The Mitral Valve and Mitral Regurgitation

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Abstract

An understanding of the normal anatomy, geometry and motion of the mitral valve is necessary to fully understand the pathophysiology of functional ischemic mitral regurgitation. The mitral valve apparatus is a dynamic structure with changes in its shape and geometry throughout the cardiac cycle. Six phases of the normal mitral valve motion and geometry can be identified.

Keywords

Mitral valve anatomy • Mitral valve motion • Mitral valve function • Mitral valve geometry • Mitral regurgitation

Structure of the Normal Mitral Valve

The mitral valve separates the left ventricle from the left atrium. It comprises two leaflets, the anterior and posterior leaflets, which are attached at their hinge ends to the mitral annulus, and at their free edge to chordae tendinae which in turn attach to papillary muscles [1] (Fig. 1.1). Some chordae tendinae, termed secondary chordae, attach to the body of the valve leaflets from papillary muscles, while others, termed tertiary chordae attach to the body of the valve leaflets directly from the mitral annulus and the left ventricle. The chordae tendinae at the free edge and body of the valve leaflets prevent excessive movement of the valve leaflets into the left atrium during left ventricular systole and are therefore essential for valve competency.

The papillary muscles are usually organized into two groups, the posteromedial and anterolateral papillary muscles, and are attached to the left ventricle wall approximately one third distance from the apex and two thirds from the annulus. The anterolateral papillary muscle usually attaches to the left ventricle at the junction between the septum and the posterior wall while the posteromedial papillary muscle usually attaches on the lateral wall of the left ventricle [1].

Each of the two mitral leaflets are further divided by two indentations at their free edges into three scallops, termed A1, A2 and A3 in the ante-

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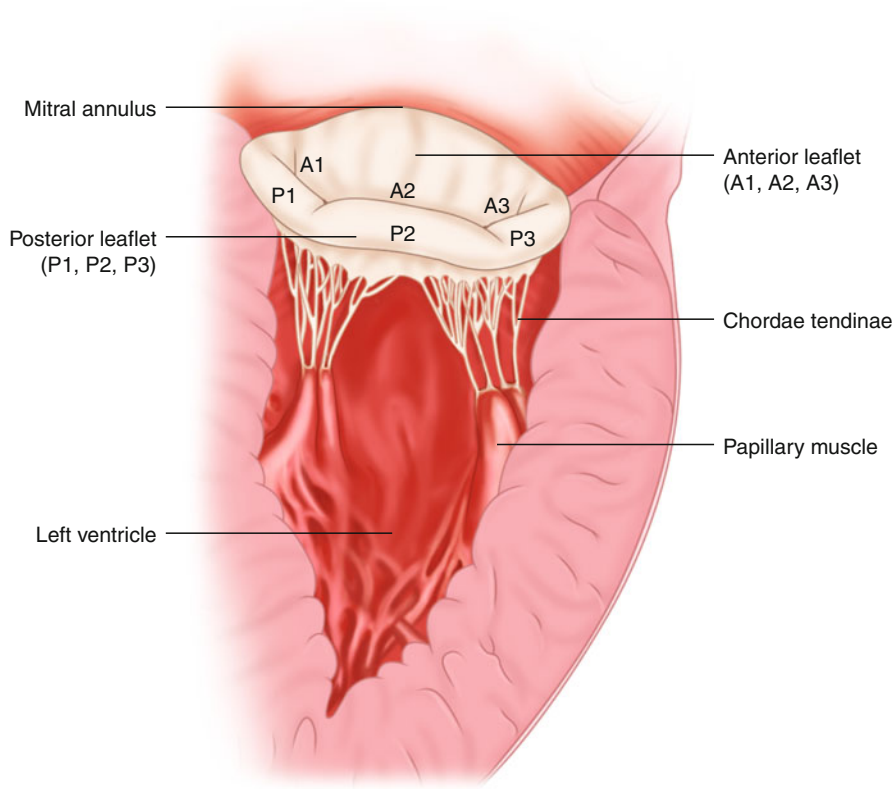

Fig. 1.1 The normal mitral valve (Adapted from Chan et al. [11]. With permission from Elsevier)

rior leaflet, and P1, P2 and P3 in the posterior leaflet (Fig. 1.1). The area between the two leaflets at the annulus is termed the commissures. A small commissural leaflet is present in this area which forms the continuity between the anterior and posterior leaflets. The anterior and posterior leaflets approximate and overlap each other at their free edges and commissures, forming a surface of coaptation of about 7–9 mm. This surface of coaptation between the free edges of the two leaflets is essential for valve competency. The normal coaptation line lies parallel to the posterior annulus [1].

Mitral Valve Motion and Geometry

The normal motion and geometry of the mitral valve has been previously studied mainly using radio-opaque markers in animals and also by echocardiography [2–5]. More recently, Chan, et al., used cardiovascular magnetic resonance to study the motion and geometry of the normal mitral valve apparatus in normal healthy volunteers; the results of this study are described here [6]. Six phases of mitral annular and leaflet motion can be identified during the normal cardiac cycle (Figs. 1.2 and 1.3):

Phase I: Ventricular systolic excursion
Phase II: Leaflet opening and annular recoil
Phase III: Leaflet approximation
Phase IV: Mid diastolic pause
Phase V: Atrial systolic excursion
Phase VI: Leaflet closure

Left ventricular systole starts in Phase VI with leaflet closure and occurs throughout Phase I. Left ventricular diastole starts in Phase II with leaflet opening, continues throughout Phases III to V, and ends in Phase VI with leaflet closure.

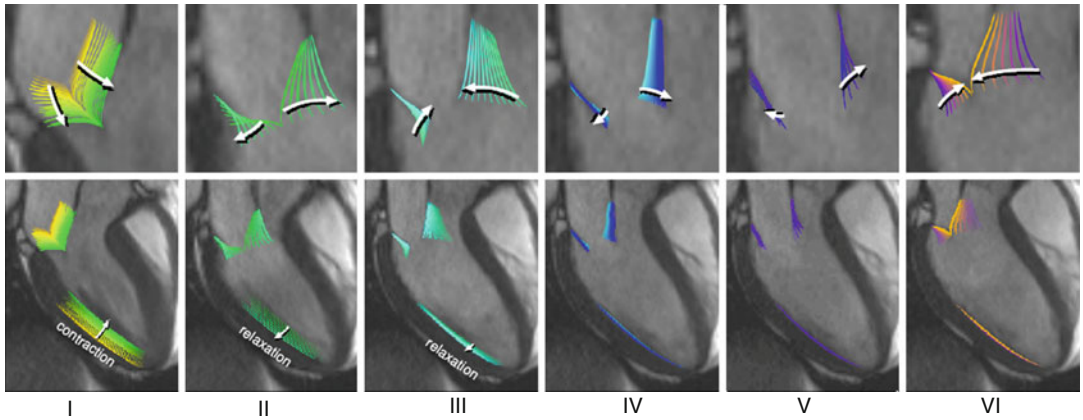

Fig. 1.2 The six phases of mitral annular and leaflet motion during the cardiac cycle in a normal individual. The motion of the mitral valve leaflets, annulus and left ventricle at its inferolateral wall were traced from individual cardiovascular magnetic resonance cine images and superimposed on each other to produce the image. Phase I represents left ventricular systole, Phases II–V represent left ventricular diastole, Phase VI represents the end of left ventricular diastole and the onset of left ventricular systole (Reprinted from Chan, et al. with permission. From Chan et al. [6])

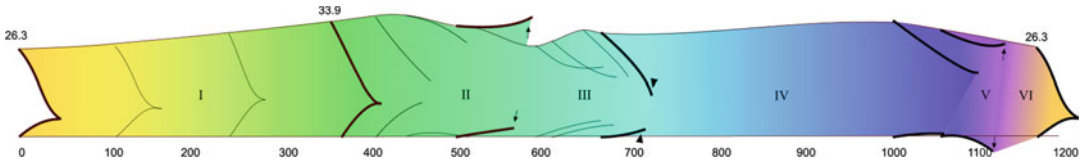

Fig. 1.3 "Two-dimensional M-Mode" display of mitral annular and leaflet motion during the cardiac cycle in a normal individual. The figure was produced by superimposing and tracing the images from individual cardiovascular magnetic resonance cine images. The view is that of the septo-lateral dimension of the mitral annulus. Phase I represents left ventricular systole, Phases II–V represent left ventricular diastole, Phase VI represents the end of left ventricular diastole and the onset of left ventricular systole. Arrows indicate maximal early and late diastolic opening of the leaflet tips, and arrow heads the diastolic leaflet approximation. Horizontal axis represents time in msec. Numbers at the top is the septo-lateral diameter in mm (From Chan et al. [6])

Phase I: Ventricular Systolic Excursion

Left ventricular systole occurs in this phase and as the left ventricle contracts, the mitral annulus moves towards the left ventricular apex. This excursion of the mitral annulus towards the left ventricular apex is asymmetrical with greater excursion of the posterior annulus and leaflets compared to the anterior annulus and leaflets (Fig. 1.2) [6]. The septo-lateral diameter of the mitral annulus increases in size during this phase but the commissure-commissure diameter remains relatively unchanged in size. The mitral leaflets remain approximated throughout this phase. During this phase of left ventricular systole, the papillary muscles contract, becoming shorter in length, and also approximate medially towards each other, towards the mitral annulus, and towards the left ventricular septum. The direction of greatest approximation is towards each other. Although both the papillary muscles and the mitral annulus move towards each other during this phase, contraction of the papillary muscles with corresponding reduction in their length means that the distance between the papillary muscle tips and the mitral annulus is relatively unchanged. Therefore, during this phase of left ventricular systole, both the anterolateral and posteromedial papillary muscles approximate towards each other and towards the long-axis midline of the left ventricle, and the anterolateral papillary muscle approximates towards the left ventricular septum. The distance between the tips of the papillary muscles and the mitral annulus

remains relatively constant throughout this phase despite the mitral annulus and the left ventricular wall moving towards each other, accounted for by the 30–40 % contraction in its length.

Phase II: Leaflet Opening and Annular Recoil

This phase corresponds to the end of left ventricular systole and the start of left ventricular diastole. At the start of this phase, the mitral leaflets open and this is followed closely by recoil of the mitral annulus back towards the left atrium (Figs. 1.2 and 1.3). The septo-lateral diameter of the mitral annulus continues to increase in size reaching its maximal size in the middle of this phase, corresponding to maximal separation of the mitral leaflets. The left ventricle starts to relax and the papillary muscles move apart from each other, away from the long axis midline of the left ventricle and from the left ventricular septum.

Phase III: Leaflet Approximation

This phase occurs during left ventricular diastole. During this phase, the mitral leaflets move passively to a partially closed position (Figs. 1.2 and 1.3).

Phase IV: Mid Diastolic Pause

Left ventricular diastole continues throughout this phase. The mitral leaflets drift about in a neutral position during this phase. This represents the phase of diastolic inactivity and is the true neutral position of the heart.

Phase V: Atrial Systolic Excursion

Left ventricular diastole continues throughout this phase. Left atrial systole pulls the mitral annulus back towards the left atrium from its neutral position. The separation of the mitral leaflet edges increases (Figs. 1.2 and 1.3). The annulus begins to contract (pre-systolic contraction) and its septo-lateral diameter starts to decrease in size during this phase.

Phase VI: Leaflet Closure

This phase marks the end of left ventricular diastole and the onset of left ventricular systole. The leaflets close. The annulus continues to contract and reduce in size at its septo-lateral dimension.

The ability of cardiovascular magnetic resonance to image the entire mitral valve apparatus (mitral leaflets, annulus and papillary muscles) and the left ventricle, and to visualise these together in a single image, has given unique insights into the function, motion and geometry of the mitral valve apparatus. The entire mitral valve apparatus changes in size, shape and position during the cardiac cycle.

Mitral annular contraction begins as left atrial systole begins (in Phase V). As the annulus is pulled towards the left atrium with left atrial contraction, it also contracts and reduces in size in its septo-lateral dimension. It reaches its smallest size just before the onset of left ventricular systole when leaflet closure occurs (in phase VI and the beginning of phase I). At this stage, the annulus has reduced in size by about 15 % from its size in the middle of left ventricular diastole (Phase IV) [6]. This helps to increase the coaptation between the mitral leaflets as left ventricular systole begins. The annulus then starts to relax as left ventricular systole begins, and throughout left ventricular systole (Phase I), the annulus continues to increase in size in its septo-lateral dimension by about 15 %, so that its diameter is maximal during leaflet opening (Phase II) (Fig. 1.3). The annulus also moves longitudinally along the long axis of the heart. Throughout left ventricular systole (Phase I), the annulus moves towards the apex of the left ventricle (Fig. 1.2). At the end of left ventricular systole, the annulus recoils back towards the left atrium (Phase II), towards its neutral position. This movement actually helps to pull the mitral leaflets further apart so that it achieves its maximal separation distance at this time (Figs. 1.1 and 1.2). The annulus

then stays in a relaxed state (Phase III and IV) until left atrial systole pulls it away from its neutral position towards the left atrium (Phase V). This movement helps to separate the mitral leaflets again. The annulus starts to contract again at this stage and its septo-lateral diameter reduces in size again until leaflet closure (Phase VI).

Several other studies have been done on mitral annular motion and function, mainly using radio-opaque markers in animal models and echocardiography. Most studies also report a pre-systolic mitral annular contraction, consistent with that reported by Chan et al. [2–5]. Ormiston et al. using transthoracic echocardiography, and Glasson et al. using radio-opaque markers in sheep hearts reported very similar results [2, 3]. There is, however, some variation in the precise changes in mitral annular size during the cardiac cycle in some studies, which may be related to differences in the imaging and analysis techniques used, and different definitions of the onset of systole and diastole. Many studies also did not analyse mitral annular size throughout the cardiac cycle, but only performed measurements at fixed time intervals, and some only measured the maximal and minimal size of the annulus. Transthoracic echocardiography, unlike cardiovascular magnetic resonance, is unable to precisely image the mitral annulus in the same position consistently between different patients, and does not always image the mitral annulus in a position which would enable accurate and reproducible measurements of its true dimensions, due to the orientation and direction of placement of the probe on the patient's chest.

The mitral leaflets show distinct motion throughout the cardiac cycle and its movement is influenced by the motion of the mitral annulus, and also by the flow of blood around it. The leaflets remain approximated throughout left ventricular systole (Phase I and VI) and separates during left ventricular diastole (Phases II–V) (Fig. 1.2). Separation of the leaflets is maximal at early diastole (Phase II) and occurs when the annulus recoils back towards the left atrium following apically directed systolic excursion in Phase I (Fig. 1.3). They then approximate to a partially closed position in the middle of diastole (Phase III) (Figs. 1.2 and 1.3). The leaflets then drift about the left ventricle in its neutral position during a period of diastolic inactivity (Phase IV). The leaflets open fully again towards the end of diastole when left atrial systole pulls the mitral annulus (and the base of the leaflets) towards the left atrium (Phase V) (Figs. 1.2 and 1.3). Finally, the leaflets close at the end of left ventricular diastole and the onset of systole (Phase VI) (Figs. 1.2 and 1.3).

The normal function of the papillary muscles is necessary to maintain mitral valve competency during left ventricular systole. The mitral annulus and the left ventricle supporting the papillary muscles move towards each other during left ventricular systole. To achieve mitral valve competency and prevent leaflet prolapse, it is necessary for the papillary muscles to contract and shorten in length so as to maintain an approximately constant distance between the tips of the papillary muscles and the mitral annulus during left ventricular systole. The papillary muscle length shortens by 30–40 % with maintenance of an approximately constant length between the tips of both papillary muscles and the mitral annulus.

The papillary muscles approximate towards each other and towards the long-axis midline of the left ventricular chamber during left ventricular systole. This realignment of the papillary muscles towards each other during left ventricular systole may be important to allow adequate coaptation between the mitral leaflets by avoiding excessive tension in the papillary muscles and chordae tendinae. In this regard, the position of the papillary muscles is dependent on both left ventricular size and function.

Mechanisms and Causes of Mitral Regurgitation

Mitral regurgitation refers to the backflow of blood from the left ventricle into the left atrium during left ventricular systole. It can occur with normal leaflet motion, so called Type I dysfunction, due to annular dilatation (e.g., due to atrial fibrillation) which pulls the leaflets apart

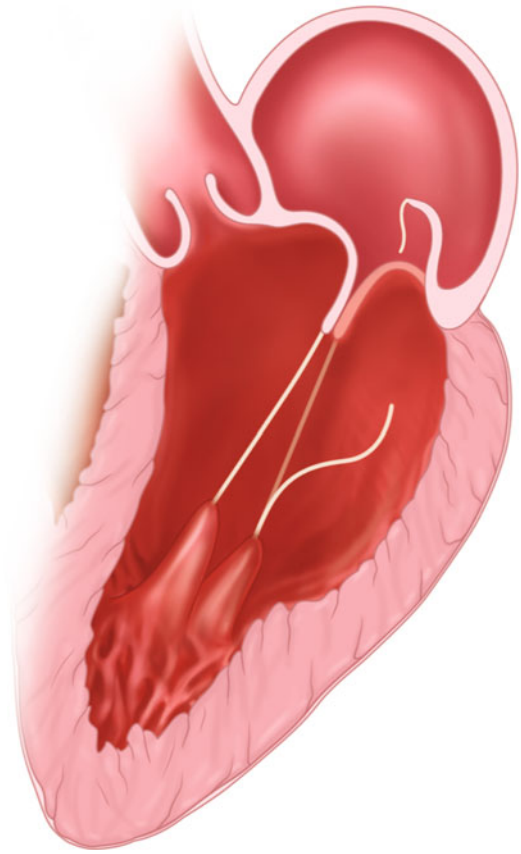

Fig. 1.4 Mitral regurgitation due to chordal rupture (Type II dysfunction) (Adapted from Amirak et al. [12]. With permission from Elsevier)

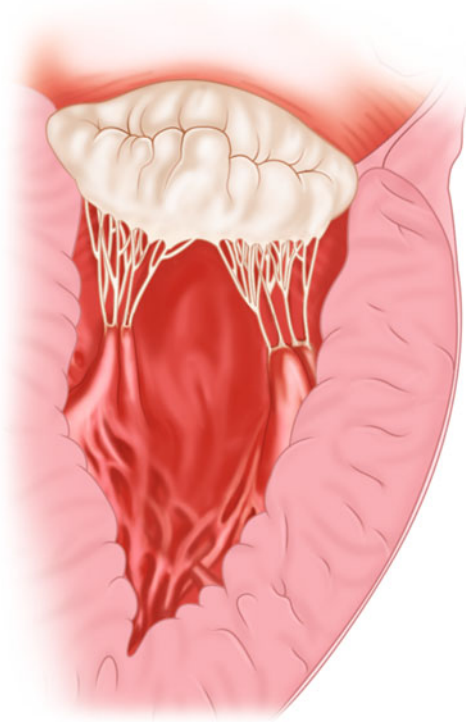

Fig. 1.5 Mitral regurgitation due to excessive leaflet tissue in Barlow's disease (Type II dysfunction) (Adapted from Amirak et al. [2]. With permission from Elsevier)

preventing adequate leaflet coaptation during left ventricular systole and resulting in mitral regurgitation, or to leaflet tear or perforation (e.g., from endocarditis), or to vegetations from endocarditis preventing adequate leaflet coaptation [1].

More commonly, mitral regurgitation occurs due to excessive leaflet motion or leaflet prolapse into the left atrium during left ventricular systole, so called Type II dysfunction. The resulting lack of leaflet coaptation results in mitral regurgitation. This typically occurs due to chordal rupture or elongation from degenerative valve disease or endocarditis, excessive leaflet tissue from Barlow's or other connective tissue diseases, or papillary muscle rupture from myocardial infarction (Figs. 1.4 and 1.5) [1].

Mitral regurgitation can also occur due to restricted leaflet motion, so called Type III dysfunction. This is seen in rheumatic valve disease where thickened and fused leaflets limit leaflet motion and prevent adequate leaflet coaptation resulting in mitral regurgitation. It can also occur due to dilatation and dysfunction of the left ventricle following myocardial infarction or due to dilated cardiomyopathy. In these cases, the dilated or poorly contracting left ventricle pulls the mitral valve leaflets apart through their attachments to the chordae tendinae and papillary muscles, preventing adequate leaflet coaptation and resulting in mitral regurgitation (Figs. 1.6 and 1.7). This type of mitral regurgitation is also termed functional mitral regurgitation as the mitral valve apparatus is normal in structure but its function is abnormal secondary to the effects of a dilated or dysfunctional left ventricle. It will be discussed in more detail in Chap. 2.

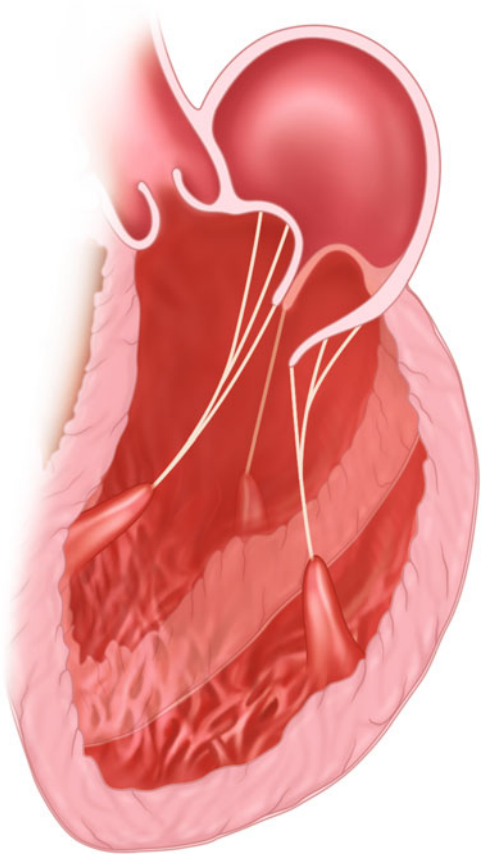

Fig. 1.6 Mitral regurgitation due to posterior leaflet restriction or tethering in functional ischaemic mitral regurgitation (Type III dysfunction). Left ventricular remodelling occurs following an inferior myocardial infarction. The resulting dilatation and displacement of the left ventricle wall tethers or restricts the posterior mitral valve leaflet during left ventricular systole preventing adequate leaflet coaptation and resulting in mitral regurgitation (Adapted from Chan et al. [11]. With permission from Elsevier)

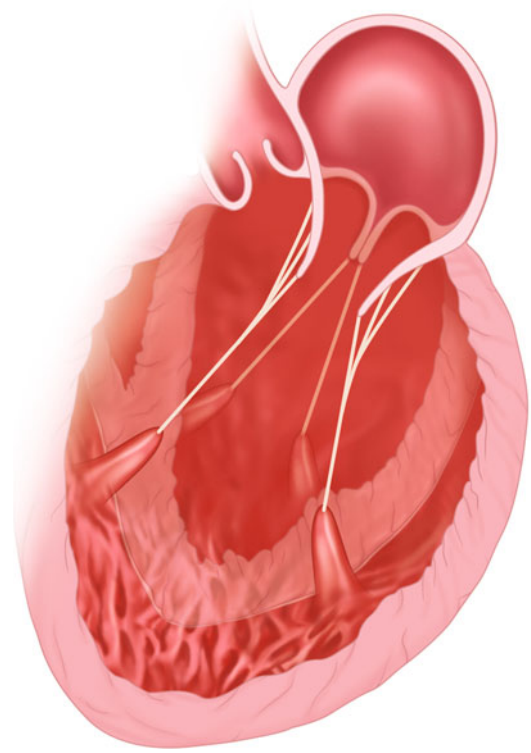

Fig. 1.7 Mitral regurgitation due to restriction or tethering of both anterior and posterior leaflets in functional mitral regurgitation due to dilated cardiomyopathy (Type III dysfunction). Global left ventricular dilatation and dysfunction tethers or restricts both mitral valve leaflets during left ventricular systole preventing adequate leaflet coaptation and resulting in mitral regurgitation (Adapted from Chan et al. [11]. With permission from Elsevier)

Consequences of Mitral Regurgitation

Mitral regurgitation results in an increase in preload and a decrease in afterload due to the backflow of blood into the left atrium. To maintain forward stroke volume, the left ventricle adapts by dilating in order to increase forward cardiac output with each systolic contraction; eccentric left ventricular hypertrophy occurs with the laying down of extra sarcomeres in series [7, 8]. This is also triggered as a result of increased left ventricular wall stress due to the increased preload. Geometrical changes also occur in the left ventricle which becomes more spherical. The left atrium enlarges as a result of the regurgitant flow into it. The enlargement of the left atrium results in left atrial pressures remaining normal or only being slightly elevated. As a result, pulmonary oedema is avoided and significant increases in pulmonary vascular resistance seldom occur. During this period of compensation, the patient may remain entirely asymptomatic but the heart is getting larger [9, 10].

After a period of compensation, with progressive left ventricular dilatation, contractile dysfunction occurs with increased myocyte length and decreased myofibril content. Consequently, left ventricular systolic contractility becomes

progressively impaired as mitral regurgitation progresses. However, the calculated ejection fraction and stroke volume may still remain normal despite impaired left ventricular contractility, because the calculated ejection fraction and stroke volume includes the backflow of blood into the left atrium. The forward stroke volume will be decreased with progressive impairment of left ventricular contractility. Pulmonary congestion eventually ensues [9, 10].

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