## SECTION 2

### Foundations of Cardiovascular Medicine

<table>
<thead>
<tr>
<th>Chapter</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>Functional Anatomy of the Heart</td>
<td>69</td>
</tr>
<tr>
<td>5</td>
<td>Normal Physiology of the Cardiovascular System</td>
<td>105</td>
</tr>
<tr>
<td>6</td>
<td>Molecular and Cellular Biology of the Heart</td>
<td>129</td>
</tr>
<tr>
<td>7</td>
<td>Biology of the Vessel Wall</td>
<td>148</td>
</tr>
<tr>
<td>8</td>
<td>Molecular and Cellular Development of the Heart</td>
<td>165</td>
</tr>
<tr>
<td>9</td>
<td>Genetic Basis of Cardiovascular Disease</td>
<td>178</td>
</tr>
<tr>
<td>10</td>
<td>Stem Cells and the Cardiovascular System</td>
<td>209</td>
</tr>
</tbody>
</table>
CHAPTER 4
FUNCTIONAL ANATOMY OF THE HEART

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BACKGROUND / 69
ORIENTATION OF THE HEART WITHIN THE THORAX / 70
METHODS USED TO STUDY CARDIAC ANATOMY / 71
TOMOGRAPHIC METHOD / 72
CORRELATIVE ANATOMY / 73
Pericardium / 73
Cardiac Skeleton / 75
Tricuspid Valve / 76
Mitral Valve / 76
Aortic Valve / 81
Pulmonary Valve / 84
Age-Related Valve Changes / 84
Cardiac Grooves, Crux, and Margins / 84
Right Ventricle / 85
Left Ventricle / 85
Ventricular Septum / 87
Atrial Septum / 87
Right Atrium / 89
Left Atrium / 89
Coronary Arteries and Veins / 90
Regional Coronary Artery Supply / 96
Coronary Collaterals and Microcirculation / 96
Cardiac Lymphatics / 97
Great Vessels / 97
Cardiac Conduction System / 99
NEW DEVELOPMENTS AND FUTURE CHALLENGES / 100
ANATOMY NOT ADDRESSED AND QUANTUM COMPUTING / 101

BACKGROUND
The study of the heart and great vessels has expanded since the days of Andreas Vesalius, the great 16th-century anatomist who recognized the impact of anatomy on the practice of medicine. During the European Renaissance, the tomographic approach to the study of cardiac anatomy became popular because of its artistically based correlations. This is vividly depicted in the drawings of Leonardo da Vinci (Fig. 4–1), the first comparative anatomist since Aristotle (see Chap. 1). During the ensuing nearly 400 years, however, interest in cardiac anatomy has been sporadic and limited to a few zealous and pioneering physicians, anatomists, and artists. The 19th century ushered in the era of anatomic dissection for the study of physiologic and pathophysiologic processes, and correlations. Virchow in 1885 described the inflow-outflow method of cardiac dissection, which followed the direction of blood flow.2 It was quick and simple and became the dissection method of choice. The works of Virchow and Osler paved the way to understanding the pathophysiologic basis of such diseases as pulmonary embolism, endocarditis, and heart failure.2 Renewed interest in the study of cardiac anatomy and pathology was facilitated by the rise in autopsy rates in Europe and North America during the first half of the 20th century.7 Herrick described the clinical features of coronary thrombosis.8 Later, Blumgart, Schlesinger, and Zoll advanced our understanding of coronary artery disease through elegant clinicopathologic correlations.9

These achievements notwithstanding, however, were limited to postmortem examinations. The advent of cardiac surgery in the 1950s, followed by coronary angiography, was a major impetus for promoting the study of in vivo clinicopathologic anatomic correlations. Although cardiac surgeons were quick to appreciate the importance of having a detailed understanding of cardiac anatomy, clinical cardiologists were more interested in pathophysiology. However, with the introduction of noninvasive imaging techniques (echocardiography, computed tomography [CT], magnetic resonance imaging [MRI], and single photon-emission computed tomography [SPECT]) over the past three decades, the perception of cardiac anatomy and pathophysiology radically changed for all of medicine in general and cardiology in particular.

With increasing use of tomographic techniques in the diagnosis and management of cardiovascular diseases, there has been a corresponding decrease in the use of autopsies for anatomic correlations. The reasons for this decrease are complex and controversial and include an increased confidence in antemortem laboratory and imaging technologies, and rescinding the mandate for autopsies for hospital accreditation.10 Nonetheless, autopsy still uncovers unexpected processes in upward of 40% of cases and remains an invaluable tool for quality assurance and educational programs.

Today, there is a growing resurgence in the clinicopathologic correlation approach to cardiovascular morphology. In particular, the tomographic presentation of cardiac structure, which had remained dormant for more than a century, has become relevant because the diagnostic techniques used today are tomographic in nature.3 The specialties associated with cardiovascular diseases have been quick to embrace these newer anatomic presentations. Echocardiography was brought into the operating room and cardiac catheterization laboratory, and with the advent of transesophageal echocardiography, the cardiologist became an indispensable member of the surgical team and structural heart disease interventional cardiology team (see Chap. 15).4,5

The interest in cardiac anatomy among cardiologists is by no means limited to those instances involved in imaging the heart. Over the past few years, there has been an explosion of interest in anatomically guided electrophysiologic mapping and ablation techniques, which are increasingly guided by intracardiac ultrasound (see Chap. 15).7,8 It has thus become feasible to accurately pinpoint the anatomic location of the source of many arrhythmias9 (Figs. 4–2 and 4–3). By providing the electrophysiologist with a real-time visual road map, the search-and-destroy mission during an ablation procedure will be made much easier, and results, as well as complications, will be recognized immediately.9,10 By providing a new window to the heart, real-time anatomic-electrophysiologic correlations can also help to enhance our understanding of the mechanisms of propagation of various arrhythmias. There has also been a recent surge in anatomically guided percutaneous catheter-based procedures to repair or replace a diseased native or bioprosthetic cardiac valve, and to close atrial and ventricular septal defects.9 The successful implementation of these
increasingly more sophisticated surgical and percutaneous catheter-based techniques requires closer interaction among the cardiac surgeon, interventional cardiologist, and the noninvasive cardiologist, as well as precise diagnostic tools with greater spatial and temporal resolution to guide the planning of these procedures.5,6,9,10

This is being met by rapid technological advances that currently include the ability to reconstruct and display cardiac anatomy/pathology in a readily familiar three-dimensional (3D) format, be it imaging alone or translation to a physical model with the increased utilization of 3D printing.9-12

Because of all these developments, a new appreciation of cardiac anatomy has emerged as the cornerstone for clinical cardiology. The purpose of this chapter is to describe the anatomy of the heart by principally using the tomographic format prevalent in current CT, MRI, and echocardiography, with special emphasis and focus on clinically relevant anatomic details. We will make only passing note of the next generation of imaging techniques (ie, molecular, parametric, quantum). The intent is to emphasize the important anatomic features of various cardiovascular disease processes relative to diagnosis and management.

ORIENTATION OF THE HEART WITHIN THE THORAX

The body can be viewed in three standard orthogonal anatomic planes: (1) frontal (coronal), (2) horizontal (transverse), and (3) sagittal.4,5 However, the three primary planes of the heart (short axis [transverse], four-chamber [frontal], and long-axis [sagittal]) do not correspond to the standard anatomic planes of the body4,5 (Fig. 4–4). Incorrect photographic or artistic orientation of surgical or autopsy specimens of the heart, presented out of context, can result in the display of two-dimensional (2D) images in nonanatomic positions and actually contribute to misconceptions regarding the position of the heart within the thorax5 (Fig. 4–5). As we enter into the 3D era, anatomic realism will become absolutely essential.

Thus, first, in describing the orientation of a specific organ such as the heart, one must take into account both the position of the heart...
CHAPTER 4: Functional Anatomy of the Heart

and the position of adjacent structures such as the thoracic aorta and esophagus. In interpreting 2D or 3D images, clinicians must avoid making correlations that yield impossible anatomy (Fig. 4–6). Accurate anatomic diagnoses require close interdisciplinary interactions among cardiovascular pathologists, cardiologists, radiologists, anesthesiologists, and surgeons and emphasize a critical need for teamwork and a “common language” in describing cardiac anatomy and pathology. This is of paramount importance when guiding catheter-based interventions (see Sec. 8 and Chap. 88).

FIGURE 4–5. A. Anterior view of the heart in its usual anatomic position with its apex directed from right to left. Arrows point to the anterior interventricular groove. B. Nonanatomic positioning of the normal heart with its apex directed downward, thereby resembling a “valentine.” The position of the cardiac apex is normally leftward (levocardia) but can anomalously be rightward (dextrocardia) or midline and inferiorly (mesocardia). Ao, ascending aorta; LV, left ventricle; PT, pulmonary trunk; RV, right ventricle; SVC, superior vena cava.

METHODS USED TO STUDY CARDIAC ANATOMY

The two conventional approaches to the study of cardiac anatomy that have stood the test of time are (1) the inflow-outflow method (Fig. 4–7) and (2) the tomographic ventricular slice method (Fig. 4–8). Although the inflow-outflow method readily demonstrates disease processes in a given cardiac chamber or valve, it does not allow simultaneous visualization of the effects of that process on contiguous structures. Furthermore, the inflow-outflow method does not correspond

FIGURE 4–6. Apex-down four-chamber view of the heart (left) and mirror-image photograph (right). Mirror-image depiction (commonly used in publications) to depict normal four-chamber, apex-up echocardiographic anatomic images does not correspond to normal anatomic reality. Obviously, 3D anatomic correctness is essential for accurate clinicopathologic correlations. LA, left atrium.
well to clinical tomographic imaging modalities except possibly cavitary angiography. With the ventricular slice technique (see Fig. 4–8), the ventricles are “bread sliced” perpendicular to the plane of the ventricular septum. This technique is ideal for the evaluation of ischemic heart disease but may have to be carried basally, well beyond the papillary muscle tips.

**TOMOGRAPHIC METHOD**

Renaissance anatomists such as da Vinci used the tomographic approach principally because of its artistic correlations. Modern anatomists and pathologists have resorted to this method because it correlates with conventional tomographic imaging techniques. With this method, cardiac dissection involves bisecting the heart into two pieces using a single plane of section. Anatomy contained within the depth of each section fosters a perception of 3D anatomy. Commonly used planes bisect the heart perpendicular to the base-apex axis (short-axis transverse views) (Fig. 4–9) or parallel to it (long-axis and four-chamber frontal views) (Fig. 4–10). Planes that bisect the heart parallel to the conventional body planes (frontal coronal, transverse short-axis, and sagittal long-axis views) (Fig. 4–11) replicate body tomography.

The short-axis tomographic planes of the heart (Fig. 4–12) are similar to the ventricular slice method but differ in two important respects. The “bread slicing” of the heart is continued to the base of the heart and great vessels, and the slices are oriented as though the heart were being viewed from the apex toward the base rather than in the opposite direction, as has been the case with the ventricular slice technique. Photographs should correspond with diagnostic tomographic scans.

The long-axis and four-chamber planes are orthogonal to the short-axis planes. The four-chamber planes of cardiac dissection (Fig. 4–13) involve sectioning the heart along both lateral walls, from apex to base, such that both ventricles and both atria are included in the plane of section. The long-axis two-chamber method (Fig. 4–14) involves bisecting the heart from the left ventricular apex through the mitral orifice and into the left atrium. The long-axis plane can cut through both the left ventricular inflow tract (including the left atrium and mitral valve) and the left ventricular outflow tract (including the ventricular septum, anterior mitral leaflet, and ascending aorta) (Fig. 4–15A). This plane also cuts obliquely through the right ventricular outflow tract.

These three anatomic tomographic planes of the heart have been particularly useful in echocardiography and more recently CT and MRI (Fig. 4–15B). Serial sections within each plane produce a collage of anatomic slices (Fig. 4–16) that can be used for 3D and higher-dimensional reconstructions, which is beyond the scope of this chapter. The tomographic planes of section can be tailored to the different imaging modalities. Thus echocardiography and SPECT generally use the primary planes of the heart. In contrast, CT and MRI use the primary planes of the body. The parasagittal or oblique planes of the body serve radionuclide angiography and left ventriculography. When the tomographic examination is not configured to the primary planes of the heart but rather to the planes of the body, the terms short, long, and frontal can be misleading (Figs. 4–17 and 4–18).

Pathologic lesions in both congenital and acquired heart diseases often involve contiguous chambers, valves, or vessels. The tomographic
METHOD is the optimal technique for demonstrating intracardiac relationships and is ideal for any disease that involves several cardiac chambers. The proliferation of noninvasive tomographic imaging techniques makes this method particularly ideal for clinicopathologic correlations. Limitations of tomographic dissection can be overcome by photography, computer imagery, and interestingly, the use of glue. After each tomographic section has been produced and photographed, the bisected specimens can be glued back together using any cyanoacrylate glue, such as Krazy Glue or Superglue, and resected along a different tomographic plane. Step-by-step photographic documentation is necessary, because once the specimen has been glued and recut, the preceding tomographic plane of section will be available only in the photograph and not in the actual specimen.

CORRELATIVE ANATOMY

This section provides an illustrated review of applied cardiac anatomy. The clinical significance of the anatomy described is highlighted in italics.

PERICARDIUM

The pericardium is a vaguely conical structure that adheres to and envelops the heart as the serous (visceral) and fibrous (parietal) pericardia, respectively. The fibrous pericardium is a resilient sac that surrounds the heart and attaches onto the great vessels. The main pulmonary artery, portions of both venae cavae, distal pulmonary veins, and nearly the entire ascending aorta are intrapericardial (Fig. 4–19). These are important anatomic features to remember in evaluating diseases of the pericardium. Given the intrapericardial location of the ascending aorta, diseases such as localized aortic wall hematomas, aortic dissection, or aortic rupture can produce a rapidly fatal hemopericardium. Because the sac is collagenous, with little elastic tissue, it cannot stretch acutely. However, the pericardium is capable of remodeling such that chronically it can accommodate relatively large volumes in excess of one liter without causing tamponade. In patients with total anomalous pulmonary venous connection, the confluence of pulmonary veins is intrapericardial. In
FIGURE 4–11. Tomographic cardiac dissection along the body primary planes. **A, B.** Transverse sections (looking from head toward feet) at the level of the great vessels (A) or the cardiac chambers (B). The aortic arch travels over the left bronchus and the right pulmonary artery. **C, D.** Frontal sections (looking from anterior to posterior) through both ventricles (C) or left ventricle and right atrium (D). **E, F.** Parasagittal sections looking from right (E) to left (F). Ao, ascending aorta; CS, coronary sinus; E, esophagus; IA, innominate artery; IVC, inferior vena cava; LA, left atrium; LAA, left atrial appendage; LB, left bronchus; LCX, left circumflex coronary artery; LIV, left innominate vein; LLPV, left lower pulmonary vein; LPA, left pulmonary artery; LUPV, left upper pulmonary vein; LSA, left subclavian artery; LV, left ventricle; MS, membranous ventricular septum; MV, mitral valve; PS, pericardial sac; PT, pulmonary trunk; PV, pulmonary valve; RA, right atrium; RAA, right atrial appendage; RPA, right pulmonary artery; RUPV, right upper pulmonary vein; RV, right ventricle; RVO, right ventricular outflow; SVC, superior vena cava; TV, tricuspid valve.
contrast, the right and left pulmonary arteries and ductal artery (ductus arteriosus) are extrapericardial structures.

The serous pericardium forms the delicate inner lining of the fibrous pericardium and continues onto the surface of the heart and great vessels at the pericardial reflection. Over the heart, it is referred to as the epicardium, and it contains the epicardial coronary arteries and veins, autonomic nerves, lymphatics, and a variable amount of adipose tissue. The junctions between the visceral and parietal pericardium lie along the great vessels and form the pericardial reflections. The reflections along the pulmonary veins and venae cavae are continuous and form a posterior midline cul-de-sac known as the oblique sinus (Fig. 4–20). Behind the great arteries, the transverse sinus forms a tunnel-like passageway that separates the great arteries anteriorly from the great veins posteriorly (see Fig. 4–20). After open-heart surgery, localized accumulation of blood within the oblique sinus can produce isolated left atrial tamponade. Similarly, a hematoma adjacent to the low-pressure right atrium can cause isolated right atrial compression tamponade. With increasing age and with obesity, fat can accumulate within the parietal pericardium and epicardium (see Fig. 4–33). In imaging the heart, it is important not to misinterpret epicardial fat as an abnormal structure or a tumor.

### CARDIAC SKELETON

The four major cardiac valves are anchored to their annuli, or valve rings. These fibrous rings, at the base of the heart, join to form the fibrous skeleton of the heart (Fig. 4–21). The centrally located aortic valve forms the cornerstone of the cardiac skeleton, and its fibrous extensions about each of the other three valves. The cardiac skeleton contains not only the four major valve annuli but also the membranous septum and the aortic intervalvular, right, and left fibrous trigones. The fibrous trigones form the anatomic substrate for mitral-aortic continuity (Fig. 4–22; see also Fig. 4–21). The intervalvular fibrosa also forms part of the outer wall of the transverse sinus (see Fig. 4–22). In patients with infective endocarditis of the mitral or aortic valves, infection can burrow through the intervalvular fibrosa and produce characteristic fistulas between the left ventricle and the adjacent left atrium, ascending aorta, or transverse sinus (see Chap. 67). The right fibrous trigone (see Fig. 4–21), also known as the central fibrous body, welds together the aortic, mitral, and tricuspid valves and forms the largest and strongest component of the cardiac skeleton. It is through the right fibrous trigone that the atrioventricular (AV) (His) bundle passes. Otherwise, the fibrous cardiac skeleton serves to electrically isolate the atria from the ventricles. Diseases or surgical alterations of one valve can affect...
the shape or angulation of adjacent valves (eg, aortic valve replacement causing severe mitral regurgitation) and can affect the nearby coronary arteries or conduction tissue.17

■ TRICUSPID VALVE

The tricuspid valve is comprised of five components (ie, annulus, leaflets, commissures, chordae tendineae, and papillary muscles). The anterior tricuspid leaflet is the largest and most mobile and forms an intracavitary curtain that partially separates the inflow and outflow tracts of the right ventricle (Fig. 4–23). The posterior leaflet is usually the smallest. The septal leaflet is the least mobile because of its many direct chordal attachments to the ventricular septum. A relatively distensible annulus is unique to the tricuspid valve owing to discontinuity of collagenous tissue on the right AV free wall.17 Consequently, dilatation of the right ventricle commonly produces circumferential tricuspid annular dilatation resulting in variable degrees of tricuspid valve regurgitation (see Chap. 51).16

■ MITRAL VALVE

The mitral apparatus is composed of the same five components as the tricuspid valve. Competent mitral valve function is a complex process that requires the proper interaction of all components, as well as adequate left atrial and left ventricular function. Abnormalities of the mitral valve apparatus can involve any of these components or combinations thereof. The pattern of pathologic involvement often determines the feasibility of mitral valve repair (surgical or percutaneous) (see Chaps. 48, 49, and 50).18 The mitral valve annulus forms a complete fibrous ring that is firmly anchored along the circumference of the anterior leaflet by the tough fibrous skeleton of the heart17 (see Fig. 4–21). Therefore, dilatation of the mitral valve annulus primarily affects the posterior leaflet. All current operative mitral valve repair techniques are based on this principle of asymmetric annular dilatation. Mitral valve annuloplasty reduces the mitral valve inlet area by reducing the contribution of the posterior leaflet.17 This is the rationale for using a partial posterior annuloplasty ring. The mitral and tricuspid annuli are not planar but rather saddle-shaped with dynamic motion throughout the cardiac cycle. The nonplanar conformation is maintained throughout systole and diastole but is slightly more pronounced during systole.19

The mitral valve has only two leaflets. The anterior leaflet is large and semicircular, and it partially separates the ventricular inflow and outflow tracts (see Fig. 4–23). However, unlike its right-sided counterpart, it also forms part of the outflow tract. In patients with hypertrophic obstructive cardiomyopathy, the anterior mitral leaflet can be pulled anteriorly toward the basal ventricular septum, resulting in midystolic outflow obstruction and mitral regurgitation.20 The posterior mitral

FIGURE 4–13. Tomographic cardiac dissection along the heart’s primary four-chamber plane. The heart is viewed as though one were looking from the anterosuperior surface toward the posteroinferior surface. In the floor of the right atrium is the orifice of the inferior vena cava (IVC). The pulmonary veins (PulV) enter the posterior aspect of the left atrium. AL, anterolateral mitral papillary muscle; AS, atrial septum; LA, left atrium; TV, tricuspid valve; VS, ventricular septum.

Fig. 4–14. Tomographic cardiac dissection along the heart’s primary long-axis plane. A. Tomographic section showing the left ventricle and left atrium. The mitral valve is also well demonstrated. The left atrial appendage is located anteriorly. The specimen is viewed as though one were looking from the tip of the left scapula toward the right nipple. B. Two-chamber transesophageal echocardiography (TEE) analogous to the two-chamber transthoracic echocardiography (TTE). Arrowheads point to the left atrial appendage. AW, anterior wall; DAo, descending thoracic aorta; E, esophagus; IW, inferior wall; LA, left atrium; LAA, left atrial appendage; LB, left bronchus; LPA, left pulmonary artery; LV, left ventricle; MV, mitral valve; PulV, pulmonary vein; Tr, trachea.
CHAPTER 4: Functional Anatomy of the Heart

FIGURE 4–15. A. Left ventricular long-axis method of tomographic cardiac dissection (looking from left flank toward the midsternum). Continuity between mitral and aortic valves is clearly seen. The transverse sinus (*) abuts the wall of the left atrium; arrows point to the right upper and lower pulmonary veins. B. Comparable MR long-axis view. Black arrow points to the anterior mitral leaflet and white arrow points to posterior mitral leaflet. A, anterior mitral leaflet; Ao, ascending aorta; CS, coronary sinus; LA, left atrium; LV, left ventricle; P, posterior aortic cusp; PM, posteromedial mitral papillary muscle; R, right aortic cusp; RVO, right ventricular outflow; SVC, superior vena cava.

FIGURE 4–16. Collage of four-chamber tomographic sections cutting from inferior wall to anterosuperior wall showing A. coronary sinus, B. internal cardiac crux (*), and C. aortic valve. Ao, ascending aorta; CS, coronary sinus; IVC, inferior vena cava; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle; arrow in A points to a fenestrated eustachian valve.
leaflet is more shallow and is usually divided into three scallops (ie, P1, P2, and P3). The middle scallop (P2) is the largest of the three in more than 90% of normal hearts. Occasionally, however, either the anterolateral (P1) or the posteromedial scallop (P3) is larger, and rarely there are accessory scallops.\(^{16,17}\) (Fig. 4–24). Posterior mitral leaflet prolapse usually involves the middle scallop and can be associated with chordal rupture. Both mitral leaflets are normally similar in area. The anterior leaflet is twice the height of the posterior leaflet but has half its annular length.\(^ {17}\) With advanced age, the mitral leaflets thicken somewhat, particularly along their closing edges.\(^ {17}\)

The commissures are incomplete splits in the leaflet tissue that represent the sites of separation of the leaflets (Figs. 4–25 and 4–26A). Beneath the two mitral commissures lie the anterolateral and posteromedial papillary muscles, which arise from the left ventricular free wall (see Figs. 4–18B and 4–25). Commisural chords arise from each papillary muscle and extend in a fan-like array to insert into the free edge of both leaflets adjacent to the commissures (major commissures)\(^ {37}\) (see Figs. 4–24 and 4–26A) or into two adjacent scallops of the posterior leaflet (minor commissures) (see Figs. 4–24 and 4–25). Normal posterior leaflet interscallop indentations formed by minor commissures are superficial and do not affect mitral valve closure. Commissures, both major and minor, are to be distinguished from gaps and clefts. Clefts divide one leaflet into two and may or may not reach the level of the annulus. They are most commonly seen in the setting of AV canal defects. Unlike commissures, clefts have no papillary muscles beneath them and no chordal insertions along their edge. Some authors have also used the term cleft to describe deep divisions between scallops that are occasionally observed in myxomatous mitral valve disease, which may impact valve function and/or repair (Figs. 4–27A–C). Gaps are abnormal spaces between two leaflets, often at the usual site of a commissure. They are most frequently encountered in the setting of AV canal defects and tetralogy of Fallot, where the anterior and septal tricuspid leaflets may exhibit separation. Because the commissural chords are seldom elongated, they serve as accurate reference points for determining the proper
CHAPTER 4: Functional Anatomy of the Heart

Percutaneous edge-to-edge mitral valve repair involves grasping the free edges of the anterior and posterior leaflets at the site of mitral regurgitation (see Chap. 48).

The anterolateral papillary muscle is commonly single and usually has a dual blood supply from the left coronary circulation. In contrast, the posteromedial papillary muscle usually has multiple heads and is most commonly supplied only by the dominant coronary artery. Small left atrial branches supply the most basal aspects of the mitral leaflets.

Papillary muscle contraction pulls the two leaflets toward one another and thereby promotes valve closure. The line of closure for either mitral leaflet is not its free edge but an ill-defined junction between a thin, clear zone and a thicker, rough zone (see Fig. 4–26).

Chordae tendineae progressively arborize from their anchoring point on the papillary muscle to their ultimate insertion on the free edge (so-called primary chords) and the rough zone (so-called secondary chords). The primary chords serve to prevent leaflet prolapse during ventricular systole, whereas the secondary chords help to maintain left ventricular geometry throughout the cardiac cycle. Two particularly prominent secondary chords, referred to as strut chords, insert along each half of the ventricular surface of the anterior mitral leaflet and provide additional leaflet support. They can contain cardiac tissue.
muscle and tend to calcify with age. Unlike the tricuspid valve, the normal mitral leaflets have no chordal insertions into the ventricular septum. The functional orifice of the mitral valve is defined by its narrowest diastolic cross-sectional area. This can be at the annulus when there is extensive annular calcification or close to the papillary muscle tips in patients with rheumatic mitral stenosis.

Mitral valve prolapse is characterized by thickened and redundant leaflets, annular dilatation (with or without calcium), and thickened and elongated chordae tendineae (with or without rupture). Prolapse of the posterior leaflet occurs more frequently than that of the anterior leaflet.

FIGURE 4-19. Anterior view of the heart. The anterior portion of the parietal pericardium has been removed, exposing the intrapericardial portions of the superior vena cava (SVC), ascending aorta (Ao), and pulmonary trunk (PT). LV, left ventricle; RA, right atrium; RV, right ventricle.

FIGURE 4-20. Tomographic section in the short-axis plane of the body, looking from apex toward the base, showing the oblique (OS) and transverse (TS) pericardial sinus. Ao, ascending aorta; DAo, descending thoracic aorta; LA, left atrium; LAS, left anterior sinus; LMA, left main coronary artery; PS, pericardial sac; PV, pulmonary valve; RAA, right atrial appendage; SVC, superior vena cava.

FIGURE 4-21. Base of heart. A. Section through the base of the heart, looking from base toward apex, with the atria and great arteries removed, shows all four cardiac valves. B. A comparable schematic diagram of the fibrous cardiac skeleton. The centrally located aortic valve forms the cornerstone of the cardiac skeleton. Its fibrous extensions anchor and support the other three valves. A, anterior; AoV, aortic valve; AV, atriocoronary sinus; IV, interventricular; L, left; LCX, left circumflex coronary artery; MV, mitral valve; P, posterior; PV, pulmonary valve; R, right; RCA, right coronary artery; S, septal; TV, tricuspid valve.

FIGURE 4-22. Long-axis section of the left ventricle. The intervalvular fibrosa (dashed triangle) lies between the anterior mitral leaflet and the posterior cusp of the aortic valve and abuts the floor of the transverse pericardial sinus (+). Ao, ascending aorta; IIV, inferior wall; LA, left atrium; LV, left ventricle; RVO, right ventricular outflow; VS, ventricular septum.
Rheumatic involvement of the mitral valve causes chordal shortening and thickening without annular dilatation. Rheumatic mitral stenosis is produced by chordal and commissural fusion, often with calcification, whereas rheumatic mitral insufficiency results from scar retraction of leaflets and chords. Chronic postinfarction mitral regurgitation is associated with left ventricular dilatation and scarring of a papillary muscle and its subjacent ventricular free wall. Leaflet tethering and annular dilatation cause malcoaptation of the contact surfaces of the mitral leaflets. Acute postinfarction mitral regurgitation can be associated with partial or complete rupture of a papillary muscle, usually the posteromedial one. Three-dimensional imaging of the atrial (surgical view) and ventricular (interventional cardiologist fluoroscopic view) surfaces of the mitral valve is now possible with echocardiography (see Chap. 15).

Anatomically important structures during percutaneous or surgical mitral valve repair or replacement include the left circumflex coronary artery, which courses within the left AV groove near the anterolateral commissure, and the coronary sinus, which courses within the left atrioventricular groove adjacent to the annulus of the posterior mitral leaflet (see Fig. 4–21A).

AORTIC VALVE

The aortic valve, like the pulmonary valve, is composed of three components (ie, annulus, cusps, and commissures). In contrast to the mitral and tricuspid valves, the two semilunar valves have no tensor apparatus (ie, chordae tendineae or papillary muscles). The commissures form tall, peaked spaces between the attachments of adjacent cusps (Figs. 4–28 and 4–29) and attain the level of the aortic sinotubular junction, the ridge that separates the sinus and tubular portions of the ascending aorta (originally described by da Vinci as the "supraortic ridge") (see Fig. 4–29). The functional aortic valve orifice can be at the sinotubular junction or proximal to it. The three half moon–shaped (semilunar) aortic cusps form pocket-like tissue flaps that are avascular. In only approximately 10% of hearts are they truly equal in size. In 67% of hearts, either the right or posterior cusp is larger than the other two. Just below the free edge of each cusp is a biscalloped ridge-like closing edge (see Fig. 4–29). At the center of each cusp, the closing edge meets the free edge and forms a small fibrous mound, the nodule of Arantius (see Fig. 4–29). Between the free and closing edges, to each side of the nodule are two crescent-shaped areas known as the closing surface or lunulae, which represent the sites of cusp apposition during valve closure. Lunular fenestrations, near the commissures, are common and increase in size and incidence with age (Fig. 4–30). However, owing to their position distal to the closing edge, they rarely produce valvar incompetence. When viewed from above, the linear distance along the closing edge of a cusp is much greater than the straight-line distance between its two commissures (see Fig. 4–28). This extra length of cusp tissue is necessary for nonstenotic opening and nonregurgitant closure of the valve. A virtual line connecting the bases of each cusp forms the basal...
FIGURE 4–26. Components of the mitral valve. A. Each leaflet has a large clear zone (CZ) and a smaller rough zone (RZ) between its free edge and closing edge (dotted line). A fan-like commissural chordae tendinea (+) connects the tip of the papillary muscle to the commissure. B. Schematic diagram of an open anterior mitral leaflet comparable to A. Section obtained along the dotted lines shows the relationship of the mitral annulus and free edge to the closing edge.

FIGURE 4–27. Flail P2 segment. Enface three-dimensional TEE LA view of mitral valve showing mitral cleft (red arrow) between the P1 and P2 scallops. Arrowheads point to ruptured chordae (A). Mitral regurgitation (yellow arrow) through the P1-P2 cleft (B). Intraoperative view of the mitral cleft (arrow) (C). A1-A2-A3, lateral to medial anterior leaflet scallops; LA, left atrium; P1-P2-P3, lateral to medial posterior mitral leaflet scallops; TEE, transesophageal echocardiogram. Used with permission of Mayo Foundation for Medical Education and Research. All rights reserved.
CHAPTER 4: Functional Anatomy of the Heart

ring, although the true ventriculoarterial junction is just distal to it. The diameters of the basal ring and sinotubular junction are usually similar and represent the narrowest portions of the aortic root. Accurate measurements of these regions are critical to success of transcatheter or surgical aortic valve replacement. Lunular fenestrations (see Fig. 4–30), depending on their severity, may preclude the ability to perform valve-sparing aortic root replacement in patients with aortic root aneurysms. In hearts from adults with bicuspid valves and other congenital aortic valve disease, the annular diameter is usually enlarged. In contrast, patients with normal aortic cusps and central aortic regurgitation show enlargement at the level of the sinotubular junction. A prebypass intraoperative transesophageal long-axis view of the left ventricular outflow tract is used to measure the aortic valve annular diameter prior to replacement by a homograft. In doing so, precious bypass time is saved while the homograft is being prepared. Disease processes that produce commissural fusion such as rheumatic valvulitis or decrease cusp mobility such as fibrosis or calcification can lead to aortic stenosis. In contrast, those disorders that decrease cusp size, such as rheumatic valvulitis, or that cause aortic root dilatation can lead to aortic regurgitation. Combinations of these processes can produce combined stenosis and regurgitation.

The commissure between the right and posterior aortic cusps overlies the membranous septum (Fig. 4–31) and contacts the commissure between the anterior and septal leaflets of the tricuspid valve (see Fig. 4–42). The commissure between the right and left aortic cusps contacts its corresponding pulmonary commissure and overlies the infundibular septum (see Fig. 4–12D). The intervalvular fibrosa, at the commissure between the left and posterior aortic cusps, fuses the aortic valve to the anterior mitral leaflet.

The central location of the aortic valve (see Fig. 4–21) provides the electrophysiologist with a unique vantage point from which arrhythmias in the right or left ventricular outflow tract, right or left atrium, can be ablated. During surgical or transcatheter aortic valve replacement, the anterior mitral leaflet, left bundle branch, or coronary ostia can be injured inadvertently. Annular abscesses caused by infective endocarditis involving the aortic valve can burrow into adjacent structures and thereby produce endocarditis of the other valves; conduction disturbances with septal involvement; aortoatrial, aortopulmonary artery, or aortoventricular fistulas; pericarditis; or fatal hemopericardium.

**FIGURE 4–28.** Each cusp of a semilunar valve is pocket-shaped. The aortic valve is viewed from above in simulated closed (A) and open (B) positions, showing the three commissures (arrows). Note that the length of the closing edge exceeds the straight-line distance between the commissures.

**FIGURE 4–29.** An opened aortic valve shows the right (R), left (L), and posterior (P) cusps. The dashed line marks the closing edge. Between the free and closing edges of each cusp are two lunular areas, representing the surfaces of apposition between adjacent cusps during valve closure. The commissures (*) attain the level of the aortic sinotubular junction (STJ). Conus, conus coronary ostium; LC, left coronary ostium; LV, left ventricle; N, nodule of Arantius; RC, right coronary ostium.

**FIGURE 4–30.** Aortic cusp fenestrations (arrows) occurring in the lunular regions near the commissures. This is a common age-related degenerative finding and normally accounts for little or no aortic valve regurgitation.

**FIGURE 4–31.** The commissure between the right and posterior aortic cusps (arrow) overlies the transilluminated membranous septum (arrowhead). A, anterior mitral leaflet; Ao, ascending aorta; LV, left ventricle; P, posterior aortic cusp; R, right aortic cusp.
§ PULMONARY VALVE
The pulmonary valve is virtually identical in design to the aortic valve.27 The pulmonary artery sinuses are partially embedded within the muscle bundles of the right ventricular infundibulum, particularly adjacent to the right and left sinuses.28,29 In pulmonary valve atresia with an intact ventricular septum, hypertrophy of the muscle bundles is rare and the narrow right ventricular outflow tract accentuates this relationship.30 Also, unlike the aortic valve, which is continuous with the mitral valve, the pulmonary and tricuspid valves are separated by infundibular muscle.27 Ventricular arrhythmias can originate from the pulmonary valve or supravalvular portion of the pulmonary arteries.4 The pulmonic valve is typically cephalad to the aortic valve (see Fig. 4–35) such that the supravalvular portion of the aortic valve lies in immediate proximity to portions of the pulmonary valve. This relationship is important for the electrophysiologist.8

§ AGE-RELATED VALVE CHANGES
Several age-related changes in the cardiac valves can have clinical significance.21 In normal hearts, the thickness of the aortic and mitral leaflets increases progressively with each decade, particularly along their closure margins.20 Probably the most common clinical manifestation of these changes is aortic valve sclerosis, characterized by valve thickening without hemodynamic dysfunction.21 However, age-related degenerative calcification of an otherwise anatomically normal-appearing aortic valve can result in progressive aortic stenosis.21 Age-related thickening along the nodule of Arantius and closing edges can be associated with the formation of whisker-like projections called Lambi excrescenses. These fine fibrous-like strands also can develop on the mitral valve.17 Lambi excrescences can be detected by echocardiography and have been associated with cardioembolic stroke.22 Larger lesions with complex branching, having the appearance of a sea anemone, are considered to be either neoplastic or reactive and are known as papillary fibroelastomas.23

The circumferences of all four major cardiac valves increase with age in normal hearts. This is particularly evident in the semilunar valves.21 Age-related annular dilatation of the aortic valve can result in aortic regurgitation.21 Mitral annular calcification (MAC) is rare in women younger than 70 years of age but is present in 40% of women older than 90 years.22 MAC almost invariably involves only the posterior leaflet and forms a C-shaped ring of annular and subannular calcium.23 It can impede subannular ventricular contraction, thereby resulting in mitral regurgitation, and marked MAC can result in severe mitral inflow stenosis that may be amenable to valve-in–native valve implantation (see Chap. 50). Three-dimensional reconstruction of intracardiac structures such as heart valves from CT or MRI images using 3D printing is now available20,22 and allows for testing of feasibility of transcatheter interventions. Because of the proximity of the posteromedial commissure to the AV (His) bundle, MAC can be associated with AV block.20 Because of the increasing size of the aging population, degenerative calcific aortic disease is increasing in frequency.21

§ CARDIAC GROOVES, CRUX, AND MARGINS
The AV groove (or sulcus) encircles the heart and defines its base. It separates the atria from the ventricles (Fig. 4–32). The two ventricles are separated by the anterior and posterior (inferior) interventricular grooves (or sulci), which define the plane of the ventricular septum (see Figs. 4–5A and 4–32).

With age, fat tends to accumulate in increasing amounts in the epicardium, particularly in the AV grooves.21,24 Increased epicardial fat deposits can be associated with increased risk of cardiac rupture after acute transmural myocardial infarction.24 Excess fat in the atrial septum is called lipomatous hypertrophy of the atrial septum (see Fig. 4–48) and can result in a thickness exceeding that of the ventricular septum. Fat in the right ventricular free wall can be detected on CT (Fig. 4–33); its excess

**FIGURE 4–32.** External cardiac crux. View of the diaphragmatic aspect of the heart shows the intersection of the atrioventricular (arrowheads), posterior interventricular (long arrow), and interatrial (small arrow) grooves at the external cardiac crux (A). A. Diagram. B. Cardiac specimen. LA, left atrium; LV, left ventricle; RV, right ventricle.

**FIGURE 4–33.** Noncontrast CT four-chamber view of the heart. Fat is seen within the epicardium (solid lines), and right ventricular free wall (dashed lines). Arrowheads point to fat in the atrioventricular groove. RV, right ventricle.
accumulation can be associated with increasing age, obesity, myocardial injury or arrhythmogenic cardiomyopathy.\textsuperscript{15}

Along the surface of the heart, the right and circumflex coronary arteries travel in the right and left AV grooves, respectively, and the left anterior and posterior descending coronary arteries course along the anterior and posterior (or inferior) interventricular grooves, respectively (see Figs. 4–5A and 4–32). The external cardiac crux is the cross-shaped intersection between the atrioventricular, posterior interventricular, and interatrial grooves (see Fig. 4–32). Its internal counterpart (internal crux) is the posterior intersection between the mitral and tricuspid annuli and the atrial and ventricular septa (see Figs. 4–16B and 4–36).

The junction between the anterior and inferior free walls of the right ventricle forms a sharp angle known as the acute margin. The rounded lateral wall of the left ventricle forms the obtuse margin.\textsuperscript{14}

\section*{RIGHT VENTRICLE}

The right ventricle is a right-anterior structure. It is comprised of an inlet and trabecular and outflow segments\textsuperscript{14} (Fig. 4–34). The inlet component extends from the tricuspid annulus to the insertions of the papillary muscles. An apical trabecular zone extends inferiorly beyond the attachments of the papillary muscles toward the ventricular apex and about halfway along the anterior wall.\textsuperscript{14} This muscular meshwork is the usual site of insertion of transvenous ventricular pacemaker electrodes and the preferred site for positioning of the tip of an implantable cardioverter defibrillator lead. During right ventricular endomyocardial biopsy, tissue generally is obtained from the septum, usually under echocardiographic or fluoroscopic guidance. Disruption of a portion of the tricuspid valve tensor apparatus is a potential complication of right-sided cardioverter defibrillator lead placement.\textsuperscript{24}

The moderator band (\textit{moderator} means moderator, or controlling factor) is a muscle band that lies between the leaflets of the tricuspid valve and connects the endocardium of the right ventricular wall to the endocardium of the right atrium. It is made up of three components (ie, parietal band, infundibular septum, and septal band) that can appear as distinct structures or can merge together\textsuperscript{24,25} (see Fig. 4–34). The parietal band is a free-wall structure, whereas the adjacent infundibular septum is intracardiac and separates the two ventricular outflow tracts beneath the right and left cusps of the semilunar valves\textsuperscript{24,25} (Fig. 4–35; see also Fig. 4–12D). The septal band forms a Y-shaped muscle, the two upper limbs of which cradle the infundibular septum. From this branching point of the septal band emanates the medial tricuspid papillary muscle\textsuperscript{25,26} (see Fig. 4–34). The moderator band forms an intracavitary muscle that connects the septal band with the anterior tricuspid papillary muscle (see Fig. 4–34A).

\section*{LEFT VENTRICLE}

The left ventricle, like the right ventricle, is made of an inlet portion comprised of the mitral valve apparatus, a subaortic outflow portion, and a finely trabeculated apical zone.\textsuperscript{27} The left ventricular free wall is normally thickest toward the base and thinnest toward the apex, where it averages only 1 to 2 mm in thickness, even in hypertrophied hearts.\textsuperscript{27} Structurally, the left and right ventricles differ considerably.\textsuperscript{24,27}

Normally, the left ventricular free-wall and septal thicknesses are three times the thickness of the right ventricular free wall. The mitral and aortic valves share fibrous continuity, whereas the papillary band separates the tricuspid and pulmonary valves. Whereas the mitral valve has an elliptical orifice and no septal attachments, the tricuspid valve has a triangular orifice and numerous direct septal attachments (see Fig. 4–23). The right ventricular apex is much more coarsely trabeculated than the aortic valve.

\section*{SUPRAVENTRICULARIS}

The supraventricularis (CSV), which consists of the parietal band (PB), infundibular septum (IS), and septal band (SB)\textsuperscript{14} (Fig. 4–34), serves as the typical site of insertion of transvenous ventricular pacemaker leads. The moderator band (\textit{moderator} means moderator, or controlling factor) forms an intracavitary muscle that controls the endocardium of the right ventricular wall to the endocardium of the right atrium. It is made up of three components (ie, parietal band, infundibular septum, and septal band) that can appear as distinct structures or can merge together\textsuperscript{24,25} (see Fig. 4–34). The parietal band is a free-wall structure, whereas the adjacent infundibular septum is intracardiac and separates the two ventricular outflow tracts beneath the right and left cusps of the semilunar valves\textsuperscript{24,25} (Fig. 4–35; see also Fig. 4–12D). The septal band forms a Y-shaped muscle, the two upper limbs of which cradle the infundibular septum. From this branching point of the septal band emanates the medial tricuspid papillary muscle\textsuperscript{25,26} (see Fig. 4–34). The moderator band forms an intracavitary muscle that connects the septal band with the anterior tricuspid papillary muscle (see Fig. 4–34A).

\section*{REFERENCES}

FIGURE 4–36. Internal cardiac crus. Four-chamber slice of the heart shows the characteristic normal apical displacement of the tricuspid valve septal leaflet insertion (arrowhead) when compared with septal insertion of the mitral valve (solid arrow). This tomographic section also shows the interatrial septum (IAS), anteroseptal septum (AVS), and interventricular septum (IVS). Open arrow points to fossa ovalis. LA, left atrium; LLPV, left lower pulmonary vein; LV, left ventricle; RA, right atrium; RLPV, right lower pulmonary vein; RV, right ventricle.

its counterpart on the left (see Figs. 4–9B and 4–18C). The distinctive differences in apical trabeculations persist even in markedly hypertrophied or dilated hearts.17

The annular attachment of the septal leaflet of the tricuspid valve inserts more apically than that of the anterior mitral leaflet, allowing distinction between the right and left ventricles by four-chamber imaging (Fig. 4–36). Exceptions include partial AV septal defects and double-inlet ventricles in which the two valve annuli are at the same level. Ebstein anomaly is characterized by exaggeration of apical displacement of the septal and posterior tricuspid leaflets resulting in an atrialized portion of the right ventricular chamber.16,17 Morphologic differentiation of the right and left ventricles is particularly important in congenital heart disease. The morphologic tricuspid valve virtually always connects to a morphologic right ventricle, whereas the morphologic mitral valve connects to a morphologic left ventricle.14,16 Because of the rightward bulging of the ventricular septum, the left ventricular chamber appears circular in cross-section, whereas the right ventricular chamber has a crescentic appearance (see Fig. 4–23). Tomographic segmental left ventricular anatomy is reviewed in the following section on the coronary arteries.

Left ventricular false tendons, also referred to as pseudotendons or bands,26 are discrete, thin, cord-like fibromuscular structures that connect two walls, the two papillary muscles, or a papillary muscle to a wall, usually the ventricular septum (Fig. 4–37). However, false tendons, as the name implies, are not attached to the mitral leaflets. Chordal attachments between the mitral leaflets and the ventricular septum are abnormal and are usually associated with AV septal defects or straddling AV valves.16 False tendons are common anatomic variants of the normal left ventricle, occurring in 50% of hearts, and can become calcified with age (Fig. 4–38). They are more frequently observed in men, but their incidence does not appear to be age related.26 It has been suggested that they can be the cause of innocent systolic musical murmurs and rarely form part of an arrhythmogenic conduction circuit.26 Although they are readily detectable by echocardiography, they can be misinterpreted by the inexperienced sonographer as pathologic structures such as ruptured chords, mural thrombi, or vegetations.25,26

Prominent left ventricular trabeculations27 are another common anatomic normal variant that can be an even greater source of misinterpretation by 2D echocardiography in patients with suspected mural thrombus. They are defined as discrete, thick muscle bundles that generally connect the free wall to the septum (Fig. 4–39). Less common

FIGURE 4–37. Various locations of left ventricular false tendons. A. Two false tendons (arrow) from posteromedial mitral papillary muscle (PM) to ventricular septum (VS), representing the most common location. B. Complex branching false tendon (arrow) with origin from the left ventricular free wall (FW) and insertions into the ventricular septum (VS) and base of posteromedial mitral papillary muscle (PM).

FIGURE 4–38. Calcified left ventricular false tendon (arrows) seen in short-axis view.
FIGURE 4–39. Prominent left ventricular trabeculations. Multiple large muscle bundles extend from the anterior free wall to the septum (probe with white arrow), and one bundle extends from one portion of the posterior septum to another (probe with black arrow). Such trabeculations are more prominent in noncompaction cardiomyopathy.

Attachments include papillary muscle to the septum, septum to septum, or free wall to free wall. In noncompaction of the left ventricular myocardium, also known as spongy myocardium, there is persistence of prominent ventricular trabeculations and deep intertrabecular recesses caused by arrest in the normal in utero process of myocardial compaction. The associated clinical manifestations and age at onset of symptoms (ie, typically a dilated cardiomyopathy) are highly variable.

VENTRICULAR SEPTUM

The ventricular septum is a complex intracardiac partition and is comprised of four parts: (1) inlet, (2) trabecular, (3) membranous, and (4) infundibular. The plane of the infundibular portion (see Figs. 4–12D and 4–35) is different from that of the three other portions. This anatomic relationship is important in many forms of congenital heart disease in which the infundibular septum is dissociated from the remainder of the ventricular septum (eg, malalignment forms of ventricular septal defects in tetralogy of Fallot and in double-outlet right ventricle).

■ VENTRICULAR SEPTUM

The ventricular septum also can be divided into muscular and membranous portions (Figs. 4–40 and 4–41). The membranous septum lies beneath the right and posterior (noncoronary) aortic cusps (see Fig. 4–31) and contacts the mitral and tricuspid annuli (Fig. 4–42). The membranous septum in conjunction with the right fibrous trigone with which it is continuous fuses the commissure between the anterior and septal tricuspid leaflets (see Fig. 4–21B). The majority of clinically significant ventricular septal defects involve the membranous septum. Because of normal angulation between the infundibular septum and remaining ventricular septum, the septal surface follows the course of an inverted “S” (moving from apex to aortic valve). The basal half of the ventricular septum is smooth walled, whereas the apical half is characterized by numerous small and irregularly arranged trabeculations.

Clinically relevant age-related anatomic changes include a disproportionate increase in ventricular septal thickness regardless of sex and in the absence of a history of hypertension. This is associated with an appreciable increase in the ratio of ventricular septal to left ventricular free-wall thickness, often exceeding 1.3 in patients older than 60 years (Fig. 4–43). This can be caused in part by the accentuation of the sigmoid shape of the basal septum (Fig. 4–44). Age-related ventricular septal angulation can have clinical importance because it can morphologically mimic hypertrophic cardiomyopathy, particularly if complicated by the indiscriminate use of volume-depleting diuretics or afterload-reducing agents.

ATRIAL SEPTUM

When viewed from its right aspect, the atrial septum is composed of interatrial and AV regions (see Fig. 4–36). The interatrial portion is characterized by the fossa ovalis, which is the anatomic hallmark of a morphologic right atrium (Fig. 4–45A). Its outer muscular rim is a horseshoe-shaped limbus, and its central depression is the valve of the fossa ovalis (see Fig. 4–45A). The potential interatrial passageway...
between the limbus and the valve (which is patent throughout fetal life) is the foramen ovale (Figs. 4–45B and 4–46). When viewed from the left atrium, the atrial septum is entirely interatrial because the AV component lies below the mitral annulus between the left ventricle and right atrium. Likewise, the limbus of the fossa ovalis is completely covered by its opaque valve and is not directly visible from the left atrium. The fossa ovalis and surrounding anatomy can be readily imaged by 3D transesophageal echocardiography (Fig. 4–47A and B).

The foramen ovale is anatomically closed in approximately two-thirds of adults, but in the remaining one-third it remains patent and, therefore, a potential source for shunts and paradoxical embolism. Stretching of the atrial septum, when the atria are markedly dilated, can transform a patent foramen ovale into an acquired atrial septal defect. The posterior aortic sinus abuts against the interatrial septum anterior and superior to the limbus (see Fig. 4–12D). During transseptal procedures, care must be taken to stay within the confines of the valve of the fossa ovalis to avoid perforation of an aortic sinus. Besides fluoroscopy, transesophageal echocardiography may be used to guide the transseptal puncture during catheter-based left heart interventions. Congenital atrial septal defects most often occur in the region of the valve of the fossa ovalis (so-called secundum-type defects). Redundant valve tissue can form an aneurysm of the valve of the fossa ovalis.

The AV portion of the atrial septum is made of major muscular and minor membranous components and separates the right atrium from the left ventricle. The AV septum corresponds roughly to the triangle of Koch (see Fig. 4–77), an important anatomic surgical landmark because it contains the AV node and proximal portion of the AV (His) bundle. Thus, during tricuspid annuloplasty procedures and patch closures of membranous ventricular septal defects, care must be taken to avoid injury to the conduction system. The muscular component of the AV septum is interposed between the membranous septum anteriorly and the internal cardiac crux posteriorly.

When defects occur in the muscular AV septum, the mitral annulus usually drops to the same level as the tricuspid annulus, so the defect becomes primarily interatrial (primarily atrial septal defect), and the AV conduction tissues are displaced inferiorly. Lipomatous hypertrophy of the atrial septum is characterized by excessive accumulation of adipose tissue within the limbus of the fossa ovalis but always sparing the valve of the fossa (Fig. 4–48). Lipomatous hypertrophy of the atrial septum occurs commonly, but not exclusively, in older and obese persons. Although readily detected by echocardiography, it can be misinterpreted as a thrombus or neoplasm.
CHAPTER 4: Functional Anatomy of the Heart

eustachian valve toward the foramen ovale, and superior vena caval blood is directed toward the tricuspid valve (Fig. 4–52). Thus trans-septal cardiac catheterization is more easily accomplished via the inferior vena cava, whereas instrumentation of the right ventricular apex (eg, endomyocardial biopsy or placement of a lead) is more easily accomplished via the superior vena cava.

■ LEFT ATRIUM

The pulmonary vein orifices lie on the posterolateral (left pulmonary veins) and posteromedial (right pulmonary veins) aspects of the left atrial cavity. The left and right upper pulmonary veins are directed anterosuperiorly, whereas the lower veins enter the left atrium nearly perpendicular to the posterior atrial wall (Fig. 4–53). Left atrial muscle extends some distance within the pulmonary veins. The resultant cuff of muscle acts as a sphincter during atrial systole and can be the source of focal atrial fibrillation that is amenable to catheter ablation (see Fig. 4–2).

The atrial appendage arises anterolaterally and lies in the left AV groove atop the proximal portion of the left circumflex coronary artery and, in some individuals, the left main coronary artery (see Figs. 4–21A and 4–53). The left atrial appendage (LAA) is usually multilobed (Fig. 4–54) and narrower than its right atrial counterpart, and it exhibits more variability in shape (Fig. 4–55). Four basic morphologic patterns of LAA have been described: windsock, cactus, cauliflower, and chicken wing (Fig. 4–55). Importantly, there is some relationship between stroke and LAA morphology, with the chicken wing variety showing less likelihood of embolic events. There are also age- and sex-related differences in the dimensions of the appendage. With increasing use of transesophageal echocardiography to search for a cardiac source of embolism and to guide cardioversion and percutaneous balloon valvuloplasty procedures, a thorough appreciation of the variations in normal left atrial appendage morphology has become important, because a thrombus can be missed if all lobes in the appendage are not visualized. Appreciation of LAA morphology and accurate measurements of LAA dimensions are crucial to successful device closure of the LAA in patients with atrial fibrillation. In contrast to the right atrial free wall, the left has no crista terminalis and no pectinate muscles outside its appendage. Either atrial appendage may serve as a vantage point to access and ablate arrhythmias in the adjacent segment of the right ventricular outflow tract (see Figs. 4–34A and 4–53).
SECTION 2: Foundations of Cardiovascular Medicine

FIGURE 4–47. Three-dimensional TEE RA view of the fossa ovalis and surrounding anatomy. The fossa ovalis is seen as an oval-shaped crater (arrowheads). The atrial wall anterior to the fossa (double-headed arrow) abuts the posterior aortic sinus. White arrow points to aortic root. Yellow arrow points to the SVC (A). Spatial relationships of fossa ovalis (red arrows). White arrow points to SVC superiority, an asterisk marks the eustachian ridge inferiorly (B). A, anterior; I, inferior; P, posterior; RA, right atrium; S, superior; SVC, superior vena cava; TEE, transesophageal echocardiogram. Used with permission of Mayo Foundation for Medical Education and Research. All rights reserved.

FIGURE 4–48. Four-chamber slice through the heart showing lipomatous hypertrophy of the atrial septum (arrows) and descending thoracic aortic aneurysms can compress this chamber. A large hiatal hernia also can abut against the left atrium and present as a mass.

The marked increase in the incidence of atrial fibrillation from the fourth to the ninth decades of life can be caused by dilatation of the left atrium consequent to left ventricular diastolic dysfunction or mitral valve disease.

CORONARY ARTERIES AND VEINS

A detailed description of the spectrum of coronary artery anatomy including the many variations in the number and size of branches and course of the different arteries is beyond the scope of this chapter. The interested reader is referred to the elegant anatomic work by Wallace McAlpine published in 1975. The focus of the following discussion, therefore, is to introduce the reader to the clinically relevant anatomy of the coronary circulation, with special emphasis on tomographic analysis of regional blood flow.

From the right and left aortic sinuses arise the right and left coronary arteries, respectively, and their ostia, which normally originate about two-thirds the distance from the aortic annulus to the sinotubular junction and about midway between the aortic valve commissures (Fig. 4–56; see also Fig. 4–29). Whereas the right coronary artery arises nearly perpendicularly from the aorta, the left arises at an acute angle (Fig. 4–57). Rarely, the anterior descending and circumflex arteries arise separately from a double-barrel left coronary ostium. Ostial stenosis most commonly results from atherosclerosis and degenerative calcification of the aortic sinotubular junction, which often overlies the right aortic sinus. Less often it is caused by aortic dissection or by aortitis associated with syphilis or ankylosing spondylitis. Stenosis of the right coronary ostium is much more frequent than that of the left. Intraluminal injury can complicate coronary angiography, intraoperative coronary perfusion, or aortic valve replacement. Atherosclerosis or thrombosis of the most proximal portion of either coronary artery can mimic true ostial stenosis.

The right coronary artery is embedded in adipose tissue throughout its course within the right AV groove. Tricuspid annuloplasty or replacement can be complicated by injury to the right coronary artery.
to 60% of persons, its first branch is the conus artery (Fig. 4–58), which supplies the right ventricular outflow tract and forms an important collateral anastomosis (circle of Vieussens), just below the pulmonary valve, with an analogous branch from the left anterior descending (LAD) coronary artery. Among the numerous marginal branches of the right coronary artery that supply the remainder of the right ventricular free wall, the largest branch travels along the acute margin from base to apex (Fig. 4–56). In at least 70% of human hearts, the posterior descending artery arises from the distal right coronary artery (see Fig. 4–56). The posterior descending and distal posterolateral branches of a dominant right coronary artery supply the basal and middle inferior wall, basal (inlet) inferior septum, right bundle branch, AV node, AV (His) bundle, posterior portion of the left bundle branch, and posteromedial mitral papillary muscle. The infundibular septum is supplied by the descending septal artery, which usually originates from the proximal right or conus coronary artery. Among the numerous marginal branches of the right coronary artery that supply the remainder of the right ventricular free wall, the largest branch travels along the acute margin from base to apex (see Fig. 4–56). In at least 70% of human hearts, the posterior descending artery arises from the distal right coronary artery (see Fig. 4–56). The posterior descending and distal posterolateral branches of a dominant right coronary artery supply the basal and middle inferior wall, basal (inlet) inferior septum, right bundle branch, AV node, AV (His) bundle, posterior portion of the left bundle branch, and posteromedial mitral papillary muscle.
The left main coronary artery travels for a very short distance along the epicardium between the pulmonary trunk and left atrium (see Figs. 4–56 and 4–58). It then divides into anterior descending and circumflex arteries (see Figs. 4–56 and 4–58). An intermediate artery (ramus intermedius) also may arise at this division, thus forming a trifurcation rather than a bifurcation, and follow the course of a circumflex marginal branch\textsuperscript{14,16,17} (see Fig. 4–58).

The LAD courses within the epicardial fat of the anterior interventricular groove, wraps around the cardiac apex, and travels a variable distance along the inferior interventricular groove toward the cardiac base. Its septal perforating branches supply the anterior septum and apical septum. The first septal perforating branch supplies the AV (His) bundle and proximal left bundle branch\textsuperscript{17} (Fig. 4–59). In patients with symptomatic hypertrophic obstructive cardiomyopathy, nonsurgical septal reduction by percutaneous transluminal occlusion of septal branches of the LAD is a therapeutic approach aimed at reducing the outflow gradient.\textsuperscript{14} The epicardial diagonal branches of the LAD supply the anterior left ventricular free wall, part of the anterolateral mitral papillary muscle, and the medial one-third of the anterior right ventricular free wall.\textsuperscript{14,16,17} Although short segments of the LAD can travel within the myocardium (covered by a so-called myocardial bridge) (Fig. 4–60), the resulting systolic luminal narrowing is probably benign in the vast majority of people.\textsuperscript{27} However, whereas the prevalence of angiographically recognized myocardial bridging is only 0.5% to 1.6% in the general population, it is reported to be 28% in children and 30% to 50% in adults with hypertrophic cardiomyopathy.\textsuperscript{35} More important, myocardial bridging appears to be associated with a poor prognosis (higher incidence of myocardial ischemia and sudden death) in patients with hypertrophic cardiomyopathy regardless of age.\textsuperscript{35}

The left circumflex coronary artery courses within the adipose tissue of the left atrioventricular groove (see Fig. 4–21A) and commonly terminates just beyond its large obtuse marginal branch (see Fig. 4–56). It supplies the lateral left ventricular free wall and a portion of the anterolateral mitral papillary muscle.\textsuperscript{14,16,17}

Along the inferior surface of the heart, the length of the right coronary artery varies inversely with that of the circumflex artery.
The artery that crosses the cardiac crux and gives rise to the posterior descending branch represents the dominant coronary artery. Dominance is right in 70% of human hearts, left in 10%, and shared in 20%. In patients with a congenitally bicuspid aortic valve, the incidence of left coronary dominance is 25% to 30%. Recent advances in CT technology allow for 3D reconstruction of the epicardial coronary arteries and veins (Fig. 4–58).

The coronary venous circulation is comprised of coronary sinus, cardiac veins, and thebesian venous systems (Fig. 4–59).
emission tomography [PET], and MRI), for clinicopathologic correlations (Fig. 4–64), a combined tomographic and segmental approach to coronary artery anatomy is recommended.\textsuperscript{17,37,38}

Ventricular mass is made of the left and right ventricular free walls and the partitioning ventricular septum. Three levels (ie, basal, midventricular, and apical) are used to divide the base-apex length of the left ventricle into thirds (Fig. 4–65). The basal third includes that portion between the mitral annulus and the tips of the papillary muscles. The midventricular third is from the papillary muscle to the most apical insertion point of these muscles into the left ventricular free wall. The apical third includes the remainder of the ventricle, from the insertion of the papillary muscles to the left ventricular apex. A similar approach can be applied to the right ventricle.\textsuperscript{14,16,17}

The ventricular septum can be divided into anteroseptal and inferoseptal segments, and the left ventricular free wall is divided into anterior, lateral, and inferior segments at the basal and midventricular levels (see Fig. 4–65). The left ventricular apical level consists of four segments (ie, septum, inferior, lateral, and anterior) (see Fig. 4–65). This regional approach is not arbitrary and has been verified by studies of normal, dilated, and hypertrophied hearts. According to this system, there are 16 left ventricular segments that can be evaluated for regional abnormalities. This regional approach can also be used to assess transmural infarct size, because the percentage of left ventricular

During cardiac operations, cardioplegic solution can be administered retrograde into the coronary sinus. In patients with the Wolff-Parkinson-White preexcitation syndrome and left-sided bypass tracts, the ablation catheter during electrophysiologic studies can be positioned within the coronary sinus and great cardiac vein adjacent to the mitral valve ring to localize the aberrant conduction pathway.\textsuperscript{17} Coronary venous anatomy is extremely variable, however. This can have important practice implications. The coronary veins, via the coronary sinus, provide access to percutaneous epicardial mapping and pacing of the ventricles and ablation of subepicardial arrhythmogenic foci\textsuperscript{36} (Fig. 4–63). For biventricular pacing, optimal left ventricular pacing lead position is within the posterolateral branch of the coronary sinus, followed by the lateral branch.

Coronary artery disease is associated with regional abnormalities in ventricular structure and function. Because analysis of segmental myocardial perfusion or contractility is the cornerstone of tomographic imaging techniques (stress echocardiography, SPECT imaging, positron emission tomography [PET], and MRI), for clinicopathologic correlations (Fig. 4–64), a combined tomographic and segmental approach to coronary artery anatomy is recommended.\textsuperscript{17,37,38} Ventricular mass is made of the left and right ventricular free walls and the partitioning ventricular septum. Three levels (ie, basal, midventricular, and apical) are used to divide the base-apex length of the left ventricle into thirds (Fig. 4–65). The basal third includes that portion between the mitral annulus and the tips of the papillary muscles. The midventricular third is from the papillary muscle to the most apical insertion point of these muscles into the left ventricular free wall. The apical third includes the remainder of the ventricle, from the insertion of the papillary muscles to the left ventricular apex. A similar approach can be applied to the right ventricle.\textsuperscript{14,16,17}

The ventricular septum can be divided into anteroseptal and inferoseptal segments, and the left ventricular free wall is divided into anterior, lateral, and inferior segments at the basal and midventricular levels (see Fig. 4–65). The left ventricular apical level consists of four segments (ie, septum, inferior, lateral, and anterior) (see Fig. 4–65).

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FIGURE 4–64. Short-axis views. A. Collage of anatomic sections obtained by “bread slicing” the heart in its short-axis plane viewed from the apex toward the base of the heart. B. Comparable MRI images. C. Comparable sestamibi SPECT images of the left ventricle showing normal myocardial perfusion at rest and with exercise. SA, short axis.

FIGURE 4–65. Schematic diagram of the three levels of short-axis tomographic views used in echocardiography for 16-segment wall motion analysis. A, anterior; AL, anterolateral; AS, anterior ventricular septum; I, inferior; IL, inferolateral; IS, inferior ventricular septum; L, lateral; LV, left ventricle; LVOT, left ventricular outflow tract; P, posterior; PL, posterolateral; PS, posterior ventricular septum; RV, right ventricle; S, septum. The most basal segment of the inferior wall is the anatomically true posterior segment. At this level, the adjacent ventricular septum is commonly referred to as either the basal posterior septum or the basal inferior septum and the adjacent lateral wall as either the basal posterolateral wall or the basal inferolateral wall.
mass contributed by any particular region is not altered in any significant manner by symmetric hypertrophy or dilatation.\textsuperscript{17}

\section*{Regional Coronary Artery Supply}

The ventricular regions described tend to correlate well with common patterns of coronary artery distribution\textsuperscript{14,16,17} (Figs. 4–66 and 4–67). Any specific epicardial coronary artery generally will supply a certain cluster of regions. For example, in a typical right-dominant system, the LAD would supply the midventricular and basal segments of the anterior and anterolateral walls and anterior septum and all apical segments. The left circumflex artery would supply the midventricular and basal inferolateral segments, and the right coronary artery would supply the midventricular and basal inferior wall and inferior septum (see Fig. 4–67). However, because the patterns of coronary distribution are so highly variable, these correlations between coronary blood flow and regional anatomy are not precise. For example, a hyperdominant right coronary artery can supply the apex, and a large, obtuse marginal branch of the circumflex artery can supply the anterolateral or inferior wall. Also, any given myocardial region can, in some people, receive its blood supply from the branches of two independent major epicardial arteries.\textsuperscript{14,16,17}

In old age, the coronary arteries become dilated and tortuous (Fig. 4–68). Ultrafast electron beam CT is very useful for the detection of calcified plaques within the coronary arteries.

\section*{Coronary Collaterals and Microcirculation}

Collateral channels provide communication between the major coronary arteries and their branches.\textsuperscript{17} If stenosis of an epicardial coronary artery produces a pressure gradient across such a vessel, the collateral channel can dilate with time and provide a bypass avenue for blood flow beyond the obstruction. Such functional collaterals can develop between the terminal extensions of two coronary arteries, between the side branches of two arteries, between branches of the same artery, or within the same branch (via the vasa vasorum). These are most common in the ventricular septum (between septal perforators of the anterior and posterior descending arteries), in the ventricular apex (between anterior descending septal perforators), in the anterior right ventricular free wall (between anterior descending and right or conus arteries), in the anterolateral left ventricular free wall (between anterior descending diagonals and circumflex marginals), at the cardiac crux, and along the atrial surfaces (between the right and left circumflex arteries).\textsuperscript{17}
The intramural coronary vessels form the microcirculation. There are age-related variations in the pattern of distribution of the coronary microcirculation. Angina-like chest pain in some patients with angiographically normal epicardial coronary arteries (ie, syndrome X, or microvascular angina) can be secondary to abnormal vasodilator reserve or vasoconstriction of the coronary microcirculation. Abnormal flow reserve of the coronary microcirculation is seen in both dilated and hypertrophied hearts. In the latter, structural changes in the coronary arterioles can be found on histologic examination of the myocardium. In patients with symptomatic hypertrophic cardiomyopathy without angiographic evidence of epicardial coronary artery disease, myocardial tissue obtained during surgical myectomy can show smaller than normal coronary arteriolar lumina. Postmortem analysis of hearts with hypertrophic cardiomyopathy also has revealed coronary arterioles with abnormally thick walls. With contrast echocardiography, it can be possible to noninvasively visualize intramyocardial arterioles and study coronary flow reserve. Demonstration of an intact microvascular circulation in akinetic myocardium following acute myocardial infarction, using PET or SPECT imaging or contrast echocardiography, is evidence of viability of the affected segment. The creation of intramyocardial channels with CO₂ laser transmyocardial revascularization has been associated with augmentation of collateral flow to ischemic myocardium through angiogenesis.

CARDIAC LYMPHATICS

The myocardial lymphatics drain toward the epicardial surface, where they merge to form the right and left lymphatic channels, which travel in retrograde fashion with their respective coronary arteries. These two lymphatic channels travel along the ascending aorta and merge before draining into a pretachael lymph node beneath the aortic arch. This single lymphatic channel then travels through a cardiac lymph node, between the superior vena cava and innominate artery, and finally empties into the right lymphatic duct. Obstruction of epicardial lymphatics by a metastatic process can produce a pericardial effusion.

GREAT VESSELS

The subclavian and internal jugular veins merge bilaterally to form the right and left innominate veins (Fig. 4–69). Valves in the subclavian and internal jugular veins, near their junctions with the innominate veins, are important anatomic structures that help maintain unidirectional antegrade blood flow not only in the normal state but also in the setting of elevated right-sided heart filling pressures. Subclavian and internal jugular venous valves are absent in 2% and 6% of individuals, respectively, and venous valves can be damaged by catheter-induced trauma or age. Absent or malfunctioning valves can interfere with the success of closed-chest cardiopulmonary resuscitation and contribute to the development of brain edema during such a procedure.

The left innominate vein is two to three times the length of its right-sided counterpart. It travels anteriorly to the aortic arch along the right anterolateral border of the ascending aorta, where it joins the right innominate vein to form the superior vena cava (SVC). The left innominate vein is two to three times the length of its right-sided counterpart. It travels anteriorly to the aortic arch along the right anterolateral border of the ascending aorta, where it joins the right innominate vein to form the superior vena cava (SVC). The superior vena cava lies anterior to the right pulmonary artery (Fig. 4–70) and receives theazygos vein posteriorly before draining into the superior aspect of the right atrium, just posterior to the atrial appendage (see Figs. 4–49, 4–52, and 4–70). The superior vena cava has important spatial relations with the right upper pulmonary vein (see Fig. 4–58) and the aortocaval ganglion, each an important target site when ablating atrial fibrillation. The vein of Marshall forms the terminal connection between a persistent left superior vena cava and the coronary sinus. Its vestigial remnant in normal adults is the ligament of Marshall (Fig. 4–71). Both vein and ligament are a potential source of arrhythmias. The ostium of the inferior vena cava is guarded by a crescent-shaped, often fenestrated flap of tissue, the eustachian valve (see Figs. 4–16A), which is readily seen by echocardiography. Although generally small, the eustachian valve can become so large that it can produce a double-chambered right atrium.

FIGURE 4–69. The longer left (LIV) and shorter right (RIV) innominate veins normally join to form the right superior vena cava (SVC). Ao, ascending aorta; PT, pulmonary trunk.

FIGURE 4–70. Long-axis view of the superior vena cava (SVC) and inferior vena cava (IVC). The specimen is viewed from the left looking toward the free wall of the right atrium. The right atrium (RA) and its appendage (RAA) are anterior. This is a commonly used tomographic plane in transesophageal echocardiography (TEE). AS, atrial septum; LA, left atrium; LB, left bronchus; RPA, right pulmonary artery.
ectasia is associated with hypertension, aortic medial degeneration, and advanced age and can produce aortic regurgitation, ascending aortic aneurysm, or aortic dissection.\textsuperscript{14,16,17} Sleeves of left ventricular myocardium can extend above the aortic valve and to variable lengths into the aorta. A variety of arrhythmias can be ablated in the aorta and aortic valve cusps.\footnote{The superior vena cava and proximal ascending aorta are closely related (see Fig. 4–20). As a result of this proximity, supra-aortic valvular arrhythmias may be mapped or ablated from the superior vena cava.\textsuperscript{14,16,17}}

Sleeves of left ventricular myocardium can extend above the aortic valve and to variable lengths into the aorta. A variety of arrhythmias can be ablated in the aorta and aortic valve cusps.\footnote{By echocardiography, a Chiari net can be misinterpreted as a mass lesion. The thoracic aorta arises at the level of the aortic valve and is divided into three segments: ascending aorta, aortic arch, and descending thoracic aorta (Fig. 4–73). The ascending aorta consists of sinus and tubular portions, which are demarcated by the sinotubular junction (Fig. 4–74; see also Fig. 4–29). This is the site at which supravalvular aortic stenosis is often most severe.\textsuperscript{14,16,17} The entire thoracic aorta can be readily imaged by CT and MRI (Fig. 4–75).}

Behind the aortic valve cusps are three outpouchings, or sinuses (of Valsalva). The right aortic sinus abuts against the ventricular septum and right ventricular parietal band and is covered in part by the right atrial appendage (see Figs. 4–31 and 4–58). In contrast, the left aortic sinus rests against the anterior left ventricular free wall and a portion of the anterior mitral leaflet, abuts the left atrial free wall, and is covered in part by the pulmonary trunk and left atrial appendage (see Figs. 4–20 and 4–21A). The posterior (noncoronary) aortic sinus overlies the ventricular septum and a part of the anterior mitral leaflet, forms part of the transverse sinus, abuts the atrial septum, and indents both atrial free walls\textsuperscript{14,16,17} (see Figs. 4–12D and 4–22). Rupture of the right and posterior aortic sinuses of Valsalva can result in a communication with the right ventricular outflow tract or right atrium, whereas rupture of the left aortic sinus of Valsalva leads to a communication with the left atrium or left ventricular outflow tract. Anulooaortic ectasia is associated with hypertension, aortic medial degeneration, and advanced age and can produce aortic regurgitation, ascending aortic aneurysm, or aortic dissection.\textsuperscript{14,16,17} The superior vena cava and proximal ascending aorta are closely related (see Fig. 4–20). As a result of this proximity, supra-aortic valvular arrhythmias may be mapped or ablated from the superior vena cava.\textsuperscript{14,16,17}

Most coarctations occur just distal to the left subclavian artery. When thoracic aortic dissection does not involve the ascending aorta (DeBakey type III and Stanford type B), the intimal tear is commonly near the ligamentum arteriosum or eustachian or the adjacent thebesian valve of the coronary sinus is large and fenestrated, it is referred to as a Chiari net (Fig. 4–72).\textsuperscript{14,16,17} By echocardiography, a Chiari net can be misinterpreted as a mass lesion. The thoracic aorta arises at the level of the aortic valve and is divided into three segments: ascending aorta, aortic arch, and descending thoracic aorta (Fig. 4–73). The ascending aorta consists of sinus and tubular portions, which are demarcated by the sinotubular junction (Fig. 4–74; see also Fig. 4–29). This is the site at which supravalvular aortic stenosis is often most severe.\textsuperscript{14,16,17} The entire thoracic aorta can be readily imaged by CT and MRI (Fig. 4–75).
the ostium of the left subclavian artery. Nonpenetrating deceleration-related chest trauma, as can occur in motor vehicle accidents, commonly involves the aorta in the region between the aortic arch and descending thoracic aorta and can be associated with aortic transection or pseudoaneurysm formation.

The descending thoracic aorta lies adjacent to the left atrium, esophagus, and vertebral column. The pulmonary trunk (or main pulmonary artery) emanates from the right ventricle and travels to the left of the ascending aorta. As it bifurcates, the left pulmonary artery courses over the left bronchus, whereas the right pulmonary artery travels beneath the aortic arch and behind the superior vena cava (see Figs. 4–11A and 4–70). Thus the left bronchus and the right pulmonary artery normally travel beneath a left-sided aortic arch. In cases of right-sided aortic arch, commonly seen with conotruncal malformations (eg, tetralogy of Fallot), the arch will travel over the right bronchus and the right pulmonary artery. Thus, the laterality of the aortic arch is determined by the bronchus over which it travels.

### CARDIAC CONDUCTION SYSTEM

The cardiac conduction system consists of the sinus node, internodal tracts, AV node, AV (His) bundle, and right and left bundle branches (Fig. 4–76). The sinus node is located subepicardially in the terminal groove, close to the junction between the superior vena cava and right atrium. The sinus node artery arises from the right coronary in 55% of persons. Its course can place it in contact with the base of the right atrial appendage and the superior vena cava–right
atrial junction (see Fig. 4–58). When the sinus node artery arises from the left circumflex artery (45%), it can course close to the left atrial appendage. During such surgical operations as the Mustard and Fontan procedures, the sinus node and its artery are susceptible to injury.16,17 By light microscopy, there are no morphologically distinct conduction pathways between the sinus and AV nodes.17 However, electrophysiologic studies support the concept of functional preferential pathways that travel along the crista terminalis and atrial septum including the limbus but not the valve of the fossa ovalis.17 Internodal conduction disturbances therefore are not expected as a result of transseptal procedures. With the Mustard operation for complete transposition of the great arteries, there can be severe disturbance of internodal conduction because the entire septum is resected, and the surgical atriotomy can disrupt the crista terminalis.17 Lipomatous hypertrophy of the membranous septum can interfere with internodal conduction and induce a variety of atrial arrhythmias. Ventricular preexcitation is most commonly associated with aberrant bypass tracts that span the annulus of the tricuspid or mitral valve (see Fig. 4–2).

The AV node, in contrast to the sinus node, is a subendocardial structure that is located within the triangle of Koch (Fig. 4–77). The triangle of Koch is bordered by the coronary sinus ostium posteroinferiorly and the septal tricuspid annulus anteriorly. Because of its right atrial location near the tricuspid annulus, the AV node is susceptible to injury during tricuspid anuloplasty and during plication procedures for Ebstein anomaly.14,15,17

The AV (His) bundle arises from the distal portion of the AV node and travels along the ventricular septum adjacent to the membranous septum14,15,17 (see Fig. 4–77). The AV node tissue is generally remote from the defect in the outlet, inlet, and muscular forms of ventricular septal defect but travels along the inferior margin of a membranous ventricular septal defect. The AV bundle travels through the central fibrous body (right fibrous trigone) and therefore is closely related to the annuli of the aortic, mitral, and tricuspid valves. Thus, during operative procedures involving these valves or a membranous ventricular septal defect, care must be taken to avoid injury to the His bundle. Whereas in normal hearts, the AV bundle courses along the posteroinferior rim of the membranous septum, it courses along the anterosuperior rim of the membranous septum in hearts with AV discordance. The AV bundle receives a dual blood supply from the AV nodal artery and the first septal perforator of the left anterior descending coronary artery.17

The right bundle branch emanates from the distal portion of the AV bundle and forms a cord-like structure that travels along the septal and moderator bands toward the anterior tricuspid papillary muscle (see Fig. 4–76). In contrast, the left bundle branch represents a broad fenestrated sheet of subendocardial conduction fibers that spread along the septal surface of the left ventricle14,15,17 (see Fig. 4–76). The right and left bundle branches receive dual blood supply from the septal perforators of the LAD and posterior descending coronary artery.17 Left ventricular pseudotendons can contain conduction tissue from the left bundle branch.17 The left bundle branch can be disrupted following surgical myectomy, whereas the right bundle branch can be damaged during percutaneous alcohol septal ablation.18 Following right ventriculotomy for reconstruction of the right ventricular outflow tract, the electrocardiogram shows a pattern of right bundle-branch block even though the right bundle is not disrupted.16

**NEW DEVELOPMENTS AND FUTURE CHALLENGES**

The future holds promise for an integrated multidimensional approach to the study of cardiac anatomy that incorporates static 3D data, the elements of time (the fourth dimension) and motion, and physiologic (pressure and perfusion) and metabolic parameters.49,50 Until recently, the geometric fusion of anatomy and function was not possible without physically invading the body. With the currently available imaging techniques, multidimensional anatomy and physiology are mentally reassembled from the sequential tomographic images using echocardiography, MRI or CT, or multiple scintigraphy, as well as SPECT imaging.49 With the advances in medical technology propelled by the rapid developments in computer technology, digital imaging, and data-storage techniques, it has become possible to electronically perform virtual dissection and reconstruction of the heart and cardiovascular system.49,50 Furthermore, multidimensional imaging allows continued study of any human organ of interest because of the ability to permanently store anatomic images and the contained physiologic features for retrieval, comparison for change, and ultimately, replication in a more familiar 3D and four-dimensional (4D) presentation.49,50

The potential realization of virtual anatomy notwithstanding, standardization of the various tomographic approaches to image acquisition in a manner that conforms to familiar anatomic presentation remains a major challenge that has to be overcome if multidimensional cardiac imaging is to become a clinical reality. There is current progress in this direction. Real-time 3D reconstruction of the heart using identical CT and 2D tomographic sectioning of the heart is now possible. Virtual human vivisection might soon become reality. The goal is virtual surgery (dry runs prior to the actual operation) and dissection of the heart into its various components, be it anatomic, functional, or metabolic, either separately or in various combinations. Moreover, 3D printing11,12 allows for imaging data to be translated to the physical realm whereby specimens can be studied in detail to assist in planning for intervention or education. Because of advances in multimedia technology, the centuries-old great divide between physiologists and anatomists is about to become relegated to the history books.

**FIGURE 4–77.** The atrioventricular node (AVN) lies within the triangle of Koch (dashed triangle), and the AV (His) bundle (AVB) travels through the tricuspid annulus to rest along the summit of the ventricular septum. CS indicates coronary sinus; FO, fossa ovalis; IVC, inferior vena cava; S, septal leaflet of the tricuspid valve; SVC, superior vena cava.
ANATOMY NOT ADDRESSED AND QUANTUM COMPUTING

Fine-detailed anatomy such as that of the conduction system and microvasculature is not readily available to the usual anatomic dissection by computer-based imaging. Additionally, tissue histology or molecular biological assessment is not obtained routinely by imaging. At the other end of the spectrum, 3D gross anatomical dissection of contiguous structures is also normally not available (eg, how does metastatic cancer throughout the system relate to a primary tumor in the gut?).

These and other macroscopic and microscopic dissections await the future of increasingly sophisticated computer technology and information management. Both pathologic and living tissues someday will be dissected and analyzed not by destructive cutting but by higher dimensional imagery. Today’s computers have introduced the information era. Information has become a commodity expanding our ability to access useful data. It is possible that we will soon evolve into the “Quantum Era,” where all that has been discussed in this chapter plus gross and microscopic anatomy will be possible within an electronic environment. Reality will be expressed as base parts or characteristics (eg, quanta, molecules, pixels) and reformatted in 3D or 4D geometry relative to the desired information. Gross anatomy, physiology, tissue characteristics, and even histopathology will be dissected and presented as a quantifiable geometric image. The concept of a living autopsy may become a reality.

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