THE SURGICAL AND PATHOLOGICAL ANATOMY OF THE MITRAL VALVE

BY

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The great practical importance that mitral valve disease has now assumed in surgery demands a consideration of the anatomy and function of the normal valve and the pathological anatomy of the diseased valve. Observation on many normal and diseased specimens and direct study of the valve at some 150 operations has made it clear to me that much that is now taught is quite unacceptable. The revision and extension of existing knowledge has often been seen in the development of the surgery of all parts of the body; relatively recently we have seen the great expansion of pulmonary surgery and the corresponding intensive study of pulmonary anatomy and pathology.

In considering valvotomy for the relief of mitral stenosis we are too inclined to concentrate our attention on the valve cusps, forgetting that they are but one part of the valve mechanism. Although the complex nature of the valve mechanism is well known it seems to be largely ignored in dealing with the diseased valve, which is often approached as merely a fibrous, obstructing diaphragm with a small hole that must be made larger. The popular conception of the valve in mitral insufficiency seems to have no logical or scientific basis and is wholly unsatisfactory.

The valve mechanism extends over a distance of some 5 cm., and a lesion may affect all parts of it or one part more severely than another. A lesion at any level can seriously interfere with the mechanism, and it is not always the actual valve cusps that are most seriously affected.

THE NORMAL MITRAL VALVE

The normal mitral valve consists of the atrio-ventricular fibrous ring to which the cusps gain their attachment, of the two cusps, and of the papillary muscles with their chordæ tendineæ. The two cusps are anteromedial and posterolateral, being so arranged that the axis of the valve orifice is directed obliquely forwards from right to left (Fig. 1, 18, and 22).

The *anteromedial* or aortic cusp is much the larger and the more important in that during ventricular systole it receives a great part of the stream of emergent blood which it prevents being driven back into the left atrium and helps to direct into the aortic outlet.

The *posterolateral* cusp fulfills a secondary and supporting part in the closing of the mitral orifice in ventricular systole.

The *papillary muscles* are typically two in number and lie opposite the intervals between the valve cusps; they arise from the ventricular wall at about the junction of the apical and middle thirds, one being anterolateral and one posteromedial. Both muscles are situated in the lower inflow portion of the left ventricle; the anterolateral one arising from the concavity of the anterolateral wall of the
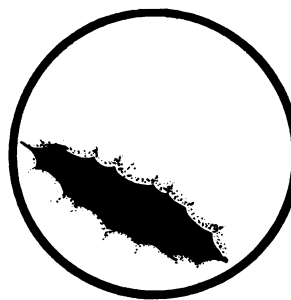

Fig. 1. — Diagram of the arrangement of the mitral cusps.

ventricle and the posteromedial from the junction of the septal and posterior surfaces. Each muscle may be a single papilla or may be split to a greater or lesser degree so that it is either notched at its apex, deeply grooved along its sides, or bifid. Extra muscles may exist. Rusted *et al.* (1951) have recently analysed the arrangement of the paillary muscles in 250 hearts.
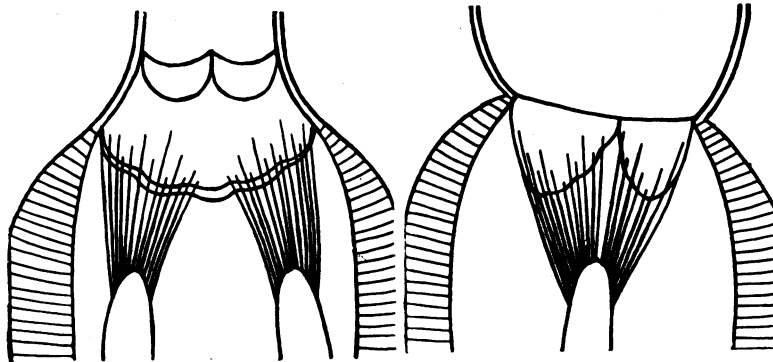

Fig. 2.—Diagram of the arrangement of the papillary muscles and chordæ tendineæ. Each muscle and its tendons controls the corresponding half of the opposing cusps.

The *chordæ tendineæ* pass from each papillary muscle to gain attachment to both valve cusps in such a way that the anterolateral muscle and its tendons control the anterolateral half of the valve, and the posterolateral muscle and its tendons control the corresponding posterolateral half (Fig. 2, 3, and 5). The chordæ tendineæ are of three orders, according to Quain (1929).

(1) Those which are inserted on the free edge of the cusps. They are numerous delicate threads which arise from the other cords near the cusp margin, and often form a fine network before they are attached to it.

(2) Those which are inserted at intervals on the ventricular surface of the cusp near its free edge, which they pass over to the attached border. They are distinctly thicker than the chordæ of group 1, and those attached to any one cusp are derived from two different papillary muscles or from one papillary muscle and the wall of the ventricle.

(3) The short, broad fibres that stretch across the perivalvular groove from the ventricular wall to the under surface of the cusp near its base and run along the cusp a short distance towards its free margin.
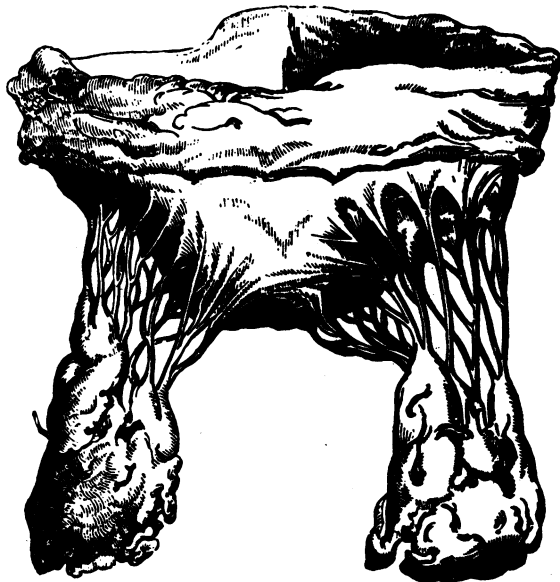
The Critical Areas of Tendon Insertion

Although the arrangement of the chordæ tendineæ can be correctly described in this fashion, the total picture presented is too complex—too complete—for practical purposes. It needs simplification. Study of the arrangement has impressed me with the essential importance of the direct or primary attachment of the tendons.

The primary attachments are two on each

Fig. 3.—The mitral valve. Reproduced from an article in the *Guy's Hospital Reports*, by T. Wilkinson King, 1840.

cusp, being situated about 2 cm. apart; these I have termed *the critical areas of tendon insertion*, for reasons which will become apparent later.

These areas are depicted in Fig. 4 and 5 and are seen to be formed by those tendons that arise from the very summit of the papillary muscles and gain the most direct attachment to the valve cusps. These tendons are often slightly thicker and stronger, and clearly must exercise the most powerful selective action on the cusps since they are in the line of the direct pull of the papillary muscles. The more obliquely placed tendons which gain insertion on each side are of a secondary supporting nature.

The disposition or shape of the cusps is such that there exists, fundamentally, a distal or central horizontal portion and two lateral or receding oblique portions. The critical areas of tendon in-
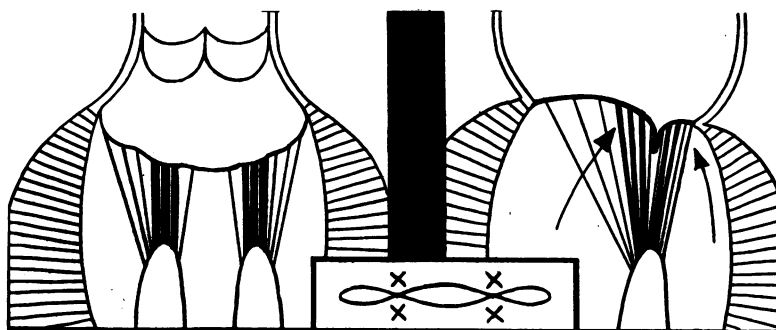

FIG. 4.—Diagram to show the probable mode of closure of the mitral cusps. The thicker chordæ tendineæ are those in the direct line of pull from the papillary muscle to the cusps. The small inset indicates the critical areas of tendon insertion thus formed; in these areas the cusps are held most tightly together.
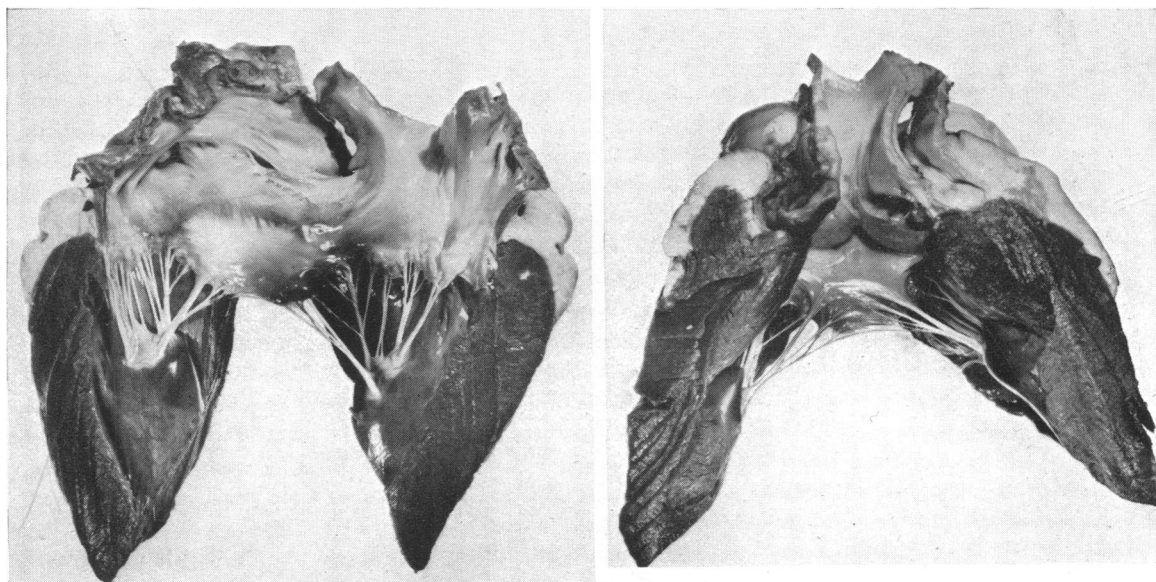

FIG. 5.—Photograph of bullock's heart opened to show the mitral cusps. The arrangement of the tendons from each papillary muscle controlling half of each cusp is well shown. The large, prominent cusp is the anteromedial or aortic, and the critical areas of tendon insertion should be noted with the central blood pathway between.

sertion are at the junction of the horizontal part and the lateral receding parts of the valve cusps. Most of the oblique or secondary tendons are inserted in relation to the lateral parts of the cusps; fewer pass medially to what may be termed the central pathway of the valve (Fig. 3 and 5). This is especially so in the case of the anteromedial cusp. It is true that the shape of the free edge of the cusps may be described as semicircular or semi-ovoid, but the above suggested resolution into a somewhat arbitrary transverse or horizontal distal central portion and the two lateral receding portions is of great practical importance and, I think, aids clarity of understanding.

The Closure of the Normal Mitral Valve

Although the mode of closure of the mitral valve may appear to be fairly obvious and simple, it is a good policy to take nothing for granted but to re-examine the existing conceptions from time to time. Actually, there is room for considerable difference of opinion in the exact mode of closure of the valve.

It is almost certain that the two cusps come together along their opposing surfaces, being driven into this position by the rise in pressure during ventricular systole (Fig. 4). The anterior cusp is 15–18 mm. long at its middle part and the posterior cusp is 10–12 mm. The extent of their probable contact can be judged from the observation (Quain, 1929) that the area of the atrioventricular orifice is 855 sq. mm. in a man and the surface area of the two cusps is 1868 sq. mm. The proportion of valve area to orifice area, therefore, appears to be from 1:1·5 to 1:2·2. The overlap may, in fact, be more than this, because when the ventricle contracts the A-V ring is reduced in circumference and the area of the orifice is correspondingly reduced.

On the other hand Quain does not state the conditions under which these measurements were obtained, although presumably in the cadaver, and we must make allowance for much variation in the degree of contraction and dilatation of the heart and mitral orifice that may be present, whether caused by disease or by *rigor mortis* affecting the heart muscle. Nature is always prodigal in her reserve and it may well be that the overlap described above pertains to quiet resting conditions of the healthy heart, but is greatly lessened during exercise or illness, especially when the heart muscle is affected by the disease.

A possible alternative mechanism of closure is that the orifice is closed entirely by the anteromedial or aortic cusp forcing the much smaller posterolateral cusp against the posterior wall of the ventricle (Fig. 6).
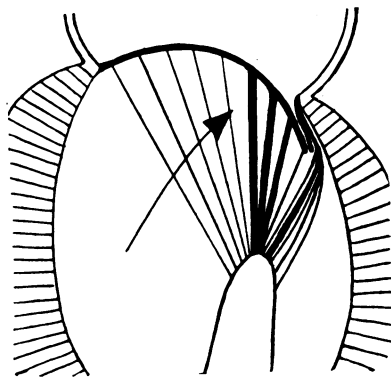

Fig. 6.—Diagram to show a possible alternative mode of closure of the mitral valve in which the posterolateral cusp is forced against the wall of the ventricle by the anteromedial cusp.

It is doubtful if this is the usual mechanism, but it may well happen sometimes; it is unlikely to occur if the A-V ring is at all dilated. In systole the inflow portion of the left ventricle is tightly contracted so that its cavity is almost completely obliterated, but sufficient space remains postero-inferiorly for the hydrostatic pressure to reach the ventricular aspect of the posterolateral cusp and force it away from the wall of the ventricle. The greater part is in contact with the opposing surface of the aortic cusp and its " free " area must be small. When the finger is introduced into the left atrium during an operation for mitral stenosis, the anteromedial cusp is usually very easily felt billowing upwards into the atrium like a sail filled with the wind; it is much more difficult to feel the posterolateral cusp, and, indeed, its presence has often rather to be assumed by judging the distance that lies between the actual mitral orifice and the posterior part of the A-V ring.

Lutembacher (1950) describes the mechanism of closure as a bunching up in which the valve cusps, the chordæ tendineæ, and the papillary muscles come together in such a way that the closure of the orifice is aided and secured by an actual interlocking or inter-opposing of these structures, and he describes and depicts specimens in such a way as to indicate that the partly cockled-up mitral orifice in mitral stenosis actually occupies grooves on the papillary muscles.

This is a conception reached by inspection of the dead contracted heart; from what the finger feels when inside the contracting heart it cannot be a true one. I have felt the mitral valve functioning on some 150 occasions and the aortic cusp at least has always been forced or billowed towards the atrium; it has never been gripped or bunched up within the orifice.

Although it is difficult to feel the posterolateral cusp billowing out, it would be strange if it did not do so in conformity with the aortic cusp, for the very presence of the musculo-tendinous mechanism indicates that this is the action of the cusp in systole. It cannot fail to be forced upwards and backwards in this manner unless it is entirely protected from the high ventricular systolic pressure by complete apposition with the posterior wall of the ventricle.

There is little doubt that the aortic cusp is the main factor in effective closure of the orifice and the posteromedial cusp occupies a subsidiary or adjuvant function in sealing a narrow and probably variable posterior crescent of the orifice. The variability of its apposition with its fellow cusp in varying degrees of cardiac activity or dilatation has already been mentioned.

According to Quain the distance between the apex and the base of the left ventricle does *not* shorten during contraction of the ventricle; the atrioventricular region moves upwards and backwards, but the length of the cavity remains constant. This makes it easier to understand how the comparatively small contraction of the papillary muscles is able to ensure efficient closure of the valve. Actually, all that is needed is quite a small tensing of the chordæ tendineæ to prevent the cusps being blown open into the atrium. The apposition of the cusp surfaces ensures their water-tight closing, but the pressure-resisting mechanism comes from the musculo-tendinous support and its neuromuscular co-ordination with ventricular systole. The mode of insertion of the tendons from one papillary muscle to each of the opposing cusps increases the efficiency of the mechanical closure of the valve.

The important, indeed fundamental, part played by the papillary muscle mechanism was dramatically, albeit tragically, emphasized for me in one unfortunate operative experience (Brock, 1950). After dividing the stenosed orifice in a case of mitral stenosis I tried to reach the aortic valve by passing the index finger around the aortic cusp of the mitral valve. I had just reached the valve with the finger-tip when something gave way and immediately severe mitral regurgitation occurred; a powerful regurgitant stream of blood could be felt entering the atrium, the walls of which could be seen to distend freely with each ventricular systole. Clearly the high ventricular pressure was being transmitted directly to the left atrium. I thought that the aortic cusp had been torn transversely across its substance. The patient, unable to tolerate this severe acute mitral incompetence, died some 48 hours later and autopsy revealed that the anterolateral papillary muscle had been torn from its ventricular attachment; the simple mechanical apposition of the two cusps had not been enough to prevent the severe and fatal regurgitation.

During ventricular diastole the papillary muscles and the tendons are lax and the cusps float open before the stream of blood from the atrium. During ventricular systole the valve cusps are flung tight together and the papillary muscles and tendons become taut and straight; the maximum, the most powerfully supported, closure must occur in relation to the critical areas of tendon insertion already described. At the height of systole the tendons passing from the summit of each papillary muscle direct to these two critical areas on the valve cusps must be tense and parallel and very close to one another (Fig. 4); in contrast with during diastole when they are lax and directed outwards towards the wide open margins of the orifice. At these two critical areas the cusps must be held together very tightly indeed; the significance of this will become apparent when we consider the state of the diseased valve. Sokoloff *et al.* (1950), as a result of a study and survey of the chordæ tendineæ in 200 hearts of all ages, have shown that the more centrally placed chordæ of the anterior cusp are thickened and more powerful in every heart and this can be noted at all ages and in animals (bovine) when rheumatic infection can be excluded. The changes are less marked in the chordæ of the posterior cusp, as would be expected.

The size of the mitral orifice. I have always been intrigued by the difference in size between the A-V orifice and that of the aorta or pulmonary artery. The explanation that first suggests itself is that this is related

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