

Comparative study between

- **Open mitral valvotomy**
- and
- **Balloon mitral valvuloplasty**

Thesis

**Submitted: in partial fulfillment of
M.D Degree in Cardiothoracic surgery**

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Dedication

To my parents who gave and still giving all things without taking any thing.

To my wife not only for here great support and continuos help during this work and all through life but also for here presence in my life.

To my professors and colleagues who gave me direction and advice.

ACKNOWLEDGEMENT

All thanks are due to God.

Many thanks to Prof. Dr. Ahmed El kerdany Professor of Cardiothoracic Surgery, Faculty of medicine, Ain Shams University for his encouragement, advice and great support.

I would like to express my deepest gratitude to my Prof. Dr.

Mohsen Abdel Karem Fudala .Professor of cardio -thoracic surgery, Faculty of medicine, Ain Shams University for his very kind supervision, direction and continuous help in all stages of this work.

I would like to thank Prof. Dr Hesham Abdel Rohman Ass. Professor of cardiothoracic Surgery Faculty of medicine Ain Shams University for his advice and usefull support.

I would like to thank Prof. Dr Azza Abdel Abealla El Fiky Ass. Professor of cardiology Faculty of medicine Ain Shams University for her advice and usefull support.

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INTRODUCTION

Mitral stenosis is either congenital or rheumatic. Congenital mitral valve stenosis is a developmental malformation of one or more of the components of the mitral valve apparatus. (Khalil,et al., 1975).

The prevailing cause of mitral stenosis (MS) is a progressive reaction to an injury caused by one of the major manifestations of rheumatic fever (RF): rheumatic carditis. The mitral valve is the valve most commonly prone to suffer a rheumatic process. Therefore, the prevalence of MS reflects the frequency of RF in the community and, consequently, in the region. (Bruce CJ, et al.,(1998).

In developed countries, with the decline of new RF cases to 0.5 cases/100,000 inhabitants in the early 1980s, 3 rheumatic cardiac diseases began to be seen as "geriatric diseases. However, from 1987 on, the ratio and distribution of invasive streptococcus serotypes have increased, since RF cases reappeared in those countries. In the poorest and densely populated areas of our planet, however, the devastating effects of RF remained constant, since its prevalence in poor countries is at least 10 times higher than in developed countries. Additionally, in lower income population areas, the course of MS differs much from the classic one. Due to a more severe initial rheumatic bout or to repeated bouts of rheumatic carditis secondary to recurrent streptococcal infections, the progression of MS in those areas tends to be much faster, leading to functional disability at a much earlier stage. In underdeveloped countries, MS is a major public health issue. (Bonow RO, et al., 1998).

Mitral stenosis (MS), resulting from thickening and immobility of the mitral valve leaflets causes an obstruction in blood flow from the left atrium to left ventricle. As a result, there is an increase in pressure within the left atrium, pulmonary vasculature, and right side of the heart, while the left ventricle is unaffected in isolated MS. However, MS often coexists with mitral regurgitation and occasionally with aortic valve dysfunction, which may cause left ventricular dysfunction. (Marcus, et al., 1994).

The predominant cause of mitral valve stenosis is rheumatic fever. It occurs approximately in 40% of all rheumatic heart diseases. Two thirds of all mitral stenosis(70%)are females. (Henary, et al., 1977).

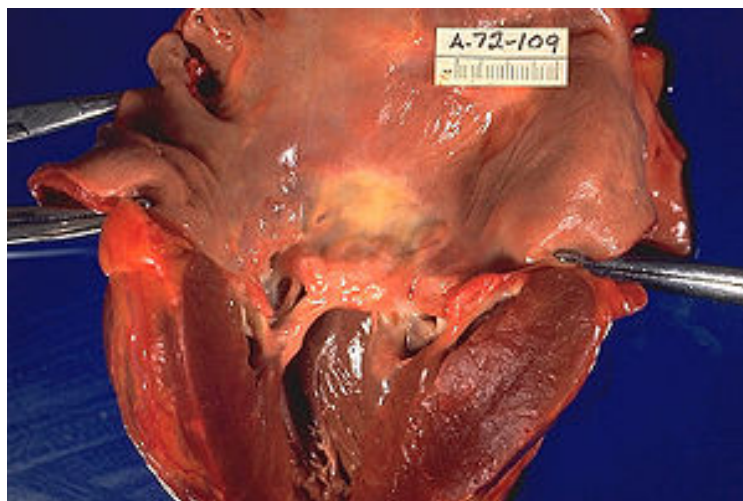


Fig.1 Rheumatic heart disease at autopsy with characteristic findings (thickened mitral valve, thickened chordae tendineae, hypertrophied left ventricular myocardium).

Rheumatic fever results in four types of fusion of mitral valve apparatus leading to stenosis: (i) commissural, (ii) leaflet thickening and calcification, (iii) chordal thickening, shortening, and fusion, (iv) papillary muscle length and fusion. In addition, myocarditis caused by

rheumatic fever affect the left ventricular wall and attached valvular apparatus. It takes approximately 2-10 years or more after acute attack of rheumatic fever for mitral stenosis to develop and approximately a decade, before patient becomes symptomatic. (Carpentier, et al., 1979).

The mean pressure gradient across the mitral valve on Doppler echocardiography (echo) in MS is at least 5 mm Hg; in severe stenosis, it is usually higher than 10 mm Hg. Because the gradient across the mitral valve is flow dependent, the severity of MS is more accurately defined by the mitral valve area (MVA). The normal valve area is 4 to 5 cm². In mild mitral stenosis, the MVA is 1.5 to 2 cm², in moderate stenosis it is 1 to 1.5 cm², and in severe stenosis it is less than 1 cm².

Patients with mitral stenosis may present with exertional dyspnea, fatigue, atrial arrhythmias, embolic events, angina-like chest pain, hemoptysis, or even right-sided heart failure. Previously asymptomatic or stable patients may decompensate acutely during exercise, emotional stress, pregnancy, infection, or with uncontrolled atrial fibrillation.

If untreated, MS often progresses to significant symptoms (eg, dyspnea and fatigue) and serious complications (eg, pulmonary edema, systemic arterial embolism, pulmonary hypertension, and death). Medical therapy can relieve symptoms but does not affect the obstruction to flow. Among patients with severe symptomatic MS, numerous studies have demonstrated a significant reduction in mortality with surgery compared to medical. (Bonow et al., 2005).

Mitral stenosis can be diagnosed clinically on the bases of ; history, physical examination including a loud first heart sound, an opening snap,

diastolic rumble with a pre-systolic crescendo when sinus rhythm is present, chest radiology shows left atrial enlargement, the left ventricle is normal in size but the right ventricle and pulmonary artery are somewhat enlarged, ECG is not diagnostic but shows P-wave abnormalities characteristic of left atrial enlargement (p-mitral) or atrial fibrillation and right ventricular hypertrophy. (Spencer, 1990).

Two-dimensional (2D) and Doppler echocardiography is indicated for all patients with suspected MS to confirm the diagnosis and determine its severity.

Echocardiography also allows assessment of pulmonary artery pressure, detection of other valve disease, visualization of left atrial thrombus, and identification of important differential diagnoses, such as left atrial myxoma. Trans-esophageal echo is superior to trans-thoracic echo at identifying left atrial thrombus in patients who are being considered for percutaneous mitral balloon valvotomy or cardio-version. Stress echocardiography may be helpful if there is a discrepancy between a patient's severity of symptoms and the baseline hemodynamic data. An exercise mean trans-mitral gradient of more than 15 mm Hg and peak right ventricular systolic pressure of more than 60 mm Hg indicate hemodynamically significant MS.

Cardiac catheterization is not necessary in all cases but, like stress echocardiography, may be helpful in characterizing the severity of mitral stenosis when there is a discrepancy between symptoms and findings on echocardiography.

According to the severity of the symptoms (NYHA classification), degree of mitral stenosis, mitral valve area, gradient across the mitral valve, mitral valve score, pulmonary artery pressure; surgical correction of mitral valve stenosis involves three general classes of techniques; repair (open mitral commissurotomy), replacement, and trans-catheter intervention. (Patel et al., 1991)

Aim of the work

This study aimed to compare and evaluate the results and the effects of the two techniques through analysis of two groups of patients who underwent balloon mitral valvuloplasty, and open mitral commissurotomy.

SURGICAL ANATOMY OF THE MITRAL VALVE

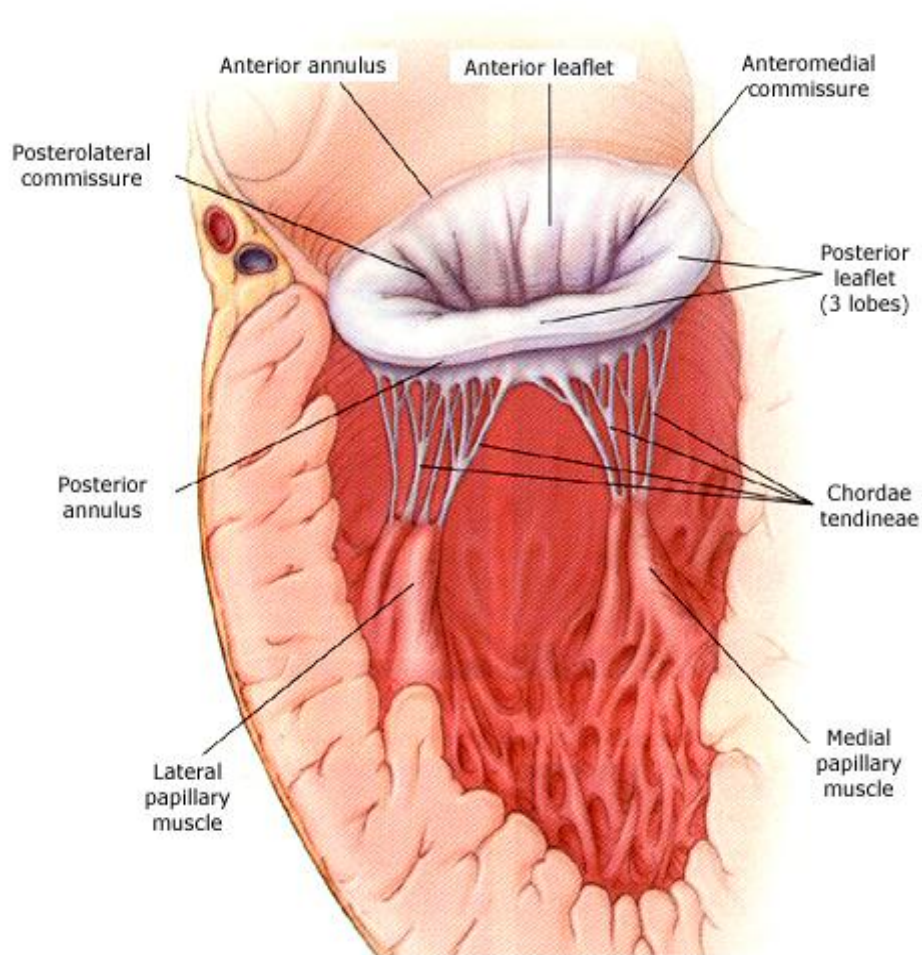


Fig. 2.

The mitral valve consists of the mitral annulus, anterior and posterior leaflets, chordae tendineae, and the papillary muscles. (Otto et al, 2001).

GENERAL ANATOMY

The valvar complex comprises the annulus, the leaflets, the tendinous cords, and the papillary muscles. Also important for its functioning is the left atrial musculature inserting to the leaflets and the myocardium to which the papillary muscles are inserted. The valve is obliquely located in the heart and has a close relation to the aortic valve

(fig 3A). Unlike the tricuspid valve which is separated by muscle from its counterpart, the pulmonary valve, the mitral valve is immediately adjacent to the aortic valve. (fig 3 B)

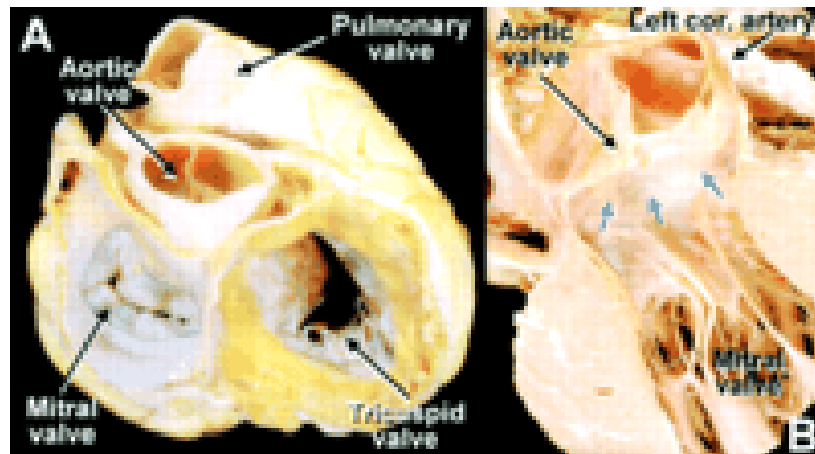


Fig. 3(A) View of the base of the heart in anatomical orientation shows the spatial relations of the four cardiac valves. The left heart valves are close together whereas the right heart valves are separated by myocardium. Dotted line marks the limit of atrial myocardium around the mitral orifice. **Fig. 3 (B)** This dissection of the heart viewed from the anterior aspect shows the close relation between aortic and mitral valves in situ. Fibrous continuity between the valves (blue arrows) is related to the non- and left coronary sinuses of the aorta. (Perloff JK, et al., 1972)

The left atrial wall:

Although generally not appreciated as part of the mitral apparatus, the important role of the atrial wall since left atrial enlargement can contribute to mitral regurgitation. The continuity of the atrial myocardium over the atrial surface of the mural ("posterior") leaflet makes this leaflet vulnerable to being displaced when the atrial chamber enlarges, but this hypothesis remains to be proven. Normally, extensions

of atrial muscle mark the so-called ring when the valve is viewed from the atrium (Fig. 3A) The degree of muscular extension varies from heart to heart and from area to area within the same heart. When the parietal atrio-ventricular junction is cut in profile, the variability of insertion of the hinge, or fulcrum, of the mural leaflet relative to atrial myocardium can be seen (fig. 4.A). At the aortic ("anterior") leaflet of the valve, the distal edge of atrial myocardium marks the hinge (fig. 4.B). (Perloff, et al., 1972).

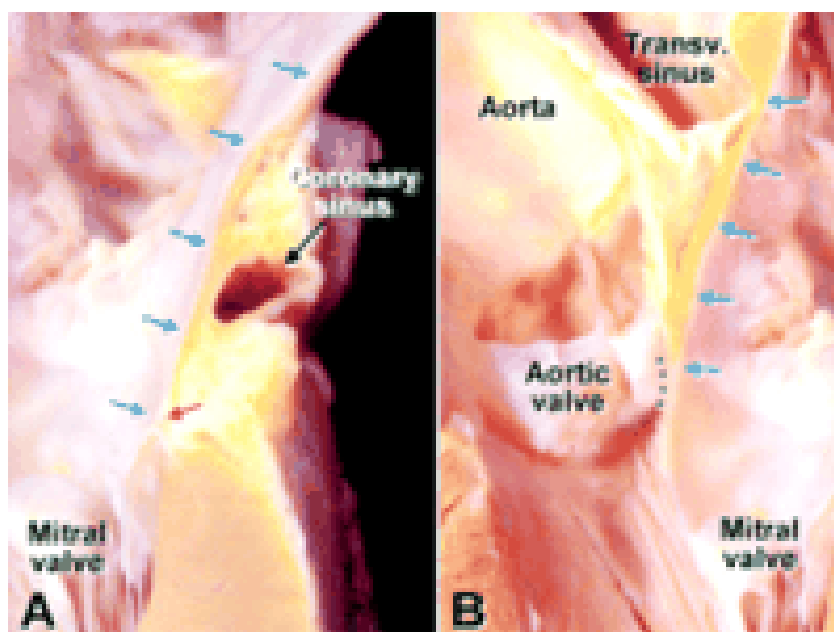


Fig. 4. (A) The left parietal atrioventricular junction in profile shows the left atrial wall (blue arrows) running to the hinge (red arrow) of the mural leaflet of the mitral valve. (B) The aortic leaflet of the mitral valve cut in profile shows the atrial wall (blue arrows) reaching the leaflet. Grey dots mark the region of fibrous continuity between aortic and mitral valves.

Mitral annulus :

The annulus marking the hinge line of the valvar leaflets is more D-shaped than the circular shape portrayed by prosthetic valves. The straight border accommodates the aortic valve allowing the latter to be wedged

between the ventricular septum and the mitral valve. In this region, the aortic valve is in fibrous continuity with one of the two leaflets of the mitral valve. Expansions of fibrous tissues at either extreme of the area of continuity form the right and left fibrous trigones. The atrio-ventricular conduction bundle passes through the right fibrous trigone. (Angelini A, et al., 1988).

Although the term annulus implies a solid ring-like fibrous cord to which the leaflets are attached, this is far from the case. In the area of aortic-mitral fibrous continuity, the distal margin of atrial myocardium over the leaflet defines the hinge-line. When viewed from the ventricular aspect, however, the hinge-line is indistinct since the fibrous continuity is a disfavoured the term annulus. It is preferring to describe the sheet-like fibrous area as the aorto-ventricular membrane that extended around the subvalvar region. Annulus opposite the area of valvar fibrous continuity tends to be "weaker" in terms of lacking a well formed fibrous cord. This is the area affected in "annular dilation" and also most often involved in calcification of the annulus. With severe dilation, the minor axis of the valvar orifice becomes so distended that the leaflets, which are of fixed lengths, become unable to approximate each other. (Angelini A, et al., 1988).

Leaflets

Distinctly different from the tricuspid valve, the mitral valve has two leaflets although some may argue that it has four leaflets. These are notably different in shape and circumferential length. Owing to the oblique location of the valve, strictly speaking, its two leaflets do not occupy anterior/posterior positions nor is one of the leaflets "septal". The septal leaflet is characteristic of the tricuspid valve whereas neither of the

mitral leaflets is attached to the septum. The corresponding terms for anterior and posterior are "aortic" and "mural". It is the aortic leaflet that is in fibrous continuity with the aortic valve. (Yacoub M, et al., 1976).

The aortic leaflet has a rounded free edge and occupies a third of the annular circumference, whereas the other leaflet is long and narrow, lining the remainder of the circumference. The aortic leaflet hangs like a curtain between the left ventricular inflow and outflow tracts. When the valve is closed, this leaflet appears to form the greater part of the atrial floor but is approximately equal in area to the mural leaflet. It meets the mural leaflet to form an arc shaped closure line, or zone of apposition, that is obliquely situated relative to the orthogonal planes of the body. With the leaflets meeting, the view of the valve from the atrium resembles a smile. Each end of the closure line is referred to as a commissure. These are designated the antero-lateral and postero-medial commissures. It is worth noting, however, that the indentations between leaflets do not reach the annulus but end about 5 mm short in the adult heart. Therefore, there are no clear cut divisions between the two leaflets. Furthermore, the free edge of the mural leaflet is often divided into three or more scallops or segments described as lateral, middle, and medial or assigned terms like P1, P2, and P3. Although three scallops are most common, the scallops are not equal in size. (Roberts WC, et al., 1972).

Normally, the valvar leaflets are thin, pliable, translucent, and soft. Each leaflet has an atrial and a ventricular surface when viewed in profile, two zones can be distinguished in the aortic leaflets and three zones in the mural leaflet according to the insertions of the tendinous cords. In both leaflets, there is a clear zone that is devoid of cordal attachments. Nearer the free edge, the atrial surface is irregular with nodular thickenings. This