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RESEARCH ARTICLE

PROFILE OF SOME CARDIAC FUNCTION TESTS IN MALE HYPERTENSIVES

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ABSTRACT

This is a cross sectional study of cardiac function tests (2D ECHO) between normotensive & hypertensive males, done in IGMC Nagpur hypertension clinics. Students''t' test is used to analyse parameters with P value of <0.05 is taken as level of significance. The changes in heart rate are insignificant. Mean values of SBP(systolic blood pressure),DBP(diastolic blood pressure),LVPWT(left ventricular posterior wall thickness), IVST(interventricular septum thickness), LVEDD(left ventricular end diastolic diameter),LVESD(left ventricular end systolic diameter), EDV(end diastolic volume) , ESV(end systolic volume),S.V.(Stroke volume), C.O.(cardiac output)statistically significant increased in all five study groups. There is statistically significant decrease in ejection fraction in all five study groups, but it is within normal limit. Present study shows presence of volume overload in addition to pressure overload (after load) which lead to cardiac failure in hypertensive patients. Present study showed that the ventricular performance was not impaired by hypertrophy as such and when ejection fraction was the only index of ventricular function studied, index remained normal.

INTRODUCTION

Prevalence of hypertension in India is 25% & 10% for urban & rural population respectively[1]. High Blood pressure is 3rdmost important risk factor for attributable burden of disease in south Asia (2010)[2].Clinical hypertension may be defined as that level of blood pressure at which the institution of therapy reduces blood pressure-related morbidity and mortality.Hypertension leads to pressure overload on left ventricle, after load is increased this leads to increased left ventricular systolic pressure, sustained pressure overload leads to concentric hypertrophy[3,4].

Chronic excessive work load on heart as in hypertensive's may lead to systolic heart failure characterized by an impaired inotropic state causing weakened contraction which leads ultimately to a decrease in stroke volume, inadequate ventricular emptying, cardiac dilatation and often increase of ventricular diastolic pressure. In many patients with LVH and dilatation, systolic and diastolic failure co-exists.[5] Diastolic failure is characterized by impaired relaxation of ventricle and leads to an elevation of ventricular diastolic pressure at normal end diastole volume. In patients with both systolic and diastolic failure, ventricle both empties and fills abnormally. There may be cardiac dilatation but the ventricular pressure volume relation is shifted, raising the ventricle pressure at any given volume.[3,4]

Aims And Objectives

- To note down the clinical parameters of study subjects and controls viz. heart rate, systolic B. P. and diastolic B. P.
- To note down the cardiac functions parameters of the controls and study group patients having same age, height, weight.
- To compare the cardiac parameters of controls with hypertensive.
- To denote the difference and locate the statistical significance of it.

METHODS

The present study was carried out in hypertension clinic of IGMC Nagpur. Institutional ethics committee permission taken to undertake study. There are two groups:

a. Study Group:Total No. (i.e.) n = 68 Subjects (only males) in the age group of 45 to 69 years, with stage I hypertension [5,6](S.B.P.>140 & D.B.P.>90) & duration of hypertension of 2 years& above visiting hypertension clinic at IGMC Nagpur were studied. Their detailed history was taken.

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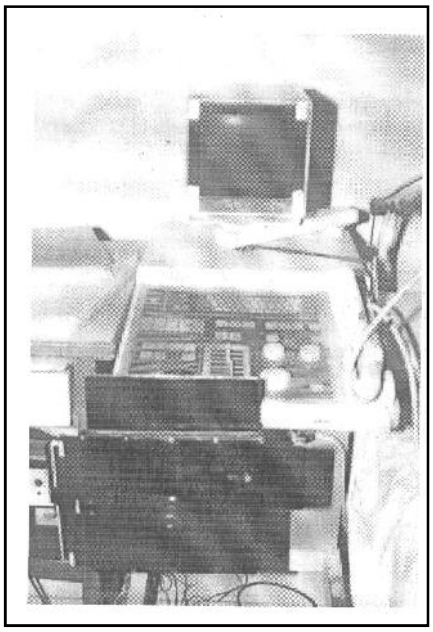
Subjects with other cause of cardiac involvement for eg. Rheumatic heart disease, valvular heart disease, congestive cardiac failure, anemia were excluded from the study.

b. Control Group: n = 57 (only males) for comparison a group of subjects who belonged to the same age group 45-69 years with nearly same anthropometric characteristics were taken. They were belonging to same socio-economic status and ethnic group as that of the study group subjects but with normal blood pressure. They were selected from staff members of IGMC as well as that of general population.



Complete general examination was done, blood pressure and heart rate was recorded. The appointment was give for ECHO.

Method for ECHO

Equipment's used

1. Aloka ECHO camera
2. Model SSD -630 UST
3. 5024N Frequency 3.5 Hz
4. Sonogel

All the echocardiography measurements were taken by an expert. A transducer of frequency 3.5 MHz was used for all the subjects. The ECHO examination was performed in left lateral positions parasternal long axis view was obtained. The transducers were placed at the 3rd or 4th intercostals spaces perpendicular to the chest wall. Following parameters for left ventricular function were taken at the chorda tendina level.[8,9] Following parameters were measured.

1. Left ventricular posterior wall thickness – LVPWT
2. Inter ventricular Septal – thickness – IVST
3. Left ventricular internal diameter — Diastole (LVEDD)
4. Left ventricular internal diameter – ESD– Systole (LVESD)
5. End diastolic volume (EDV-LV)
6. End systolic volume (ESV-LV)
7. Left Ventricular stroke volume – LVSV
8. Left Ventricular ejection fraction – LVEF
9. Cardiac output was calculated by formula C. O. = Heart rate x stroke volume

The subjects of study and control group were divided in the groups according to their age.

Group I	**45 – 49 years**
Group II	50 – 54 years
Group III	55 – 59 years
Group IV	60 – 64 years
Group V	65 – 69 years

In our study the patients were commonly treated with following anti hypertensive drugs. Maximum patients received either of these two drugs or a combination.

1. Atenolol
2. Nifedipine

Few patients also received

1. Enalapril
2. Captropril
3. Diazepam

Mean and standard deviation were calculated and significance of difference was noted with students''t' test at P <= 0.05

RESULTS

1. Significant increasein SBP & DBP in all the five study groups when compared with controls.
2. The changes in heart rate were insignificant.
3. LVP WT in the study group showed a highly significant increase in all five age groups when compared with controls.
4. There is statistically significant increase in mean IVST (CMs) of study group when compared with controls.
5. Mean values of LVEDD of study subjects were increased in all the age groups.
6. Mean values of LVESD of study subjects were increased in all the age groups.
7. EDV is increased in all five study groups and difference is statistically significant.
8. There was statistically significant increase in ESV in all five study groups.
9. Stroke volume increased in all five study groups.
10. There was statistically significant decrease in ejection fraction in all five study groups, ` but it is within normal limit.
11. C. O. also increased in all 5 study groups.

DISCUSSION AND CONCLUSION

Arterial pressure had been considered a disease of the arterioles, elevating the total peripheral resistance in a more or less uniformly distributed fashion among the regional circulatory beds. As a result the cardiac muscle was required to respond to the progressive work load by increasing its mass through the process of left ventricular hypertrophy until congestive heart failure eventually supervened. Impaired

contractility of the myocardium has been demonstrated even before LVH could be detected clinically. Then with clear cut evidence of LVH with resting systemic flow was significantly reduced. Myocardial adaptations to chronic overload as in HT lead to LVH. Four different types of hypertrophies are noticed in mildly hypertensive patients.[7,10]

Also values of LVEDD and LVESD are within normal range though a significant increase in study group is found due to adaptive mechanisms involved during the natural history of hypertension and due to ventricular after load. Responses claimed are:

AGE Gp.	N		DIASTOLIC B.P.		P VALUE	SYSTOLIC B.P.		P VALUE	HEART RATE		P value
	C	S	C	S		C	s		C	S	
I	12	13	81.3±1.7	88.2±6.1	**<0.0001**	118 ±6.12	144±11.48	**<0.0001**	72±0.6	**72**±2.5	**>0.05**
II	12	13	81±1.6	88±5.3	**0.0002**	119±3.7	142.86±11.46	**<0.0001**	73±1.25	**72**±2.3	**>0.05**
III	12	14	80±1.4	87 ±5.2	**0.0003**	121±6.57	137.86±8.6	**<0.0001**	73±1.9	**70**±1.9	**>0.05**
IV	11	14	81±1.5	87.4±2.89	**<0.0001**	125±4.2	142±11.63	**0.0002**	**72**±1.5	**73**±1.5	**>0.05**
V	11	16	81.2±1.6	88.2±5.02	**0.0002**	127±4.036	145.82±11	**<0.0001**	**72**±1.5	**71**±2	**>0.05**

Age GP.	NUMBER		LVPWT		P value	IVST		P value	LVESD		P value	LVEDD		P value
	N(C)	N(S)	C	S		C	S		C	S		C	S	
I	12	13	0.87+ 0.075	1.09+ 0.076	**<0.0001**	0.89+ 0.052	1.22+ 0.059	**<0.0001**	4.68+ 0.208	5.18+ 0.516	**0.0047**	3.29+ 0.144	3.8+ 0.33	**0.0001**
II	12	13	0.85+ 0.09	1.07+ 0.083	**<0.0001**	0.87+ 0.087	1.21+ 0.053	**<0.0001**	4.75+ 0.250	5.09+ 0.318	**0.0071**	3.37+ 0.144	3.74+ 0.227	**0.0001**
III	12	14	0.87+ 0.05	1.19+ 0.047	**0.0001**	0.92+ 0.098	1.24+ 0.134	**<0.0001**	4.77+ 0.297	5.22+ 0.578	**0.0229**	3.37+ 0.174	3.8+ 0.396	**0.0019**
IV	11	14	0.83+ 0.05	1.12+ 0.075	**0.0001**	0.87+ 0.101	1.24+ 0.072	**<0.0001**	4.67+ 0.219	5.13+ 0.554	**0.0164**	3.32+ 0.125	3.82+ 0.312	**0.0001**
V	11	16	0.88+ 0.08	1.08+ 0.075	**0.0001**	0.864+ 0.092	1.19+ 0.054	**<0.0001**	4.66+ 0.224	5.13+ 0.606	**0.0218**	3.33+ 0.127	3.77+ 0.390	**0.0014**

Age GP.	NUMBER		EDV		P VALUE	ESV		P VALUE	SV		P VALUE	EF		P VALUE
	(C)	(S)	(C)	(S)		(C)	(S)		(C)	(S)		(C)	(S)	
I	12	13	103+ 14.16	142+ 37.947	**0.0028**	37+ 4.55	58+ 15.62	**0.0002**	66+ 9.742	84+ 22.6	**0.0181**	64+ 1.03	59.3+ 1.494	**0.0001**
II	12	13	108+ 16.95	134.1+ 27.014	**0.0083**	38 + 5.29	54+ 10.21	**0.0001**	69+ 11.75	80.1+ 17.1	**0.0735**	64+ 1.24	59.7+ 1.489	**0.0001**
III	12	14	109+ 21.04	144.3+ 21.05	**0.0003**	39 + 6.36	59 + 18.26	**0.0011**	70+ 14.72	84.1+ 22.9	**0.0833**	64+ 1.14	60 + 1.946	**0.0001**
IV	11	14	102+ 14.96	144+ 37.10	**0.0019**	37+ 4.98	59+ 16.71	**0.0004**	65+ 10.14	85 + 20.6	**0.0077**	63. + 1.12	59+ 1.740	**0.0001**
V	11	16	102+ 15.29	139+ 42.68	**0.0109**	37.7+ 4.65	57+ 18.76	**0.0028**	64. + 10.68	82.1+ 24.48	**0.0319**	63+ 0.94	59+ 2.442	**0.0001**

1. Disproportionate septal hypertrophy.
2. Concentric hypertrophy.
3. Eccentric dilated LV hypertrophy.
4. Eccentric non-dilated LVH.

Myocardial Hypertrophy is one of the fundamental compensatory mechanisms to increased work load per unit of contractile tissue. In initial stages, in hypertensive, to overcome the increased after load the hypertrophied heart becomes hyper effective. It does mean, the cardiac output is increased, the heart rate is not changed thus the neural effect is minimal. Thus preload is increased. The PWT is increased also as a response to after load stress. This response in terms of hypertrophy is fit as an adaptation to create a hyper effective heart.

The IVST is also increased. The IVS is a major anatomical tissue for creation of kinetic energy required for propulsion of blood from left ventricular system to systemic circulation. Thus after load stress is mechanically transmitted to IVS and thus the thickening of IVS remains as the entity of essential nature.

Due to increase is preload the LVEDD increases. Also LVESD increases due to increase in the size of ventricles.

1. Increase Preload
2. Increase in force of contraction
3. Increase in the tension of ventricular wall.

Since preload is more, thus EDV is increased, it invokes the Frank starling mechanism. Also ESV is increased which may be due to an impaired inotropic state. By operation of all above mechanisms i.e. increased EDV, increased LVEDD, increased PWT, normal and slightly increased IVST, stroke volume is maintained. Ejection fraction serves as a left ventricular ejection phase index which is often found to be normal in patients with LVH. In our study though the ejection fraction was significantly decreased in study group, it was still maintained within normal limits (i.e.) around 60%.

Thus present study shows that the ventricular performance was not impaired by hypertrophy as such and when ejection fraction was the only index of ventricular function studied, index remained normal. Cardiac output is increased which might be due to operation of Frank Starling mechanism to maximum level, sympathetic stimulation and stretch of right arterial wall. It is thus observed that due to increase in blood volume as is evidenced by more cardiac output, increase in ventricular volume and increase in stroke volume, there is increase in

CVP, back pressure and it is responsible for right ventricular loading.

Thus heart is hyper effective but as heart rate and ejection fraction are normal, the hyper effectiveness is due to increase in EDV and stroke volume.

Therefore the early detection of cardiac dysfunction which may warn the physician regarding development of cardiovascular complications like LVF, CHF etc. ECHO investigation should be done at regular intervals. As Cuspidi C *et al* found in their finding importance of 2D echo in risk stratification of hypertensive patients our study also underlines importance of 2D echo in assessment of cardiovascular system in hypertensive patients.[11]

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