolyte disturbances, such as hyperkalemia; hypothermia; postoperative myocardial edema; or surgical injury to the conduction pathway. How the pacemaker operates and the technical factors to consider when programming a pacemaker are reviewed in this chapter.

Types of Temporary Pacing

There are many types of temporary pacing, but those used most often in the cardiac surgical suite are epicardial pacing systems. If pacing is an anticipated requirement after cardiac surgery and access to the right ventricle is deemed difficult, as in minimally invasive aortic valve surgery through mini-thoracotomy, a transvenous pacing Swan-Ganz catheter can be used. Other strategies include transcutaneous or transesophageal pacing. Temporary epicardial pacing can provide immediate and precise rate control of the heart.

Indications for therapeutic and diagnostic temporary pacing:

- Therapeutic pacing
  1. Conduction abnormalities
  2. Augmentation of cardiac output after separation from cardiopulmonary bypass
  3. Overriding of atrial pacing for treatment of atrial flutter/supraventricular tachycardia
  4. Overriding of ventricular pacing to prevent ventricular tachycardia
  5. Bi-atrial pacing for prevention of postoperative atrial fibrillation
- Diagnostic pacing

When P waves are not clearly seen on a 12-lead ECG, an atrial electrogram can be used. Some ECG machines have a specific three-lead atrial electrogram recorder. On an ECG machine without specific atrial electrogram leads, P waves can be amplified by attaching the atrial pacemaker wires to the leads of the ECG (Fig. 32.1).

1. Atrial electrogram can help distinguish junctional tachycardia from supraventricular tachycardia and sinus tachycardia.
2. Atrial electrogram can help distinguish various degrees of heart block from sinus node dysfunction.

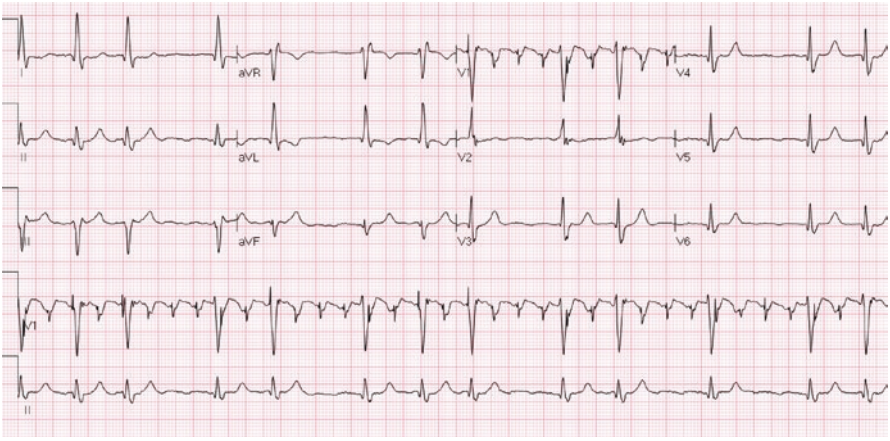

Fig. 32.1 12-lead ECG of atrial flutter. Lead V1 has been attached to the epicardial atrial wire, proving that the underlying rhythm is atrial flutter with variable conduction

Anatomy of the Epicardial Temporary Pacing System

The temporary epicardial pacing system consists of three main components: epicardial pacing leads, connecting cables, and a temporary pulse generator box. The epicardial pacing wires, with electrodes at the tip, are inserted into the epicardium and are then attached to a pacemaker box (impulse generator box) via a pacing cable (Figs.32.2 and 32.3).

Functionally, at its simplest, the electrical current delivered from the device's battery and discharged from the negative terminal of the generator box flows through a connecting wire to depolarize the myocardium, initiating contraction, and flows back into the positive terminal of the generator box to complete the circuit.

Epicardial Pacing Wires and Leads

Two temporary epicardial electrode systems are available: unipolar and bipolar. Also, the system can be a single-chamber system or a dual-chamber system.

The wire has a small curved needle at the lead side, which is used to insert the lead partially through the wall of the myocardium. The needle is cut off and discarded after the leads are placed. The other end of the wire is connected to a large, straight needle, which is used to pull the wires through the skin; this needle is also cut off, leaving pacing lead connector pins. Some wires are coiled to assist fixation.

The unipolar circuit system consists of a single wire (negative lead) attached to the epicardium, with another wire, the positive lead, attached at a distance through the skin of the epigastric area.

A bipolar circuit (two wires on the heart) can be achieved using a single-wire configuration of the bipolar temporary myocardial pacing system or the two unipolar

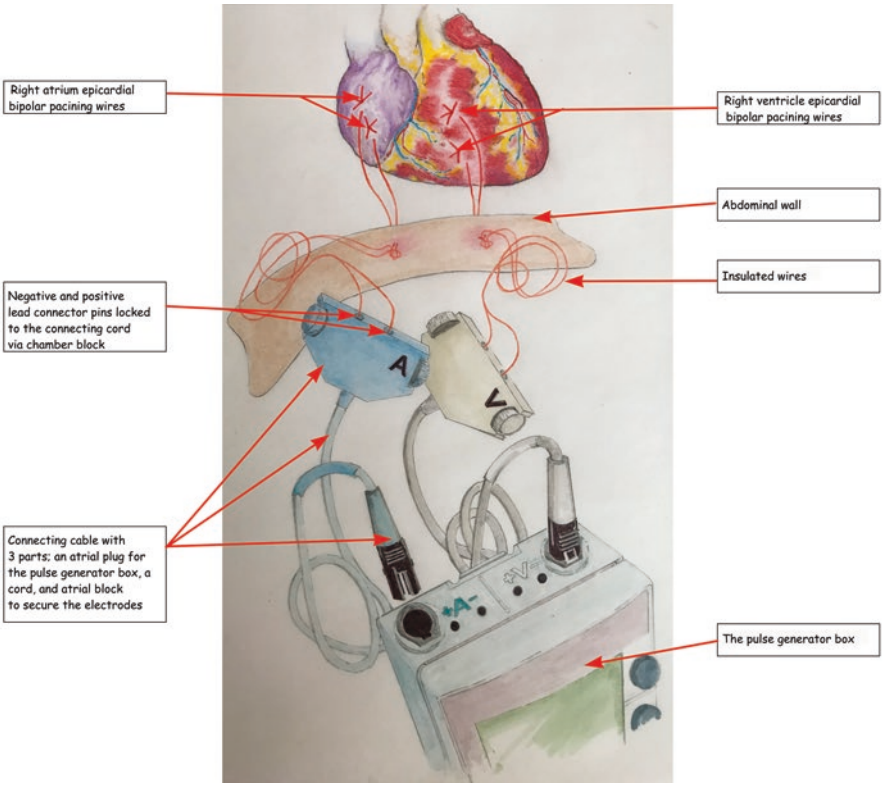

Fig. 32.2 Illustration of the complete bipolar temporary epicardial pacing system attached to the right atrium and right ventricle

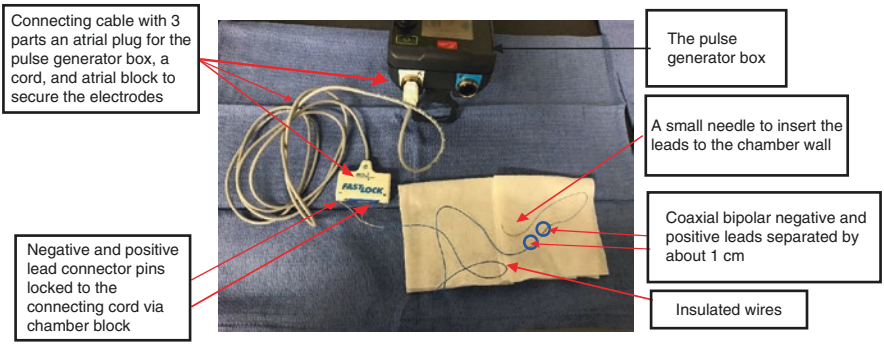

Fig. 32.3 Image of the complete bipolar temporary epicardial pacing system to be attached to the atrium

wire system (Fig. 32.4). The first system consists of a single lead that has two in-line discrete electrodes insulated electrical wires, which enable placement of both electrodes in the myocardium with only one needle puncture (Fig. 32.5). The positive wire and the negative wire are separated from each other. Both ends are sutured

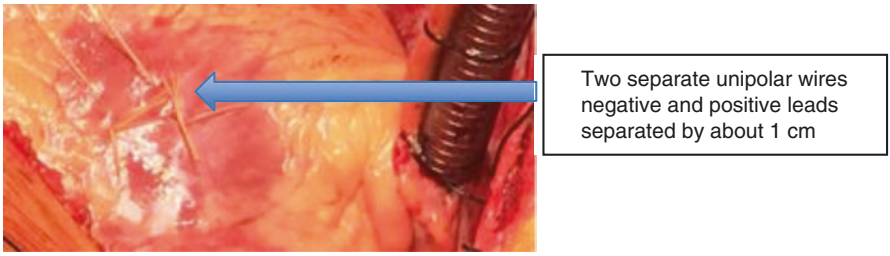

Fig. 32.4 Image showing insertion of bipolar ventricular pacing wires prior to weaning from cardiopulmonary bypass

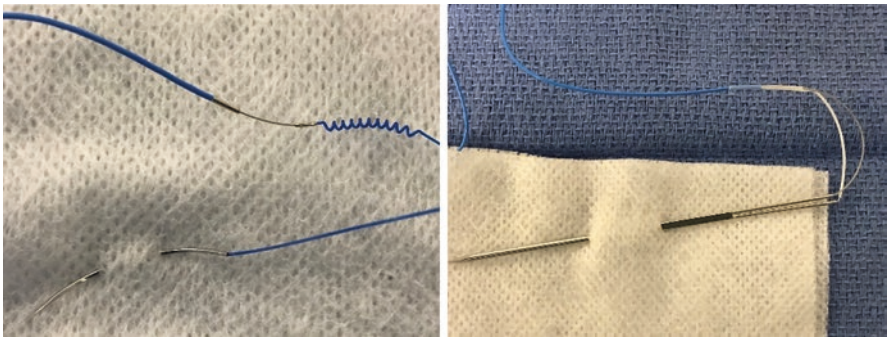

Fig. 32.5 Image showing *Left*: bipolar pacing wires insertion side *Right*: bipolar pacing wires connection side

loosely or clipped into the epicardium about 1 cm apart, where the distal wire is the negative lead and the proximal wire is the positive lead, with the myocardium completing the circuit. The second system uses two single unipolar wires attached to the heart, where one is the positive lead and the other is the negative lead, as shown below.

By convention, the atrial pacing wires exit the patient's chest and are brought out on the right side of the epigastrium, whereas the ventricular wires exit the patient's chest and are brought out on the left side of the epigastrium to allow easy identification. The electrodes can be color-coded.

The electrodes are connected by an adapter, which is attached to the pulse generator via an electrical cord. Most pulse generators have dual-chamber functionality that is easy to operate. The electrodes are designed to be removed percutaneously a few days after the operation before hospital discharge.

Patient Cable and Connector

Two types of cables (Fig. 32.6) are used in the operating room: surgical cable (disposable) and patient cable (disposable or reusable). The surgical cable has two small alligator clips at one end, where one clip (red) is the negative lead and is attached directly to the heart and the other clip (black), or the positive lead, is attached to the subcutaneous

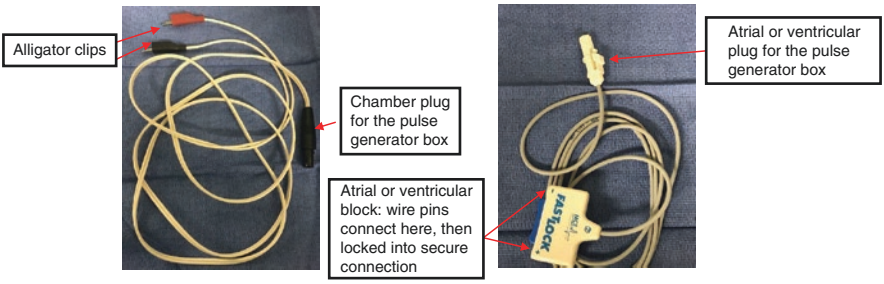

Fig. 32.6 Image showing *Left*: alligator or surgical connecting cable *Right*: connecting patient cable

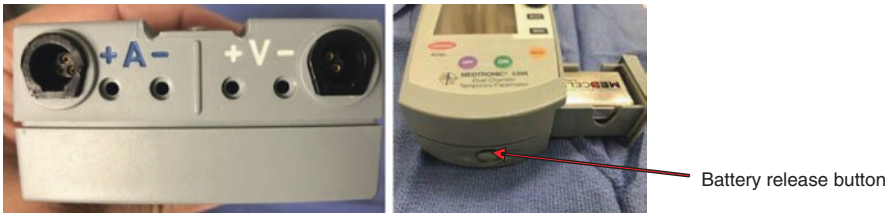

Fig. 32.7 Image showing *Left*: pacemaker connecting part *Right*: Battery housing

tissue. The other end of the surgical cable, which the surgeon hands to the anesthesia provider across the drapes, is called the generator plug and connects to the generator box. The surgical type of cable acts as the pacing lead and a connecting cable.

For the patient cable, the surgeon connects the end of the epicardial wires (pins) to an adapter at one end, called atrial or ventricular block. This provides a safe, secure connection for pacing electrodes while minding the polarity and tightening the thumb wheel. The blocks are color-coded—white for the ventricle and blue for the atrium. The other end, the generator plug, connects to the appropriate sockets in the top of the generator box (Fig. 32.7 left). One socket is marked A (atrium); the other is marked V (ventricle). These sockets also are color-coded.

The Pulse Generator Box

Pulse generators are small, battery-operated devices (Fig. 32.7 right) designed to do two things:

1. Sense the patient's intrinsic cardiac rhythm by means of a "sensing circuit"
2. Discharge electrical impulses to depolarize the myocardium and restore the intrinsic heart rhythm or increase the heart rate

Functionality of the Dials and Buttons (Fig. 32.8)

- Some pacers have a menu button that controls many submenus.
- The screen has a battery indicator.

- The screen has a lock indicator.
- The green light, when flashing, indicates pacing.
- The orange light, when flashing, indicates sensing.

The Timing of Insertion and Initiation of the Pacing

After the release of the aortic cross-clamp, three scenarios *may arise*:

- The heart is in sinus rhythm.
- The heart is fibrillating.
- The heart is in asystole or bradycardia.

Fig. 32.8 Illustration of pacemaker function dials

Table 32.1 NBG code for pacemaker programming

First Letter	Second Letter	Third Letter
Chamber(s) paced	Chamber(s) sensed	Response to sensing
A = atrium	A = atrium	I = inhibit (demand mode)
V = ventricle	V = ventricle	T = triggered
D = dual (both atrium and ventricle)	D = dual	D = dual
O = none	O = none	O = none (asynchronous)

In the first scenario, the pacer box is programmed as a backup at a lower rate than the patient's intrinsic rate. In the second scenario, defibrillation is attempted; if it is not successful after multiple attempts, measures such as correction of hyperkalemia, hypothermia, acid-base imbalance, or myocardial ischemia are applied. After defibrillation, the heart is expected to be in sinus rhythm, bradycardia, or asystole. In the third scenario, the pacer box is programmed to be the primary source of the conduction system of the heart.

The NBG Pacemaker Code

The North American Society for Pacing and Electrophysiology (NASPE) now is the heart rhythm society, and the British Pacing Electrophysiology Group (BPEG) came up with NBG, a five-letter code which stands for NASPE/BPEG Generic Code.

The first three letters are discussed here as applicable for temporary pacing in the operating room. The first letter of the NBG code refers to the chamber(s) being paced—"A" for atrium, "V" for ventricle, and "D" for dual. The second letter refers to the chamber(s) being sensed—again "A" for atrium, "V" for ventricle, and "D" for dual. The third letter refers to the system's response to a sensed event—"I" for inhibit, "T" for trigger, and "D" for dual (Table 32.1).

Modes of Pacing

The pulse generator can deliver atrial pacing, ventricular pacing, or sequential pacing in various modes, but only a few modes are commonly used in the perioperative period; they are reviewed in Table 32.2.

1. DDD pacing is dual-chamber pacing. It is the most common and most useful mode in the perioperative period for cardiac surgery patients. It is physiologic pacing, as it provides a synchronized atrioventricular (AV) contraction, and it is important when the atrial "kick" contributes substantially to the cardiac output. DDD has four distinct pacing patterns (Fig. 32.9) depending on the patient's intrinsic conduction:
   - A sense V sense, meaning complete inhibition of the pacemaker, with only sensing and observing, i.e., normal conduction.

Table 32.2 Common modes of intraoperative and postoperative pacing

Code	Chamber paced	Chamber sensed	Response trigger/ inhibit	Representing
AOO	Atrial pacing	None	None	Asynchronous atrial pacing occurs at a set rate, irrespective of intrinsic atrial impulses
VOO	Ventricular pacing	None	None	Asynchronous ventricular pacing occurs at a set rate, irrespective of intrinsic ventricular impulses
DOO	Atrial and ventricular pacing	None	None	Asynchronous atrial and ventricular pacing at a set rate, irrespective of intrinsic atrial and ventricular impulses
AAI	Atrial pacing	Atrial sensing	Inhibits pacing	Sensed P wave would inhabit atrial pacing. The absence of an intrinsic p wave will initiate atrial pacing
VVI	Ventricular pacing	Ventricular sensing	Inhibits pacing	Sensed R wave would inhibit ventricular pacing. The absence of an R wave will initiate ventricular pacing
DDD	Atrial and ventricular pacing	Atrial and ventricular sensing	Inhibition of pacing and trigger of pacing impulses	Intrinsic P and R waves will inhibit pacing. Pacing impulses will be generated when programmed intervals are surpassed

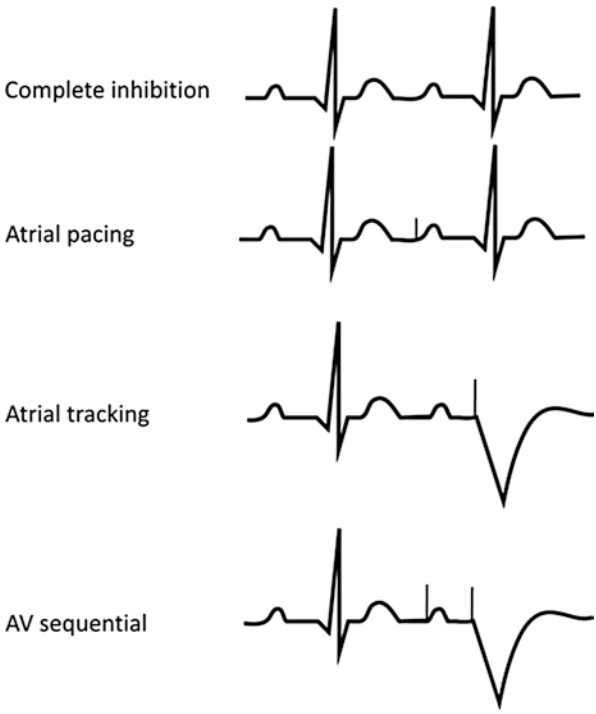

Fig. 32.9 Illustration of various DDD pacing patterns

- A sense V pace, also known as P wave tracking, meaning the sinus node function is good, but AV nodal conduction is poor (the pacemaker is tracking the patient's intrinsic atrial rate 1:1).
- A pace V sense, meaning a sinus node malfunction, but AV nodal conduction is intact.
- A pace V pace, meaning malfunction in both the sinus node in the AV node, resulting in pacing in both the atrium and ventricle.

DDD pacing is useful in patients who have sinus rhythm and complete AV block.

2. AAI pacing provides demand atrial pacing that paces and senses in the atrium, i.e., pacing at the programmed rate of the device and inhibiting atrial pacing output in response to a sensed atrial event; thus, it has two pacing patterns, depending on the intrinsic conduction (Fig. 32.10).
   - A sense, meaning inhibition of the pacemaker firing. It is only sensing the atrium and observing, i.e., normal conduction.
   - A pace, meaning a sinus node malfunction, but AV nodal conduction is intact. AAI pacing is useful for patients who have isolated sinus node dysfunction with preserved AV nodal conduction; it maintains AV synchrony.
3. VVI pacing provides demand ventricular pacing, which will pace and sense in the ventricle, pacing at the programmed rate of the device and inhibiting ventricular pacing output in response to a sensed ventricular event; thus, it has two distinct pacing patterns, depending on the intrinsic conduction (Fig. 32.11).

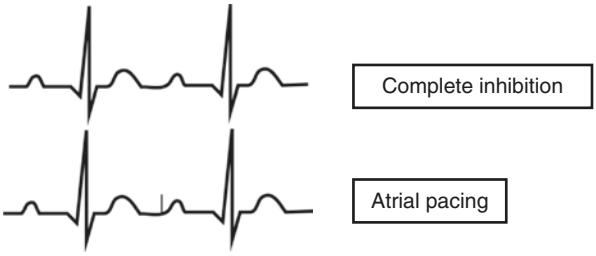

Fig. 32.10 Illustration of various AAI pacing patterns

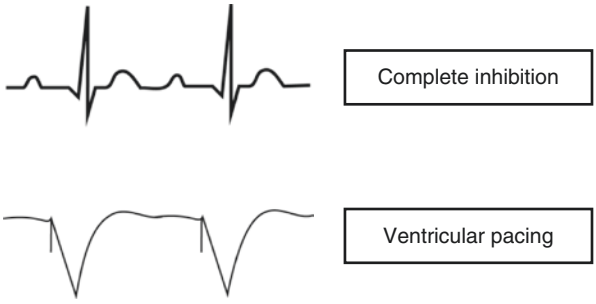

Fig. 32.11 Illustration of various VVI pacing patterns

- V sense, meaning inhibition of the pacemaker firing; it is only sensing the ventricle and observing, i.e., normal conduction.
- V pace, meaning the ventricle is paced, irrespective of intrinsic or native atrial activity.
- This mode protects against dangerous bradycardia if there are no atrial leads, and it is most useful for patients who have atrial fibrillation or flutter, where AV synchrony is not important.

4. AOO is an asynchronous or fixed atrial pacing that is used when electrocautery is being used, as cautery will trigger inappropriate sensing.
5. VOO is an asynchronous or fixed ventricular pacing that is used when electrocautery is used. Electrocautery can cause R-on-T conduction, in which an asynchronous pacing spike is superimposed on a T wave of a premature ventricular contraction, which can trigger ventricular fibrillation.
6. DOO is an asynchronous or fixed dual-chamber pacing mode. With it, the device will not sense the intrinsic rhythm, and the system will pace independent of intrinsic electrical activity.

Basic Programming Walkthrough

Commonly used temporary pulse generators: Medtronic model 5388 or model 5392 dual-chamber temporary external pacemaker (Medtronic, Minneapolis, MN, USA).

After turning on the pacing box, by pressing the on/off button, the default setting will be at rate 80, atrial output at 10 mA, ventricular output at 10 mA, and mode DDD.

Temporary pacing variables for programming (Fig. 32.12):

(i) Pacing rate: Should be set to optimize the patient's hemodynamics.
(ii) Pacing mode: Usually set to DDD; however, other modes can be used, depending on clinical circumstances.
(iii) Output and stimulation threshold: If the patient is dependent on the pacemaker, the output should be set at two or three times the minimal output necessary for the pacemaker to capture.
(iv) Sensitivity: If the patient does not have an underlying rhythm, the temporary pacer should be turned to the least sensitive value.

Duration of Usage

Atrial and ventricular temporary epicardial leads are dependable for short-term use only, as their function deteriorates because of inflammatory reaction developing around the wire; use for no longer than 7 days is recommended. The lead should be removed under aseptic precautions by gentle traction while the ECG is monitored, and the patient should be watched for a few hours after extraction because of the possibility of tamponade occurring.

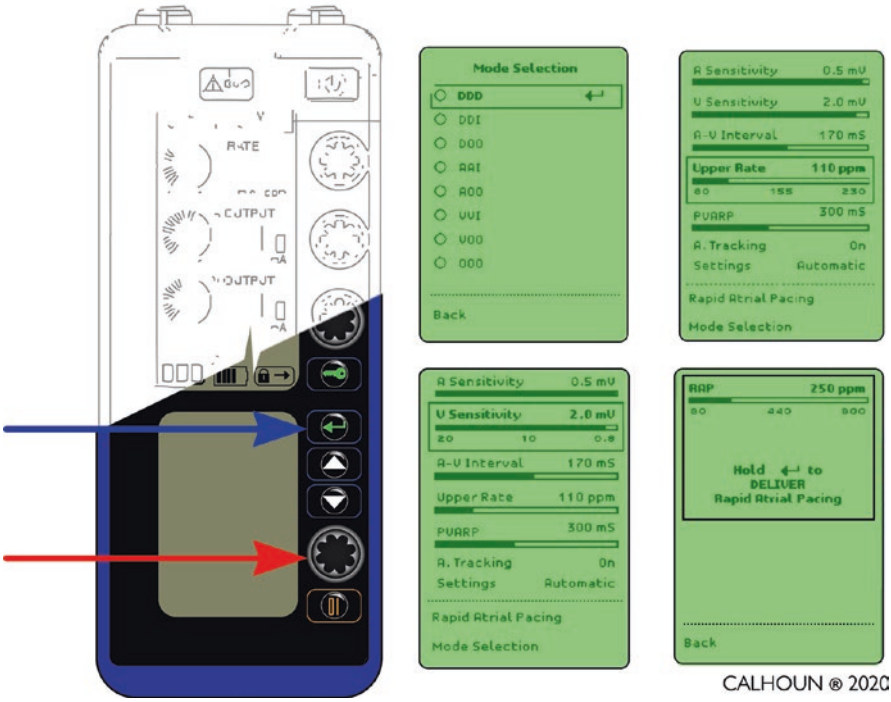

Fig. 32.12 Illustration of various menu options, use the wheel (red arrow) to toggle between menus and press enter to open (blue arrow)

Stimulation Threshold Setting

Threshold is the minimum stimulus above which myocardial capture consistently occurs. The typical capture threshold for an epicardial wire is 2–5 mA, but the threshold value can vary broadly.

Commercially available pulse generators can deliver up to 20 mA of pacing output.

Steps to take to determine safe threshold:
The first step is determining the patient's underlying rhythm. It is never acceptable to simply "turn off" the pulse generator to check for underlying conduction (Fig. 32.3) because "long short" sequences can provoke polymorphic ventricular arrhythmias, especially in patients who have severe systolic dysfunction.

- For patients in atrial fibrillation or flutter who have a VVI system I place, the rate can be lowered to 30 beats per minute for assessment of native conduction.
- For patients in sinus rhythm, the DDD mode should be switched to VVI mode or the atrial lead unplugged manually. The ventricular threshold can then be tested in the VVI mode.

- If the heart is not being paced, the pacing rate is increased to 10–20 beats faster than the patient's native rate. This practice is advised because a paced rhythm is needed to verify output threshold; then, pacing output is progressively lowered until there is loss of capture—that is, a pacing spike with no QRS.
- Testing must be performed with live monitoring, so that loss of capture is recognized immediately.
- The pacing output should be twice the measured threshold to maintain an adequate margin of safety.

Sensing

Sensing is a critical, but often overlooked (and misunderstood), part of pacing. Poor sensing can compromise both atrial and ventricular leads; however, for the purposes of simplicity and brevity, this discussion focuses on ventricular undersensing only. Failure to sense can be subtle, characterized by an unusually short interval between the native QRS and paced QRS (Fig. 32.13).

In the most extreme circumstance, failure to sense and appropriately inhibit pacing can result in "R-on-T" pacing. This rare occurrence can induce ventricular tachycardia or fibrillation (Fig. 32.14), so it must be immediately corrected.

Sensing Threshold

This is the least-sensitive programming at which the device can detect a heartbeat. The higher the number, the lower the sensitivity; the lower the number, the higher the sensitivity. An analogy for understanding the concept of sensing is to visualize a fence. Imagine that your neighbors have a 10-foot-tall fence on their property. You cannot see anyone behind the fence because it is too high. Now imagine that a new,

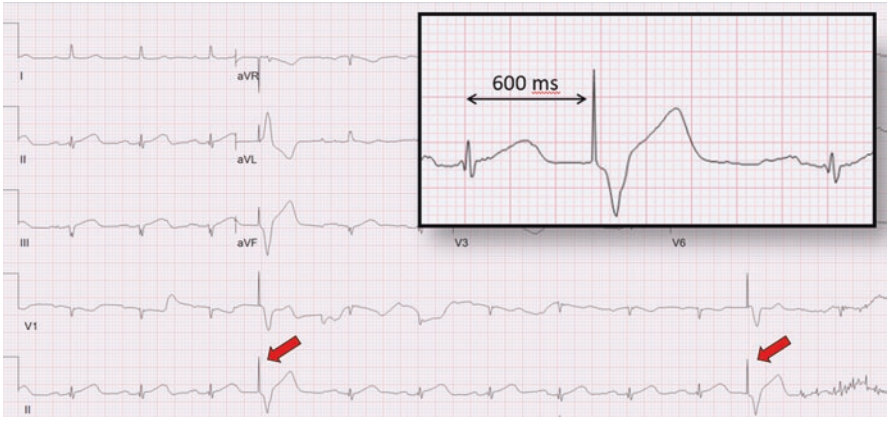

Fig. 32.13 12-lead ECG of ventricular undersensing. The pacing spikes fall approximately 600 ms after the preceding QRS complex (red arrows). The lack of pacing elsewhere on the tracing proves that there is a problem with sensing

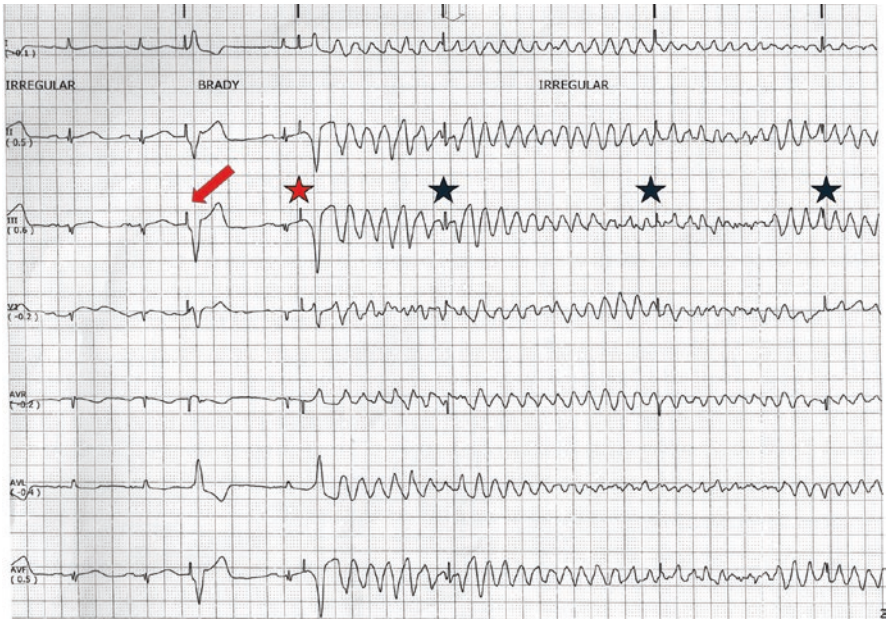

Fig. 32.14 Telemetry strip of ventricular undersensing in the same patient. The first pacing spike (red arrow) falls harmlessly after completion of repolarization, but the second pacing spike (red star) falls on the vulnerable period of ventricular repolarization, initiating ventricular fibrillation (VF). Ventricular pacing marches through VF at a variable rate due to intermittent undersensing (black stars)

more friendly neighbor reduces the height of the fence by half. Whereas, before you couldn't see even a giant on the far side of the fence, you can now easily see someone of average height. It is counterintuitive, then, that a taller fence is analogous to a lower sensitivity and a shorter fence is analogous to a higher sensitivity. A pacemaker system with a higher programmed sensitivity will have a lower programmed valve for sensing (in mV) and therefore be able to detect even small, intrinsic signals and vice versa (Fig. 32.15).

Determining the Sensing Threshold

1. Program the device rate to 10 ppm below the patient's intrinsic rate.
2. Decrease the output to the minimum value of 1 mA to prevent unintentional R-on-T waves.
3. Start decreasing the sensitivity (slowly turn the sensitivity dial clockwise) until the sense indicator stops flashing.
4. Increase the sensitivity (slowly turning the dial counterclockwise) until the sense indicator starts flashing again (sensing threshold).
5. Program the sensitivity to one-half the mV value to provide at least a two-to-one safety margin.

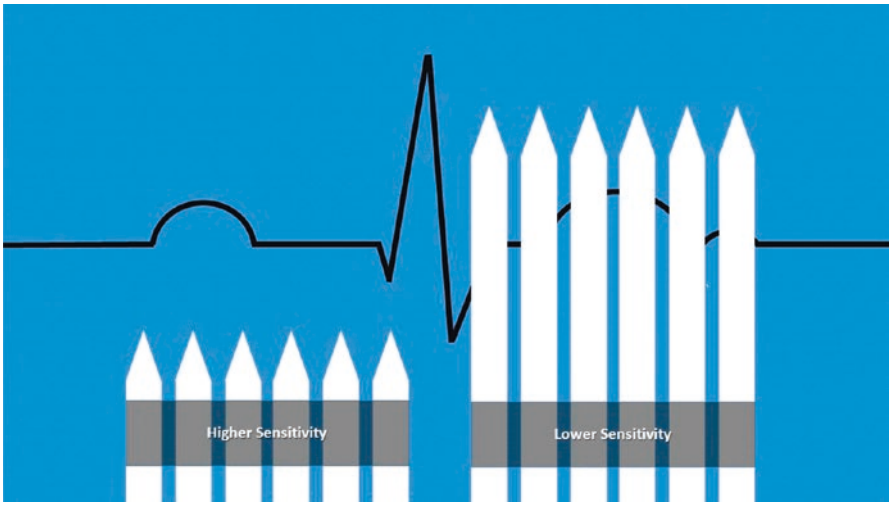

Fig. 32.15 Illustration of schematic diagram of pacemaker sensitivity

Complications of Temporary Pacing

Epicardial pacing has a low risk of complications, but they include the following:

1. Pacing capture failure. Typically, when the atrium or the ventricle is being paced, a spike, immediately followed by the P wave or QRS, respectively, is seen; there is no delay if every P or QRS wave has a spike preceding it, indicating that the chamber is being 100% paced. The term "failure to capture" means that the pacemaker senses a slow intrinsic heart rate; the pacemaker box then sends an impulse, but the heart does not respond, and there is no capture; thus, pacemaking spikes appear, but there is no P or QRS to accompany the spike.

   Pacing capture can fail for many reasons:
   - Insufficient electrical output from the pacing box
   - Broken or damaged lead
   - Pacing box failure
   - Inadequate contact or detachment of wires from the chamber surface
   - Electrical shorting of epicardial wires
2. Pacer sensing failure. If the intrinsic cardiac rhythm is appropriate, the pacemaker should be in listening mode. However, if the pacemaker fails to sense the heart's intrinsic electrical activity, paced spikes will occur during normal cardiac activity, with atrial or ventricular pacing spikes arising irrespective of the presence of P waves or QRS complexes. This situation can lead to an R-on-T superimposition, which can generate VF.

   Pacer sensing can fail for many reasons:
   - Improper sensing threshold
   - Hardware problem (lead fracture, inadequate contact, faulty insulation)
   - Insufficient native myocardial voltage
   - Electrolyte abnormality

3. Infection
4. Myocardial damage
5. Ventricular arrhythmias
6. Damage to coronary anastomosis
7. Perforation of the right atrium or the right ventricle wall and tamponade

Clinical Pearls

- The batteries must be checked by examining the battery power indicator before starting the case and one more time prior to pacing. A spare new battery is usually taped to the back of the generator box. Thirty seconds of pacing is provided while changing the battery.
- All patients especially post valve surgery must be attached to a pacing box, with backup settings in case the patient develops conduction abnormalities.
- The pacing of the RV alone creates septal wall dyskinesis and can sometimes generate severe MR due to asynchronous delayed activation of the LV. In this case, biventricular pacing can solve the problem.
- DOO (an asynchronous mode or fixed rate mode) of pacing may be utilized when electrocautery is operated to prevent inhibition of the pacemaker by electrocautery radiofrequency current. Switch back to synchronous mode (DDD) as soon as electrocautery is not used. Asynchronous mode DOO should be used with caution as it has the potential to induce VF.
- Shorting or grounding can happen if the chest is filled with fluids or blood that lead to *no* capture. Also, lung expansion during an inspiratory phase can encroach on the atrial pacing leads and lead to intermittent capture.
- Pacing leads must be handled while wearing gloves due to the potential of micro shocks as it provides a direct route for electrical current to flow directly to the heart and induce VF.
- DDD mode carries the risk of developing fast ventricular tracking in atrial tachyarrhythmias. Limiting the maximum tracking rate will avoid this problem.

Short-Term (<14 days) Mechanical Circulatory Support

Many devices that can support the circulation in the perioperative period are available. The predicted duration of circulatory support and the urgency of the patient's need are some of the factors to be considered in choosing a device. These devices support vital organ perfusion while lowering cardiac filling pressures and left ventricular volume and wall stress, resulting in improved balance of myocardial oxygen supply/demand. The devices include intra-aortic balloon pump (IABP), continuous miniature axial flow (Impella® Heart Pump), extracorporeal (outside of the body) system TandemHeart®, and extracorporeal membrane oxygenation (ECMO).

The Intra-aortic Balloon Pump

Introduction

IABP counter pulsation is one of the most commonly used short-term mechanical circulatory support devices. In cardiac surgical patients, IABP devices can be placed preoperatively, intraoperatively, or postoperatively. The IABP-SHOCK II Trial found no difference in mortality at 30 days between patients treated medically and those treated with IABP. Accordingly, the use of IABP has been downgraded to Class IIb recommendations by American College of Cardiology/American Heart Association guidelines.

Anatomy of the Intra-aortic Balloon Pump

The IABP consists of three main parts: a balloon, a vascular catheter, and a console with a pneumatic pump and helium tank (Fig. 32.16). The IABP is a helium-filled polyurethane cylindrical balloon wrapped around the distal end of a flexible introducer vascular catheter. Helium is used because of its low density, and it is relatively harmless if it leaks or the balloon ruptures; also, the balloon can be inflated quickly. The flexible vascular catheter has two lumens: the outer lumen is a path for the helium to inflate and deflate the balloon. The central lumen is a passage for inserting the guidewire and transducing the blood pressure at the tip of the catheter; it is operated and controlled by a console. Modern IABPs have fiber-optics that monitor and detect the pressure waveform while calibrating automatically in vivo for fully automatic operation.

Insertion and Proper Positioning of the Balloon

The balloon pump is usually inserted percutaneously via the left or right common femoral artery in the groin. However, the axillary, subclavian, or direct aortic route can be used for access if the femoral approach is not feasible. The IABP catheter is

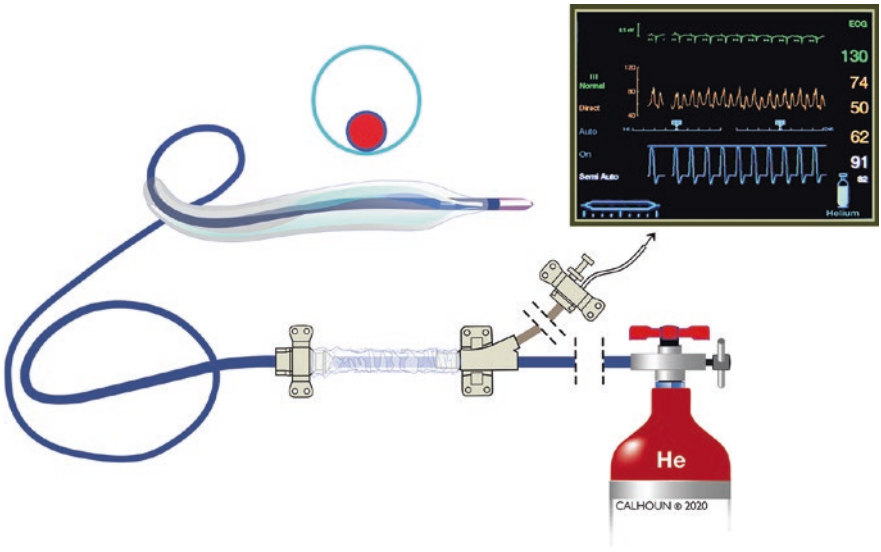

Fig. 32.16 Illustration of IABP components

threaded into the descending thoracic aorta, and the tip is positioned 2–3 cm distal to the origin of the left subclavian artery. Proper sizing and positioning of the balloon are vital to avoid visceral ischemia. The balloon size (volume ranges between 25 and 50 ml) should not exceed the distance between the left subclavian artery and the celiac trunk. The IABP is positioned under fluoroscopic guidance in the catheterization laboratory or under TEE guidance in the operating room.

Principles of Counterpulsation, Timing, and Physiology of Balloon Pumping

Principles of Synchronized Counterpulsation

The IABP does not *pump* blood. During cardiac diastole and after the aortic valve closes, the balloon inflates and displaces the blood from the descending aorta proximally toward the heart. During cardiac systole and after the aortic valve opens, the balloon deflates and creates a vaccum distally (Fig. 32.17).

Timing

Proper timing of inflation and deflation of the IABP will enable it to produce maximal hemodynamic benefit. Inflation or deflation of the balloon can be triggered by electrocardiogram waves, pacing spikes, or blood pressure waveform, either internally or through autopilot mode. The trigger initiates the inflation and deflation of the IABP in coordination with the cardiac cycle.

1. Timing by the ECG. Balloon inflation is timed after the ST-T wave; deflation is timed with sensing of the R wave, which identifies the onset of systole.
2. Timing by pacing spikes. For V or AV pacing, the ventricular spike is the trigger, whereas in A pacing, the sensed R wave is the trigger. Patient rhythm must be 100% paced.
3. Timing by blood pressure waveform. Balloon inflation is timed with the aortic dicrotic notch, whereas deflation is timed with the beginning of the systolic arterial upslope.

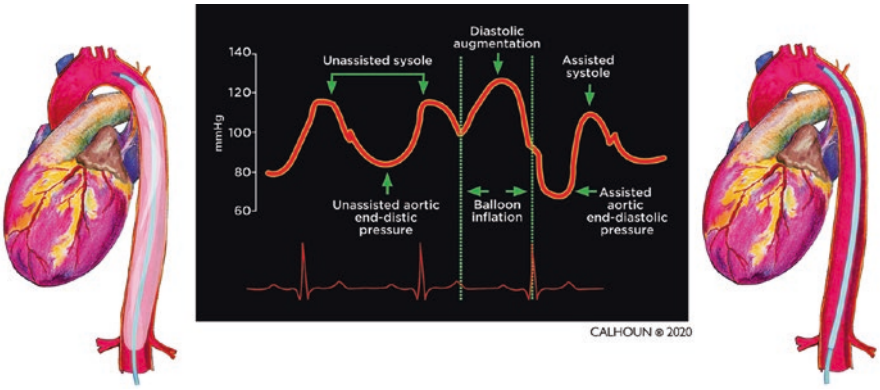

Fig. 32.17 Illustration of one whole cardiac cycle and the matching waveform of the IABP during inflation and deflation

4. Internal timing. This is used when there is no ECG or cardiac output (asystole). The balloon inflates and deflates asynchronously at a preset rate.
5. Timing through autopilot mode. If the fiber optic sensor is connected, autopilot mode is automatically selected, and the console selects the source of the trigger signal and the timing method. The console constantly monitors and selects the trigger signal best suited for the clinical condition.

Physiologic Effects

The principal objective of IABP is to enhance left ventricular performance by increasing oxygen supply and decreasing oxygen demand of the myocardium. The hemodynamic effects of IABP therapy include increasing diastolic blood pressure and coronary perfusion and decreasing left ventricular afterload during systole, which can increase stroke volume and improve cardiac output. IABP may also have indirect effects on right ventricular performance by increasing right ventricular blood flow. Also, unloading the left ventricle reduces the left atrial and pulmonary artery pressures, thus decreasing right ventricular afterload.

Indications and Contraindications for IABP

Indications

1. Preoperatively, IABP can begin in the cardiac catheterization laboratory as follows:
   - Prophylaxis for high-risk cardiac surgery patients who have severe left main or critical aortic stenosis preoperatively
   - Prophylaxis in high-risk acute myocardial infarction
   - Rescue IABP in acute myocardial infarction associated with progressive reduction in ventricular function, with deteriorating hemodynamics and developing cardiogenic shock
   - Rescue IABP in mechanical complications of acute myocardial infarction, such as postinfarction rupture of the ventricular septum
2. Intraoperatively, IABP is used for treatment of failure to wean from cardiopulmonary bypass with maximum inotropic support.
3. Postoperatively, IABP is used in the ICU for treatment of cardiogenic shock despite maximal inotropic support.

Contraindications

1. Severe vascular disorders, such as descending aortic aneurysm or dissection
2. Severe aortic insufficiency
3. Sepsis

Complications of the Balloon Pump

Complications that might be associated with IABP are as follows:

1. Inappropriately timed inflation and/or deflation
   (i) Early balloon inflation, before aortic valve closure, will increase LV afterload and decrease stroke volume as the LV ejects against the inflated balloon, with increase in LV wall tension and left ventricular end-diastolic volume.

(ii) Early balloon deflation will not reduce afterload.
(iii) Late balloon inflation will have no or minimal increase in perfusion pressure and flow to the coronary arteries, leading to less than optimal augmentation of flow.
(iv) Late balloon deflation will increase LV afterload and decrease ejection as the LV ejects against the inflated balloon.

2. Dissection or rupture of the descending aorta
3. Bleeding from vascular injury
4. Stroke from air emboli originating at the tip of the balloon from the pressure monitoring port (since the tip is near the carotid arteries), or from malposition of the balloon, with occlusion of these vessels
5. Thrombocytopenia
6. Local infections, bacteremia, and sepsis
7. Peripheral limb ischemia

Extracorporeal Pumps

Extracorporeal means that these devices have pumps that reside outside of the body.

Extracorporeal Membrane Oxygenation

ECMO is one of the most commonly used mechanical circulatory support systems for the non-pharmacologic treatment of post-cardiotomy cardiogenic shock. The indication for ECMO use in the heart room is mainly failure to wean from cardiopulmonary bypass. ECMO can be either veno-venous (VV) for oxygenation only or veno-arterial (VA) for circulatory and oxygenation assistance. The VA ECMO aids with single or biventricular failure either with or without associated respiratory failure. The circuit consists of tubing, cannulas, continuous-flow centrifugal pump for forward blood pumping, and a membrane oxygenator for gas exchange. Unlike a CBP machine, ECMO has no venous reservoir. Anticoagulation is required despite the use of heparin-bonded circuit. Deoxygenated blood is drained from the inferior or superior vena cava, pumped through the oxygenator, and returned to the descending aorta at flow rates up to 6 L/min. This system can be placed percutaneously or placed by a surgical cutdown for peripheral VA ECMO. Also, it can be placed by central cannulation as a transition from cardiopulmonary bypass for central VA ECMO.

Tandem Heart®

The TandemHeart (LivaNova, Inc., Houston, TX,) is a short-term left ventricular assist, left atrial-femoral bypass device. It consists of an inflow transseptal cannula, outflow femoral arterial cannula, control console, and centrifugal pump with flow rate up to 3.5–5 L/m depending on the arterial cannula size.

Blood is drained from the left atrium through a fenestrated cannula across the atrial septum to exit from the right femoral vein to an extracorporeal centrifugal

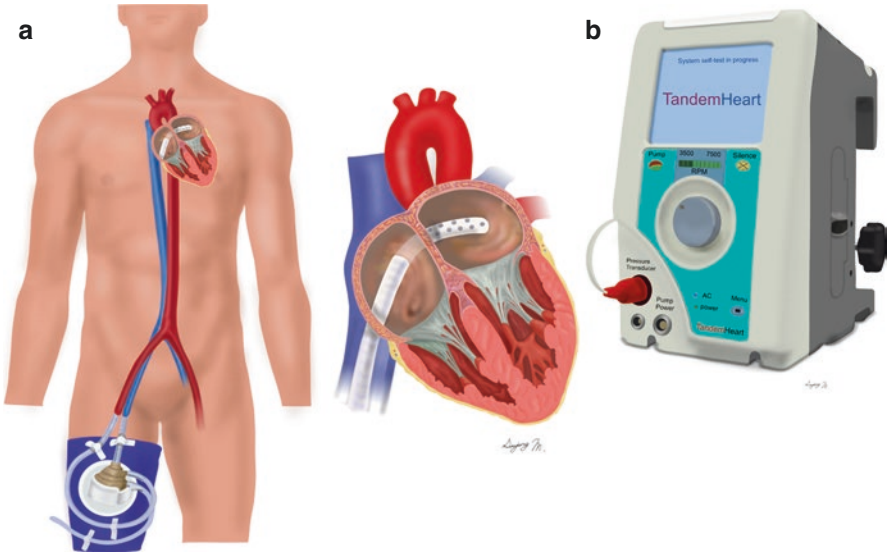

Fig. 32.18 Illustrations showing TandemHeart (**a**) the proper placement across the atrial septum and (**b**) TandemHeart controller

pump (Fig. 32.18). The blood then flows back to the descending aorta via the common femoral artery cannula. An external controller monitors and controls the function of the pump.

Miniature Axial Flow Ventricular Assist Devices

Impella®

The Impella system (Abiomed, Danvers, MA) is a short-term left ventricular assist device that provides continuous axial flow from the left ventricle to the ascending aorta, delivering up to 5.0 L/min. In the heart room, either the Impella 5.0 or Impella LD can be used. The Impella LD is inserted directly through the ascending aorta and into the left ventricle, whereas the Impella 5.0 is inserted via the femoral or axillary artery through a surgical cutdown. The tip is placed inside the left ventricle, and blood is pumped from the left ventricle to the ascending aorta. The system is composed of an Automated Impella Controller and an Impella 5.0 or LD Catheter. Components of the Impella catheter include a pigtail for stabilization; blood inlet area at the distal tip of the cannula; a cannula with a spiral-shaped body between the inlet area and outlet area; blood outlet area, where the blood leaves the cannula; motor housing with an encapsulated pump motor; and a catheter shaft placed between the motor housing and the controller. The controller has an interface for monitoring and operating the function of the Impella system (Figs. 32.19 and 32.20). The RECOVER I trial of the Impella system in patients presenting with postcardiotomy cardiogenic shock yielded favorable results.

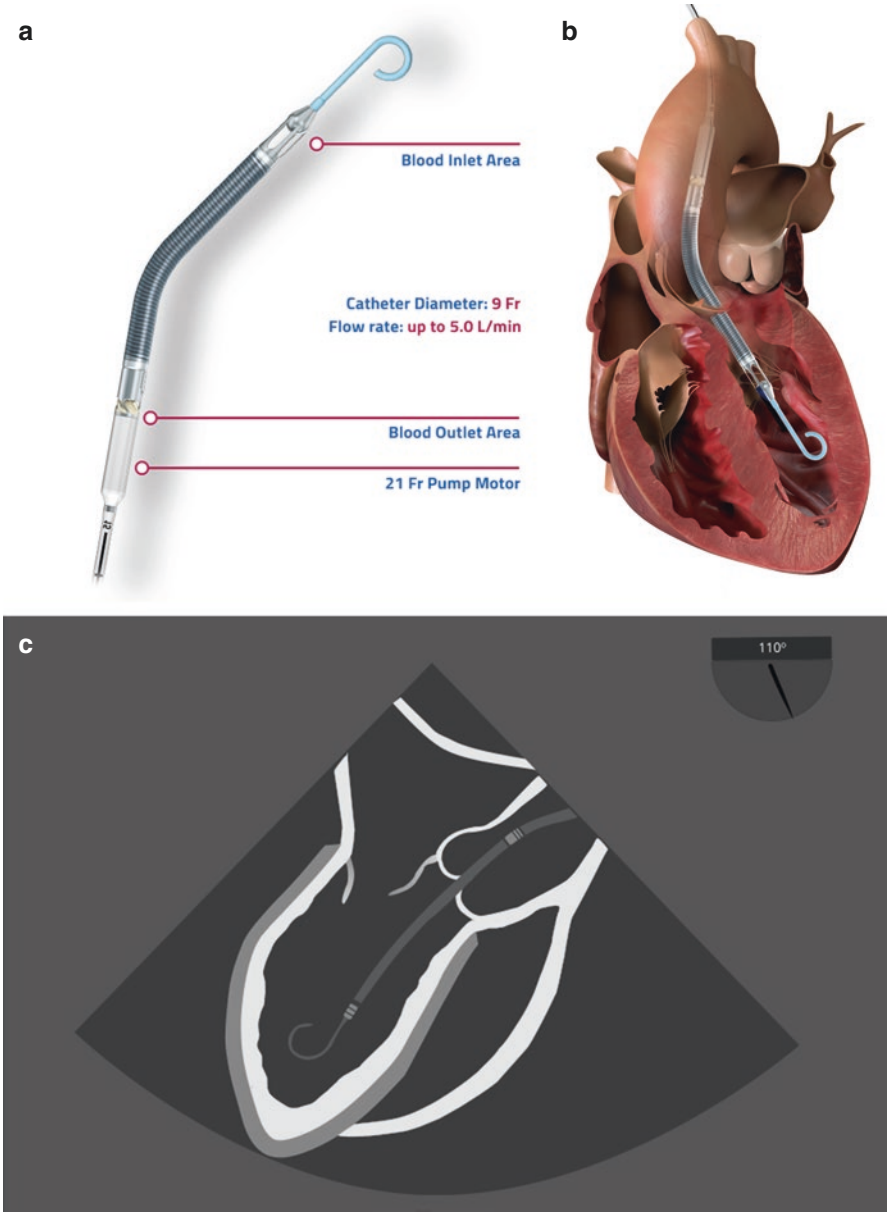

Fig. 32.19 Showing (**a**, **b**) Impella system with the inlet and outlet ports, (**c**) TEE image with the correct position of Impella (**a**, **b**). (Courtesy of Impella®)

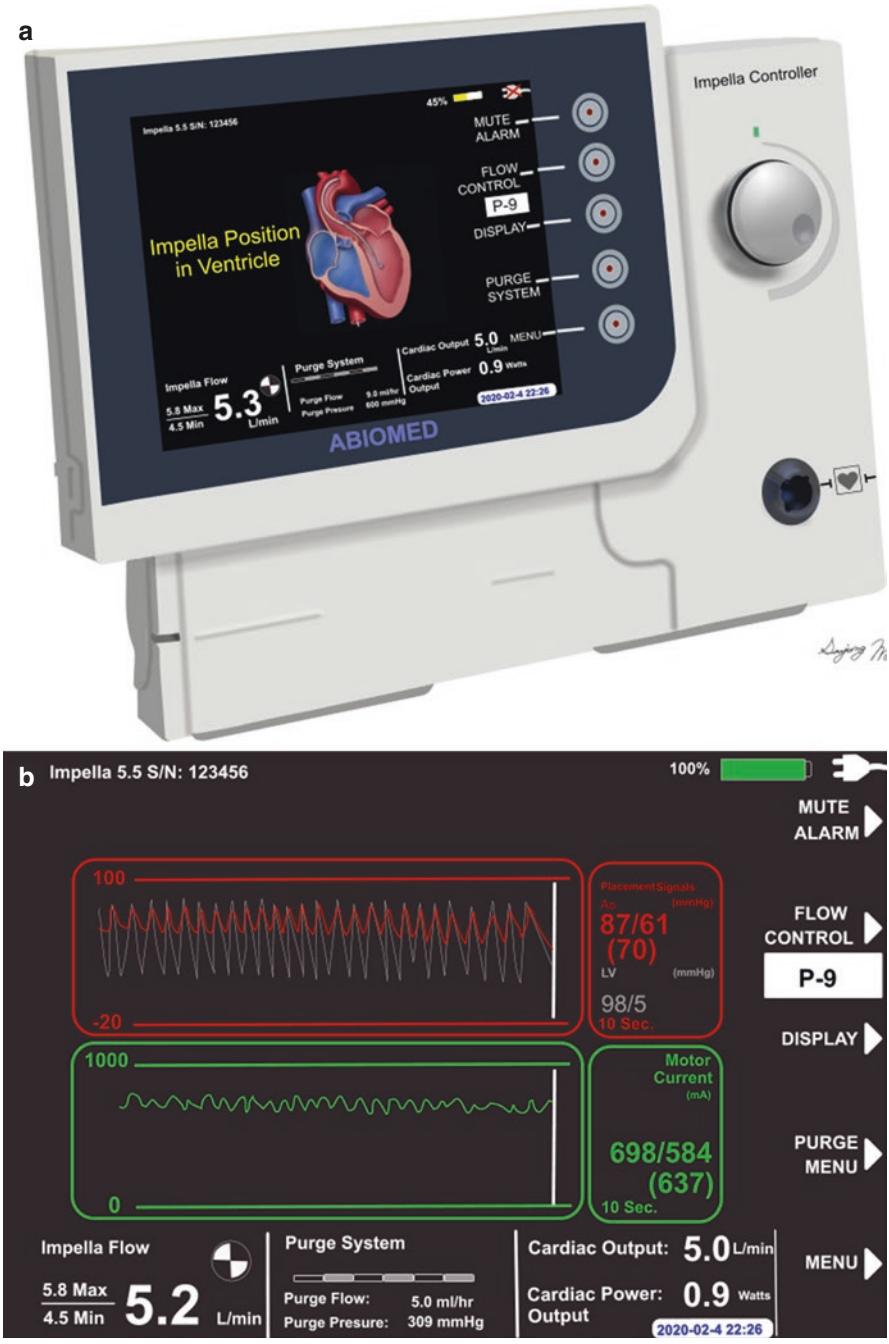

Fig. 32.20 Showing (**a**) automated Impella Controller (**b**) An aortic and ventricular waveform are displayed on the Impella Console

Table 32.3 *Summary* of short-term (<14 days) mechanical circulatory support devices commonly used in the cardiac surgery room

	IABP	ECMO®	TandemHeart®	Impella®
Type of pump	Pneumatic	Centrifugal	Centrifugal	Axial rotary
Method of insertion	Retrograde 7.5 or 8.5F femoral artery balloon vascular catheter into the descending aorta	19–29F femoral vein or internal jugular vein inflow cannula into the right atrium and 15–29 F femoral artery outflow cannula into the descending aorta	21F femoral vein inflow cannula into the left atrium via transseptal perforate and 15/19F femoral artery outflow cannula	Retrograde femoral artery 21F (Impella 5.0/ lD) catheter or anterior aorta (Impella 5.5) across the aortic valve
Amount of cardiac output enhancement	Increased CO by 0.5 L/min	Increased CO by up to 6 L/min	Increased CO by 3.5–5 L/min	Increased CO by 5–6 L/min
Flow dynamics	Balloon inflation with blood displacement, pulsatile	Continuous, nonpulsatile flow	Continuous, nonpulsatile flow	Continuous, nonpulsatile flow
Anticoagulation	May need systemic anticoagulation with heparin drip	Systemic anticoagulation with heparin drip	Systemic anticoagulation with heparin drip	Systemic anticoagulation with heparin drip
Limb ischemia	Low risk	High risk	High risk	Moderate risk
Bleeding	Low risk	Very high risk	High risk	Moderate risk
Hemolysis	Minimal risk	High risk	Moderate risk	Very high risk

Summary of short-term (<14 days) mechanical circulatory support devices commonly used in the cardiac surgery room is listed in Table 32.3.

Short-Term Right Ventricle Mechanical Circulatory Support

Protek Duo®

Protek Duo is a double-lumen cannula for support of a right ventricular assist device. Like a Swan-Ganz catheter, it is inserted as a percutaneous right-heart catheter via the internal jugular vein for right atrium-pulmonary artery placement. The inflow port of the cannula is situated in the right atrium, and the outflow port is in the main pulmonary artery. If there is respiratory failure, an oxygenator may be incorporated. A centrifugal pump provides continuous, low-speed, non-pulsatile flow. An external controller monitors and controls the function of the pump (Fig. 32.21).

Impella RP®

This system for temporary right ventricular support provides continuous axial flow from the inflow portion, positioned in the inferior vena cava to the outflow portion, positioned in the main pulmonary artery. A microaxial pump, mounted on the catheter distal to blood inlet, drives the blood across the decompensated right ventricle.

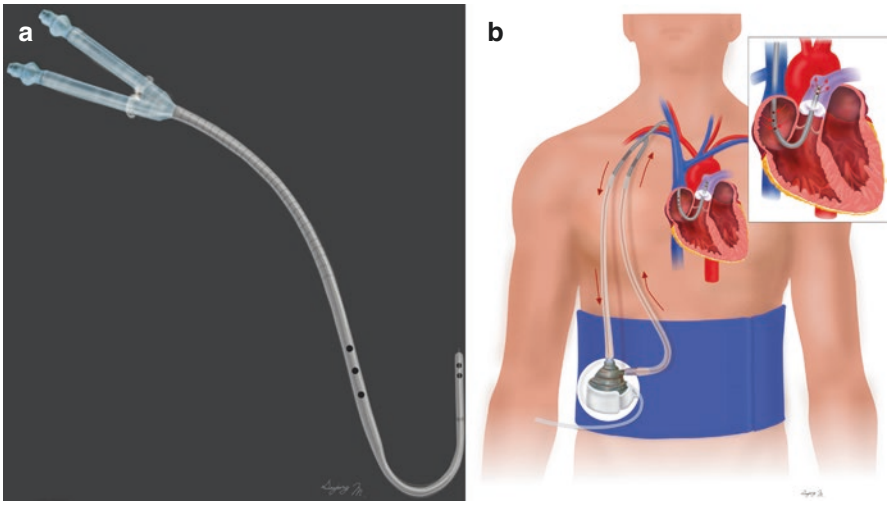

Fig. 32.21 Showing (**a**) Protek Duo dual-lumen cannula (**b**) adequate positioning from the right atrium to the pulmonary artery with inflow and outflow cannulas connected to the extracorporeal pump

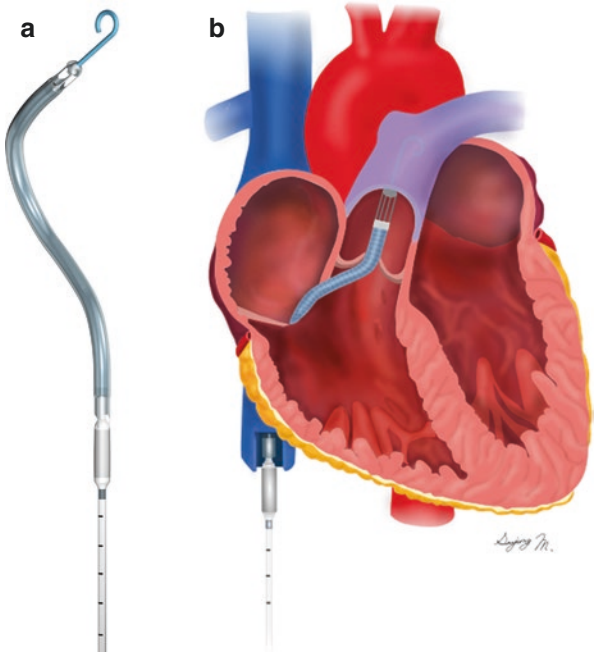

Fig. 32.22 Showing (**a**) Impella RP® device (**b**) device with the cannula in PA and the inlet and outlet ports, respectively (**a**). (Courtesy of Impella®)

The catheter is inserted percutaneously through the femoral vein. An external controller monitors and controls the function of the pump (Fig. 32.22). The RECOVER RIGHT trial of the Impella RP yielded favorable outcomes.

Further Reading

1. Reade MC. Temporary epicardial pacing after cardiac surgery: a practical review; part 1: general considerations in the management of epicardial pacing. Anesthesia. 2007;62:264–71.
2. Reade MC. Temporary epicardial pacing after cardiac surgery: a practical review; part 2: selection of epicardial pacing modes and troubleshooting. Anesthesia. 2007;62:364–73.
3. Yeh DD. Chapter 69: Intra-aortic balloon pump. In: Surgical critical care therapy. Switzerland: Springer; p. 687–97.
4. McClelland I, Kalathiya R, Shah A. Chapter 20 Percutaneous assist devices as salvage from cardiogenic shock. In: Difficult decisions in cardiothoracic critical care surgery. Springer Nature Switzerland AG: Springer.

Part IX

Anesthetic Management for Vascular Anesthesia

Anesthesia for Endovascular Aortic Aneurysm Repair

33

Christopher R. Hoffman and Michael S. Green

Everything should be made as simple as possible, but no simpler.

—Albert Einstein

Key Points

- Aortic aneurysm epidemiology
- Diagnosis and screening
- Preoperative considerations
- Intraoperative management
- Procedural setting
- Induction and maintenance
- Postoperative care and complications

Introduction

Abdominal aortic aneurysm (AAA) is defined as the localized expansion of the aorta below the diaphragm to a diameter that exceeds 3 cm or 50% larger than normal. AAAs are found in as many as 8% of men and 1.5% of women over the age of 65. They may remain completely asymptomatic for years or may result in intermittent abdominal, back, or lower extremity discomfort. The major complication resulting from the disease is eventual aneurysm rupture. The annual rate of rupture

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https://doi.org/10.1007/978-3-030-51755-7_33

Table 33.1 *Summary* of risk factors for abdominal aortic aneurysm

Major:
Smoking
Male sex
Age
Family history
Minor:
Hypertension
Hyperlipidemia
Obesity
Atherosclerotic disease
Genetic conditions:
Marfan's syndrome
Ehlers-Danlos syndrome
Loeys-Dietz syndrome
Bicuspid aortic valve
Polycystic kidney disease

relates to the aneurysm diameter: <5.5 cm, ~1%; 5.5–7 cm, ~10%; and >7 cm, ~33%. AAA rupture leads to severe abdominal or back pain, hemorrhage into the retroperitoneum, hypotension, and eventual loss of consciousness as a result of hypovolemic shock. As such, most patients with ruptured AAAs die before reaching the hospital or operative intervention. Among those who survive until hospital presentation, mortality rate is 75–90%.

The definitive treatment for AAA requires placement of a prosthetic graft to replace the weakened section of the artery. Open surgical repair has been available since the 1950s. In contrast, the first endovascular aortic aneurysm repair (EVAR) was reported in 1986. This minimally invasive approach has been associated with improved 30-day mortality, largely attributed to shorter procedural times, limited blood loss, and avoidance of aortic cross clamping. However, 6- and 8-year follow-up data suggest no difference in mortality between open repair and EVAR, but an increase in the need for secondary interventions, graft-related complications, and cost among endovascular repair patients. The selection of intervnetioanl approach must therefore stratify short- versus long-term survivability and cost on a patient-to-patient basis. Risk factors for AAA are summarized in Table 33.1.

Diagnosis

Physical examination can identify AAA, although the retroperitoneal location and large body habitus may delay diagnosis until severe dilatation has occurred. Though detectable via computed tomography angiography, ultrasonography remains the gold standard for the diagnosis of AAA with over 95% sensitivity and specificity (Figs. 33.1, 33.2, and 33.3). Screening policies vary widely and focus mainly on high-risk populations. In 2005, the United States Preventive Services Task Force (USPSTF) indicated that one-time screening for men over 65 with a smoking history or familial diagnosis carried a grade B recommendation. Men over 65 years old

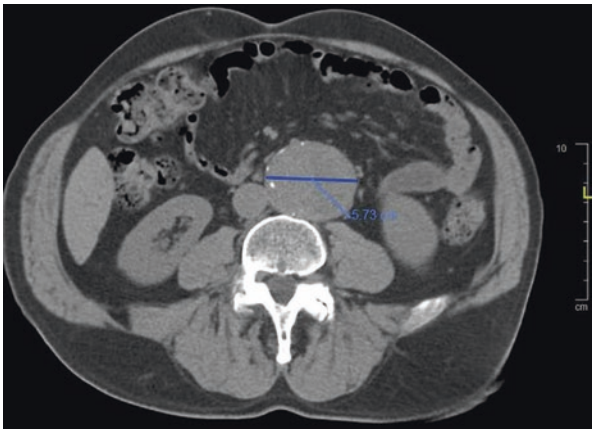

Fig. 33.1 CT imaging of abdominal aortic aneurysm with diameter measuring 5.73 cm

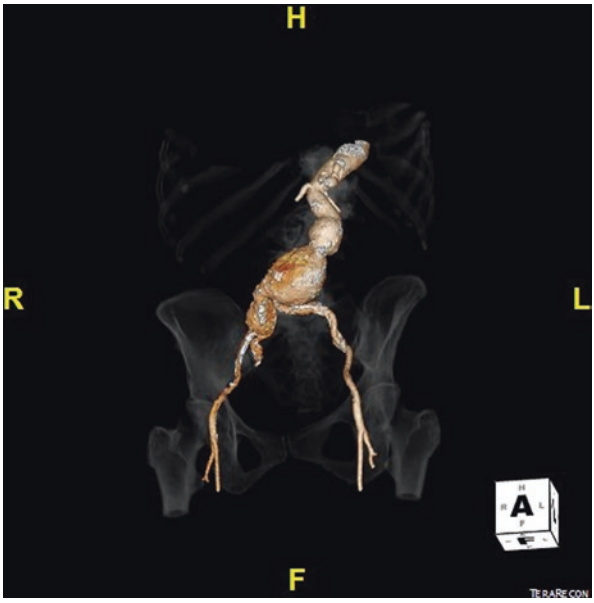

Fig. 33.2 CT reconstruction imaging of abdominal aortic aneurysm

who lacked other risk factors were given only a grade C recommendation for one-time screening. The USPSTF cited limited statistical evidence to support routine screening of other populations at this time.

Those with a known diagnosis of AAA require ongoing ultrasonographic surveillance. Smaller aneurysms are typically monitored every 3 years. When AAA diameter reaches 4.0 cm, the rate of expansion may accelerate, and the surveillance interval shortens to include annual studies. If the diameter reaches 4.5 cm, an ultrasound examination is repeated every 6 months.

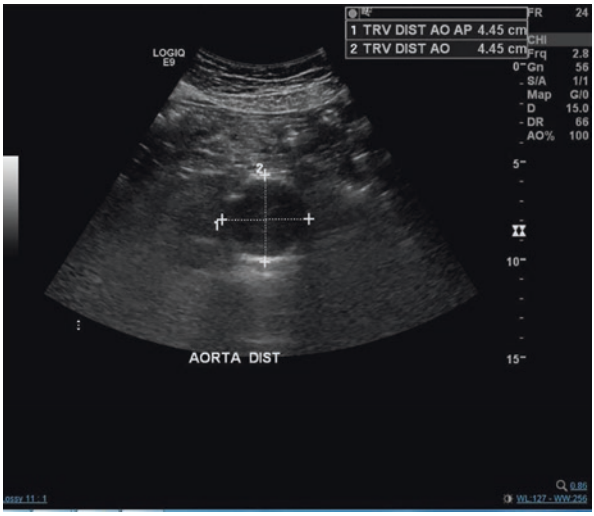

Fig. 33.3 Ultrasonography imaging of abdominal aortic aneurysm with diameter measuring 4.45 × 4.45 cm

Preoperative Considerations

No strict guidelines exist for preoperative preparation of EVAR patients. Investigation and the opportunity for optimization of comorbid medical conditions will necessarily vary according to presentation. Aneurysm rupture or dissection with end-organ ischemia is a surgical emergency. Signs and symptoms that may suggest these diagnoses include pain (stabbing or searing chest or abdominal pain radiating to the back) or hemodynamic instability. Referral for surgery for asymptomatic AAA is usually prompted by absolute aneurysm size or a significant interval enlargement. Intervention is considered when the risk of rupture exceeds the risk of surgical intervention—in general when aneurysm diameter exceeds 5.5 cm or when the rate of enlargement exceeds 1 cm in 12 months.

Consideration for endovascular repair also requires favorable aneurysmal anatomy as evaluated via computed tomography angiography. The aortic "neck," or the distance from the lowest renal artery to the origin of the aneurysm, must be of sufficient diameter, length, and angulation to allow for a stable site for endograft fixation. A small iliac artery diameter, severe tortuosity, or calcification complicate access for endograft placement.

Preoperative Assessment and Risk Stratification

Coronary artery disease is common among patients with AAA. A history of low ejection fraction, myocardial ischemiaon electrocardiogram, anemia, diuretic or digitalis therapy, or lack of statin therapy have all been associated with increased mortality rates among EVAR patients. Nonetheless, the preoperative assessment for

comorbid cardiac disease should be tailored to the individual risk factors present. The preoperative assessment and risk stratification for cardiac and non-cardiac patients are discussed in Chap. 5, while medical optimization is discussed in Chap. 7. Refer to these chapters for a more thorough discussion.

Intraoperative Management

The choice of anesthetic technique should be based on hemodynamic stability, cardiac function, anticoagulant use, and the preferences of the patient and proceduralist. The limited femoral access of the standard EVAR technique allows for a variety of approaches to anesthetic care. Regardless of the technique selected, the plan should always provide for rapid conversion to general anesthesia should an open repair become necessary.

The National Surgical Quality Improvement Program database indicates that 81% of contemporary elective EVAR cases are performed under general anesthesia, 12% received neuraxial anesthesia (7% spinal, 5% epidural), and 7% received local/monitored anesthetic care (MAC). It was noted in this retrospective report that the selection of general anesthesia for EVAR was associated with increased pulmonary morbidity and length of stay. However, patient complexity, surgeon and anesthesiologist preference, available resources, procedural complexity, and regional and institutional identity were not taken into account in this analysis.

General anesthesia may be preferred if a complex lengthy surgical procedure is anticipated, as conversion to open repair is considered likely, or access other than through the femoral vessels will be required. Neuraxial anesthesia requires that antiplatelet and anti-coagulation medications be stopped for sufficient time to allow the coagulation system to return to normal. Monitored anesthetic care (MAC) supplemented with local anesthesia may provide a sufficient level of comfort in the cooperative and motivated patient or in those where general or neuraxial anesthesia are felt to be unsafe. Another factor to consider is that controlled respiration with expiratory pauses on request may minimize the use of both fluoroscopy and intravenous contrast and can only readily be accomplished under general anesthesia.

The EVAR procedure involves the deployment of a self-expanding fabric or synthetic graft in the abdominal aorta. The stents used may be simple tube grafts that terminate above the aortic bifurcation or may be quite complex with fenestrations or arms to accommodate flow to the larger arterial branches of the aorta. Grafts are typically introduced via the femoral arteries, although alternative access sites such as the brachial or subclavian artery may be used based on the patient's individual anatomy. The graft is carefully positioned via fluoroscopy to completely overly and exclude the aneurysmal segment of the aorta. The goal is to position the stent to eliminate flow into the aneurysm sack and redirect flow exclusively through the stent graft. Without flow into the lumen of the aneurysm, the sac should thrombose and further expansion should be limited or halted altogether (Fig. 33.4). The graft is fixed to the aortic wall at the stent's proximal and distal ends via balloon expansion. Graft shape, length, and angulation are key in optimal positioning. Anesthetic

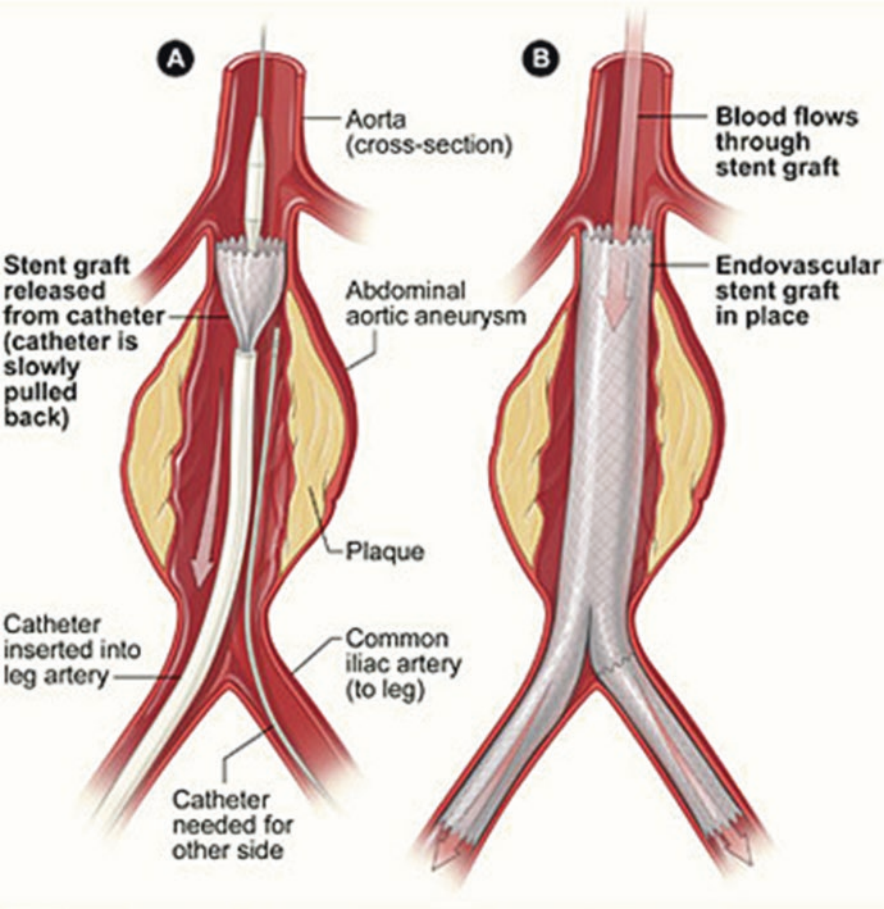

Fig. 33.4 Illustration of endovascular stent graft mechanism. (Source: National Heart, Lung, and Blood Institute; National Institutes of Health; U.S. Department of Health and Human Services)

management centers on providing anesthesia or simple analgesia for arterial cut down, while also maintaining coronary and end-organ perfusion to minimize ischemic risk during stent graft deployment.

Procedural Setting

EVAR is typically performed in a hybrid procedure room, which combines a standard surgical environment with advanced radiological imaging (Fig. 33.5). Permanent fluoroscopic equipment provides high resolution and digitally enhanced imaging while the presence of surgical equipment allows emergency conversion to an open procedure if necessary.

Fig. 33.5 Image of hybrid operating room

All of the equipment necessary to support MAC, neuraxial anesthesia, and general anesthesia should be present. Immediate access to laboratory services is required to allow for testing of hemoglobin levels, arterial blood gases, activated clotting time, and thromboelastometry.

The location should provide American Society of Anesthesiologists standard monitoring with capnograph, continuous electrocardiogram, non-invasive blood pressure measurement, temperature monitoring, and pulse oximetry with audible tone modulation and alarms. Measurement of invasive arterial pressures is routine during EVAR due to the need for beat-to-beat blood pressure control.

Two large bore peripheral venous catheters are required at a minimum. A low threshold should be maintained for securing central venous access if peripheral access is poor due to the potential for significant blood loss and vasopressor administration. Neuromonitoring via somatosensory and motor-evoked potentials may be included if aneurysm or graft location produces concern for potential spinal cord ischemia. Blood bank resources should be readily available prior to procedure initiation.

Induction and Maintenance

Induction of anesthesia should be focused on maintaining hemodynamic stability in patients with cardiac disease. General anesthesia may be induced with a balance of opioid (fentanyl) and hypnotic (propofol, etomidate). Low-dose vasopressor (phenylephrine, ephedrine, epinephrine) administration may be required if decreased systemic vascular resistance is encountered. Short-acting agents to blunt sympathetic nervous system discharge (esmolol, nitroglycerin) may be included during laryngoscopy and intubation. Vasoactive medications may also be used during the initiation of neuraxial anesthesia should significant sympathectomy occur, or if hemodynamic instability presents when initiating MAC sedation. General anesthesia may be maintained with a combination of volatile anesthetic, narcotic, and oxygen during stages of the procedure (Figs. 33.6, 33.7, and 33.8). If neuromonitoring

Fig. 33.6 Image of vascular surgeon achieving vascular access using ultrasound and fluoroscopy

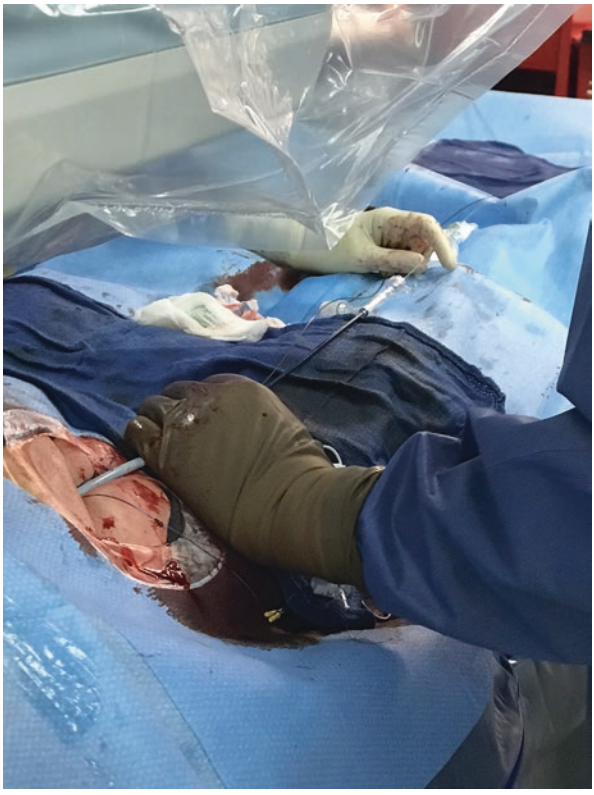

Fig. 33.7 Image of aortic endovascular stent graft delivery system

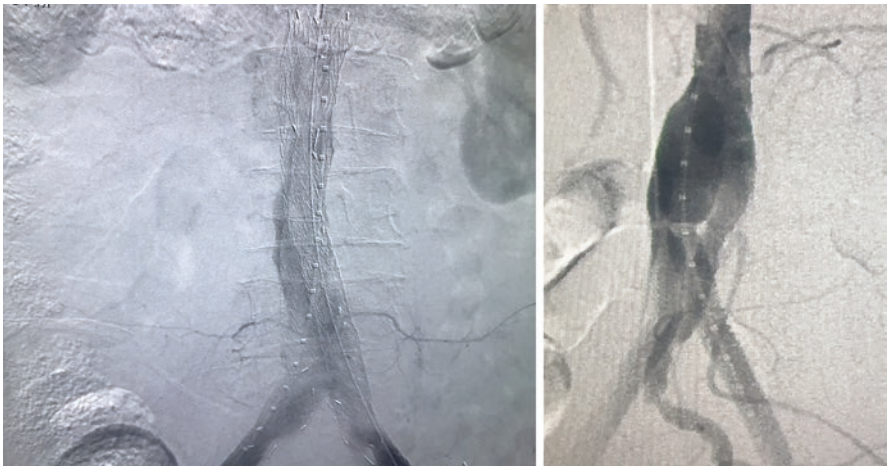

Fig. 33.8 Image of endovascular stent graft confirmed position by fluoroscopy

is being used, anesthetic maintenance may be converted to total intravenous anesthesia (propofol, remifentanil, sufentanil) to minimize anesthetic agent interference with evoked potentials.

Postoperative Care and Complications

Immediate postoperative management and patient disposition are dictated by the outcome of the procedure and the final hemodynamic status. Recovery commonly takes place in an intensive care unit to accommodate hemodynamic monitoring, laboratory assessment, and frequent lower limb examinations for perfusion and neurological function. Immediate priorities include hemostasis at the surgical arterial access site, confirmation of arterial perfusion distal to graft, hemodynamic stability, and adequate pain control.

A constellation of abnormalities following graft deployment (post-implantation syndrome) may be seen and include leukocytosis, fever, thromboembolic events, and coagulopathy. Acute kidney injury secondary to impaired perfusion and/or contrast-induced nephropathy is not uncommon. This complication may be reduced by optimizing volume status and cardiac output. Limb ischemia, arterial laceration, graft migration, and aneurysm rupture are all potential complications that warrant emergency return to the operating room.

Spinal cord ischemia is a rare complication of EVAR, but carries the potential for a catastrophic outcome. The incidence following EVAR is 0.21% and is associated with graft occlusion, prolonged aortic clamping, intraoperative hypotension, severe

atherosclerosis, and thromboembolic events. The final common pathway is a reduction in perfusion pressure to the vessels supplying the anterior spinal artery, most of which derive from the intercostal arteries arising directly from the aorta. Ischemia results in anterior spinal artery syndrome with loss of bowel and bladder continence and impaired motor function in the lower extremities. Preoperative placement of a catheter for intraoperative and postoperative cerebrospinal fluid (CSF) drainage has been shown to improve outcome should spinal cord ischemia occur, especially in higher risk graft locations. The mechanism of effect is felt to be an improvement in spinal cord perfusion pressure (perfusion pressure = mean arterial pressure − CSF pressure).

Other vascular beds susceptible to ischemia following EVAR include the inferior mesenteric, internal iliac, and middle sacral arteries. Longer stent grafts occluding more branch sites predictably increase the risk of ischemia. End-organ effects may include signs of impaired perfusion of the abdominal viscera or of the lower limbs. Maintenance of a high-perfusion pressure may augment collateral flow and reduce the manifestations of end-organ, limb, or spinal cord ischemia. Anesthetic management and complications for EVAR are summarized in Tables 33.2 and 33.3, respectively.

Table 33.2 Summary of anesthesia management of EVAR

Preoperative	No midazolam
Location	Abdomen via bilateral groin access
Duration	45–120 min
Anesthetic	MAC, neuraxial anesthesia, and general anesthesia
Medications	5000+ units heparin at the discretion of the surgeon Notify at 3 min Notify at 1 h for possible re-dosing
Drips	Phenylephrine, nicardipine
Positioning	Supine; bilateral arm tuck
Cases specifics	Arterial line for strict continuous blood pressure monitoring Patients with contrast allergy require 125 mg solumedrol prior to starting procedure Foley catheter for monitoring urine out put
Modifications	In some instances, aorto-uni-iliac device and femoral-femoral bypass may be required Retroperitoneal cut down and iliac conduit for small access vessels may be required Percutaneous access requires patient to lie flat and keep access leg straight for 2–4 hours to prevent bleeding Emergency conversion to open procedure for complications may be required Complex repairs may require access via arm for renal/mesenteric access or stenting Breath holds required for abdominal imaging for accurate imaging of renal/mesenteric vessels
Postoperative care	Intermediate care

Table 33.3 *Summary* of complications for EVAR

Preoperative:
Aneurysmal rupture
Poor cardiovascular optimization
Poor pulmonary optimization
Impaired renal function
Intraoperative:
Hemodynamic instability
Myocardial/cerebrovascular ischemia
Arterial cut down site hemorrhage/damage to surrounding structures
Failure to deploy stent
Incorrect stent deployment
Conversion to open repair
Conversion to general anesthesia
Postoperative:
Loss of surgical hemostasis
Hemodynamic instability
Arterial cut down site pseudoaneurysm
Compromised perfusion distal to graft
Vascular bed/end-organ/limb ischemia
Inadequate pain control
Post-implantation syndrome
Graft migration
Spinal cord ischemia

Clinical Pearls

- Risk factors for developing abdominal aortic aneurysms include smoking, male sex, age over 65, family history, vascular disease, and some genetic conditions.
- Endovascular aortic aneurysm repair has been shown to improve short-term morbidity, mortality, and length of stay. There is no known improvement in long-term mortality, and the endovascular approach is associated with an increase in secondary interventions over the long-term.
- General anesthesia may increase pulmonary morbidity and length of stay. Consideration must be given to cardiopulmonary status, hemodynamic stability, current medications, and anticipated procedural monitors and concerns when selecting type of anesthesia.

  Significant postoperative complications that warrant concern include post-implantation symptoms, acute kidney injury, limb occlusion, arterial laceration, graft migration, iatrogenic injury, aneurysm rupture, and spinal cord ischemia.

Further Reading

1. Kaplan JA, et al. Procedures in the hybrid operating room. In: Kaplan JA, editor. Kaplan's cardiac anesthesia: in cardiac and noncardiac surgery. Philadelphia: Elsevier; 2017. p. 1022–41. Print.
2. Kaplan JA, et al. Vascular surgery: endovascular and open surgery. In: Kaplan JA, editor. Kaplan's cardiac anesthesia: in cardiac and noncardiac surgery. Philadelphia: Elsevier; 2017. p. 1540–63. Print.
3. Frederick JR, Woo YJ. Thoracoabdominal aortic aneurysm. Ann Cardiothorac Surg. 2012;1(3):277–85.
4. Stather PW, Sidloff D, Dattani N, Choke E, Bown MJ, Sayers RD. Systematic review and meta-analysis of the early and late outcomes of open and endovascular repair of abdominal aortic aneurysm. Br J Surg. 2013;100(7):863–72.
5. Kothandan H, Haw Chieh GL, Khan SA, Karthekeyan RB, Sharad SS. Anesthetic considerations for endovascular abdominal aortic aneurysm repair. Ann Card Anaesth. 2016;19(1):132–41.

Anesthesia for Open Abdominal Aortic Aneurysm Repair

34

Karuna Puttur Rajkumar, Chinyere Archie, and Ksenia Guvakova

Living in a world such as this is like dancing on a live volcano.

—Kentetsu Takamori

Key Points

- Risk stratification
- Medical optimization
- Indications for repair
- Monitoring
- Aortic clamping and unclamping
- Postoperative management and complications

Introduction

Aortic aneurysm is defined as a focal and permanent dilatation of the aorta >3.0 cm in diameter or 1.5 times the normal size. The aneurysm is caused by a defect in the connective tissues involving all layers of the vessel wall. In the abdominal aorta, the infrarenal segment is the most common site of aneurysm. The prevalence of abdominal aortic aneurysms (AAAs) in screening studies is 4–8% in men and 1–2.2% in women. Elective surgical intervention may be warranted for asymptomatic patients

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if the aneurysm is >5.5 cm diameter in men or >5.0 cm diameter in women or if the aneurysm is enlarging rapidly. Emergency surgery is required for suspected rupture. For elective procedures, the anesthesia provider must ensure that comorbidities are thoroughly investigated and optimized preoperatively. Whether emergency or elective, open abdominal aortic repair is a high-risk operation that requires careful perioperative monitoring and hemodynamic management.

Preoperative Management

Open repair of AAAs is classified as a major high-risk vascular surgery. Patients presenting for open AAA frequently have coexisting medical diseases, such as hypertension, coronary artery disease, cerebrovascular disease, impaired left ventricular function, chronic obstructive pulmonary disease, pulmonary hypertension, diabetes mellitus, and renal impairment. Increased morbidity associated with emergency open AAA repair is at least partly due to the inability to achieve optimal medical treatment of these disease states prior to surgery. Preoperative assessment and risk stratification for cardiac and non-cardiac patients are discussed in Chap. 5.

A higher MET capacity indicates increased cardiovascular fitness. Patients with MET score less than 4 are at greater risk for intraoperative and postoperative adverse cardiovascular events. Here, the need for additional invasive monitoring and cardiovascular support should be anticipated.

With respect to perioperative cardiovascular complications, pharmacological stress tests are associated with a positive predictive value of only 20–30% but a negative predictive value of about 95%. A positive stress test should be followed with a coronary angiogram and preoperative therapeutic intervention if indicated for occlusive coronary disease.

Besides the Revised Cardiac Risk Index and Duke Activity Status Index, discussed in Chap. 5, there are risk stratification scoring tools specific for certain aortic operations. The Glasgow Aneurysm Score is one of the most commonly used predictors of 30-day postoperative outcomes for elective and emergent AAA repair; it is simple to use, and the results are compared with a single cutoff to predict high-vs-low mortality. The score components are age, shock, myocardial disease, cerebrovascular disease, and renal disease. A cumulative score of >84 is associated with a 30-day mortality of 65% and <84 with a 28% 30-day mortality.

$$\begin{aligned}\text{Glasgow Aneurysm Score} &= (\text{age in years}) + (17 \text{ for shock}) \\ &+ (7 \text{ for myocardial disease}) + (10 \text{ for cerebrovascular disease})\end{aligned}$$

The Hardman Index is a predictor of outcome after open repair of ruptured AAAs; it considers age, hemoglobin value, serum creatinine value, loss of consciousness, and electrocardiographic evidence of ischemia; it is the only tool validated for both immediate and 30-day mortality.

Table 34.1 Hardman Index for predicted mortality for open AAA

Number of points	Predicted mortality (%)
1	16
2	37
3	72
4	100
5	100

Hardman Index (Table 34.1)

Variables: age >76 years; creatinine >1.9 mmol/L; hemoglobin <9 g/dL; electrocardiographic evidence of myocardial ischemia; and history of loss of consciousness on arrival to hospital. One point is assigned to each of the five variables present.

Medical Optimization

Lifestyle modification, management of chronic medications, and medically optimizing patients with comorbidities are discussed in Chap. 7.

Decisions regarding the management of newer oral anticoagulant medications and antiplatelet agents must be made on a case-by-case basis. Individual indications for administration will impact the risk-versus-benefit analysis regarding when they should be stopped preoperatively. The patient, cardiologist, and surgeon should participate in the decision-making with regard to chronic medications when scheduling surgery.

Preoperative Investigations

Standard preoperative investigations should include electrocardiography (ECG), complete blood count, complete metabolic panel, and urinalysis. In many centers, an echocardiogram to assess myocardial function and rule out occult valvular disease is performed before any major vascular procedure. Blood grouping and preparation of cross-matched blood is required. Coagulation studies and liver function tests should be ordered, given the risk of perioperative complications related to bleeding abnormalities.

Imaging and physiological studies should be tailored according to comorbidities and specific risk. For example, coronary angiography, cardiac stress test, or pulmonary function tests may be indicated in patients with decreased exercise tolerance. Computed tomography (CT) imaging of the chest and upper abdomen for evaluation of the major vessels is usually part of surgical planning (Fig. 34.1). The CT scan may also provide information about pulmonary pathology.

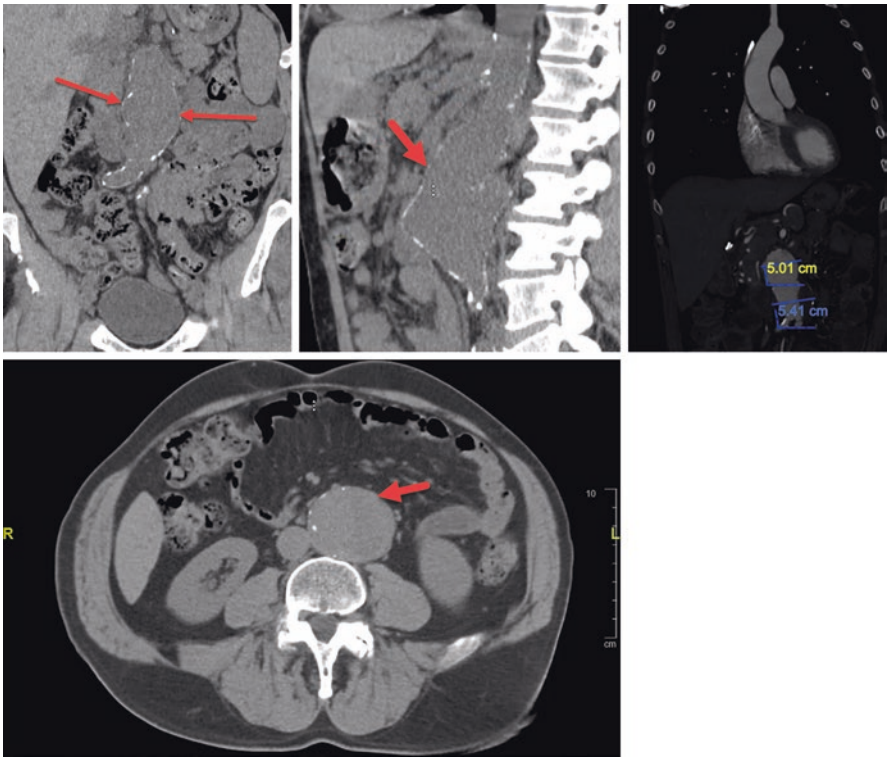

Fig. 34.1 Images of CT of different abdominal aortic aneurysms

Indications for Repair

Elective repair is indicated for fusiform AAAs with a diameter >5.5 cm, >5.0 cm with rapid growth (>1.0 cm in 12 months), or >5.0 cm in women. Elective repair is also indicated for patients with markers of proteolytic degradation and saccular aneurysm. Homocysteine, s-elastin peptides, and others are biomarkers associated with proteolytic degradation of the abdominal aneurysm wall and suggesting an impaired coagulation and fibrinolysis system. Emergency repair of AAA is required when symptoms of rupture are present or when endovascular repair has been aborted due to aortic injury or graft malposition.

There are two surgical approaches for infrarenal AAA repair: midline transperitoneal approach or the left retroperitoneal approach; both can be performed by either laparotomy or laparoscopy-assisted techniques. The retroperitoneal approach is the technique of choice for patients with previous abdominal operations, obesity, ascites, previous radiation to the pelvis, or peritoneal dialysis. Juxtarenal AAA requires suprarenal clamping, but aortic reconstruction is completed infrarenally.

Intraoperative Management

Open AAA repair requires advanced planning and coordination with the surgical team. Before the surgery begins, agreement should be reached on the monitoring techniques to be used, the form of analgesia to be employed, and the postoperative disposition of the patient. Concerns about the vascular anatomy and anticipated technical challenges should be discussed.

Planning and intraoperative management may be substantially abbreviated in emergency conditions, but the fundamental goals of anesthetic management are the same as those of elective repairs. Rapid infuser systems with warming capability should be assembled and connected to large bore IVs for blood and fluid administration. Type-specific cross-matched blood should be ordered. If cross-matched blood is not immediately available during emergency procedures, type-specific blood can be used. In the setting of severe or ongoing blood loss at any point during AAA repair, resuscitation with red blood cells, fresh-frozen plasma, and platelets in a 1:1:1 ratio by number of units may help prevent dilutional coagulopathy. Cryoprecipitate should be available if multiple units of packed red blood cells and fresh-frozen plasma are transfused, to augment fibrinogen levels. Blood salvage and autotransfusion should be used routinely. In patients with hypertensive emergency and impending rupture, short-acting beta blockers, such as esmolol, are started as an infusion and may be administered as intermittent boluses to control blood pressure if necessary.

Monitoring

All patients undergoing AAA repair should have standard American Society of Anesthesiologists monitoring, including pulse oximetry (SpO_2), continuous ECG, intermittent noninvasive blood pressure measurement, and end tidal carbon dioxide ($ETCO_2$) monitoring.

Invasive Blood Pressure Monitoring

An intra-arterial catheter is placed before the induction of anesthesia for continuous monitoring of blood pressure and pulse-pressure variation and for intermittent blood sampling for point-of-care testing.

Central Venous Catheter

A central venous catheter is often placed for monitoring central venous pressure and administering vasoactive drugs. Use of a pulmonary artery catheter can be helpful in the setting of ventricular dysfunction, severe pulmonary hypertension, and compromised renal function.

Transesophageal Echocardiography

Transesophageal echocardiography (TEE) is highly sensitive for detecting myocardial ischemia and regional wall-motion abnormalities. TEE is also useful for monitoring intravascular volume status and helping to detect hypovolemia or hypervolemia. TEE can be also used to diagnose thoracic aortic pathology, such as severe atheromatous disease or dissection.

Urinary Catheter

Catheterization of the bladder is performed after induction of general anesthesia to measure urine output and to assist in goal-directed fluid therapy.

Temperature Monitoring

The temperature probe in the urinary catheter or an esophageal temperature probe can be used to monitor core temperature and maintain normothermia. Patient-warming devices and fluid warmers are used to prevent hypothermia and shivering, thus avoiding potential complications such as myocardial ischemia and coagulopathy. Forced air warming in the lower extremities should be turned off during aortic cross clamping to avoid second- and third-degree burns due to skin hypoperfusion and heat dissipation.

Induction

Thoracic epidural analgesia is often used after elective AAA repair. For this technique, the catheter is often placed before induction of general anesthesia and may be used during the procedure for pain management. General anesthesia can be induced in a standard fashion for elective cases.

For emergency AAA repair, the surgical team should be present at the time of induction. Loss of abdominal muscle splinting and introduction of positive-pressure ventilation may exacerbate reduced venous return resulting from hypovolemia. Some form of rapid-sequence induction is typically employed due to gastroparesis and an increased risk of aspiration; a combination of etomidate, fentanyl, and succinylcholine is reasonable for this purpose. Scopolamine can be used to produce amnesia in hemodynamically unstable patients. Benzodiazepines, opioids, and inhalational anesthetics should be titrated slowly.

Fluid resuscitation and vasopressors should be administered cautiously before aortic cross clamping; aggressive resuscitation can disrupt hemostatic clot and exacerbate bleeding, and aggressive early resuscitation has been associated with worse outcomes in animal studies. After induction, central venous access can be

established and a pulmonary artery catheter inserted if the hemodynamic status allows. If severe persistent hypotension occurs, surgical intervention should proceed rapidly; monitoring and fluid resuscitation lines can be placed later in the procedure. Placement of an aortic cross clamp as soon as possible after induction is the most important determinant of survival in such patients.

Maintenance

General anesthesia can be maintained with inhalational anesthetics, total intravenous anesthesia, or both. For elective open AAA repair, a combination of epidural and general anesthesia has been shown to improve mortality. Arterial blood gases should be monitored at regular intervals for the presence of acidosis, electrolyte abnormalities, or anemia.

Incision and Aortic Exposure

Surgical exposure of the abdominal aorta is usually achieved via the left retroperitoneal or midline transperitoneal approach (Fig. 34.2).

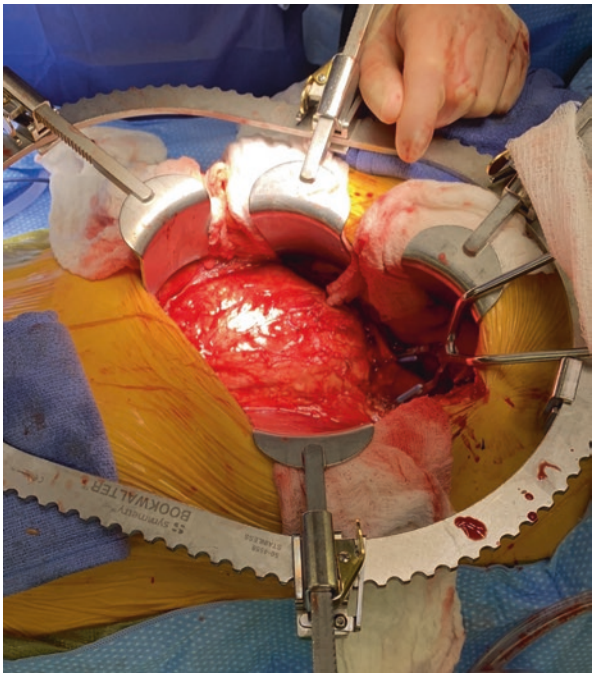

Fig. 34.2 Image of surgical exposure of open repair of abdominal aortic aneurysm before aortic cross clamping

Management of Aortic Cross Clamping

The degree of hemodynamic disturbance during aortic cross clamping depends on the location of the clamp. Infrarenal clamping is well-tolerated in most patients. Suprarenal clamping produces greater hemodynamic changes; thoracic clamping may produce severe hypertension and myocardial ischemia. Heparin (70 IU/kg) is given just prior to application of the aortic clamp to achieve activated clotting time of >200 s.

Application of the aortic cross clamp results in a sudden increase in systemic vascular resistance, arterial blood pressure, and left ventricular wall tension (Fig. 34.3).

The effect on preload is inconsistent, but it may be associated with an increase in filling pressures; stroke volume and cardiac output may decrease.

The increase in afterload and resulting systolic hypertension (>180 mmHg) can be managed by increasing the concentration of volatile inhalation anesthetic. Rapidly acting vasodilators (nitroglycerine, nitroprusside, nicardipine) should be

Fig. 34.3 Diagram of hemodynamic changes with aortic cross clamping

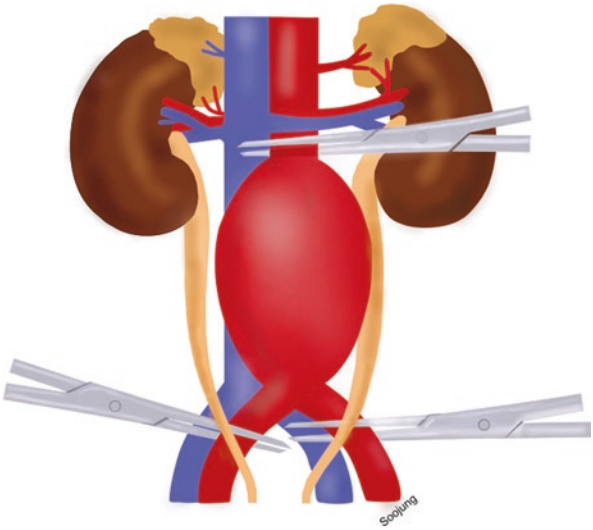

Fig. 34.4 Illustration of open repair of abdominal aortic aneurysm with the aorta and both iliac arteries are clamped

available for titration as needed. The vasodilating agents used should be short-acting, as hypovolemia and hypotension may occur rapidly after opening the aortic sac.

Immediately after the proximal aorta is clamped, the surgeon will clamp both iliac arteries to isolate the abdominal aneurysm cavity (Fig. 34.4).

Renal Dysfunction Prevention During Aortic Cross Clamping

Reduced renal blood flow from aortic cross clamping followed by the rapid restoration of blood flow from unclamping is associated with the release of free radicals and leads to ischemia–reperfusion injury of the kidneys. The severity of postoperative acute kidney injury depends on the following:

- Preexisting renal dysfunction
- The location of aortic cross clamp placement infrarenal vs juxta- or suprarenal as the latter is associated with embolization of atheroma load to the renal arteries
- Aortic cross clamping time. Short ischemia time (less than 20 min) has less damaging effects than long ischemia time (more than 50 min)
- Anemia and tissue hypoxia
- Development of abdominal compartment syndrome

Measures for renal protection may include the following:

- Intraoperative renal cooling
- Administration of mannitol (12.5–25 g) for free radical scavenging just prior to clamping
- Goal-directed fluid therapy

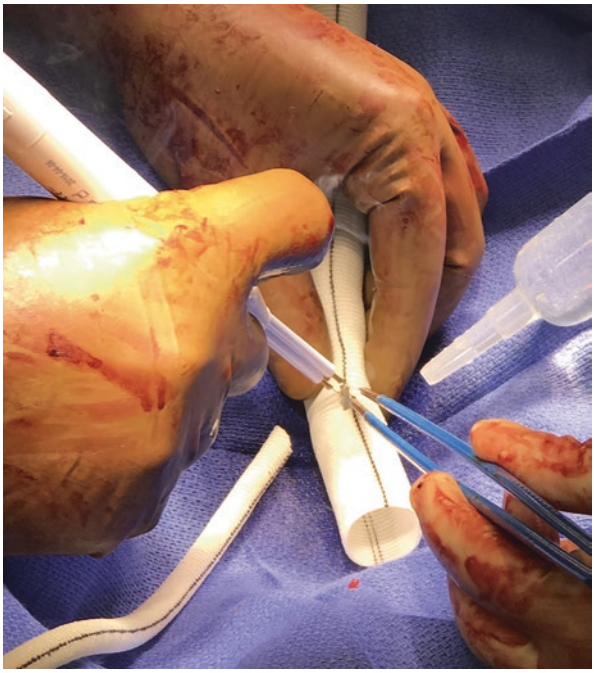

Fig. 34.5 Image of aortic synthetic graft preparation with renal branch

Aortic Graft Placement

Once isolated, the aneurysmal sac will be opened. At this point, vigorous back-bleeding commonly occurs from collateral flow to the lumbar arteries; blood loss can be rapid and sustained until the orifice of each lumbar artery is identified and over-sewn. Avoiding severe hypertension during this period may reduce collateral bleeding and assist with surgical exposure. However, as hypovolemia develops, the anesthesiologist must carefully titrate any therapy aimed at reducing afterload.

Active warming applied to the lower body should be discontinued during the period of aortic cross clamping to reduce tissue damage from decreased perfusion. An aortic synthetic graft of appropriate size and shape is prepared (Fig. 34.5). Anastomosis of the graft to the proximal segment of the aorta is performed first, followed by suturing the graft to the distal segment of the aorta.

Management of Aortic Unclamping

After vascular grafting is complete, release of the arterial clamps follows the same pattern as their application. The aortic clamp is released first so the integrity of the aortic anastomotic line can be assessed. After this, the iliac arteries are unclamped one at a time, allowing the anesthesiologist to accommodate to each change in hemodynamic state in a staged fashion.

Reperfusion of each iliac artery leads to a sudden decrease in systemic vascular resistance, venous redistribution, and release of metabolic acids and byproducts

of ischemia, which may have vasodilating and negative inotropic effects (Fig. 34.6). Hyperkalemia, malignant arrhythmias, and cardiac arrest may occur in extreme cases.

Before the arterial cross clamps are released, the patient's physiology should be optimized in anticipation of the impending hemodynamic effects. Hypercarbia, acidosis, and hyperkalemia should be treated aggressively during the period of cross clamping. Calcium chloride (1 g) and an ampule of sodium bicarbonate is usually

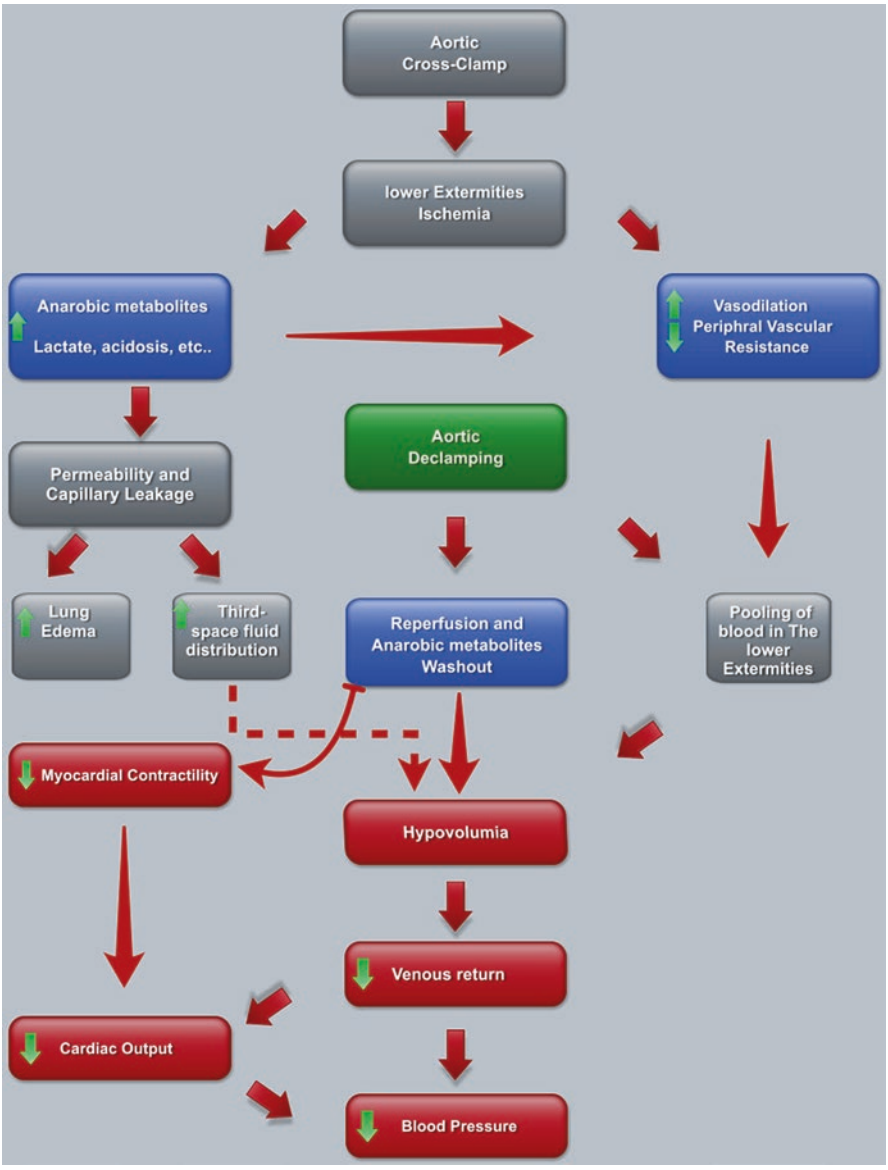

Fig. 34.6 Diagram of hemodynamic changes with aortic unclamping

given as the aortic cross clamp is removed. Intravascular volume should be supplemented, beginning shortly before the end of cross clamping period. Red blood cells should be administered if hemoglobin is ≤8 mg/dL. A bolus dose of a vasopressor may be administered just as the aortic cross clamp is removed, and a continuous infusion should be prepared. Inotropic agents may also be required during reperfusion. Metabolic acidosis is treated primarily with hyperventilation. If severe refractory hypotension persists after unclamping, the aortic cross clamp may have to be reapplied while hypovolemia, vasodilation, and acid-base abnormalities are aggressively corrected. Communication with the surgeon on a continuous basis is of paramount importance during this phase of the operation.

Abdominal Closure and Emergence

During this phase, the surgeon reapproximates the aneurysm sac using absorbable sutures to completely wrap the graft with the native aortic wall (Figs. 34.7 and 34.8). Next, while the surgeon is performing abdominal closure, and after hemodynamic stability is achieved, postoperative pain control (epidural infusion) and preparation for tracheal extubation are pursued.

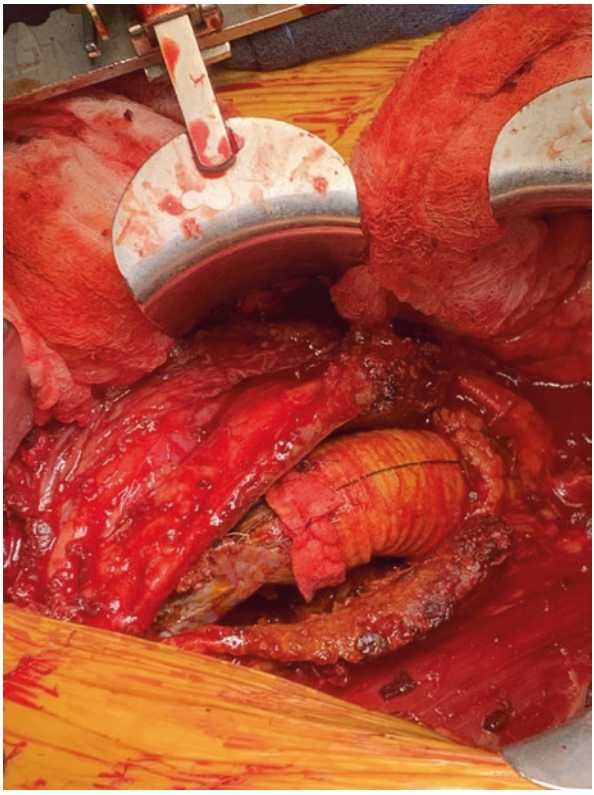

Fig. 34.7 Images of a patient after graft repair of abdominal aneurysm

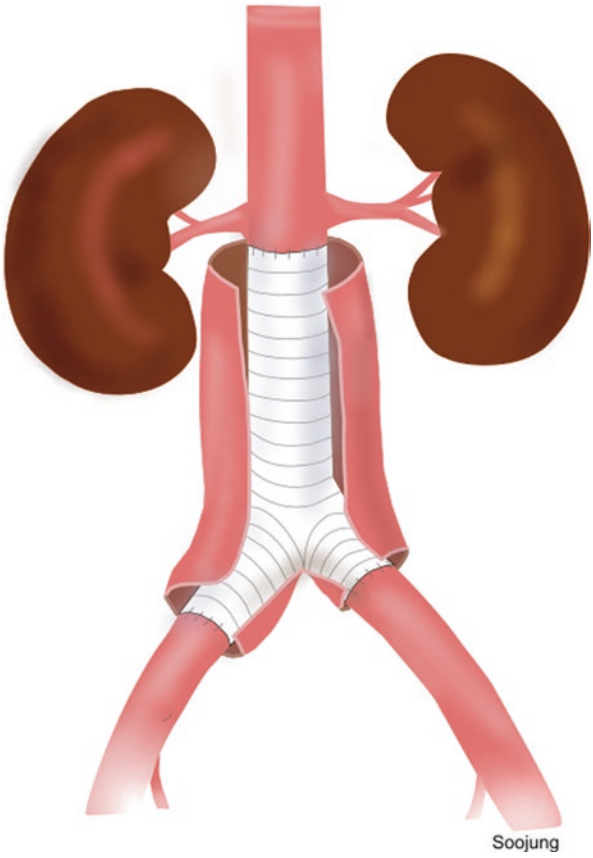

Fig. 34.8 Illustration of graft repair of abdominal aneurysm

Postoperative Management

Coagulopathy

After surgical repair, coagulopathy may persist despite initial transfusions of fresh-frozen plasma, platelets, and cryoprecipitate. Coagulation studies, such as prothrombin time, activated partial thromboplastin time, and thromboelastography, should be obtained and used to guide further management. Maintenance of normothermia is necessary during and after surgery to prevent coagulopathy and dysrhythmia.

Cardiac Complications

Postoperative myocardial infarction (MI) is the most common cause of death after AAA repair. The diagnosis is frequently made immediately postoperatively, based

on symptoms, ECG changes, and elevated serum cardiac enzyme values. An ST-segment elevation-MI (STEMI) may require percutaneous or surgical intervention. Non-STEMI can often be managed medically, with heparin, dual antiplatelet therapy, oxygen, analgesics, nitrates, beta blockers, and angiotensin-converting enzyme inhibitors.

Pulmonary Complications

Predisposing factors to pulmonary complications include advanced age, chronic obstructive pulmonary disease, cigarette smoking, and morbid obesity. Hypoventilation secondary to inadequate pain relief can be mitigated with thoracic epidural analgesia and avoidance of high-dose intravenous opioid medications. Early ambulation and frequent coaching in the effective use of incentive spirometry are the most effective means of avoiding postoperative atelectasis or pneumonia.

Renal Insufficiency

The reported incidence of renal failure after ruptured AAA repair ranges from 8% to 46% and is associated with an overall operative mortality rate of 57–97%. The most important risk factors for renal insufficiency under the control of the medical team are suprarenal cross clamping, aortic cross clamping >30 min, and prolonged periods of hypovolemia and hypotension in the perioperative period.

Neurologic Complications

The two most significant neurologic complications of open AAA repair are cognitive dysfunction and spinal cord ischemia. Risk factors for postoperative delirium include advanced age, preoperative depression, psychoactive medication use, and lower educational status. Intensive care unit delirium has been suggested to independently contribute to mortality and long-term cognitive dysfunction.

Spinal cord ischemia is a potentially devastating complication of abdominal aortic surgery; it is associated with significant morbidity and mortality, both in the immediate postoperative period and during convalescence. Spinal cord ischemia is believed to be caused by both intraoperative and postoperative spinal cord hypoperfusion as well as by reperfusion-associated cellular injury. Predictors of neurological deficits from spinal cord ischemia are total aortic clamp time, extent of aorta repair, presence of aortic rupture, patient age, proximal location of the aneurysm, and a history of renal dysfunction. For a detailed discussion of spinal cord ischemia after aortic aneurysm repair, please see Appendix 2 of Chap. 19.

Gastrointestinal Complications

Paralytic ileus commonly occurs secondary to bowel manipulation and fluid sequestration. Bowel ischemia secondary to hypovolemia and hypotension can lead to ischemic colitis; this can be prevented by maintaining adequate intravascular volume and cardiac output.

Limb Ischemia

Limb ischemia can occur secondary to anastomotic complications or distal thromboembolism. Placement of the iliac clamp before aortic cross clamping minimizes distal embolization of thrombi. Peripheral pulses should be evaluated before the patient leaves the operating room. Macroembolism can be treated surgically by thrombolysis or by amputation. Microembolism is managed by thrombolytic, anticoagulant, and antiplatelet therapy. Anesthetic management of open abdominal aortic aneurysm is summarized in Table 34.2.

Table 34.2 Summary of anesthesia management of open AAA

Preoperative	Mild sedation, e.g., midazolam
Location	Abdomen
Duration	2–4 hours
Anesthetic	GETA Epidural for postoperative pain control
Medications	5000+ units heparin at the discretion of the surgeon Notify at 3 min Notify at 1 h for possible re-dosing 25 g mannitol for suprarenal clamp
Drips	Phenylephrine, nicardipine
Positioning	Supine or Right lateral decubitus (bean bag, axillary roll, ASIS at bed break, bed cannot be reversed)
Cases specifics	Arterial line, central line Close discussion regarding clamping and unclamping Expected pressure increase with proximal clamp placement Expected pressure drop with distal clamp removal (reperfusion of legs) Avoid excessive fluid replacement, leads to postoperative fluid shifts, atrial fibrillation post-op day 3+ Communicate regarding lower urine output, may indicate problems with repair or need for diuretic at conclusion of case Communicate regarding BP drops, may indicate excessive retraction on IVC or unrecognized bleed Retroperitoneal exposures usually enter the left chest cavity with pneumothorax Cell-Saver required, estimated blood loss 1–2 L Foley catheter for monitoring urine output
Post-op care	Intensive care unit

Clinical Pearls

- Preoperative assessment for elective AAA repair requires careful risk stratification.
- Scores estimating morbidity and mortality include Revised Cardiac Risk Index, Glasgow Aneurysm Score, Duke Activity Status Index, and Hardman Index. They should be used in combination with clinical judgment.
- Emergency surgery is indicated for aneurysm rupture and carries a high perioperative mortality rate.
- Standard American Society of Anesthesiologists monitors, ECG, pulse oximetry, blood pressure, end tidal carbon dioxide monitor, temperature monitoring, arterial line, transesophageal echocardiography, central venous pressure, with or without pulmonary artery catheter, are routinely used intra-operatively.
- Ruptured AAA should be managed with conservative fluid resuscitation prior to aortic cross clamping to minimize the risk of hematoma disruption and further bleeding.
- Hypertension during cross clamping should be anticipated and managed with titration of anesthetics and intravenous vasodilators.
- The first stage of aneurysm repair is often accompanied by brisk blood loss from collateral flow to the lumbar arterial branches.
- Hypotension after releasing the aortic cross clamp is caused by hypovolemia, vasodilation, and release of byproducts of ischemia with negative inotropic effects.
- Hypotension is managed by strategic unclamping, pre-loading with volume, and titrating vasopressors and inotropes.

  Postoperative care is centered around achieving normothermia, normalizing coagulation status, and monitoring for cardiac, pulmonary, neurologic, or renal complications.

Further Reading

1. Chaikof EL, Brewster DC, Dalman RL, Makaroun MS, Illig KA, Sicard GA, et al. The care of patients with an abdominal aortic aneurysm: the Society for Vascular Surgery practice guidelines. J Vasc Surg. 2009;50(4 Suppl):S2–49.
2. Metcalfe D, Holt PJ, Thompson MM. The management of abdominal aortic aneurysms. BMJ. 2011;342:d1384.
3. Roizen MF, Fleisher LA. Anesthetic implications of concurrent diseases. In: Miller RD, editor. Miller's anesthesia. 7th ed. Philadelphia: Churchill Livingstone/Elsevier; 2010.

4. Gelzinis TA, Subramaniam K. Anesthesia for open abdominal aortic aneurysm repair. In: Subramaniam K, Park KW, Subramaniam B, editors. Anesthesia and perioperative care for aortic surgery. New York: Springer New York; 2011. p. 301–27.
5. Gelman S. The pathophysiology of aortic cross-clamping and unclamping. Anesthesiology. 1995;82:1026–57.
6. Sakalihasan N, Limet R, Defawe O. Abdominal aortic aneurysm. Lancet. 2005;365:1577–89.

Anesthesia for Lower Extremity Bypass

35

Lori Ann Edwards and Stephen A. McCaughan

It is a bad sign in acute illnesses when the extremities become cold.

—Hippocrates

Introduction

Most cases of arterial vascular disease of the lower extremities are due to obstructing atherosclerosis. Some other vascular diseases affecting the lower extremities are peripheral arterial aneurysms and pseudoaneurysms; systemic vasculitis; traumatic injury; and embolic arterial occlusion. Atheroma in atherosclerotic vessels can lead to varying degrees of occlusive peripheral arterial disease. The size of the atheroma and its location in the arterial tree define the clinical presentation. Occlusive peripheral arterial disease develops with the narrowing or complete blockage of the arteries, resulting in reduced blood flow. Symptoms start when the collateral blood flow can no longer meet the oxygen requirements of the tissues distal to the blockage. The acuity of clinical presentation depends on whether the reduction of blood flow is gradual or abrupt. Critical limb ischemia is a serious condition that requires prompt intervention. The choice of surgical interventions is determined by the anatomical location of the occlusion; the presence of collateral circulation; and the etiology of the occlusion (thrombus vs embolus).

Management strategies and surgical techniques for treating arterial disease of the lower extremity include the following:

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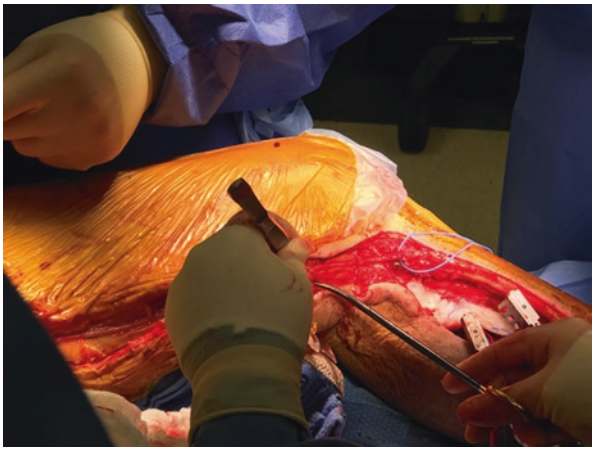

Fig. 35.1 Lower extremity revascularization using distal femoro-popliteal bypass with a reversed saphenous vein graft. First, the saphenous vein is harvested, and all tributaries are tied using longitudinal incision. Next, the common femoral artery and the popliteal arteries are exposed. Then the reversed saphenous vein is positioned in a tunnel created alongside the artery. Finally, proximal and distal anastomosis are performed

1. Angiography to delineate the arterial anatomy and assess the severity of the disease
2. Endoscopic balloon angioplasty and stenting, which can be approached through an open cutdown or percutaneous technique
3. Open surgical revascularization with bypass grafting (Fig. 35.1)
4. Combination therapy (hybrid)

Elective lower extremity bypass grafting procedures can be divided into two types based on the location of the vascular obstruction: aortoiliac disease or infrainguinal disease. Aortoiliac disease is at the level of the bifurcation of the aorta or the common iliac arteries; infrainguinal disease is at the level of the femoral or popliteal artery. Indications for lower extremity bypass include the following:

- Claudication
- Limb ischemia at rest
- Vascular anatomy not amenable to percutaneous interventions

Assessment and management of ischemic disease of the lower extremities are noted in the flow diagram in Fig. 35.2.

Surgical option for aortoiliac disease is bypass grafting, which includes anatomical and extra-anatomical grafting. Anatomical grafting with aorto-bifemoral or aorto-unifemoral grafts is usually performed through the transabdominal approach; extra-anatomical grafting is performed through a subcutaneous tunnel with a synthetic woven Dacron graft. The two extra-anatomical alternatives are (1) axillobifemoral or uni-femoral grafts and (2) femoro-femoral bypass grafts. The surgical options for treatment of infrainguinal arterial occlusive disease are femoro-popliteal bypass and endarterectomy of the femoral arteries. Because, patients with these diseases have significant multiple comorbidities, anesthesia induction can be particularly challenging for the anesthesia provider. Their perioperative care is geared toward reducing the risk of morbidity and mortality, especially of major cardiac events.

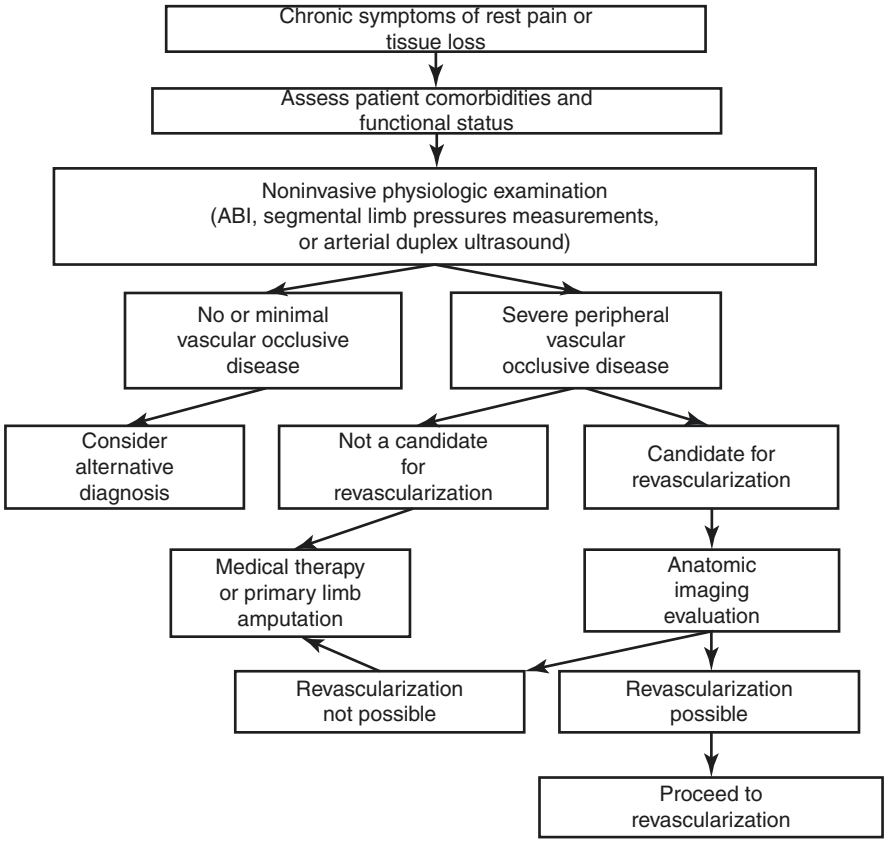

Fig. 35.2 Flow diagram of diagnostic approach to revascularization. (Tomita TM, Kibbe MR. Diagnostic approach to chronic critical limb ischemia. In: Dieter R, Dieter Jr R, Dieter III R, Nanjundappa A, editors. Critical limb ischemia. Cham: Springer; 2017)

Preoperative Assessment and Management

Most patients with peripheral vascular disease are elderly and vasculopathic, with many having comorbidities. A great number of these patients may have required multiple surgical procedures in the past. If so, their prior anesthesia records should be reviewed thoroughly. Preoperative assessment and risk stratification for cardiac and non-cardiac patients are discussed in Chap. 5.

Preoperative Optimization

All patients undergoing surgical intervention for peripheral vascular disease should have their medical conditions reviewed carefully, which may reveal that their care can be improved. The perioperative period is an important time for the review of

patients' overall health status. Studies have shown that preoperative optimization not only decreases the immediate risk of complications but also reduces morbidity and mortality over longer periods of time. However, surgery should only be delayed if an improvement in the patient's medical condition can be reasonably expected; preoperative optimization should never delay surgery for limb-threatening ischemia. Many patients with vascular disease are treated with anticoagulants. For such patients, if neuraxial anesthesia is planned, an international normalized ratio of <1.5 is required for safe conduct of anesthesia. Use of low-dose aspirin is considered safe in the perioperative period.

Medically optimizing patients with comorbidities is discussed in detail in Chap. 7.

Intraoperative Management

Lower extremity arterial bypass can be performed successfully with general, neuraxial, or regional anesthesia. The choice of these methods varies among centers, with no validated advantage associated with specific technique. Numerous studies have sought to determine if the choice of primary anesthetic affects clinical outcome, but no method has been conclusively associated with superior graft patency or fewer perioperative cardiac or pulmonary complications. Although careful, well-planned anesthetic management may improve patient outcome, the patient's primary disease and comorbidities and the operative technique used are more influential factors.

Therefore, the choice of anesthetic technique may be guided by individual patient's comorbid disease. Advantages and disadvantages that should be considered when formulating an anesthetic plan are listed in Table 35.1.

Table 35.1 Considerations when selecting anesthetic technique

	General anesthesia	Neuraxial anesthesia
Analgesia	Requires abrupt transition to postoperative analgesia on emergence	Gradual block resolution permits gradual transition to postoperative analgesics
Hemodynamics	Induction and emergence are associated with fluctuations in heart rate and blood pressure	Reduced blood pressure may compromise regional blood flow
Myocardial ischemia	Induction and emergence may be associated with demand ischemia	Increase in sympathetic tone above the block may exacerbate myocardial ischemia
Pulmonary function	May be required for patients with severe pulmonary disease who are unable to lie supine	Preserves diaphragmatic function, resulting in less postoperative atelectasis
Neurological function	May be preferred in patients with baseline cognitive impairment and difficulties cooperating	Lower incidence of postoperative delirium
Coagulation system	No restrictions	May be contraindicated by recent anticoagulant use

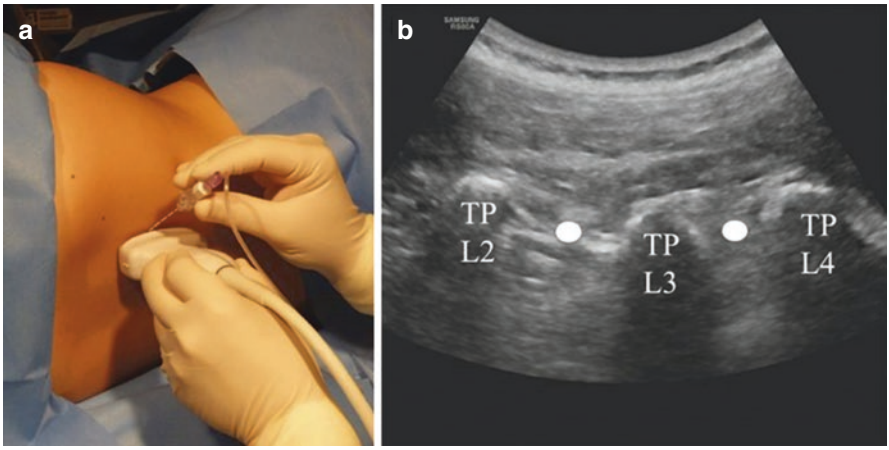

Fig. 35.3 Karmakar approach for ultrasound-guided lumbar plexus block (**a**). Patient is in the lateral decubitus position. (**b**) Sonoanatomy. The location of the tip of the needle is indicated by the white dots (between the "teeth of the trident"). *TP* transverse process. (Puntillo F, Bertini L, Bosco M, Tedesco M, Baciarello M. US-guided nerve blocks: procedure technique. In: Silvestri E, Martino F, Puntillo F, editors. Ultrasound-guided peripheral nerve blocks. Cham: Springer; 2018)

Non-neuraxial regional anesthetics also may be used for lower extremity revascularization. One such technique is continuous lumbar plexus block given via catheter infusion of local anesthetic as the primary anesthetic. For postoperative pain management, peripheral nerve blocks can be used. The use of real-time ultrasound-guided lumbar plexus block improves clinical efficacy and reduces the failure rate of the block. Two methods of performing lumbar plexus block are the paramedian longitudinal approach (Karmakar approach; Fig. 35.3) and the paramedian transverse approach (shamrock method; Fig. 35.4). Use of the lumbar plexus technique avoids sympathectomy and its associated hypotension, but the risk of systemic local anesthetic toxicity is slightly higher than that of neuraxial regional anesthesia because of a larger volume of local anesthetic agent required. As with neuraxial anesthetics, the risks versus benefits must be weighed carefully in patients who have recently used anticoagulants.

Monitoring

The goal of monitoring is to quickly identify and rapidly intervene to correct cardiopulmonary abnormalities. Standard American Society of Anesthesiologists (ASA) monitors are required regardless of the anesthetic technique employed. The monitors should include five-lead electrocardiography (ECG) with ST-segment monitoring; non-invasive blood pressure monitoring; pulse oximetry, gas analysis; capnography; and temperature monitoring.

Invasive arterial pressure monitoring is often required. Beat-to-beat measurement of blood pressure may be less important than the access provided for blood sampling

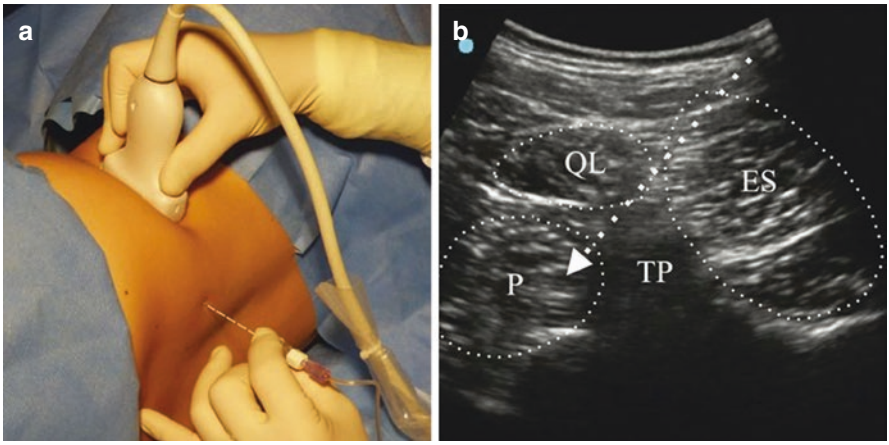

Fig. 35.4 Shamrock method for ultrasound-guided lumbar plexus block. (**a**) The patient is in the lateral decubitus position. (**b**). The sonoanatomy. *P* psoas muscle, *ES* erector spinae muscle, *QL* quadratus lumborum muscle, *TP* transverse process of L4. The nerve is in the medial and posterior part of the psoas muscle. The long white arrow indicates the needle direction, and the target point is the tip of the arrow. (Puntillo F, Bertini L, Bosco M, Tedesco M, Baciarello M. US-guided nerve blocks: procedure technique. In: Silvestri E, Martino F, Puntillo F, editors. Ultrasound-guided peripheral nerve blocks. Cham: Springer; 2018)

throughout the procedure. Frequent measuring of glucose, activated clotting time, hemoglobin, and arterial blood gases to assess acid-base status are often required.

Placement of a Foley catheter for monitoring urine output may provide some guidance in fluid management and is necessary for bladder drainage during long revascularization procedures.

Intravenous access in patients undergoing lower extremity bypass requires a minimum of two large-bore IV lines. A multi-lumen central venous catheter should be considered if need for vasopressors is anticipated, or if peripheral venous access is inadequate for administration of large volumes. Fluid-responsiveness monitoring devices can be used to monitor goal-directed fluid management; PiCCO®, LiDCO®, and Vigileo® are examples of such devices. Rapid blood and fluid infusion devices are not uncommonly required. A pulmonary artery catheter may be warranted in patients with a history of renal failure or congestive cardiac failure. In such patients, trends in filling pressures and cardiac output may guide resuscitation during significant blood loss.

Intraoperative Goals

Hemodynamic stability and adequate perfusion of vital organs during major vascular surgery help prevent major cardiac events.

A reasonable intraoperative goal is arterial blood pressure maintained within 20% of the ambulatory preoperative value. Hypertension will increase left

ventricular wall tension and may result in demand-based myocardial ischemia. Hypotension may cause hypoperfusion of arterial beds distal to focal atherosclerotic lesions, causing supply-based myocardial ischemia or adverse central nervous system events. Transient hypotension, caused by the release of vasodilating and myocardial depressant factors from ischemic tissue, should be anticipated after reperfusion of an ischemic limb and treated promptly.

Tachycardia can be especially detrimental in patients with obstructive coronary artery disease. As heart rate increases so does myocardial oxygen demand and diastolic coronary artery perfusion time decreases. The simultaneous increase in oxygen demand and decrease in oxygen delivery may result in myocardial ischemia. Conditions associated with tachycardia such as hypoxia, hypercarbia, inadequate depth of anesthesia, anxiety, and hypovolemia should be identified and treated appropriately.

Hemodynamic changes in transabdominal aorto-femoral bypass procedures are not prominent, but clamping and unclamping of the aorta may cause marked cardiovascular effects because of the absence of collateral vessels to the lower extremities. A staged unclamping can minimize the cardiovascular impact of this maneuver. Maintenance of normothermia with warming devices and administration of warm fluids is important. Hypothermia can lead to peripheral vasoconstriction and shivering, either intra-operatively or post-operatively. The increase in cardiac output induced by shivering and increase in vascular resistance can result in a rapid and dramatic increase in myocardial oxygen demand, which increases the risk of adverse perioperative cardiac events.

Significant blood loss should be anticipated as a possibility. Blood transfusion may be required to maintain oxygen carrying capacity and hemodynamic stability. It is advisable to maintain the hemoglobin concentration above 9–10 g/dL in patients who have a diminished capacity to increase cardiac output or whose myocardium is at risk for ischemia.

Postoperative Management

Prolonged stay in the post-anesthesia care unit or admission to the intensive care unit (ICU) may be required after lower extremity revascularization to allow for frequent neurovascular evaluations. Early graft failure is a risk following peripheral arterial bypass and may require emergency re-exploration. ICU admission may also be necessary for prolonged mechanical ventilation in patients with advanced pulmonary disease, need for hemodynamic support, or for the management of other comorbidities.

The risk of major cardiac or cerebrovascular events continues well into the postoperative period. Perioperative myocardial infarction may occur in as many as 15% of patients undergoing lower extremity bypass when aggressive post-operative surveillance is performed. Accordingly, rigorous blood pressure control, treatment of tachycardia, avoidance of anemia, and maintenance of intravascular volume are essential during recovery from surgery. Attention to these factors may improve the chances of graft patency and decrease the risk of postoperative morbidity and mortality.

Effective postoperative pain management is very important. Early mobilization and ambulation may decrease the incidence of postoperative complications, but mobilization and ambulation can only be achieved with adequate analgesia. A combination of regional anesthesia, intravenous opioids, and non-narcotic analgesics is commonly used for postoperative pain management. Use of narcotic analgesics alone is not recommended because of the incidence of side effects. Short-term use of non-steroidal anti-inflammatory agents is usually well-tolerated, even in patients with compromised renal function at baseline. Local anesthetic techniques are gaining popularity. Anesthetic management of lower extremity arterial bypass is summarized in Table 35.2.

Table 35.2 Summary of anesthesia management of lower extremity arterial bypass

Location of bypass	Abdomen/groin/leg
Duration of operation	2–4 hours
Anesthetic	General endotracheal anesthesia/neuraxial block/regional
Medications	≤5000 units heparin at discretion of surgeon. Notify at 3 min of administration. Notify surgical team at 1 h for possible redosing
Pain	Moderate/severe: thoracic/lumbar epidural, lower extremity nerve blocks, intravenous analgesics
Positioning	Supine
Blood loss	Variable
Case specifics	±Arterial line, Central line; Foley. May require completion angiography if distal examination is not satisfactory
Modifications	Some patients may require arm-vein harvest, rendering that arm not usable for IV access
Postoperative care	ICU for aorto-femoral bypass patients; intermediate care for all others

Clinical Pearls

- Peripheral vascular disease is commonly associated with other serious morbidities, including coronary artery disease, cerebrovascular disease, pulmonary disease, and diabetes.
- Preoperative medical optimization decreases the immediate risk of complications and morbidity and mortality over longer periods of time. However, the risks and benefits of any delay in surgical intervention must be considered.
- The ACC/AHA guidelines recommend that additional non-invasive cardiac investigations should only be performed when the results will change management.
- Graft infection is a disaster, so timely intravenous administration of prophylactic antibiotics within 1 hour prior to incision is vital. Redosing should be done at 4 hours or if massive blood transfusion is initiated.

- First dose of heparin should be delayed at least 1 hour after needle or catheter placement if regional and neuraxial blocks are used.
- Ensure that coagulation status is within safe limits before inserting epidural catheters and carefully time the window of catheter removal with regard to anticoagulant therapy to avoid adverse events.
- The specific anesthetic technique employed is not as important as the general approach to perioperative management when attempting to minimize the risk of major perioperative complications.
- Standard ASA monitors are required regardless of the anesthetic technique employed. This should include five-lead ECG with ST-segment monitoring, non-invasive blood pressure monitoring, pulse oximetry, gas analysis, capnography, and temperature monitoring.
- The surgical approach requires supine positioning with inguinal, medial thigh, and/or distal leg incisions.
- Intraoperative complications include hypertension, hemorrhage, ischemic reperfusion syndrome, and hypothermia.
- Careful blood pressure control, treatment of tachycardia, avoidance of anemia, and maintenance of intravascular volume are important during recovery. Such factors may significantly affect graft patency as well as the risk of postoperative morbidity and mortality.
- Postoperative complications can include myocardial infarction, respiratory failure, infection, graft failure or occlusion, cerebrovascular accident, and need for amputation.
- Effective postoperative pain management is very important. A combination of regional anesthesia, intravenous opioids, and non-narcotic analgesics is commonly used.

Further Reading

1. Fraser K, Raju I. Anaesthesia for lower limb revascularization surgery. BJA Educ. 2014;15(5):225–30.
2. Sanford J, Atkinson B. Anesthesia for lower extremity bypass. In: Anesthesiology. Cham: Springer; 2018. p. 625–33.
3. Wesner L, Marone LK, Dennehy KC. Anesthesia for lower extremity bypass. Int Anesthesiol Clin. 2005;43(1):93–109.
4. Fleisher LA, Fleischmann KE, Auerbach AD, Barnason SA, Beckman JA, Bozkurt B, Davila-Roman VG, Gerhard-Herman MD, Holly TA, Kane GC, Marine JE. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2014;64(22):e77–137.

Anesthesia for Carotid Endarterectomy

36

George Hsu, Daniel Rosenbaum, and Grace Dippo

The study of the causes of things must be preceded by the study of things caused.

—Hughlings Jackson

Introduction

Stroke is the fifth most common cause of death and a leading cause of serious long-term neurologic disability in the United States. Approximately 80% of all stroke cases are ischemic in origin. Carotid artery disease contributes to 20%–40% of these, while atrial fibrillation and hypertension are responsible for the remainder. Atherosclerotic lesions leading to stroke or transient ischemic attack (TIA) occur most frequently in the common carotid artery at the bifurcation of the internal and external carotid arteries (Fig. 36.1).

Carotid endarterectomy (CEA), an operation in which the carotid vessels are clamped and the plaque in the carotid artery is removed, continues to be the gold standard to prevent ischemic stroke in patients with internal carotid artery stenosis and restore carotid revascularization. According to class I evidence derived from multiple clinical trials, CEA is performed in symptomatic or selected asymptomatic patients in the following clinical scenarios: when carotid stenosis is greater than 60%, when TIAs occurring in the presence of carotid stenosis is greater than 60%, or when TIAs occur with carotid stenosis despite anticoagulation.

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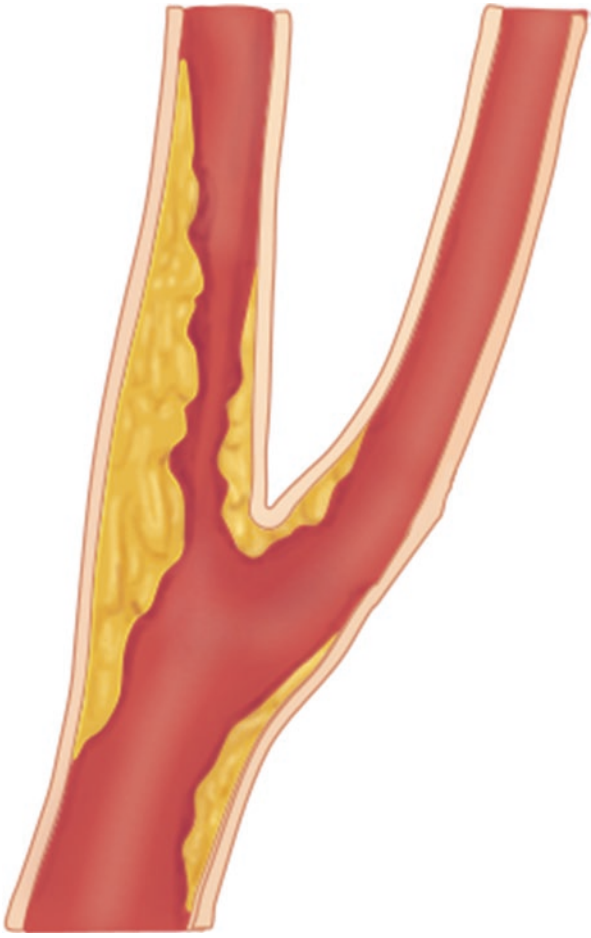


Fig. 36.1 Illustration of atherosclerotic lesions at the bifurcation

Preoperative Assessment and Management

Carotid artery disease is a manifestation of generalized arteriosclerosis, where the severity of the process parallels that of other major vessels, such as the coronary arteries. Since coronary artery disease is highly prevalent among patients with cerebrovascular occlusive disease, perioperative myocardial infarction is the major cause of morbidity and mortality following CEA. Patients undergoing surgical CEA can present significant challenges for the anesthesia provider because of their multiple comorbidities. The goal for perioperative care of these patients is to reduce the risk of morbidity and mortality, specifically preventing major adverse cardiovascular events (MACE) and cerebrovascular events. The anesthetic plan should be discussed with the patient, and informed consent for anesthesia and all related procedures should be obtained prior to surgery. The preoperative assessment and risk stratification for cardiac and non-cardiac patients are discussed in Chap. 5.

Preoperative Optimization

All patients undergoing CEA should be subject to a careful review of their comorbid medical conditions. The medical optimization of patients with comorbidities is discussed in Chap. 7.

Intraoperative Management

Case Setup

- Arterial Line.
- 2nd IV, preferably large bore (18 gauge or larger), on Ranger warmer tubing, with multiport stopcocks for multiple infusions.
- IV and arterial line extension tubing if bed and patient will be turned 180° for surgery.
- Premedication: preoperative anxiolytics such as midazolam are usually avoided when possible as they may interfere with a robust postoperative neurological exam and may contribute to emergence delirium in the elderly population.
- Induction meds: Usually fentanyl, lidocaine, and propofol are titrated to effect. Etomidate may be substituted for propofol if the patient has a very low cardiac reserve or ejection fraction. Rocuronium or succinylcholine are administered to facilitate intubation, dependent on airway exam and anticipated difficulty.
- Vasopressors such as phenylephrine and ephedrine are often used to maintain an adequate perfusion pressure during surgery. Vasodilators such as nicardipine (infusion starting at 2 mg/hour) or clevidipine may be employed to control hypertension.
- Other infusions: remifentanil (0.05–0.2 mcg/kg/min) for intraoperative analgesia and rapid emergence without residual opioid effect, dexmedetomidine (0.2–0.5 mcg/kg/hour) for additional depth of anesthesia and analgesia without hypotension but potential for bradycardia with loading bolus, and propofol at low doses (25–50 mcg/kg/min); however, propofol infusion may increase requirements for vasopressors in the elderly.
- Heparin 5000 U prior to carotid cross clamping.
- Airway setup: standard endotracheal tubes, corrugated extension tubing (gooseneck); neural integrity monitor electromyogram tracheal tube (NIM) may be needed if the vascular surgeon monitors recurrent laryngeal nerve function during surgery.

Monitoring

Standard ASA monitors, including noninvasive blood pressure, continuous waveform pulse oximetry, end-tidal capnography, gas analysis, temperature, and 5-lead electrocardiography (ECG) constitute minimum necessary monitoring. The ECG should include leads II and leads V5 for detection of arrhythmias and ischemia,

respectively. An arterial line is critical for beat-to-beat monitoring and proper management of blood pressure, particularly in patients who have abnormal cerebral autoregulation, and especially during periods of carotid cross clamping. The arterial line can also facilitate ease of blood sampling for laboratory analysis in the case of intraoperative complications. Placement of at least two large-bore IVs prior to surgery is important for assured IV access because the patient's arms are usually tucked to allow for ease of surgical access to the neck. Excellent IV access is maintained because multiple vasoactive infusions may be needed.

Positioning

The patient is positioned supine on the operating room table with upper body elevated slightly in reverse Trendelenburg or in the flexed position. A shoulder roll is placed transversely underneath the patient to achieve better extension of the neck. The head is placed within a soft gel donut-shaped head pad to support the head and prevent extreme neck extension. Then, it is rotated 45° to the contralateral side of surgery. The arms are tucked at the sides. The patient and bed are often turned 90° or 180° away from the anesthesia provider to allow the surgeon ease of access to the neck. Therefore, IV extension tubing, arterial line extension, and airway extensions are highly recommended to minimize accidental disconnections and extubation during bed-turning and surgery. If general anesthesia is chosen, an endotracheal tube should be used and secured on the contralateral side of the surgery to allow maximum surgical access to the neck, to avoid violation of the sterile field by airway equipment, and to minimize the chance of loss of secured airway.

Intraoperative Goals

CEA is associated with tremendous hemodynamic lability. Surgical manipulation of the carotid bulb and baroreceptors adds to hemodynamic instability. Maintaining hemodynamic stability and cerebral perfusion during the procedure is critical to reduce the risk of morbidity and mortality, specifically preventing stroke and MACE. Smooth and quick anesthesia emergence is essential to assess neurological function immediately postoperatively and to prevent blood accumulation in the neck. Glycemic control is extremely important because intraoperative hyperglycemia may exaggerate brain injury during the cross clamping. Since CEA is usually associated with minimal blood loss, intravenous fluid administration should be minimized to prevent discomfort and hypertension from bladder distention in an awake patient or congested lungs in diastolic dysfunction or renal failure patients. Vigilance for rapid blood pressure fluctuation allows the provider to maintain tight control of arterial blood pressure within 20% of the preoperative value using pre-spiked vasopressor and vasodilator infusions.

Options of Anesthetic Technique

General, regional, and local anesthesia have all been employed successfully in carotid surgery. The major advantages of general anesthesia are potential neurological protection (by decreasing cerebral oxygen consumption and demand) and control of airway should complications arise, while the advantage of regional anesthesia is the ability to monitor neurological status continuously in the awake patient. However, many patients are unable to tolerate the procedure while awake, and approximately 10% of patients refuse to have the surgery performed under regional anesthesia. According to the Surgical Quality Improvement Program database from the American College of Surgeons from 2005 to 2009, regional anesthesia was only employed in 15% of cases, and general anesthesia was used in 85% of cases. Although there seems to be a trend of lower mortality favoring regional anesthesia over general anesthesia, the literature thus far does not favor one over the other. Thus, the type of anesthetic can be individualized from case to case, considering the preferences of the surgeon, the patient, and the anesthesia provider. Of note, it is not recommended to perform the operation under regional anesthesia while the bed is turned away 90° or 180° from the anesthesia provider, as this positioning limits immediate access to the patient's airway.

Regional and Local Anesthesia

Regional anesthesia for CEA can be achieved with a superficial cervical plexus block, a deep cervical plexus block, a cervical paravertebral block, or a combination thereof, along with local anesthesia supplementation by the surgeon (Fig. 36.2). CEA can even be done under cervical epidural anesthesia. As discussed previously, regional anesthesia confers the benefit of continuous assessment of neurologic function intraoperatively. Additional benefits may include better postoperative analgesia, greater hemodynamic stability, and shorter hospitalization. Rare risks of regional anesthesia include infection, bleeding, local anesthetic toxicity, and very rarely, subarachnoid and phrenic nerve block. Injection of local anesthesia at the incisional site and in the surgical field is an important supplement to regional anesthesia in case of lack of local anesthetic spread due to variation in anatomy.

- Superficial cervical plexus block involves injection of local anesthetic along the posterior border of the sternocleidomastoid muscle. After intradermal injection of local anesthetics at the middle of the posterior border of the sternocleidomastoid, 5 mL of local anesthetic bupivacaine 0.25% is injected at this point using 1.5–2 in. needle. Another 10 mL of local anesthetic is injected after the needle is redirected superiorly and inferiorly along the posterior border of the sternocleidomastoid muscle with 5 mL on each side (Fig. 36.3).

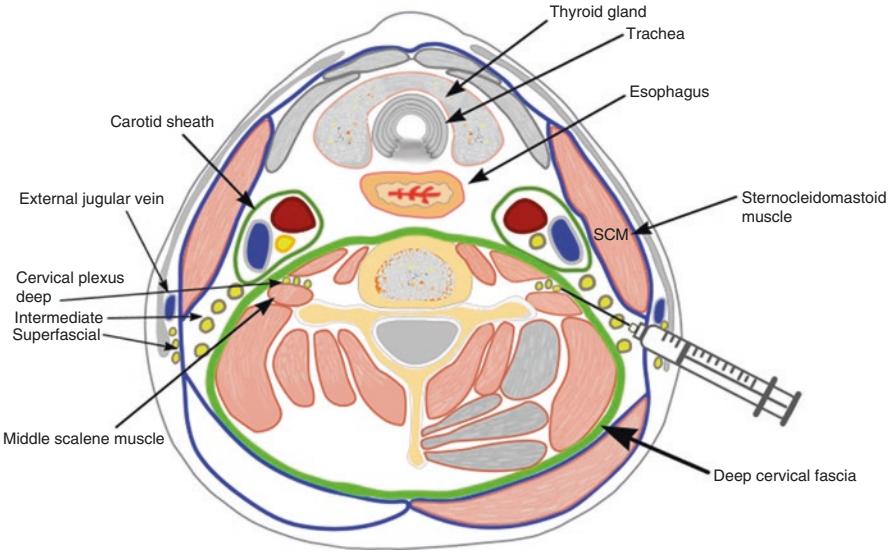
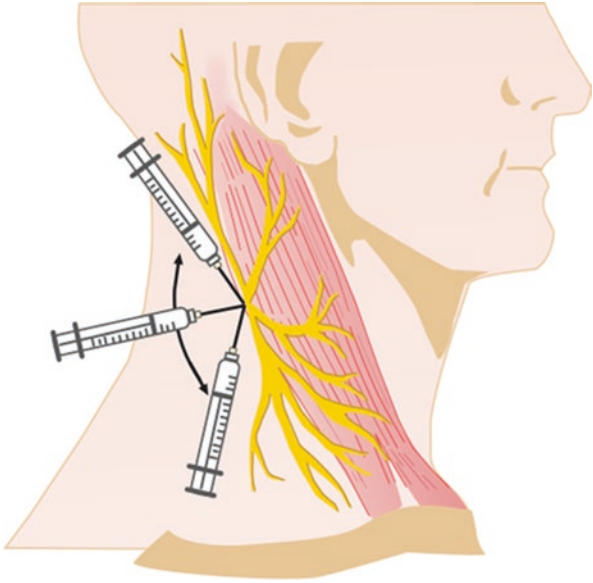


Fig. 36.2 Illustration of cervical plexus superficial, intermediate, and deep injection sites

Fig. 36.3 Illustration of site of injection of local anesthetic for superficial cervical plexus blocks



- Deep cervical plexus block includes a single injection of local anesthetic at the C3 or C4 transverse process or a three-injection technique at the C2, C3, and C4 transverse processes. It can be done using the anatomical landmark method (Fig. 36.4) or by an ultrasound-guided approach (Figs. 36.5 and 36.6). After skin preparation and draping, the C6 transverse process is palpated 1 cm behind the

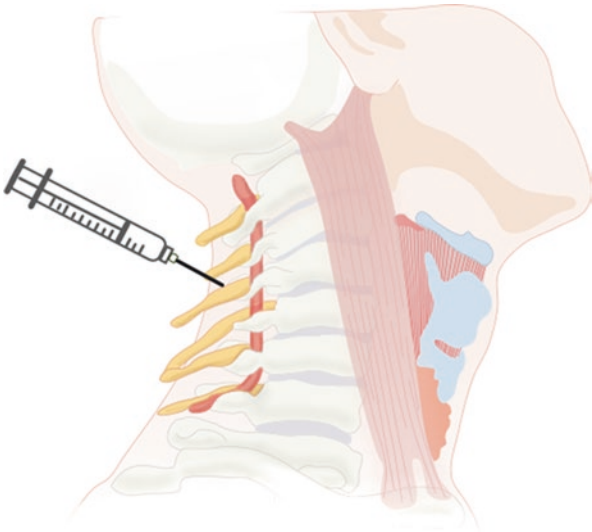


Fig. 36.4 Illustration of deep cervical plexus block

Fig. 36.5 A picture of ultrasound-guided cervical plexus block, transducer position, and scanning technique

posterior border of the sternocleidomastoid muscle at the level of the cricoid cartilage, then the C2, C3, and C4 transverse processes are identified. Three 22-gauge 1.5–2 in. needles are inserted at 2 cm depth, aiming slightly medially and caudally to avoid vertebral artery or spinal injection. The local anesthetic bupivacaine (0.25%, 3–4 mL) is given through each needle.

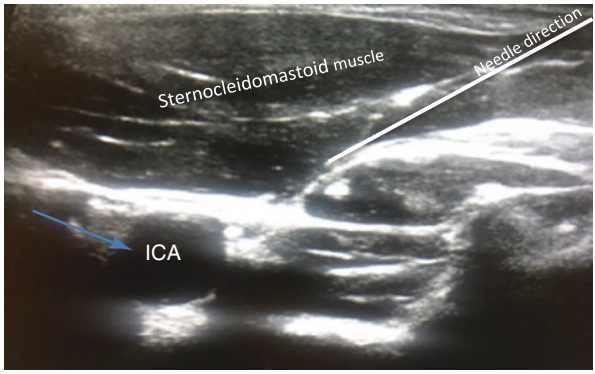


Fig. 36.6 Image of sonogram of the a C4 nerve root cervical plexus

- Cervical epidural anesthesia consists of initial access with an epidural catheter at C6–C7 or C7–T1 interspace followed by slow titration of local anesthetics and a short-acting opioid. An undesirable side effect is it produces a bilateral block.

Management of General Anesthesia

Induction

After application of standard ASA monitors, anesthesia may be induced in the patient with propofol or etomidate, a fast-onset muscle relaxant such as rocuronium, and lidocaine plus a short-acting opioid such as fentanyl to blunt hemodynamic response to tracheal intubation. After tracheal intubation, an arterial line and a second IV are established. The arterial line can be placed prior to induction if the patient has depressed cardiac function. Ketamine should be avoided or used sparingly due to its potential to cause tachycardia and hypertension in this patient population with a high incidence of comorbid coronary artery disease. Additionally, ketamine may interfere with neurologic monitoring by increasing the amplitude and latency of somatosensory-evoked potentials (SSEPs). High-dose opioid induction is undesirable since it has the potential to delay emergence and impair postoperative neurologic assessment.

Maintenance

Inhalational agents (sevoflurane, desflurane, and isoflurane) can be used safely for maintenance of anesthesia and may have the advantage of neuroprotection via uncoupling (increasing supply by cerebral vasodilation while decreasing demand by reducing cerebral metabolism). Total intravenous anesthesia with propofol and remifentanil may have advantages of greater hemodynamic stability and preserved cerebral autoregulation. However, propofol should be titrated to avoid suppression of the electroencephalogram (EEG) monitoring. A combination of inhalational and intravenous anesthetics is also appropriate in selected patients. Whether used in combination with inhalational agents or propofol, a remifentanil infusion is

recommended to provide intraoperative analgesia while allowing for immediate postoperative neurologic examination without residual opioid interference. Moreover, the use of a short-acting high-potency opioid can also reduce the anesthetic depth required for surgery and provide greater intraoperative hemodynamic stability. Dexmedetomidine is an effective adjunct as it can decrease anesthetic and opioid requirements without causing respiratory depression. Throughout the perioperative course, blood pressure should be maintained within 10%–20% of normal baseline to ensure adequate cerebral perfusion. If the blood pressure is low, addition of phenylephrine or norepinephrine infusion or bolus can maintain hemodynamics, while hypertension may be managed with a vasodilator such as nicardipine, clevidipine, or nitroglycerine.

Emergence

At the conclusion of surgery, a focused neurological examination is ideally performed prior to extubation to rule out intraoperative stroke. Deep extubation, although best for preventing coughing and bucking, is not recommended since a neurological examination would not be possible. Reversal of neuromuscular blockade can be achieved during skin closure. Titration of remifentanil to respiratory rate and a bolus of IV lidocaine (50–100 mg) prior to extubation may be helpful in minimizing coughing and bucking during the extubation process. Vasodilator agents are typically necessary to control the increased sympathetic tone during emergence and maintain systolic blood pressure of less than 150 mmHg or mean arterial blood pressure of less than 90 mmHg. Careful control of blood pressure during emergence can prevent cerebral hyperperfusion and neck hematoma. Boluses of esmolol (30–100 mg) may be helpful in controlling tachycardia, especially if the patient has co-existing coronary disease, to minimize myocardial strain and ischemia. Considerations when selecting anesthetic technique are noted in Table 36.1.

Table 36.1 Considerations when selecting anesthetic technique

	General anesthesia	Regional anesthesia
Pulmonary function	May be required for patients with severe pulmonary disease who are unable to lie flat	May affect diaphragmatic function
Anesthesia	Effective	Block may fail, risking need to convert to general anesthesia
Immobility	Effective	Patient may not stay stand still; claustrophobia can be a problem
Neurological function	May be preferred in patients with baseline cognitive impairment and difficulty cooperating	Ability to assess the patient's neurological status during cross clamping
Coagulation system	No limitations	May be contraindicated by recent anticoagulant use
Levels of CO_2	Controlled	Hypercarbia can develop
Shunt usage	Increased rate	Decreased rate

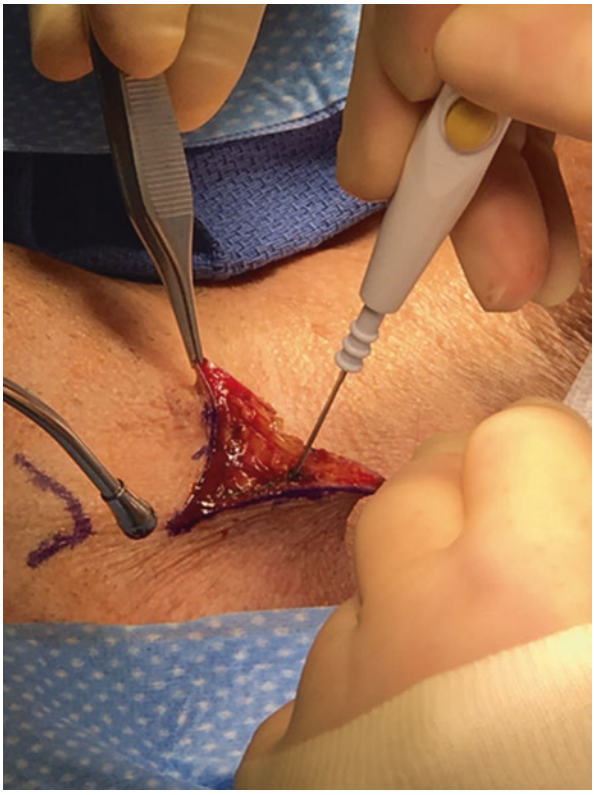


Fig. 36.7 A picture of step 5: Skin incision

Operative Technique

The steps of the procedure are briefly summarized:

1. EEG electrodes are placed on the scalp.
2. A baseline EEG is obtained.
3. GA is induced and lines are obtained.
4. The patient's position is optimized to ensure adequate surgical exposure.
5. Incision is made (Fig. 36.7), centered along the medial border of the sternocleidomastoid muscle.
6. Dissection continues to identify the common carotid artery, external carotid artery, and internal carotid artery, which are then isolated with an umbilical tape (Fig. 36.8).
7. An anticoagulant (5000 U of heparin) is administered to the patient.
8. After 3 minutes, the internal carotid artery is cross-clamped first to prevent any distal emboli from reaching the brain. The common carotid artery and external carotid artery are then clamped simultaneously to complete circulatory isolation of the surgical field.
9. Arteriotomy of the common carotid artery is made and extended to the internal carotid artery past the plaque.

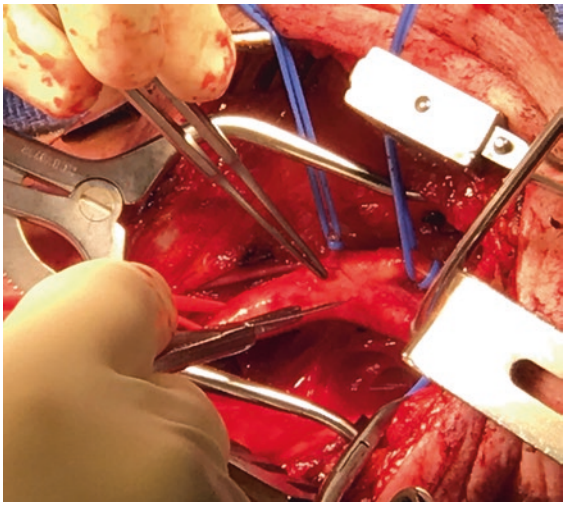


Fig. 36.8 A picture of step 9: Arteriotomy

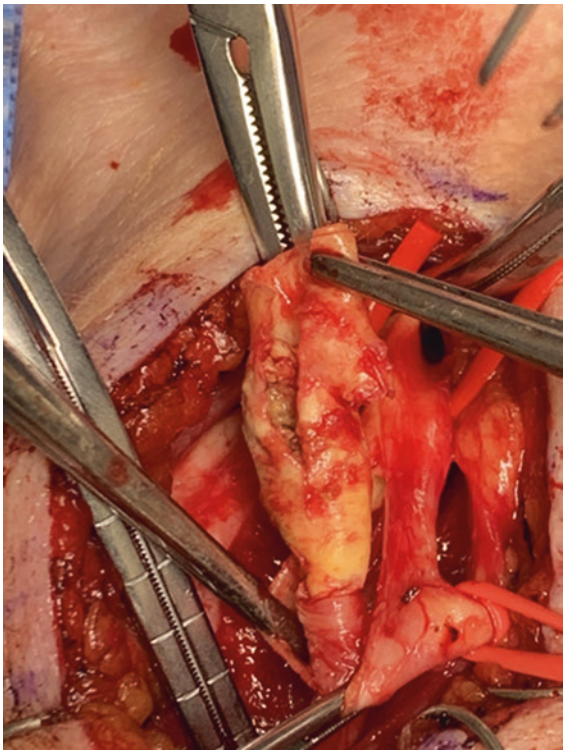


Fig. 36.9 A picture of step 10: A picture of plaque removal

10. The plaque is accessed with circumferential dissection (Figs. 36.9 and 36.10).
11. The arteriotomy is closed by direct closure with suture. An autologous or prosthetic patch may be used in vessel closure if there is concern for the internal carotid artery narrowing with direct closure (Fig. 36.11).

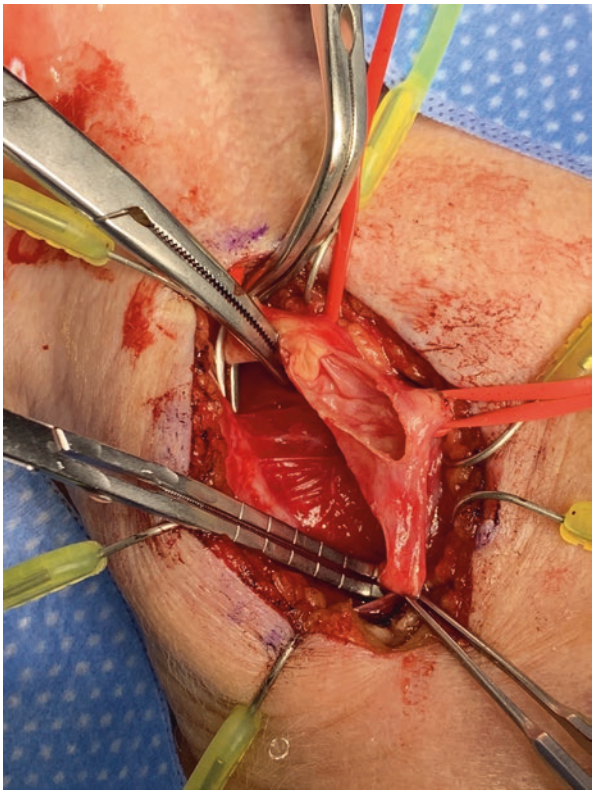


Fig. 36.10 A picture of the carotid after the plaque is removed

Special Intraoperative Consideration

Carotid Sinus Reflex

Surgical traction of the carotid sinus may result in bradycardia, bradyarrhythmia, hypotension, and even asystole via the baroceptor reflex. Immediate cessation of surgical manipulation is usually sufficient for prompt hemodynamic recovery, but atropine (0.2–0.5 mg) may be needed to treat persistent bradycardia and hypotension. Pretreatment with glycopyrrolate (0.1–0.2 mg) and/or surgical infiltration of the carotid sinus with lidocaine prior to surgical manipulation can be effective in preventing this reflex. However, lidocaine injection into the carotid sinus may cause the very same bradycardic response. If bradycardia, hypotension, or asystole persists despite these interventions, transcutaneous pacing and advanced cardiac life support should be initiated.

Carotid Cross Clamping

Cross clamping of the carotid artery is one of the most critical events during CEA. The carotid cross clamping technique creates a non-pulsatile and bloodless field to facilitate plaque removal. Prior to carotid occlusion, a heparin dose of 5000–7000 U, or 100 U/kg, is given systemically to reduce the risk of thromboembolism, with the goal of an activated clotting time (ACT) between 200 and 250 seconds. Cerebral perfusion is maintained via the collateral vessels in the circle of Willis (Fig. 36.12).

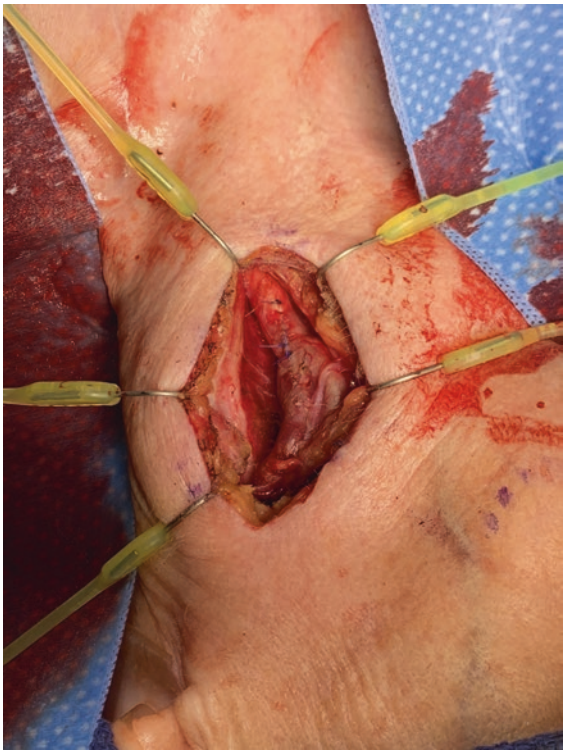


Fig. 36.11 A picture of step 11: the carotid after the arteriotomy is closed

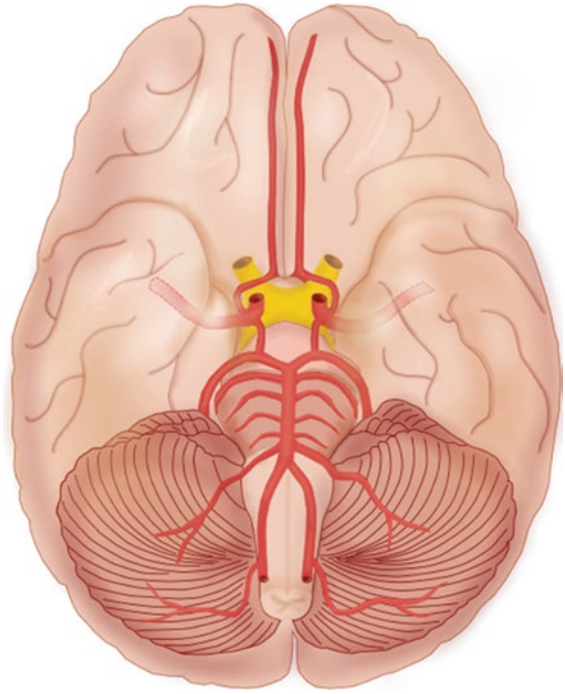


Fig. 36.12 Illustration of anatomy of the carotid arteries and circle of Willis

Around 15% of the population has a congenitally incomplete circle of Willis, and so regional blood flow may be compromised while the cross clamp is placed. Preexisting cerebrovascular disease in one or more vessels within the circle is also a risk factor for compromised perfusion. To optimize blood flow to cerebral tissues while the carotid cross clamp is in place, blood pressure should be at the patient's baseline or greater. Neurologic monitoring is an important tool for continuous assessment of overall cerebral perfusion and regional blood flow. If the neuromonitors show signs of cerebral ischemia, vasopressors may be needed to maintain perfusion. If pressors are inadequate for restoring cerebral perfusion, a shunt may be placed at the discretion of the surgeon. After the carotid cross-clamp is released, the surgeon may ask for heparin reversal with protamine. Routine reversal of heparin with protamine is controversial. Protamine seems to reduce the risk of postoperative bleeding; however, it may increase the risk of postoperative stroke. The typical dose is 1 mg of protamine for every 100 U of heparin administered. Protamine should be administered slowly and carefully in aliquots of 10 mg, as rapid administration often causes profound hypotension. Protamine is also known to cause anaphylaxis. Specifically, patients who have had cardiac surgery or who have used NPH insulin may be at risk due to sensitization with prior exposure. After release of the carotid cross clamp, blood pressure should be maintained at the patient's baseline or slightly lower to avoid cerebral hyperperfusion and resultant cerebral edema.

Carotid Shunting

The routine usage of carotid shunts during CEAs is controversial. However, if cerebral ischemia is detected after carotid clamping and persists despite efforts to maximize cerebral perfusion by increasing blood pressure, the surgeon can place a carotid shunt to restore adequate blood flow. The arterial shunt allows partial blood flow from the common carotid artery to the internal carotid artery. The overall incidence of stroke seems to remain the same whether or not a shunt is used. The incidence of embolic stroke, however, is increased with shunt placement, as are other complications such as cranial nerve damage, infection, and hematoma formation.

Neurologic Monitoring

Several types of neurologic monitoring can be utilized during CEA. These include assessment of the awake patient, EEG, SSEPs, motor-evoked potentials (MEPs), transcranial Doppler (TCD), bispectral index (BIS), near-infrared spectroscopy, and carotid stump pressure monitoring.

Assessment of the Awake Patient

As a direct monitor of neurologic function, frequent assessment of the awake patient is considered the gold standard. Neurologic assessment includes asking the patient to perform simple tasks such as squeezing hands or a bulb. The biggest disadvantage to this technique is that it necessitates a regional anesthetic.

Electroencephalogram

A trained neurophysiologist places the EEG wires and leads on the patient (Fig. 36.13) and interprets the electrical signals during surgery. The EEG readout can be affected by electrical interference, changes in anesthetic depth, type of anesthetic used, and changes in patient temperature. Abrupt generalized slowing seen on EEG shortly after carotid cross clamping is suggestive of cerebral ischemia. Compared to assessment of the awake patient, sensitivity for ischemic events with EEG monitoring is only about 60%. The reason for this relatively low sensitivity may be that the EEG waveform only reflects electrical activity generated by the cerebral cortex and not from deeper brain structures.

Somatosensory-Evoked Potentials and Motor-Evoked Potentials

After stimulation of either the median or the tibial nerve, the amplitude and latency of the signal over the cortex is measured and interpreted by a dedicated neurophysiologist. Although the sensitivity for detection of cerebral ischemia is reported to be 81%, SSEPs and MEPs are sensitive to inhalational anesthetics.

Transcranial Doppler

This technology utilizes the Doppler shift to measure changes in the blood velocity of the middle cerebral artery by placing a probe external to the temporal region. TCD is unique in that it can also detect thromboemboli via auditory variation. However, it is often difficult to obtain a signal, and the rate of failure to capture is between 10% and 21%.

Bispectral Index

BIS is a noninvasive monitoring device using EEG waveforms to assess the depth of anesthesia. Some reports suggest that it can predict cerebral ischemia after carotid artery clamping when there is a sudden change in EEG patterns. However, its main utility is for titrating the anesthetic depth and for reducing anesthetic-induced hypotension.

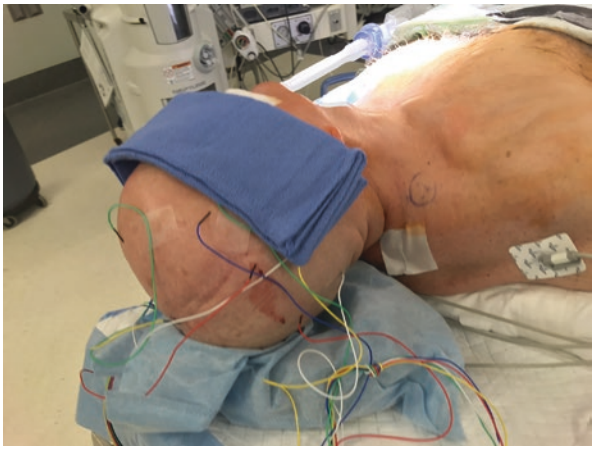


Fig. 36.13 A picture of somatosensory evoked potentials (SSEPs)

Near-Infrared Spectroscopy

Near-infrared spectroscopy is a noninvasive monitoring device that can be used to measure regional cerebral oxygenation. However, because of the conflicting reports about its predictive value, its use is not widespread.

Carotid Stump Pressure

The carotid stump pressure is obtained by inserting a small needle distal to the carotid cross clamp and transducing the arterial pressure. A stump pressure of 40–50 mmHg or above is considered adequate for cerebral perfusion. A carotid stump pressure of less than 40–50 mmHg would alert the surgeon to consider placing a shunt.

Postoperative Care

After extubation, patients are transferred to the post anesthesia care unit (PACU) or ICU for close observation of neurologic status and hemodynamics, dependent on postoperative concerns or medical comorbidities specific to the patient. Incisional pain from CEA is usually minimal and is well-controlled with short-acting opioids such as fentanyl. Hypertension, whether secondary to pain, sympathetic outflow, surgical denervation of carotid sinus receptors, or bladder distention, warrants immediate treatment. Uncontrolled hypertension has the potential to cause demand myocardial ischemia (MI), cerebral hyperperfusion syndrome, hemorrhagic stroke, or hematoma formation at the surgical site. Antihypertensives such as labetalol and hydralazine (both in 5 mg increments) are effective first-line agents. Alternatively, a nicardipine infusion may be continued or started postoperatively to ensure continuous control of hypertension. Hypotension secondary to hypovolemia, anti-hypertensives, and possibly increased sensitivity of the carotid sinus after surgical exposure, could lead to supply myocardial ischemia, cerebral hypoperfusion, or ischemic stroke and requires prompt treatment as well.

Complications

Myocardial Ischemia

As mentioned previously, MI is the major cause of morbidity and mortality after CEA, with an estimated incidence of 1%–2%. Most MIs after CEA are non-ST segment elevation MIs and confer poor long-term outcomes. Postoperative hyper- or hypotension may disrupt myocardial supply-demand balance and contribute to MI.

Stroke and Neurological Deficits

As the second most common cause of death after CEA, stroke occurs with an incidence of 1%–2% and up to 3–5%. The majority of intraoperative and perioperative strokes are thromboembolic, rather than ischemic or hemorrhagic. Even so, perioperative hypotension may contribute to cerebral ischemia, while perioperative hypertension may cause cerebral hemorrhage.

Neck Hematoma

Wound hematomas develop in 5%–8% of CEAs and 1%–3% require surgical reexploration. Uncontrolled intraoperative and postoperative hypertension, as well as rigorous coughing and bucking during emergence, may contribute to neck hematoma formation. Patients on perioperative anticoagulation may also be at risk for hematoma. Hematomas of arterial origin can be extremely dangerous due to their potential for rapid expansion and resultant airway compromise. Evidence of expanding neck hematoma or stridor in the PACU should prompt immediate surgical reexploration. Emergency endotracheal intubation may be required. Surgical airway equipment such as cricothyrotomy or tracheostomy kit should be available.

Cerebral Hyperperfusion Syndrome

Cerebral hyperperfusion syndrome (CHS) is a spectrum of clinical symptoms believed to be related to increased regional cerebral blood flow secondary to loss of cerebrovascular autoregulation and usually occurs in the first few hours after CEA. Loss of vasomotor tone, carotid baroreceptor denervation, and uncontrolled hypertension all contribute to the development of cerebral hyperperfusion. Symptoms of CHS include ipsilateral headache, eye pain, seizures, cerebral hemorrhage, and coma. Though uncommon with an incidence of 1%–5%, CHS carries a mortality rate of up to 40%. Therefore, it is imperative to maintain systolic blood pressure of less than 150 mmHg or mean arterial pressure less than 90 mmHg immediately after surgery and even for weeks postoperatively. Prompt investigation with TCD, MRI, or computed tomography (CT) scan may also be warranted to determine the degree of hyperemia. Control of systemic blood pressure, even when it is normal, with beta-blockers or clonidine remains the cornerstone for treatment of CHS.

Injury to the Recurrent Laryngeal, Hypoglossal, and Superior Laryngeal Nerves

Postoperative dysfunction of the recurrent laryngeal, hypoglossal, and superior laryngeal nerves due to surgical traction is usually transient. Dysfunction of the recurrent laryngeal nerve is characterized by hoarseness of voice. Bilateral damage to the recurrent laryngeal nerve can pose a risk of airway compromise but is theoretical not possible since surgery is unilateral, unless the contralateral nerve was compromised due to previous carotid or thyroid surgery.

Table 36.2 Summary of anesthesia management of carotid endarterectomy

Preoperative	No midazolam
Location	Neck
Duration	60–120 min
Anesthetic	GETA (rarely awake, which requires regional block)
Medications	5000+ U heparin at the discretion of the surgeon Notify at 3 min Notify at 1 hour for possible re-dosing Atropine ready in room, bradycardia with carotid bulb manipulation
Drips	Phenylephrine, nicardipine
Positioning	Supine; bilateral arm tuck, gel donut for head, shoulder roll
Cases specifics	Arterial line for strict continuous blood pressure monitoring Maintain high pressures while clamped (MAP 90s or SBP > 150), goal MAP 60–90 mmHg after clamp released Affix ET tube on side opposite surgical side (i.e., right CEA corresponds to ET tube secured on the left side) EEG monitoring requires placement of scalp electrodes and adjustment of anesthetic to prevent suppression of signals
Post-op Care	Intermediate care; no minimal opioids as needed; consistent neurological examination given the risk of stroke
Surgeon specifics	Yes/No EEG monitoring Clamp and sew Yes/No shunt Occlusion time ~ 8–30 min Selective shunt with patch Rotate bed 180 ° Completion angiogram

GETA general endotracheal anesthesia, *ET* endotracheal tube, *CEA*

Summary of Anesthetic Technique

We prefer to perform carotid CEA under general anesthesia to secure the airway since most of our surgeons turn the head of the bed to a 180° angle. Anesthetic management of CEA is summarized in Table 36.2.

Trans-Carotid Artery Revascularization (TCAR)

In certain patients with significant internal carotid artery tortuosity, or excessive calcification, the trans-carotid artery revascularization (TCAR), a relatively new procedure, can be used as an alternative carotid artery stenting to transfemoral filter-protected stenting instead of the standard open surgical procedure. It uses a special device (ENROUTE™ neuroprotection system) that temporarily reverses the direction of blood flow without the need to clamp the carotid artery, thereby avoiding potential dislodging of atheromatous debris to the brain while applying the clamp. The patient is positioned the same as for classic carotid endarterectomy. Exposure to the carotid artery is via a small transverse incision in the neck to dissect the common carotid artery (Fig. 36.14).

After exposure and isolation of the artery, a microcatheter is placed just below the common carotid bifurcation and arteriogram is performed to delineate anatomy (Fig. 36.15). A venous sheath is placed in the contralateral femoral vein and secured

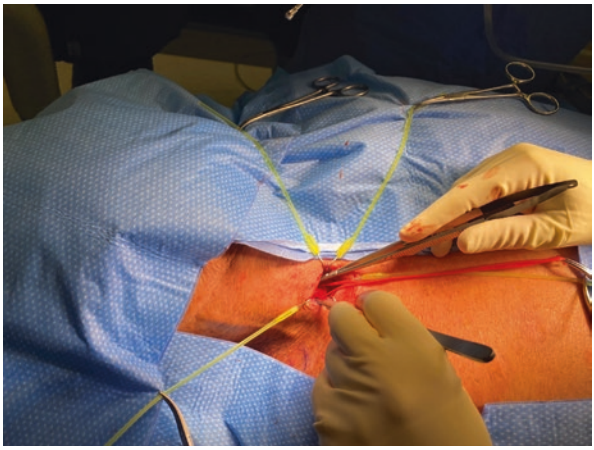


Fig. 36.14 A photo of exposure to the carotid artery is via a small transverse incision

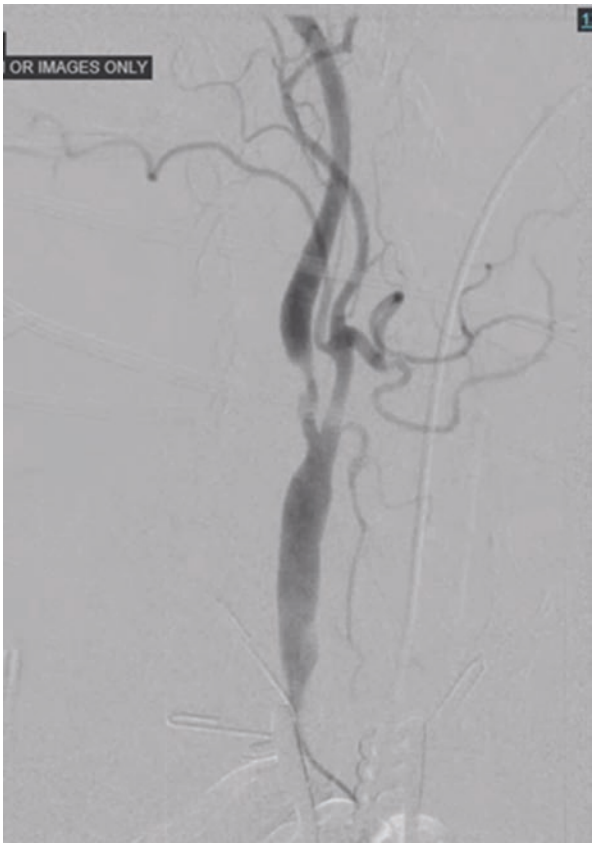


Fig. 36.15 Angiographic image of carotid arteries to delineate anatomy

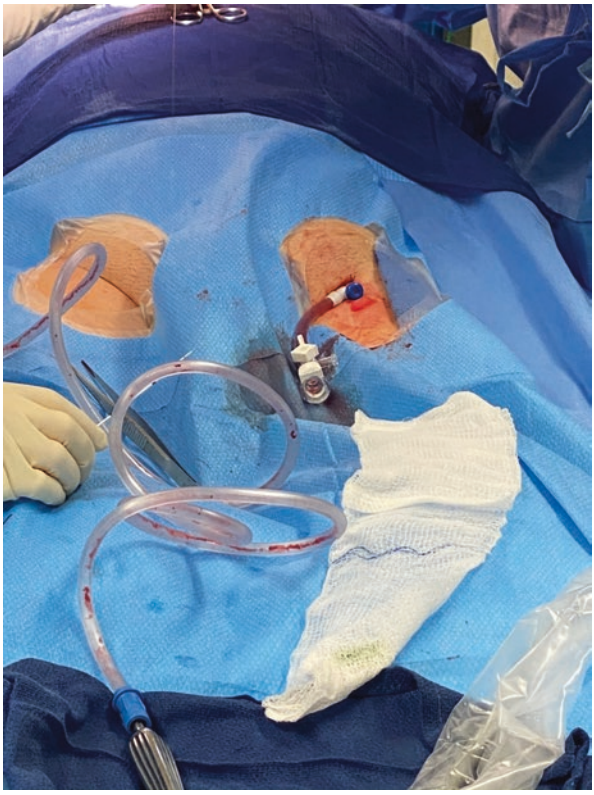


Fig. 36.16 A photo of venous sheath that is placed in the contralateral femoral vein

in place (Fig. 36.16). The microcatheter is replaced with the ENROUTE® carotid sheath. The retrograde filter flow apparatus is attached first to the carotid sheath and then to the venous sheath (Fig. 36.17).

An arterio-venous shunt is created with passive retrograde flow without arterial clamping. Next, the stent is positioned and deployed. Completion angiographic images are then obtained (Fig. 36.18). The retrograde filter system is disengaged and drained into the femoral sheath and normal antegrade flow allowed.

Anesthetic Management for TCAR

Anesthetic management is the same as CEA surgery with some modifications.

1. The procedure can be approached either under general or regional anesthesia.
2. Systolic blood pressure maintained between 140 and 160 mmHg is crucial to maintain trans pressure difference between carotid artery and femoral vein and effective reverse flow.

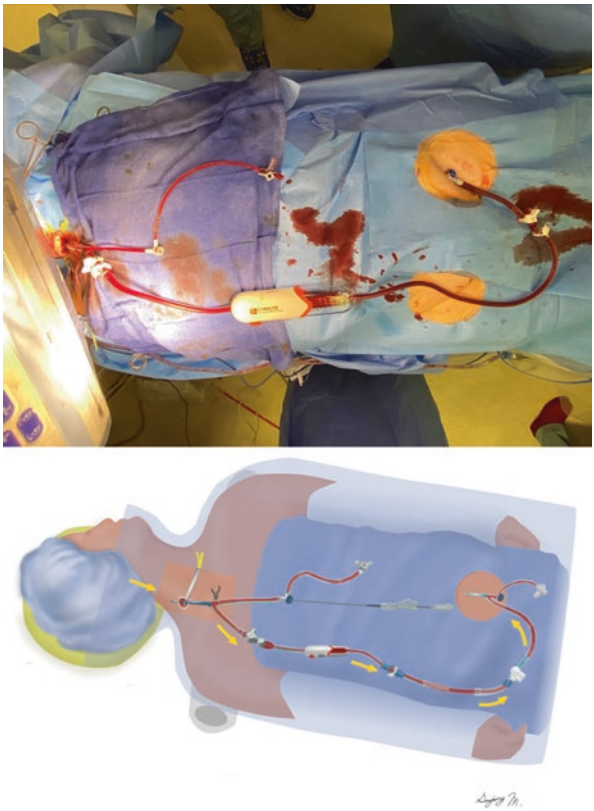


Fig. 36.17 A photo (top) and an illustration (bottom) of the ENROUTE® retrograde filter flow apparatus

3. Vasopressor drip is usually used to maintain target pressure.
4. Anticoagulation with target ACT of 250 s is recommended.
5. Post-stent deployment is associated with hypotension and bradycardia secondary to carotid body receptors stimulation from stent pressing against the receptors. Therefore, prophylactic atropine (0.5 mg intravenously) or glycopyrrolate (0.4 mg intravenously) is recommended.

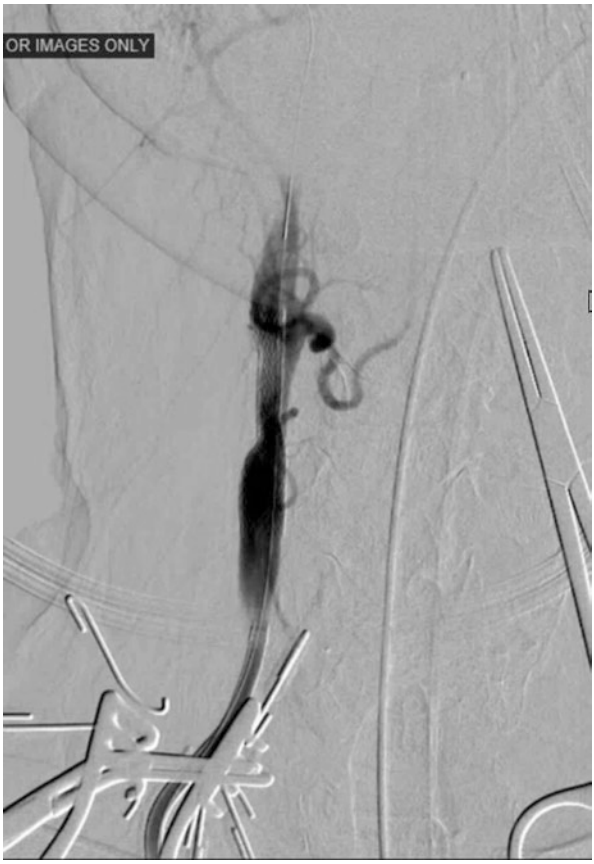


Fig. 36.18 Angiographic image of carotid arteries post stent deployment in the same patient

Further Reading

1. Apinis A, Sehgal S, Leff J. Intraoperative management of carotid endarterectomy. Anesthesiol Clin. 2014;32(3):677–98.
2. Abigail M, Gaurav B. Anesthesia for carotid endarterectomy. In: Anesthesiology: a practical approach. Cham: Springer; 2018. p. 645–55.
3. Kadoi Y. Anesthesia for carotid endarterectomy. In: Neuroanesthesia and cerebrospinal protection. Tokyo: Springer; 20015. p. 320–30.
4. Stoneham MD, Stamou D, Mason J. Regional anaesthesia for carotid endarterectomy. Br J Anaesth. 2015;114(3):372–83.
5. Dragana, US, Djordje R, Gojkovic T, Matic P, Rankovic L, Jovic M. Anesthesia for carotid endarterectomy: where do we stand at present? 2015. http://www.signavitae.com.

Anesthesia Considerations for Upper Extremity Arteriovenous Fistulas

37

Victoria N. Pham, Diego Urdaneta, Robert Gessman, and Michael S. Green

Fast is fine, but accuracy is everything.

—Wyatt Earp

Key Points

- Medical management of ESRD patients
- General anesthesia
- Regional anesthesia
- Local anesthesia

Introduction

End-stage renal disease (ESRD) requires long-term vascular access in order for intermittent hemodialysis to be performed. An arteriovenous fistula (AVF) is the preferred form for such access due to favorable long-term patency rates and a low incidence of infectious complications. Approximately 68% of all hemodialysis performed annually in the United States use an AVF.

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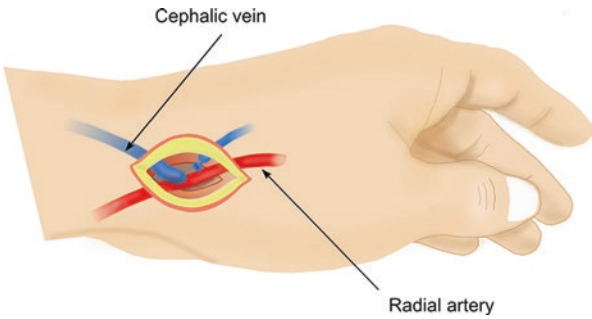

Fig. 37.1 Illustration of surgical creation of a radiocephalic fistula

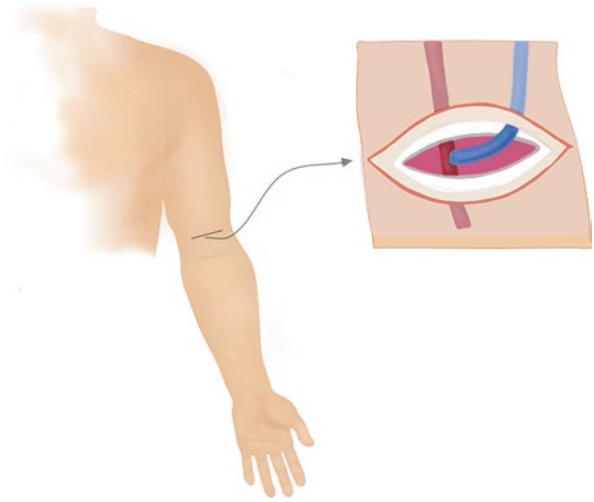

Fig. 37.2 Illustration of surgical creation of a brachiocephalic fistula

ESRD patients have many comorbidities that may make them challenging to manage during the perioperative period. AVF can be placed under general anesthesia (GA), regional anesthesia (RA), and local anesthesia (LA). The type of anesthetic technique employed is influenced by several factors including co-existing disease states and the technical requirements of the operating surgeon. Examples of the different surgical techniques employed are illustrated in Figs. 37.1 and 37.2.

Preoperative Assessment and Management of ESRD Patients

Preparation of ESRD patients for surgery should begin with a thorough review of the medical history. An understanding of the current state of all ongoing health conditions is required. Many chronic disease states will not be amenable to

improvement. Nonetheless, it is important to identify when decompensated conditions are present and to intervene prior to elective surgery in order to minimize complication rates. Physical examination should focus on airway in addition to the heart, lungs, and extremities with particular attention given to any signs of pericarditis and assessment of fluid volume status. Physical examination findings may include pericardial friction rub, pulsus paradoxus, hypotension, muffled heart sounds, and jugular venous distention. Preoperative assessment should include metabolic panel with serum concentration of magnesium and phosphorus to evaluate for critical values and electrolyte abnormalities.

Cardiac risk assessment

All dialysis patients should be considered as being subject to a relatively high risk of major cardiovascular complications regardless of the surgical procedure to be performed.

The incidence of ischemic heart disease in dialysis patients is twice that in the general population. A baseline electrocardiogram should be obtained for all patients with known coronary or other cardiac disease. Exercise or pharmacological stress testing is warranted if the patient provides a history consistent with new or unstable angina.

ESRD patients may be hypervolemic and often have hypoalbuminemia, which may contribute to symptoms of congestive heart failure. Poor exercise tolerance or shortness of breath on minimal exertion should prompt an assessment of both systolic and diastolic left ventricular function via echocardiography.

Hypertension is also common due to chronic hypervolemia and disorder of the renin-angiotensin-aldosterone system. Preoperative blood pressure control should be confirmed, and an assessment made for any associated end organ damage.

Pulmonary risk assessment

Shortness of breath should be assessed via chest x-ray. Pulmonary edema may be seen as a result of fluid overload, and pneumonia also occurs more frequently in this patient population due to impaired immune system functioning.

Endocrine

Diabetes mellitus is common among ESRD patients as it is the most frequent cause of chronic renal failure. Renal failure is also associated with an elevated risk of hypoglycemia through several different mechanisms. Perioperative management must include adequate blood glucose monitoring to avoid both hyper- and hypoglycemia.

Renal

The timing of surgery relative to the patient's hemodialysis schedule is of significant importance and should always be ascertained during the preoperative interview. Immediately following hemodialysis most patients will be relatively hypovolemic. If hemodialysis has been delayed beyond the patient's normal interval, hypervolemia may be present. These differences may translate into widely varying hemodynamic response patterns during the perioperative period. Ideally dialysis should occur within 24 hours of anesthesia and surgery with a gap of at least 6–12 hours to allow re-equilibration of the patient's total body water.

Dialysis disequilibrium syndrome (DDS) is a rare but serious complication that usually occurs with the patient's first HD session. While the exact mechanism is unclear, DDS is thought to be due to cerebral edema, increased intracranial pressure, and acute hyponatremia. Neurologic symptoms are the most common and include restlessness, headache, and coma.

Hematologic

The most common hematologic derangement seen in ESRD patients is a normochromic, normocytic anemia. This anemia results from lack of erythropoietin, bone marrow suppression, nutritional deficiencies, and diminished red blood cell survival time. ESRD patients usually compensate for this chronic anemia by increasing cardiac output and 2,3-DPG levels. If a transfusion of red blood cells is required other than during acute blood loss, it may be optimally given during dialysis when the intravascular volume status can be controlled. Accumulation of waste products in ESRD patients can inhibit platelet function, impair in vivo whole blood coagulation, and lead to microvascular bleeding during surgery.

Gastrointestinal

Uremic patients may have a higher incidence of hiccups, anorexia, nausea, vomiting, and diarrhea. Renal failure leads to delayed gastric emptying. Premedication with metoclopramide and ranitidine will decrease gastric volumes and pH.

ESRD and Anesthetic Technique

The physiological and metabolic changes that accompany ESRD necessitate greater caution in administering anesthetic medications. Hypervolemia may result in an increased volume of distribution for hydrophilic drugs. Reduced plasma protein concentration may result in decreased volume of distribution for highly protein-bound medications. Hypovolemia and/or autonomic dysfunction in diabetic patients may result in exaggerated cardiovascular instability. Chronic metabolic acidosis will affect the degree of ionization of many drugs, potentially altering both the pharmacokinetics and pharmacodynamics.

Rocuronium and vecuronium both rely partially on renal excretion, and the duration of clinical action may be slightly prolonged.

Sugammadex and the sugammadex-rocuronium complex are renally excreted. There is evidence for slower clearance, but not increased harm when this NMB reversal agent is used in renal failure. Regardless, the manufacturer's recommendation is that sugammadex should not be used in patients with creatinine clearances of less than 30/ml/min. Acetylcholinesterase inhibitors such as neostigmine and pyridostigmine rely on glomerular filtration and active tubular secretion for elimination. Dose adjustments of these drugs are necessary in ESRD patients due to reduced clearance.

Morphine-6-glucuronide is an active metabolite of morphine that is renally excreted and may accumulate during repeated dosing in ESRD patients. Fentanyl is suitable for post-operative pain control due to its fast onset and redistribution and lack of active metabolites. Oxycodone prolongation is less pronounced.

General Anesthesia

Although the National Surgical Quality Improvement Project (NSQIP) data identified a trend toward higher mortality in GA compared to RA or local/MAC in patients undergoing AVF creation, the most common technique in the United States is GA. Careful consideration should be taken with regard to devising an anesthetic management plan such that patient comorbidities are not underestimated. GA may be warranted where RA or local/MAC is contraindicated, such as in patients with significant cardiac dysfunction, coagulopathy, and altered mental status who will not cooperate with local/MAC. Also, it may be indicated due to difficult anatomy, or if regional anesthesia fails. A history of either significant neuropathies or gastroesophageal reflux may warrant endotracheal intubation, rather than the use of a supraglottic airway.

GA does result in peripheral venodilation and enhanced blood flow to the operative limb. However, these effects are limited to the intraoperative period. Conversely, general anesthesia can also be associated with increased stress response, hemodynamic instability, complications from neuromuscular blockade and controlled ventilation, and poor post-operative pain control.

Induction with propofol and low-dose fentanyl (25–50 mcg) is usually well-tolerated; etomidate can be used in case of hemodynamic instability. Succinylcholine can be safely used if potassium concentration is less than 5.5 meq/L. Cisatracurium (0.1–0.2 mg/kg) may be the preferred non-depolarizing neuromuscular blocking agent in ESRD because its mechanism of elimination is completely independent of renal function. Neither the action nor elimination of inhalational agents is affected by renal function. All of the volatile gases are safe for use in renal failure patients.

Regional Anesthesia

Major peripheral nerve blocks are growing in popularity as an anesthetic option for AVF surgery. These regional anesthetic techniques are accompanied by blockade of sympathetic nervous system fibers. The resulting vasodilatation and enhanced blood

flow to the operative limb may facilitate surgical creation of the AVF. There is evidence that regional anesthetics may also help maintain graft patency during the early post-operative period. Increased vein diameter as well as higher vessel flow rates are both positive predictors for long-term graft viability. Thus, the use of RA during fistula formation may contribute to shorter maturation times and higher overall patency rates. Furthermore, the excellent pain relief provided by regional anesthesia may reduce the need for narcotic analgesics and accelerate discharge times.

Risks of RA include nerve injury, infection, and hematoma formation. The risk of direct nerve trauma may be reduced by the use of ultrasound guidance. Evidence of any pre-existing neuropathy may preclude the use of a regional technique. All local anesthetics are potentially neurotoxic and pre-existing damage to a peripheral nerve increases the likelihood of long-term sequelae. Platelet dysfunction is common in chronic renal failure. Patients who are being treated with additional antiplatelet medications may present an excessive risk for bleeding complications. Patients with metabolic acidosis may have decreased uptake or onset of block with local anesthetic. Bicarbonate may be considered to counter this problem if it is suspected.

The brachial plexus is the appropriate target for facilitating upper extremity AVF creation. Commonly performed nerve blocks include the supraclavicular, axillary, or infraclavicular block.

A supraclavicular nerve block is associated with a 1% risk of pneumothorax. Concurrent interruption of phrenic nerve function occurs in approximately 50% of cases but has little clinical significance in the absence of severe lung disease. This block has the benefit of being able to be performed regardless of arm position.

The Infraclavicular block should be performed with the arm abducted, flexed at the elbow, and externally rotated. This is a deeper block that can be more technically challenging in some patients. Both the supraclavicular and infraclavicular approaches will provide adequate anesthesia for AVF creation in the antecubital fossa or upper arm.

The axillary block should likewise be performed with the arm abducted and externally rotated. The axillary approach carries a decreased risk of pneumothorax and phrenic nerve blockade. Disadvantages include risk of intravascular injection or hematoma. During an axillary block, the intercostobrachial nerve (T2/T3) is spared. This structure innervates the axilla and upper medial arm and requires supplementary local anesthetic to be injected in the superficial skin across the axilla in order to adequately cover the surgical field reaching into the upper medial arm. The axillary approach targetting the radial nerve is usually used for AVF creation in the forearm.

Local Anesthesia

It is possible to complete certain surgical procedures by injecting local anesthetic directly in the area where the AVF will be created (field block technique). Any AVF created in this manner will generally be superficial—within 1 cm of the skin surface—and limited in terms of the lengths of the vessels exposed.

The use of local anesthetics in patients with ESRD requires some caution. Metabolic acidosis may reduce both the duration of action and the protein binding of local anesthetics. The greater percentage of unbound drug and reduced seizure threshold increases the potential for local anesthetic toxicity.

It is important to reassure and instruct patients during RA and local/MAC techniques to avoid over-sedation. These patients may be exquisitely sensitive to narcotics or propofol sedation due to their comorbidities. It is important to note that if local/MAC is chosen, local anesthetic infiltration does not provide motor block and can be inadequate when a large amount of vein needs to be transposed.

Monitoring

Care should be taken to place pulse oximetry monitors and BP cuff on the nonoperative upper extremity to avoid the surgical field and obstruction of blood flow to the new fistula site. If needed, the lower extremity may be utilized for BP monitoring if use of the nonoperative upper extremity is contraindicated due to a previous or failed AV fistula site or previous axillary lymph node dissection.

Fluid Management

Careful management to minimize intravenous fluid (IVF) administration should be considered for these patients due to their likelihood of fluid volume overload prior to anesthetic care. Normal saline is the preferred solution to avoid hyperkalemia. IVF should be given via a pump upon entrance into the operating room to ensure avoidance of unintentional IVF administration. Anesthetic management of upper extremity arteriovenous fistula creation is summarized in Table 37.1.

Table 37.1 Summary of anesthesia management of upper extremity arteriovenous fistula creation

Location of bypass	Typically, upper extremity
Duration of operation	60–90 min
Anesthetic	MAC with local (1% lidocaine), need to know maximum dose, may need general/LMA for lower extremity versus RA
Medications	2000–5000 U heparin at the discretion of the surgeon. Notify at 3 min of administration.
Pain	Mild PO analgesics
Positioning	Supine operative arm out on arm board
Blood loss	Typically, little to no expected blood loss
Case specifics	Graft creation and basilic vein transposition require creation of subcutaneous tunnel and may require additional anesthesia No IV access or BP cuff on ipsilateral limb
Modifications	Revision cases May take longer and carry higher risk of blood loss May require angiography, no electrodes over chest on operative side
Post-op care	Return to room vs. discharge home

Clinical Pearls

- Arteriovenous fistulas (AVFs) are the most common form of dialysis access.
- Placement of AVF can be performed under general anesthesia, regional anesthesia, or local anesthesia.
- Anesthetic technique is selected after considering comorbidities (anticoagulation, severe pulmonary disease), surgeon requirements, and patient preference.
- Comorbid conditions unrelated to the surgery often present the greatest challenge during perioperative patient management.

Further Reading

1. Bradley T, Teare T, Milner Q. Anesthetic management of patients requiring vascular access surgery for renal dialysis. BJA Educ. 2017;17(8):269–74.
2. Cole N, Vlassakov K, Brovman E, Heydarpour M, Urman R. Regional anesthesia for arteriovenous fistula surgery may reduce hospital length of stay and reoperation rates. Vasc Endovasc Surg. 2018;52(6):418–26.
3. Siracuse JJ, et al. In anesthetic considerations for arteriovenous fistula creation. J Vasc Access. 2014;15(5):364–9.
4. Reynolds TS, et al. Pre-operative regional block anesthesia enhances operative strategy for arteriovenous fistula creation. J Vasc Access. 2011;12(4):336–40. https://doi.org/10.5301/JVA.2011.8827.
5. Farag E, Mounir-Soliman L, Brown DL. Brown's atlas of regional anesthesia. 5th ed. Philadelphia: Elsevier; 2017.
6. Nguyen H, Pai SL, Thammasithiboon S, Gasanova I. Anesthetic considerations for vascular access placement in patients with end-stage renal disease. SM J Anesth. 2017;3(1):1–9.
7. Pardo M, Miller RD. Chapter 18: Peripheral nerve blocks (2018). In: Basics of anesthesia. 7th ed. Philadelphia: Elsevier; 2018.
8. Shemesh D, Raikhinstein Y, Goldin I, Olsha O. General, regional or local anesthesia for successful radial cephalic arteriovenous fistula. J Vasc Access. 2017;18(Suppl. 1):S24–8.
9. Son A, Mannoia K, Herrera A, Chizari M, Hagdoost M, Molkara A. Dialysis access surgery: does anesthesia type affect maturation and complication rates? Ann Vasc Surg. 2016;33:116–9.

Part X

Post-Operative Care for the Cardiac Surgery Patient

Postoperative Care for the Adult Cardiac Surgery Patient

38

Michael Kouch, Akhil Patel, and Talia K. Ben-Jacob

I was a little surprised to find myself recovering after the surgery. Then gratified to have been given a second life

—Michael DeBakey MD

Introduction

Cardiac surgery, which encompasses coronary artery bypass grafting (CABG), cardiac valve and aortic procedures, and heart assist or replacement, is one of the most frequently performed surgery in the United States. Successful outcomes after cardiac surgery depend on excellent postoperative critical care. To improve morbidity and mortality in cardiac surgery patients, multidisciplinary teams in a cardiac ICU provide care for the first 24–48 hours after their operation. These teams consist of experienced intensivists, nurses, house staff, respiratory therapists, pharmacists, and physical therapists. They work closely with the cardiothoracic surgeons to ensure optimal patient care. These critical-care team members must have broad understanding of cardiopulmonary physiology, pathophysiology, clinical sequelae

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Fig. 38.1 Operating room to intensive care unit handoff and patient care transition

of cardiopulmonary bypass, and consequences of intraoperative decisions and their management in the cardiac ICU. With the use of protocols for perioperative management, outcomes have improved, extubation occurs earlier, less blood products are transfused, and length of stay is decreased. This chapter describes the management of post-cardiac surgery patients for the first 24–48 hours while they are being treated in the ICU.

ICU Transfer, Handoff, Initial Workup, and Monitors

Postoperative management most often begins with "hand-off" within the ICU. During transfer to the ICU, at minimum continuous, arterial blood pressure monitoring, pulse oximetry recording, and electrocardiographic (ECG) monitoring should be provided. As most patients will remain intubated during this period, an Ambu-bag with an adequately filled oxygen tank or portable ventilator is needed. During the transport period, vasopressors should be continued if needed, and close attention should be paid to catheters, lines, and chest tubes. The ICU team should be notified before the patient leaves the operating room and a brief report given.

Upon arrival in the ICU, the patient should be transitioned to ICU monitors and connected to the ventilator with the assistance of nursing staff and respiratory therapists. Handoff should be given in a structured, standardized way, with nursing, respiratory, surgical, anesthesia, and ICU teams present (Fig. 38.1). Focus should be on transfusion and vasopressor requirements, echocardiography findings, hemodynamics, perioperative antibiotics, and intraoperative events.

After handoff, the ICU team will thoroughly examine and evaluate the patient, including assessment of the chest tubes and drainage, laboratory work, electrocardiogram (ECG), and X-ray imaging for confirmation of the positioning of endotracheal tube and vascular or support devices.

Fluid Management and Blood Transfusion

Proper fluid management in the postoperative period is essential in optimizing hemodynamics. Assessment of volume status often is evaluated with static and dynamic measures. If a pulmonary artery catheter is used, pulmonary artery diastolic or occlusion pressures are used as markers of volume status. Although these measures may be poor predictors of volume responsiveness in shock states, they are used regularly in postoperative management to help guide the resuscitation. Noninvasive devices may be used to assess other dynamic measures, such as stroke volume and pulse pressure variation. Used in isolation, measurement of central venous pressure is not a reliable tool for determining volume status discussed in Chap. 9.

Goal-directed volume administration can be delivered with albumin or crystalloid. Third spacing due to cardiopulmonary bypass-induced systemic inflammatory response causes a need for volume expansion early in the postoperative period; higher amounts may be needed with longer bypass times. Over-resuscitation with fluids should be avoided; it increases the risk of pulmonary edema and transfusion requirements, delays extubation, and increases length of stay.

Establishment of blood transfusion thresholds has been controversial, and values vary among studies. Based on the current literature, restrictive transfusion strategy appears to be safe, and it reduces the frequency of adverse effects. A hemoglobin value of more than 7–8 mg/dL or hematocrit of >24% appears to be as effective as a more liberal threshold. Nonetheless, oxygen delivery and volume status should be evaluated for each patient before intravascular volume expansion or blood product transfusion is used.

Clinical Pearls

- Communication is a key part of the postoperative care and handoff for cardiac patients.
- Patient transfer should only occur when the patient appears to be stable and all necessary emergency medications and monitors are present.
- Post-cardiac surgery patients are at high risk for arrhythmias.
- Over-resuscitation with fluids increases the risk of pulmonary edema, transfusion requirements, delays extubation, and increases length of stay.
- Hyperglycemia should be avoided to allow for proper wound healing, decreasing wound infection, and decreasing overall morbidity and mortality.
- Early mobilization and good nutrition have decreased hospital stay and enhanced recovery after surgery.
- Post-CABG patients should be given aspirin and statins to improve graft patency and decrease the risk of myocardial infarction, stroke, and renal disease.
- Patients undergoing aortic arch operations and operations in which the aorta is cross clamped are at risk for stroke due to manipulation of the arch. Frequent neurological examinations allow appropriate interventions if necessary.

Systems Management

Cardiac

After the initial evaluation of the patient in the ICU, a more thorough examination of each system is required for continued care. Postoperative cardiac surgery patients are prone to arrhythmias, a topic that is discussed in Chap. 4. A postoperative ECG should be viewed in the context of the patient's clinical condition and compared to the preoperative ECG. Nonspecific ST changes are common and should be correlated with the type of procedure and preoperative coronary artery anatomy. The presence of bundle branch blocks, prolonged PR intervals, and concurrent ST changes should prompt evaluation for ischemia, systemic inflammatory response syndrome, or tamponade. Electrolytes (particularly magnesium, potassium, and calcium) should be repleted as abnormalities in their values increase the risk of arrhythmia. Hypothermia, myocardial irritation, and acidosis also increase the risk of arrhythmia. Patient should be warmed to normothermia and acidosis treated with mechanical ventilation or sodium bicarbonate.

Temporary cardiac pacing is often required in cardiac surgery patients, especially in those who have valvular surgery. If temporary pacing is required, testing for the presence of native rhythm should be conducted regularly. Placement of a permanent pacemaker is often required if bradycardia or conduction deficits persist beyond 4 days.

For off-pump cases, the patient is usually able to retain their native rhythm, as long as perfusion is adequate throughout the operation. If the surgeon places lead wires, they are set as a backup. For on-pump cases, the patient might require temporary pacing especially when coming off bypass. For more information about temporary pacing please refer to Chap. 32.

One of the most common postoperative arrhythmias detected is atrial fibrillation. It is particularly common after CABG, and its highest incidence is in combined CABG and aortic valve replacement surgery. Atrial fibrillation limits the atrial contribution to cardiac performance (atrial kick), which can be as high as 25% of the stroke volume. Thus, rate or rhythm control of atrial fibrillation is necessary for adequate perfusion. Rate control can be accomplished with beta-blockers or calcium channel blockers to achieve a proper heart rate with adequate filling times. Rhythm can be controlled with medications such amiodarone or occasionally sotolol. Beta-blockers and amiodarone are the most commonly used medications for this purpose. They are used also as prophylaxis for tachyarrhythmias, which may decrease their occurrence almost 50%. Immediate electrical cardioversion is indicated if the patient is acutely unstable. Lastly, magnesium is often used as prophylaxis for atrial fibrillation development and has a low side-effects profile.

Crisis Management

Hemodynamic management focuses on optimizing end organ perfusion. Oxygen delivery can be maximized with the use of nasal cannulas, high-flow oxygenation, continuous positive airway pressure (CPAP), bilevel positive airway pressure (BiPAP), or mechanical ventilation based on the patient's needs and physiology.

If oxygenation and ventilation are preserved, preload and cardiac output should be managed. Albumin and crystalloid are appropriate volume expanders, and the choice of fluid is often based on institutional preference. Vasodilation after cardiopulmonary bypass may be managed with vasopressors, which is discussed in Chap. 3. Due to overexpression of inflammatory mediators resulting from cardiopulmonary bypass, along with possible concurrent use of angiotensin converting enzyme inhibitors and angiotensin II receptor blockers for outpatients, patients may have refractory hypotension, termed vasoplegia. This pathologic syndrome of low systemic vascular resistance in the presence of normal or raised cardiac output may require the use of vasopressin or methylene blue in refractory cases. Angiotensin II is a relatively new drug that has shown promise in treatment of refractory vasoplegia discussed in Chap. 3.

Left and right ventricular dysfunction may be transient or persistent in cardiac surgery patients. ECG changes in ventricular dysfunction are often nonspecific. However, diffuse ST depressions in the setting of hemodynamic changes should prompt consideration of echocardiographic evaluation or coronary arteriography. Pericardial tamponade is a critical diagnosis to consider in the hemodynamically unstable postoperative patient, especially within the first 24 hours. Suspicion of tamponade is especially warranted when there is abrupt cessation of chest-tube drainage as blood may collect in the mediastinum and impair heart chambers filling. Symptoms in tamponade may be classic or atypical for example when it occurs in focal hematoma formation behind the right atrium. In this situation, the patient is hemodynamically unstable; however, the hematoma may be missed on transthoracic echocardiography. Findings of tamponade may include posterior bowing of the right ventricle (RV) into the left ventricle (LV), decreased cardiac output, tachycardia, hypotension, increased cardiomegaly seen on chest radiograph, elevated jugular venous pressure, pulsus paradoxus, and equalization of diastolic pressure on pulmonary artery catheter readings.

Acute RV failure may be driven by pulmonary hypertension, which can be exacerbated by pain, hypercarbia, hypoxia, excessive positive end expiratory pressure, and hyperinflation. Other causes include new-onset mitral regurgitation, pulmonary embolism, and volume overload. Increased RV pressure or volume overload may place right coronary artery perfusion at risk and may inhibit LV filling, leading to shock. Correcting poor oxygenation and hypercarbia, and setting lower positive end expiratory pressure and tidal volumes, will decrease pressure on the RV. Inotropes

that reduce pulmonary vascular resistance, such as milrinone or dobutamine, and pulmonary artery vasodilators, such as nitric oxide, nitroglycerin, and inhaled prostaglandins are also treatment modalities.

Cardiac stunning is present in some postoperative cardiac surgery patients and requires continued inotropic and vasopressor support. If a patient is in refractory heart failure that is not responding to interventions with inotropic support and volume optimization, mechanical circulatory support may be required. Since high-dose pharmacologic support is associated with poor outcome in post-cardiotomy shock, mechanical support should be considered early in the course of treatment. Intra-aortic balloon pump counter-pulsation or, more recently, the Impella® device (Abiomed), is first line of mechanical support; extracorporeal membrane oxygenation is being used in cases of more severe cardiac impairment.

Management of cardiac arrest in cardiac surgery patients is slightly different from that used in advanced cardiac life support. Ventricular tachycardia and ventricular fibrillation arrest should be managed with early defibrillation. In pulseless electrical activity arrest, sternotomy should be considered, with advice and input from the cardiac surgeon. If the patient is paced, it should be turned off to exclude underlying ventricular fibrillation, which should be defibrillated.

Pneumothorax may occur in cardiac surgery patients. Chest X-ray imaging should be done for confirmation of the positioning of chest tubes and absence of pneumothorax. Tension pneumothorax should be suspected if sudden hemodynamic instability occurs.

Postoperative acute prosthetic valve dysfunction, although rare, may occur following valve operations. Improper closure or opening of the prosthetic valve leaflets, valve dehiscence, and significant perivalvular leak can produce hemodynamic instability. High index of suspicion and missing arterial wave tracing while ECG tracing is not lost should prompt the ICU provider to confirm the diagnosis of valve failure using transesophageal echocardiography. Prompt surgical intervention and reexploration should be considered.

Respiratory

Nearly all cardiac surgery patients present to the ICU mechanically ventilated and often sedated. Protocolized, aggressive management to facilitate extubation within 6–8 hours postoperatively is associated with improved outcomes. Our protocol includes attempted extubation within 2–6 hours postoperatively. Lung-protective ventilation using low tidal volume ventilation (6 mgL/kg predicted body weight) should be utilized for most patients to limit the risk of barotrauma and volutrauma which can put the patient at risk for development of acute respiratory distress syndrome. Hypoxia should be avoided to maintain oxygen delivery. Hypercarbia should be avoided as well to prevent acute right ventricular failure as described previously. As the patient is rewarmed and resuscitated, the minute ventilation needs to change to match the increased oxygen consumption and should be monitored and adjusted to the individual patient. Frequent arterial blood gases measurement can guide ventilator management.

Weaning can commence when the patient is normothermic, hemodynamically stable, and has no acid-base disturbances, with all bleeding sites are controlled. If the patient is awake, alert, following commands, and has passed a spontaneous breathing trial, extubation can be attempted. Use of noninvasive ventilation such as BIPAP and CPAP can facilitate successful extubation in select patients. Post-extubation management includes incentive spirometry, Acapella use, moving the patient from a bed into a reclining chair, and early mobility with physical therapy assistance. These measures will help in preventing and treating atelectasis, which can worsen hypoxia.

Renal

Renal dysfunction is not uncommon after cardiac surgery. It is most likely due to hypoperfusion and overexpression of inflammatory mediators occurring during the operations. Patients most at risk are those with preexisting renal insufficiency, diabetes, long bypass or aortic cross-clamp times, and hypotension. Nonsteroidal anti-inflammatory agents should be avoided in these patients. Acute kidney injury increases length of stay and mortality especially in those requiring renal replacement therapy.

Measurement of urine output is a nonspecific way to measure renal perfusion and function. Twice daily laboratory tests, with serum creatinine and electrolytes determination, also help in monitoring kidney function and can help in differentiating the causes of the kidney injury. Diuresis often is needed in patients after bypass, as they have an increase in interstitial fluid compared with that in patients who do not have bypass run.

Postoperatively, electrolyte abnormalities are common and often problematic. During on-pump operations, patients receive cardioplegia, which can increase their serum potassium level. Acidosis can cause shifts of potassium into the intravascular space. Insulin administration may cause hypokalemia. Patients may lose potassium and magnesium from the effects of cardiopulmonary bypass, which can increase the likelihood of arrhythmia as described previously. Serum calcium concentrations may also be decreased due to citrate given in transfused blood products. Repletion of calcium is necessary for stabilizing cardiac myocytes. Phosphate is another important electrolyte as it can cause changes in the oxygen hemoglobin dissociation curve and change the shape of red blood cells. Repletion of phosphate is often needed in the ICU.

Both metabolic and respiratory acidosis may be encountered. Metabolic causes are largely due in part to reduced oxygen delivery. This can be managed by increasing minute ventilation and optimizing cardiac index with inotropes, vasopressors, and fluids. Bicarbonate administration is often used as a temporizing measure, but it may cause hypernatremia, volume overload, and intracellular acidosis. Therefore, improved perfusion should be the main goal in treating acidosis. Shivering should be considered as a cause of acidosis as it increases oxygen consumption, which can lead to a mismatch of oxygen delivery. Adequate rewarming will prevent and treat hypothermia that causes shivering.

Endocrine

All patients undergoing major surgical procedures have substantial stress responses, with increased catecholamines, which can cause increased blood sugar concentrations. Also, cardiopulmonary bypass prime may contain large amounts of corticosteroids. Tight glycemic control decreases morbidity and mortality and decreases the risk of sternal wound infections and sepsis. Current guidelines recommend keeping blood glucose values below 180 mg/dL, especially for the first 2 postoperative days. Most patients will require insulin to maintain this value even if they have no history of diabetes. Hyperglycemia will further promote a ketotic state and often osmolar diuresis.

Postoperative adrenal insufficiency should be considered in patients who remain hypotensive and have persistent vasodilation despite seemingly adequate vasopressor support. Postoperative adrenal insufficiency is a clinical condition, and treatment should not be guided by response to adrenocorticotrophic hormone challenge; stress-dose corticosteroids should be given when this diagnosis is considered.

Surgical Bleeding and Chest Tubes

Continued blood loss after cardiac surgery is common, and some is expected. Monitoring of chest tube output is essential for detecting bleeding beyond that which is expected. Customarily, a mediastinal tube is placed, and other tubes are placed in the pleural or pericardial space. Drainage from the tubes is monitored hourly or as frequently as every 15 minutes. Packed red blood cells trasfusion trigger and timing was described earlier.

When evaluating a patient with bleeding, the ICU provider must determine if it is “medical” or “surgical” bleeding. “Medical bleeding” may be caused by coagulopathy due to factors such as thrombocytopenia, hypothermia, insufficient coagulation factors, or insufficient heparin reversal, which may need correction. Transfusion of platelets, fresh-frozen plasma, and coagulation-factors concentrates may be needed. Fibrinogen values may be monitored, and cryoprecipitate given if values are abnormal. Administration of Factor VII is controversial because of the associated increase in thromboembolic adverse events, but it may be necessary; consideration of its use should be discussed with the surgical team. The possibility of “surgical bleeding” from causes such as bleeding vessels or anastomoses should be discussed promptly with the surgical team; sudden cessation of bleeding with associated hypotension should raise concern for occluded chest tube and the development of tamponade. On occasion, a large amount of blood may drain several hours or even days after operation without hemodynamic instability or fall in hematocrit. This condition may be due to the release of a “sequestered” accumulation that drains suddenly. It is important to keep this in mind as a potential occurrence.

Infection Prevention

Prevention of postoperative infection is essential. Twenty-four hours of prophylactic antibiotics are used to limit surgical-site infection; the most common medications used are cefazolin and vancomycin.

Prompt removal of lines and catheters that are no longer needed will help prevent infection. Arterial and central venous catheters should be removed as hemodynamics improve and vasoactive drugs are no longer required. For patients who remain intubated, the head of the bed should be elevated more than 30 degrees and good oral hygiene maintained. As discussed earlier, tight glycemic control will prevent infections, especially sternal wound infections.

Rehabilitation and Mobilization

Early postoperative mobilization of cardiac surgery patients is essential to prevent postoperative complications, decrease length of hospital stay, and optimize their functional status. Multiple techniques and protocols for early mobilization have been proposed in the literature. Whatever approach is used, limiting prolonged bedrest is key for preventing complications. Typically, ambulation on postoperative day one is suggested for patients who are hemodynamically stable, are not receiving inotropic or vasoactive medications, and whose mechanical support devices have been removed.

Nutrition

Nutrition and feeding in postoperative cardiac surgery patients is similar to that in the general intensive care population. Preoperative nutrition assessment will identify patients who are at high risk for malnutrition postoperatively. Generally, enteral nutrition is preferred over parenteral nutrition. Vasopressor use is not necessarily a contraindication to enteral nutrition. Early enteral nutrition is likely beneficial in patients after initial resuscitation from critical-organ failure. Nutrition requirements should be assessed daily in patients during their ICU stay, with a goal of providing at least 80% of their energy and protein requirements.

Reestablishment of Preoperative Medications

Preoperative medications are usually restarted on postoperative day 1 if patients can resume oral intake. If they cannot tolerate a diet, home medications are usually replaced with intravenous formulations. Preoperative blood pressure medicines are often resumed at lower doses since patients may still be recovering from cardiogenic shock. Home anti-arrhythmic medications are dose-adjusted or withheld depending on the current cardiac rhythm.

Surgery-Specific ICU Management

CABG

CABG is the most common cardiac operation. Postoperative CABG management focuses on maintaining graft patency and modifying of long-term cardiovascular risk factors.

Aspirin should be given within 6 hours postoperatively and continued indefinitely; this practice is associated with improved graft patency at the lead 60 days and 1 year. Aspirin therapy in the early postoperative period also is associated with reduction in in-hospital mortality and rates of myocardial infarction, stroke, acute renal failure, and bowel infarction. Alternative antiplatelet agents may be used in aspirin-allergic patients.

Statin therapy should be reinstituted early in the postoperative course. In the CABG population, statins inhibit the development of vein graft disease, and they decrease the rates postoperative of atrial fibrillation, renal disease, and all-cause mortality.

Mitral Valve Operations

Mitral valve replacement or repair may be performed independently or in conjunction with CABG and/or intervention on the tricuspid or aortic valve. With mitral valve operations changes in hemodynamics in the postoperative period likely are due to a change in loading conditions. For example, after surgical correction of a regurgitant mitral valve, the LV must work harder to pump against increased afterload that is developed by removing of the least resistance path and the back flow across the mitral valve. Afterload reduction and inotropic support is often required to maintain stroke volume and prevent LV failure. Another situation may arise after mitral valve repair, i.e. as a consequence of the repair and ring annuloplasty, dynamic LV outflow obstruction may develop due to systolic anterior motion of the anterior leaflet of the mitral valve, in which the anterior mitral leaflet paradoxically moves anteriorly and obstructs the LV outflow tract during systole. This abnormality is typically detected on transesophageal echocardiography as cardiopulmonary bypass is terminated, but it may present later, in the ICU. Postoperative management of mitral valve surgery patients involves gradual expansion of intravascular volume, limiting use of inotropic agents, and minimizing tachycardia, which may require beta blockage. Long-standing mitral valve disease can result in pulmonary hypertension and right ventricular dysfunction. Surgical intervention aims to alleviate these sequelae, but right ventricular failure can be exacerbated postoperatively; treatment regimens for this includes phosphodiesterase inhibitors (milrinone) and inhaled vasodilators such as nitric oxide and prostacyclins.

Aortic Valve Procedures

Postoperative management of patients with aortic valve interventions is like that of patient with CABG and mitral valve operations, as noted above. However, patients with long-standing volume overload from chronic aortic insufficiency may require aggressive volume expansion to maintain cardiac output. Also, aortic valve patients, especially those with bicuspid aortic valve disease, often have thin-walled aortas; consequently, avoiding hypertension in them early after operation to limit stress on the aortotomy repair is important. Monitoring and treatment of conduction disturbances by pacing may be required post-aortic valve surgery. The reported rates of high-degree heart block in these patients vary, but heart block may be common because of the immediate anatomical vicinity of the AV node and the proximal conduction bundle. Monitoring and medical treatment of complete heart block should be initiated while decision for permanent pacemaker is pending.

Aortic Root/Arch Operations

Management of patients with ascending aorta and aortic arch surgeries should include focus on potential neurologic complications, including stroke, spinal cord injury, and delirium. These complications can result from hypotension or hypoxia, air embolism, or thromboembolism of intravascular plaque during manipulation of the heart and aorta. Treatment of embolization includes its rapid identification and prevention of further insults. With the management of large-vessel occlusion in stroke, mechanical thrombectomy should be a component of the treatment algorithm. Spinal cord injury may result from disruption of the spinal artery blood supply. Indication of therapeutic hypertension and decreasing cerebral spinal fluid pressure by lumbar drain placement should be considered in patients at risk. In operations that include reimplantation of coronary arteries into a tube graft, coronary artery occlusion may occur, leading to myocardial infarction. Postoperative management of patients who have had these operations should include suspicion for kinking or occlusion if evidence of myocardial ischemia is present.

Minimally Invasive

Minimally invasive interventions are attractive for treatment of appropriate patients, with a goal of decreasing pain and shortening recovery times. While most postoperative care of patients who have had minimally invasive procedures is like that of conventional cardiac procedures, specific consideration should focus on prevention and treatment of postoperative atelectasis and monitoring of lower extremity perfusion. Since single-lung ventilation is required during minimally invasive cardiac procedures, risk of atelectasis is higher than in conventional surgery. Adequate pain

control (enteral, parenteral, and nerve block) and aggressive pulmonary toilet is essential in these patients. If peripheral cannulation for cardiopulmonary bypass was used, neurovascular perfusion and function of the lower extremities should be monitored closely.

Further Reading

Grimm, J. C., & Whitman, G. (2016). Postoperative care of the cardiac surgical patient. In Surgical intensive care medicine. 3rd ed. pp. 653–667. Springer International Publishing. https://doi.org/10.1007/978-3-319-19668-8_48.

Humanez JC, Singh V, Zaky A. Chap. 16 Postoperative care for the adult cardiac surgery patient. In Everything you need to know: out of the operating room and minimally invasive cardiothoracic procedures. 1st ed. Nova science publishers. 2018. ISBN: 1536129178, 9781536129175.

Stephens RS, Whitman GJ. Postoperative critical care of the adult cardiac surgical patient. Part I: routine postoperative care. Crit Care Med. 2015;43:1477–97. https://doi.org/10.1097/CCM.0000000000001059.

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;43:1995–2014. https://doi.org/10.1097/CCM.0000000000001171.

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