Mitral Annulus

Anatomy, size, and shape:

The mitral annulus constitutes the anatomical junction between left ventricle and left atrium, and serves as an insertion site for the leaflet tissue ***(McAlpine., 1975).*** It is often divided segmentally according to the site of leaflet insertion in to the anterior and posterior annulus ***(Hueb et al., 2002).***

The anterior portion of the mitral annulus is attached to the fibrous trigone and is generally more developed than the posterior annulus (Figure 1) ***(Berdajs et al., 2007).*** The right fibrous trigone is a dense junctional area between the mitral, tricuspid, non-coronary cusp of the aortic annuli and the membranous septum. The left fibrous trigone is situated at the junction of both left fibrous borders of the aortic and the mitral valve ***(Goetz et al., 2006).***

Mitral annulus in normal subjects, is roughly elliptical (or kidney-shaped), being less circular in systole than in diastole. In three-dimensional space, the annulus appear as a saddle-shaped, with its high (farthest from apex) point located anteriorly near the aortic root and posteriorly near the posterior left ventricular wall, and its low points located at the anterior and posterior commissure sides ***(Salgo et al., 2002)***. It is surrounded by several important anatomic structures which must be respected, including the aortic valve, the coronary sinus, and the circumflex artery ***(Ahmad et al., 2004).***

Mitral annulus is non conductive, fibrofatty membrane that is divided into anterior and posterior portions (Figure1). The anterior annulus is anatomically coupled to the aortic annulus; the posterior annulus is externally related to the musculature of the LV inflow region and internally to the left atrium ***(Pai et al., 2003).***

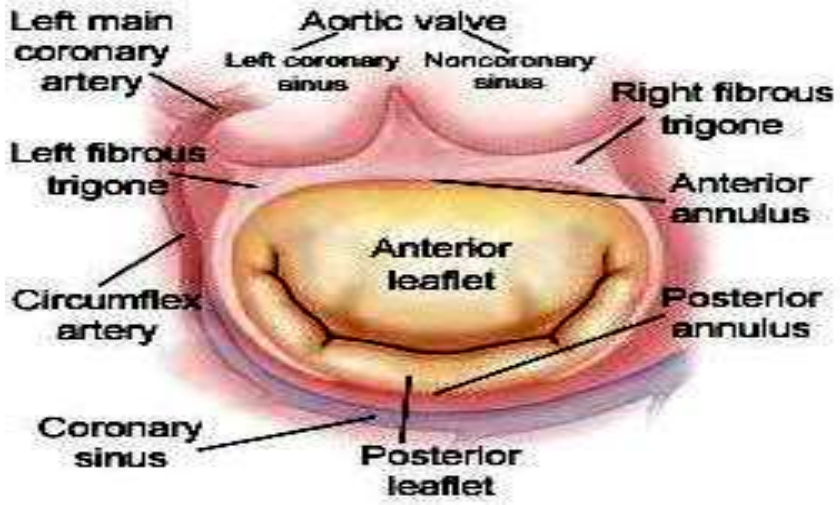

Figure (1): Surgical anatomy of the mitral annulus with important surrounding structures ***(Ahmad et al., 2004).***

The average mitral annular cross-sectional area ranges from 5.0 to 11.4 cm^2 in normal human hearts (average is 7.6 cm^2), the annular perimeter of the posterior leaflet is longer than the anterior leaflet by a ratio of 2:1; i.e., the posterior annulus circumscribes approximately two-thirds of the mitral annulus ***(Flachskampf et al.,2000).***

In normal subjects the annular area increased during diastole to a maximum of 3.8 ± 0.7 cm^2/m^2 in late diastole. There was presystolic followed by systolic narrowing to a minimum in mid systole. The mean reduction in area was 26 ± 3% ***(Ormiston et al., 1981).***

Annular area varies during the cardiac cycle and is influenced directly by left atrial (LA) and LV contraction, size, and pressure. The magnitude of change in mitral annular area is 20 to 40% during the cardiac cycle ***(Parish et al., 2004).*** Annular size increases beginning in late systole and continues through isovolumic relaxation and into diastole; maximal annular area occurs in late diastole around the time of the P-wave on the electrocardiogram. Importantly, half to two-thirds of the total decrease in annular area occurs during atrial contraction ***(Carlhall et al., 2004).***

Annulus Dynamics and Motion:

The mitral annulus moves upward into the left atrium in diastole and toward the LV apex during systole; the duration, average rate, of annular displacement correlate with the rate of LA filling and emptying ***(Van Rijk-Zwikker et al., 1990).*** The annulus moves slightly during late diastole (2 to 4 mm toward the left atrium during atrial systole). This movement does not occur in the presence of atrial fibrillation and may be an atriogenic contractile property.

The annulus moves a greater distance (3 to 16 mm toward the LV apex) during isovolumic contraction and ventricular ejection phases ***(Keren et al., 1988)***, this systolic motion which aids subsequent LA filling, occurs in the presence or absence of atrial fibrillation (AF) and is related to the extent of ventricular emptying; thus it is likely driven by LV contraction subsequently.

The annulus moves very little during isovolumic relaxation but then exhibits rapid recoil back toward the left atrium in early diastole ***(Glasson et al., 1997).***

Motion of the mitral annulus represents changes in left ventricular long-axis dimension as the cardiac apex is relatively fixed during the cardiac cycle ***(Ashman et al., 1988),*** in the absence of gross distortion of the ventricular shape or severe regional wall motion abnormalities, the changes in long-axis dimension could reflect left ventricular volume changes ***(Carlhall et al.,2004).***

During the cardiac cycle, the mitral annulus undergoes complex changes. Two types of motion have been described: sphincter contraction and translation. Studies have demonstrated that sphincteric contraction reduces annular area by approximately 25% ***(Pai et al., 1995).*** The mitral annulus also

undergoes translational motion along the LV major axis as torsion of the base of the LV.

The decrease in the LV long-axis dimension produced during annular translation. Systolic translation of the annulus produces increase in left atrial filling ***(Komoda et al., 1994).***

Left Atrium

Normal size and anatomy of the LA:

In the early history of anatomy, the left atrium was one of the first cardiac structures to be identified, recorded and analyzed; as a relatively oval shaped chamber with thin, muscular wall (Figures 2, 3). The left atrium is easily visualized posterior to aortic root and superior to the left ventricle.

The left atrium serves as a reservoir for blood draining the pulmonary veins during ventricular systole and as a conduit for that blood during early diastole. In late diastole; the LA becomes a muscular pump to complete the process of the left ventricular filling before ventricular contraction and mitral valve closure. Thus changes in LA dimensions and volumes are a mirror of this continuous process of filling and emptying ***(Harvey Feigenbaum Textbook, 2005).***

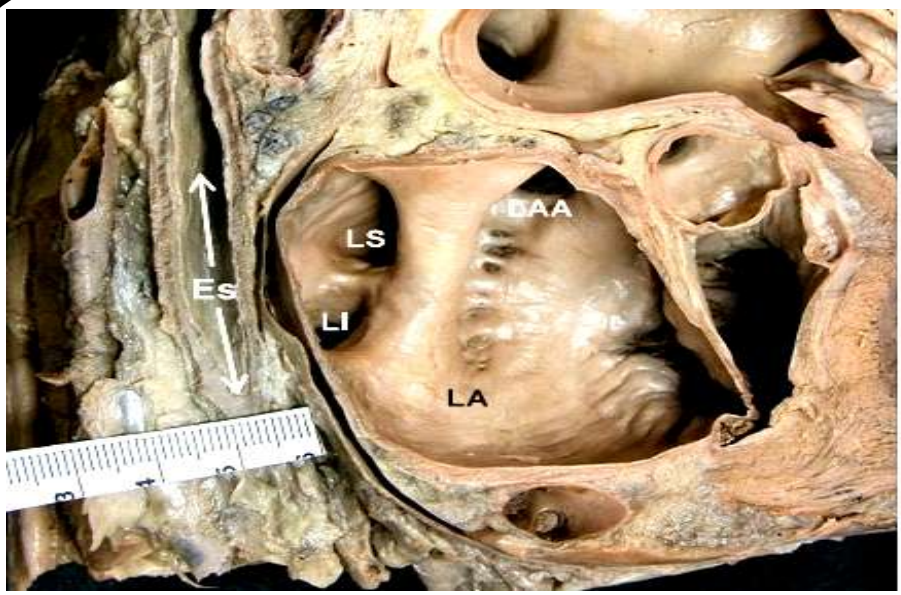

Figure (2): Sagittal section through the left atrium shows the proximity of the esophagus (Es) to the posterior wall of the left atrium (LA) ***(Mansour, etal., 2006)***

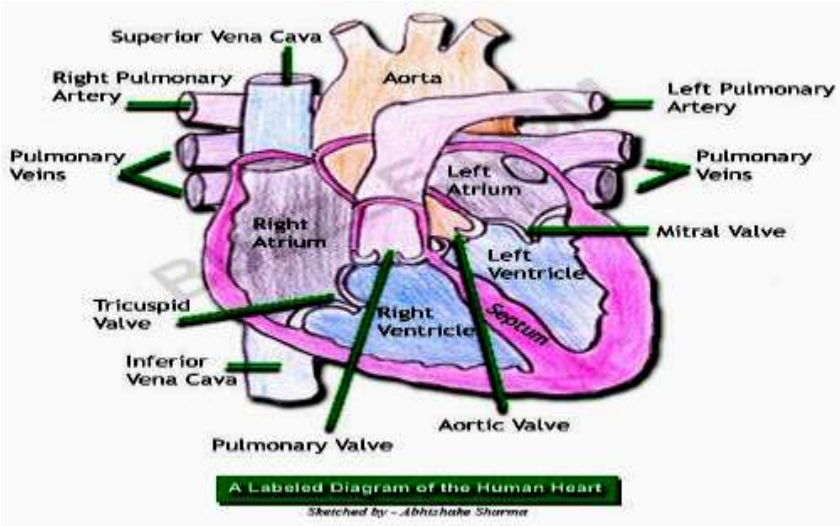

Figure (3): Diagram explains anatomical structure of the heart ***(Scharf et al., 2003)***

Demographic and Anthropometric Determinants OF LA Size:

Age: In healthy individuals, LA volume index is independent of age. Changes in LA volume with age are attributable to the pathophysiologic changes associated with normal aging process as opposed to the result of normal aging itself .This difference can be attributed to the variations in relative weight and body size ***(Pritchett et al., 2003).***

Body Size: Body size is the most important determinant of LA size, which increases with increasing body size. Therefore, LA size should be indexed to a measure of body size ***(Pearlman et al., 1990).***

Body surface area (BSA): is most commonly used for LA volume indexation; however, this might attenuate the adverse influence of obesity on LA size. A recent analysis showed that LA ‘volume indexed to BSA may over or underestimate the prevalence of LA enlargement in patients depending on their obesity status. However, LA volume indexed to height or height with algometric powers was unaffected by the level of obesity and appears to be preferable to indexing by BSA, which will need to be independently assessed in other populations ***(Thomas et al., 2003).***

Left Atrial Mechanical Function

The LA mechanical function can be described by three phases within the cardiac cycle:

First, *during ventricular systole and isovolumic relaxation phases:* the LA functions as a "reservoir" that receives blood from pulmonary venous return and stores energy in the form of pressure ***(Pagel et al., 2003).***

Second, *during the early phase of ventricular diastole*: LA operates as a "conduit" for transfer of blood into the LV after mitral valve opening via a pressure gradient, and through which blood flows passively from the pulmonary veins into the left ventricle during LV diastases ***(Mitchell et al., 1969).***

Third, *during the late phase of ventricular diastole:* the "contractile" function of the LA normally serves to augment the LV stroke volume by approximately 20%. The relative contribution of this "booster pump" function becomes more dominant in the setting of LV dysfunction ***(Hoit et al., 1993).***

The left atrium modulates left ventricular filling through three components: a phase of reservoir or expansion during systole, a conduit phase during diastole ***(Grant et al., 1963).*** And an active contractile component -when sinus rhythm is present- during late diastole, this active contractile component of the left atrium has an important role in patients with ventricular dysfunction as a ‘booster pump’ ***(Spencer et al., 2001).***

LA booster function is one of the mechanisms compensating for decreased early filling in patients with reduced left ventricular compliance**,** whereas loss of atrial contraction as a result of AF or ventricular pacing reduces cardiac output by approximately 15–20% (***Stephanotis et al., 1999)*.**

During exercise LA reservoir and booster functions are augmented. Increased reservoir function may play an important role in accelerating LV filling by helping to maintain atrio-ventricular pressure gradient during diastole ***(Kagawa et al., 1994).***

Evaluation of LA mechanical function by TTE echo:

Occur by measure LA function in three phases of cardiac cycle:

Left atrial volumes were measured echocardiographically by the biplane area length method from the apical four-chamber views. LA maximal volume (Vol_{max}) was recorded at the onset of the mitral opening, LA minimal volume (Vol_{min}) at the onset of mitral closure, and LA presystolic volume (Vol_{p}) at the beginning of the atrial systole (P-wave on ECG) ***(Aydin et al., 2004).***

LA emptying function parameters were calculated according to the LA maximal, minimal and presystolic volumes (LA passive emptying volume = Vol_{max}-Vol_{p}, LA passive emptying fraction = [Vol_{max}-Vol_{p}]/Vol_{max}, LA active emptying volume = Vol_{p}- Vol_{min}, LA active emptying fraction = [Vol_{p}-Vol_{min}] / VP and total emptying volume = Vol_{max}-Vol_{min}). All volume measurements were corrected with respect to the body surface area (BSA) ***(Acar et al., 2009).***

Mitral Annulus Calcification (MAC)

Pathology of (MAC):

Mitral annular calcification (MAC) begins as one or more small hard nodules at the base of the posterior mitral leaflet, commonly the middle of this leaflet. As it increases in size, the small calcific nodules coalesce to form a continuous bar or ridge projecting in to the angle between the posterior mitral leaflet and LV posterior wall ***(Kronzon et al., 1978)***.

The calcification occurs in the annulus region and basal part of the leaflets sparing the free edges of the leaflets; this distribution of calcium is useful in distinguishing it from rheumatic calcific MS ***(Boon et al., 1997).***

Caseous calcification, formed from a mixture of calcium, cholesterol and fatty acids, is an uncommon variant found on echocardiography with MAC. The characteristic echocardiographic appearance is of a large echo-dense structure with echolucent center and smooth borders ***(Marcu et al., 2006).***

Microscopically:

MAC appears as solid, amorphous masses arising in the fibrosa of the valve or in the posterior leaflet near its attachment. With further growth, MAC penetrates adjacent myocardium into the posterior sub-mitral angle ***(Roberts et al.,***

1973). About 3% of the patient with MAC undergoes unexplained central softening so that a sterile abscess containing caseous material formed ***(Tajik et al., 1984).*** It may resemble a tuberculoma or gamma. This unusual MAC variant is of relatively larger size and round shape. It is of important value because it stimulates neoplastic or thrombotic left atrial mass ***(Carpentier et al., 1998).***

Macroscopically:

The gross appearance of MAC may vary from small, localized calcified spicules to massive, rigid bars up to 2 cm in thickness.

Initially, calcification begins at the mid-portion of the posterior annulus; as the process progresses, the leaflets become upwardly deformed, stretching the chordae tendineae, and a rigid curved bar of calcium surrounding the entire posterior annulus or even a complete ring of calcium may encircle the entire mitral orifice ***(Feindel et al.,2003).***

Invasion of the calcific spurs into the LV myocardium and the conduction system can result in atrioventricular and/or intra-ventricular conduction defects ***(Fulkerson et al., 1979).***

Annular calcification causes mitral regurgitation by displacing and immobilizing the mitral leaflets **(**thereby preventing their normal systolic coaptation**)** or impairing the presystolic sphincteric action of the annulus. As the degree of MR worsens over time, LV volume overload can lead to heart

failure. Systemic embolization can occur if the annular calcific debris is extensive and friable ***(Messer et al., 2008).***

Pathogenesis:

The Pathogens of mitral annular calcification is not known, but it appears to be a stress-induced phenomenon. Annular calcification can be associated with systemic hypertension HNT, hypertrophic cardiomyopathy, aortic stenosis, and other predisposing conditions include chronic renal failure and DM. Aortic valve calcification is an associated finding in 50% of patients with severe MAC ***(Fenster et al., 1995).***

Mitral annular calcification is usually benign, asymptomatic, and without physical findings. It may be responsible for an 'innocent' 'heart murmur, conduction abnormalities, mitral regurgitation, mitral stenosis, or subacute bacterial endocarditis ***(Watanakunakorn et al., 1972).***

Pathophysiology of (MAC)

The pathophysiologic basis of MAC represents a chronic degenerative, non inflammatory condition in the fibrous base of the mitral valve apparatus ***(Adler et al., 2001).*** It also involves calcium and lipid deposition.

There is an association between MAC and systemic atherosclerotic disease. Some uncontrolled studies were suggesting that MAC or AVC may be related to AS ***(Movahed et***

al., 2007) (Figure 4). MAC, AVC, and calcific aortic valve stenosis represent a degenerative process that occurs mainly in the elderly. Calcific deposits lead to cardiac conduction disturbances in these patients or to embolism due to dislodgement of calcified material in the blood stream ***(Mathieu et al., 2007).*** MAC was present in 15% of patients with AS compared to only 6% of patients without AS. Similarly, in a large population-based study from the Mayo Clinic, patients with AVC progressed to calcific AS faster ***(Messika-Zeitoun et al., 2007).***

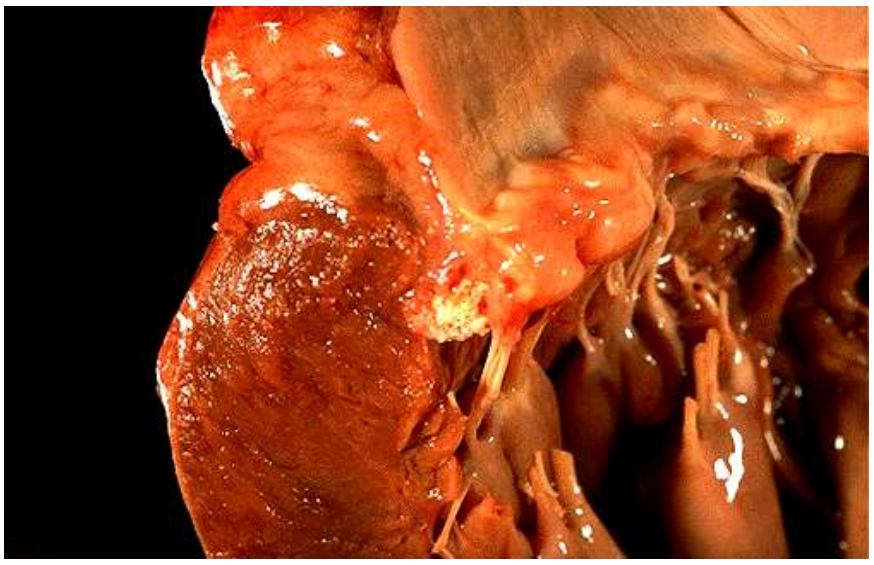

Figure (4): Mitral annulus calcification ***(Harpaz et al., 2001).***

In the Framingham study, a prevalence of 11.9% was found in women and 6.0% in men.

Why mitral annulus calcification prevails in women is unclear, although a role of increased serum parathyroid hormone resulting in hypovitaminosis D has been proposed ***(Shiraki et al., 1988).*** Hemodynamic complications of MAC include MR and MS. Sever extensive MAC is believed to cause MR by deforming and restricting motion of the posterior mitral leaflet thus interfering with systolic valve closure ***(Grande-Allen et al., 2005).***

The mitral annulus area normally is smaller in systole than diastole. Increased rigidity of the annulus as in MAC impairs systolic contraction of the annulus leading to MR ***(D'Cruz et al., 1979).***

Alternatively, the calcification may actually displace the posterior mitral leaflet and prevent its systolic coaptation with the anterior leaflet. Also, mitral annular calcification create pressure overload on the left ventricle, simulating systemic HNT and AS ***(Kronzon et al., 1978).***

The anterior leaflet can become thick and immobile LV inflow obstruction also results from calcification of the posterior mitral valve leaflet. Calcific protrusions into the ventricle and extension of the calcium into the leaflets further narrow the valve orifice, resulting in mitral stenosis ***(Allison et al., 2006)***. In such cases, more pressure is exerted on the left atrium to pump blood in sufficient amounts to the left ventricle ***(Fox C et al., 2004).*** In other words, the left atrium has to work