Postoperative Evaluation Of The Left Ventricular Function After Mitral Valve Replacement In Patients With Border Line Left Ventricular Contractility

A THESIS

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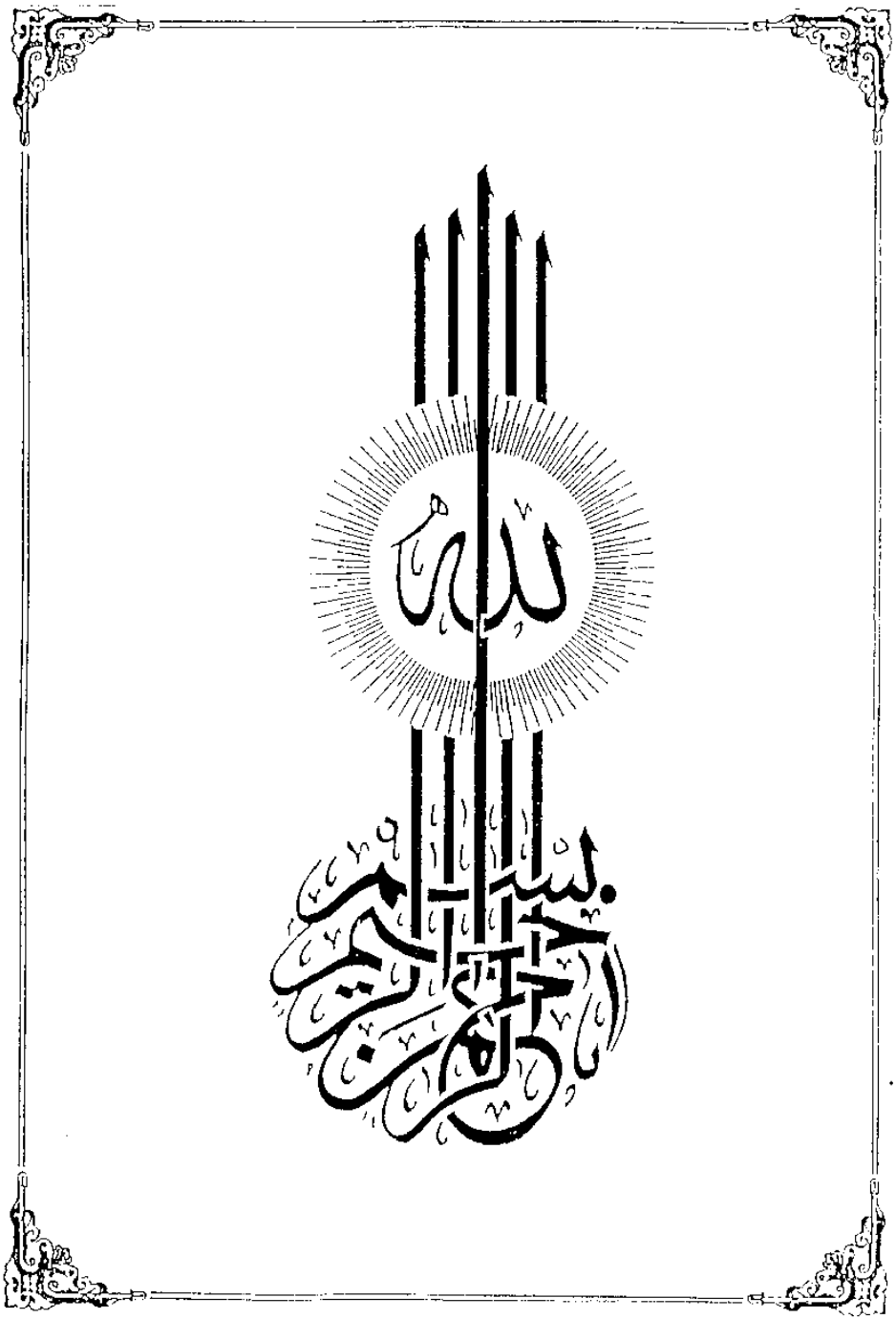

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ABBREVIATIONS

A C E I	= angiotensin enzyme inhibitores
B P	= blood pressure
Cir. Fi. S.	= circumferential fibre shortening
E C G	= electrocardiogram
E D D	= end-diastolic diameter
E D V	= End-diastolic volume
E F	= ejection fraction
E S D	= End-systolic diameter
E S V	= End-systolic volume
F	= female
F S	= fraction shortening
H R	= heart rate
L V	= Left ventricle
L V H	= Left ventricular hypertrophy
M	= male
MI	= Myocardial infarction
MR	=Mitral regurge
MVR	= Miteral valve replacement
NHI	= National heart Institute
N Y H A	= New York Heart Association
No	= Number
Postop	= Postoperative
Preop	= Preoperative
Rg V	= Regurgitation volume
S E F	= Systolic ejection fraction
Std Dev	= Standard deviation
Y	= Year

INTRODUCTION

In chronic compensated MR; dilatation and hypertrophy of the left ventricle proceed and chamber compliance of the ventricle increases permitting a larger stroke volume to be ejected with a decrease (or at least without a further increase) in preload.

Several studies indicate that at this stage of the disease, systolic stress is generally within the normal range (Wisenbaugh et al 1988, Zile et al 1985).

According to Carbello, this chronic compensated phase of MR is characterized by increased preload, normal contractile function, and an afterload that has increased from subnormal to normal levels as a consequence of an increased ratio of chamber radius to wall thickness. (Carbello, B. 1988).

Elimination of the mitral leak results in a further increase in left ventricular afterload. When a chronic shunt from the left ventricle to the left atrium was eliminated, LV wall stress increased acutely (Renkin, et al. 1975) and a similar finding was observed by Wong and Spotnitz in six patients with MR studied intraoperatively immediately following valve replacement (Wong and Spontize, 1981).

However; numerous studies indicate that MVR in patients with chronic MR results in a fall in SEF regardless of the preoperative. State of LV function. In a recent review of the response to surgical correction of chronic AR and MR; Gaasch and Coworkers in 1985 reported that in chronic MR; an average decrease in SEF was found following MVR in each of 10 separate reports, with an overall reduction in SEF among the 10 studies from 0.59 to 0.49 (Gaasch et al. 1985).

Logic would suggest that explanation for this fall in SEF is the combined decrease in preload and increase in afterload observed following MVR. There are , however several reasons to question this conclusion. First, although LV wall stress increases immediately following MVR, when regression of hypertrophy and remodeling of the ventricle has occurred months later, this may not be the case.

Zile and associates reported no change in systolic wall stress one year following M.V.R for chronic MR. (Ziel, et al., 1985).

Second, whereas SEF usually falls following MVR, this does not appear to be the case when surgical correction of MR is accomplished by mitral valve repair. Of three studies of systolic function following repair of MR, SEF rose in two, and the overall SEF for the three groups increased

from 0.59 to 0.62 (Duran et al 1980, Bonchek et al 1982, David et al 1983).

It has been suggested that loss of the integrity of papillary muscles and chordae tendineae following MVR is an important factor responsible for postoperative reduction in systolic function and that valve repair with preservation of the continuity between mitral annulus and the LV wall plays a critical role in sustaining LV function(Duran, et al 1980, David et al 1983) .

Although a fall in SEF is uniformly observed following MVR in chronic MR; this phenomenon cannot be attributed to elimination of the MR per se. (Herbert J., 1992)

In patients whose SEF is between 0.55 to 0.70 one may suspect that LV function is likely to be normal, but it behooves the clinician to examine LV chamber volumes carefully, particularly ESV, and to proceed with an analysis of stress-shortening relation in an effort to characterize myocardial function better. (Herbert J., 1992)

Serial examination using echocardiography or radionuclide ventriculography at 4 to 6 month intervals should be helpful in identifying a trend in the patient's hemodynamic profile. (Herbert J., 1992).

If the SEF is less than 0.55, one must assume that there is some LV dysfunction and both the extent and duration of this dysfunction should be documented as accurately as possible. (Herbert J. 1992)

In the relatively asymptomatic patient with severe mitral regurgitation, the possibility of developing "silent" irreversible myocardial dysfunction poses an important problem(Ross J Jr., et al. 1985).

Mitral regurgitation places relatively favorable systolic loading conditions on the left ventricle, and eccentric hypertrophy coupled with the low-impedance leak early and late in systole yields a high normal ejection fraction when contractility is not reduced. (Eckberg DL., et al. 1973)

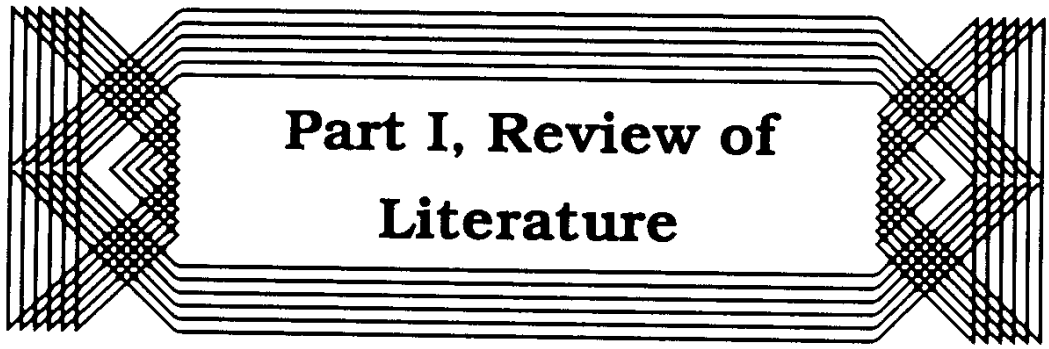
Even when myocardial contractility becomes severely depressed ; a relatively normal ejection fraction can be maintained although mean Vcf is sometimes reduced. (Eckberg DL., et al 1973).

Thus if significant cardiomegaly is seen on the chest roentgenogram or found on physical examination in such patients, a baseline echocardiographic study should be obtained to assess left ventricular performance (Ekberg DL., et al 1973).

In contrast to aortic regurgitation; studies before and after replacement indicate that left ventricular function falls to some degree

AIM OF THE WORK

Assessment of ejection fraction after mitral valve replacement in patients with borderline left ventricular contractility which ranges between 0.50-0.55 before miteral valve replacement.



CLINICAL ADAPTATION, LEFT VENTRICULAR MECHANICS AND CONTRACTILE MUSCLE FUNCTION IN CHRONIC MITRAL REGURGITATION

Mitral regurgitation develops as a result of slow relentless disease processes or as a result of one or more abrupt decrements in valve function. The eventual results may be quite similar, but the differences between acute and chronic mitral regurgitation are profound (Table I). The major burden and threat of acute mitral regurgitation is to the pulmonary venous circulation and the lungs, whereas in chronic mitral regurgitation, the major burden is carried by the left ventricle. (Braunwald, et al., 1969; Rackley et al., 1970).

Table 1 Left Ventricular (LV) and Circulatory Dynamic in Mitral Regurgitation (MR)

		Chronic MR	
	Acute MR	Compensated	Decompensated
Heart rate	↑	N	↑
Effective cardiac output	↓	N	N-↓
Systemic vascular resistance	↑	N	↑
LV end-diastolic pressure	↑↑↑	N-↑	↑-↑↑↑
LV end-diastolic volume (EDV)	↑	↑↑	↑↑-↑↑↑
LV end-systolic volume	N-↓	↑	↑↑
Ejection fraction	↑	N	↓-↓↓
Total stroke volume (TSV)	↑	↑↑↑	↑↑
Regurgitant volume (RgV)	↑↑	↑↑-↑↑↑	↑↑-↑↑↑
Forward stroke volume	↓	N	N-↓
Ratio of RgV/TSV	↑↑↑	↑	↑↑
Ratio of RgV/EDV	↓↓↓	↑↑	↑
LV mass	N	↑-↑↑	↑↑
Ratio of EDV/LV mass	↑	N-↑	↑↑
LV systolic stress	↓	N	↑-↑↑
LV chamber stiffness	N	N-↓	↓
Myocardial stiffness	N	N	↑-↑↑
LV oxygen consumption	N-↑	↑-↑↑	↑↑-↑↑↑

RgV = Regurgitation volume

The volume of mitral regurgitant flow is determined hydraulically by the size of the regurgitant orifice and the magnitude and duration of the systolic pressure gradient across the valve. (Gaasch et al., 1985). Thus, by rearranging the factors in the Gorlin formula for valve orifice area, the equation representing mitral regurgitant volume (MRV) is:

$$MRV = MROA \sqrt{LVP_S - LAP_S} \,.Ts.C$$

where MROA = mitral regurgitant orifice area, LVPs

LAPs = left ventricular and left atrial mean systolic pressures respectively,

Ts = time of duration of systole, and

C = constant.

Recognizing that the pressure gradient across the valve is variable and that the regurgitant orifice area in some forms of mitral regurgitation is dynamic, one may explain the effects of a variety of hemodynamic interventions and therapies on regurgitant volume. For example, venodilators can be of value in reducing mitral regurgitant flow in patients with ventricular enlargement and papillary muscle dysfunction by reducing ventricular size and the functional mitral regurgitant orifice area. (Bargemhagen et al., 1977; Keren et al., 1989). In some patients with mitral valve prolapse or hypertrophic cardiomyopathy, however, venodilators may augment mitral regurgitant flow through an influence on ventricular size and mitral leaflet coaptation. In patients with fixed-orifice mitral regurgitation (i.e., those with chronic rheumatic disease or annular and valvular calcification), venodilation might reduce pulmonary venous hypertension and congestion in association with an increased regurgitant volume; this could occur if the fall in left atrial pressure was greater than the reduction in left ventricular systolic pressure. By contrast, a pure reduction in left ventricular systolic pressure would reduce mitral regurgitation, particularly in the hypertensive patient. These considerations of the dynamic determinants of the mitral regurgitant volume are especially relevant to the development of a rational therapeutic strategy (Keren et al., 1989).

The etiology of mitral disease has an important effect on the magnitude of regurgitation and its reversibility. In coronary heart disease, for example, the development of significant mitral regurgitation requires a critical combination of papillary muscle dysfunction and abnormal wall motion; the extent of the wall motion dysfunction and its location interact as important determinants of the regurgitant volume (Rackley, et al., 1970; Rackley et al., 1977).

In idiopathic dilated cardiomyopathy, mitral regurgitation is determined by altered leaflet function and annular size. In such patients annular dilatation does not develop in direct proportion to the degree of left ventricular enlargement; (Baltwood et al., 1983; Chandraratna et al., 1981), ventricular volume appears to be less important than annulus size. Changes in ventricular geometry (i.e., a more spherical shape) also affect competence of the valve. These and other nonrheumatic alterations in the functional components of the mitral valve influence the severity, operability, and prognosis (Klughaupt, 1969). This is especially the case in patients with mitral valve prolapse where a variety of pathophysiologic mechanisms contribute to the hemodynamic lesion and its consequences (Jeresaty, 1973), (Pastermac, 1982).