Evaluation of Mitral Valve Replacement on Beating Heart in Patients with Chronic Severe Mitral Regurge and Left Ventricular Dysfunction; Safety and Potential Benifit

Thesis

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بسم الله الرحمن الرحيم

قالوا

سبحانك لا علم لنا إلا ما علمتنا إنك أنت العليم الحكيم

صدق الله العظيم

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Hani Mahmoud Soliman Albatrek

List of Contents

Title	Page No.

List of Tables

List of Tables cont…

Table No.	Title	Page No.

List of Figures

Fig. No.	Title	Page No.

List of Figures cont…

List of Figures cont…

Abstract

There was significantly longer mechanical ventilation time and ICU stay in cold group I compared to beating group II.

Both the early (in hospital) and late (6 months) postoperative echocardiography showed a similar improvement in the two groups regarding left ventricular end diastolic diameter (LVEDD) and left ventricular end systolic diameter (LVESD) but a significant difference in left ventricular function in favor of the beating technique as the left ventricular ejection fraction (LVEF) and left ventricular fraction shortening (LVFS) were better in beating group II than cold group I.

The significant differences in EF and FS which was in favor of beating group II over cold crystalloid cardioplegic technique in early and late postoperative echocardiographic results, explains the generally better course of patients of this group in hospital and during the early follow up period, this together with the absence of related perioperative mortality and the major complications suggests that it is safe and beneficial to LV function.

Keywords: LV end-systolic volume **-** ejection fraction- LV end-systolic volume-

INTRODUCTION

After the initial compensatory phase of chronic mitral regurgitation, left ventricular (LV) systolic contractility becomes progressively impaired [1]. The calculated ejection fraction (EF) is usually falsely high. LV End-systolic dimension or LV end-systolic volume (LVESV) is less dependent on preload than is ejection fraction and can be used as a better measure of left ventricular systolic contractile function [2].

When LV systolic function is affected, the attention is directed to the myocardial protection during mitral valve surgery. The term "myocardial protection" refers to strategies and methodologies used either to attenuate or to prevent post ischemic myocardial dysfunction that occurs during and after heart surgery, which is attributable, in part to a phenomenon known as ischemia/reperfusion-induced injury [3].

Most of the current myocardial protective strategies utilizing cold cardioplegia have withstood the test of time, providing safe and effective myocardial protection during various cardiac operations. Nevertheless, all cardioplegic myocardial protective strategies devised to date subject the heart to a period of the so called "mandatory ischemia", where the heart is without circulation. This subsequently leads to reperfusion injury when the aorta is de-clamped [4].

Then the concept of warm heart surgery with the use of continuous warm blood cardioplegia as a mean of myocardial protection and prevention of ischemia evolved. Since its introduction, multiple studies have shown that warm heart surgery is comparable to cold cardioplegia in its safety record. From a metabolic standpoint, it provides superior myocardial protection. As well, high-risk patients who may have metabolically compromised hearts show greater benefit from reduced ischemic damage of the myocardium [5,6].

As a natural extension of warm heart surgery, beating heart valve surgery was born. Fundamental question was posed: why arrest the heart if technically adequate valve procedures could be accomplished with continuous warm perfusion? [7].

Preliminary data suggest that beating heart valve surgery is safe and that there may be a benefit to high risk patients [8].

AIM OF THE WORK

The aim of this work is to evaluate mitral valve replacement on beating heart in patients with chronic severe mitral regurgitation and left ventricular dysfunction.

PATHOPHYSIOLOGY OF MITRAL REGURGITATION

The functional competence of the mitral valve relies on the interaction of the mitral annulus and leaflets, chordae tendineae, papillary muscles, left atrium, and left ventricle. Dysfunction of any one or more components of this valvular ventricular complex can lead to mitral regurgitation [11].

Chronic mitral regurgitation is a volume overload lesion of the left ventricle. With gradual development of a regurgitant orifice, an abnormal orifice develops between the left ventricle and the left atrium during systole, allowing blood to regurgitate backwards into the left atrium. This regurgitated volume in the next diastole returns to the left ventricle, thus increasing the left ventricular end diastolic volume by the Frank Starling mechanism, there is an increased force of contraction, thus increasing total left ventricular stroke volume [12].

Since the regurgitant mitral orifice is in parallel with the aortic orifice, the resistance to ventricular emptying (left ventricular afterload) is reduced in mitral regurgitation. Left ventriculoatrial regurgitation occurs not only during ejection but during isometric contraction as well [13].

The large left ventricular end diastolic volume increases resting left ventricular wall tension, stimulating myocardial hypertrophy, which allows the left ventricle to grow larger to accommodate the large diastolic volume (so, called eccentric hypertrophy). This enlargement of the ventricular capacity allows for greater diastolic volume without raising left ventricular diastolic filling pressure [12].

The reduced load on the ventricle allows a greater proportion of the myocardium contractile energy to be expanded in shortening than in tension development and explains how the left ventricle can adapt to the load imposed by chronic mitral regurgitation. Thus it appears to be the reduction in left ventricular tension in mitral regurgitation that allows the left ventricle to increase its total output and ultimately accounts for the fact that patients with moderately severe mitral regurgitation can sustain large regurgitant volumes for prolonged periods, while maintaining forward cardiac output at normal levels for many years [14].

Factors which Influence Volume of Mitral Regurgitation Blood Flow

The volume of mitral regurgitant flow depends on the size of the regurgitant orifice as well as on the pressure gradient between the left ventricle and left atrium [14].

However, it is obvious that regurgitant orifice and heart rate are more influential in determining regurgitant volume than the gradient across the valve, since during systole, flow can occur out of the left ventricle in two directions, normally into