DETERMINANTS OF ATRIAL FIBRILLATION IN PATIENTS WITH CHRONIC RHEUMATIC MITRAL VALVE DISEASE

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THESIS

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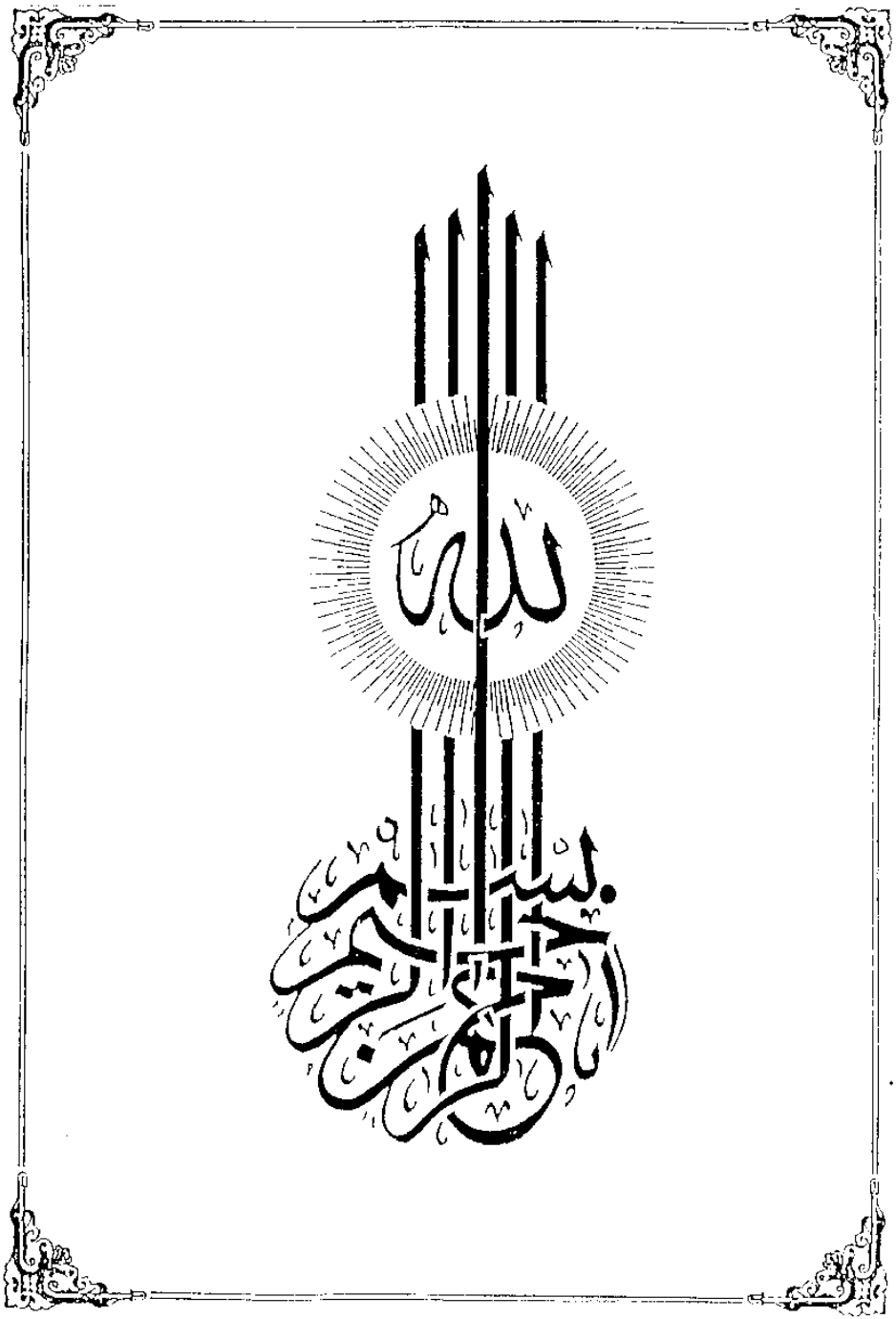

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INTRODUCTION

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Rheumatic heart disease is still a common cause of morbidity and mortality in our country. It still casts its gloomy shadow on the future of those patients affected by this disabling illness.

The mitral valve is the commonest valve to be affected by the rheumatic process. The resultant mitral stenosis or incompetence or both, cause haemodynamic disturbance which is reflected on the clinical condition of the patient.

Atrial fibrillation is a common and serious complication of rheumatic mitral valve disease . Not only does it cause marked haemodynamic deterioration, but it also increases the risk of embolization whether arterial or venous.

Since atrial fibrillation occurs at first in paroxysms then it becomes permanent, several trials were done in order to delineate the possible factors which may be responsible for the genesis of atrial fibrillation, and thereby, it may be possible to anticipate this serious arrhythmia.

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Moreover, it is mandatory to define the patient at risk of developing atrial fibrillation by detecting some variable factors which may pave the way for atrial fibrillation.

It was known that atrial fibrillation was common in cases of mitral stenosis over the age of 40 years (Wood, 1954). Spann in 1973 postulated that 20-30 years at least should elapse before atrial fibrillation supervenes.

According to Henry 1976, the left atrial size measured by M-mode is an important factor in the genesis of atrial fibrillation whereas pulmonary wedge pressure and severity of mitral stenosis were unimportant determinant factors.

Fucus 1982 showed that severe mitral incompetence was associated with atrial fibrillation.

In our study, we aim at delineating the possible factors incriminated in the genesis of atrial fibrillation so that we may be able to identify the patient at risk of developing atrial fibrillation .

These factors include:

1. Age of the patient.
2. Duration of the rheumatic process represented by the time interval between the initial rheumatic attack

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REVIEW OF LITERATURE

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NATURAL HISTORY OF RHEUMATIC MITRAL VALVE DISEASE

The natural history of rheumatic mitral valve disease is no longer easily observed, since it would be improper to withhold surgical help in certain advanced stages. Consequently natural history often has to be extrapolated from older studies. The study of the natural history is beneficial in that it throws light on the impact of the disease on the patient's condition and possible complications which may supervene, thus the proper management may be done at the proper time with less morbidity and mortality.

The mitral valve leaflets are affected during the initial attack of rheumatic carditis, however these changes are incapable of producing actual narrowing of the valve orifice, instead mitral regurgitation occurs and it may progress with permanent damage.

NATURAL HISTORY OF MITRAL STENOSIS

It is generally agreed that a time interval of several years has to elapse between the initial attack of carditis and the time when definite clinical evidence of mitral stenosis becomes apparent. This time interval variesfrom 2-8 years (Coombs,1924). Yet in the majority of cases it

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seems that it is longer than a decade. The majority of patients with fully developed mitral stenosis remain asymptomatic for a varying length of time. Thus, two "latent" phases are described, one is the stage of formation of mitral stenosis, and the other is the asymptomatic stage of fully developed mitral stenosis. Wood's series (1954) showed that the overall latent period was about 19 years; the mean age of the attack of carditis was 12 years and the age at which symptoms appeared was 31 years. Wood also estimated that from the onset of symptoms to the stage of total disability, on average of 7 years elapsed.

Rowe et al.(1960) followed a group of 250 patients for 20 years or until death. About half ofthe patients were under 30 years of age. 52% were asymptomatic. At the end of the study, 79% were dead and 13% were unchanged. Of those who were asymptomatic, 59% remained unchanged after 10 years, 24% did so after 20 years.

This shows that asymptomatic patients may remain asymptomatic indefinitely while mildlysymptomatic patients may remain unchanged for as long as 20 years.

Most patients with mitral stenosis tend to develop symptoms in the 4th or 5th decade of life, in about half symptoms develop gradually, and in the other half, abruptly

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being commonly precipitated by atrial fibrillation or other complications (Selzer, 1972).

The rapid progression of mitral stenosis and its tendency to cause serious disability in early life, is known to occur in under developed countries.(Al Bahrani,1966).

The most important complications that occur during the course of mitral stenosis are atrial fibrillation and embolization both will be discussed later. Other complications include respiratory infections pulmonary apoplexy and very rarely infective endocardites.

Progressive mitral stenosis in absence of clinically apparent attacks of carditis is known to occur. This may be due to chronic rheumatic activity or repetitive and rheumatic insults , both are usually subclinical . An alternative explanation is that the intial scarring of rheumatic valvulitis produces changes upon the valve that are capable of perpetuating themselves in a non specific manner. Edwards (1966), postulated that a valve can be traumatized by abnormal flow patterns and that such trauma may cause thickening fibrosis or calcification of the valve cusps. This may be the cause of developing calcific aortic stenosis on a bicuspid value.

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Similarly,there is possibility of platelet break down at the scarred areas of the mitral valve and this initiates more fibrosis and scarring .

The result of mitral stenosis is progressive rise in the left atrial pressure and left atrial dilatation. Pulmonary venous congestion is a natural result and reactive vasoconstriction occurs in long standing cases, pulmonary hypertension is the ultimate result. It is regarded as a protective mechanism for pulmonary oedema and it is usually reversible after surgical relief of mitral stenosis, except in late conditions where octual obliterative changes occur in the pulmonary arterioles.

Right ventricular hypertroply followed by dilatation and failure occur in response to pulmonary hypertension so that functional tricuspid regurgitation may occur due to dilated tricuspid valve ring.

NATURAL HISTORY OF MITRAL INCOMPETENCE

It seems that mitral regurgitation is well tolerated by the patients more than mitral stenosis although the pathological changes in the mitral apparatus are complete shortly after the initial attack of carditis. Mitral incompetence causes a chronic preload on the left ventricle

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which responds by both hypertrophy and dilatation thus maintains a relatively fair cardiac output with little rise in the wall tension.

It was found that approximately 80% of patients survived 5 years after the diagnosis of mitral incompetence has been established and almost 60 percent survived 10 years (Rapoport ,1975). Munoz et al., 1975 in studying a group of patients with greater disability found that cases of severe mitral regurgitation had a 5 year survival rate of only 45 percent . The arteriovenous oxygen difference and ventricular end diastolic volume were significantly inversly proportional to the expected survrival rate. Patients with combined mitral stenosis and regurgitation had poorer prognosis, with only 67 percent surviving 5 years and 30 percent surviving 10 years after the diagnosis (Ropoport, 1975).

In late cases of pure mitral regurgitation, left ventricular failure occurs and end diastolic pressure and hence left atrial pressure rises more than before . It is then when pulmonary venous congestion is marked and pulmonary hypertension follows it finaly.

The left atrial size increases more in cases of mitral incompetence than in mitral stenosis yet left atrial

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pressure is only mildlyelevated in long standing cases with huge left atrium (Braunwald, 1984). Atrial fibrillation is more common in mitral incompetence than in mitral stenosis and this holds true for infective endocardites yet embolization is still common with mitral stenosis (Abernathy,1973).

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LEFT ATRIAL FUNCTION IN MITRAL VALVE DISEASE

The left atrium is a muscular contractile chamber located in the inflow path of the ventricle which provides transportation of blood from pulmonary veins to the ventricle in various ways. Not only does the left atrium serve as a conduit for left ventricular filling , but it also serves as volume reservoir receiving blood from the pulmonary veins during left ventricular systole. When the elastic chamber dilates during filling, energy is absorbed in stretching the wall with elevation of the atrial pressure, acting in this way, the chamber serves as a passive energy reservoir. The potential energy stored during ventricular systole becomes kinetic energy which is released byatrial emptying. With the onset of atrial systole, the atrium performs the function of active energy transport (Sassayama et al., 1984). The booster pump action of atrial systole allows more volume and pressure delivered than would be discharged by passive energy reservoir alone . however, it may be difficult to separate clearly the contribution of the individual function, because all of these functions operate with varying degrees of influence upon ventricular filling depending upon the loading condition of the atrium.

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Importance of atrial transport has been demonstrated repeatedly by the intensified depression of ventricular performance with the sudden loss or reversal of the normal sequence of atrioventricular contraction (Carleton,1967; Cohn,1982).

ATRIAL FUNCTIONS IN NORMAL CARDIAC CYCLE

Left atrial diameter and pressure changes were studied in the normal canine heart (Sassayama,1984). Subsequent to the P-wave of the ECG,left atrial pressure increases to form a small a wave 50 ms. After the onset of the atrial pressure rise , the left atrial wall begins to shorten leading to a decrease in left atrial diameter by an average of 4.6% from the end diastolic value of 19.7 mm. With atrial contraction, the left ventricle is expanded to a certain extent. The nadir of the atrial chamber shortening is during the isovolumic contraction phase of left ventricle. With the onset of ventricular contraction, the left atrium begins to expand, rapidly in the beginning and slowlyat the end, until the openning of the mitral valve.

The atrial diameter averaged 20.5 mm at the point of peak V-wave in the atrial pressure. After the mitral valve openning, the left atrial diameter remains constant until

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