Mitral Insufficiency Resulting from
“Overshooting” of Leaflets

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SUMMARY
Mitrail insufficiency may result from abnormal degrees of upward extension of valvular tissue, a condition which may be termed “overshooting.” This condition may result either from rupture of elements in the papillary muscle-chordal mechanism or from inadequate support of leaflets without rupture. The latter may occur in myocardial infarction or in instances of intrinsic weakness of connective tissues of leaflets and/or chordae tendineae.

Additional Indexing Words:
Myocardial infarction  Emphysema  Arachnodactyly  Billowing mitral valve
Rupture of chordae tendineae  Floppy mitral valve

Among the anatomic causes of mitral insufficiency is a variety of changes in the valve mechanism. Inadequacy of or restricted motion of leaflets and fusion of commissures are recognized among the several anatomic causes. Another group of conditions includes upward extension of leaflet tissue beyond the optimal level for apposition of the two leaflets, a condition which may be termed “overshooting.” Several anatomic states may result in this functional abnormality. These may be placed into two groups with respect to continuity of papillary muscle-chordal support of leaflets as follows: (a) with loss of continuity and (b) with maintenance of continuity.

In the first category, the involved portions of the mitral leaflets are flail. In each of the latter groups, various degrees of abnormal upward movement of the leaflets during ventricular systole are responsible for corresponding degrees of incompetence of the valve.

Loss in continuity of papillary muscle-chordal support may be manifested as rupture either of a papillary muscle or of chordae tendineae.

Rupture of a papillary muscle may result from myocardial infarction or trauma. The classical instance of rupture secondary to myocardial infarction occurs in subjects with coronary atherosclerosis and in whom the infarct is located in the inferior and adjacent septal wall of the left ventricle. Classically, the posteromedial papillary muscle is involved. Usually the entire muscle ruptures, and the resulting mitral insufficiency is of overwhelming proportion. The collapse in the circulation may be similar to that in rupture of the ventricular septum. Rarely, the two conditions are associated (figs. 1a and b). Uncommonly, rupture of a papillary muscle involves only a limited number of its heads, so that the resulting mitral insufficiency is of limited degree and may be tolerated for months.1, 2 A rare cause of rupture of a papillary muscle is infarction secondary to coronary embolism, as in bacterial endocarditis (figs. 1c and d).

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Figure 1

(a and b) Acute inferior myocardial infarction with rupture of papillary muscle and of ventricular septum. (a) Interior left ventricle (L.V.). A portion of the posteromedial papillary muscle has ruptured (between arrows). Beside the base of the involved papillary muscle, a rupture of the ventricular septum (probe) is also present. (b) Right ventricular (R.V.) view showing opening of ruptured ventricular septum (probe).

(c and d) Rupture of anterolateral papillary muscle secondary to bacterial endocarditis of aortic valve and coronary embolism. (c) A portion of the left atrium (L.A.) and left ventricle (L.V.). The anterolateral papillary muscle (A.L.) has ruptured secondary to infarction, which was the result of coronary embolism from the infected aortic valve shown in d. (d) A portion of the aortic valve. The left aortic leaflet (above arrow) shows vegetations and loss of substance incident to bacterial endocarditis.

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(a) Rupture of papillary muscle secondary to external trauma, viewed from interior of left ventricle (L.V.). The anterolateral (A.L.) papillary muscle has been ruptured at its base. Laceration with perforation of the adjacent ventricular wall is also present. (b) Chordae of the posterior mitral leaflet (P.) have ruptured. The unsupported portion of the leaflet shows characteristic “hooding” toward the left atrium (L.A.). (c) Mitral valve viewed from interior of left atrium (L.A.) in a case of rupture of chordae of the posterior mitral leaflet (P.). The leaflet shows characteristic “hooding.” The probe indicates the direction of the regurgitant stream which strikes the atrial septum. In this position, jet lesions are present. The aortic valve lies beyond. The systolic murmur generated by impingement of the jet-like stream against the atrial septum may be responsible for a systolic murmur in the “aorta area.” (d) A
Rupture of a papillary muscle secondary to trauma includes accidental laceration during mitral commissurotomy. More commonly, the trauma is of the external variety. The force necessary to rupture a papillary muscle is of great magnitude, so that traumatic rupture of a papillary muscle is usually associated with other cardiac injuries, including rupture of a septum or of the free wall of a chamber (fig. 2a).

Rupture of chordae tendineae is to be distinguished from rupture of a papillary muscle both as to etiology and, in a general way, as to severity of the resulting mitral insufficiency. Chordal rupture is not to be viewed as a complication of myocardial infarction. Usually, the mitral insufficiency which results from ruptured chordae is of protracted nature.

An interesting feature of chordal rupture is that the unsupported flail segment of the leaflet rises, as a hood, beyond the general level of the supported part of the leaflet (fig. 2b). The hood-like deformity tends to direct the regurgitant stream in the direction opposite the hood. Thus, with rupture of chordae to the anterior leaflet, the regurgitant stream is directed against the posterior wall of the left atrium. Contrariwise, when chordae of the posterior leaflet are ruptured, the regurgitant stream impinges against the atrial septum (fig. 2c). In this position, the site of impact of the stream is at the same level as the aortic valve. The resulting systolic murmur may be best heard in the “aortic area,” leading to confusion with aortic stenosis.8

As to the causes of chordal rupture, the destructive effects of bacterial endocarditis, whether or not clinically recognized, are clearly established4 (fig. 2d). In a significant proportion of cases the etiology is unknown, while, in other instances, a condition of recognized weakness of connective tissue such as arachnodactyly5 (Marfan’s syndrome) (fig. 3a) or gargoylism may be associated. Although rheumatic endocarditis has been claimed as a cause of chordal rupture, I have not satisfied myself as to this as an etiologic factor.

Maintenance of continuity of papillary muscle-chordal support may be associated with: (1) infarction of a papillary muscle, (2) unusually long chordae tendineae, and (3) laxity of leaflet tissue. In this group, the most common condition is acute or healed myocardial infarction of nonruptured papillary muscular tissue.

Infarction of a papillary muscle with mitral insufficiency is usually associated with infarction of the adjacent free wall of the left ventricle (fig. 3b). According to recent clinical8 and experimental evidence,9 incompetence of the valve depends not entirely upon intrinsic dysfunction of the papillary muscle but also upon distortion of the papillary muscular foundation by asynergic contraction of the related free wall.

Abnormally long chordae tendineae are seen most commonly as part of arachnodactyly. The basis for overshooting of the mitral leaflets which may occur in this condition may be contributed, in part, by the abnormally long chordae8 and, in part, by coincidental laxity of the connective tissue composing the leaflets. Laxity of leaflet tissue may be evidenced by hood-like deformity of the segments of leaflets between sites of chordal insertion.

Hooding of mitral valvular tissue is seen in a variety of circumstances. It may occur as an isolated entity or it may be a manifestation of conditions with recognized congenital weakness of connective tissues, such as arachnodactyly (Marfan’s syndrome) or gargoylism. We have been interested in the frequent association with pulmonary emphysema,9 suggesting a common factor or weakness in connective tissues of the lungs and cardiac valves10 (figs.

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portion of the left atrium (L.A.) and left ventricle (L.V.) and part of the mitral valve. Near the posteromedial commissure, the anterior mitral leaflet shows vegetation (V.) of bacterial endocarditis. The corresponding and adjacent portions of the leaflet are devoid of chordae. Residual stumps of ruptured chordae are evident (arrows).
Figure 3

(a) A portion of left atrium (L.A.) and left ventricle (L.V.) showing several ruptured chordae (arrows) attached to the anterior leaflet of the mitral valve. From a patient with extensive cystic medial necrosis and dissecting aneurysm of the aorta, suggesting a common denominator of connective tissue weakness in the aorta as well as in the tissues of the mitral valve. (b) Healed myocardial infarction. In the left ventricle (L.V.) the posteromedial (P.M.) papillary muscle shows atrophy and scarring incident to healed myocardial infarction. The adjacent subendocardial region of the free wall of the left ventricle also shows scarring and hollowing as a result of loss of muscle. The illustration supports the concept that mitral insufficiency associated with infarction of a papillary muscle is, in part, contributed by asynergic contraction of the related free wall of the left ventricle.

(c and d) Hooding of mitral valve associated with emphysema. (c) Close-up view of posterior mitral leaflet (P.) which shows upward hooiding toward the left atrium. (d) From the case shown in c. Extensive pulmonary emphysema. Illustrations c and d suggest that a common factor of connective tissue weakness may exist in the pulmonary tissue and in the mitral valve.
Figure 4

Changes associated with Marfan's syndrome. (a) The unopened mitral valve is viewed from above. Upward protrusion of the posterior mitral leaflet (P.) is a result of unusually long chordae tendineae as well as laxity of the valvular tissue. (b) Close-up view of a portion of an opened mitral valve showing distinct interchordal hooding of the mitral leaflet tissue. Unopened valve from this case shown in c. (c) Unopened mitral valve viewed from above in the case shown in b. The interchordal hooding of the mitral valvular leaflets is represented by a
3c and d). The association of mitral valvular hooding with supraavalvular aortic stenosis has been observed in this laboratory.\textsuperscript{11}

The most striking examples of interchordal hooding are seen in arachnodactyly (figs. 4a–c), in extreme cases of which segments of upward ballooning of leaflet tissue may be identified in angiograms as distinct serrations (fig. 4d).

The phenomenon of primary interchordal hooding of mitral valvular tissue brings up for consideration the condition that has been called the floppy\textsuperscript{12} or the billowing\textsuperscript{13, 14} mitral valve. The clinical phenomena of the latter condition may be the counterpart of primary interchordal hooding seen pathologically. Yet, with regard to the billowing mitral valve, one must be prepared to accept that cases of rupture of chordae or even of part of a papillary muscle may present clinically and radiographically in a similar manner.

References


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series of serrated protrusions of the mitral valvular leaflets toward the left atrium. (d) Left ventriculogram. In addition to signs of mitral insufficiency, the serrated character of the mitral valvular leaflets (between arrows) is evident.
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