A STUDY OF ANATOMY AND VARIATION OF THE HUMAN MITRAL VALVE APPARATUS IN AUTOPSY SPECIMENS

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CERTIFICATE

This is to certify that Dr.T.M.PONNUSAMI postgraduate student (2003 - 2006) in the Department of cariothoracic surgery, Government General Hospital Chennai & Madras Medical College, Chennai - 03, has done this Dissertation of "A STUDYOF NORMAL ANATOMY AND VARIATIONS OF THE HUMAN MITRAL VALVE APPARATUS IN AUTOPSY SPECIMENTS under my guidance and supervision in partial fulfillment of the regulations laid down by TheTamil Nadu Dr.M.G.R. Medical University, Chennai, for MCh cardiothoraci - Branch II examination to be held in August 2006.

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CONTENTS

INTRODUCTION

One hundred human hearts from autopsies are studied to clarify controversies in the literature about commissures, slits, chordae and leaflets of the mitral valve. This study revealed that no 2 hearts are alike in the morphology of commissures, slits and scallops. We have designed perpendiculars drawn from the annulus to the free edge at the shortest height of the mitral valve on either side of the aortic leaflet as the anterolateral (AL) and posteromedial (PM) commissural lines. These lines divide the mitral valve precisely into aortic and mural leaflets. Slits in the leaflets are easily identified by the dipping in of the free edge into the cusp tissue. There are no slits in the aortic leaflet. Ninety-eight hearts has slits in mural leaflet varied from one to five. Unlike the relatively static and straight annulus of the aortic leaflet. The curved annulus of the mural leaflet contracts and changes in contour during systole. Hence slits are necessary in the mural leaflet to help it fold and adapt to the reduced orifice during systole and unfold during diastole. In addition to this coarse adjustment of the mural leaflet, both leaflets are pleated due to "hooding up" of the leaflet tissue between chordal attachments during systole, providing the fine tunning to enable the leaflets to adapt themselves to the reduce systolic orifice. The nodular appearance (rather than a straight ridge) of the line of apposition of the leaflet is evidence of this pleating mechanism during valve closure. Slit lines, designed as perpendiculars drawn from the summit of the slits to the annulus, are used arbitrarily to divide the mural leaflet into 2 to 6 scallops. When slits are absent there are no scallops. The slits and scallops are best serially numbered counterclockwise from the surgeon's view through the atrium. The scallops immediately behind the commissural lines, customarily labeled as the commissural scallops, are best defined as mural leaflet tissue between the AL or PM

commissural line and the closet AL or PM slit line. All commissural scallops are seen in 81 hearts and PM commissural scallops in 76 hearts. There is no consistent fan-shaped chordal pattern at the commissural lines or at the slits. However the chordae arising from each papillary muscle group, in total, from a fan, reaching out to the corresponding adjacent halves of the 2 leaflets, restraining their splaying out in diastole and upward bulge during systole. These findings are relevant to the pathogenesis of mitral valvular disease and reparative procedures.

The Central Mitral Plane passing through the middle of the aortic and mural leaflets divides the chordopapillary support of the mitral value into anterolateral and posteromedial halves. The papillary muscles of the mitral valve are studied in 100 human autopsy hearts collected at random. the anterolateral papillary support has 1 belly in 67 hearts, 2 in 27, 3 in 4, 4 in 1, and 5 in 1 heart. Likewise, the postermedial papillary support has 1 muscle belly in 50 hearts, 2 in 36, 3 in 11, and 4 in 3. The single papillary muscle is conical, mammilated, flat topped, grooved, stepped, wavy, arched, sloped or saucerized. When there are two bellies they presented as two tiered, interlinked, parallel, arched, V, Y, or H configuration. Three papillary muscles formed a parallel, interlinked arrangement with the third belly separately. When four or five bellies existed, they are parallel or interlinked. In the anterolateral and posteromedial group, the papillary muscle bellies are mostly intraluminal in 14% and 11%, mostly intraluminal with the tip anchored in 19% and 28% equally sessile and intraluminal in 54.5% and 41.5% mostly sessile in 12.5% and 19.5% respectively. In the anterolateral group 19% of papillary muscle bellies arises from the upper third of the ventricle, 70.5% from middle third, and 1.5% from lower third, the corresponding figures for postermedial group are 6% 92.5% and 1.5%, respectively. Four to 22 chordae originated from the anterolateral papillary group, ending

in 14 to 72 chordale arises from the posteromedial papillary group ended in 12 to 89 leaflet insertions. The chordae in each group are best considered in to as a fan. The configuration of the fan is unique in each heart. Imaging techniques need to be refined to outline these variations more precisely. the relevance of chordopapillary variations in rheumatic heart disease, reparative procedures, papillary muscle dysfunction, mitral value prolapse, mitral valve replacement, and use of mitral valve homograft for mitral / tricuspid replacement is discussed.

MATERIAL AND METHODS

One hundred normal human autopsy hearts collected at random are studied. The age and sex of persons are not known. Looking through the left atrium, the mural leaflet is divided between its anterolateral and posteromedial chordopapillary support. A few false chordae connecting the two groups of papillary muscles are divided to splay the valve open with the aortic leaflet in the middle, the anterolateral half of the mural leaflet with anterolateral group of papillary muscles on the left, and the posteromedial half of mural leaflet with posteromedial group of papillary muscles on the right.

The number of papillary muscle bellies in each varied from 1 to 5. When single, the shape and size of the muscle belly varied. When there are 2 to 5 muscle bellies they form various configurations.

The papillary muscles varied in the extent of protrusion into the ventricular cavity and adherence to the ventricular wall. When mostly intraluminal, the tip of the muscle may be anchored to the ventricular wall by a small mjuscle band. Commonly, papillary muscle are related to the middle third of the ventricle.

The variations in number of chordae arising from each group of papillary muscle is studied. The number of chordae inserting into the free edge of the leaflets in the corresponding half of the valve is also noted.

In two hearts, the anterolateral papillary muscle remained muscular upto the annulus also, the strut chordae to the aortic leaflet in two hearts had separate muscular bellies.

AIM OF STUDY

AIM : Study of anatomy and Anatomical variations of mitral valve and nature of sub valvular

apparatus helps to preserve anatomical position of mitral valve in its natural position by preserving all relations of mitral valve in repair of mitral valve instead of replacing with mechanical valve.

The mechanical valve replacement is a long procedure with life long anticoagulant like warfarin.

In view of, life long management of mechanical valve with anticoagulants it is cumbersome for Indian Poor People. So, it is well advised to preserve mitral valve with repair instead of replacement with mechanical valve. So by studying the normal anatomy of mitral valve apparatus we can preserve the natural parts of mitral valve in its normal anatomical position.

DEVELOPMENT OF MITRAL VALVE

The atrioventricular valves are complex entities made up of the annulus, the leaflets, and the supporting tension apparatus, the latter comprising the tendinuous cords and the papillary muscles. The leaflets are hinged from the annulus, which is an integral part of the atrioventricular junction. In the definitive heart, the tricuspid and mitral valves are separated by septal structures, which are absent in hearts having a common atrioventrcular junction.

To understand the development of these valvular complexes, particularly the presence in malformed hearts of a common atrioventricular valve, valvar formation must be examined in the context of cardiac development as a whole. In addition to cardiac septation, interdependent and mutually generative processes closely linked to the formation of the atrioventricular valves include the mechanisms of connection of the right atrium to the right ventricule, and incorporation of the subaortic outlet into the left ventricle, and incorporation of the subaortic outlet into the left ventricle. In this review, we will seek to integrate these themes as we describe how an initially unseptated and valveless tube is changed into a complex four chambered organ, with the chambers and arterial trunks separated by a system of one-way valves, concentrating attention of the atrioventricular valves. We will also speculate on how normal development can be perturbed to produce some of the lesions seen in modern surgical practice.

Primary Heart Tube

After the third week of gestation, a lumen forms within the primary endocardium of the heart that, when enveloped by myocardial cells formed in the primary cardiac crescent,

becomes converted into the initially valueless and inverted Y-shaped tube that initiates the circulation. With time, this primordium will become the larger part of the left ventricle and part of the atrial chambers. The future right ventricle and outflow tract, along with the remainder of the left ventricle and the atriums, take their origin from a spatially distinct area, known as the secondary heart field, with the cells migrating at a later state from the area to become integrated within the arterial and venous poles of the developing heart tube.

The atrioventricular junction, the seat of the future atroiventricular valves, comes into prominence following rightward looping of the heart tube, after the 25th day of gestation of development. Looping occurs as the initial attachment of the tube to the body wall disappears, this connection being the so called dorsal mesocardium. The atrial remnant of this structure then serves as the site of entry of the pulmonary vein and the vestibular spine during the 7th week. As we will see, the spine then plays a crucial role in atrial septation. Subsequent to looping, the developing heart takes on the more characteristic three-dimensional appearance of the mature organ. By the end of the 5th week, the developing ventricles have become visible as pouches that balloon from the primary tube, with the primordium of the muscular ventricular septum also being visible. At this stage of development, the primordial left ventricle supports the developing right ventricle provides most of the muscular support for the developing ventricular outflow tract. The lumen of the atrioventricular canal is largelyoccupied by two large mesenchymal masses, the superior and inferior atrioventricular endocardial cushions. Initially unfused, the cushions face each other within the canal, leaving slits on each side between their edges and the lateral margins of the canal. These slits will eventually expand to become the right and left atrioventricular junctions. Even before fusion of the cushions, the

right-sided slit provides continuity between the developing right atrium and right ventricle through the lumen of the primary heart tube, with the inner curve of the tube forming the roof of this communication, which is called the primary interventricular foramen. Studies of human embryos stained with an antibody revealed that the myocardium surrounding the foramen is distinct from the remainder of the primary myocardium with part becoming transformed into the atrioventricular node and bundle. It is expansion and remoulding within this region of primary myocardium, known as the primary ring, that provides the substrate for formation of first the right ventricular inlet, and then the tricuspid valve.

This right side of the atrioventricular canal expands across the developing muscular ventricular septum as the superior and inferior cushions fuse within the lumen of the canal, their rightward margins then becoming draped across the crest of the septum. Parts of the fused cushions, nonetheless, remain to the left side of the septal crest, with their bulk protruding in to the cavity of the left ventricle, where they will form the aortic leaflet of the mitral valve. The parts spanning the crest of the developing muscular septum, which are the first parts to fuse, will form the larger part of the membranous septum. This structure, along with contributions from the outflow cushions, will eventually partition the aorta into the left ventricule.

Fate of the Primitive Atrioventricular Canal

Fusion of the atrioventricular endocardial cushions during the sixth week of development divides the atrioventricular canal into the primordiums of the right and left atrioventricular junctions, to which the developing leaflets of the mitral and tricuspid valves will eventually be anchored. Septation of the canal by the cushions goes hand in hand with the

beginnings of not only ventricular, but also atrial septation. The earliest indication of atrial septation is the downgrowth of the primary septum, or "septum primum", from the atrial roof. As it grows towards the cushions within the atrioventricular canal, this primary septum carries a cap of mesenchyme on its leading edge. At the same time, contiguous with the right margin of the dorsal mesocardial; connection, a further mass of mesenchyme grows into the heart at the level of the base of the developing atrial septum. This latter structure is the so-called " spina vestibuli", or vestibular spine, which is separated from the mesenchymal cap.

The tissue of the spine, together with the mesenchymal cap clothing the leading edge of the primary atrial septum, merges with the atrial margins of the fused atrioventricular endocardial cushions to close the primary atrial foramen, or "ostium primum". The vestibular spine itself then muscularizes to form the thick base of the atrial septum. By this time, the initial musculature of the atrioventricular canal is becoming incorporated into the now divided atrioventricular junctions as the atrial vestibules. The final separation of the musculature from the ventricular walls does not occur until much later in development, when the fibro-adipose tissues of the atrioventricular grooves separates the atrial and ventricular muscular segments at all sites other than the location of the bundle of His. The point of penetration of the bundle of His marks the site within the septal components of the initial atrivoentricular canal musculature.

Failure of fusion of the superior and inferior cushions is the process usually held responsible for producing atrioventricular septal, or "canal", defects. Indeed, for many years this group of malformations is labeled as "endocardial cushion defects". Recent research, however, suggests that it is deficiency of the vestibular septum, that is responsible for the

common atrioventricular junction, this feature being the halmark of the malformations. Failure of this mesenchymal front to contribute to the base of the developing atrial septum permits the primary foramen to remain patent and, at the same time, ensures persistence of the common atrioventricular junction. When a valve is eventually formed in the setting of this common junction, it bears scant morphologic resemblance to the normal mitral and tricuspid valves. This is because the space between the bridging leaflets formed from the superior and inferior atrioventrucular cushions in an integral part of the valvar orifice. The location of this space reflects the arrangement seen very early during normal development. It is also case that, subsequent to separation of the left atrioventricular junction, clefts of varying depth can be found in the aortic leaflet of the otherwise normally formed mitral valve. Such clefts can also be found when the mitral valve straddles through a ventricular septal defect opening to the outlet of the right ventricle. Thus, although the zone of apposition found between the left ventricular components of the bridging leaflets in a heart with an atrioventricular septal defect with common atrioventricular junction, like the cleft of the aortic leaflet of an otherwise normal mitral valve, exists because of failure of fusion of the atrioventricular endocardial cushions, it is only the leftward tips of the cushions that have failed to fuse when there is an otherwise normally structured mitral valve.

Development of the Mitral Valve

Formation of the normal mitral valve not only required division of the atrioventricular canal, but also cannot proceed until the developing aorta becomes committed to the left ventricle. In the definitive heart, almost always there is fibrous continuity between two of the leaflets of the aortic valve, and one of the leaflets of the mitral valve. The leaflet of the mitral

valve in continuity with the aortic root named the aortic leaflet, thus distinguishing it from the mural leaflet, which is hinged from the parietal atrioventricular junction. As well will see, these morphologic difference in the hinges of the leaflets reflect their development heritage.

The building blocks of the valvar leaflets are the endocardial cushios. Formation of the definitive valve, therefore, requires reorientation of the newly separated left atrioventricular junction, which expands in inferior direction. This inferior reorientation occurs concomitant with incorporation of aorta into the outlet part of the left ventricle. Blood from the left ventricle initially reaches the developing aorta through the primary interventricular foramen, with the fused atrioventricular cushions forming the left ventricular border of the roof of this pathway. As the outflow cushion fuse, and muscularize to separate the subpulmonary infundibulum from the subaortic outlet, they also fuse with the crest of the muscular ventricular septum, thus walling the developing aortic valve into left ventricle. The aortic root then occupies the space that has appeared, concomitant with the expansion and reorientation of the left atrioventricular junction, between the ventricular septum and the fused left ventricular components of the atrioventricular cushions. This space forms a bay on the ventricular aspect of the atrioventricular cushions. When the aorta is first walled into this newly created by within the left ventricle, the myocardium of the inner heart curve continues to separate the developing aortic valver leaflets from the cushions fusing to form the aortic leaflet of the mitral valve. Only subsequent to the completion of septation does this muscle disappear, thus establishing the definitive arrangement of fibrous continuity between the aortic leaflet of the mitral valve and the noncoronary and left coronary leaflets of the aortic valve.

With this remoulding, the valvar orifice changes its shape from a slit to a crescent within

the ventricle, the two ends of this expanded crescent are associated with compacting columns in the trabecular, or spongy, layer of the ventricular muscle. These columns, which will form the papillary muscles, are positioned to support not only the ends of the lateral cushion, but also the distal ends of the fused atrioventricular cushions. The layer of spongymyocardium that initially supports the lateral cushion, however, will subsequently disappear. As it does so, the endothelially derived lateral cushion itself becomes transformed into the mural leaflet of the mitral valve. The liberated myoardium that initially joined the cushion to form papillary muscles will also disappear with time, the myocardial, cells being replaced by fibrous tissue. A similar process occurs at the interface between the edges of the developing aortic leaflet and the tips of the papillary muscles, with fibrous tension apparatus eventually replacing the myocardium.

Excessive or abnormal compactness of the trabecular layer of the developing ventricular myocardium is responsible for producing the so-called "parachute" deformity of the valve, either with a solitary papillary muscle supporting the entirety of the valvular complex, with a leash of cords fanning out from this signal locus, or with incomplete formation of one of the two papillary trabecular columns. Failure of formation of the tendinous cords from the original myocardial primordiums results in the "hammock" or "arcade" lesions of the mitral valve, with the musculature extending from the edge of the leaflets to the papillary muscles.

THE NORMAL MITRAL VALVE APPARATUS

The normal mitral valve apparatus is composed of various structures including the mitral ring, the valve proper, the chordae tendineae, and the papillary muscles.

The Mitral Ring

The mitral (atrioventricular) ring or annulus in an important part of the skeleton of the heart. It is an anteriorly incomplete, circular, and fibroelastic structure. It represents the dividing line between the atrial and ventricular musculature from the eighth week of intrauterine life and appears in the embryonic heart as a result of the crowding together of the connective framework of the organ. The ring's upper border is the point of origin of the ventricular muscular. It has recently been shown that the atrioventricular annulus decreases its diameter during each systolic contraction. This event, which takes an effective part in the closure of the normal mitral valve, is probably the result of the systolic contraction of the deep bulbo and Sino Spiral muscles with the superficial bulb spiral muscle assisting in a minor role. An impression is gained that the orifice is completely obstructed during the late stage of the cardiac systole by the sphincter like action of the annulus.

TABLE - I

MEASUREMENTS (IN CM) OF NORMAL MITRAL VALVES

(100 HEARTS)

Structures Measured	MEN				WOMEN			
	Height		Breadth		Height		Range	
	Average	Range	Average	Range	Average	Range	Average	Range
Aortic Leaflet	2.4	3.2-1.9	3.7	4.5-2.5	1.2	2.7-1.8	3.3	4.2-2.4
Ventricular Leaflet	1.4	2.5-1.0	3.3	4.1-2.5	1.2	2.4-0.8	3.0	3.6-2.3
Anterior Accessory Leaflet	1.1	1.8-0.8	1.5	1.8-1.1	1.0	1.3-0.7	1.2	1.6-1.0
Posterior accessory leaflet	0.9	1.2-0.6	1.1	1.5-0.8	0.9	1.0-0.7	0.8	1.2-0.7
Anterior junctional tissue	0.8	1.2-0.6	1.7	2.4-0.7	0.7	1.1-0.6	1.5	2.1-0.7
Posterior junctional tissue	0.7	0.9-0.5	1.3	1.8-0.7	0.6	0.8-0.4	1.2	1.6-0.7
Circumference of valve ring	-	-	10.0	11.5-8.5	-	-	9.0	10.5-8.5

The outer aspect of the mitral annulus constitutes the deepest part of the atrioventricular groove on the external surface of the heart. The mitral valve takes origin as a continuous veil from the inner aspect of the ring and inserts around tee entire circumstance of the mitral orifice.

In this study we found that the circumstance of the mitral annulus is in average 10cm (range: 8.5 to 11.5 cm) in the male specimens and 9 cm (range: 8 to 10.5 cm) in the female specimen (Table 1). The readings obtained are very seldom found at variance with these figures.

THE MITRAL VALVE PROPER

The mitral valve is represented by a continuous veil of valvular tissue attached as a muff to the entire circumference of the atrioventricular annulus. Its free edge is split indentations, none of which reaches the mitral annulus. These clefts divide the indentations, none of which reaches the mitral annulus. These clefts divide the valvular tissue into two major leaflets which are always present and into two minor accessory cusps not invariably represent. These minor cusps, when present, are at the area of the junctional tissue.

The valve tissue consists of a fibroelastic texture covered on the atrial and ventricular aspect by a coat of endocardium. The so-called fibrous skeleton which gives strength and shape to the valve is furnished by a layer of connective tissue protruding from the inner aspect of the mitral ring. Important contributions to the valve-supporting framework are the chordal insertions. Rusted and his associates noted that the chordal insertions passes through the substance of the leaflet for a considerable distance. This particular arrangement, as pointed out by Mayo Clinic Group, is often seen on the transiluminated posterolateral leaflet, but more often a fibrous band running along the margin of closure is seen in this leaflet. This fibrous structure can be considered as the point of insertion of the chordae. Neither the junctional tissue nor other additional valve leaflets show any such arrangements of their connective layers.

The two major cusps are attached to the ring in such a way as to be in anteromedical and posterolateral locations. The anteromedial cusp has been referred to as the anteromedial, aortic, septal or greater leaflet. The posterolateral has been designated as the posterlateral, ventricular or lesser leaflet. The major axis of the mitral orifice is consequently directed obliquely forward

form right to left.

The Anteromedial or Aortic Leaflet

The aortic leaflet is the most important from the anatomical as well as the physiological standpoint. Table-I shows the linear measurements of the various value leaflets. Table II lists the surface measurements of the same structures, obtained by planimetric integration of the removed valvular tissue. The aortic cusp is by far the largest. It exceeds every other leaflet in height and breadth at the mitral ring. Its surface area gives a better idea of the importance of this cusp, because it is equal to almost one-half of all tissue surface area.

TABLE - II

Structures Measured	**MEN (8 Subjects)**		**WOMEN (8 Subjects**	
	Average	**Range**	**Average**	**Range**
	651	833-427	563	714-413
Ventricular leaflet	384	532-270	334	456-413
Anterior accessory leaflet	227	257-193	203	220-183
Posterior accessory leaflet	136	148-110	120	128-112
Anterior junctional tissue	171	200-129	144	171-125
Posterior junctional tissue	92	110-70	78	94-67
Valvular tissue area	1398	1770-1000	642	1518-970
Mitral orifice area	793	1020-572	642	875-490

The anteromedial cusp is roughly triangular in shape and, in our experience, its free edge has never been found split.

It is the only dividing structure between the mitral and the aortic orifice. Directly inferior to the aortic canal, it constitutes an integral part of the outflow tract of the left ventricle. Due to this anatomical location, a considerable portion of its function is believed to be the direction of the flow of blood toward the aorta. This has been described by Harken as the aortic baffle. In other words, the leaflet apparently acts as a watershed which deflects the blood toward the aorta in the ejection of the ventricular contraction.

Close anatomical observation of the normal heart as well as experimental investigation of the valvular function would not be in favour of this view. The leaflet has the greatest importance in the closure of the mitral valve, but it has no baffling action on the blood flow, indeed, the blood driven forcefully from the inflow into the outflow tract has no other outlet but the aortic orifice, since the mitral valve is, at this stage of the systole, hydraulically closed. The physiological lines of progression upon which the blood is forced out into the aorta run directly from the apex of the heart to the aortic canal. The contribution of the anteromedial leaflet to the information of these lines of direction is certainly not greater than that of the interventricular septum or the anterior left ventricular wall. The last mentioned two structures together with the septal cusp constitute the walls of the outflow tract of the left ventricle. If a defect is produced in the aortic leaflet, it is possible to do in experimental surgery, a great valvular incompetence ensues since here and there are no compensatory mechanisms likely to limit the mitral reflux.

Likewise, a defect of the interventricular septum will permit on escape of blood into the right ventricle. The two situations, the mitral reflux and the escape of blood through an interventricular septal defect, are indeed dynamically analogous since both are governed mainly by the intracavity pressure. On the other hand, the muscular fibers of the outflow tract appear to play an important role in giving a definite direction to the blood flow. This fact is demonstrated by the position of the left ventricle in the cases of prevailing hypertrophy of the left ventricular outflow tract.

The Posterolateral or Ventricular Leaflet

The smaller ventricular leaflet is not as important as the aortic leaflet, but by no means should it be disregarded in the mechanism of valve closure. This is confirmed by experimental and clinical facts. The posterolateral cusp is roughly equal to one-third of all the valvular tissue surface area. Its breath at the line of insertion is only a few millimeters less than that of the aortic leaflet, while its height is almost one-half that of the anteromedial cusp (Table 1). The contour is usually quadrangular with sloping sides and its free edge is often notched. However, the notching is seldom deep enough to be mistaken as a cleft separating the poseterolateral leaflet from an additional cusp. The importance of the ventricular leaflet in the normal mitral valve to have been generally under estimated.

It was originally thought that the complex mechanism of the normal mitral valve function could be simply described as an unpretentious or moderate systolic and diastolic swinging movement of the septal leaflet. According to the see, the contraction of papillary muscles would cause the anteromedial leaflet to act like a "curtain stretching obliquely from the

right half of the mitral ring an across the orifice, which would remain entirely hidden." At the same time, the posterolateral ventricular wall "would march" toward the septal leaflet and come into contact with its free margin. In diastole, the aortic leaflet would swing back opening the valve. Consequently, the ventricular leaflet appeared to be a worthless structure as far as the mechanism of the mitral valve closure was concerned. The perivalvular space was thought to be entirely obliterated in systole by the posterior ventricular wall moving toward the aortic cusp.

The posterior leaflet is sqeezed and butterssed by the posterolateral wall of the left ventricle in systole. In diastole, the myocardium swings, away from the normal veli-like, posterior mitral leaflet", mechanism of closure may well happen sometimes.

In the first place, the digital exploration of a normal human mitral valve in a mistaken preoperative diagnosis of mitral stenosis, revealed a remarkable billowing effect of the ventricular leaflet toward the left atrium. The ventricular leaflet could be forced out into the atrium only by the blast of blood squeezed into the perivalvular space. The same could not be produced by the ventricular wall being pushed against and butterssing the posterolateral leaflet. Moreover, the digital pressure on the ballooned-out ventricular leaflet produces a feeling of resilience like that of squeezing a fluid filed bag which would hardly be felt were the muscular wall responsible for the bulging. In addition, this same maneuver causes an immediate mitral reflux, which further suggests that the perivalvular space is not obliterated in systole.

Second, the chordae tendineae of third order, which constantly connect the ventricular leaflet with the ventricular wall, would be devoid of any importance if we were to admit that

the heart wall buttresses the leaflet.

Commissural lines

The mitral leaflet tissue formed a continuous veil hanging down from the mitral annulus except in 2 specimens. A perpendicular line from the annulus to the free edge was used to measure the height of the leaflet tissue at a given point.

The aortic leaflet had maximum height in its middle. On either side, it is tapered. The transitional lines at which the height of the leaflet tissue started increasing again are labeled as AL and PM commissural lines. These passes through the highest point of the broad indentation or concavity between the 2 leaflets. The height of the commissural lines varied from 2 to 8 mm.

The aortic leaflet is defined as the portion of the mitral curtain included between these 2 commissural lines anteriorly, hanging down from a relatively straight fibrous annulus shared with the aortic valve. Posteriorly the rest of the curtain of leaflet tissue formed the mural leaflet, hanging down from a curved annulus, related to the atrioventricular musculature.

Slits

Apart from tiny cremations between chordal insertions, the free edge of the leaflets may exhibit deep indentations, which we have designated as slits. We prefer the term "Slit" rather than "cleft" which usually connotes a congenital defect in the aortic or rarely mural leaflet.

The aortic leaflet had no slits. Slits are seen in the mural leaflet in 98 hearts. The number of slits varied : 0 in 2 hearts, 1 in 20, 2 in 56, 3 in 15, 4 in 6 and 5 in 1. As a surgeon would view the valve through the atrium, these slits are serially numbered anticlockwise, from the AL commissural line to the PM posteromedial in 76. The varied location of slits is noted.

Slit lines and scallops

The perpendicular drawn from the annulus to the deepest part of a slit is designated as the slit line. Slit line subdivide the mural leaflet into 2 to 6 scallops. It is best to number these serially. The leaflet tissue between the AL commissural line and closet AL slit line, when present, is usually labeled as the AL commissural scallop. We saw this in 81 hearts.

Commissural and slit chordae

The chordae in relation to each commissural line, and the closet slit line, are traced down to their origin and studied, All these chordae start as a single stem from their origin and subdivided prior to insertion into the leaflet tissue either at the free edge, rough zone, or both. Rarely there are 2 parallel chordae on either side of the commissural or slit line. The gross appearances of the free edge chordae, in relation to the commissural or slit, varied Only 47% of AL commissural chordae, 53% of PM commissural chordae. 39% of first AL slit chordae and 29% of last PM slit chordae were fan shaped. The length of these chordae span of attachment at the free edge varied widely. The main stem usually inserts into the submit of the commissure or slit.

When the commissural chordae are parallel with chordal insertion either side of the commissural line, a segment of the free edge is left unsupported over a distance varying from 1

to 5mm in 9 hearts. In 3 hearts the commissural scallops had basal chordae. The commissural chordae had varied origins. The origins of slit chordae are found to be similarly varied.

Muscular Chordae

In 2 hearts the mitral veil of the leaflet tissue was interrupted at the location of the AL commissural line by a muscular chorda which extended up to the annulus. The leaflet tissue is attached to the sides of this muscular chorda close to the annulus.

Line of apposition

In order to observe nodular thickening at the line of apposition, the leaflet tissue adjacent to commissural lines is transilluminated. Normally in both the leaflets, the line of apposition consisted of a row of thickened nodules rather than a linear ridge. These are absent in the scallops behind the commissural lines in 95 hearts and faintly observed in 5 hearts.

Discussion

Several tissues regarding the anatomy of the normal mitral value remain to be clarified.

Commissures

The free edge of the mitral skirt needs to have indentations, slits and pleats to allow it to splay open, providing a large orifice during diastole, and to close neatly during systole. Two constant, shallow, wide indentations divide the mitral value into an aortic leaflet and mural leaflet. The junctions between the 21 leaflets commonly called commissures have been variously defined as :

- Tissue joining the 2 leaflets;
- Junctional zones of valvular tissue;
- Point of attachment of mitral annuals to the fibrous trigones;
- Indentations at either and of the aortic leaflet;
- Angles at which the 2 leaflets meet;
- Clefts separating anterior and posterior leaflets;
- The space between identifiable components of the skirt of leaflet tissue;

Area of leaflets covered by typical commissure chordae have been considered as the commissural area, which would include adjacent leaflet tissue. There has been dispute as to whether the commissural is an anatomical reality or a pathologic entity. Papillary muscles and chordal grooves have also been taken as guides to the commissures. We propose AL and PM commissural lines as precise lines separating the 2 leaflets.

Slits and scallops of mural leaflet

The relatively straight intervalvular segment of the mitral annulus between the 2 fibrous trigones does not alter in length during the systole. Hence the aortic leaflet hanging down from this segment has no need to become folded. An un-slit aortic leaflet offers a smooth outflow from the left ventricle.

In contrast, the C-shaped annulus of the mural leaflet, related to the atrioventricular musculature, changes in contour and size during atrial and ventricular systole. Hence the mural

leaflet needs to have slits, to enable it to vary in size and contour. These slits divide the leaflet into segments which vary in size and number and are unique in each heart. Instead of giving names which may be confusing, it is best to number serially the segments or scallops, commencing from scallop 1, behind the anterolateral commissural line. When there are no slits, the mural leaflet is undivided, and its free border forms an uniform are.

The scallops just behind commissural lines have special pathological and surgical significance. They have been labeled as commissural leaflets, accessory leaflets commissural scallops or projections. The commissural scallops considered as separate leaflets of a quadricuspid valve. Without AL or PM slits, there would be no separate demarcation of a commissural scallop, which implies that such a demarcation is not essential for normal mitral valve function.

The inconstant commissural scallops, varying in size and number, do not merit an exclusive status. They are, when present, the first and last of the serially numbered scallops of the mural cusp. This part of the mural leaflet tissue, whether it contains slits and scallops or not, allows the central part of the mural leaflet to move well away from the aortic leaflet during diastole. During systole, this segment does not appear to bear the brunt of force of closure of the valve because it is usually devoid of nodular thickenings seen at the line of apposition of the rest of the leaflets.

We noted lack of fibrous thickening of the junctional tissue. This indicates that at this region, the leaflet tissue merely gets folded and gently plugs the two ends of the cresentric line of closure of the value by rolling over of the leaflet tissue on either side of commissural and slit

lines, an unique mechanism designed for closure of an asymmetrical value, with a curvilinear closure line.

Commissural and slit chordae

Typical fan-shaped chordae help to locate the commissures and slits. The indentations on either side of the anterior leaflet and slits in the mural leaflet are obvious. It is unnecessary to depend on any chordae to locate them. Moreover, the chordae in relation to commissural and slit lines are neither typical nor always fan-shaped. The configuration in each heart is unique.

Their primary function is to prevent the leaflet tissue on either side of the commissural or slit lines from splaying apart too much during diastole. In fact all the chordae emanating from the papillary muscles fan out like parachute strings giving a long rope for the leaflets to the left atrium during systole. These basic functions are achieved with varying chordal configurations.

Pleating of the leaflets

While intention between the 2 leaflets and slits in the mural leaflet provide a coarse adjustment for closure of the reduced systolic orifice, it is the pleating of the leaflets like pleating of a skirt, that provides precision for the closing mechanism. This is achieved by upward bulging of the leaflet tissue in between the chordal insertions, causing multiple hoods in the value leaflets, and pleating of the leaflet tissue.

In a study of 100 hearts, we found upward bulging or hooding of interchordal leaflet tissue especially in elderly patients and those with left ventricular hypertrophy. If this pleating

does not occur, the line of apposition of the leaflets would be a fairly uniform, straight and thickened ridge of tissue. Instead, invariably we see a row of nodular thickenings. The nodules occur due to impact of apposition of the convex hoods. The pleats could be visuvalized during surgery or in autopsy specimens. Obviously when slits are absent, pleating assumes greater importance.

CONCLUSION

Detailed knowledge of the anatomy of slits and scallops of the mitral valve provides improved understanding of mitral valve function, which aids the surgeon in understanding valve pathophysiology and in designing reconstructive procedures.

TABLE

THE FREQUENCY OF ACCESSORY

MITRAL VALVE

	CASES	**PERCENT**
Mitral valve with anterior and posterior accessory leaflets	20	20.9
Mitral valve with anterior accessory leaflet	30	28.6
Mitral valve with posterior accessory leaflet	10	11.5
Mitral valve without accessory leaflets	40	39.0
TOTAL	**100**	**100.0**

TYPES OF PAPILLARY MUSCLES IN 100 NORMAL HEARTS

Types of papillary muscles	**Cases**	**Percent**	**Cases**	**Percent**
Single	87	82.8	31	29.5
Double	15	14.3	57	54.3
Triple	3	2.9	12	11.4
More than three	-	-	5	4.8

TABLE

TYPES OF INNER SURFACE OF PAPILLARY MUSCLES NORMAL HEARTS

Types of Papillary Muscles	Cases	Percent	Cases	Percent	Cases	Percent	Cases	Percent
Single	84	80.0	3.	2.8	31	29.5	-	-
Double	8	7.7	7	6.8	15	14.3	41	39.0
Triple	1	0.9	2	1.8	4	3.8	14	13.4

TABLE

Patterns of Papillary Muscles

Number of Bellies	Pattern	Anterolateral Group	Postermedial Group
Single	Conical	32	12
	Mammillated	14	12
	Flat topped	9	8
	Grooved	2	6
	Stepped	-	5
	Wavy	-	3
	Arched	-	2
	Sloped	-	1
	Saucerized	-	1
Two	Two tiered	11	9

	Interlinked	4	10
	Parallel	7	6
	Arched	1	5
	V	1	4
	Y	1	1
	H	2	2
Three	Parallel	2	3
	Interlinked	-	5
	Two Interlinked	2	-
	+1 separate arched	-	1
	Two tired +1 separate	-	1
Four	Parallel	1	2
	Interlinked	-	1
Five	Scattered	1	-

TABLE

Extent of Protrusion of Papillary Muscles

	Anterolateral Group	**Posteromedial Group**
Mostly Intraluminal	14%	11%
Mostly intraluminal with tip anchored	19%	28%
Equally sessile and intraluminal	54.5%	41.5%
Mostly sessile	12.5%	19.5%

TABLE

Origin of Papillary Muscles

Site	Anterolateral	Posteromedial
Upper third	19%	6%
Middle Third	79.9%	92.5%
Lower Third	1.5%	1.5%
Mostly sessile	12.5%	19.5%

TABLE

Number of Chordae at Origin

Number of Chordae	Anterolateral Group	Posteromedial Group
1-3	-	1
4-6	21	31
7-9	51	31
10-12	20	22
13-16	6	10
17-20	1	2
21-23	1	-
Range	4-22	2-18

TABLE

Number of Chordae at Origin

Number of Chordae	Anterolateral Group	Posteromedial Group
10-20	4	9
21-30	40	31
31-40	31	45
41-50	17	9
51-60	5	4
61-70	2	1
71-80	1	1
Range	14-72	12-80

MITRAL VALVE - C0MMISSURAL / SLIT LINES AND SCALLOPS OF THE MURAL LEAFLET

DIAGRAMS & PHOTOGRAPHS

DISCUSSION & INTERPRETATION

The mitral valve apparatus, including the papillary muscles, is as unique to each individual as one's own finger prints.

The papillary muscles occupy either side of the mid-mitral line, passing through the middle of the aortic and mural leaflects. The anterolateral papillary muscle group provides chordal supports for the anterolateral halves of the aortic and mural leaflets separated by the anterolateral commissural line. The posteromedial halves of the two leaflets separted by the posteromedial commissural line. The adjacent chordae from either group "shake hands" across the mid-mitral line, two with a flimsy narrow band of leaflet tissue uniting the chordae across the line on either side. It is of interest that a midtricuspid line passing through the middle of the septal and "anterior" component of the mural leaflet of the tricuspid valve also divides the chordopapillary support into two (superior and inferior) groups. The direction of chordae in the groups is different as in the mitral valve. The direction of chordae in the two groups is different as in the mitral valve. The chordae band of leaflet tissue at the free edge, as in the mitral valve. Similarity in divisibility of mitral and tricuspid valves in this fashion is in consonance with the similarity of the basic bicuspid design of these valves.

The anterolateral and posteromedial groups of chordae tendineae radiate from either group like struts the "Commissural chordae" in relation to the commissures and "cleft chordae" in relation to the clefts in the mural leaflet have been described as fan shaped. We have shown that these commissural and slit chordae are not necessarily fan shaped, and vary in configuration from heart to heart. From the surgeon's angle it is advisable to consider the chordal arrangement on either side in to as a fan. The fan forms various configurations, depending upon the number and site of origin of the chordae, their pattern of branching, and the number and mode of insertion into the leaflets. When there is a single muscle belly the chordae arise from its upper edge and / or sides. When there are multiple muscle bellies, chordae from each muscle belly radiate to the corresponding segment of the leaflet in varied patterns. When there is a two tiered arrangement of chordal support, the chordae to the commissural region may arise from muscle bellies of various size and shape, or consist of short direct chordae arising close to the leaflet edge. The variations would influence the pathophysiological effects of various disorders. During surgery, the chordae can be studied by traction on the concerned papillary muscle group, or the middle of the aortic and mural leaflets can be splayed apart, using long angled hooks and the chordal fan assessed on either side.

In essence, the reparative procedure adopted for mitral incompetence should restore the chordal fan on either side. With this aim, it is necessary to judge the type of repair required, and to access the length and number of

chordal substitutes and location of site for re-implantation of ruptured chordae and papillary muscles.

In rheumatic mitral stenosis, thickening, shortening, fusion, and eventually disappearance of the chordal fan impair the mechanism of fanning out. There is fibrosis / fusion of the papillary muscles and leaflets. It is likely that short chordae in relation to the commissural line emanating from the papillary muscles reaching close to the free edge of the leaflet, are more likely to be destroyed earlier by the rheumatic process.

Impairment of fanning out of the chordae results is failure of separation of leaflets during diastole. Consequently, cobwebs of fibrinous strands and later fibrorus tissue unite the two leaflets. Current techniques of closed, open, or balloon mitral valvotomy merely disrupt the fibrous union and split fused chordal pillars and papillary muscles lengthwise, and do not restore the normal fan. Fortunately, such crude procedures widen the mitral orifice, improve the mobility of the leaflets to varying extents, and are functionally adequate; though residual stenosis, mitral incompetence, and restenosis would continue to post problems. Ideally, one should restore to normalcy the papillary muscles, chordal fan, and leaflet.

When there is left ventricular dilatation, the papillary muscles are displaced, with alteration in the line of their long axis and resultant impairment of closure of mitral valve. In mitral valve prolapse syndrome, the left ventricle

assumes various configurations, which is possibly dependent on the architecture and location of papillary muscles, subject to pull by the prolapsing leaflet.

In mitral valve replacement, retention of chordopapillary support is being favoured to preserve optimum function of left ventricle. However, if the native valve has too many chordae and papillary muscle bellies, these may interfere with the function of the disc or ball, especially if they are mostly intraluminal.

Replacement of mitral valve with mitral homograft has been tried and is reemerging as a surgical alternative. Obviously, it is preferable to use homograft mitral valves with single anterolateral and posteromedial papillary muscle rather then multiple papillary muscles, which will be cumbersome to fix. Location of the donor papillary muscles in the recipient heart needs to be tailored, ensuring optimum fanning out of chordae to ensure its systolic and diastolic function. Very long chordae in the homografts may be prone to rupture. Very short chordae may favour early fibrous fusion between the papillary muscle and the leaflet tissue. Of course, the transplanted valve is devascularized and denervated and this would affect its function and durability. The blood supply of the papillary muscle is less critical when tethered, rather than when it is wholly protuberant into the ventricular cavity. It is advisable to retain the patients own papillary muscle whenever possible, to preserve its vascularity, inervation, and continuity with ventricular musculature, which

could improve the function of the homograft.

Homograft mitral valve has been used to replace the mitral valve. The two groups of mitral papillary muscles are located in the non-septal wall, and designed to draw the mobile aortic leaflet toward the relatively static mural leaflet. In contrast, the two groups of papillary muscle of the tricuspid valve are mostly scattered over the septum, drawing the more mobile mural leaflet towards the less mobile septal leaflet. These factors should be considered while expecting the leaflets and papillary muscles of the donor mitral valve to function in the tricuspid location.

Papillary muscles are formed due to delamination of the ventricular musculature. Later, they differentiate into muscle bellies and chordae. Aberrations in this process would lead to persistence of muscle, to a varying extent. When the muscle reaches upto the annulus, it should not be mistaken for pathological fusion between papillary muscles and leaflet tissue, in which condition Fibrotic changes would obvious.

Function of the mitral valve has been observed refinements in echo-cardiography would make this possible in man.

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