سامية محمد مصطفى

شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

سامية محمد مصطفى

شبكة المعلومات الجامعية

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التوثيق الالكتروني والميكروفيلم

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سامية محمد مصطفى

جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

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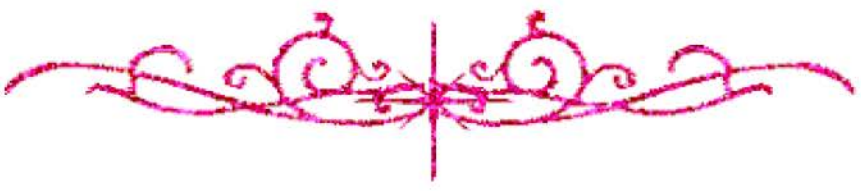

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بالرسالة صفحات لم ترد بالأصل

LEFT ATRIAL DYSFUNCTION IN MITRAL STENOSIS: THE EFFECTS OF BALLOON MITRAL VALVULOPLASTY.

Thesis

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INTRODUCTION

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CONTENTS

INTRODUCTION

THE MITRAL VALVE

The mitral valve is attached to the fibrous ring, which encircles the left atrioventricular orifice. It consists of two triangular cusps, formed by duplication of the endocardium, strengthened by fibrous tissue and containing a few muscular fibers.

The cusps are of unequal size, and are larger, thicker and stronger than those of the tricuspid valve. The larger cusp is placed in front and to the right between the atrioventricular and aortic orifices, and is known as the *anterior cusp.* Nearly all the chordae tendineae, which reach it, are attached near the margin of this cusp, which is, smooth on both its surfaces. The smaller or *posterior cusp* is placed behind and to the left of the opening. Close to their bases the cusps are always joined at their edges for a distance of 0.5 to 1 cm; this union is often termed a commisure. Rarely two small accessory cusps are present in the angles between the larger ones.

The cusps of the mitral valve are furnished with a number of delicate tendinous chords which are termed the *chordae tendineae*, which are attached to the apices of two muscular structures which project from the inner surface of the ventricle they are termed *papillary muscles.*

As the blood flows from the atrium to the ventricle the cusps of the mitral valve float upwards towards the orifice, and as the ventricular muscle contracts they are prevented from passing into the atrium by the pull of the papillary muscles which enable them to resist the force exerted by the out flowing stream. For this purpose the papillary muscles should contract at the outset of ventricular systole and in this connection it should be noted that the earliest branches given off from the left limb of the atrioventricular bundle are distributed to the papillary muscles.

The anterior cusp measures 15-18 mm in length while the posterior cusp measures 10-12 mm so that when the valve is closed there must be an appreciable degree of overlapping. This helps to ensure its competence because it covers the points where the tension is believed to be greatest [1]

MITRAL STENOSIS

DEFINITION:

Mitral stenosis is defined as obstruction to blood flow between the left atrium and ventricle caused by abnormal mitral valve.

PATHOLOGY:

Rheumatic mitral stenosis is more common in females. It results from repeated episodes of rheumatic endocarditis alternating with healing and fibrous tissue deposition.

Mitral stenosis can be subdivided into four distinct types:

1) *Comissural type:* The severest changes occur in the commisures that fuse and narrow the valve orifice.
2) *Cuspal type:* Affects the anterior leaflet more than the posterior leaflet.
3) *Chordal type:* Here the chordae tendinae are thickened, shortened and fused contributing to the stenosis.
4) *Combined type*: There is combination of two or three of the previous types [2].

PATHOPHYSIOLOGY:

The narrowed mitral orifice limits blood flow into the left ventricle with three pathophysiologic consequences:

1- Elevated left atrial pressure with consequent pulmonary congestion and pulmonary venous hypertension.

2- Right ventricular pressure overload secondary to increased pulmonary artery pressure.

3- Reduced LV ejection performance in some cases.

1) Pulmonary Venous Hypertension

As the orifice size decreases, the gradient across the mitral valve increases, thereby increasing both left atrial pressure and pulmonary venous pressure. As the pulmonary venous pressure approaches 25 mmHg, transudation of fluid into the alveolar spaces occurs which may lead to pulmonary edema.

Protective mechanisms against the occurrence of pulmonary edema include: Lymphatic hyperfunction to move alveolar fluid, high pulmonary vascular resistance, the development of a physical barrier at the capillary-alveolar interface from thickening of the capillary walls, increased interstitial tissue formation, and alveolar membrane; and some decompression of the pulmonary veins into the bronchial veins.

However, increased left atrial pressure results in pulmonary congestion which is responsible for the symptoms of exertional dyspnea, orthopnea and paroxysmal nocturnal dyspnea and lastly pulmonary edema [3].

2) Right Ventricular Pressure Overload

The right ventricle's main physiologic function is to generate the pressure needed (about 10 mmHg between pulmonary artery and vein) to drive blood across the pulmonary bed and to provide the force needed to fill the left ventricle.

Mitral stenosis obstructs normal left ventricular filling, requiring the right ventricle to perform increased pressure work to overcome the obstruction.

As mitral stenosis worsens, there is a secondary increase in pulmonary vascular resistance, adding a burden on the right ventricle, increasing the right ventricular pressure overload. To overcome this overload mismatch, the right ventricle makes use of its preload volume [4].

As end diastolic volume increases, end diastolic pressure also increases, leading to systemic venous congestion. Despite the pressure overload, right ventricular contractile function in mitral stenosis appears to be normal.