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Original Article

Left ventricular wall mechanics in hypertension - an echocardiographic study

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ABSTRACT

The present study defines relationship between the extent of left ventricular hypertrophy and left ventricular systolic performance in hypertensive patients. Fractional fibre shortening was used as determinant of systolic performance in left ventricular hyertrophy. End systolic wall stress, obtained using the systolic cuff blood pressure combined with M-mode study as an index of left ventricular hypertrophy. The study was conducted on 66 subjects of both sexes, age group ranging from 40 to 60 years in hypertensive patients at Kilpauk Medical College. The standard t-test was used for comparative analysis. Ejection fraction (EF%) is normal and even above the control group, but end systolic stress is significantly higher in hypertensive group. Fractional Fiber shortening (FFS%) in group with hypertension was significantly less than the control group. M-mode echocardiography remains relatively inexpensive diagnostic tool in assessing the myocardial performance in hypertensive patients for prevention of onset of target organ damage.

1. Introduction

Hypertension is a hemodynamic derangement. It is the primary and most important manifesting symptom of hypertensive vascular disease.Cardiac compensation for the excessive workload imposed by increased systemicpressure is at first sustained by left ventricular hypertrophy. Left ventricular hypertrophy is a powerful independent predictor of cardiovascular morbidity and mortality. The heart undergoes hypertrophy without ventricle dilation, resulting in a decreased contractile ability and subsequent failure[1].It is concluded that the occurrence of impaired left ventricular systolic function with increasing left ventricular mass in hypertensive subjects reflects a deficiency in intrinsic contractile performance of the hypertrophied myocardium.

Recent works have shown that patients with increased left ventricular mass index and high relative wall thickness have depressed mid wall fractional fibre shortening despite normal ejection fraction[2] .M-mode echocardiography is a non - invasive excellent tool for assessment of left ventricular (LV) structure and function in hypertensive subjects. Left ventricular hypertrophy is a physiological process of adaptation of the heart to mechanical load increase. In the presence of hypertrophy, the thick walled ventricle allows increased pressure generation without improved myocardial contractility[3].

This study was undertaken to define the relationship between the extent of left ventricular hypertrophy and left ventricular systolic performance in hypertensive patients. Left ventricular cavity measurements and wall thickness at the end diastole and end systole and shortening fraction were obtained with the precision by M - mode echocardiography. Fractional fibre shortening was used as the determinant of left ventricular systolic performance[4]. End systolic wall stress obtained using the systolic cuff blood pressure combined with M-mode study was used as an index of the adequacy of left ventricular hypertrophy. This index was significantly raised in Hypertension[5].

Hence we planned to assess the Left ventricular systolic function in patients with pressure overload hypertrophy by stress-shortening relationship. Increased left ventricular wall thickness exhibits reduced shortening velocity at the mid wall and moreover that is not apparent by any endocardial or conventional measurements. As the significant impairment of fractional fibre shortening is due to alteration in dimensions of LV wall thickness, LV cavity, LV geometry and fibrous changes in LV myocardium the abnormal feature of M-mode parameters in hypertensives have been evaluated from comparison with data from normal subjects in our study. Relationships between the quantities measured have

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been studied to give fuller understanding of changes taking place in the ventricle, so that the data can be employed as the basis for a non -invasive assessment of left ventricular systolic function in hypertensive subjects.

2. Materials And Methods

The study was conducted on 66 subjects of both sexes, age group ranging from 40 to 60 years, who have a previous diagnosis of hypertension, which was established by assessment of medical records in Hypertension clinic, Medical Department, Kilpauk Medical College. These subjects later underwent M-Mode Echocardiography analysis in the cardiology department, Kilpauk Medical College. Most patients (85%) were under treatment and the remaining patients were out of medications prior to echocardiography.

The following exclusion criteria were considered. The subjects excluded in our studies were patients of severe hypertension [pressure levels(systolic BP > 170 mmHg, diastolic BP > 104 mmHg)], Secondary hypertension, diabetes, Chronic renal failure, Coronary heart disease, patients having clinical signs of congestive heart failure, and all patients with an inadequate echocardiography window or with valvular lesion evidenced on echocardiogram.Out of the local referential population, a group of 45 normotensive individuals of both sexes were chosen to undergo M-Mode echocardiography assessment as a control group.

On M-Mode echocardiography the following structural parameters were assessed.

I. Thickness of posterior wall and the interventricular septum.
II. Left ventricular diameter during systole and diastole.
III. All measurements of the septum and the posterior wall were performed at the end of diastole according to recommendation of the American Society of Echocardiography[6].
IV. Direct measurement of ventricular diameter and volume of the chambers obtained in echocardiography allowed obtainment of parameters of systolic function and hemodynamic derivatives such as Stroke Volume-(SV) was measured by considering LV as a prolate ellipse, in this geometrical concept, the SV was calculated by following formula[7].

a. $SV = (LVID_d)^3 - (LVID_s)^3$, where
$LVID_d$ - Left ventricular internal diameter at diastole
$LVID^s$ - Left ventricular internal diameter at systole
b. Cardiac output (CO) = Stoke volume x heart rate
c. Cardiac index (CI) = Cardiac output/Body surface area
d. Ejection fraction $(EF)\% = \frac{(LVID_d)^3 - (LVID_s)^3}{(LVID_d)^3}$

This EF can be regressed to corrected EF by Teichholz's formula[20]

$$\text{Ef Teichholz} = \frac{7.0}{2.4 + (LVID_d)} \times EF^3$$

e. Percentile change in FFS%[20] = $\frac{EDD-ESD}{EDD} \times 100$

V. Left ventricular load parameters[20]

a. Index of Preload=EDV/BSA
where EDV=End diastolic volume
BSA=Body surface area

b. Assessment of after load

1. Peripheral Vascular Resistance Index

Peripheral vascular resistance does not reflect the after load status at LV myocardial level. The after load seen at LV myocardial level is actually the systolic wall force or stress. This wall force or stress can be assessed by use of law of Laplace (T= P x r / Th).

2. End systolic stress (ESS): Echocardiography provides adequate data to measure the left ventricular wall stress, by using the methods described by Gross Man et al.,[8] Richeck et al. [9]and Quinones et al[3]. Based on the their concept, following mathematical formula measures the end systolic stress.

$$ESS = \frac{0.33 \text{ X ESD x SBP}}{PWT\ (1 + PWT/ESD)} \text{ din / seg / m}^2$$

Where ESD - End systolic diameter of LV
EDD - End diastolic diameter of LV
PWT - Posterior wall thickness of LV
SBP - Systolic blood pressure

VI. Left Ventricular Mass Index =
$(0.8 \text{ x } (1.04 \text{ x EDD} + IVSTd + PWD)^3 - EDD3 + 0.6) / BSA$
in g / m^2

VII. LV Hypertrophy Index (h/r)
or RWT[20] (Relative wall thickness) = IVSTd + PWT + EDD
IVSTd - Inter ventricular septum thickness
PWT - Posterior wall thickness
EDD - End diastolic diameter

3.1 Statistical Test

For statistical analysis, the parameters were recorded with SPSS for windows (V -11.5) software. Demographic and pressure parameters as well as echocardiography analysis were presented as mean ± standard error, between the normotensive and hypertensive groups.

The standard t-test was used for comparative analysis. The tests were considered statistically significant if P - value were < 0.05 (The null - hypothesis was applied). To know the inter - relationship between the parameters of determinants of LV function , graphic method was used for the correlation analysis.

4.Result

Table I showing parameter of left ventricular function among normotensive and hypertensive subjects determined by M-Mode echocardiography
Group Statistics / t - test for equality of mean

L.V.function paramters	Normotensive group n = 45		Hypertensive group n = 66		P.Value
	Mean	S.D	Mean	S.D	
S.V.Index (ml/min/sqm) Stroke volume index	35.28	4.17	36.09	9.3	.589
Cardiac index (L/min/Sqm)	2.6	.36	2.8	.93	.108
EF% Ejection fraction	59.05	3.9	67.32	7.17	.000*
FFS% Fractional fibre shortening	39.56	3.6	35.45	7.6	.001*
PVRI (din/seg/m^2) Peripheral Vascular Resistance Index	1045.94	191.60	1554.95	509.46	.000*
ESS (x 10^3 dynes/cm^3) End systolic stress	84.596	14.24	96.97	24.13	.003*
Preload index	60.01	7.9	53.73	12.9	.004*
Systolic BP mm Hg	122.18	6.77	160.55	15.51	.000*
Diastolic BP (mm Hg)	76.22	4.73	99.21	8.17	.000*
LVMI (gm/m^2) Left Ventricular Mass Index	95.79	10.7	115.46	32.88	.000*
LV Hyp. Index (h/r) (RWT) LV Hypertrophy Index(h/r) or RWT (Relative wall thickness)	.3933	3.425	.4976	8.877	.000*

* P < 0.05 Statistically significant

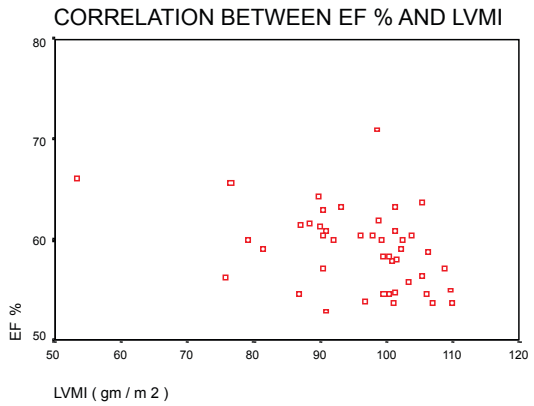
CORRELATIONS - 1
Control group

Descriptive Statistics			
	Mean	Std. Deviation	N
LVMI (gm/m^2)	95.7569	10.7469	45
EF%	59.0509	3.9328	45

Correlations			
		LVMI (gm/m^2)	EF%
LVMI (gm/m^2)	Pearson Correlation	1.000	-.393**
	Sig. (2-tailed)	.45	.008
	N		45
EF%	Pearson Correlation	-.393**	-.393**
	Sig. (2-tailed)	.008	.008
	N	45	.45

**. Coorelation is significant at the 0.01 level (2-tailed)

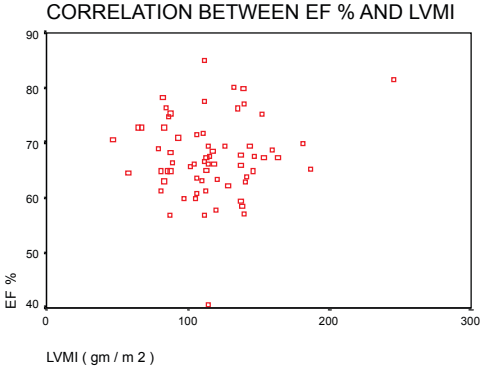
GRAPH



CORRELATIONS - 2
Hypertensive group

Descriptive Statistics			
	Mean	Std. Deviation	N
LVMI (gm/m^2)	115.4682	32.8853	66
EF%	67.3238	7.1754	66

GRAP



Correlations			
		LVMI (gm / m^2)	EF %
LVMI (gm/m^2)	Pearson Correlation	1.000	-.393**
	Sig. (2-tailed)	.66	.008
	N		45
EF%	Pearson Correlation	.087	1.000
	Sig. (2-tailed)	.490	.66
	N	66	

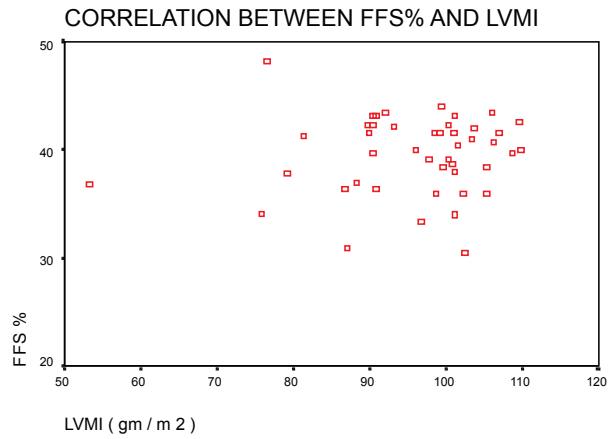
**. Corelation is significant at the 0.01 level (2-tailed)

CORRELATIONS - 3

Control group

Descriptive Statistics			
	Mean	Std. Deviation	N
FFS%	39.5631	3.6208	45
LVMI (gm/m^2)	95.7569	10.7469	45

GRAPH



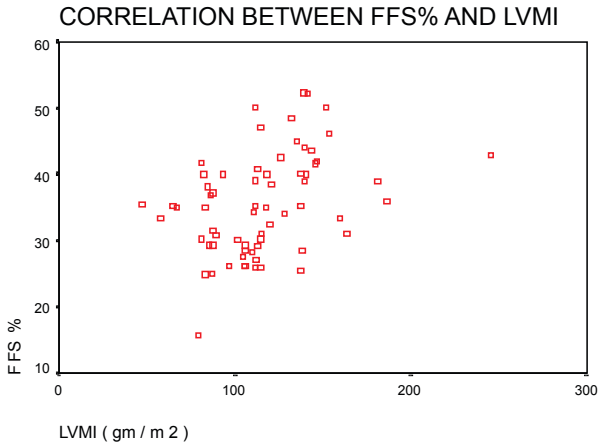
Correlations			
		LVMI (gm / m^2)	EF %
FFS %	Pearson Correlation	1.000	0.079
	Sig. (2-tailed)	.45	.607
	N		45
LVMI (gm/m^2)	Pearson Correlation	.079	1.000
	Sig. (2-tailed)	.607	
	N	45	.45

CORRELATIONS - 4

Hypertensive group

Descriptive Statistics			
	Mean	Std. Deviation	N
FFS%	35.4597	7.6531	66
LVMI (gm/m^2)	115.4682	32.8853	66

GRAPH



Correlations			
		LVMI (gm / m^2)	EF %
FFS %	Pearson Correlation	1.000	.383**
	Sig. (2-tailed)	.66	.002
	N		66
LVMI (gm/m^2)	Pearson Correlation	-383**	1.000
	Sig. (2-tailed)	.002	
	N	66	.66

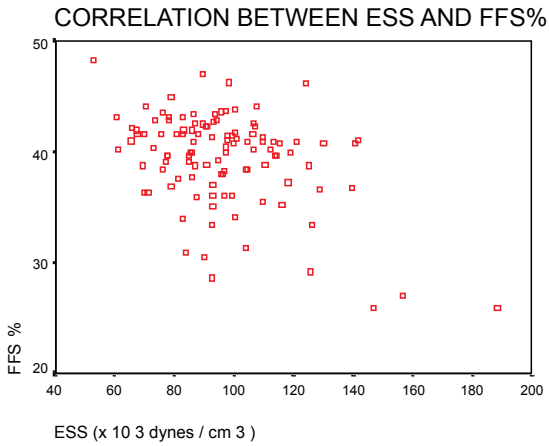
**. Corelation is significant at the 0.01 level (2-tailed)

T-TEST

Hypertensive and control group

Paired Samples Statistics				
	Mean	N	Std. Deviation	Std.Error Mean
FFS %	39.4778	111	4.1705	.3958
ESS (x 10.3dynes/cm3)	96.0728	111	21.4051	2.0317

GRAPH



Paired Samples Correlations

	N	Correlation	Sig
FFS % & ESS (x 10 3 dyens / cm^3)	111	-.422	.000

5. Discussion

The assessment of left ventricular function is important in many circumstances in which information on myocardial status may influence decision in patient management [10].

Quantitative echocardiography is the most convenient way to study LV function, because the procedure is relatively of low cost and the possibility to gain information to single beat intrinsic myocardial contractility, by assessing the ability of left ventricle minor axis to shortening during systole at the level of midwall [11].

After load (ESS) is the force distributed in the ventricular wall during ventricular ejection. This force is complex in the intact heart and dependent on viscous and inertial properties of the blood, ventricular volume, LV wall thickness and peripheral arterial resistance.

Afterload is never constant during ventricular ejection, but continuously decline as the LV volume and mid wall radius decrease as predicted by Laplace relations i.e = P x r/ Th where = wall stress / tension, P = pressure, r = radius of LV and Th = thickness of LV wall.

This type of cardiac contraction with continuously varying load is called auxotonic contraction. Increasing afterload (ESS) causes immediate changes in myocardial wall shortening and contractile state [12].

For many years, it was unclear whether myocardium, that had undergone pressure over load hypertrophy, exhibited normal contractility during systole. Most invitro studies on myocardium obtained from animal models of cardiac hypertrophy had suggested that such heart muscle was hypocontractile, by contrast, studies in human with left ventricular hypertrophy (LVH) had suggested that contractility of hypertrophic myocardium was normal or even perhaps increased. It has been suggested that this difference might be due to the fact that in human studies, chamber volume had been studied, rather than myocardial mechanisms [13]An improved understanding of left ventricular systolic function might also result from the use of modified midwall measurement. Quinones et al[14] calculated left ventricular stress - velocity relationship from endocardial and conventional midwall measurement. In the current study, mid wall fractional fibre shortening rate is less than the endocardial shortening in the hypertrophied heart.

Active reduction of left ventricular chamber size during systole is the final effect of complex interaction mechanism involving layers of differently oriented myocardial fibres, the shortening of which is less than the one measured as shortening of the left ventricular diameter at the level of the endocardium. This biological phenomenon is particularly evident in conditions such as arterial hypertension, in which left ventricular geometry is altered[15] Due to the double effect of contraction on both the longitudinal (shortening) and transverse (thickening) axes of the myocardial fibers the shortening of single myocardial fibres is amplified at the level of the endocardium and this amplification is a function of wall thickness. Increase wall thickness can enhance at the endocardial level the effect of myocardial fibers with reduced shortening[16], allowing preservation of ejection fraction despite depressed midwall shortening, through a "contractile gradient", proceeding from epicardium to endocardium. This is detectable using tagged MRI or even quantitative echocardiography [17] Chronic pressure over load of the left ventricule results in an appropriate increase in myocardial mass (concentric hypertrophy) that is characterized by low chamber radius and wall thickness R/Th ratio. The increase in myocardial mass that accompanies chronic volume overload (eccentric hypertrophy) is characterized by a normal or only minimally increased r/Th. As long as the ventricle remains compensated, the increased thickness is proportional to the increase in radius and r/Th remains normal[14]. Krayen buchi et al developed the theory that midwall shortening was less than normal in patients with concentric LVH and greater than normal in those with eccentric LVH. In Krayen buchi analysis, endocardial shortening was equal in normal subjects and patients with concentric and eccentric hypertrophy[2]

It was found that left ventricular hypertrophy is an important and independent predictor of cardiovascular morbidity and mortality[18]. The question arises as to the mechanism by which increased left ventricular mass may increase cardiovascular risk. Several possibilities suggest themselves. In the first place, LVH increases myocardial oxygen demand, while decreasing coronary blood flow reserve, creating a supply demand mismatch, which will predispose to cardiac ischaemia and sudden death[19] . Secondly many factors that predispose to LVH (especially in hypertension) are also associated with atherosclerotic disease, including coronary heart disease. Finally LVH is known to predispose to ventricular dysrhythmias and hence sudden death, even in the absence of overt coronary disease[12].

Assessment of LV function by midwall fractional fibre shortening has indeed been shown to be more physiologically accurate in representing myocardial performance than is endocardial shortening, a measure of chamber performance especially in the presence of LV geometry abnormalities such as those occurring in arterial hypertension[20].

Large number of pharmacological and non - pharmacological methods have been recommended for the management of hypertension. Their role at the level of LV wall stress and LV contractile state is established in various large scale multicentre study[15].

It is worth to consider the Framingham study which have stated that the most important means of preventing the cardio - vascular complication is to identify and treat hypertension before complication develop. In this point of view, the echocardiography is very useful tool in demonstrating abnormalities in LV performance and supplying additional clue to a newer concept of hypertension as a disease process rather than a manifestation of underlying disease.

6 .Conclusion

The major index of left ventricular systolic function is end systolic stress in hypertension. Hypertensive patients with left ventricular hypertrophy have increased left ventricular wall stress and impaired fractional fibre shortening.In patients with left ventricular hypertrophy the ejection fraction, calculated by Teichholz's formula is a poor measure of left ventricular function. Reduced fractional fibre shortening is an independent predictor of cardiac morbidity. M-mode echocardiography will remain a relatively inexpensive diagnostic tool in assessing the myocardial performance in hypertensive patients for prevention of onset of target organ damage. The main objective of management of hypertensive subject should be, to reduce the afterload and to improve the LV contractile state. Being a pilot study , we want to under take this study on a large scale to standardize the various M- mode parameters among Indian population

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