Left Ventricular Papillary Muscles

Description of the Normal and a Survey of Conditions Causing them to be Abnormal

By William C. Roberts, M.D., and Lawrence S. Cohen, M.D.

SUMMARY
The left ventricular papillary muscles appear to be the last portions of the heart to be perfused by coronary arterial blood. As a consequence they are sensitive anatomic markers of myocardial ischemia. Foci of necrosis or fibrosis therefore are commonly seen in these structures, particularly the posteromedial papillary muscle, which has a poorer blood supply than does the anterolateral muscle. Coronary arterial luminal narrowing is the most common cause of necrosis or fibrosis of the left ventricular papillary muscles. Other conditions, all associated with inadequate cardiac output, which may produce these lesions include left ventricular outflow tract obstruction, especially that resulting from congenitally malformed aortic valves, acute valvular regurgitation (infective endocarditis), various cardiomyopathies, and primary endocardial fibroelastosis with or without anomalous origin of one or both coronary arteries from the pulmonary trunk. Various infiltrative diseases, including inflammation (Aschoff bodies, sarcoid, abscesses), amyloid, iron, and neoplasms, also may involve the papillary muscles. Their most common congenital malformation is the parachute or single papillary muscle. Fibrosis or necrosis of adjacent left ventricle free wall without involvement of the papillary muscles themselves may simulate clinically “papillary muscle dysfunction.” The anterior papillary muscle of the right ventricle is frequently affected by conditions which also affect the left ventricular papillary muscles. Whether or not necrosis or fibrosis of the right ventricular papillary muscle causes tricuspid regurgitation, however, is unknown at present.

Additional Indexing Words:
Coronary heart disease  Congenital heart disease  Myocardial infarction
Aortic stenosis  Idiopathic cardiomegaly  Cardiac surgery

ONE OF THE MOST significant advances in clinical cardiology during the decade of the 1960's was the appreciation of the importance of the left ventricular papillary muscles to closure of the mitral orifice during ventricular systole. It is now a well-recognized fact that hypoxia, necrosis, or fibrosis of the left ventricular papillary muscles may be associated with varying degrees of mitral regurgitation. Although coronary atherosclerosis is the most common cause of papillary muscle disease, scarred or necrotic papillary muscles have been observed in a number of conditions in which the coronary arteries were normal. Despite our increased awareness of disorders of the papillary muscles, a number of discrepancies have appeared which indicate that our knowledge about these structures is incomplete. For example, a number of patients without precordial murmurs during life have been observed at necropsy to have severe necrosis or fibrosis or both of one or both left ventricular papillary muscles; severe mitral regurgitation during or after acute myocardial infarction has been found at necropsy to be

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associated with normal papillary muscles, normal mitral leaflets and chordae tendineae, and normal-sized mitral annulae. This report attempts to clarify some of these discrepancies by reviewing some necropsy observations on the cardiac papillary muscles and correlating them with clinical findings.

Normal Left Ventricular Papillary Muscles

Each of the two left ventricular papillary muscles receives chordae tendineae from each mitral valve leaflet (fig. 1). Thus, damage to either papillary muscle may affect both leaflets. Each papillary muscle may be viewed as consisting of a major trunk from which an average of six heads or fingers project (fig. 2). Each papillary muscle has an average of 12 chordae tendineae per head. Each primary or first-order chorda tendinea divides into an average of two secondary or second-order chordae tendineae. Each second-order chorda divides into two or three tertiary or third-order chordae tendineae which attach to the mitral leaflet. Thus, for each primary chorda an average of five tertiary chordae result, and each head of a papillary muscle anchors two first-order and 10 third-order chordae. Consequently, each papillary muscle supports an average of 62 chordae actually attached to mitral leaflets, or both papillary muscles support about 124 third-order chordae or 24 first-order chordae. There is considerable variation, however, in the number of chordae tendineae attached to either papillary muscle or to either mitral leaflet.

Generally in a normal heart the thickness of either papillary muscle is about the same as is that of the left ventricular free wall or ventricular septum. The anterolateral (A-L) papillary muscle normally is slightly larger than the posteromedial (P-M) one. Just as the

![Figure 1](https://circ.ahajournals.org/content/46/7/139)

*Normal left ventricular papillary muscles. The posteromedial (P-M) papillary muscle, its chordae tendineae, and portions of anterior (Ant) and posterior (Post) mitral leaflets attached to it are enclosed by dotted lines. Each papillary muscle receives chordae from both mitral leaflets. Thus, rupture of one head or of the entire trunk of a papillary muscle alters support to both leaflets.*

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P-M and A-L papillary muscles can differ from each other in the same heart, considerable morphologic variation is observed in comparing either or both muscles from one heart with those of another. The A-L papillary muscle usually (75%) consists of a single major muscle group, whereas the P-M papillary muscle often (65%) consists of two or three major muscle groups. The basal to apex lengths of the papillary muscles also vary considerably. In most individuals the papillary muscles are attached to the left ventricular free walls over a large base and often by several trabecular bridges. Sometimes the site of attachment or anchorage is only slightly larger than the widest circumference of the papillary muscle. Ranganathan and Burch called the former type of papillary muscle tethered, in contrast to the fingerlike type which protrudes freely into the left ventricular cavity with few or no trabecular attachments. Mixtures of these two types also exist. It is probable that the tethered type has a more abundant blood supply. The axis of orientation of the papillary muscles is generally parallel to the long axis of the left ventricular cavity which is nearly perpendicular to the left atrioventricular valve annulus.

The A-L papillary muscle appears to have a richer blood supply than does the P-M one. Myocardial infarction involving the posterior left ventricular wall usually results in necrosis of the P-M papillary muscle, whereas anterior-wall infarction may spare the A-L papillary muscle. The A-L papillary muscle is supplied by branches from both left anterior descending and left circumflex coronary arteries. The major supply of the P-M papillary muscle is dependent on which coronary artery is dominant. When the right coronary is dominant, and this is the situation in 90% of human hearts, its major supplier is the right coronary artery, and when the left one is dominant the major supplier is the left circumflex. The left circumflex contributes some blood, however, to the P-M papillary muscle no matter which coronary is dominant. The arrangement of the intramural coronary arteries supplying the papillary muscles appears to depend on some extent on their gross structure. When they have a fingerlike configuration one or several major intramural vessels (class B arteries of Estes et al.) arise from the epicardial branch, extend through the left ventricular free wall to the bases of the papillary muscles, turn uphill, so to speak, coursing to the apices of the muscles giving off branches along the way. When this artery is single it has been called the "central artery." Papillary muscles or portions of them receiving the central artery usually have few or no anastomotic connections with the extrapapillary subendocardial plexus. The tethered papillary muscles usually

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**Figure 2**

Diagram of a single normal left ventricular papillary muscle and its attached chordae tendineae. Each left ventricular papillary muscle contains an average of six "heads," each of which contains two primary or first-order chordae tendineae. Each primary cord subdivides into two secondary chordae, each of which divides into two or three tertiary or third-order chordae. The number of chordae attached to each left ventricular papillary muscle thus averages 12, and the number of chordae inserting directly into the mitral leaflets from a single papillary muscle averages 62. (These numbers resulted from counting chordae and papillary muscle heads in 12 normal hearts.) Thus, rupture of one papillary muscle head, which contains usually two primary chordae, causes loss of function of at least 10 tertiary chordae.

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do not have a single central artery but many small ones (class A arteries of Estes et al.3,5) with rich anastomotic connections among themselves as well as with the extrapapillary subendocardial plexus.2,3

Although their major blood supply is from intramural coronary arteries, the most peripheral portions of the left ventricular papillary muscles are perfused by intracavitary blood. The actual mechanism by which oxygen diffuses into the immediate subendocardial regions is uncertain. Possibly endocardial pores provide the means.

Abnormal Left Ventricular Papillary Muscles

Types of papillary muscle damage and the causes of the various types are outlined in table 1.

Anatomically Normal Papillary Muscles but Papillary Muscle Dysfunction

Transient ischemia of the papillary muscles is believed to be extremely common and probably results in varying degrees of mitral regurgitation.6–8 A murmur of mitral regurgitation may occur during an attack of angina pectoris and be absent during pain-free periods.6–10 Transient systolic murmurs during and after myocardial infarction probably result from papillary muscle ischemia. Arterial perfusion of the left ventricular papillary muscles may be influenced by body position. Brody and Criley9,10 documented severe mitral regurgitation and interscapular back pain in a man when he lay supine but both disappeared when he assumed an upright position. Possibly, transient ischemia of the papillary muscles may occur during emotional stress in individuals with normal or nearly normal coronary arteries in a manner similar to that which leads to constriction of the peripheral vascular beds during anxiety or during cigarette smoking.

Left ventricular dilatation of any origin is a frequent cause of papillary muscle dysfunction.6–8 Under such circumstances the papillary muscles may contract normally, but the spatial relationships between the papillary muscles, the chordae tendineae, and the orifice are altered by the caudal and lateral migration of the left ventricular wall away from the mitral annulus. The valve leaflets are thus pulled downward into the left ventricle, and consequently the mitral orifice becomes incompetent. Also, the axes of the papillary muscles become more oblique with respect to the mitral annulus.

Although it may accompany left ventricular dilatation, mitral annular dilatation is probably a rare cause of mitral regurgitation, and most patients in the past with mitral regurgitation believed to be the result of mitral annular dilatation probably had papillary muscle dysfunction instead. Mitral annular dilatation to the degree capable of causing mitral regurgitation probably does not occur because the surface area of the mitral leaflets is about two times the area of the mitral orifice,11 and the annulus contracts during ventricular systole.12 Indeed, the mitral ring has a sphincter-like action since the mitral orifice is smaller during systole than during diastole.12 In patients with dilated left ventricles, the widest diameter occurs not at the left ventricular base, the area which includes the mitral annulus, but in the midportion of the chamber between apex and base. Indeed, the base of the left ventricle is prevented from dilating freely because the “fibrous skeleton” is attached to it whereas the midportion of the left ventricle is not thus inhibited. Severe left ventricular dilatation may occur without any dilatation of the mitral annulus.

Although it is now well appreciated that mitral regurgitation may occur in patients with dilated left ventricles from any cause without associated left ventricular necrosis or fibrosis, it is less well recognized that necrosis or fibrosis of the left ventricular free wall unassociated with left ventricular dilatation or papillary muscle or mitral tissue lesions also may be associated with mitral regurgitation (fig. 3). Generally, the scarring or necrosis of the myocardium adjacent to the papillary muscles fails to move or moves paradoxically during ventricular systole and the abnormal ventricular contraction may lead to abnormal papillary muscle anchoring with resultant

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**Table 1**

*Spectrum of Left Ventricular Papillary Muscle Disease*

<table>
<thead>
<tr>
<th>I.</th>
<th>Anatomically normal papillary muscles but papillary muscle dysfunction:</th>
</tr>
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<tbody>
<tr>
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<td>Transient ischemia</td>
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<tr>
<td>B.</td>
<td>Left ventricular dilatation from any cause</td>
</tr>
<tr>
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<td>Generalized</td>
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<tr>
<td>2.</td>
<td>Localized</td>
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<td>C.</td>
<td>Necrosis or fibrosis in adjacent left ventricular free wall</td>
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<tr>
<td>1.</td>
<td>With coronary arterial narrowing</td>
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<tr>
<td>2.</td>
<td>Without coronary arterial narrowing</td>
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<tr>
<td>D.</td>
<td>Small left ventricular cavity (hypertrophic cardiomyopathy)</td>
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<th>Necrosis or fibrosis of papillary muscle(s) without rupture:</th>
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<td>2.</td>
<td>Healed myocardial infarction</td>
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<tr>
<td>B.</td>
<td>Without coronary arterial narrowing</td>
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<tr>
<td>1.</td>
<td>Acute</td>
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<tr>
<td>a.</td>
<td>Shock</td>
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<td>b.</td>
<td>Acute valvular regurgitation (infective endocarditis)</td>
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<td>2.</td>
<td>Chronic</td>
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<tr>
<td>a.</td>
<td>Anemia</td>
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<td>Left ventricular outflow obstruction</td>
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<td>Valvular aortic stenosis</td>
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<td>Discrete and diffuse subaortic stenosis</td>
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<td>3.</td>
<td>Supravalvular aortic stenosis</td>
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<td>c.</td>
<td>Systemic hypertension</td>
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<td>d.</td>
<td>Origin of left coronary artery or of both coronary arteries from pulmonary trunk</td>
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<td>e.</td>
<td>Primary endocardial fibroelastosis of left ventricle</td>
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<td>f.</td>
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<td>Löeffler's fibroplastic parietal endocarditis</td>
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<td>g.</td>
<td>Idiopathic cardiomegaly (primary or diffuse myocardial disease)</td>
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<td>h.</td>
<td>Focal myocardial disease</td>
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<td>1.</td>
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<td>2.</td>
<td>&quot;Neurogenic&quot; heart disease</td>
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<td>Friedreich's ataxia</td>
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<td>Myotonic muscular dystrophy</td>
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<th>Necrosis or fibrosis of papillary muscle(s) with rupture:</th>
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<td>Trauma</td>
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<td>Miscellaneous</td>
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<td>Types of papillary muscle rupture</td>
<td></td>
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<tr>
<td>1.</td>
<td>Total (&quot;belly or trunk&quot;) → rapid death</td>
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<tr>
<td>2.</td>
<td>Partial (&quot;head&quot;)</td>
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<tr>
<td>a.</td>
<td>Rapid death</td>
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<td>b.</td>
<td>Survival but chronic congestive heart failure</td>
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<th>IV.</th>
<th>Infiltrative diseases of papillary muscles:</th>
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<td>Granulomas</td>
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<td>Aschoff bodies</td>
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<td>2.</td>
<td>Sarcoïd</td>
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<td>C.</td>
<td>Amyloid</td>
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<td>D.</td>
<td>Neoplasm</td>
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<td>E.</td>
<td>Calcium</td>
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F. Iron
G. Other

V. Congenital malformations of papillary muscle(s):
   A. Single papillary muscle (parachute mitral valve syndrome)
   B. Accessory papillary muscle
   C. Abnormally large and malpositioned papillary muscles
   D. Abnormally small and malpositioned papillary muscle
   E. Insertion of papillary muscle directly into mitral leaflet

VI. Miscellaneous afflictions of papillary muscle(s):
   A. Excision, partial or complete, during mitral valve replacement
      1. Atrophy of nonexcised portion of papillary muscle after valve replacement
      2. Left ventricular aneurysm at site of papillary muscle excision
   B. Disuse atrophy after excision of mitral leaflets and chordae without excision of papillary muscle(s)

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Figure 3

**Mitral regurgitation from scarring of the left ventricular free walls but not of the papillary muscles.** The heart shows severe scarring of the left ventricular wall from the base of the papillary muscles to the cardiac apex, but the posteromedial (P-M) and anterolateral (A-L) muscles are spared from significant scarring. This 72-year-old man (A68-83) had an acute myocardial infarct at age 60 years. He was well thereafter until age 69 (3 years before death) when mild congestive cardiac failure appeared, but no precordial murmur was heard. On examination 9 months before death a grade 1/VI pansystolic apical murmur was heard. Five months later severe congestive heart failure appeared, and cardiac catheterization was performed. The left atrial v waves ranged from 60 to 75 mm Hg and left ventricular cineangiogram disclosed 3+/4+ mitral regurgitation. Despite this angiographic documentation of mitral regurgitation the precordial murmur was never louder than grade 1/VI intensity and located in early and midsystole only. Cardiomyopathy was performed 3 months before death. The anterior mitral leaflet was found to herniate into the left atrium during ventricular systole but the mitral leaflets and chordae appeared normal. The mitral valve was plicated but congestive cardiac failure, which ultimately was fatal, persisted postoperatively.

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mitral regurgitation.\textsuperscript{8, 12} Anatomic left ventricular aneurysms unassociated with papillary muscle necrosis or fibrosis may cause mitral regurgitation by the same mechanism (fig. 4).

Abnormal pulling on the papillary muscles unassociated with left ventricular necrosis or fibrosis or with cavity dilatation may occur in patients with hypertrophic cardiomyopathy, and this mechanism may account for the mitral regurgitation in them.\textsuperscript{14} The huge thickening of the ventricular septum—the thickest portion is midway between left ventricular base and apex—\textsuperscript{10}—may distort or bend the anterolateral papillary muscle and prevent proper contraction of this structure during ventricular systole.

\textbf{Necrosis or Fibrosis of Papillary Muscle(s) without Rupture}

Necrosis and fibrosis of one or both left ventricular papillary muscles is extremely common. The necrosis or fibrosis may be either focal or diffuse, involving only one papillary muscle or both, and auscultatory mitral regurgitation may or may not be present. When they are focal, the papillary muscle lesions are generally of two types: (1) involve nearly all the distal or apical portion of the papillary muscle, or (2) involve many areas throughout the entire papillary muscle. The latter lesions usually are small and spare areas adjacent to intramural coronary arteries.\textsuperscript{16} When only one papillary muscle contains foci of necrosis or fibrosis it is virtually always

\textbf{Figure 4}

Radiograph of heart specimen (a) and longitudinal section of heart (b) in a 65-year-old man (A67-71) who had an acute myocardial infarct 2 months before death with subsequent development of a left ventricular (L.V.) aneurysm (An.) located in the lateral wall. He developed a murmur consistent with mitral regurgitation. Both left ventricular papillary (pap.) muscles grossly appeared normal but their bases were adjacent to the aneurysm. The mitral regurgitation can probably be attributed to the poor anchorage of the papillary muscles as a result of the aneurysm. R.A. = right atrium; R.V. = right ventricle; V.S. = ventricular septum; L.A. = left atrium; A.V. = aortic valve; Ao. = aorta.
the P-M muscle, since this one has the poorer blood supply.

It is now clear from experimental studies\textsuperscript{17-19} that mitral regurgitation is not a consequence of fibrosis involving only papillary muscles themselves. If one or both left ventricular papillary muscles are made fibrotic in the dog, either by injection of formalin or by ligating the base of the muscle, no regurgitation of contrast material from left ventricle to left atrium later occurred on angiographic studies. If, however, the free wall beneath the papillary muscle was made fibrotic at the same time so that ventricular contraction was impaired, mitral regurgitation did result. We have observed a number of patients at necropsy with extensive fibrosis or necrosis of one or both left ventricular papillary muscles, and no precordial murmur had been audible during life. Fibrosis or necrosis of the left ventricular papillary muscles and of the free walls beneath them, however, does not necessarily assure the appearance of a precordial murmur of mitral regurgitation during life. Several patients with silent mitral regurgitation by auscultation have been shown to have mitral regurgitation when left ventricular injection of contrast material also was performed.\textsuperscript{20, 21} Necrosis or fibrosis has been observed at necropsy in these patients to involve the papillary muscles extensively and the entire thickness (transmural) of the left ventricular free wall.\textsuperscript{22} Silent mitral regurgitation during acute myocardial infarction has been attributed to a diminished flow velocity across the mitral valve secondary to diminished myocardial contractility.\textsuperscript{20}

The most common cause of papillary muscle necrosis or fibrosis is narrowing of the coronary arterial lumen by atherosclerosis.\textsuperscript{4, 22, 23} The P-M papillary muscle is usually involved in acute posterior myocardial infarction. In anterior-wall infarction, however, the A-L papillary muscle may be spared, presumably because its blood supply is better than that of the P-M muscle. Since infarction limited to the ventricular septum or to the lateral portion of the left ventricular free wall (i.e. the portions of the left ventricular wall unassociated with papillary muscles) is rare, the papillary muscles are usually involved when myocardial infarction occurs. It, therefore, is surprising that papillary muscle dysfunction is not more frequently observed during or after acute myocardial infarction.

Necrosis or fibrosis of one or both left ventricular papillary muscles is frequent in a number of conditions unassociated with luminal narrowing of the extramural coronary arteries. Inadequate oxygenation of the papillary muscles may occur if the amount of oxygen in the blood is low (i.e., anemia) or if the cardiac output for any reason is inadequate. It is well to keep in mind that the left ventricular papillary muscles are the last portions of the heart to be perfused with coronary arterial blood. To perfuse the apices of the papillary muscles the coronary artery must extend through the entire thickness of the myocardial free wall, turn up hill, and ascend a distance of at least one and often two thicknesses of the free wall. Consequently, it is of little wonder that these structures often show evidences of inadequate oxygenation. Furthermore, the papillary muscles serve as the most sensitive markers of inadequate myocardial oxygenation. Since the P-M papillary muscle is less well perfused than the A-L one, if only one shows foci of necrosis or fibrosis it will nearly always be the P-M muscle. We have observed foci of necrosis in these papillary muscles in patients with anemia from a variety of causes, particularly chronic anemias like sickle-cell disease (fig. 5).

In patients with left ventricular outflow obstruction, lesions are often observed in the papillary muscles.\textsuperscript{24-28} Valvular aortic stenosis, whether in infants\textsuperscript{24, 25} or in adults,\textsuperscript{26-28} when superimposed on a congenitally malformed valve, either unicuspid or bicuspid, is nearly always associated with fibrosis and atrophy of at least the P-M papillary muscle. Rheumatic valvular aortic stenosis combined with organic mitral stenosis or regurgitation, however, infrequently is associated with papillary muscle fibrosis or
The heart in a 14-year-old girl (A69-13) with sickle-cell disease diagnosed by hemoglobin electrophoresis when she was 4 years old. She had multiple sickle-cell crises and at age 12 years developed congestive cardiac failure. During her last 6 months she had nightly paroxysmal dyspnea and multiple episodes of exertional syncope. She had a grade II/VI systolic murmur over the pulmonic area. Electrocardiogram showed right-axis deviation, right ventricular hypertrophy, and P-pulmonale. The pulmonary arterial pressure was 47/13 mm Hg, and the cardiac output was 6.5 liters/min. (a) Chest roentgenogram. (b) Posteromedial (P-M) left ventricular papillary muscle. (c) Section of P-M papillary muscle showing focal fibrosis. (d) Opened right ventricle showing fibrosis of the anterior (Ant.) papillary (pap.) muscle. P.V. = pulmonic valve. (e) Section of scarred anterior right ventricular papillary muscle. The papillary muscle fibrosis is presumably related to the chronic anemia.

atrophy. Nearly all adult patients with discrete subaortic stenosis or hypertrophic cardiomyopathy with or without diffuse subaortic stenosis have small fibrous scars in both left ventricular papillary muscles, but severe atrophy of one or both muscles is uncommon.

Foci of necrosis also are common in patients with fatal severe valvular regurgitation of recent onset. Among 47 patients dying of active valvular infective endocarditis, 34 (72%) had necrotic lesions in one or both left ventricular papillary muscles, and none had
Severe diffuse fibrosis and calcification of the anterolateral left ventricular papillary muscle in a 9-month-old girl (A56-14) in whom the left coronary artery arose anomalously from the pulmonary trunk. She had a grade III/VI systolic precordial murmur. Diffuse endocardial fibroelastosis of the left ventricle and left atrium also was present. (a) Opened left ventricle, aortic valve, and aorta. The ostium of the right coronary artery is designated by the arrow. No left ostium is observed. The distal half of the A-L papillary muscle is severely scarred and calcified. (b) Section of the fibrotic, atrophied, and calcified A-L papillary muscle. (Hematoxylin and eosin stain, × 4.)

Figure 6

significant narrowing of the extramural coronary arteries.29 Most patients had pure aortic regurgitation, but some had pure mitral regurgitation secondary to the active infective endocarditis. Severely diminished cardiac output with resultant poor myocardial perfusion was believed to be the cause of the papillary muscle necrosis in them.

Small fibrous scars in the papillary muscles are common in patients with systemic hypertension but atrophy of these structures in this condition, unless coronary heart disease also is present, is infrequent. When left ventricular hypertrophy occurs from any cause, the hypertrophy of the papillary muscles probably should be proportional to that of the left ventricular free wall or ventricular septum. This proportional hypertrophy usually exists in cases of systemic hypertension, but infrequently in patients with left ventricular outflow obstruction.

Fibrosis with atrophy of the papillary muscles is to be expected in patients with primary endocardial fibroelastosis of the left ventricle.30 Histologically, the thickened endocardium in this condition is similar to the media of the aorta, being characterized by the presence of elastic fibrils running parallel to one another and to the surface. Primary left ventricular endocardial fibroelastosis may represent simply an anatomic expression of chronically inadequate coronary arterial perfusion of the left ventricle, and, since the P-M papillary muscle is the least well-perfused portion of the heart, this structure is affected the most. In the congenital condition, origin of the left31 or both32 coronary arteries from the pulmonary trunk, diffuse endocardial fibroelastosis of the left ventricle is nearly always an associated lesion. Fibrosis and atrophy of the left ventricular papillary muscles is to be expected in this anomaly, and these patients may present clinically with features of pure mitral regurgitation33 (fig. 6).
Diagram depicting two major types of papillary muscle rupture. It is likely that rupture of the entire trunk (acute myocardial infarction or trauma) (left) is incompatible with survival since a major portion of the support to both valve leaflets is destroyed. With rupture of an apical head (right), survival would appear to depend upon the extent to which the function of the left ventricle has been impaired by necrosis. With severely impaired ventricular function, the additional burden of even modest mitral regurgitation may be intolerable, and death is quick. If the left ventricle is less severely compromised, survival is possible for weeks or months, but congestive cardiac failure will almost invariably develop.

Foci of fibrosis or necrosis in the left ventricular papillary muscles are infrequent in patients with the congestive or dilated type of cardiomyopathy. Severe scarring of one papillary muscle and of the free wall beneath it was responsible for severe mitral regurgitation in a patient with primary myocardial disease studied by Marcus et al.44 Generally, however, left ventricular cavity dilatation alone is the cause of mitral regurgitation in these patients. Rarely, myocarditis may be localized to papillary muscle and adjacent left ventricular free wall.45 Patients with the neurogenic heart diseases (Friedreich’s ataxia, progressive muscular dystrophy, and myotonic muscular dystrophy)45 generally have scarred left ventricular papillary muscles, especially the P-M one. The African cardiomyopathy—endomyocardial fibrosis—may be associated with extensive scarring of the papillary muscles.47 Usually the P-M one in this condition is covered by a thrombus or by dense fibrous tissue which may represent organization of thrombus. Likewise, Löeffler’s fibroplastic parietal endocarditis, which may represent one stage of endomyocardial fibrosis,48 is usually associated with extensive scarring of one or both papillary muscles.49

Necrosis or Fibrosis of Papillary Muscle with Rupture

In contrast to necrosis of a papillary muscle which occurs in over 50% of patients with fatal acute myocardial infarction,4 rupture of a papillary muscle is rare, occurring in <1% of patients with fatal acute myocardial infarction. The rupture may be of two types (fig. 7). One involves the entire central muscle belly of the papillary muscle and this type rupture is incompatible with survival since half the support to each valve leaflet is destroyed and mitral regurgitation of overwhelming severity occurs. The second type of rupture involves only one or two apical heads of a papillary muscle. The resulting mitral regurgitation is of lesser magnitude, and immediate survival is then dependent upon the degree to which the function of the left ventricle has been impaired by the infarct. In patients who survive after papillary muscle rupture, the functional capacity of the left ventricle also will govern the extent of clinical and hemodynamic improvement after mitral valve replacement.50

Although the entire papillary muscle is usually necrotic, the mitral regurgitation resulting from rupture of an entire trunk of a papillary muscle may justifiably be attributed entirely to the rupture. In contrast, the mitral regurgitation following rupture of only one head of a left ventricular papillary muscle cannot necessarily be attributed entirely to the rupture since the remainder of the papillary muscle is nearly always also necrotic. What percentage of the regurgitant volume is due to the rupture of a single head and what percentage to the associated papillary muscle necrosis is uncertain. Rupture of a single

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Operatively excised mitral valves in four patients demonstrating rupture of a papillary muscle head in each. Acute myocardial infarction (AMI) had occurred in each 15 months (a), 13 months (b), 3 months (c), and 14 months (d) earlier. All four patients were men, aged 51 to 69 years at the time of operation, and all developed loud (grade III-IV/VI) systolic murmurs typical of mitral regurgitation at the time of AMI. Congestive cardiac failure persisted after AMI. The arrows designate the ruptured papillary muscle heads. All four patients died within 3 years of operation. (The valve replacements were performed by Dr. Andrew G. Morrow.)

Although coronary arterial luminal narrowing by atherosclerosis accounts for most cases of ruptured papillary muscle, trauma may cause either partial or complete rupture of these structures. Blunt trauma, however, when severe enough to cause papillary muscle rupture, is also usually severe enough to rupture the left ventricular free wall. Brock described total rupture of a papillary muscle during mitral commissurotomy, and death ensued in two days.

Infiltrative Diseases of Papillary Muscles

The papillary muscles are subject to various infiltrative processes just like any other portion of myocardium. Pyogenic abscesses, Aschoff bodies, granulomas (fig. 9), neoplasms, amyloid, and iron have all been observed in these structures. Congestive heart failure from severe mitral regurgitation may be the initial symptom of sarcoidosis (fig. 9).

Congenital Malformations of the Left Ventricular Papillary Muscles

The most frequent congenital malformation of these structures is the occurrence of only one papillary muscle (fig. 10). This condition, described as the parachute mitral valve, consists of only one left ventricular papillary muscle to which all mitral chordae tendineae are attached. The resulting mitral valve is usually stenotic, but it may be purely incompetent or it may function normally. The single papillary muscle is usually associated with other malformations including a supramitral valve ring, diffuse subaortic stenosis, and coarctation of the aorta. Other malformations, including valvular aortic stenosis, ventricular septal defect, and valvular
pulmonic stenosis also may occur in association with the single left ventricular papillary muscle.\textsuperscript{47} The single papillary muscle malformation may be the most common cause of congenital mitral stenosis.\textsuperscript{48}

Not only may one too few papillary muscles occur, but one too many also may occur. An accessory papillary muscle is usually of no functional significance but it has been seen in association with congenital mitral regurgitation.\textsuperscript{49} Two papillary muscles may be present and yet one or both still be congenitally malformed. Each of the two papillary muscles may be abnormally large and malpositioned so that the primary orifice of the valve is narrowed.\textsuperscript{49, 50} The malpositioning consists of origin of the papillary muscles at sites higher in the left ventricle than normal. Both papillary muscles also may be poorly developed and small. This finding is particularly characteristic of origin of the left coronary artery from the pulmonary trunk. In the latter

Figure 9

Anterolateral (a) and posteromedial (b) left ventricular papillary muscles in a 26-year-old woman (PGGH A-70-541) who was asymptomatic until 10 days before death when dyspnea appeared. The dyspnea rapidly worsened, and when hospitalized on the day of death she was in acute pulmonary edema. The blood pressure was 80/70 mm Hg, heart rate 160 beats/min, and a grade III-IV/VI pansystolic blowing apical murmur, which radiated into the axilla, was audible. Chest roentgenogram showed congested lungs, cardiomegaly, and prominent hilar adenopathy. Electrocardiogram showed nonspecific ST-T wave changes and atrial hypertrophy. Several hours after admission ventricular fibrillation occurred, she was resuscitated, but complete heart block appeared. A transvenous pacemaker was inserted, but asystole occurred shortly thereafter. At necropsy, large firm white deposits were present in the walls of all four cardiac chambers and completely replaced both left ventricular papillary muscles (a and b). On histologic section, the firm white areas represented hard granulomas typical of sarcoidosis as seen in (c). (Hematoxylin and eosin stain, \( \times 400 \).) Similar hard granulomas were present in lymph nodes, liver, spleen, and lung. Stains for acid-fast organisms, other bacteria, and fungi were negative. (Specimen was kindly provided by Dr. James Hutchinson.)
LV PAPILLARY MUSCLES

Figure 10

Single left ventricular papillary muscle or parachute mitral valve syndrome. Most patients with a single left ventricular papillary muscle also have a partial or completely circumscribing ring just above the mitral valve or at its annulus, subaortic stenosis, and aortic isthmic coarctation (a). Other anomalies, particularly ventricular septal defect and valvular aortic stenosis, as shown here (b), are also common. A single left ventricular papillary muscle with a parachute mitral valve from a 14-year-old boy (GT#71A-215) is shown in (b). In addition, this child had severe valvular and discrete subvalvular aortic stenosis as well as spontaneous closure of a ventricular septal defect. The aortic valve was congenitally bicuspid. The peak systolic pressure gradient between left ventricle and systemic artery was 80 mm Hg. He died in acute pulmonary edema. He had been asymptomatic until 1 year before death. No abnormality of the mitral apparatus was apparent clinically. (This child was cared for by Dr. Joseph K. Perloff.)

Consequences of Operative-Excision of the Papillary Muscles at the time of Mitral Valve Replacement

Both left ventricular papillary muscles are excised by most surgeons at the time of mitral valve replacement. The myocardium of the free wall at the sites of excision of these muscles is always infiltrated by inflammatory cells if this area is examined in the early postoperative period. If the papillary muscles are not excised when the mitral leaflets and chordae tendineae are excised during mitral valve replacement, the papillary muscles atrophy and are focally replaced by fibrous tissue.

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When the papillary muscles are excised at operation, each is grasped by a clamp extending through the mitral orifice from the left atrium. If the papillary muscles are pulled too vigorously by the clamp when the muscle is excised a portion of left ventricular free wall beneath the left ventricular papillary muscle also may be excised. This may result in perforation of the left ventricular wall, or it may cause severe thinning of the wall at this point with formation of a functioning or anatomic aneurysm or both.

**Anterior Papillary Muscle of the Right Ventricle**

This structure appears to be susceptible to all the afflictions which affect the left ventricular papillary muscles. This structure in length is usually equivalent to several thicknesses of right ventricular wall, and blood also is required to course “up hill” to perfuse this papillary muscle. This muscle is also most frequently made necrotic or fibrotic by myocardial infarction from coronary arterial atherosclerosis. Its involvement usually indicates “massive” anterior-wall myocardial infarction. Whether or not tricuspid regurgitation is a consequence of necrosis or fibrosis or of infiltrative disease of the right ventricular papillary muscle is uncertain. Necrosis of the distal portion of this papillary muscle is common in coronary heart disease, acute valvular dysfunction as in infective endocarditis, and in chronic anemias (fig. 5).

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