The Mitral Apparatus

Functional Anatomy of Mitral Regurgitation

By Joseph K. Perloff, M.D., and William C. Roberts, M.D.

SUMMARY

This review deals with the functional anatomy of the six components of the mitral apparatus, namely, the left atrial wall, annulus, leaflets, chordae tendineae, papillary muscles, and left ventricular wall. Each component is considered individually, in the context of the apparatus as a whole, in relation to the mode of closure of the normal mitral valve, and in the light of many acquired and congenital disorders that disturb the harmony of the finely coordinated mitral mechanism and render it incompetent.

The left atrium is related to mitral valve competence in terms of contraction and relaxation and in terms of dilatation of its posterior wall. The annulus not only serves as a fulcrum for the leaflets but exhibits sphincteric contraction in systole that decreases the size of the orifice. The two leaflets differ in shape but are nearly identical in area, and together are about two and one half times the area of the orifice that they are required to close. Leaflet abnormalities causing acquired or congenital mitral regurgitation result from deficient leaflet tissue, excessive leaflet tissue, or restricted leaflet mobility. Chordae tendineae are considered according to their leaflet attachments, ventricular attachments, thicknesses, lengths, and arborization patterns. Mitral regurgitation due to chordal abnormalities results from chordae that are abnormally long, abnormally short, ectopically inserted, or ruptured. In this context, systolic clicks and late systolic murmurs are discussed, and severe acute mitral regurgitation is contrasted with severe chronic mitral regurgitation. The papillary muscles and the left ventricular wall represent the two muscular components of the mitral apparatus. An appraisal of papillary muscle dysfunction includes dysfunction with loss in continuity (rupture) and dysfunction without loss in continuity (fibrosis, ischemia, replacement). Finally, the role of altered left ventricular shape is discussed in the context of mitral regurgitation, and the effect of dilatation is ascribed chiefly to alterations in the position of papillary muscles and their directional axes of tension.

Additional Indexing Words:
Left atrial wall Mitral annulus Leaflets Chordae tendineae
Papillary muscles Left ventricular wall Systolic clicks
Late systolic murmurs

Our current conceptions as to the dynamic consequences of a leaking mitral valve . . . are still at variance. It is pertinent, therefore to discuss a few of the supposed consequences of mitral regurgitation.—Carl J. Wiggers, 1922

THE MITRAL APPARATUS is a complex, finely coordinated mechanism that can be deranged by a multiplicity of acquired and congenital disorders and requires for its competence the functional integrity of six anatomic elements working in delicate concert. These anatomic elements are: (1) posterior left atrial wall, (2) annulus, (3)

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leaflets, (4) chordae tendineae, (5) papillary muscles, and (6) left ventricular wall.

The exact mode of closure of the normal mitral valve is still uncertain.\(^2\)\(^,\)\(^3\) Left ventricular systole begins with contraction of the papillary muscles. The vertical forces exerted by the contracting papillary muscles move the leaflets into apposition. As the intraventricular pressure rises, the free edges of the cusps firmly coapt, mutually supporting each other along a comfortable margin of their atrial surfaces, and firmly sealing the orifice. The remainder of each leaflet bulges like a parachute toward the left atrium.\(^4\) The annulus not only serves as a fulcrum for the leaflets, but during ventricular systole decreases its circumferential size, thus reducing the area that the leaflets are required to bridge.\(^5\)\(^,\)\(^6\) Even so, the surface area of the leaflets is about two and one half times the area of the orifice, thus providing a comfortable reserve. As the left ventricle ejects, its apex and the mitral orifice approach each other. Shortening of the vertical axis of the left ventricle is accompanied by synergistic contraction of the papillary muscles and adjacent left ventricular wall so that an appropriate vertical anchoring force is applied to the chordae tendineae, that prevents eversion of the leaflets. Let us now turn attention to the six anatomic components of the mitral apparatus which when faulty can disturb the harmony of the valvular mechanism and render it incompetent. Coordinated interaction among these elements—which are interdependent—usually means that more than one, or even several, are deranged at any one time although with varying degrees of importance.

**Left Atrial Wall**

Two contributions of the left atrium have been related to competence of the mitral valve: (1) contraction and relaxation\(^7\)\(^,\)\(^8\) and (2) atrial dilatation.\(^4\) Even recently, it has been affirmed that atrial contraction and relaxation result in mitral valve closure in man.\(^8\) Contractility rather than flow has been considered the chief determinant of mitral valve closure as a result of atrial activity.\(^7\) Vigorous atrial systole followed by a rapid decline in atrial pressure has been held responsible for substantial changes in the rate of development of ventriculoatrial pressure gradients before the onset of ventricular systole.\(^7\) Although atrial contraction and relaxation seem capable of closing the mitral valve in man, the absence of such activity does not cause mitral regurgitation.\(^9\) Competence of the mitral apparatus depends in part on a normal sequence of ventricular activation (see below) but not on a normal sequence of atrial and ventricular contraction. Loss of effective atrial contraction (atrial fibrillation, complete heart block, and others) does not necessarily result in regurgitation.\(^9\)

Left atrial enlargement itself can contribute to mitral regurgitation\(^4\) (fig. 1). Dilatation of the left atrium does not affect the anterior leaflet since that leaflet is anchored to the root of the aorta.\(^4\) The posterior leaflet, however, can be directly affected\(^4\) (figs. 1 and 2). As the left atrium enlarges, its posterior wall is displaced posteriorly and downward. Because of the continuity of the atrial endocardium and posterior mitral leaflet, this displacement exerts tension on the posterior leaflet.\(^4\) The displacement may prevent that cusp from contacting its mate or may aggravate pre-existing leaflet malapposition. Thus, as mitral regurgitation provokes left atrial enlargement,

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*Figure 1*

As the left atrium dilates, its posterior wall tends to hang over the atrioventricular sulcus. The dilatation pulls the posterior mitral leaflet cephalad since this leaflet is continuous with the left atrial endocardium.
Mitral Apparatus

The annulus forms an important part of the basal attachment or fulcrum of the mitral leaflets. A traditional point of view states that if the atrioventricular ring dilates sufficiently, the cusps are unable to meet and mitral regurgitation results. This explanation is an oversimplification. Left ventricular dilatation—an undoubted cause of mitral incompetence—may exert its effects independently of dilatation of the mitral ring (see below). Nevertheless, the size of the ring does play a role in preserving competence of the mitral valve, albeit a role that relates less to dilatation than to failure to decrease its circumference during systole. The tissue at the basal attachments of the mitral leaflets is tough but pliable. Toughness opposes dilatation; pliability permits sphincter-like contraction of the annulus during systole, which reduces the area that the apposing leaflets must bridge by an estimated 20 to 50%. Left ventricular dilatation may exert an unfavorable effect on the annulus chiefly by applying a distending pressure that opposes systolic annular contraction. With calcification of the annulus—in itself a cause of mitral regurgitation—the mechanism of regurgitation is believed to stem from a loss of sphincteric action of the basal attachments of the mitral cusps. In such patients, calcific deposits commonly coexist on the aortic surfaces of otherwise normal trileaflet aortic valves; the resulting obstruction to left ventricular outflow augments incompetence associated with the calcified mitral ring. Calcification of the annulus is found not only in the elderly but occasionally in young patients with the Marfan syndrome and represents one of the two organic causes of mitral regurgitation in such individuals (see below). In addition, mitral annular calcification also occurs in children with Hurler’s syndrome.

Leaflets

Proper closure of the leaflets represents an ultimate goal of the entire mitral mechanism. The areas of the two leaflets are nearly identical, but the basal to free edge length of the anterior leaflet is two or more times that of the posterior (fig. 2). Consequently, the anterior leaflet is more mobile, while the posterior leaflet fulfills a secondary or supporting role. Although the mitral leaflets, like other cardiac valves, have been looked upon as passive connective tissue structures, recent investigations have found within their substance striated muscle bundles to which an active function has been assigned. It is uncertain whether such muscle fibers have physiologic significance in promoting closure of the valve.

Leaflet abnormalities can be acquired or congenital, with regurgitation resulting from deficient leaflet tissue, excessive leaflet tissue, or restricted leaflet mobility. Inadequate leaflet tissue results from a number of causes,
the best known of which is rheumatic endocarditis which provokes scarring and contractures. Clefts which penetrate a leaflet from its free edge can be acquired (infective endocarditis) or congenital (cleft anterior leaflet of endocardial cushion defect). Acquired clefts lack chordal support so that mitral regurgitation is obligatory. Congenital clefts differ in that chordae tendineae attach to their margins and can offer support that is adequate for valve competence, or if too short, can restrict mobility of the cleft leaflet and cause regurgitation. Inadequate leaflet tissue can also result from perforations or holes within a leaflet remote from its free edge. Such perforations are typically acquired (infective endocarditis). Double-orifice mitral valve is, strictly speaking, not a congenital perforation, but rather two mitral valves equipped with supporting chordae.

Excessive leaflet tissue may result in a hoodlike deformity in which the unsupported segment of leaflet (ruptured chordae tendineae or abnormally long chordae) balloons upward as ventricular systolic pressure is exerted against its undersurface. Perhaps akin to interchordal hooding is the disorder known as the billowing or floppy mitral valve or the systolic click-late systolic murmur syndrome.

An occasional patient exhibiting this disorder has complete or partial stigmata of the Marfan syndrome with fibromyxomatous changes in valve tissue, marked redundancy of leaflets, and elongated chordae (fig. 3). More recently, systolic clicks with the late systolic murmur have been found in patients with isolated thoracic bony abnormalities. As a rule, however, the patient, usually female, otherwise judged as normal presents with systolic clicks and a late systolic murmur, and on angiography or echocardiography shows billowing mitral cusps, generally posterior, and relatively mild late systolic regurgitation. In the latter half of ventricular systole the redundant leaflet, poorly anchored by its

**Figure 3**

Opened left heart of a 2-day-old infant with features of the Marfan syndrome and auscultatory evidence of mitral regurgitation. (Left) Opened aorta, aortic valve, and left ventricle. The wall of the aorta was abnormal and consistent with cystic medial necrosis. The aortic valve appears normal. (Right) Opened dilated left atrium, mitral valve, and dilated left ventricle. The mitral leaflets are floppy, i.e., elongated in both longitudinal and transverse dimensions.

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long chordae, becomes slack and prolapses into the left atrium. A late systolic murmur and occasionally bizarre musical late systolic whoops or honks \(^{24}\) may accompany late systolic regurgitation.

Osler’s account of the systolic whoop or honk is apt. \(^{25}\) He wrote, “As she [a 12-year-old girl] sits upright in the chair, the heart sounds at apex and base loud and clear; no murmur. When she stands, a loud systolic murmur is heard at apex, high-pitched, somewhat musical. . . . It disappeared quite suddenly, and could not be detected on most careful examination. The child then suggested that she heard it most frequently when in the stooping posture; and on causing her to lean forward and relax the chest, the murmur was at once heard, and with greatly increased intensity.”

Clicks, single or multiple, occur alone, may introduce the murmur, may be intermittent, persistent, or absent, and are believed to be due to tension of the elongated chordae (chordal snaps) or the billowing leaflet. \(^{20}\) Migration of the click (or clicks) toward the first heart sound is known to occur during inspiration and after amyl nitrite inhalation. \(^{26}\) Recently, persistent early timing, i.e., early systolic clicks due to mitral valve prolapse, has been identified. \(^{27}\) When such early clicks introduce mitral regurgitation, the accompanying murmur \textit{sounds} holosystolic although a small gap exists between first heart sound and click. When a pressor agent is administered to subjects with isolated systolic clicks, the clicks move toward the second sound, and the rise in left ventricular systolic pressure occasionally produces a late systolic murmur presumably identifying the clicks as intracardiac and associated with potential mitral regurgitation. \(^{28}\) Accordingly, subjects with clicks alone, clicks and a late systolic murmur, or a late systolic murmur alone are at risk of infective endocarditis \(^ {29} \) and should be so managed even though some systolic clicks may prove to be extracardiac. Patients with clicks and late systolic murmurs often have abnormal electrocardiograms, especially T-wave inversions in diaphragmatic leads. \(^ {20,21} \)

These auscultatory and electrocardiographic patterns are occasionally familial. \(^ {20} \) An increased incidence of ectopic rhythms \(^ {30} \) and an unknown incidence of sudden death occur in such patients. \(^ {20} \) One who died suddenly had a markedly redundant posterior leaflet and thin elongated chordae at necropsy. \(^ {29} \) Recently systolic clicks and late systolic murmurs or whoops with billowing posterior leaflets have been found in ostium secundum atrial septal defect \(^ {24,31} \) and in Ebstein’s anomaly of the tricuspid valve. \(^ {32} \) In addition, an abnormality of the inferior wall of the left ventricle has been proposed as a contributory factor in rendering the posterior leaflet slack. \(^ {21} \) It is not known whether the bifid apical systolic impulse in these patients relates to such an abnormality of left ventricular wall, to abrupt mid systolic decompression of the left ventricle by the prolapsing mitral leaflet, or to an unidentified cause. \(^ {33} \)

Not all whoops or honks are confined to late systole, and the etiology is not always a floppy mitral leaflet. \(^ {24} \) Furthermore, late systolic murmurs without clicks occasionally occur in rheumatic mitral regurgitation, \(^ {34} \) and systolic clicks may introduce the late systolic murmur of papillary muscle dysfunction in ischemic heart disease \(^ {35} \) (see below).

Mitrail regurgitation due to restricted leaflet mobility has already been related to abnormally short chordae. Peculiarities of mitral commissural fusion following rheumatic endocarditis also keep the leaflets apart and contribute to incompetence, \(^ {4} \) but most patients with fused commissures have some degree of stenosis as well. In pure rheumatic mitral regurgitation, the chordae tendineae are generally not shortened. At other times, leaflet mobility may be restricted by abnormal attachments to the endocardium of the left ventricle. Fusion of the posterior cusp to the underlying ventricular endocardium has been related to organization of vegetations of infective endocarditis located in the angle formed by the posterior leaflet and left ventricular wall. \(^ {4} \) Occasionally leaflet motion may be restrained because of the abnormal directional pull exerted by chordae tendineae.
with ectopic ventricular attachments. Another type of leaflet restriction accompanies congenitally corrected L-transposition of the great vessels (ventricular inversion). In this anomaly, the mitral orifice is equipped with a tricuspid valve (inversion, i.e., right-to-left interchange). The left-sided tricuspid valve is often the site of an Ebstein-like anomaly in which the basal portion of a leaflet is attached to the left ventricular endocardium. Restricted cusp mobility is a chief cause of mitral regurgitation in this anomaly.

**Chordae Tendineae**

Abnormally long chordae, abnormally short chordae, and ectopically inserted chordae have been discussed. This section deals with ruptured chordae.

Chordae tendineae must be considered according to their leaflet attachments, their ventricular attachments, and their individual thicknesses, lengths, and arborization patterns. Several different orders of chordae are recognized. The physiologic consequences and clinical signs of rupture depend to a large extent on where in the chordal pattern discontinuity occurs.

The majority of chordae that attach to the leaflets form a fine network of delicate threads that spring from larger chords near the papillary muscles (fig. 4). Some chords run as single nonbranching bundles of collagen from papillary muscle to leaflet. Each papillary muscle gives rise to chordae that cross to and support both anterior and posterior cusps (fig. 4). The etiology of chordal rupture is often unclear (spontaneous). Infective endocarditis is one established cause, but rupture of chordae tendineae is not a complication of myocardial infarction. Rupture of one or

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**Figure 4**

*Schematic diagrams. (Left upper) Showing that the posterior (P-M) and anterior (A-M) papillary muscles each gives rise to branching chordae that lend support to both anterior and posterior leaflets. (Left lower) Showing the posterior papillary muscle with its multiple heads. P.M.L. = posterior mitral leaflets, L.A. = left atrium, L.V. = left ventricle. (Right and center) Showing that rupture of the central body (entire trunk) results in overwhelming regurgitation, and rupture confined to one head of a papillary muscle results in regurgitation analogous to that following rupture of major chords.*

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Opened left atrium (a) and opened left heart (b) of a 37-year-old woman who developed sudden severe mitral regurgitation 7 months earlier. The left atrium is of normal size. A club-shaped ruptured chorda tendinea (arrows) from the anterior mitral leaflet was responsible for acute severe mitral regurgitation. The nonruptured chordae are normal. The chest X-rays show cardiomegaly due to left ventricular dilatation, but the left atrium is not enlarged. The left atrial v wave was 52 mm Hg.

more chordae results in acute loss of leaflet support and in the abrupt onset of appreciable mitral regurgitation\(^{37,38}\) (fig. 5). Nevertheless, in contrast to papillary muscle rupture (see below) patients with chordal rupture tend to survive for months\(^{38}\) or even years.\(^{39}\) Such
patients present in sinus rhythm with a history of sudden heart failure and are found to have a decrescendo apical systolic murmur that tends to radiate to the left sternal edge and base; both third and fourth heart sounds are present with presystolic distention of the left ventricle, physical and electrocardiographic signs of pulmonary hypertension, but little or no radiologic evidence of left atrial enlargement ($^{37,38}$) (fig. 5). The key to the physiologic disturbance and clinical picture is the sudden development of severe regurgitation into a small left atrium-high pressure left atrial enlargement, and the right atrium and ventricle are normal. When severe mitral regurgitation appears suddenly in individuals with previously normal or near normal hearts, the left atrium (LA) is relatively small, and the high pressure within it is transmitted into the pulmonary vessels and the right ventricle (RV). The anatomic indicator of these physiologic events is severe hypertrophy of the left atrial and right ventricular walls and marked intimal proliferation and medial hypertrophy of the pulmonary veins (PV) and arteries (PA). (Lower panel) Severe mitral regurgitation is also present, but the regurgitation is chronic. The left atrium is enormously dilated and is therefore capable of "absorbing" the regurgitant flow without elevating the pressure in the pulmonary vessels and right heart. Consequently, there is no intimal proliferation or medial hypertrophy of the pulmonary veins or arteries and the right ventricular wall is not hypertrophied. PT = pulmonary trunk.

**Figure 6**

Diagrammatic representation of the two extremes of severe mitral regurgitation, i.e., acute, with small high-pressure left atrium, and chronic with large low-pressure atrium. (Upper panel) When severe mitral regurgitation appears suddenly in individuals with previously normal or near normal hearts, the left atrium (LA) is relatively small, and the high pressure within it is transmitted into the pulmonary vessels and the right ventricle (RV). The anatomic indicator of these physiologic events is severe hypertrophy of the left atrial and right ventricular walls and marked intimal proliferation and medial hypertrophy of the pulmonary veins (PV) and arteries (PA). (Lower panel) Severe mitral regurgitation is also present, but the regurgitation is chronic. The left atrium is enormously dilated and is therefore capable of "absorbing" the regurgitant flow without elevating the pressure in the pulmonary vessels and right heart. Consequently, there is no intimal proliferation or medial hypertrophy of the pulmonary veins or arteries and the right ventricular wall is not hypertrophied. PT = pulmonary trunk.
previously normal, small, and therefore relatively noncompliant left atrium (figs. 5 and 6). The steeply rising left atrial v wave approaches left ventricular pressure in the latter part of systole, resulting in late systolic decline in both regurgitant flow and murmur intensity (holosystolic but decrescendo). The previously normal left atrium responds by increasing its wall thickness (fig. 6) and its contractile force and thus causing presystolic distention of the left ventricle and a fourth heart sound. The direction of the regurgitant jet may be forward and medial toward the atrial septum adjacent to the aortic valve, so that radiation of the murmur is toward the left sternal edge and base; this pattern accounts for “mitral incompetence simulating aortic stenosis.” In other patients, the direction of the jet is posterior or lateral.

**Chronic severe mitral incompetence** stands in contrast to acute severe mitral regurgitation (fig. 6). In chronic severe mitral incompetence, there is a long history of a murmur which is holosystolic but not decrescendo; radiation is to the axilla and back; atrial fibrillation is the rule with a prominent third heart sound; physical signs of pulmonary hypertension are absent because a distensible left atrium enlarges strikingly and “absorbs” the regurgitant flow (fig. 6).

**Papillary Muscles and Left Ventricular Wall**

These two elements represent the muscular components of the mitral apparatus. The papillary muscle, as a functional unit, includes a portion of the adjacent left ventricular wall.

Although rupture of a papillary muscle has been known since 1803, the term “papillary muscle dysfunction” was not introduced until 1963. Dysfunction can occur either with loss in continuity (rupture) or without loss in continuity (fibrosis, ischemia, replacement; table 1).

The papillary muscles emerge as single bodies from the left ventricular wall and divide into a variable number of heads, each of which serves as an anchor for chordae tendineae (fig. 4). Rupture of a papillary muscle typically results from infarction of the posteromedial segment. Traumatic rupture is rare. Sudden loss in continuity results in acute severe regurgitation. The specific anatomic site at which papillary muscle rupture occurs is an important determinant of the magnitude of regurgitation and the subsequent clinical course (fig. 4). If rupture is confined to one papillary muscle head, the physiologic derangement is quantitatively similar to rupture of major chordae. If rupture involves the entire papillary muscle, i.e. its central body, approximately half of the support of each leaflet is lost so that regurgitation is overwhelming and rapidly fatal (fig. 4). In either case, the mitral incompetence of papillary muscle rupture is superimposed upon an acutely infarcted left ventricle, whereas acute regurgitation from chordal rupture usually occurs in an otherwise normal or nearly normal heart.

The papillary muscles contract before the left ventricular wall, bracing the leaflets and chordae for the ensuing abrupt rise in intraventricular pressure. When this sequence is reversed by a ventricular ectopic beat, a whiff of mitral regurgitation occurs (this is a common observation during left ventricular angiography). Papillary muscle contraction normally continues synchronously with left ventricular contraction, and thereby supports the chordae tendineae and prevents overshoot of the leaflets during ventricular systole. If papillary muscle contraction is absent or ineffective, i.e., papillary muscle dysfunction is present, then, as the left ventricular apex moves toward the annulus in systole, the chordae slacken, the leaflets are not held in

**Table 1**

**Papillary Muscle Dysfunction**

<table>
<thead>
<tr>
<th>1. With loss in continuity (rupture):</th>
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<tbody>
<tr>
<td>a. Infarction</td>
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<tr>
<td>b. Trauma (rarely)</td>
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</table>

<table>
<thead>
<tr>
<th>2. Without loss in continuity:</th>
</tr>
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<tbody>
<tr>
<td>a. Infarction — persistent mitral incompetence</td>
</tr>
<tr>
<td>b. Ischemia without infarction (angina) — transient mitral incompetence mild to severe</td>
</tr>
</tbody>
</table>
place, and prolapse into the left atrium occurs.

The anatomic basis of papillary muscle dysfunction without loss in continuity is still not completely settled. Burch and co-workers\textsuperscript{43} originally called attention to the effect of coexisting aneurysm of the adjacent left ventricular wall from which the infarcted papillary muscle took origin. Subsequently, Shelburne and associates\textsuperscript{47} commented on the role of infarction of the portion of left ventricular wall that forms the base of the papillary muscle. These authors believed that infarction at that site resulted in failure to provide an adequate anchor for the contiguous papillary muscle or, by forming an aneurysm, changed the directional axis of the papillary muscle itself. Infants with severe congenital valvular aortic stenosis exhibit atrophy of one or both papillary muscles, with mitral regurgitation ascribed to that cause.\textsuperscript{48} Yet many older patients with valvular aortic stenosis have papillary muscles that are small in comparison with the hypertrophied left ventricular wall, but the mitral valve remains competent.\textsuperscript{49} Mittal and co-workers\textsuperscript{46} and Tsakiris and associates\textsuperscript{50} have clarified these discordant views by showing that experimental damage\textit{confined} to a canine papillary muscle does\textit{not} necessarily render the mitral valve incompetent unless the damage extends to the adjacent left ventricular wall (fig. 7). Accordingly, the papillary muscle as a functional unit is now believed to include its muscular foundation in the contiguous left ventricular wall.\textsuperscript{19, 46, 50}

Papillary muscle dysfunction without loss in continuity can result in either persistent mitral incompetence of varying severity\textsuperscript{43, 51} or in intermittent mitral incompetence during episodes of ischemia (angina) without infarction (table 1).\textsuperscript{2, 52} Occasionally, intermittent, severe mitral regurgitation (table 1) is accompanied by precipitous elevations of left atrial v waves and pulmonary edema due to transient ischemic papillary muscle dysfunction.\textsuperscript{52}

Mitrval regurgitation occurs not only with a classic holosystolic murmur but also with an early systolic decrescendo murmur, a late

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\textbf{Figure 7}

Histologic sections of anterolateral (a) and posteromedial (b) left ventricular papillary muscles in a 51-year-old man with type II hyperlipoproteinemia and two myocardial infarcts who died suddenly. At no time was a murmur heard. Each papillary muscle is focally scarred, but the left ventricular free walls beneath them are normal.
systolic murmur, or a midsystolic murmur. The mitral incompetence of papillary muscle dysfunction encompasses all of these types. In fact, the configuration sometimes changes from beat to beat. Variations in the functional state of the papillary muscle and left ventricular wall and in the alignment (directional tension) exerted by the papillary muscle on its chordae are likely to influence the timing of the murmur. Some late systolic murmurs of papillary muscle dysfunction have recently been shown to follow one or more midsystolic clicks. Accordingly, the systolic click-late systolic murmur syndrome must now be extended to include ischemic heart disease.

Now let's turn attention to those aspects of the left ventricular free wall related to the shape of the ventricle per se. It is axiomatic that enlargement of the left ventricle may be accompanied by mitral regurgitation. The explanation generally offered is dilatation of the annulus, but this appears to be inadequate even though admittedly the mitral orifice can dilate. How then might left ventricular enlargement render the mitral valve incompetent? In part, dilatation may prevent the annulus from decreasing its circumferential size during ventricular systole. The answer, however, seems to lie chiefly in the effect of altered left ventricular shape on the position of the papillary muscles and their directional axes of tension. The papillary muscles normally arise from the left ventricular wall at its apical and middle thirds (fig. 1). This position permits the contracting papillary muscles to exert a desirable vertical force on the chordae tendineae, effectively moving the leaflets together during isovolumetric contraction and restraining them during ventricular ejection. In contrast, when the papillary muscles are not vertically aligned with the annulus (lateral migration due to spherical dilatation of the left ventricle), the systolic forces exerted on the leaflets via the chordae are in a lateral as opposed to a vertical direction. This lateral tension, especially on the anterior leaflet, opposes apposition, and renders the valve incompetent. Lateral enlargement of the left ventricle is a feature of mitral regurgitation itself, so that once initiated, "regurgitation begets regurgitation." On the other hand, lesions such as aortic incompetence result principally in downward elongation of the left ventricle. If the papillary muscles are pulled in the same direction, the distance between the mitral annulus and the apex increases so that chordae become too short to permit leaflet closure. Compensatory elongation of the chordae under these circumstances serves to preserve mitral valve competence, but if the annular-apical distance subsequently decreases (correction of aortic regurgitation), these elongated chordae might result in leaflet overshoot.

A different alteration in left ventricular shape has been held responsible for another variety of mitral regurgitation, namely, the late regurgitation of idiopathic hypertrophic subaortic stenosis. Echocardiography has shown that in this disorder the anterior mitral leaflet reopens in midsystole after normal initial closure. This reopening times with the onset of outflow obstruction as the anterior leaflet and septum approach each other. It has been postulated that the abnormal systolic movement of the hypertrophied interventricular septum alters the axis of the anterolateral papillary muscle by pulling it from its closed position. Such movement may not only provoke late systolic mitral regurgitation but may also contribute to obstruction to ventricular outflow.

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