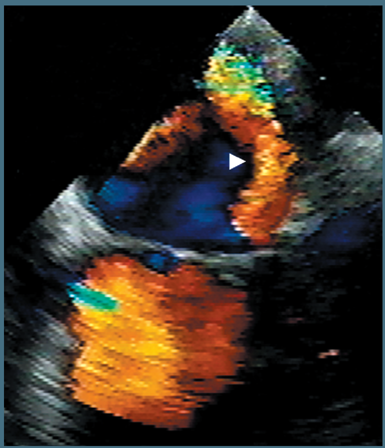
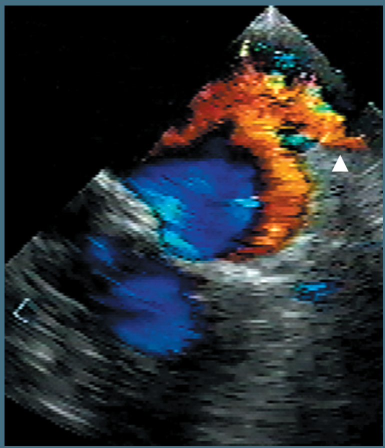
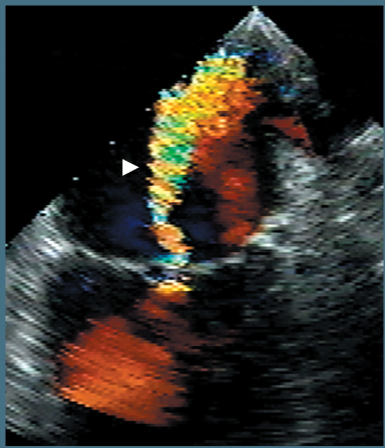

Mitral Valve Transesophageal Echocardiography

Martin G St John Sutton • Alan R Maniet

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Mitral Valve Transesophageal Echocardiography

MARTIN G ST JOHN SUTTON, MB, FRCP, FACC, FASE
John W Bryfogle Professor of Cardiac Imaging
University of Pennsylvania Medical Centre
Philadelphia, PA
USA

ALAN R MANIET, DO, FAAIM
St Louis University Medical Center
St Louis, MO
USA

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Preface

Over the past few years transesophageal echocardiography has become one of the most exciting imaging modalities available today in modern clinical cardiology. Transesophageal echocardiography and MRI imaging has substantially improved our understanding of mitral valve anatomy and physiology with multiplane transesophageal echocardiography having a true advantage through its portability, relatively inexpensive equipment and seminoninvasive nature. Multiplane transesophageal echocardiography provides high resolution cardiac images in an infinite number of planes. When combined with conventional and color Doppler modalities, this offers a superlative diagnostic tool for evaluating cardiac structure and function. Multiplane transesophageal echocardiography provides a three-dimensional perspective, especially of the mitral valve, that cannot be appreciated even by the cardiac pathologist. This point is illustrated by multiple carefully prepared anatomic sections, matched with diagrams and multiplane transesophageal images. After finishing our original text, the *Atlas of Multiplane Transesophageal Echocardiography*, we were approached by many physicians to provide a smaller concise reference just on the mitral valve, as the importance of mitral regurgitation has blossomed in modern cardiology. The aim of this atlas is to provide medical students, anesthesiologists, cardiac surgeons and cardiologists with an in-depth analysis of the mitral valve from an experience of over 15,000 transesophageal echocardiograms performed by the authors, with over 15 years in the operative arena studying mitral repair techniques. This atlas may also serve as a reference for diagnostic examples of mitral pathology for physicians who practice transesophageal echocardiography. The text is not meant to be a complete or authoritative reference for the mitral valve, but to serve as a starting point or "how-to" reference for studying the mitral valve structure and function. With the new emphasis on the possibility of replacing or repairing mitral valves when patients are asymptomatic, it is extremely important for echocardiographers to possess a clear understanding of mitral valve abnormalities in order to address surgical decision-making properly with our surgical colleagues. The format begins with normal mitral valve structure and function, followed by abnormalities of the mitral valve unit. Chapters include evaluation of prostheses, interventional cardiology techniques, and intraoperative transesophageal echocardiography, especially how it relates to mitral valve repair. A concise explanation of measurements of cardiac chamber sizes and function and Doppler are provided only for transesophageal echocardiographic applications. As in the original atlas, transesophageal echocardiographic images are juxtaposed with correlative anatomic specimens to provide a graphical understanding of normal and abnormal mitral valve anatomy.

Martin G St John Sutton
Alan R Maniet

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1

Introduction

Mitral valve repair • Mechanical valves • Biological valves • Further developments in mitral valve repair • The impact of echocardiography

The mitral valve, with its unique and delicate structure, has intrigued physicians and scientists for centuries. Indeed, the quest to understand the function of the mitral valve is a thread that runs through the entire history of modern cardiology.

Some of the earliest descriptions of the mitral valve were made in the 16th century by Leonardo da Vinci. His drawings and notes, based on dissected bovine and human hearts, highlighted the complex architecture of the valve and alluded to the unknown function of the individual elements of the subvalvular apparatus.[1–3] The mitral valve was named for its resemblance to a bishop's mitre, as depicted in da Vinci's drawings.

In 1543 the first complete textbook of human anatomy by Andreas Vesalius released the study of human anatomy from the restrictions of Galenic doctrine. [4] Vesalius described the heart as the center of the vascular network, and proposed that the pulmonary veins carried air from the lungs to the left atrium. Vesalius' and da Vinci's historic theories opened the doors to further study and discovery.

In the 19th century Prussian physiologists attempted to explore the form and function of the mitral valve apparatus *in vitro* using primitive organ baths. Based on simple geometric principles, Woods was the first to propose a pivotal role for the papillary muscles and mitral subvalvular apparatus in left ventricular systolic function. [5]

MITRAL VALVE REPAIR

Attention turned to the clinical recognition of mitral valve disease in the late 1800s, when Samways[6] and Brunton[7] proposed that mitral stenosis could be treated by performing a transventricular valvotomy. This was of particular importance because, at the time, operating on the heart was in direct violation of common thought and practice. Samways hypothesized that "the severest cases of mitral stenosis will be relieved by slightly notching the mitral orifice and trusting to the auricle to continue its defence." Brunton had carried out preliminary experiments on diseased cadaveric mitral valves and healthy feline valves, and believed that mitral stenosis could be corrected despite the risks of surgery. He suggested "elongating the natural opening, cutting through the thickened edge of the commissures along their natural openings or incising the leaflets in the middle through a ventricular approach", considering that the thicker wall of the left ventricle would be less prone to bleeding than the thinner left atrium. With the exception of the ventriculotomy, this description closely resembles modern-day surgical techniques. Nevertheless, Brunton was severely criticized by his peers for even proposing cardiac surgery.[8,9]

Mitral valve surgery lay dormant until the 1920s when the first valvotomies were performed by Cutler and Levine at the Peter Bent Brigham Hospital in Boston, [10] and by Souttar at The London Hospital. [11] Cutler attempted surgery from the auricular approach, since the difficulty with the transventricular approach "lay in locating the mitral valve so that the valve could be engaged between the cutting edges of the instrument." Despite choosing the safer atrial route, Cutler soon discovered that those patients who had parts of their mitral valve accidentally excised by the cardiovalvulotome had almost a 100% mortality; only one patient, whose mitral valve was left intact, survived. Henry Souttar circumvented this problem by performing the first finger dilation on mitral valve stenosis through a transatrial approach. He stated that, "the information given by the finger is exceedingly clear, and personally I felt an appreciation of the mechanical reality of stenosis and regurgitation which I never felt before." Souttar's technique of finger dilation of mitral stenosis ultimately led to more successful surgeries and fewer mortalities in a time before direct visualization was possible. However, the number of failed cases in the 1920s was high enough to discourage mitral valvotomies despite many new proponents for heart surgery.

Mitral valvotomy for mitral stenosis was redefined during the late 1940s and early 1950s by Bailey[12,13] and Harken[14–16] in the USA, and by Brock[17] and Tubbs in the UK. Harken (who continued the work of Cutler) and Bailey had accumulated a wealth of surgical experience prior to performing their first valvotomies. Harken's experience had come during World War II repairing hearts injured during battle, while Bailey's was gained through laboratory surgeries on canine mitral valves. Both men believed that "palpation of the valve [was] critical to ensuring an equilibrium between stenosis and regurgitation and [that] selecting patients with stenosis and no complications was most important". In 1948 Bailey and Harken performed their first transatrial commissurotomies, on June 10th and June 16th respectively. Bailey had to endure four surgical mortalities before his first success at Philadelphia's Episcopal Hospital, where he used a hooked knife guided by his index finger to perform the procedure. By 1956, Bailey had performed over 1000 commissurotomies with an operative mortality of 7.9%. Harken performed over 1000 valvulotomies over a 20 year period, including a large percentage in patients under the age of 40 with mostly noncalcified valves. Although these patients had improved long-term survival rates, he was quick to point out that the results were palliative rather than curative.

Enthusiasm for mitral valve surgery continued to grow in the 1950s. The development of the heart–lung machine and mechanical oxygenation allowed Gibbon[18] to perform the first successful open-heart operation with direct visualization of the heart in a bloodless field at the Thomas Jefferson University Hospital. Bigelow[19] used hypothermia to reduce oxygen usage during heart surgery, an approach that Lewis[20] used in conjunction with caval occlusion. These technical advances played a crucial role in providing surgeons with an opportunity to look inside the heart and directly view the damaged mitral valve, thereby enabling more extensive mitral valve surgeries.

Meanwhile, other physicians were investigating mitral insufficiency. Griffith[21] and Hall[22] in 1903 proposed that an apical late systolic murmur might denote mitral regurgitation. In 1937, White[23] suggested that mid-systolic sounds might sometimes arise from abnormal chordae tendineae. This theory was revived by Reid[24] in 1961, who postulated that clicks and late systolic murmurs were of mitral valvular origin. The clicks arose from the chordae, and Reid used the terms "chordal snap" and "nonejection systolic click" in order to differentiate them from aortic and pulmonary ejection sounds. The physiologists Rushmer, a bioengineer at the University of Washington, and Tsakiris, working at the Mayo Clinic, further defined chordae tendineae. In 1956, Lillehei[25,26] in Minnesota attempted to repair leaky valves under direct vision, and McGoon[27] at the Mayo Clinic introduced "repair of mitral regurgitation for ruptured chordae tendineae by performing a triangular resection of the prolapsing leaflet segment."

MECHANICAL VALVES

Although initial surgical repairs of regurgitant and stenotic mitral valves were now being commonly performed, the long term success rate was unsatisfactory. The search for alternative, more successful treatment strategies led to the development of prosthetic valves.

The first steps towards valvular replacement came with the selective mechanical replacement of specific parts of the mitral valve. These included Gott's replacement of the posterior mitral leaflet, and King's replacement of the chordae tendineae.[24] While these procedures looked promising theoretically the results were disappointing; either the replacement parts failed to achieve full function or the rigidity of the prosthetic parts worsened mitral valve function by causing stenosis. The first mitral valve replacement was performed by Chesterman[28] in England on July 22nd 1955.

The design of the valve used by Chesterman closely resembled that of the first successful mitral valve replacement – the Starr-Edwards ball-and-cage valve. The original idea for the Starr-Edwards valve came from a bottle stopper patented in 1858. The design was modified and refined based on data from canine experiments. Starr used strict selection criteria to maximize the success rate of the procedure, focusing on patients whose mitral valves had been damaged beyond repair and who needed surgery quickly. The Starr-Edwards valve led to the first ever published report of survival beyond three months for patients with total valvular replacement. Following the success of the Starr-Edwards valve, mitral valve mechanical replacement flourished in the 1960s under Starr, Braunwald and Harken, even though initial mitral valve replacement mortality rates were 30% and higher.[29–33] New prosthetic mitral valve designs proliferated, using a range of synthetic materials (from Teflon to Mylar and Dacron) and shapes (depending on the designer's understanding of how normal physiology could be maintained).

BIOLOGICAL VALVES

The high rate of thrombosis experienced with the mechanical valves available at the time led to the idea of using biological valves (bioprostheses). Homografts (transplants from human cadavers) were introduced by Ross and Barratt-Boyes in the 1960s.[34,35] Binet and associates developed tissue valves from formaldehyde-fixed xenografts.[36,37] The initial results were promising, but within a few years these valves began to fail because of degeneration and calcification of the tissues. Carpentier and associates studied the valves of various animal species and found that porcine valves closely resembled human valves.[38] In 1966 Carpentier began working with porcine valves fixed in glutaraldehyde rather than formaldehyde and mounting them on a stent, allowing the valve to be placed in the mitral position. He found that although the use of gluteraldehyde almost eliminated the inflammatory reaction, the implants had to be replaced within five years due to calcification and cuspal tearing. Further research built on this early work and led to the development of the Carpentier-Edwards, Hancock and Angell-Shiley bioprostheses.

Mitral valve replacement, whether mechanical or biologic, was associated with an unacceptably high surgical mortality, due to the removal of the leaflets and subvalvular apparatus. In 1972, Kirklin remarked that "we should expect to see a lower LV ejection fraction after mitral valve replacement for mitral regurgitation due to elimination of the 'systolic pop-off' mechanism, resulting in higher left ventricular systolic wall stress (or afterload)".[39] In the 1980s, David, Hagl and surgeons at Stanford found that preserving the mitral subvalvular apparatus improved clinical outcome, preserved LV shape and systolic function and reduced mortality.[40]

FURTHER DEVELOPMENTS IN MITRAL VALVE REPAIR

Mitral valve repair continued into the late 1950s, but it was technically challenging and infrequently performed, and most surgeons chose to use prosthetic valves. However, Wooler, Reed and Kaye refined the techniques for mitral annuloplasty, allowing some surgeons to continue repairing stenotic and regurgitant valves. [41–44]

At about this time the epidemiology of mitral valve pathologies began to change. Rheumatic mitral valve disease declined, due largely to the introduction of penicillin and a general improvement in socioeconomic conditions.[45,46] Degenerative diseases of the mitral valve thus became more prominent, with mitral valve prolapse taking center stage as the most important cause of mitral regurgitation.

In 1963 Barlow and associates[47–50] identified late systolic murmurs as an indicator of billowing mitral leaflets or mitral valve prolapse allowing regurgitation of the mitral valve (a condition now known as Barlow's syndrome). During the 1970s the cardiology literature experienced an "information explosion" relating to mitral valve prolapse syndrome.[51] The frequency of mitral valve prolapse and regurgitation as defined with echocardiographic reports ranged between 4% and 21% of the normal population. As far back as 1953, McKenzie and Lewis had stated that systolic murmurs were harmless in the absence of the evidence of myocardial disease. Nevertheless, it soon became clear that a differentiation would have to be made between patients with severe mitral regurgitation, and patients with trivial or mitral valve reflux, as detected by echocardiographic and Doppler techniques. Over the next decade the etiology, diagnosis, prognosis and management of patients with mitral valve prolapse and regurgitation was defined. In the early 1980s, Carpentier redefined the essential and important differences between "prolapse" and billowing by careful analysis of mitral valve pathology during mitral valve repair.

During the 1980s mitral repair continued to flourish under the direction of Carpentier[52–54] and Duran,[55–57] both of whom introduced several reparative techniques using annuloplasty rings determined by the functional classification of the specific mitral valve lesion. Using these techniques mitral valve repairs were performed with: lower operative and late mortality rates; improved hemodynamics; fewer thromboembolic complications with a reduced risk of anticoagulant-related hemorrhage; a reduced risk of infective endocarditis. These factors combined to make mitral valve repair especially appealing to developing countries.

Although mitral valve reparative techniques were initially largely used for cases of mitral regurgitation, they were also employed in the treatment of mitral stenosis. With the expansion of the field of interventional techniques in the cardiac catheterization laboratory, Inoue[58] and Lock[59] pioneered balloon catheters to dilate stenotic mitral valves in 1984, avoiding the risks associated with thoracotomy. Long-term studies at three and five years showed that balloon valvuloplasty and open-heart surgical commissurotomy had similar survival and restenosis rates.[60,61]

THE IMPACT OF ECHOCARDIOGRAPHY

Over the last decades, echocardiography has played an important role in understanding mitral valvular disease. In the early days of M-Mode echocardiography, the mitral valve was frequently the only discernible cardiac structure, allowing mitral stenosis to be diagnosed noninvasively.[62] With the development of two-dimensional echocardiography a spatial evaluation of the mitral valve could be obtained, and the importance of the mitral subvalvular apparatus, the valve leaflets and their motion was recognized for the first time, eliminating many of the misconceptions about the mitral valve[63–68]. The mitral valve prolapse syndrome was highlighted by echocardiography. Doppler echocardiography in conjunction with two-dimensional imaging has provided a method of quantifying the degree of stenosis and regurgitation in mitral valvular disease.

In surgical repair of mitral valvular disease,[69] transesophageal echocardiography gives the precise assessment of mitral valvular structural abnormalities that is necessary to direct appropriate treatment.

Prior to the mid-1990s, the decision to intervene in mitral valvular disease was based on the presence of severe stenosis or regurgitation, and was followed by replacement of the valve with a prosthesis. This strategy frequently resulted in an imperfect functional result, substituting one disease process for another, and led to disconcertingly high morbidity and mortality.[70] A better appreciation of mitral valvular disease has evolved, largely due to the improvement in imaging techniques. The ability to define specific structural abnormalities of the valve leaflets and subvalvular apparatus has allowed the development of reparative surgical techniques that overcome the shortcomings prosthetic valves. The success of managing and surgical techniques have improved the longevity of patients with mitral valve disease.[70–72]

It is essential that the physician performing transesophageal echocardiographic studies has a complete and thorough understanding of normal mitral valvular anatomy, in order to recognize abnormalities. The physician must be able to evaluate the motion of the valve leaflets as well as the subvalvular apparatus, to describe anatomical abnormalities due to the disease process.[73–75] The physician must be well versed in the principles and practice of Doppler methodology in order to identify abnormalities. Finally the echocardiographer must possess the technical ability to obtain all of the views necessary to evaluate the whole structure and function of the mitral valve, which are uniquely acquired with multiplane transesophageal echocardiography, in order to provide a complete assessment.

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2

Normal Anatomy, Normal TEE, Normal Doppler

Normal mitral valve anatomy • Normal multiple transesophageal echocardiographic valve analysis • Multiplane Doppler examination of the normal mitral valve

NORMAL MITRAL VALVE ANATOMY

Leaflets

The mitral valve sits in the fibrous skeleton of the heart separating the left atrium from the left ventricle (Figure 2.1). The normal mitral valve has two leaflets that act in conjunction with the subvalvular apparatus as one functional unit.[1–6] The mitral leaflets are attached at their bases to the fibro-muscular ring or annulus fibrosis, and by their free edges to the subvalvular apparatus consisting of chordae tendineae and papillary muscles.

The two leaflets, anterior and posterior, differ in size and shape but share equal leaflet surface area (Figures 2.1 and 2.2). The anterior leaflet, also known as the septal or aortic leaflet, is triangular in shape, with a convex free edge. The width (base to free edge measurement) of the anterior leaflet is about one third larger than the posterior leaflet. The posterior leaflet, also known as the mural or inferolateral leaflet, is narrower, attaches to a larger portion of the annulus than the anterior leaflet, and has a concave free edge. Two natural indentations or clefts in the posterior leaflet produce three segments, which subdivide the posterior leaflet into lateral, central, and medial scallops. The lateral and medial scallops are smaller in surface area than the central scallop. The two leaflets are joined at their lateral free edges near the annulus just above the papillary muscles, to form two hinge points or commissures (Figures 2.1 and 2.3). When the mitral valve is closed during systole and viewed *en face*, the line of valve closure resembles a smile. When the mitral valve is open during diastole and viewed *en face*, it has an elliptical shape that gradually tapers like a funnel into the ventricle. When imaged sagittally the mitral valve resembles a trapdoor with the anterior to posterior leaflet length ratio being 2:1.

The leaflet tissue is thin and transparent near the annulus and the central portion (body) of the valve, gradually thickening near the free edge, becoming opaque and very thick at the opposing surface (rough zone). The rough zone is created by the points of attachment for each chorda, and permits a seal between the two leaflets when they approximate during closure. The leaflet edges fold over the chordal attachment (hooding), which somewhat protects the insertion point and provides increased surface area for approximation.

The posterior leaflet, with its narrow height, serves as a valance for the anterior leaflet to swing up and close against, providing a seal during closure. This leads to the concept [7] of

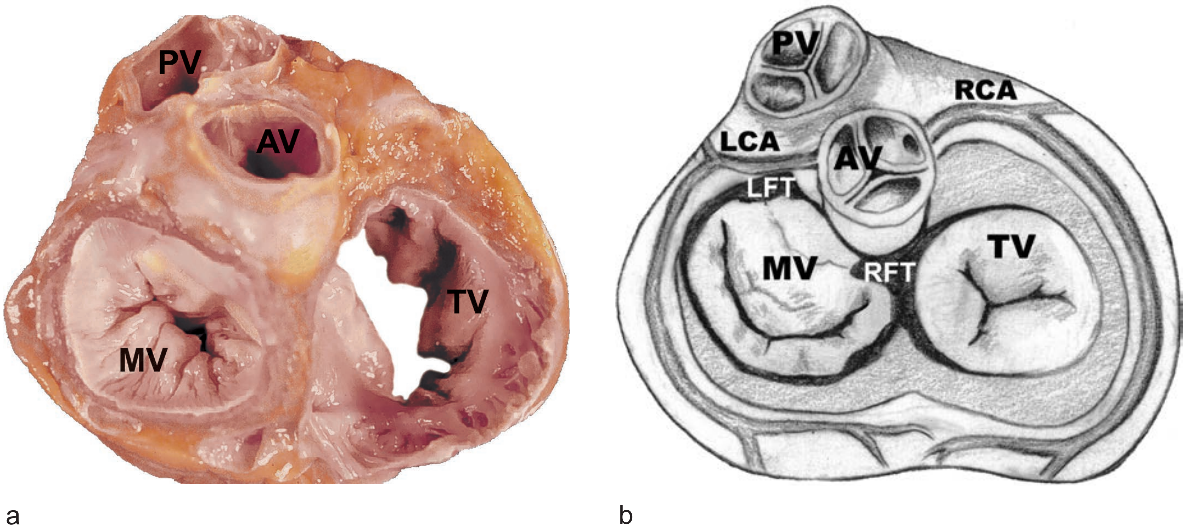
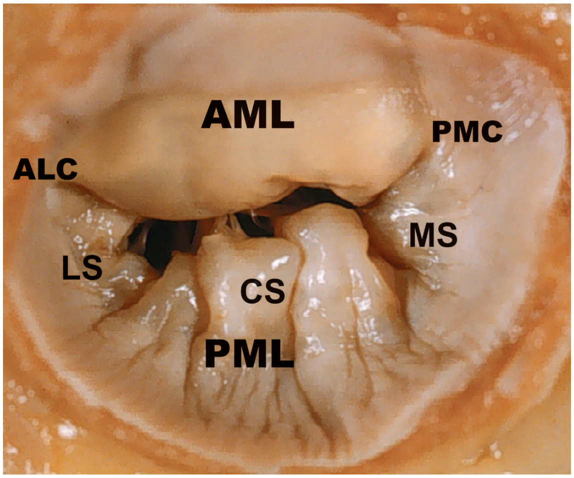


Fig. 2.1 Normal mitral valve anatomy. (a) Anatomical section at the base of the heart illustrating the position of the four cardiac valves. The mitral valve (MV) is seated posterior to the semilunar valves and is lateral and to the left of the tricuspid valve (TV). The anterior leaflet of the mitral valve is contiguous and opposite the left and non-coronary cusps of the aortic valve. The posterior medial commissure of the mitral valve lies opposite the septal leaflet of the tricuspid valve. The posterior position of the mitral valve allows optimal examination by multiplane transesophageal echocardiography. PV, pulmonary valve; AV, aortic valve. (b) Corresponding drawing of the fibrous skeleton of the heart. The anterior mitral leaflet is firmly attached to the aortic annulus by dense fibrous connective tissue forming the mitral curtain or mitral-aortic fibrous continuity. Two fibrous trigones situated near each commissure represent fixation points for the fibrous skeleton providing stability for cardiac contraction and the valve annulus. The right fibrous trigone (RFT) is centrally located between the aortic valve (AV), the mitral valve (MV) and the tricuspid valve (TV). The left fibrous trigone (LFT) extends superiorly and blends into the aortic root. Both trigones project connective tissue fibers laterally and posteriorly to encircle the mitral annulus. In the most posterior portion of the annulus the fibers progressively diminish leaving the central posterior annulus devoid of dense collagen fibers and only composed of loose connective tissue. This composition of the mitral annulus allows for annular dilatation to occur largely in the posterior aspect of the valve. The left coronary artery (LCA) courses superiorly and laterally around the mitral annulus and the circumflex artery wraps around the posterior aspect of the valve to the crux of the heart. RCA, right coronary artery; PV, pulmonary valve.

the anterior leaflet being the active “velocity leaflet”, largely responsible for the dynamics of opening and closing of the mitral orifice. The posterior leaflet is the “passive leaflet” and is the buttress that the anterior leaflet closes against to absorb the strain transmitted to the valve from the pressure generated in the ventricle during systole. The smaller height of the posterior leaflet also defines the orientation of the inflow tract, directing the flow of blood to the posterior aspect of the left ventricle.

Annulus

The two mitral leaflets are attached at their bases to a ring of fibromuscular tissue in the fibrous skeleton of the heart, called the annulus fibrosus (see Figure 2.1).[8,9] The fibrous trigones are the major components of the annulus fibrosus and sit adjacent to the commissures. The right fibrous trigone (central fibrous body) is the more prominent of the two and is centered between the tricuspid, mitral, and aortic valves. The left fibrous trigone is seated



c

Fig. 2.1 (contd.) (c) Anatomic specimen of the mitral valve leaflets. The posterior mitral leaflet (PML) is divided by prominent indentations in the leaflet margin into three scallops: the lateral scallop (LS); the central scallop (CS), usually the largest; and the medial scallop (MS). Note that the anterior mitral leaflet (AML) is free of these indentations or scallops. The anterior leaflet is rather triangular in shape. The posterior leaflet is more rectangular in shape and wraps around the free margin of the anterior leaflet. Although the width of the anterior leaflet is larger than the posterior leaflet, both leaflets are approximately equal in surface area. In addition, both leaflets are really continuous as demonstrated at the commissures, and do not extend entirely to the annulus. ALC, anterolateral commissure; PMC, posteromedial commissure; PV, pulmonary valve.

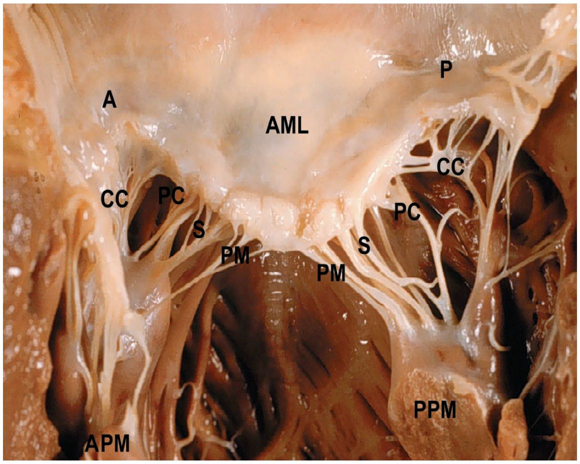
between the mitral and aortic valve towards the peripheral and most posterior portion of the fibrous skeleton. The two trigones are connected anteriorly with a thick band of collagenous connective tissue (the mitral-aortic fibrous continuity), which lies between the aortic and mitral valve and which forms the annulus for the entire anterior mitral leaflet. The anterior leaflet occupies only 40% of the total annular perimeter, with the entire basal portion of the anterior leaflet being attached to rigid collagen tissue opposite the aortic valve, which allows commissural attachments to be very close to the trigones. These characteristics allow rigid support of the anterior leaflet. The trigones project connective tissue laterally and posteriorly, surrounding the posterior leaflet to form the remainder of the annulus. The posterior annulus is thinner and weaker than the anterior annulus – as the connective tissue progresses posteriorly it gradually tapers to leave the posterior area around the central scallop with only loose connective tissue almost completely devoid of collagen fibers. This provides less annular support for the posterior leaflet, especially near the central scallop,[10,11] allowing the annulus to stretch in this area, under the stress of ventriculoatrial dilatation.

The dimensions of the annulus are characterized by a height (anterior-posterior dimension) to width (transverse dimension) ratio of 3:4 (Figure 2.1).[7]

Chordae tendineae

The chordae tendineae are tendinous supporting structures that originate from the tips of the papillary muscles and fan out by branching before inserting into the ventricular surface of the leaflet (Figure 2.5).[3] Chordal insertion is equally spaced (0.5 mm apart) on the margin of the leaflet edge, which promotes symmetrical motion. Normal chordae tendineae are arranged in an architectural alignment and spacing allowing each chorda to remain free from touching each other. Each chorda remains free from the other chordae and from the ventricular myocardium at all phases of the cardiac cycle. Occasionally, some chordae may originate directly from the ventricular myocardium and insert into the leaflets. Small false chordae may originate from the ventricular myocardium and attach to the papillary muscles, and are thought to act as supporting apparatus for the latter. Aberrant chordae – chordae that are attached from myocardium to myocardium – have unknown significance and probably serve no real purpose. Variations in the number of chordae and in the branching or insertion patterns account for the major aberrations of the chordae tendineae.

Chordal systems differ according to whether they are attached to the anterior or posterior leaflets (see Figure 2.2).[3,12–16] Chordae to the



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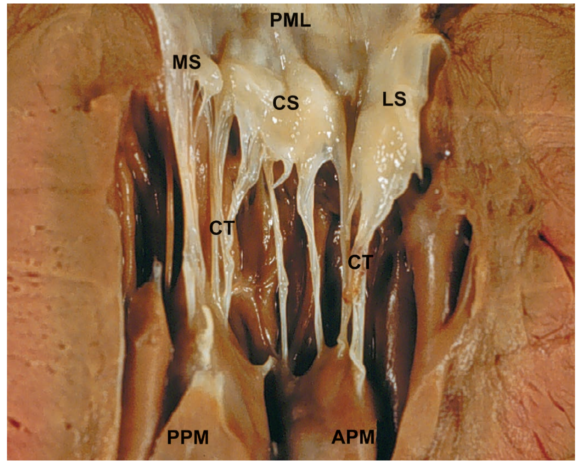

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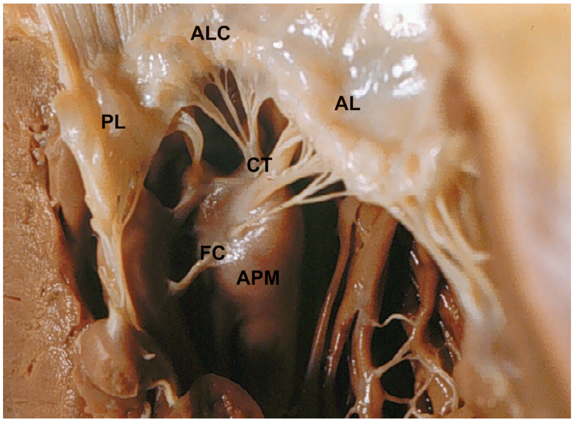
Fig. 2.2 Normal anterior and posterior leaflet anatomy. (a) The anterior mitral leaflet (AML) is triangular in shape with a broad base of attachment and a rounded apical contour. The body of the leaflet is thin and translucent in comparison to the leaflet margin. Chordal insertion to the ventricular surface of the free margin thickens the leaflet and produces the rough zone, which aids in providing for maximum leaflet coaptation. Both papillary muscles supply chordae tendineae in an oblique, symmetrical fashion to the anterior leaflet. The oblique orientation of the chordae allows for maximum and unrestricted movement of the anterior leaflet. Anterior leaflet chordae tendineae are labeled according to their point of attachment to the leaflet, and to some degree, to their shape. AML, Anterior mitral leaflet; A, anterior commissure; P, posterior commissure; CC, commissural chords; APM, anterior papillary muscle; PPM, posterior papillary muscle; PM, paramedical chordae; S strut chordae; PC, paracommissural chordae. (b) The posterior mitral leaflet (PML) is rectangular in shape. It has a larger circumference than the anterior leaflet and attaches to a larger portion of the annulus. The three scallops of the posterior leaflet are readily identifiable with the central scallop (CS) being the largest scallop. MS, medial scallop; LS, lateral scallop. Both papillary muscles (PPM and APM) supply evenly spaced, parallel chordae tendineae (CT) to the posterior leaflet. This parallel arrangement, a result of the shorter distance between the papillary muscles posteriorly in the ventricle, allows the chordae to act as supporting columns for the 'passive' or 'buttressing' posterior leaflet (see text for details). The arrangement of the chordae tendineae is such that each chord remains free of the others during all phases of the cardiac cycle.

posterior leaflet project and attach to the ventricular surface of the leaflet in a parallel manner. These chordae act as supporting columns to the posterior leaflet. Branching posterior leaflet chordae insert into the central scallop at the free edge, the body (mid surface), or the basal portion near the annulus. Chordae to the lateral and medial scallops insert in a more variable arrangement. Chordae to the anterior leaflet are projected in an oblique manner, which aids in opening and closing of the leaflet. Anterior leaflet chordae insert into the ventricular surface of the leaflet at the leaflet margin (primary chordae) or slightly behind the free edge (secondary chordae). Chordae to the anterior leaflet can be further subdivided into paramedial, central strut, or paracommissural chordae, depending on their position on the anterior leaflet's free edge.

The commissures have a unique chordal system that provides an anchor for the hinge point[3,13] (see Figure 2.3). Each papillary muscle gives rise to a single commissural chorda which fans out distally into about five small chordae inserting into the corner of both leaflets, at the free edges of the commissural tissue. Due to the slightly oblique position of the valve in the annulus fibrosus in relation to the papillary muscles, the commissural chordae to the posterior medial commissure are



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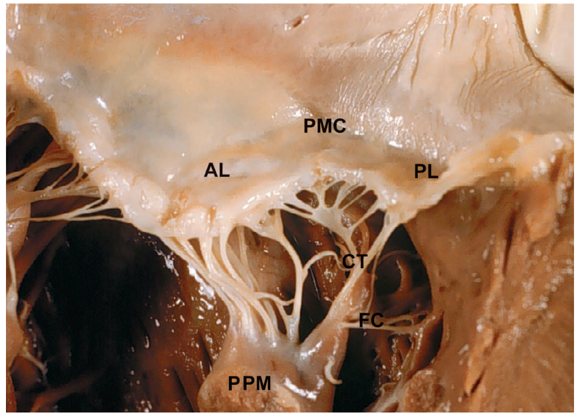

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Fig. 2.3 Normal anterior and posterior commissures. Each commissure receives one chorda tendinea (CT) from its respective papillary muscle (APM and PPM). Commissural chordae are broad at their origin, project perpendicularly and fan out to intersect the commissural portion of each leaflet. In this manner, adequate support is provided to the whole commissure serving as a hinge point for leaflet motion. (a) Anterolateral commissure (ALC). (b) Posteromedial commissure (PMC). Due to the oblique orientation of the mitral valve in the fibrous skeleton in relation to the position of the papillary muscles, the commissural chordae to the posteromedial commissure are usually thicker, longer, and more widely branching than the chordae to the anterolateral commissure. This may be the reason why the leaflet tissue surrounding the anterolateral commissure is less likely to prolapse (a feature that provides a good reference point at surgery for demonstrating prolapse in other areas). AL, anterior leaflet; FC, false chords; PL, posterior leaflet.

usually more widely spaced, thicker, and longer, than the chordae to the anterior lateral commissure. The proper identification of the commissures can therefore be aided by identifying the respective commissural chordal pattern.

Papillary muscles

The papillary muscles originate as muscular projections or columns from the ventricular myocardium, between the apical and middle portion of the ventricular chamber (Figure 2.6).[17,18] The exact positioning of both papillary muscles may vary considerably. The anterior papillary muscle usually arises from the lateral border of the anterior wall, and the posterior papillary muscle usually originates from the medial aspect of the posterior wall where it abuts the ventricular septum. There is usually only one anterior papillary muscle with often two or three posterior papillary muscles. The volume or mass of the anterior and posterior muscle groups are usually equal.

When a papillary muscle group is present the individual muscles wrap around each other in a concave-convex fashion, allowing the whole group to act as a single unit during contraction. The base and body of the papillary muscles are anchored to the ventricular free wall by muscular or tendinous chordae known as false chords, which are believed also to serve as channels for Purkinje fibers. The tips or heads of the papillary muscles taper into tendinous projections to give rise to the chordae tendineae. Each papillary muscle group gives off chordae tendineae in a symmetrical fashion to their respective halves of both the anterior and posterior leaflets (see Figure 2.5). It is well established that the papillary muscles play a vital part in valve integrity and valve motion.

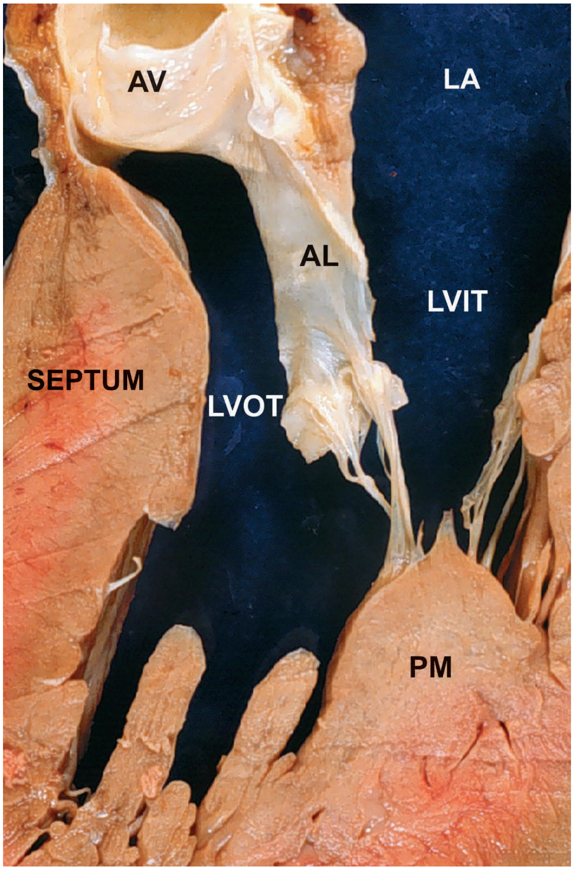


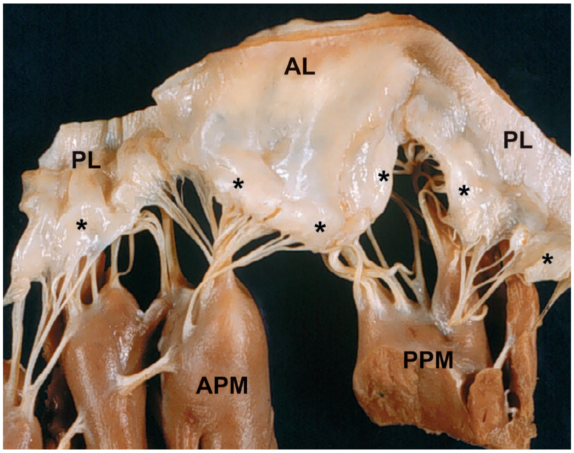
Fig. 2.4 Anatomical specimen demonstrating how the mitral valve produces functional left ventricular inflow and outflow tracts. Unlike the right ventricle, the left ventricle has no true anatomical inflow and outflow tracts. The inflow and outflow tracts are produced by the normal anatomical relationship of the mitral valvular apparatus in its posterior position and orientation in the left ventricle. LA, left atrium; LVIT, left ventricular inflow tract; LVOT, left ventricular outflow tract; PM, papillary muscle; AL, anterior mitral leaflet; AV, aortic valve; SEPTUM; interventricular septum.

The blood supply to the papillary muscles is highly variable.[19] The anterior papillary muscle is supplied by the second septal branch of the left anterior descending artery and by a branch of the circumflex artery. The posterior papillary muscle is usually supplied from septal branches of the posterior descending artery, in addition to a small branch of the circumflex artery. The confluence of coronary artery branches for each papillary muscle joins into a single central artery or remains as a meshwork of small branches. These vessels taper and project longitudinally into the muscle. This framework of blood supply probably allows the head of the muscle to be more prone to ischemia. When the blood supply is left-dominant, the posterior papillary muscle is especially prone to ischemia. In addition to blood supply, the papillary muscles are also perfused by the diffusion of oxygen from blood in the ventricular cavity.

NORMAL MULTIPLANE TRANSESOPHAGEAL ECHOCARDIOGRAPHIC VALVE ANALYSIS

Transesophageal echocardiography (TEE) has allowed many of the intricacies of the mitral valve apparatus to be elucidated.[20–28] It is now widely recognized that function of the mitral valve depends on the normal function and integrity of the leaflets, annulus, chordae tendineae, papillary muscles, and subjacent left ventricular myocardium. Abnormalities in any of these components, either individually or in combination, produce dysfunction of the mitral valve unit. Multiplane TEE can define mitral valve structure and function. To obtain a comprehensive and accurate description of the whole mitral apparatus it is useful to have a systematic approach when performing the multiplane TEE examination. Beginning with the multiplane TEE transducer in the stomach the papillary muscle structure can be assessed along with their interaction with the left ventricle. With slow withdrawal of the probe into the esophagus the chordae tendineae, and the valve leaflets including commissural areas and annulus, can be assessed in succession (see Figures 2.8–2.11). With a careful and systematic TEE assessment, a three-dimensional picture of the whole mitral apparatus can be constructed.

With the multiplane TEE transducer at 0°, 40–45 cm from the incisors in the transgastric position, the probe is slightly anteflexed and maneuvered to obtain a short axis view of the left ventricle at the mid papillary muscle level (Figures 2.16, 2.18). Depending on the position



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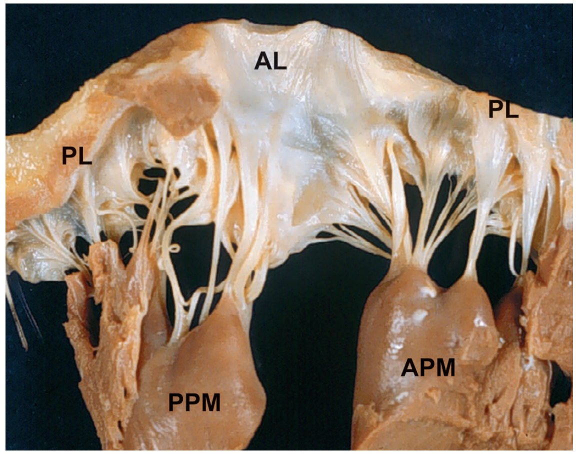

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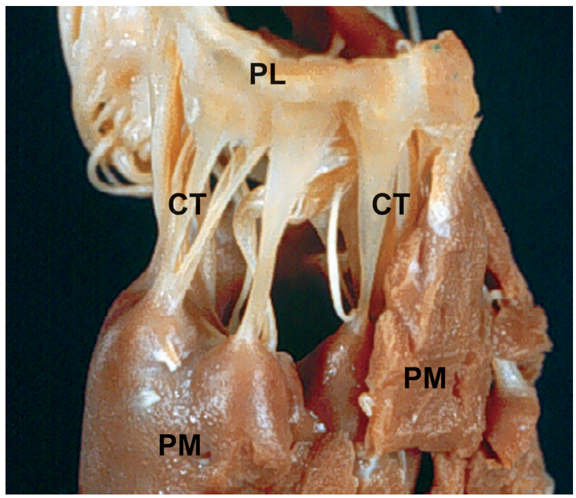
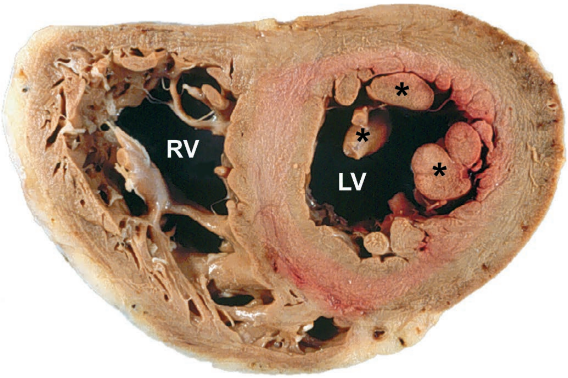
Fig. 2.5 Normal mitral valve anatomy. (a) Postmortem mitral valve specimen viewed from the atrial aspect of the leaflets. Chordae tendineae attach to the undersurface of the leaflet margins producing hooding of the leaflet edge for the rough zone (stars). The greater thickness of the rough zone of the leaflet margin provides greater surface area for leaflet coaptation. In addition, false chordae tendineae are demonstrated bridging one papillary muscle to another. (b) Postmortem mitral valve specimen viewed from the ventricular aspect of the leaflets. Chordae tendineae insert into the ventricular or undersurface of the leaflet margin. Chordae tendineae only insert to the peripheral margin of the anterior leaflet. (c) Postmortem mitral valve specimen viewed from a lateral ventricular aspect of the posterior leaflet surface. The parallel projection of chordal insertion to the posterior leaflet from the papillary muscles is demonstrated nicely. Chordae tendineae insert into the whole undersurface of the posterior leaflet including the leaflet margin, the body of the leaflet and the annular insertion portions. This chordal arrangement again lends credence for the pressure support function of the posterior mitral leaflet. AL, anterior leaflet; PL, posterior leaflet; APM, anterior papillary muscle; PPM, posterior papillary muscle; CT, chordae tendineae.



c

in the chest, either vertical or horizontal, the transducer may to be rotated from 0° to about 15° to obtain a true, non-oblique short axis view of the left ventricle. With the depth of the echocardiographic image set to 12–16 cm, the normal left ventricle should fill most of the image sector and allow good visualization of the papillary muscles for examination. The papillary muscles are visualized in the short axis of the left ventricle in cross-section. The posteromedial papillary muscle is at the top of the echocardiographic display at approximately 1 o'clock, and the anterolateral papillary muscle is located between 4 and 5 o'clock.



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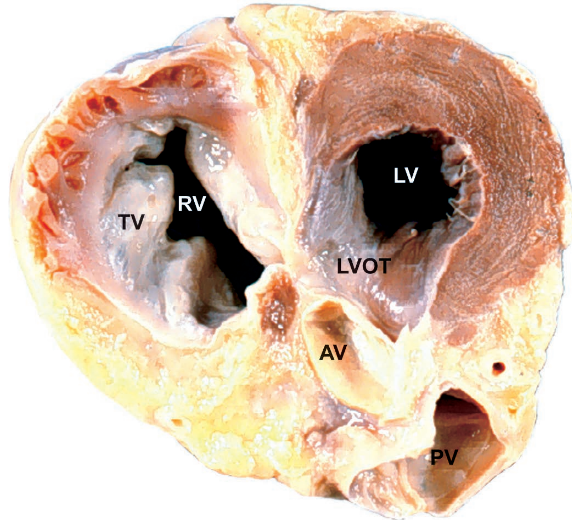

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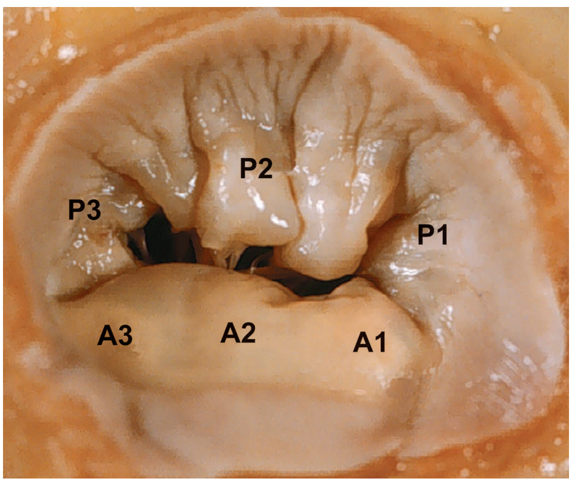

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Fig. 2.6 Short-axis anatomical preparations corresponding to the standard echocardiographic short-axis views obtained from the gastric window at 0°. (a) Ventricular short axis at the papillary muscle level. (b) Ventricular short axis at the basal level. (c) Short-axis orientation of the mitral valve as typically projected from the transesophageal gastric window. RV, right ventricle; LV, left ventricle; TV, tricuspid valve; LVOT, left ventricular outflow tract; AV, aortic valve; PV, pulmonary valve; A1,A2,A3, anterior mitral leaflet; P1,P2,P3, posterior mitral leaflet.

The exact position of the papillary muscles varies in different patients, and this is better appreciated by TEE imaging than by transthoracic echocardiography. Both papillary muscles are normally situated in the longitudinal, posterior half of the left ventricle. Therefore in the short axis view the distance between the papillary muscles in the inferior aspect of the left ventricle is less than half the distance around the anterior aspect of the left ventricle. The anterolateral papillary muscle is usually larger than the posteromedial muscle and usually comprises one muscle, in contrast to the posteromedial muscle, which is made up of a group of two or more smaller muscles. Both papillary muscles should appear in close approximation with the ventricular myocardium, and should have the same texture as the surrounding myocardium. The size or diameter of the papillary muscles should be slightly smaller than the myocardial thickness. Occasionally, both papillary muscles may exhibit isolated hypertrophy,[19] appearing thicker or slightly out of proportion to the ventricular myocardium, but are in normal positions in the ventricular cavity.

The transducer is then rotated 90° from the true cross-section to assess the papillary muscles longitudinally (Figure 2.19). In the long axis view, the papillary muscles appear in the middle of the echocardiographic image. In the apex-up orientation, with the transducer at

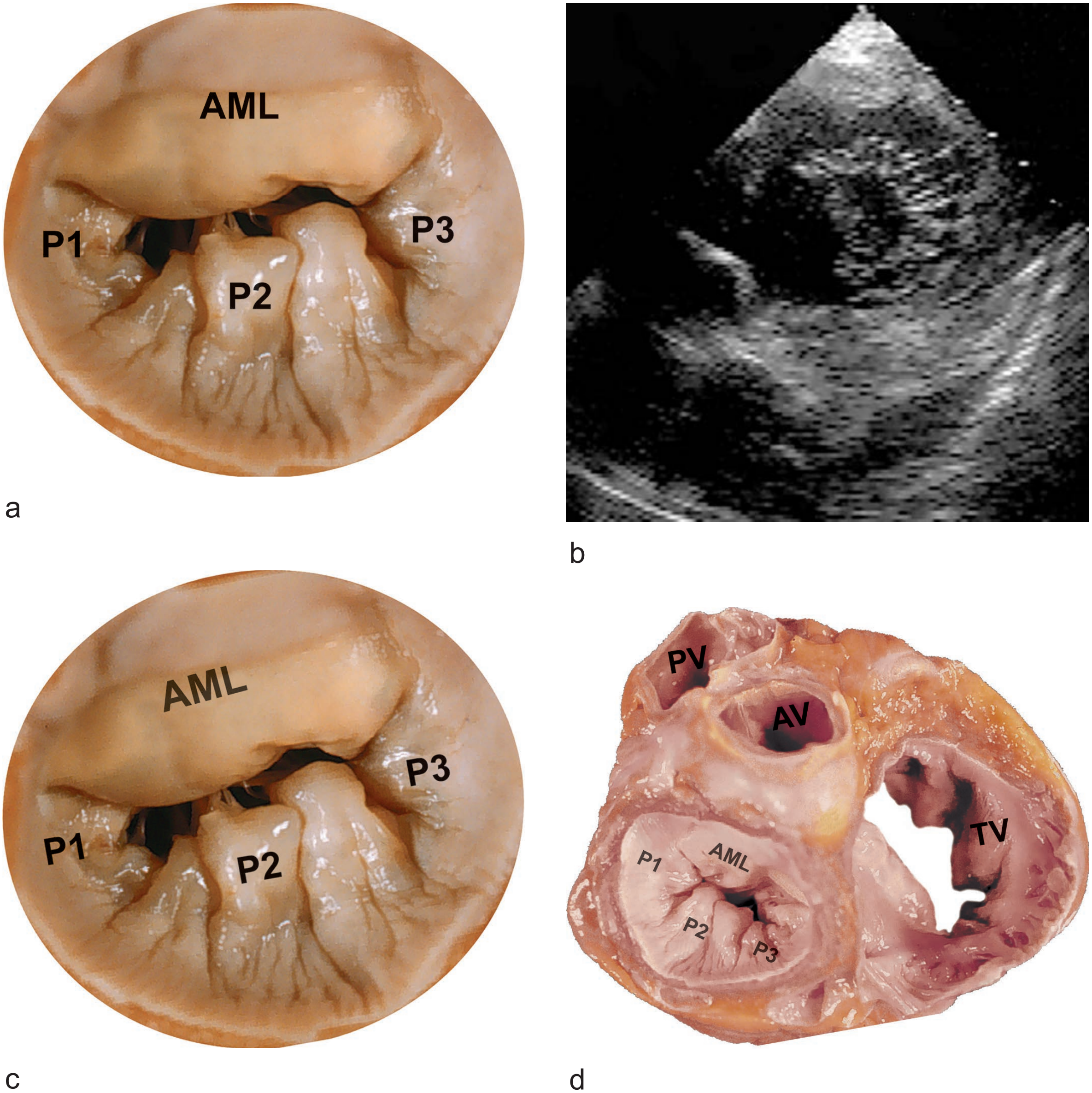


Fig. 2.7 Orientation of the mitral valve from different perspectives in the operating room. It is helpful to understand the orientation of the mitral valve from different perspectives so that meaningful and accurate communication can occur between the surgeon, the echocardiographer, and the anesthesiologist, especially during mitral valve reparative procedures. (a) Surgeon's anatomical view. (b) Short-axis echo image. (c) Echocardiographer's orientation. (d) Anesthesiologist's anatomical perspective from the head of the operating table. AML, anterior mitral leaflet; P1,P2,P3, posterior mitral leaflet.

the top of the screen, the posteromedial muscles with multiple heads appear directly at the top of the image and project towards the base of the heart, with the anterolateral muscle at the bottom of the image. Either papillary muscle may be better defined with minor angulations of the probe. The papillary muscles take origin from the myocardium in the mid third of the

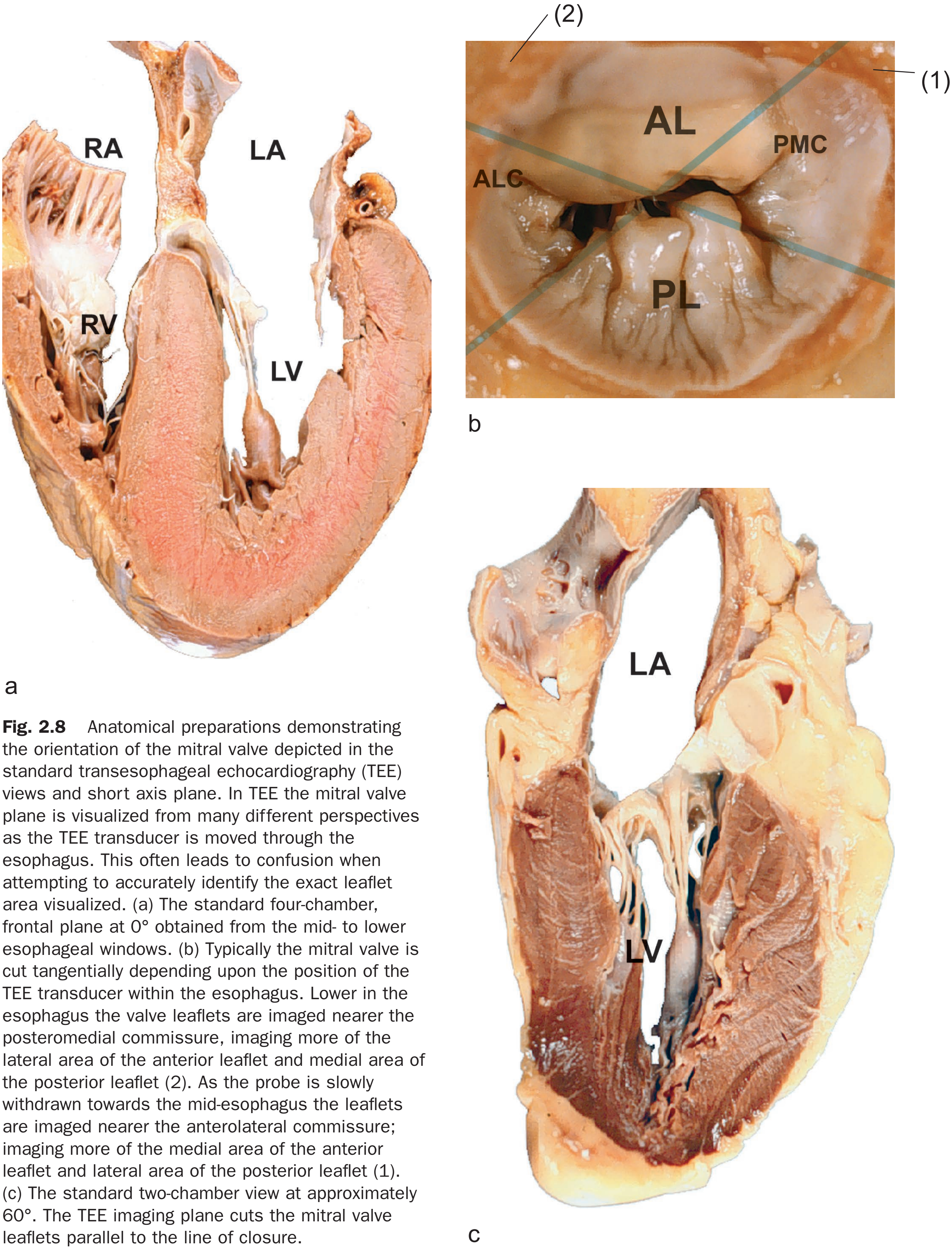
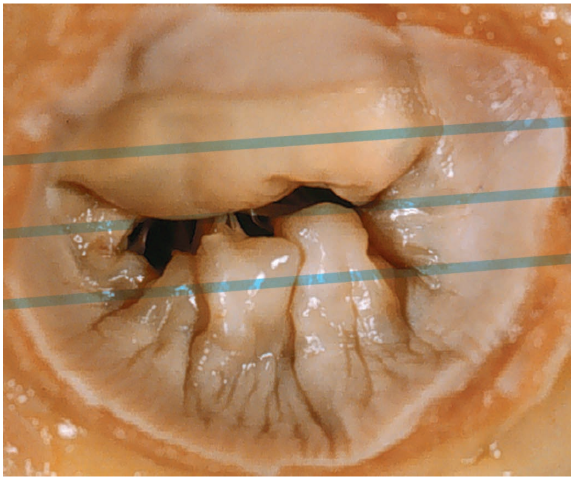


Fig. 2.8 Anatomical preparations demonstrating the orientation of the mitral valve depicted in the standard transesophageal echocardiography (TEE) views and short axis plane. In TEE the mitral valve plane is visualized from many different perspectives as the TEE transducer is moved through the esophagus. This often leads to confusion when attempting to accurately identify the exact leaflet area visualized. (a) The standard four-chamber, frontal plane at 0° obtained from the mid- to lower esophageal windows. (b) Typically the mitral valve is cut tangentially depending upon the position of the TEE transducer within the esophagus. Lower in the esophagus the valve leaflets are imaged nearer the posteromedial commissure, imaging more of the lateral area of the anterior leaflet and medial area of the posterior leaflet (2). As the probe is slowly withdrawn towards the mid-esophagus the leaflets are imaged nearer the anterolateral commissure; imaging more of the medial area of the anterior leaflet and lateral area of the posterior leaflet (1). (c) The standard two-chamber view at approximately 60°. The TEE imaging plane cuts the mitral valve leaflets parallel to the line of closure.



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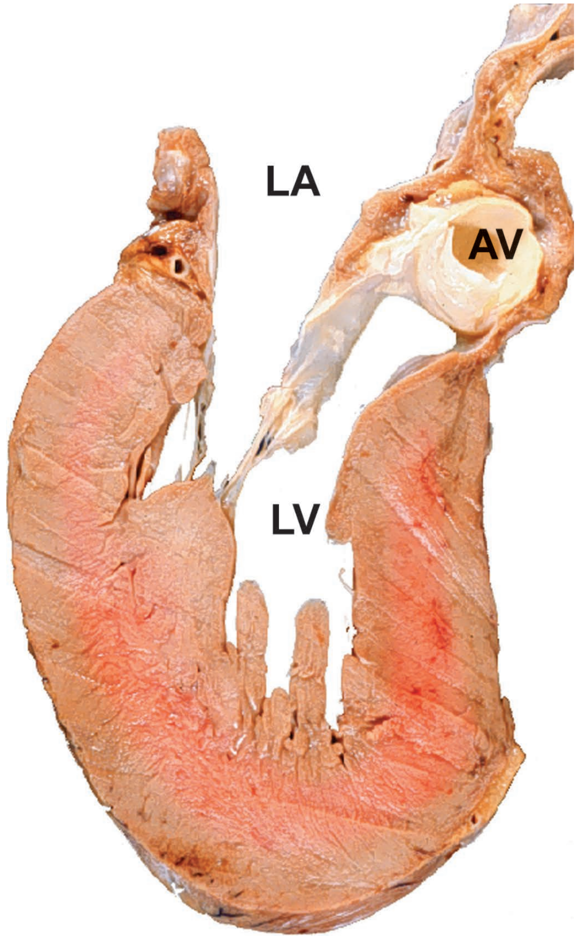

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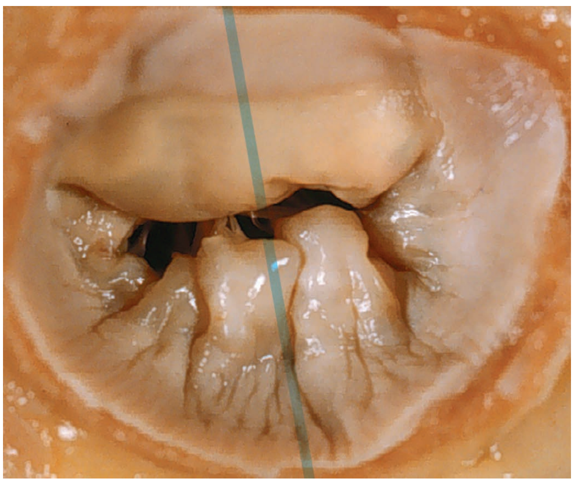

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Fig. 2.8 (contd.) (d) With a slight rotational movement of the transducer the plane can be directed through the anterior leaflet and posterior leaflet line of closure. (e) The standard three-chamber view at 135°. (f) This view produces an imaging plane nearest to truly cutting the mitral valve in an anterior–posterior orientation. RA, right atrium; LA, left atrium; RV, right ventricle; LV, left ventricle; AV, aortic valve; AL, anterior leaflet; PL, posterior leaflet; ALC, anterolateral commissure; PMC, posteromedial commissure.

ventricle, with the anterolateral muscle generally appearing longer and narrower in the longitudinal projection. In all cases, the papillary muscles should have similar size and motion. The papillary muscles are attached to the ventricle with a broad base and gradually taper to a head in a conical fashion, at the same level in the ventricular cavity. Occasionally, a small accessory papillary muscle may be seen emanating from the apex. All muscles should have similar echogenicity, signifying the same muscle texture and density of the surrounding ventricular myocardium. In older patients, a small degree of increased echogenicity is frequently observed in the papillary muscle tip, signifying calcification, which probably represents the normal aging process. Normal motion of the ventricular myocardium should also be

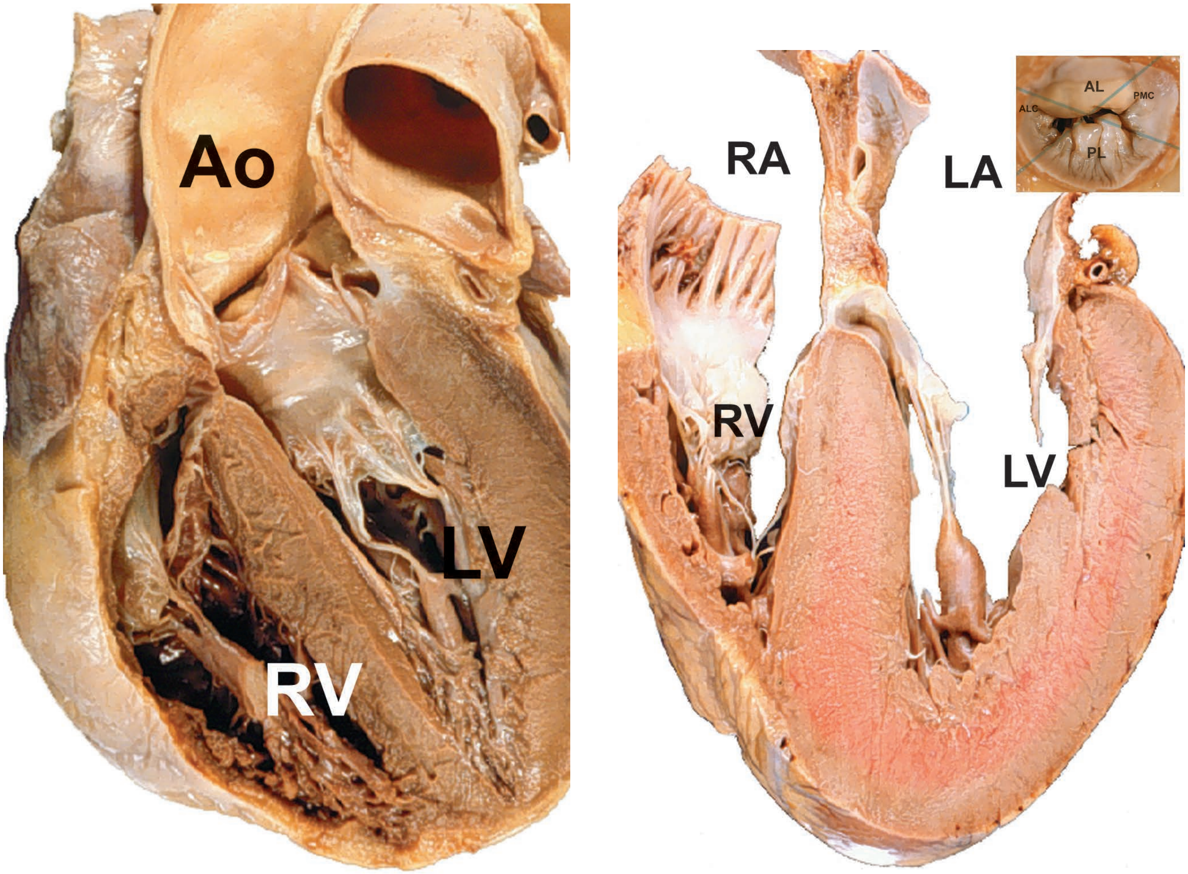

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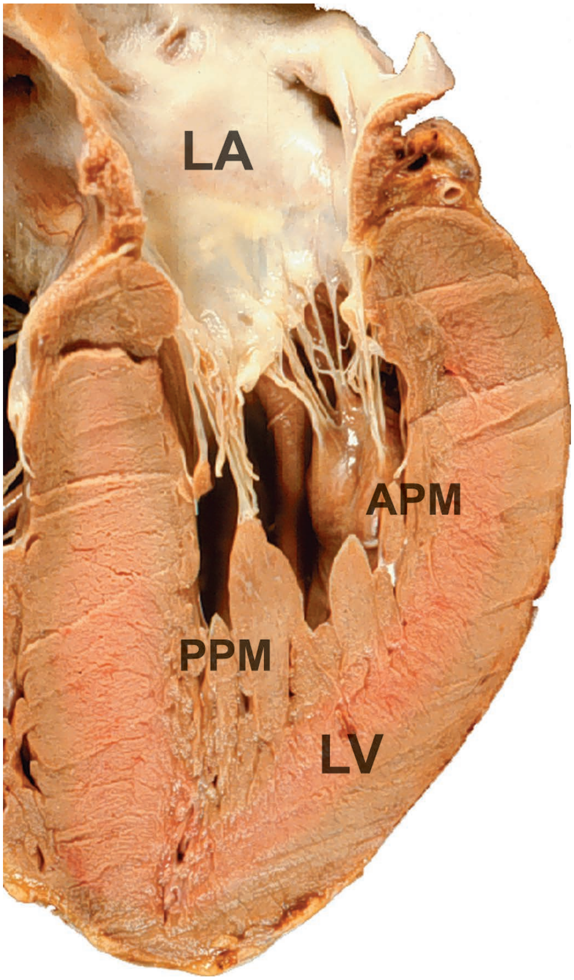
Fig. 2.9 Normal echocardiographic anatomy demonstrating mitral valve anatomy. In the lower esophageal window the mitral valve is imaged in different planes as the transducer is rotated between 0° and 180°. In examining the mitral leaflets and subvalvular apparatus it is important to have a conceptual idea of the true anatomy in each plane in order to mentally construct a three-dimensional perspective of the mitral anatomy. Mitral valve disease or abnormalities can more accurately be described and understood in this manner. Frontal projections. (a) Anatomical preparation of the heart at –10°. (b) Enlargement of mitral apparatus –10°.

observed in relation to normal papillary muscle motion. Fractional shortening of the papillary muscle (FSPM, Figure 2.20) can be determined in this view by measuring the end-diastolic (EDL) and end-systolic longitudinal lengths (ESL), so that:

$$\mathrm{FSPM} = [\{\mathrm{EDL} - \mathrm{ESL}/\mathrm{EDL}\} \times 100].$$

Normal papillary muscle fractional shortening in is 30% ±8%.[19]

The chordae tendineae are visualized as thin horizontal, continuous structures emanating from the heads of the papillary muscles, projecting towards the annulus (base of the heart) and inserting into the ventricular surface of the leaflets (see Figure 2.17). The chordae tendineae appear as multiple, thin, linear structures that move in and out of plane between systole and diastole. Normal chordae emanate from the apex or head of the papillary muscles and



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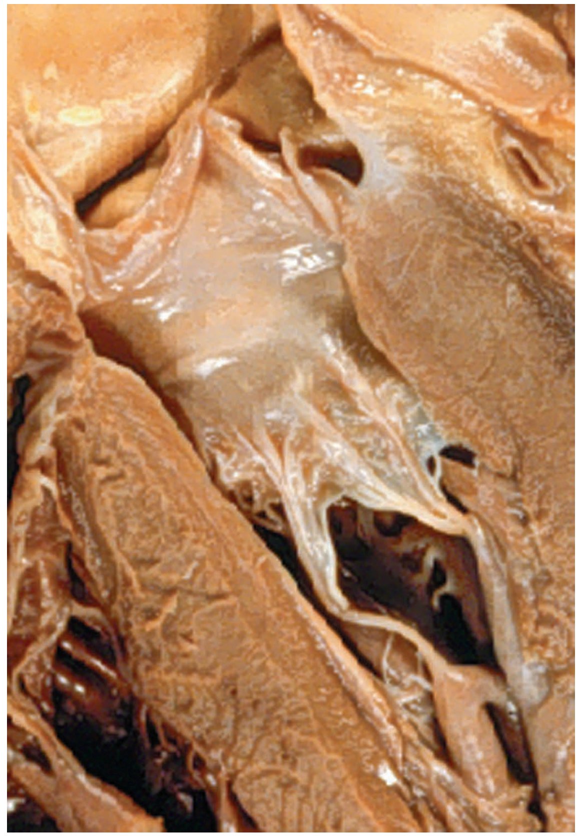


d

Fig. 2.9 (contd.) (c) Anatomical preparation of the heart at 0°. (d) Enlargement of mitral apparatus 0°.

project in a straight, nearly horizontal manner as they fan out towards the leaflets. In the longitudinal projection, the chordae that insert into the posterior leaflet appear nearly horizontal, whereas the chordae to the anterior leaflet are more oblique. It should be noted that all chords, whether they insert into the anterior or posterior leaflet, should appear straight – almost under tension – in both diastole and systole. With the currently available multiplane probes, branching of the chordae tendineae is apparent, but even in the zoom modes the image resolution does not allow for the reliable labeling of chordae as primary, secondary, or tertiary branches. Occasionally, chordae may be identified that emanate from the heads of the papillary muscles and insert into the ventricular myocardium. These chordae are frequently identified by transthoracic imaging, but are more readily apparent with transesophageal

e

f

Fig. 2.9 (contd.) (e) Anatomical preparation of the heart at 30°. (f) Enlargement of mitral apparatus 30°.

echocardiography, and are known as ventricular chordae or aberrant chordae.

The probe is slowly advanced to approximately 50 cm at 0° to the deep transgastric level, with anteflexion until the aortic root is imaged obliquely and the mitral subvalvular apparatus is well seen (see Figure 2.16). The papillary muscles are visualized obliquely with a foreshortened view of the left ventricular apex near the top of the echocardiographic screen. Usually both papillary muscles are visualized between 0° and 20°. The central portions of both papillary muscles are easily inspected along with the chordae tendineae. The chordae tendineae appear straight, all about the same width, and lie in a vertical position in the image. The valve leaflets, however, are too foreshortened to be fully appreciated, especially when they are normal or show hooding, but it is possible to distinguish the anterior from the posterior leaflet. In our experience, this is the best view to assess the chordae tendineae. Elongation of the chordae is easily seen, as the chordae appear redundant, bowed, and curvi-

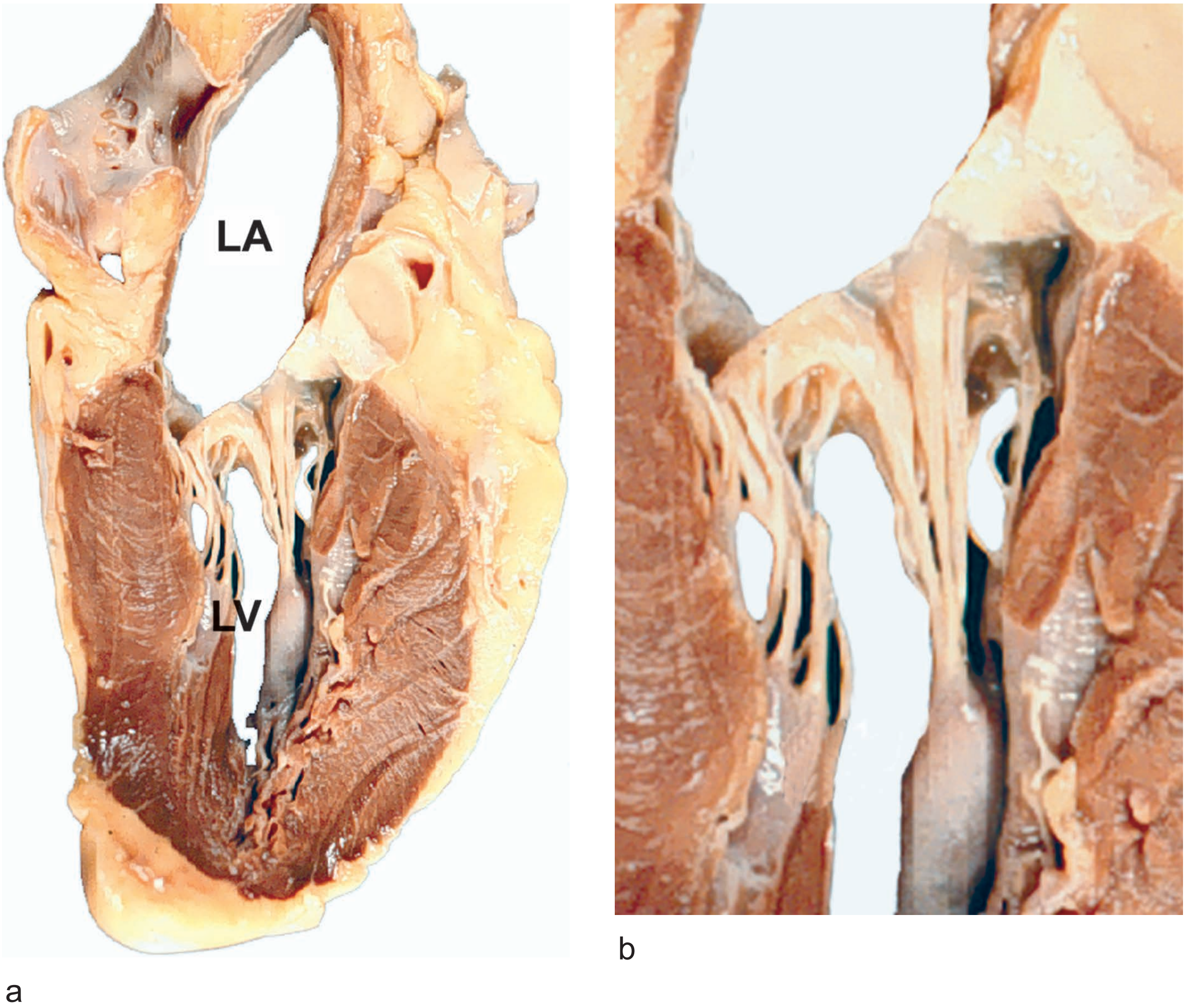
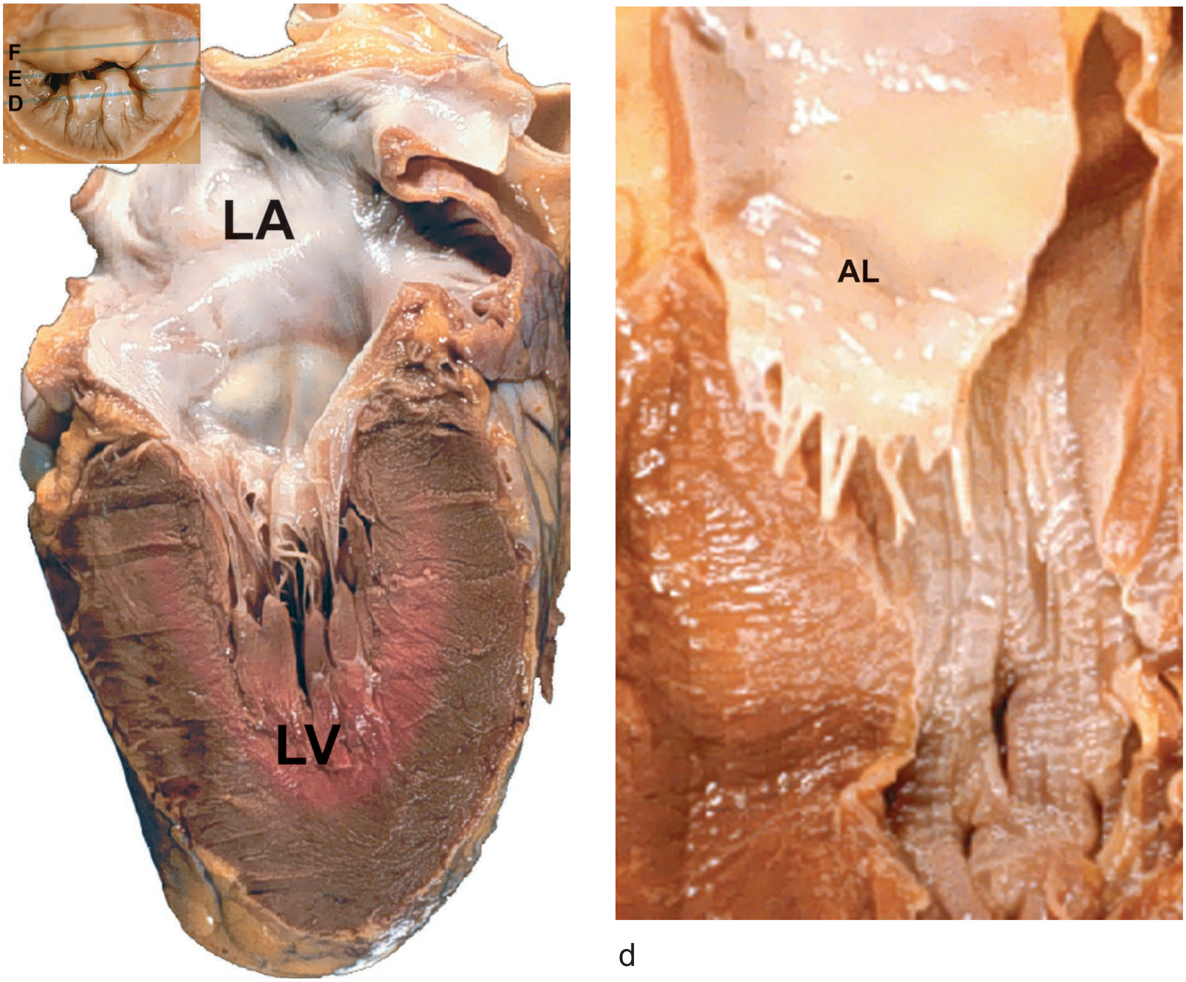


Fig. 2.10 Normal echocardiographic anatomy demonstrating mitral valve anatomy (continuation of Figure 2.9). Longitudinal projections. (a) Anatomical preparation of the heart at 60° with (b) enlargement of mitral apparatus.

linear, instead of straight. The chordae to the anterior leaflet appear to project at an angle away from the papillary head, and the chordae to the posterior leaflet appear to be parallel and vertical. With further rotation of the transducer from 45° to 50°, a sweep of the whole subvalvular apparatus is completed and nearly all of the chordae can be assessed.

The multiplane probe is then slightly withdrawn at 90° to center the annulus and leaflets in the echocardiographic image. With slight angulations of the probe, the full breadth of the anterior, posterior, or both leaflets are seen vertically, opening and closing with changes in the cardiac cycle. The mitral annular plane is readily apparent, separating the left ventricle (to the left of the image) and the left atrium (to the right of the image), and the normal mitral valve leaflets do not protrude significantly past this imaginary plane towards the left atrium. During diastole when the two leaflets are imaged, the posterior leaflet is seen at the top of



c

Fig. 2.10 (contd.) (c) Anatomical preparation of the heart at 75° with *en face* view of the mitral leaflets illustrating rotational cuts and corresponding enlargements showing (d) predominately anterior leaflet.

the echocardiographic image and the anterior leaflet at the bottom of the image. Rotating the transducer from 90° to 135° shifts the image towards the left ventricular outflow tract and the aortic root, displaying the anterior leaflet continuous with the aortic root at the bottom of the display. Care should be taken in assessing the thickness of the leaflets at the points of chordal insertion so as not to overestimate leaflet thickness.

The transducer is returned to 0° and the probe is withdrawn towards the lower esophageal level (about 35 cm from the incisors) to image the base of the left ventricle and project the mitral valve leaflets and annular plane *en face* (Figures 2.12 and 2.21). The probe