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# THE MITRAL VALVE

## A Pluridisciplinary Approach

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# 7 Pathology of the Mitral Valve

Introduction to Plastic and  
Reconstructive Valve Surgery

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The development of techniques for mitral valve reconstruction presupposes a precise knowledge of the lesions encountered. The present study analyzes the anatomic findings at autopsy of 100 human hearts with either rheumatic or dystrophic noncalcified valvular disease, and those observed at the time of operation in a series of 400 mitral valve repairs. Anatomic study consisted of a precise measurement of the various valve structures; intra-operative observations allowed us to analyze accurately the shape of the annulus and the physiology of the leaflets of the living, beating heart.

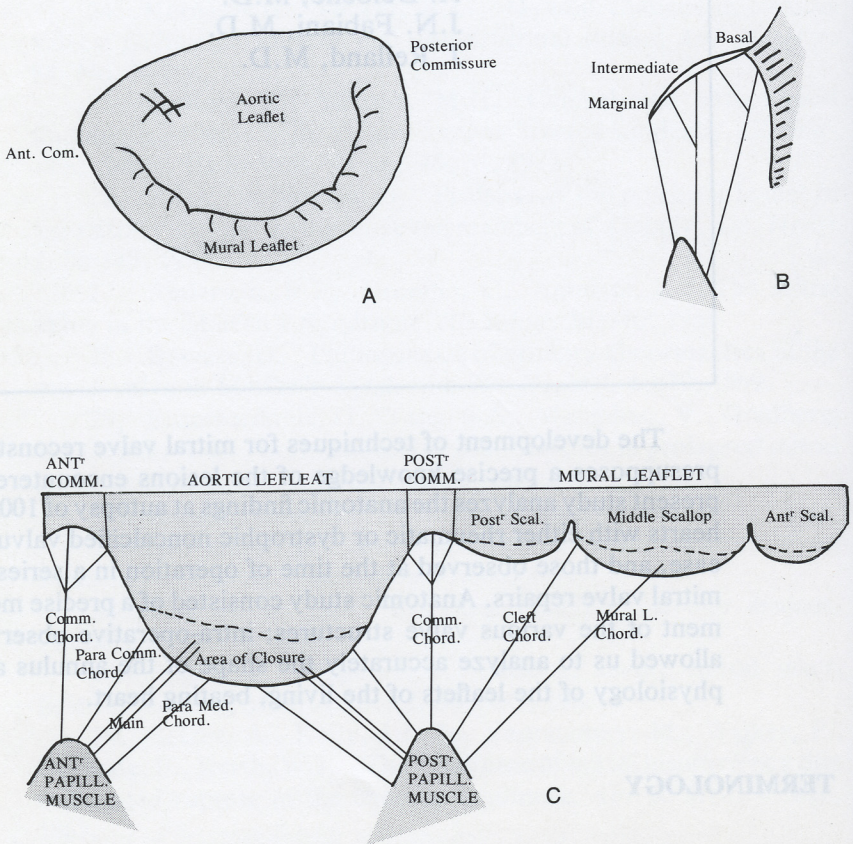
## TERMINOLOGY

Since several terms are used in the literature to define the same structure of the mitral valve, it is necessary to begin by reviewing the terminology used in the present study. Aortic (anterior) and the mural (posterior) leaflets are separated by antero-septal and postero-medial



commissures, which can be identified by the tips of the corresponding papillary muscles,<sup>9,10</sup> and by the commissural chordae.<sup>7</sup> The commissural chordae arise from the tip of the papillary muscle as a single stem that branches radially in a fan-like fashion to insert into the free margin of the commissural regions<sup>6</sup> (Fig. 1).

The anterior and posterior leaflets present a rough zone which is the area of contact of both leaflets during systole. In this study, this zone is called the "closure area." The margin of the mural leaflet generally presents two indentations, giving rise to a scalloped appear-



**Figure 1.** Schematic representation of the normal anatomy of the mitral valve apparatus. A—View from left atrium. B—Cross sectional view showing site of insertion of the chordae to the leaflet. C—Mitral apparatus opened showing the various components.



Mural leaflet chordae: Chordae of the middle scallops—thin chordae of the marginal intermediate or basal type. Cleft chordae corresponding to the indentations of the leaflet—thin chordae of the marginal type.<sup>6</sup>

## MATERIAL AND METHODS

The 100 human hearts with rheumatic or dystrophic valvular disease were compared with 50 normal hearts which served as reference. The average weight of the normal hearts was 340 gm. The 100 pathological hearts were divided into four groups.

Rheumatic valvular insufficiency: 30 specimens—average weight: 620 gm. This group was characterized by major insufficiency with a commissural fusion of less than 5 mm and without ruptured chordae.

Rheumatic combined valvular insufficiency and stenosis: 30 specimens—average weight: 580 gm. This group consisted of cases of non calcified mitral valve disease with associated insufficiency and mild stenosis (1 to 2 finger breadths).

Dystrophic mitral insufficiency: 20 specimens—average weight: 630 gm. This group included cases of mitral valve insufficiency with no previous history of rheumatic fever, and without typical rheumatic lesions, i.e., fusion of the commissures, shortening and fusion of the chordae, thickening and shrinkage of the leaflets.

Dystrophic mitral insufficiency with ruptured chordae: 20 specimens—average weight: 560 gm. Ruptured chordae of rheumatic origin or due to bacterial endocarditis were excluded from this group. The ruptured chordae belonged to the mural leaflet in 11 cases, to the aortic leaflet in 5 cases, and to both leaflets in 4 cases.

The hearts used in the anatomic study were obtained at autopsy. In contrast to most studies, no distinction was made between the measurements on the basis of sex, because it was felt that the differences were related more to body size than to sex. The average height of the subjects studied measured 1.66 m, with a range of 1.50 m to 1.82 m ( $\pm 10\%$ ). All measurements represent averages which have been rounded off to the nearest millimeter.

Intra-operative observations were made on a series of 400 mitral valve repairs carried out under extra-corporeal circulation. The mitral valve was approached through a median sternotomy and a left atriotomy. The heart was kept beating. The shape of the annulus was analyzed while the aorta was unclamped. The motion of the leaflets was observed, with the aorta crossclamped to avoid air embolism.



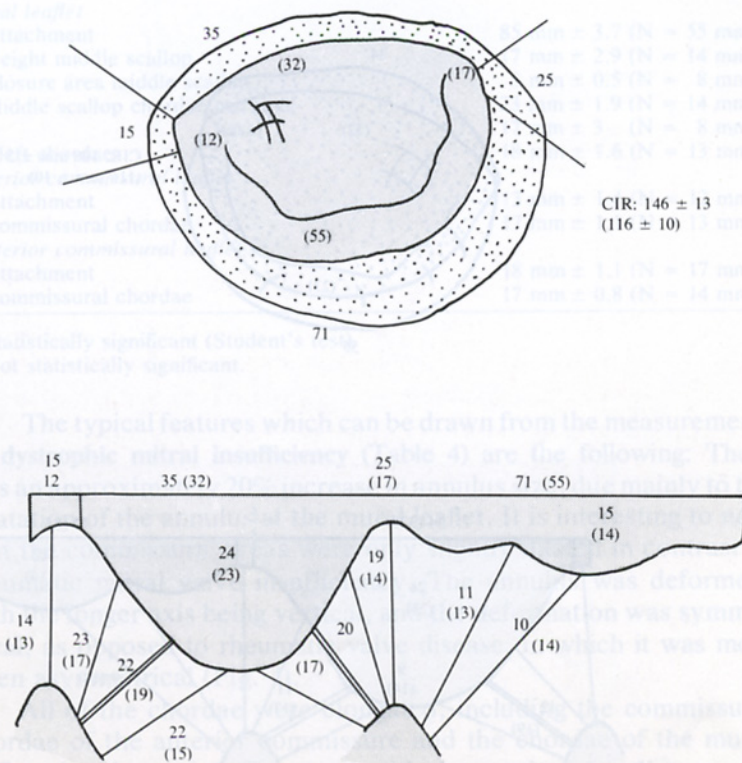




missures. The shape of the annulus was changed: the main axis became vertical instead of horizontal, and in most cases the deformation was asymmetrical, the dilatation being more marked at the posterior commissure. With the heart beating, there was no change in the shape of the orifice and no reduction in its size during systole.

Table 3 shows that, in the case of **combined mitral valve disease and insufficiency**, the size of the annulus was significantly reduced due to the retraction of the commissures and the mural leaflet. The aortic valve attachment was normal or even slightly dilated. The valvular tissue was thickened and retracted, particularly at the mural leaflet and the commissure (Fig. 3).

All of the chordae were shortened, thickened, or fused. Some were selectively elongated. It is of some practical importance to point out that thickening and rigidity of the mural leaflet were often due



**Figure 2.** Rheumatic valvular insufficiency. Measurements of the various structures in comparison with normal measurements shown in parentheses.









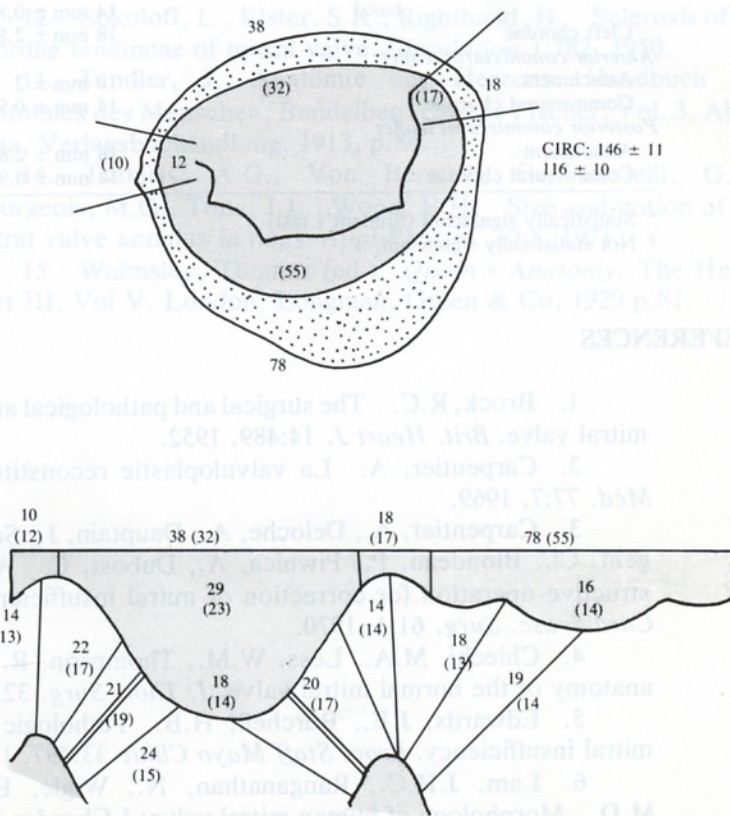




preceding group. They show that both groups represent different evolutions of the same pathological process (Fig. 5).

## CONCLUSION

This study is, to our knowledge, the first to present detailed, numerical data on the pathological findings of usual causes of mitral valve insufficiency. The differentiation of these heterogeneous lesions into their components provides a rational basis for the development of a variety of new mitral-valve reconstructive techniques and for the specific indications governing their use in each of the disease groups.



**Figure 5.** Dystrophic mitral insufficiency with ruptured chordae. Measurements of the various structures in comparison with normal measurements shown in parentheses.

**Table 5**  
**Dystrophic Mitral Insufficiency with Ruptured Chordae**

Circumference: 144 mm $\pm$ 5.1 (N = 116 mm) p 0.005	
<i>Aortic leaflet</i>	
Attachment	38 mm $\pm$ 2.2 (N = 32 mm)*
Height	29 mm $\pm$ 1.5 (N = 23 mm)*
Closure area	18 mm $\pm$ 1.2 (N = 14 mm)†
Main chordae anterior	21 mm $\pm$ 0.3 (N = 19 mm)*
posterior	20 mm $\pm$ 1.9 (N = 17 mm)†
Paracommissural anterior	22 mm $\pm$ 1 (N = 17 mm)*
Paramedial anterior	24 mm $\pm$ 0.9 (N = 15 mm)*
<i>Mural leaflet</i>	
Attachment	78 mm $\pm$ 3.6 (N = 55 mm)*
Height middle scallop	16 mm $\pm$ 1.5 (N = 14 mm)†
Closure area middle scallop	9 mm $\pm$ 0.9 (N = 8 mm)†
Middle scallop chordae marginal	19 mm $\pm$ 1.4 (N = 14 mm)*
basal	14 mm $\pm$ 0.8 (N = 8 mm)*
Cleft chordae	18 mm $\pm$ 2.8 (N = 13 mm)*
<i>Anterior commissural leaflet</i>	
Attachment	10 mm $\pm$ 2 (N = 12 mm)†
Commissural chordae	14 mm $\pm$ 0.9 (N = 13 mm)†
<i>Posterior commissural leaflet</i>	
Attachment	18 mm $\pm$ 2.8 (N = 17 mm)†
Commissural chordae	14 mm $\pm$ 0.9 (N = 14 mm)†

\* Statistically significant (Student's test)

† Not statistically significant.

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Exclusive of dynamic heart disease, the causes of mitral valve disease in children are numerous and are related to local cardiac abnormalities as well as generalized disease processes. The pathological changes in the mitral valve may be confined to that structure or may be part of a life generalized anomaly involving several other cardiac structures. Of note in this group is papillary muscle dysfunction which is secondary in conditions such as aortic stenosis or endocardial fibroelastosis. When the heart is part of a generalized disease process involving other organs of the body, such as in the inherited disorders of metabolism, the mitral valve is commonly the predominant cardiac structure involved. In these generalized conditions, the mitral valve involvement in some cases may be an incidental finding at autopsy; in other cases, the incompetent valve may be the cause of congestive cardiac failure and death.

Irrespective of whether the mitral pathology is isolated or part of a cardiac syndrome or some generalized condition, the pathological features are best understood by classifying the lesions according to