Assessment of ventricular elements of mitral valve by left ventriculography

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In the left ventriculogram in the right anterior oblique projection the plane of the mitral valve is seen in profile and the papillary muscle shadows are outlined. The distance from the middle of the papillary muscles to the plane of the mitral valve during systole was used to assess the average length of the chordae tendineae, and the area of the papillary muscle shadows was measured as an index of hypertrophy in a series of hearts with mitral valve disease. Valvar mitral stenosis is characterised by slight reduction in the length of chordae tendineae and more hypertrophy of the papillary muscles, while in subvalvar mitral stenosis there is more shortening of the chordae tendineae and less papillary muscle hypertrophy. Valvotomy may lengthen the chordae tendineae in subvalvar mitral stenosis. In rheumatic mitral regurgitation length of chordae tendineae and papillary muscle size were normal. The measurements were not useful in assessing non-rheumatic mitral regurgitation.

Clinical evaluation of the mitral valve must take into consideration the functional activity of each element that participates in valve movement. Some information can be obtained from physical examination and from the electrocardiogram and echocardiogram, but assessment of the morphology and the movement of the valve and its subvalvar elements requires left ventriculography. In the right anterior oblique projection the ostium of the left ventricle, the mitral valve, and the supporting papillary muscles are seen in profile (Ross and Criley, 1964); in the left anterior oblique projection the anterior (aortic) leaflet is in profile (Bittar and Sosa, 1972).

We describe here procedures developed to assess (a) the average length of the chordae tendineae and (b) papillary muscle hypertrophy in mitral valve disease. On the left ventriculogram in the right anterior oblique projection, the distance from the middle of the papillary muscles to the plane of the mitral valve was used to represent chordae tendineae length and the area of the papillary muscles was measured by planimetry to assess papillary muscle hypertrophy. Both measurements were related to the end-systolic longitudinal axis of the left ventricle and expressed as the chordae tendineae and papillary muscle hypertrophy indices.

A close relation was found between the chordae tendineae index and the measured length of chordae tendineae from the surgical specimens of mitral stenosis. Both the chordae tendineae and the papillary muscle hypertrophy index levels are larger with valvar mitral stenosis than with subvalvar mitral stenosis. When these indices are measured in patients with restenosis of the mitral valve after valvotomy, additional insight into the pathophysiology of mitral stenosis is obtained. The indices are not useful in assessing the pathology of mitral regurgitation alone or in combination with mitral stenosis.

Methods

Records of cardiac catheterisation and cineangiography from consecutive patients with mitral valve disease studied at Guy’s Hospital (London) and at Jackson Memorial Hospital (University of Miami) provided the material for this investigation. The 'normals' were matched for age as closely as possible with the study group and were subjects who were catheterised to exclude organic heart disease and found to have no haemodynamic or cineangiographic abnormality. The stroke index was determined by standard procedures.

Left ventriculograms were obtained at Guy’s Hospital with a Siemens radiographic generator, Arriflex cinecamera, and Kodak XX film exposed at 30 frames per second, and at the University of Miami with a Picker generator using grid pulsing fluorographic control and Kodak Shellburst cineangiographic film exposed at 60 frames per second.
Angiographic assessment of mitral stenosis

At Guy's the contrast medium was Hypaque 85 per cent and was delivered by Talley injector; Renografin-76 (30 to 34 ml) was injected through a Gensini or pigtail catheter, using a Viamonte-Hobbs injector, at the University of Miami. In both institutions, a 30° right anterior oblique position was used.

The ventriculograms were analysed using the Tage-Arno projector. Outlines of the left ventricular cavity and of the papillary muscles at end-systole were traced and the distance between the middle of the papillary muscles and the centre of the left ventricular ostium was measured. Papillary muscle area was determined by planimetric measurements of the papillary muscle shadows (Fig. 1).

Results

The distance from the middle of the papillary muscles to the left ventricular ostium and measurements of long axis of the left ventricle at end-systole in normal hearts and in hearts from patients with mitral stenosis are shown in Table 1. Differences caused by variations in radiographic imaging are eliminated by relating the chordal measurement to the long axis of the ventricle; the ratio was described as the chordae tendineae index.

Traces of the end-systolic left ventriculograms from a normal heart and hearts with dominantly valvar or mixed valvar and subvalvar ('subvalvar') mitral stenosis are shown in Fig. 1. The lines used for measuring chordae tendineae distance, the papillary muscle area, and the long axis of the ventricle at end-systole are illustrated. a is from a heart with subvalvar mitral stenosis, b from valvar mitral stenosis with mild aortic regurgitation, and c from a normal heart. The shorter chordae tendineae distance in subvalvar mitral stenosis is appreciated by comparing a with b or c. (The greater length of the ventricle in b results from ventricular enlargement secondary to aortic regurgitation.)

The idea that the chordae tendineae index reflects the length of the chordae tendineae is supported by the close relation between the index and the average chordae tendineae length from 5 surgical specimens of mitral valves (Table 2). Measurements of the surgical specimens were made after suturing the annular margin of the resected valve to 'dacron' fabric that was then mounted on a wooden frame. Heavy suture material was passed through the body of the papillary muscles and the chordae tendineae were extended. Multiple measurements of the shortest and longest chordae tendineae inserted into the rough zone of the valve leaflet were averaged. Mounted valves viewed from the left atrium and from a simulated right anterior oblique projection are shown in Fig. 2. The chordae tendineae index for the anterior and posterior measurements ranged from 0·38 to 0·23 (Table 2). The lowest values for this index occurred with severe shortening of the chordae tendineae (cases 4 and 5; Fig. 1 and 2) and the higher values with longer chordae (cases 2 and 3). The specimen with the longest chordae (case 1) had low values because of the increase in length of the ventricle as a result of aortic regurgitation (trace b and Fig. 2).

Table 1 Angiographic assessment of end-systolic length of chordae tendineae in mitral stenosis

<table>
<thead>
<tr>
<th>Normal subjects</th>
<th>Mitral stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Length of chordae (cm)</td>
</tr>
<tr>
<td>Series</td>
<td></td>
</tr>
<tr>
<td>British</td>
<td>3·6 ± 0·2 (7)</td>
</tr>
<tr>
<td>U.S.A.</td>
<td>6·2 ± 0·4 (8)</td>
</tr>
</tbody>
</table>

*CTI, chordae tendineae index (i.e. ratio between length of chordae and ventricular long axis). Measurements expressed as mean ± standard deviation (number of hearts in parentheses).
The chordae tendineae index in 64 patients with mitral stenosis is shown in Fig. 3. Twenty-six of these were classified at operation or at necropsy as having major shortening of the chordae tendineae and were considered as subvalvar mitral stenosis; 38 had dominant valvar mitral stenosis. The chordae tendineae index for the normal hearts (0.53 ± 0.02) is significantly higher than the value for either group of mitral stenosis (P < 0.001). The value for subvalvar mitral stenosis is significantly lower than in valvar mitral stenosis (P < 0.001).

In some hearts, both papillary muscle shadows are poorly outlined; these were excluded from the study. In approximately 1 out of 5 hearts with mitral stenosis, the anterior papillary muscle of the left ventricle could not be outlined; in these the measurement for the posterior papillary muscle was used. In the other hearts the areas of the anterior and posterior papillary muscles were averaged.

The papillary muscle hypertrophy index in

Table 2  **Length of chordae tendineae in mitral stenosis**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Left ventriculographic measurement (CTI)*</th>
<th>Chordae tendineae length of surgical specimen (mm)</th>
<th>Surgeon’s remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Anterior 0-38</td>
<td>Posterior 0-32</td>
<td>Anterior 18</td>
</tr>
<tr>
<td>1†</td>
<td>0-36</td>
<td>0-31</td>
<td>20</td>
</tr>
<tr>
<td>2</td>
<td>0-34</td>
<td>0-31</td>
<td>12</td>
</tr>
<tr>
<td>3</td>
<td>0-28</td>
<td>0-33</td>
<td>8</td>
</tr>
<tr>
<td>4</td>
<td>0-25</td>
<td>0-23</td>
<td>7</td>
</tr>
<tr>
<td>Normal</td>
<td>0-53</td>
<td></td>
<td>18-14*</td>
</tr>
</tbody>
</table>

*CTI, chordae tendineae index.
†Mild aortic regurgitation.
‡Chordae tendineae inserting into the rough zone of the valve leaflet (Lam et al., 1970).

Fig. 2  **Surgical specimens of mounted valves with mitral stenosis** (a) annular margins sutured to a ‘dacron’ support viewed from above, (b) chordae tendineae from heart of case 1 with valvar mitral stenosis, and (c) severely shortened chordae tendineae from case 5 with valvar plus subvalvar mitral stenosis. Note the villous-like Lambl’s excrescences (Magarey, 1949) on the chordae tendineae and the papillary muscle.
Angiographic assessment of mitral stenosis

Valvar mitral stenosis is significantly higher than in the normal hearts (P < 0.01), and probably higher than in subvalvar mitral stenosis (P < 0.05) (Fig. 4). As in Fig. 3, triangles refer to hearts with restenosis.

Table 3 shows that the chordae tendineae index in hearts with sinus rhythm is similar to that in hearts with atrial fibrillation, even though atrial fibrillation is associated with lower stroke indices.

The angiographic indices are of limited value in rheumatic or non-rheumatic mitral regurgitation. The chordae tendineae index in 24 hearts with rheumatic mitral regurgitation of 0.48 (Table 3) is not significantly different from that in normal hearts.

Fig. 3 Chordae tendineae indices (CTI) in (a) 26 hearts with subvalvar mitral stenosis, (b) 38 hearts with valvar mitral stenosis, and (c) 15 normal hearts. Horizontal line represents the mean value, and vertical bar the standard error. Triangles denote hearts with restenosis after valvotomy.

Fig. 4 Papillary muscle hypertrophy index (PMHI) in (a) 26 hearts with subvalvar mitral stenosis, (b) 38 hearts with valvar mitral stenosis, and (c) 15 normal hearts. Horizontal line represents the mean value, and vertical bar the standard error. Triangles denote hearts with restenosis after valvotomy.

Fig. 5 Left ventriculogram in patient with prolapsed mitral valve, showing (a) outline of left ventricular ostium, (b and c) anterior and posterior papillary muscles, and (d) prolapsed mitral valve.

Table 3 Chordae tendineae index in rheumatic mitral valve disease in sinus rhythm and atrial fibrillation

<table>
<thead>
<tr>
<th></th>
<th>Stroke index ml/beat per m²</th>
<th>CTI*</th>
<th>Atrial fibrillation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Sinus</td>
<td>Atrial fibrillation</td>
<td>Sinus</td>
</tr>
<tr>
<td>Mitral stenosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Valvar</td>
<td>39 ± 9 (18)</td>
<td>25 ± 11 (19)</td>
<td>0.46 ± 0.05</td>
</tr>
<tr>
<td>Subvalvar</td>
<td>36 ± 7 (10)</td>
<td>25 ± 8 (15)</td>
<td>0.38 ± 0.09</td>
</tr>
<tr>
<td>Mitral regurgitation</td>
<td>34 ± 11 (13)</td>
<td>26 ± 11 (11)</td>
<td>0.48 ± 0.12</td>
</tr>
</tbody>
</table>

*CTI, chordae tendineae index.
Measurements expressed as mean ±standard deviation (number of hearts in parentheses).
With a prolapsed mitral valve, the aberrant shape and position of both leaflets of the mitral valve obscure the outline of the left ventricular ostium and the profile of the papillary muscles is often displaced or deformed (Fig. 5).

Discussion

Post-rheumatic mitral stenosis is initiated by endothelial damage along the line of apposition of the valve cusps and deposition of fibrin on the smooth surface of the valve and in the commissures (Magarey, 1951), but the nature of the ultimate deformity of the chordae tendineae may be influenced by the geometry of the scarred valve. Rusted et al. (1956) examined the pathology of mitral stenosis and placed valves into one of 3 general categories. The categories and relative incidence are (1) scarring of cusps or commissures or both (61%); (2) scarring of cusps or commissures, with shortening of the chordae tendineae (30%); and (3) chordal shortening alone (9%). Harken et al. (1951) classified mitral stenosis as type I, in which there is a rigid, scarred, and diaphragm-like valve with essentially normal chordae tendineae, or type II, in which the valve forms an elastic funnel with fusion of the chordae tendineae. Both Harken et al. (1951) and Rusted et al. (1956) found that the diaphragmatic type was more common than the funnel-shaped valve.

Angiographically, mitral stenosis may appear as a relatively immobile diaphragm-like structure at the ostium of the left ventricle with a slight reduction in the length of the chordae tendineae and chordae tendineae index, and enlarged papillary muscles with high papillary muscle hypertrophy index; or as a less discrete valve shadow with greater reduction in length of the chordae tendineae and chordae tendineae index, and slight enlargement of the papillary muscles with slightly increased papillary muscle hypertrophy index (Fig. 3 and 4). The Harken et al. (1951) classification into type I and type II closely corresponds to these two angiographic patterns.

The possibility that the length of chordae tendineae in mitral stenosis is influenced by the geometry and mobility of the valve is suggested by studies of valvar restenosis. Chordae tendineae and papillary muscle hypertrophy indices in patients with restenosis of the funnel-shaped subvalvar mitral stenosis are generally distributed above the mean for unoperated hearts (triangles in Fig. 3 and 4). This skewed distribution of data suggests that valvotomy increases the mobility of the valve and causes an elongation of the chordae tendineae and enlargement of the papillary muscle. By contrast, symmetrical distribution of chordae tendineae and papillary muscle hypertrophy indices in patients with restenosis of diaphragmatic valvar mitral stenosis about the mean for unoperated hearts suggests that movement and ventricular systolic forces exerted on the valve are essentially unchanged by valvotomy.

The finding that the chordae tendineae may lengthen and the papillary muscles enlarge after valvotomy for subvalvar mitral stenosis may have therapeutic implications. After viewing the thickened and shortened chordae tendineae of subvalvar mitral stenosis (type II, Fig. 2), the prospect of restoration of valve movement and elongation of the chordae tendineae after valvotomy might appear remote. The results of these ventriculographic studies may, however, provide additional incentives for reconstructive rather than replacement valve surgery for subvalvar mitral stenosis.

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References


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