MEASUREMENT OF BRONCHIAL HYPERREACTIVITY IN PATIENTS WITH RHEUMATIC MITRAL VALVE DISEASE

THESIS

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Master Degree of Medicine

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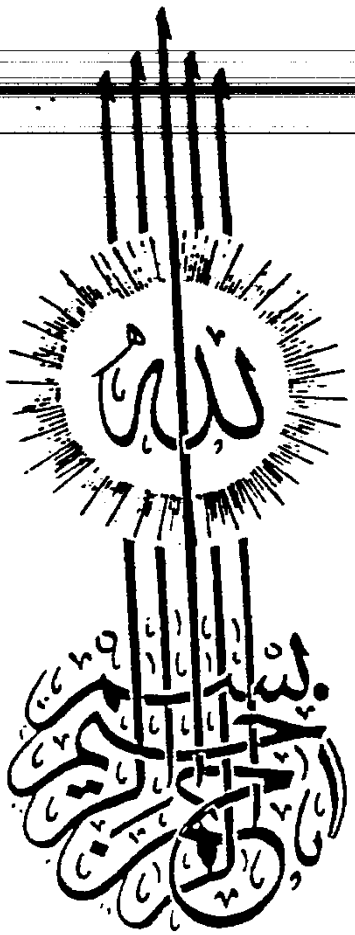

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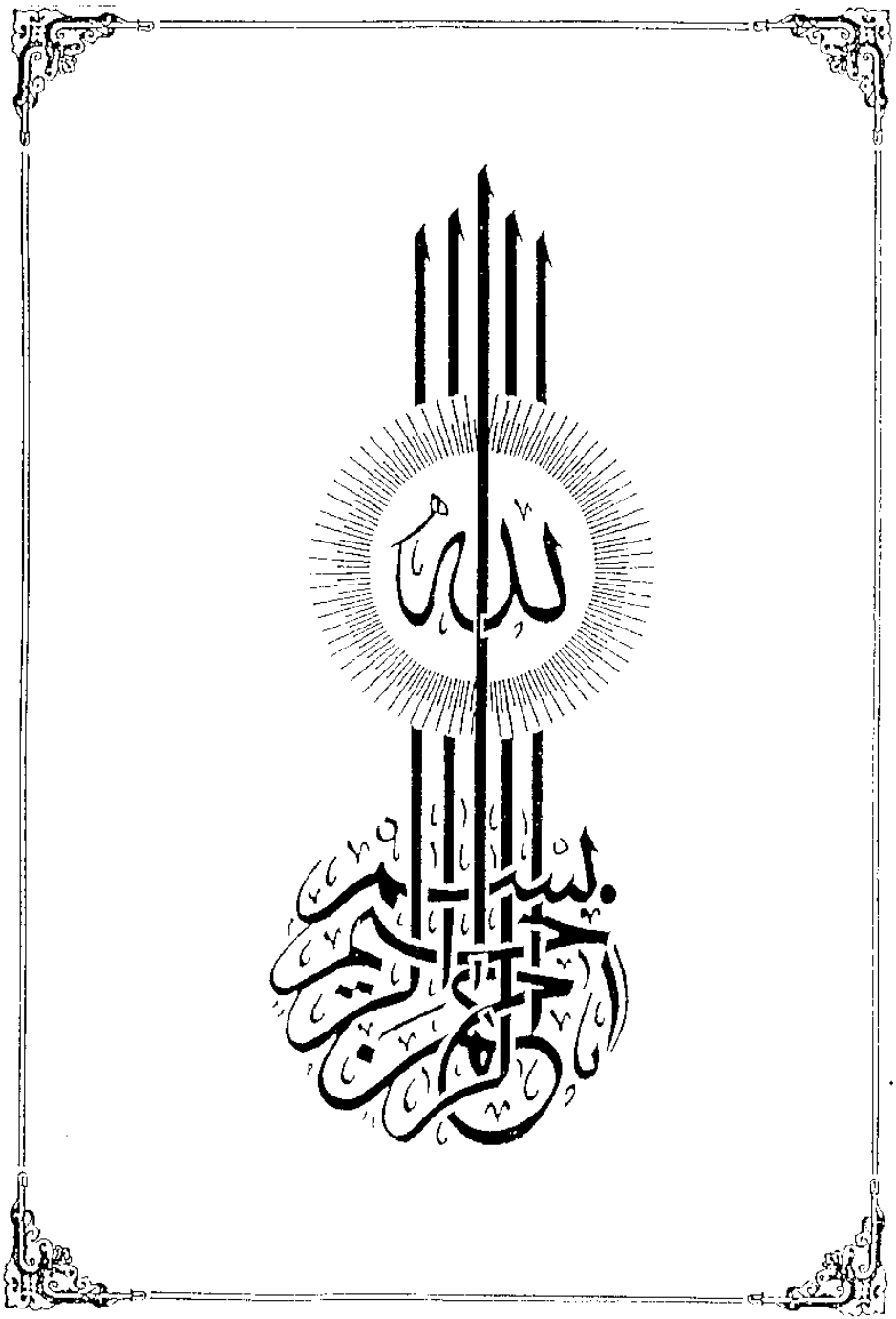

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C O N T E N T S

INTRODUCTION

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Bronchial hyperreactivity in response to infection, allergy and non-immunological stimuli such as histamine, methacholine, cold air and exercise has been well documented.

Patients with rheumatic mitral valve disease, especially with mitral stenosis, sometimes shows signs of airways obstruction, as well as signs of infection, in addition to signs of pulmonary venous congestion. Clinical situations sometimes arise in which we have to differentiate between those patients having asthma like symptoms due to congestive bronchitis secondary to mitral valve disease from those patients having bronchial asthma as a separate disease coincident with their rheumatic heart disease. To do this a prerequisite is to test allergic factors versus congestive factors, to know the effect of bronchial congestion on bronchial reactivity or allergy.

The aim of the present study is to evaluate bronchial congestion and pulmonary venous congestion in patients with rheumatic mitral valve disease especially mitral stenosis as a new mechanism or a predisposing

cause for increased bronchial reactivity and allergy.

Although, the subject of bronchial hyperreactivity in response to various stimuli has been well documented, we have been unable to find any medical records by preceeding investigators on the effect of bronchial congestion on bronchial reactivity. Hence, this study is quite unique in its subject.

Glossary of frequently used terms

- A.V. gradient. Atrio-ventricular gradient.
- M.S. Mitral stenosis.
- IgE. Immunoglobulin E
- ED_{50}. Is the dose which cause a 50 % change in SR_{aw}
- SR_{aw}. Specific airways resistance
- FEV_1. Forced expiratory volume in one second
- PC_{20}. Provocation concentration producing a 20 % reduction in FEV_1 or PEFR
- PEFR. Peak expiratory flow rate in litres/minute.
- Hist. PC_{20}. the concentration of histamine producing a 20 % reduction in FEV_1 or PEFR.

REVIEW OF LITERATURE

RHEUMATIC MITRAL VALVE DISEASE

Mitral Stenosis

"In examining the extraordinary dilatation of the body of the pulmonary veins, and its common openings, I perceived that the mouth of the left ventricle appeared very small and that it had an oval oblong shape". (Vieussens, 1715).

Etiology

Rheumatic fever accounts for about 99 % of cases of mitral stenosis and 50 % of patients give a history of rheumatic fever with male to female ratio 1:4. Conditions which impair flow through the mitral valve are rheumatic valvulitis, thrombus formation, atrial myxoma, bacterial vegetation , and calcium accumulation. (Perloff J.K. and Roberts, W.C., 1972).

Pathology

Mitral stenosis usually is the result of recurrent rheumatic endocarditis. The valvular leaflets and the chordae tendineae are affected by scarring

with concomittant contracture. An additional feature is that at each of the two junctional areas (the commissures) between the two major leaflets there is interadhesion between the two leaflets. This process, along with concomittant shortening of the chordae, causes the two interadherent leaflets to be held downwards. The entire process is manifested by the leaflets forming a funnel shaped structure. The inlet of the funnel is at the level of the left atrial floor and is wider than the apex, which presents in the left ventricular cavity. In the normal heart, blood flows freely through the mitral valve. It may flow through the principal orifice, or through multiple secondary orifices, which are the spaces between the chordae. (Bonnabeau R.V. et al, 1965).

Secondary effects of mitral stenosis include calcification of leaflet tissue, left atrial enlargement and signs of pulmonary venous hypertension, including right ventricular hypertrophy. (Wooley C.F. et al, 1974).

Episodes of acute pulmonary edema and right ventricular failure are common lethal complications.

Mural thrombosis of the left atrium and systemic arterial embolism are very common among patients with isolated mitral stenosis. (Jordan R.A. et al, 1951).

Abnormal Physiology

The physiological abnormality produced by obstruction to mitral valve flow is a pressure gradient between the left atrium and left ventricle during diastolic filling. The pressure gradient is related to the orifice size and diastolic flow through the mitral valve. Mitral valve flow is determined by the cardiac output and duration of diastole. The diastolic pressure gradient across the mitral valve abnormally increases left atrial pressure and volume which are further reflected to the pulmonary veins, capillaries and eventually the pulmonary arteries. Elevation of left atrial pressure distends the chamber and pulmonary viens. If the pulmonary capillary pressure exceeds the osmotic pressure of the plasma proteins, pulmonary edema develops. Chronic elevation of left atrial pressure will lead to hyperplasia and hypertrophy of pulmonary arterioles. Ultimately, pulmonary vasoconstriction and hypertension develop. Eventually, right ventricular hypertrophy and dilatation result from significant

pulmonary hypertension. In the early phase of mitral stenosis the pulmonary blood volume may be increased, but as non-compliant changes develop in the vessels there may be a reduction in pulmonary blood volume with a redistribution of the flow patterns from the base to the apex of the lungs. Finally, the critical narrowing of the mitral orifice will reduce the cardiac output. In this manner, chronic mitral stonosis imposes a pressure overload on the left atrium, the pulmonary vascular tree, and the right ventricle. (Kennedy J.W. et al, 1970).

Left ventricular function may gradually deteriorate as a result of diminished diastolic filli ng from the left atrium. (Curry G.C. et al, 1972 and Silverstein D.M. et al, 1980).

The Hemodynamic Effects of Mitral Stenosis can be Summarized as follows :

1- The left ventricle: Evidence of left ventricular dysfunction can be detected in 20 % of patients with isolated M.S. This is shown by diminished ejection fraction of the left ventricle.