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Anatomy of the Mitral Valve Apparatus – Role of 2D and 3D Echocardiography

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Abstract

The mitral valve apparatus is a complex three–dimensional functional unit that is critical to unidirectional heart pump function. This review details the normal anatomy, histology and function of the main mitral valve apparatus components 1) mitral annulus, 2) mitral valve leaflets, 3) chordae tendineae and 4) papillary muscles. 2 and 3 dimensional Echocardiography is ideally suited to examine the mitral valve apparatus and has provided insights into the mechanism of mitral valve disease. An overview of standardized image acquisition and interpretation is provided. Understanding normal mitral valve apparatus function is essential to comprehend alterations in mitral valve disease and the rationale for repair strategies.

Keywords

mitral valve apparatus; mitral valve; mitral annulus; papillary muscles; chordae tendineae; mitral regurgitation; echocardiography

Introduction

The normal mitral valve apparatus is a dynamic three-dimensional system that allows brisk left ventricular (LV) blood-inflow during diastole and ensures unidirectional heart pump function by sealing the left atrium from the LV during systole. Key components are the mitral annulus, the mitral valve leaflets, the chordae tendineae, and the LV wall with its attached papillary muscles (PMs) (Figure 1). Proper valve function is dependent on the integrity and harmonious interplay of these components – and an imbalance can result in a leaking (regurgitant, insufficient, incompetent), stenotic, or combined regurgitant and stenotic valve dysfunction. A detailed understanding of mitral valve apparatus development, anatomy and function is important for cardiac imaging interpretation, for disease diagnosis, and comprehending the rationale for repair strategies. Repair of the diseased mitral valve

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requires understanding the *dysfunction* caused by the disease *lesion* in order to restore a normally functioning valve, a lesson learned from one of the foremost pioneer's in mitral valve repair – Prof. Alain Carpentier.

Mitral Valve Apparatus Development

Mitral valve development is complex and still under investigation (1). The developing heart tube consists of extracellular matrix sandwiched by myocardial and endothelial layers, and has folded into a basic four-chamber configuration by the end of fetal week 4. By week 5 the outer myocardial layer has compacted and trabecular structures start to form within the future LV cavity. At this point, superior, inferior and lateral cardiac cushions can be detected in the common atrioventricular (AV) canal (2). These 5 cushions are formed by endothelial cells that migrate into the mesenchyme and change into an interstitial fibroblasts cell-type, a process termed endothelial–mesenchymal transdifferentiation or transformation (EMT) (1). The superior and inferior cardiac cushions fuse at week 7 to 7 ! and divide the common AV canal into right and left portions. The anterior (aortic) leaflet of the mitral valve originates from the fused superior and inferior cushion tissue in the left AV canal and starts to delaminate or separate from the myocardial wall shortly thereafter. The posterior (mural) leaflet forms from the left lateral cushion (2). Over weeks 8–10 the LV intracavitary trabecular bridges compact, and by week 10 small (antero)lateral and (postero)medial papillary muscles (PMs) can be observed delaminated in the LV cavity. Actually both are located posteriorly relative to the anterior chest wall - one medial, the other lateral. Both PMs attach directly to both leaflet-forming cushions. From week 11 to 13 the leaflets continue to form, the PMs become more distinct, and rudimentary chordae develop. By week 15 the MV leaflets, chordae and PMs become developed (2). The mitral valve apparatus then continues to grow to meet the needs and demands of the developing organism (3).

Mitral Valve Apparatus Anatomy & Histology

1) Mitral annulus

The mitral annulus (MA) is defined by the tissue juncture of the left atrium, the left ventricle and the mitral leaflets. It is a dynamic, anatomically ill-defined structure. En face the MA resembles a kidney-bean; in three dimensions it is a non-planar saddle shape (4–6).

The anterior flatter portion of the mitral annulus is continuous with the aortic annulus, is the elevated (most atrial) "horn" of the saddle shape and consists of parallel collagen fibers (4,7). The MA-to-aortic annular angle changes dynamically over the cardiac cycle, with displacement coupled through the fibrous continuum (8). Reciprocal systolic and diastolic aortic and mitral annulus area changes have been observed (9–11). The posterior part of the MA runs distal to the left (lateral) and right (medial) fibrous trigones and includes the low points of the saddle close to the lateral and medial commissures and the posterior saddle horn. Compared to the anterior portion, the posterior mitral annulus is more loosely anchored to the surrounding tissue, allowing it to move freely with myocardial contraction and relaxation (7). It is basically a junction of leaflet and myocardium. Less static in all dimensions than the anterior MA portion, the posterior MA allows for systolic apical bending along a mediolateral commissure axis, with increased saddle height and circumferential area decrease (12). The saddle-shaped MA and its dynamic change reduces leaflet tissue stress and is important for coaptation geometry (13–20). The MA is innervated and supplies blood vessels to the leaflets (21–23). From week 18 to 24 on to adulthood the MA area increases 25-fold (24) and, contrary to the widely published "normal" mitral annular orifice area of 4–6cm^2, multiple investigations with differing imaging modalities report an average mitral annular area of ~10cm^2 in healthy subjects (25–30). MA area can

significantly increase in patients with dilated LVs (27,29,30). This is accompanied by MA flattening (29,31) and decrease and delay of systolic sphincter-like mitral annular area reduction (29,32). The final result of these changes is altered leaflet stress and unfavorable mitral leaflet remodeling (Table 1). MA flattening has also been recently described in myxomatous valve disease, associated with more severe MR and chordal rupture, potentially related to increased out-of-plane stresses (20,33).

2) Mitral valve leaflets

The mitral valve has anterior and posterior leaflets and variable commissural scallops to occlude medial and lateral gaps (Figure 1C, Figure 2). Leaflet tissue circumferentially attaches to the mitral annulus with a minimum tissue length of 0.5 to 1cm (34). Redundant leaflet tissue is critically important for leaflet apposition and tight leaflet coaptation. In normal and dilated LVs a leaflet to MA area ratio of 1.5 to 2 has been found sufficient to prevent significant mitral regurgitation (30,34). The atrial surface of the leaflets is smooth and the leaflet body translucent (Figure 2). A hydrophilic protein rich zone, termed rough zone, starts ~1 cm from the distal leaflet edge. When the leaflets coapt, the irregular, soft surface of this zone helps to maintain and insure a seal (Figure 2, Coaptation zone). The ventricular surface of especially the anterior leaflet is a basket-weave of criss-crossed collagen strands that originate at the chordal insertion and continue into the annulus (35,36). Secondary chordae insert close to the rough zone, while primary chordae insert at the free leaflet tips (35,37).

The anterior (also labeled aortic or septal) mitral leaflet is trapezoid- or dome-shaped, anchored to the fibrous portion of the mitral annulus, and shares a fibrous tissue continuity mainly with the noncoronary cusp of the aortic valve (Figure 2). Its collagen fiber orientation suggests tight anchoring into the left (anterolateral) and right (posteromedial) fibrous trigones (38). The anterior leaflet is larger, longer and thicker than the posterior leaflet (Table 2, Figure 2). To facilitate diagnostic and therapeutic medical communication, the anterior leaflet can be divided into lateral (A1), central (A2) and medial scallops (A3). For the anterior leaflet, this nomenclature does not represent anatomicaly distinct structures (Figure 1C).

The posterior (mural) leaflet is crescentic with a long circumferential base (~5cm vs ~3cm anterior leaflet, (39)) and relatively short radial length (Figure 1C and Figure 2; Table 1). It is attached to the posterior portion of the mitral annulus. Similar to the anterior leaflet, the posterior leaflet can be divided into lateral (P1), central (P2) and medial scallops (P3). Slits and indentations within the posterior mitral tissue demarcate these scallops (Figure 1C) (37).

Additional leaflet tissue termed commissural, accessory or junctional can be found at the anterolateral (A1-P1) and posteromedial (A3-P3) commissures (Figure 1B and 1C, Figure 2). Their tissue length measured form annular insertion varies from 0.5 to 1cm. (34,35).

The MV leaflets fully open in less than 100ms 3 billion times throughout a lifetime and are exposed to a wide range of LV pressures (40). Despite this demanding environment, significant MV disease is uncommonly seen in patients younger than 65 years (41). Influenced by the immediate environment and mechanical needs, the MV leaflet tissue is trilaminar, consisting of fibrosa/ventricularis, spongiosa and atrialis layers. Valvular endothelial cells cover the blood-interfacing surfaces. Each layer has unique extracellular matrix (ECM) characteristics: Hemodynamically exposed to LV pressures, the fibrosa/ventricularis is composed of dense collagen, which is important for mechanical stability. The spongiosa has less organized collagen, but especially at the leaflet tip rough zone, is rich in water absorbent proteins. This protects the leaflet edges and ensures a tight seal. The atrialis contains a network of collagen and elastin and appears to play a critical role in leaflet

remodeling and adaptation (42). While trilaminar, the layer distribution of the anterior and posterior leaflets differs significantly: Much of the anterior leaflet thickness is due to a dominant fibrosa layer, which allows this leaflet to withstand a significantly higher tensile load without tissue disruption. The posterior leaflet is thinner and more flexible (43,44). The anterior mitral leaflet has an especially dense innervation, and nerve terminals close to smooth muscle cells and fibrobasts suggest a potential neural feedback or regulatory mechanism (45). Interestingly, but of unknown significance, this neural innervation greatly diminishes with age (45,46). Cardiac muscle cells can be detected in both leaflets close to the annulus. This tissue is excitable via the atria, isolated from LV excitation, and resembles atrial myocardium (36). The healthy, mature MV has a very rudimentary vascular and lymphatic system (23,47), interstitial cells are mostly dormant, and the ECM turnover is slow. Physiologic or pathologic-induced leaflet stress can induce prominent EMT, interstitial cell activation & proliferation, ECM remodeling and neovascularization (3,42,48,49). Regulation of mitral leaflet adaptation is not well understood, but the wide range of total leaflet area (Table 2) suggests a potent adaptation mechanism that has to be further explored and therapeutically targeted.

3) Chordae tendineae

The chordae tendineae are fibrous strings that originate with highly variable branching from the PM tips (heads) and insert fan-like into the ventricular aspects of the anterior, posterior and commissural leaflets (Figure 1B and 1C, Figure 2) (37,50). Occasionally chordae originate from the basal posterior myocardium and insert directly into the posterior leaflet (34). Two main types of chordae can be distinguished based on leaflet insertion: Primary (marginal) chordae, which attach to the leaflet free edges, and secondary (basal) chordae which insert into the anterior leaflet edge rough zone and throughout the posterior leaflet body (51). Chordae are composed of an interfacing, tightly linked collagen and elastin network that dampens PM-leaflet force transmission (50).

Secondary chordae are thicker than primary chordae and have more tightly crimped collagen, making them more extensible (52). Commonly a pair of thick secondary chordae, termed strut chordae, insert at 4 and 8pm into the ventricular aspect of the anterior leaflet; additional strut chords, including to the posterior leaflet have been described. Basket-woven collagen fibers distribute chordal force over the leaflet surface from insertion to the annulus (34,36,51). To relieve pathologic apical leaflet tethering and restore mitral leaflet coaptation in patients with functional/ischemic mitral regurgitation, selected secondary chordae can be cut without deleterious effects on LV function (53–55).

Primary chordae are thinner, insert at the leaflet tips and have limited extensibility due to higher collagen fibril density and reduced crimping (52). These characteristics prevent leaflet edge eversion (=flail leaflet) (56). There is a wide variability in chordal anatomy and branching patterns (37) which makes correct anatomic labeling and measurement comparisons difficult. Most consistent and reproducible results are available for the anterior strut chordae. Their normal average length and thickness are reported at ~20mm and 1–2mm respectively (37,39,42,51). Similar to mitral leaflets, chordae adapt to altered loading conditions (42).

4) Papillary muscles

The papillary muscles are labeled by their projected relationship to the mitral commissures as lateral and medial (Figure 1B and 1C) (35). Their bodies originate from the apical 1/3 of the LV and protrude finger-like into the cavity (2). Chordal fans extend from the PM heads to the corresponding anterior, posterior and commissural leaflet portions (Figure 2) (34). The lateral PM in the majority of cases has a single head and dual blood supply from the left

circumflex and left anterior descending artery. The medial PM most commonly has 2 heads and is either supplied by the right or circumflex coronary artery based on dominance (34,35,57). The PM-chordal system is finely tuned so that PM contraction maintains the systolic spatial relationship between the mitral annulus and the PM heads as the intervening myocardium contracts, akin a shock absorber, thereby preventing leaflet prolapse (58–60). The PM head positions and relative distance to each other keep both leaflets under outwardly-directed tension and therefore posteriorly restrained to prevent anterior motion (see hypertrophic 12 cardiomyopathy below). The anterior, posterior and commissural leaflets are therefore in optimal position and configuration to form an effective systolic coaptation seal (61).

2D and 3D Echocardiography of the Mitral Valve Apparatus

Echocardiography is the clinical tool of choice for diagnosing, assessing and following patients with valvular heart disease (62). It is a noninvasive, non-ionizing imaging test with excellent spatial and temporal resolution. Two-dimensional (2D) and threedimensional (3D) Echocardiography (Echo) provides detailed morphologic and functional assessment, while Doppler Echocardiography evaluates hemodynamics. Indeed, the functional mechanisms of mitral regurgitation in many conditions were first clearly defined by Echo. Constant advances in information technology make Echo very portable (63) and an increasingly important tool to guide minimally invasive percutaneous valve repairs (64).

3D Echo has been pivotal to today's understanding of the normal and diseased mitral valve apparatus: 3D Echo established the saddle-shaped, non-planar shape of the mitral annulus (4,6), helped explore the complex geometric relationship of PM and leaflet position relative to the mitral annulus and LV outflow tract (61,65–67) and recently made it possible to measure mitral leaflet size in the beating heart (20,30,42,68). Consequences have been the development of non-planar mitral annuloplasty rings (69), and a detailed understanding of the mechanisms of ischemic/function mitral regurgitation (MR) and systolic anterior motion (SAM) of the anterior leaflet in hypertrophic cardiomyopathy (HCM) (61,65–67), a redefinition of the diagnostic criteria of mitral valve prolapse (MVP) (70,71) and recent evidence of mitral valve adaptation and leaflet growth (30,42,68).

Correct diagnosis of mitral valve disease is dependent on optimally acquired 2D Echo views. A three-dimensional cardiac anatomical understanding is paramount for 2D image acquisition and interpretation. Figure 3 shows the LV and mitral valve in a schematic short-axis view and the Echo beam planes for the most common 2D Echo views. Following the projected 4-Chamber view (4C) Echo beam helps to understand that the mitral valve scallops A3, A2 and P1 are typically shown in this very common Echo view. One can however also appreciate that a slight Echo probe and beam angulation will image a different plane and set of scallops while presenting an apparently similar image (72). 3D Echo controls for such uncertainty since the acquired 3D data can be precisely sliced in every dimension until the optimal and desired 2D view is obtained (73). The middle panel in figure 4 shows a 3D-rendered mitral valve from the surgical perspective (compare with Figure 1C). Slicing the 3D image set perpendicular (red line) to a mediolateral commissural axis (green line) will create 2D Echo views that accurately show the paired leaflet scallops in systole (upper panel) and diastole (lower panel).

Normal and Abnormal Mitral Valve Apparatus Function

Mitral valve anatomy is designed to promote and maintain normal mitral valve apparatus function; perturbations of the normal anatomic relations can result in mitral valve dysfunction (Table 3).

Tightly sealed MV leaflet coaptation depends on the balance of systolic tethering and closing forces on the valve and the amount of leaflet tissue available to cover the mitral annulus (74). Tethering forces are transmitted via the LV wall - PM - chordae system and keep the leaflets from prolapsing into the left atrium (Figure 1A). Closing forces depend on the pressure generated by the LV to close the mitral valve (67,68,75). Disturbance of this finely tuned spatial and temporal interplay of LV contraction, PMs, MV leaflets and mitral annulus can unsettle the tethering force-closing force balance relationship. If this leads to a deficit of leaflet area relative to annulus area coaptation will be impaired and mitral regurgitation occur (67,68,75). A vicious cycle may begin: Significant MR volume will overload the LV; to restore wall tension, the LV will remodel and dilate. Altered LV geometry will consequently remodel the mitral annulus, leaflets and chordae. MR severity will dynamically change throughout these adaptation processes, which conceivably are aimed to restore the tethering force-closing balance relationship (30,42,68). The natural disease course, however, suggests this process is comparable to destructive resonance, with each remodeled component causing disturbance in its relation to all the others. Throughout this, MR severity is a moving target, which, if progressive, will fuel ongoing LV remodeling.

Mitral Valve Prolapse

MVP is defined as mitral leaflet billowing by more than 2mm above the anterior and posterior horns of the mitral annulus during ventricular systole (Figure 5A) (70,71). MVP is usually diagnosed by echocardiography and is a manifestation of degenerative mitral valve disease. Coaptation geometry is altered due to a combination of leaflet and chordal extensibility, redundancy and elongation; whether superior papillary muscle displacement or traction is cause or effect has yet to be determined (76,77) (Figure 5A, dashed line & arrow). The billowing leaflets may appear diffusely thickened “Barlow’s syndrome” or thin except in flail portions “Fibroelastic deficiency” (39). Severe leaflet prolapse or flail leaflets can result in important MR (Table 3). Repair strategies aim to restore effective leaflet coaptation by reducing leaflet redundancy and mitral annular dimensions, and if needed implanting artificial chordae. Suitable mitral valve leaflet characteristics may also allow leaflet free edge approximation at the site of regurgitation (edge-to-edge technique) by a stich (Alfieri stitch) (78) or a clip (MitraClip) (79). Repair techniques, pioneered by Alain Carpentier, aim to restore leaflet function while preserving the native valve (80). Mitral valve replacement is not commonly indicated with a skilled surgeon.

Functional/Ischemic Mitral Regurgitation

Functional or ischemic MR is caused by MV leaflet tethering to displaced papillary muscles in the setting of a distorted, remodeled LV (Figure 5B). Pathophysiologic changes of LV form or function that increase the distance between PM heads and mitral annulus interfere with adequate leaflet coaptation (81–84). Key LV changes are LV remodeling with global dilatation and increased LV sphericity (67,85–88), or localized LV remodeling (89). If LV remodeling affects the PM-bearing ventricular walls, apical/posterior/posterolateral and outward PM displacement is likely to develop (89–94) (Figure 5B, arrows). Subsequent tethering restricts systolic closure motion of the MV leaflets. Affected leaflets stay “tied back” in the LV cavity, and this prevents a tightly sealed MV closure (Table 3) (66). Repair strategies aim to restore effective leaflet coaptation by reducing leaflet tethering at the PM and mitral annular ends. Therapeutic PM repositioning targets are LV shape (95–97) and function (revascularization (98–100), gene/cell therapy (101–103), cardiac resynchronization therapy (104,105)), PM approximation (106), chordal cutting (53–55,107), leaflet edge-to-edge approximation technique (78,79), mitral annulus area reduction or valve replacement. Overall, functional/ischemic MR therapy strategies that deal with the

annulus, but not the ventricular tethering are often limited, with recurrent MR(108–111); this can be reduced, for example by chordal cutting (112).

Hypertrophic cardiomyopathy

HCM is an autosomal dominant disease of myocyte disarray and fibrosis, morphologically characterized by significant LV hypertrophy in the absence of chronically elevated afterload or infiltrative diseases (e.g. Cardiac amyloidosis) (Figure 5C double-arrow) (113). HCM also involves the papillary muscles and mitral leaflets: Total mass (2-fold) and number of PM heads are increased (114). The PMs are anteriorly displaced (Figure 5C arrow) and the heads positioned closer to each other (61,115). This increases leaflet slack, and like a sail catching a breeze, the anterior leaflet is at risk of being displaced into the LV outflow tract by blood-flow drag forces (systolic anterior motion, SAM) (61,116–120). If anterior leaflet displacement is severe enough to impair posterior leaflet apposition, mitral regurgitation will occur (Figure 5C red lines) (121). Since PM position is a major culprit, septal reduction therapy does not always eliminate SAM (122). In HCM, the leaflets are also frequently elongated (123,124), contributing to leaflet slack and positioning the coaptation point anteriorly to increase the flow drag forces on the enlarged “sail”. After obstruction begins, LV outflow tract narrowing increases velocity above the valve, propagating SAM through airplane like lift forces (125,126). Because of these mechanisms, repair strategies include papillary muscle repositioning and reducing leaflet redundancy (Table 3).

In summary therefore, the MV is an elegantly constructed, well-balanced mechanism. Normal anatomic relations maintain the leaflets within the LV, preventing prolapse, and maintain them below LV outflow tract flow and taut, preventing SAM. Altered leaflet length and PM position can lead to MVP and obstructive SAM, which are both related to leaflet excess, as shown by their occurrence in the same patient (127,128). Conversely, the PM tethering conducive to normal function becomes maladaptive in ischemic MR when the PMs are displaced. Understanding MV anatomic relationships, appreciated by Echo as well as MRI and CT, is therefore essential to understanding its dysfunction in disease and developing optimal therapies (80).

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Keypoints

- The mitral valve apparatus is a complex three–dimensional functional unit that is critical to unidirectional heart pump function.
- The main mitral valve apparatus components are 1) mitral annulus, 2) mitral valve leaflets, 3) chordae tendineae and 4) papillary muscles.
- Tight sealed mitral leaflet coaptation depends on the balance of systolic tethering and closing forces on the valve and on the amount of leaflet tissue available.
- Echocardiography is ideally suited to examine the mitral valve apparatus and has provided insights into the mechanism of mitral valve disease.
- Understanding normal mitral valve apparatus function is essential to comprehend alterations in mitral valve disease and the rationale for repair strategies.

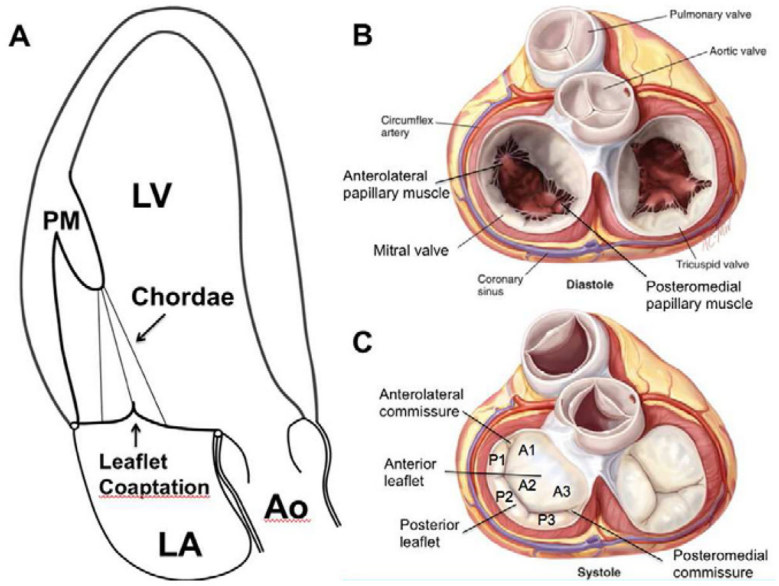

Figure 1.

A. Schematic apical long-axis view of the heart in systole with the apex on top. There is normal function and spatial relationship of the left ventricular myocardium, the papillary muscles (PM), chordae, leaflets and mitral annulus. The tethering force closing force balance relationship is normal, both leaflets are normally configured, concave toward the LV, and coapt without mitral regurgitation. B. Surgical view of the open mitral valve in diastole with the atrial walls removed. C. Surgical view of the closed mitral valve is systole. (Ao, aorta; LA, left atrium; LV, left ventricle; PM, papillary muscle; Panels B and C are adapted from Carpentier A et al. Carpentier's Reconstructive Valve Surgery. From Valve Analysis to Valve Reconstruction. 2010 Saunders Elsevier)

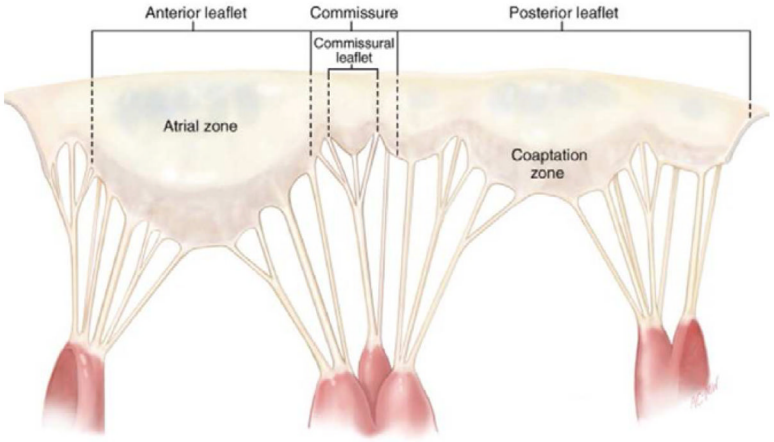

Figure 2.

The mitral valve is unfolded and the atrial leaflet surface exposed. The papillary muscles have been dissected and the heads remain attached via chordae tendineae to the anterior, posterior and commissural leaflets (Adapted from Carpentier A et al. Carpentier's Reconstructive Valve Surgery. From Valve Analysis to Valve Reconstruction. 2010 Saunders Elsevier)

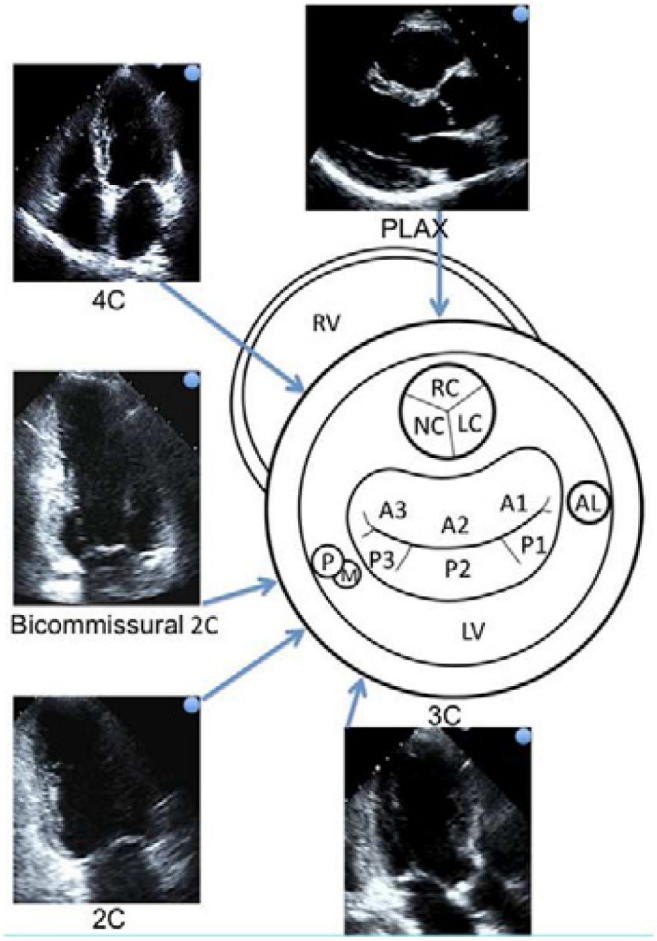

Figure 3.

The central schematic shows the left ventricle in short axis view seen from the apex. The approximate locations of the aortic valve, mitral valve and papillary muscles are projected. The most common and standardized 2D Echo views are arranged around the schematic. The blue arrow-tipped lines indicate the direction of the Echo beam and are connected to the corresponding Echo views. The blue dots on the Echo images 19 relate to the orientation of the blue arrows. Extrapolating the Echo beam lines allows us to estimate the 2D Echo mitral scallops represented in the corresponding Echo views. (2C, 2-Chamber view; 4C, 4-Chamber view; AL, anterolateral papillary muscle; LC, left aortic cusp; LV, left ventricle; NC, noncoronary aortic cusp; PLAX, parasternal long axis view; RC, right coronary cusp; RV, right ventricle; PM, posterolateral papillary muscle).

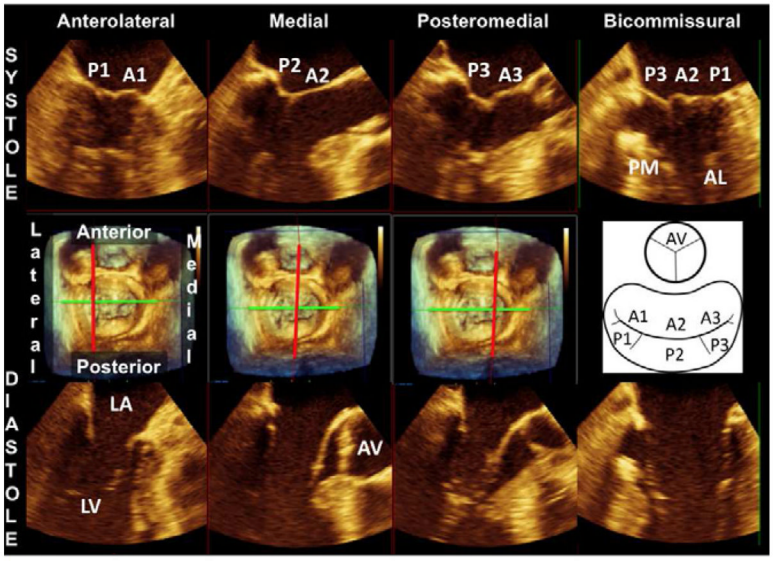

Figure 4.

The middle panel shows a 3D-rendered surgical view of the mitral valve. A schematic on the right side of the middle panel helps identify the mitral valve scallops and aortic valve in this view. The upper panel shows 2D Echo views of precisely known orientation because they are derived as slices of the 3D mitral valve apparatus in systole. The slice plane is indicated by the red line in the middle plane (perpendicular to a bicommissural axis, green line). The lower panel shows the same slice planes in diastole with the mitral leaflets open. (AL, anterolateral papillary muscle; AV, aortic valve; LA, left atrium; LV, left ventricle; PM, posteromedial papillary muscle)

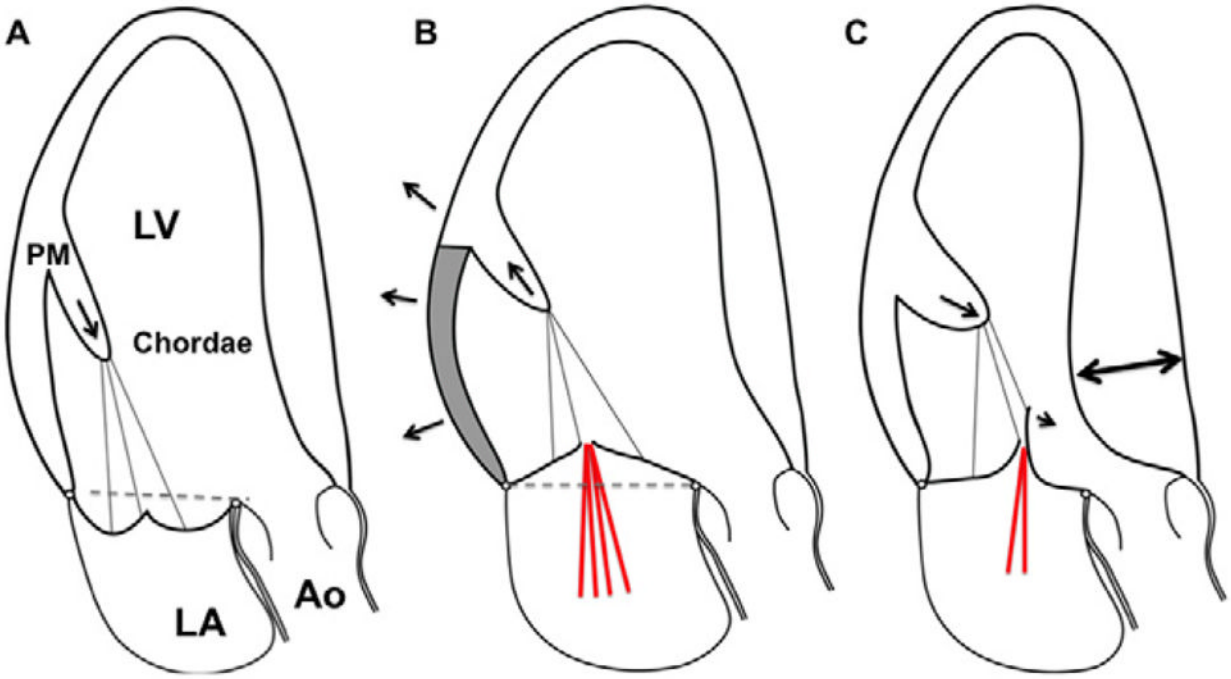

Figure 5.

A. Mitral valve prolapse: The schematic shows bileaflet mitral valve prolapse, with superior displacement of the papillary muscle tip, “tugged” by the leaflets, and excessive leaflet and chordal tissue and mobility. Leaflet coaptation is displaced into the left atrium superior to the annular plane (dashed line). B. Functional/ischemic mitral regurgitation: The papillary muscle (medial in inferior myocardial infarction) is displaced posteriorly, laterally and, to the extent allowed by the chords, apically (arrow) due to left ventricular local dilatation & remodeling (arrows) caused by MI (shaded area). The LV wall-PM displacement tethers the mitral leaflets apically and limits coaptation. There is 20 often not enough leaflet tissue to compensate for leaflet tenting (area apical to the dashed line), resulting in mitral regurgitation (red lines). C. Hypertrophic cardiomyopathy: The geometry of the left ventricle and papillary muscles is altered by myocardial hypertrophy (interventricular septum, double arrow). The papillary muscles are enlarged and displaced anteriorly (arrow) and closer to each other (not shown). This decreases intercommissural leaflet tension and moves the coaptation point and distal leaflets toward the left ventricular outflow tract. Like a sail catching a breeze, the distal anterior leaflet and/or posterior leaflet if elongated, is at risk of being displaced into the LV outflow tract by blood-flow drag. If anterior leaflet displacement is severe enough and posterior leaflet apposition restricted, mitral regurgitation will occur (red lines). (Ao, aorta; LA, left atrium; LV, left ventricle; PM, papillary muscle).

Table 1

Mitral annulus dimensions in normal human hearts and patients with dilated cardiomyopathy.

	Normal	**Dilated Cardiomyopathy**
Non-planar shape	++(4,5).	--(29,31)
Area (cm^2)	~7–12cm^2 (25–30)	~11–20cm^2 (27,29,30)
Circumference (cm)	7–11cm (35,129)	8–18cm (129)
% Area change diastole/systole	~ 20–42% (27–29,32,130)	13 – 23% (29,32)

Table 2

Mitral valve leaflet dimensions in healthy subjects, patients with dilated cardiomyopathy (CMP) and hypertrophic cardiomyopathy (HCM).

	Normal	**Dilated CMP**	**HCM**
Anterior leaflet area (cm^2)	4 – 7 (34,124,131,132)	7 (131)	6 – 14 (124,131,132)
Posterior leaflet area (cm^2)	2 – 3 (34,131,132)	2 (131)	3 – 7 (124,131,132)
Total leaflet area (cm^2)	9 – 15 (34,131,132)	12 – 20 (30,131)	13 – 21 (124,131,132)
Anterior leaflet length (mm)	18 – 24 (30,35,39,131,132)	24 (30,131)	22 (131,132)
Posterior leaflet length (mm)	11 – 14 (30,39,131,132)	13–14 (30,131)	14 (131,132)

CMP, cardiomyopathy; HCM, hypertrophic cardiomyopathy

Table 3

Mitral valve apparatus components in normal and diseased states

	Normal	Mitral Valve Prolapse	Functional/ischemic MR	Hypertrophic CMP
Papillary muscles	Parallel to the LV long axis	Superior traction	Apical/posterior/posterolateral displacement	Hypertrophied, anteriorly displaced, PM heads closer to each other
Chordae tendineae	Normal	Elongated, thick or thin, rupture-prone	Elongated, thick	
Leaflet area/length	Normal	Increased/Elongated	Increased/Elongated vs thick	Increased/Elongated in many
Mitral annulus shape/area	Saddle shaped/normal	Flattened/Normal - increased	Flattened/Increased	Saddle shaped/normal - decreased
Leaflet coaptation	At the annular level	At or superior to the annulus	Significantly apical to the annulus	Shifted towards the LVOT in SAM

CMP, cardiomyopathy; HCM, hypertrophic cardiomyopathy; LV, left ventricle, LVOT, left ventricular outflow tract; MR, mitral regurgitation PM, papillary muscles; SAM, systolic anterior motion;