# ASSESSMENT OF PAPILLARY MUSCLE TRACTION IN MITRAL VALVE PROLAPSE BY TWO DIMENSIONAL ECHOCARDIOGRAPHY AND INCIDENCE OF ARRHYTHMIAS BY 24 HOUR HOLTER MONITORING



A Thesis

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1994

# بسم الله الرحمن الرحيم


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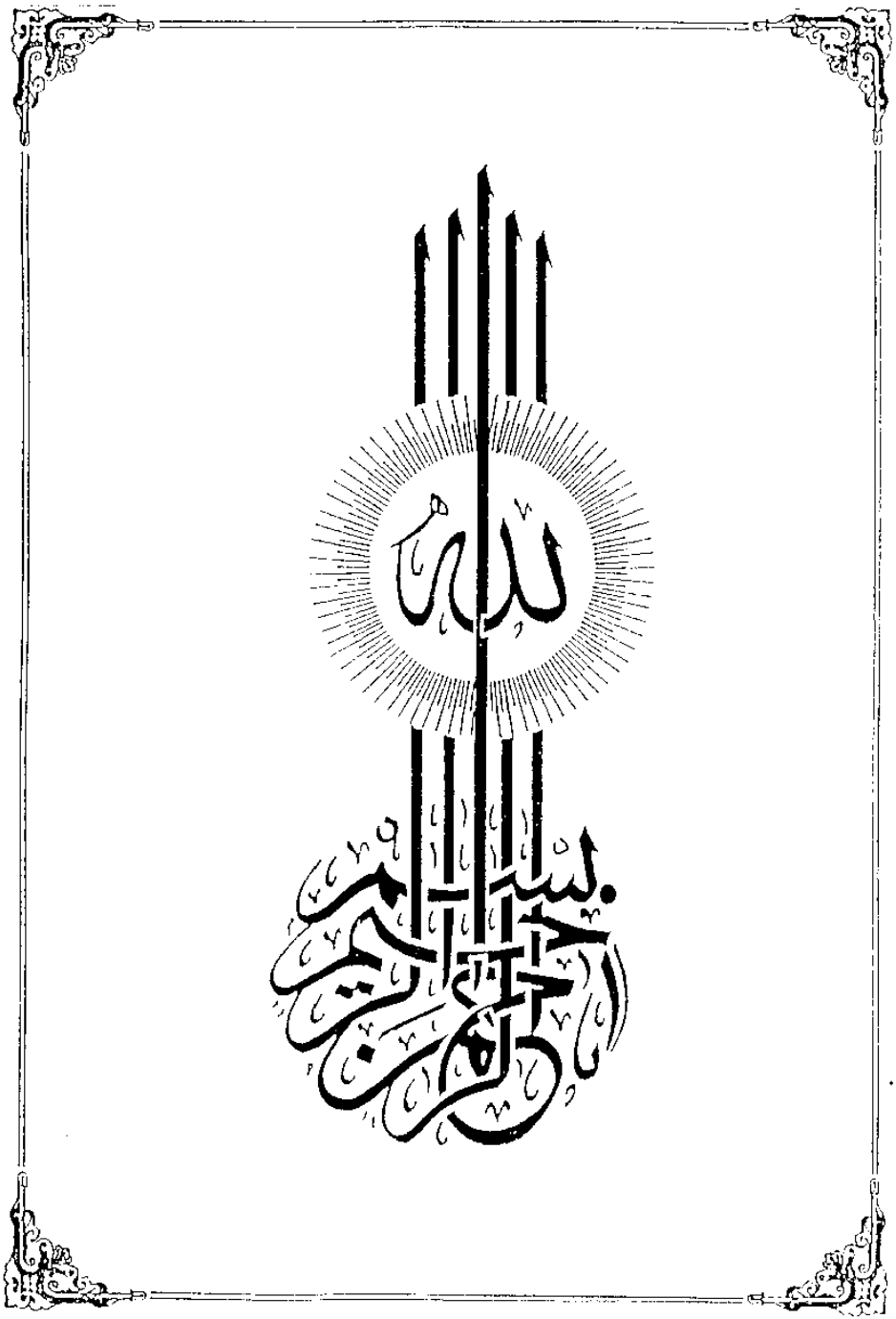

**This work**
**is dedicated to**
**My Father, Mother**
**&**
**Brothers**
**Tarek, Essam & Osama**

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**All thanks are due to God.**

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# LIST OF CONTENTS


**INTRODUCTION** 1

**AIM OF THE WORK** 4

**ANATOMY OF THE MITRAL VALVE** 5

*Developmental anatomy* 5
*Structural anatomy of the mitral valve* 8
*Blood supply of the mitral complex* 10
*Innervation of the mitral complex* 11
*Functional anatomy of the mitral valve* 13

**ÆTIOLOGY OF MITRAL VALVE PROLAPSE** 17

**CLINICAL PICTURE OF MITRAL VALVE PROLAPSE** 25

**PHYSICAL EXAMINATION OF A PATIENT WITH MVP** 37

**ECHOCARDIOGRAPHY OF MITRAL VALVE PROLAPSE** 45

*M-mode echocardiography* 46
*Two-dimensional echocardiography* 53

**OTHER INVESTIGATIONS OF MVP** 64

*Roentgenographic signs* 64
*Carotid pulse tracing* 65
*Jugular venous tracing* 66
*Electrocardiogram* 66
*Stress myocardial imaging* 67
*Cardiac catheterization and angiography* 69

**DIAGNOSTIC CRITERIA OF MVP** 73

**ARRHYTHMIAS AND MITRAL VALVE PROLAPSE** 78

*Pathogenesis of arrhythmia* 78
*Analysis of arrhythmia* 81

PATIENTS AND METHODS 91

RESULTS 98

DISCUSSION 130

SUMMARY AND CONCLUSION 139

RECOMMENDATIONS 142

REFERENCES 143

ARABIC SUMMARY

# INTRODUCTION AND AIM OF THE WORK

# INTRODUCTION

The term « mitral » is derived from the latin word « mitra » meaning cap, which in turn was derived from the greek word « mitra », meaning turban had dress. Bishops of the christian church wore had dresses which they called « miters », fashioned after those of ancient hebrew high priests (Robert & Perloff., 1972).

Mitral valve prolapse [MVP] is defined as mitral leaflet displacement beyond the normal range of leaflet motion relative to some reference structure, usually taken to be the mitral annulus (Robert., 1989).

MVP has been given many names including the systolic click murmur syndrome, Barlow syndrome, Billowing MVP syndrome, Ballooning mitral cusp syndrome, Floppy valve syndrome and redundant cusp syndrome.

MVP is considered as one of the most prevalent cardiac valvular abnormalities affecting as much as 5-10% of the population and is detectable in patients of all ages and both sexes.

MVP exists in the setting of a hereditary background. It is transmitted as an autosomal trait. It may represent one manifestation of a number of systemic connective tissue disorders and/or thoracic abnormalities. MVP can co-exist with rheumatic mitral stenosis. Both ischemic heart disease and MVP are common disorders that could co-exist not infrequently (Braunwald., 1992).

Previous angiographic studies of MVP are characterized by abnormal superior displacement of mitral leaflets above the level of the mitral annulus during systole. It has been postulated that this leaflet displacement may exert abnormal tension on the papillary muscle tips causing their superior traction or displacement and that such traction may have adverse pathophysiologic effects.

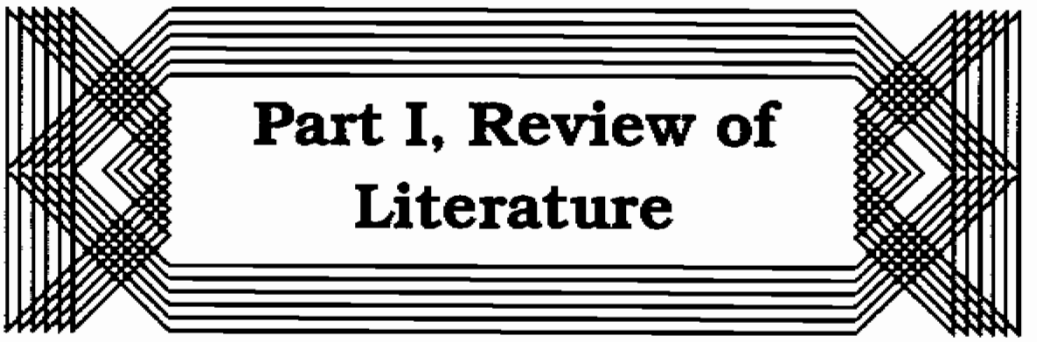
Recently, it has been observed that similar motion could be identified during echocardiographic examination of patients with classical MVP. Some studies suggested that in such patients, the papillary muscle and mitral leaflets move in parallel toward the left atrium in systole. Whereas in normal subjects, the papillary muscles move apically during systole in parallel with mitral annulus,

maintaining a relatively constant distance with respect to the annulus (Anthony., 1992).

# AIM OF THE WORK

In attempt to predict the occurrence of arrhythmia in patients with MVP, this study aims at detecting and assessing the papillary muscle traction non-invasively by two-dimensional echocardiography in patients with classic mitral valve prolapse.

It also aims at assessing the prevalence and type of arrhythmia and its relation to traction on papillary muscle in these patients by 24-hour Holter monitoring.



# Part I, Review of Literature

Chapter 1

# ANATOMY OF THE MITRAL VALVE

## Developmental Anatomy of the Mitral Valve

The atrioventricular valves develop from the endocardial cushions. In a 10 to 12 mm embryo, both atrioventricular orifices are surrounded by mesenchymal tissue. The definitive atrioventricular valves are derived from endothelium, mesenchyme and ventricular muscle tissue. In principle, a skirt of ventricular muscle tissue covered on its atrial side by mesenchymal tissue is formed at each atrioventricular orifice. The muscular tissue is attached to the wall of the ventricles by the trabeculae.

The chordae are initially thick, muscular and few in number. With further development, however, they are transformed into delicate fibrous stands (Clark & Von Mieropl.. 1989). As the junction between the left atrium proximally and the primitive ventricle distally is forming a relatively narrow AV canal, the endocardial cushions proliferate and not only form the tricuspid and mitral valves but also

close the ostium primum and the secondary interventricular foramen, between the 29$^{th}$ and 55$^{th}$ days (Netter., 1969). These AV endocardial cushions represent bulky mounds of mesenchyme, which functions either as a tissue adhesive to seal opposing masses during septal formation or as primitive valves (Edward., 1987). By approximately the 29$^{th}$ day, the bulky superior and inferior cushions grow toward each other and fuse by the 35$^{th}$ day, and they thereby form two orifices, the tricuspid and mitral orifices. Furthermore, tissue from these two cushions extends onto the septum primum along the rim of the ostium primum and actively closes this orifice by the 35$^{th}$ day (Netter., 1969).

Attenuation and relative absorption of the bulbo ventricular flange not only affects leftward shifting of the aorta toward the primitive LV but also allows rightward shifting of the AV canal with alignment between the tricuspid orifice and the proximal bulbus cordis [right ventricle]. Accordingly, the mitral orifice maintains its LV connection and the tricuspid orifice gains right ventricular connection by the thirty first day (Edward., 1987).

Only the free-floating anterior leaflet of the tricuspid and MV are derived primarily from the mesenchymal cells of the endocardial cushions, and they develop earlier than the remaining leaflet (Netter., 1969). The septal tricuspid leaflet and both posterior leaflets form by