Exercise Doppler Assessment Of Hemodynamic Response After Balloon Mitral Valvuloplasty Compared To Closed Mitral Commissurotomy

Thesis
Submitted In Partial Fulfillment Of
The Doctorate Degree In Cardiology

By

Mohsen Mahmoud Mahdy
M.B., B.Ch., M.S.

Under The Supervision Of

Prof. Mohamed Mamdouh El-Ashry
Professor of Cardiology
Ain Shams University

Prof. Wagdy Galal
Professor of Cardiology
Ain Shams University

Dr. Samir Saleh Wafa
Lecturer of Cardiology
Ain Shams University

Faculty of Medicine
Ain Shams University
1993

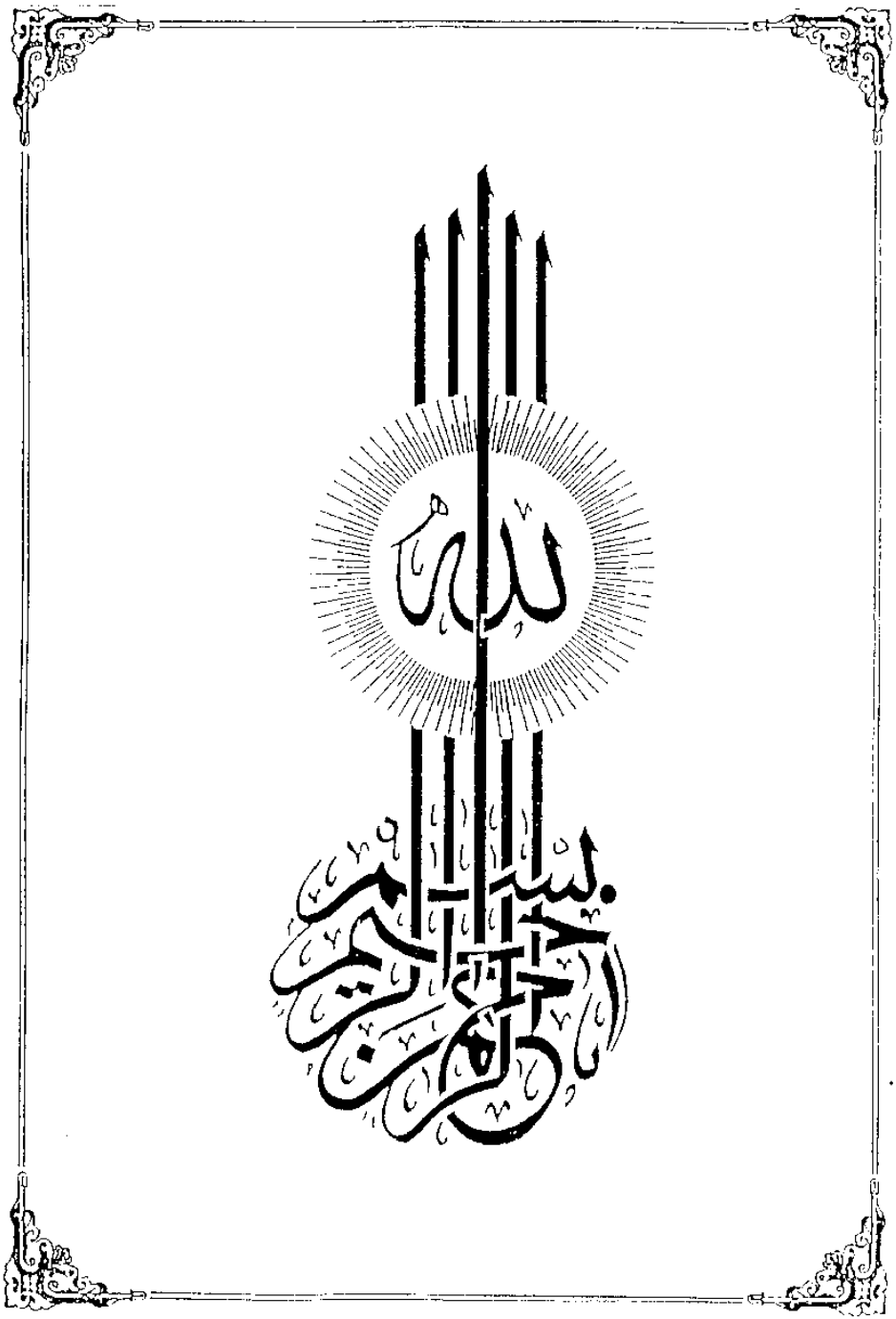
Acknowledgment

I wish to express my deep gratitude to Professor Mohamed Mamdouh El-Ashry, Professor of Cardiology, Ain Shams University, both for suggesting and supervising the whole study. Thanks for his valuable and appropriate suggestions.

I am greatly indebted to Professor Wagdy Galal, Professor of Cardiology, Ain Shams University, for his kind guidance and continuous encouragement , to him goes my sincere gratitude.

I wish to thank also Dr. Samir Saleh Wafa, Lecturer of Cardiology, Ain Shams University, for his kind care and assistance.

I am also grateful to Dr. Hany Fouad who helped me in the practical part of my work.



Dedication

To My Beloved Daughter Nada

Contents

Introduction and Aim of the Work

Introduction and Aim of the Work

Although the prevalence of rheumatic heart disease is declining in most industrialized nations, the disease continues to be endemic in Africa and the Middle East including Egypt, and continues to cause significant cardiac disability with subsequent considerable risk of mortality. Therefore, early and optimal management is mandatory.

Treatment of rheumatic mitral stenosis by surgical closed commissurotomy was reported to be successful in the 1940s ***(Harken et al., 1948)***. Balloon mitral valvuloplasty (BMV) was first described in 1984 as an alternative to the surgical approach ***(Inoue et al., 1984)***. Descriptive studies have reported successful short term results ***(Lock et al., 1985)***.

However, most of the previous studies demonstrated the beneficial effect of balloon mitral valvuloplasty or surgical closed commissurotomy on resting hemodynamics. It did not take into account the hemodynamics during activity and when performing various types of work. Such conditions may be simulated by subjecting the patient to physical exercise. The response to exercise helps to estimate the degree of mitral stenosis on the basis of increased pressures proximal to the mitral valve and increased transmitral pressure gradients, in addition, physical exercise may also help to uncover further disturbances resulting from pathological conditions of the myocardium.

Exercise hemodynamic assessment is difficult to perform during cardiac catheterization in a premedicated recumbent patient, and is only feasible when the brachial approach is used. Therefore, in the catheterization laboratory, exercise hemodynamic measurements have important practical limitations.

On the other hand, Doppler echocardiography has proved to be a useful, non-invasive tool for evaluation of the mitral flow dynamics during exercise in patients with mitral stenosis and can substitute for cardiac catheterization in most patients ***(Moro et al., 1986)***.

Aim of the Work

This study represents an attempt to evaluate resting and exercise hemodynamic changes produced by balloon mitral valvuloplasty and surgical closed commissurotomy in order to gain some information as to the efficacy of both procedures.

Review of Literature

Exercise Physiology and Hemodynamics in Normal Subjects

Dynamic exertion is the major form of exertion in every day activity. The hemodynamic effects of exercise involves complex and integrated hemodynamic and neurohormonal functions which culminate in increased oxygen delivery to and extraction by the exercising muscles. This hemodynamic response to exercise consists of effects of tachycardia, catecholamine stimulation and Frank-Starling mechanism.

In normal sedentary individuals, there is a tenfold increase in oxygen consumption from rest to maximal exertion ***(Bruce et al., 1973)***. In well-trained athletes the increase in maximal oxygen consumption can be as great as 20 to 30 times resting values. The various factors which contribute to oxygen consumption can be illustrated by the Fick equation :

$$VO_2 = HR \times SV \times (A - VO_2)$$

where VO_2 = Oxygen consumption,

HR= Heart rate,

SV = stroke volume,

$(A - VO_2)$ = difference between arterial and mixed venous oxygen content. Thus, in any individual, the maximal oxygen consumption depends on the integration of cardiac, metabolic, vascular and pulmonary responses.

The most striking cardiovascular alteration with exercise is an increase in heart rate. The main cause of the rise in heart rate with

exercise is sympathetic stimulation. Parasympathetic withdrawal also plays a role, although to a far lesser degree. The ability of the heart to increase its output appropriately or the increase in oxygen consumption at any level of exercise is met predominantly by an increase in heart rate. This is supported by the observation that stroke volume shows only minor increase during vigorous exercise ***(Donald et al., 1955)***.

Ross and Coworkers compared the response of cardiac output, stroke volume, and heart rate to a given level of bicycle exercise in normal subjects and also found that the increase in cardiac output was due to an increase in heart rate with a negligible contribution by change in stroke volume. However, during repeat exercise when heart rate was hold constant, there was comparable increase in cardiac output due to marked increase in stroke volume. Furthermore, when heart rate is artificially increased by electrical pacing in the absence of dynamic exercise, a major fall in stroke volume occurs ***(Ross et al., 1965)*** indicating that further cardiovascular adjustments are required for an adequate hemodynamic response to dynamic exercise.

The earliest hemodynamic response to acute hemodynamic exercise is probably a fall in systemic vascular resistance that reflects a marked vasodilatation of the resistance vessels in the exercising muscles. This is most marked at mild level of exercise intensity, with only minimal further decrease in vascular resistance occurring at nearly maximal work loads. The hemodynamic effects of this reflex are twofold. After-load falls and cardiac output is redistributed, the delivery of blood borne oxygen and glucose to working skeletal muscle is enhanced in the

presence of normal vasculature by reduction in skeletal muscle vascular resistance mediated by metabolic byproducts, fall in PO_2 or by release of locally acting vasodilators such as adenosine as well as the effects of circulating catecholamines and stimulation of sympathetic vasodilator nerve fibers and by sympathetically mediated vasoconstriction elsewhere which cause redistribution of blood away from the renal and splanchnic beds to exercising muscle.

Left Ventricular Systolic Performance During Dynamic Exercise:

Augmented left ventricular systolic performance during dynamic exercise results from the interaction of several factors. Tachycardia exerts a positive inotropic effect (Treppe phenomenon), but increased sympathetic nervous system activity appears to be the most significant factor, leading to enhanced myocardial contractility ***(Sonnenblick et al., 1965)***. The increase in cardiac output are paralleled by a marked increase in systolic blood pressure. Diastolic blood pressure remains unchanged and may even fall slightly. Mean arterial blood pressure increases only moderately reflecting the significant decrease in peripheral vascular resistance.

In some normal subjects, an increase in end-diastolic fiber tension (preload) may also occur, leading to improved cardiac performance by means of the Frank-Starling mechanism. The fact that left ventricular end-diastolic volume may not change or even decrease during dynamic exercises has been taken as evidence that the Frank-Starling mechanism does not usually contribute to the cardiac response to exercise. However, the effect of dynamic exercise on left ventricular

end-diastolic volume in normal subjects are complex and are influenced by contractile state (end-systolic volume), posture, heart rate and age. End-diastolic volume at rest is near maximum when a normal subject is supine, smaller when he is sitting and smallest when he is standing ***(Crawford et al., 1979)***.

In most normal subjects, supine bicycle exercise is accompanied with an increase in ejection fraction and other ejection indices of the left ventricle, a decrease in left ventricular systolic volume, and decrease or no change in end-diastolic volume ***(Gorlin et al., 1965)***. In occasional normal subject, the end-diastolic volume increases during supine bicycle exercise, thus an increase in stroke volume contributes to the rise in cardiac output ***(Gorlin et al., 1965)***. In upright position, left ventricular end-diastolic volume, cardiac output and stroke volume are lower than in supine position ***(Thadani and Parker., 1978)***. It has been shown that left ventricular end-diastolic volume, end-systolic volume, and stroke volume are larger during exercise than during comparable heart rates induced by pacing ***(Sonnenblick et al., 1965)***. Thus, it seems likely that the Frank- Starling mechanism is active during dynamic exercise, but may be masked by the salutary effects of tachycardia and increased myocardial contractility, both of them tend to reduce left ventricular volume.

The effect of dynamic exercise on left ventricular filling pressure is variable. ***Thadani and Parker*** (1978) observed an increase in both left ventricular filling pressure and mean capillary wedge pressure during supine and sitting bicycle exercise although the absolute values

for these pressures during exercise were lower in the sitting than in supine posture. On the other hand, some published data reported that left ventricular filling pressure and volumes during supine exercise either did not change or actually decreased from the resting values ***(Ross et al., 1965)***. These differences may be due to varying methods of carrying out exercise in the supine posture. The study done by ***Thadani and Parker***, the bicycle ergometer was placed so that the legs were elevated above the chest during supine exercise. Thus, increased venous return might have been responsible for the increased left ventricular filling pressures, but this seems unlikely because the left ventricular filling pressure in the same patients increased by a similar amount during upright bicycle exercise.

Caution must be used during interpreting left ventricular function obtained during dynamic exercise. The effect of advancing age on the exercise response should be kept in mind. With advancing age, there is reduction in heart rate and contractility response during exercise. The increase in cardiac output during exercise is accomplished by a significant increase in end-diastolic volume and in stroke volume ***(Port et al., 1980)***.

The diminished heart rate and contractility responses and resultant increased dependence on the Frank-Starling mechanism during exercise in the elderly may reflect an age-related decrease in responsiveness to beta adrenergic stimulation.