Original Research Article

Evaluation of left ventricular diastolic function in hypertensives

C. Venkateshwarlu[1], B. Praveen Kumar[2], Md. Yousuf Khan[3*]

[1]Associate Professor, Department of General Medicine, Government Medical College and Government General Hospital, Nizamabad, Telangana State, India
[2]Associate Professor, Department of General Medicine, Gandhi Medical College, Secunderabad, Telangana State, India
[3]Assistant Professor, Department of General Medicine, Government Medical College and Government General Hospital, Nizamabad, Telangana State, India
[*]Corresponding author email: **dr.yusuf_mohd@rediffmail.com**

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Abstract

Background: Recently much of attention is focused on the diastolic function of the heart. Since the systolic performance of the heart in hypertensive patients has been examined so far, but the study of the diastolic function of heart has emerged as a newer mode of study.
Aim: To study non-invasively the diastolic function of left ventricle in patients with systemic hypertension.
Materials and methods: Twenty patients either admitted or as outpatients in outpatients Department, Gandhi Hospital with isolated systemic hypertension. (HTN) who satisfied inclusion and exclusion criteria were taken up for the study with five age matched individuals who along served as controls assessed by Doppler Echo-cardiography and their findings were compared with five normotensive age matched controls.
Results: Bedside post valsalva maneuver, this test was positive in 7 5% of the test subjects. Of the 25% who had a negative test, 40% had evidence of diastolic dysfunction as assessed by pulsed wave Doppler. Left ventricular hypertrophy was present in 55% of the patient subgroup and all (100%) of these patients had significant diastolic dysfunction. The rest of the 45% of the patients did not have left ventricular hypertrophy. 75% of these non-hypertrophic hypertensives had diastolic dysfunction as seen by PWD. Increased left ventricular mass index has been observed in 85% of the hypertensives but has not correlated statistically with either severity of blood pressure or diastolic filling variables or isovolumic relaxation time. The left atrial size an indirect indicator of LV diastolic function was increased in 50% of the hypertensive subjects when compared with the controls but the statistical

difference was insignificant. LV diastolic function as assessed by PWD mitral flow velocity profile and isometric volume relaxation time was abnormal in 85% of the hypertensive subgroup and was highly significant statistically (P value <0.005). The deceleration time (MS) was similar in both the controls and the hypertensives making it an insignificant parameter for assessing LV diastolic function. The peak filling rate was reduced in the hypertensive subgroup and was of moderate statistical significance.

Conclusion: Doppler Echocardiography, an easily available non invasive technique today, can be utilized for identifying hypertensives with diastolic dysfunction and thus treat this group with specific therapy (Beta Blockers) so as to arrest or reverse the pathological changes produced in left ventricle due to hypertension.

Key words

Doppler Echocardiography, Hypertension, Valsalva maneuver.

Introduction

Recently much of attention is focused on the diastolic function of the heart. Since the systolic performance of the heart in hypertensive patients has been examined so far, but the study of the diastolic function of heart has emerged as a newer mode of study. Diastology implies evaluation of diastolic function of the heart. The normal function of diastole is to allow ventricular filling within physiological rates without elevation of pressure. Although both systolic and diastole are placed differently in the cardiac cycle, they are interrelated to each other. Diastolic dysfunction implies elevation of the left ventricular end - diastolic pressure (LVED) with normal LV end - diastolic volume (LVED) i.e., (60 - 88 M'/M2) and normal ejection fraction (EF). Systolic dysfunction is more common (70%) than diastolic dysfunction 30%. In several diseases like coronary artery disease (CAD) etc., both may co-exist. The systolic dysfunction can be evaluated by 2- D echo but for diastolic dysfunction echo - doppler is mandatory. Congestive heart failure (CHF) is a common and often lethal complication of chronic systemic hypertension. After extensive research, certain factors are believed to have caused the syndrome of CHF. These factors include the effects of ischemia, hypertrophy of heart chambers, changes in the peripheral circulation, alteration in reflex and neurohumoral function, the interdependent function of the right and left ventricles and of signal importance to the clinician - the difference between systolic and diastolic dysfunction of the left ventricle. Diastolic dysfunction of the left ventricle (LV) is defined as an impaired capacity to accept blood or fill without a compensatory increase in left arterial pressure. In its mildest form, diastolic dysfunction may appear as a slow or delayed pattern of relaxation and filling with little or no elevation of LV diastolic pressure. Thus in patients with LV hypertrophy an alteration in diastolic filling can serve as a sensitive indicator of disease. Abnormal diastolic LV function may result in symptoms of congestive heart failure despite a normal systolic contractile state. In some way normal diastolic dysfunction is similar to mitral stenosis; in this case however, impaired filling is due to a reduced effective mitral orifice area. Thus the concept of diastolic dysfunction as a mechanism underlying heart failure is similar to the description of backward failure. Simply stated, systolic dysfunction is the inability of the myofibrils to shorten against a load. Thus the LV loses its ability to eject blood into a high pressure aorta. Diastolic dysfunction implies that the ventricle cannot accept blood at low pressures; ventricular filling is slow, delayed or incomplete unless arterial pressure increases. Consequently pulmonary and / or systemic venous congestion develops, thus the signs and symptoms of pulmonary and / or systemic venous congestion are not necessarily the result of systolic dysfunction instead they are related to alterations in diastolic properties of the LV chamber. These

properties may consist of changes in the passive (or static) diastolic properties of the LV and / or changes in the active processes of relaxation and filling. For most clinical situations, the LV ejection fraction provides a simple, easily interpreted and generally reliable measure of LV systolic performance. In contrast a simple, single measure of LV diastolic function is not available. An ever expanding and confusing array of measures of diastolic function has been derived from invasive and more recently from non - invasive tests. Interest in diastolic properties of the LV and the nature of relaxation had slowly evolved for almost many centuries. Then, in the past two decades, owing in part to the development of methods to measure intra cardiac pressure and volume, research in this area has exhibited an explosive growth. During the 1970's investigators studied the basic mechanism that under lie diastolic dysfunction. These studies emphasized the physical properties of the fully relaxed ventricle (i.e., the process of relaxation) as well as factors extrinsic to the LV (pericardium, RV etc.). In the 1980's numerous articles reflecting the clinical importance of diastolic dysfunction appeared in the literature [1, 2]. Studies reported that more than 1/3rd of patients referred to their nuclear cardilolgylab for evaluation of CHF had normal systolic function, a high prevalence of diastolic dysfunction was found in patients with hypertensive heart disease. Also recurrent pulmonary edema with preserved systolic function has been reported in elderly patients with IHD and a syndrome of hypertensive hypertrophic cardiomyopathy [3]. In this later condition Cittadini A, et al. [4] documented abnormal diastolic function in the presence of excessive LV emptying (high normal ejection fraction) most important, however was their observation that Beta adrenergic receptor antagonists or calcium channel blocking agents generally produced symptomatic improvement whereas vasodilators not only were ineffective but on occasion produced intolerable hypotension and rarely death. Thus there is no longer any doubt that the clinical syndrome of CHF should be evaluated and treatment designed with an application of differences between systolic and diastolic dysfunction [5]. A complete evaluation of the diastolic properties of the LV requires calculation of LV chamber and myocardial stiffness, LV pressure and volume relationships etc. This is a formidable task requiring massive catheterization techniques and therefore a complete assessment of cardiac diastole is rarely made in clinical practice. Also for serial evaluation of Progression of LV diastolic dysfunction invasive cardiac catheterization seems superfluous. Hence a clinical assessment of diastole requires less invasive methods such as M. Mode Echo; 2D - Echo and Doppler Echocardiography for routine or serial evaluation [6]. In some disease states especially HTN diastolic function precedes the onset of systolic dysfunction.

Materials and methods

Patients presenting to Outpatient Department of Gandhi Hospital and inpatients with systemic hypertension were taken up for the study.

Inclusion criteria

Age Range of 20 - 65 years, Patients with systemic hypertension - with systolic blood pressure > 160 mm Hg and diastolic blood pressure of >95 mm Hg were enrolled. Informed consent was obtained from all patients. Newly diagnosed or diagnosed but untreated hypertension or treated but inadequately controlled hypertensive patients were enrolled.

Exclusion criteria

The exclusion criteria were the following: Coronary artery disease either by history or ECG or segmental wall motion abnormalities. Heart rate < 50 beats / min or > 100 beats / minutes. Arrthythmias such as first degree heart block, AF, Atrial flutter multiple ectopics. Insulin dependant diabetes mellitus. Chronic obstructive lung disease and related disorders like bronchial asthma. Valvular heart disease by physical examination or echocardiography. Pericardial disease by 2D - echo. Hypertrophic cardiomyopathy by 2D - echo.

Methods of study

Patients presenting with systemic hypertension fulfilling the inclusion and exclusion criteria were assessed by a detailed history, physical examination as outlined in the proforma. Appropriate baseline investigations such as biochemical and hematological were done on enrolling them into the study. Other necessary investigations such as ECG, X - ray chest, urinalysis was also done. Normotensive patients of similar mean age without hypertensive heart disease and fulfilling exclusion criteria were also studied as normal control subjects.

Method of Echo - Doppler evaluation

2D - Echo and pulsed wave Doppler was done with 0ewlett Packard. In the 2D Echo evaluation of patients with hypertension, a short axis and long axis view of the heart was obtained with the patient in semi left lateral position. An apical four chamber view was also seen- This was done to rule out any subclinical valvular heart disease, pericardial disease especially constrictive pericarditis and hypertrophic cardiomyopathy. In the short axis view with the cursor aligned just distal to the tips of the mitral valve, an M-Mode echocardiogram was obtained to take the various dimensions such as left ventricular dimensions in diastole and systole, septal thickness and posterior wall thickness. Next, a colour Doppler evaluation was done to detect any subtle regurgitant lesions. From an apical four chamber view the pulsed wave Doppler cursor was aligned parallel to the stream of inflow of blood from left atrium to left ventricle. A site was chosen along the cursor for sampling the mitral flow velocity profile such that the sample volume was taken just internal to the tips of the mitral leaflets. A pulsed wave Doppler tracing was obtained and the following parameters were measured. E wave (Early diastolic flow velocity) cm/ s. A wave (late diastole flow velocity) cm/ s. E/ A Ratio .Peak filling rate (Ex Mitral valve area) .Normalized peak filling rate (PFR/ LV and diastolic volume) .Isometric volume Relaxation time (ms), Deceleration time (ms). Total Diastolic flow duration. The mitral valve area was calculated by pressure half time. As per the recommendations of American society of Echocardiography (I0), the left ventricular mass was calculated by the formula : Left ventricular mass = 0.80 x [1.04 x (LVEDD-EVS-i-PW)^3 - (LVEDD)^3] where LVEDD = LV end diastolic dimension (cm) VS = Ventricular septum (cm) PW = Posterior wall thickness (cm).

Results

Twenty patients either admitted or as outpatients in outpatients Department, Gandhi Hospital with isolated systemic hypertension. (HTN) who satisfied inclusion and exclusion criteria were taken up for the study with five age matched individuals who along served as controls.

The age of the patients ranged from 35 years to 65 years with a mean of 50 + 8.7 years. The age range of control volunteers ranged from 37 - 60 years with a mean of 47 ± 10 years. Of the twenty patients studied seventeen were male and three female. Males and females had no significant differences (**Table – 1**).

The control normotensive subjects had a systolic blood pressure with a mean of 117.20 + 6.42 mm of Hg whereas the hypertensive subjects had a mean Systolic blood pressure of 178 + 16.7 mm of Hg and a diastolic pressure with a mean of 107 + 7.9 mm of Hg. The heart rate in both study groups was well within the limits of inclusion criteria. The body surface area (m^2) in controls was 1.67 + 0.11 m2 versus 1.65 0.12 m^2 in hypertensive. The controls and the test individuals were subjected to the bedside 10 second valsalva maneuvre. The response of korotkoff sounds at systolic blood pressure after 10 seconds of valsalva maneuvre. Disappearance of korotkoff sounds was taken as a normal response the test being negative where as the persistance of korotkoff sounds was taken as an abnormal response - the test being positive indicating elevated left atrial pressures. 15 out of 20 Patients (75%) had a positive test. All the 5 control normotensives had a negative test. 35% of patients had a fourth heart sound. These same 35% of patients had their cardiac apical impulse

outside midclavicular line with prominent LV impulse. One patient had a third heart sound with accentuated aortic component, normal CT ratio with pulmonary basal rales. One patient had a short systolic murmur at the base of the heart over 2nd left inter costal space. About 50% cardiomegaly defined of the test subjects had as CT ratio greater than 0.55. Electrocardiogram revealed left ventricular hypertrophy in 65% of patients. Of these 20% had left atrial overload and 15% had strain pattern (**Table – 2**).

Table - 1: Patient characteristics.

Age Range (Years)	Mean	Sex	Number of Patients
35-65	50±8.7 years	Males	17
37-60	47±10 years	Females	3

Table - 2: Subject characteristics controls and cases.

Details	Controls	Hypertensive	P Value
No of subjects	5	20	
Age (years)	47.20 ± 10.43	49.50 ± 8.70	NS
Body Surface area (m^2)	1.67 ± 0.11	1.65 ± 0.12	NS
Systolic Blood Pressure (mm of Hg)	117.20 + 8.32	177.05 + 16.71	***
Diastolic Blood Pressure (mm of Hg)	76.80 + 6.42	107.05 + 7.90	***
Heart Rate (beats/ min)	74.82 ± 9.34	82.85 + 8.37	***
Body mass Index	2.43 ± 0.23	2.40 + 0.25	NS

1. Values given are Mean ± Standard deviation
2. NS .7.-- Not significant
3. *** $p < 0.005$ - Highly significant
4. * $P < 0.05$ - Significant.

Echocardiographic evaluation by 2D-Echo and M-, Mode revealed the following. The ventricular septal wall thickness in normotensives had a mean of 0.81 0.07 cm versus 1.15 + 0.24 in the hypertensive group. The left ventricular posterior wall thickness in control subjects had a mean of 0.81 + 0.08 cm versus 1.13 + 0.22 cm in the hypertensive subgroup. 55% of the patients had concentric LV hypertrophy defined by posterior inter ventricular septal thickness > 1.1 cm. The left ventricular end diastolic dimension (LVEDD) in the test population was 4.53 + 0.49 cm and the left ventricular end systolic dimension was 2.97 ± 0.36 cm. The left ventricular mass index (LVMI) in the control subgroup was 64.66 + 10.8g/ m2 versus 114.19 + 38.09 g/rr12 in the hypertensives. 85% of the patients had increased LVMI. Acute off point of 90 g/m2 was decided on to recognise increased LVM and thus LVH. The systolic function as assessed by fractional shortening and ejection fraction in controls and hypertensive had the following values. Fractional shortening (%) 38.15 + 1.64 versus 34.20 + 6.79.Ejection fraction (%) 64.80 + 2.93 versus 58.46 + 11.73. The left arterial size was increased in majority of the participants of the test group but aortic size did not differ much between the two groups. The mitral valve area did not differ much between -.47 + 0.46 sq. cm. in control the two groups 4 versus 4.31 + 0.47 sq. cm in the hypertensive subgroup (**Table – 3**).

The pulsed wave Doppler echo findings were as follows: The early peak velocity y (E) ranged from 64 to 108 on/sec• in controls with a mean of 80.84 + 16.8 cm/sec. whereas the range of E in test group ranged from 36 to 89 cm/sec with a

The late atrial velocity (A) in the hypertensive ranged group from 50.8 to 98 cm/sec. with a mean of 67.67 + + 16.59 cm/sec. When compared with a mean of 38.56+ 14.32 cm/sec. in controls. The Early Peak velocity to late Arterial velocity ratio E/A revealed a range from 1.4 to 2.9 with a mean of 2.23 + 0.53 in the control cases whereas the E/A ratio in hypertensive group ranged from .7 to 1.28 with a mean of 0.96 + 0.21. The Isometric volume relaxation time (IVRT) in the control group ranged from 71 to 89 ms with a mean of 76.40 + 8.17 ms whereas in hypertensive group the range was from 90 to 160 ms with a mean of 122.95 + 20.21 milliseconds. The deceleration time in the control volunteers had a mean of 159.20 ± 35.85 ms, 157.15 + 38.15 ms versus a value of in the hypertensive cases. The peak filling rate (E x Mitral valve area) in 6. The control volunteers had a mean of 365.62 + 109.24 74.89 + 79.28 was found in the test whereas a value of 2 subgroup (**Table – 4**).

Table - 3: 2D - Echo & M-Mode variables - controls and cases.

Parameter Value	Controls	Hypertensives	P-value
No of subjects	5	20	
Venticular Septal thickness (cm)	0.81 + 0.07	1.15 + 0.22	***
Posterior Wall thickness (cm)	0.81 + 0.08	1.13 ± 0.22	***
LV End Diastolic Dimension (cm)	4.31 + 0.58	4.53 + 0.49	NS
LV End Systolic Dimension (cm)	2.66 ± 0.29	2.97 + 0.36	NS
Fractional Shortening%	38.15 ± 1.64	34.20 + 6.79	NS
Ejection fraction %	64.80 ± 2.93	58.46 + 11.73	NS
LV Mass Index g/m^2	64.66 ± 10.81	114.19 + 38.09	***
Aorta (root size) (cm)	2.68 ± 0.22	2.09 + 0.42	NS
Left Atrial size (cm)	2.80 + 0.29	3.16 + 0.45	NS
Mitral Valve area (cm)	4.47 + 0.46	4.31 + 0.47	NS
Body mass Index g/m2.	2.43 + 0.23	2.40 + 0.25	NS

1. Values given are mean ± standard deviation.
2. NS = Not significant.
3. *** $p < 0.005$ = highly significant.

Discussion

The concept of diastolic clinical cardiology dysfunction in is relatively new. Before 1981 diastolic function was assessed by invasive procedures such as cardiac catheterization. Since the introduction of non-invasive methods such as radionuclide ventriculogram and Doppler echocardiography, these techniques have become the modalities of choice for assessment of LV diastolic function. These have the advantage of ease of performance and repeatability. Diastolic dysfunction can be identified in most patients with ischemic heart disease, hypertension, hypertrophic cardiomyopathy and pericardial disease.

Presence of ischemic heart disease, hypertrophic cardiomyopathy and pericardial disease by 2 D Echo or other parameters such as ECG for IHD excluded the patient from the study. An attempt was made to study non-invasively by pulsed wave Doppler the diastolic function of the heart in hypertensives only. About shortness of III with ventricular interestingly function of half of the patients had exertional breath in the range of grade II to grade 3 patients also giving a history of left failure following severe hypertension. All these patients had a normal systolic the heart as assessed by fractional shortening and ejection fraction normally presence of exertional dyspnoea implies an impaired or poor LV systolic function. The presence of exertional

SOB in the presence of normal systolic function could be explained by diastolic dysfunction. Diastolic dysfunction of LV as already defined 1 is an impaired capacity to accept or fill blood without a compensatory increase in left atrial pressure. A rise in LV filling pressures because of diastolic dysfunction increases the left atrial pressure and this in turn being transmitted to the pulmonary circulation producing a congested state and the symptom of dyspnoea. Accordingly a normal systolic LV function does not rule out the possibility of a cardiac cause of dyspnoea. Easy fatiguability was the next common cardiac symptom in the patient subgroup after exertional dyspnoea. Now again easy fatiguability usually indicates a reduced cardiac output commmensurate with the body needs thus implying a poor or impaired contractile state (systolic function) of the heart. Depression of systolic contractile state of the LVmay result in clinical manifestation of limited cardiac output. Regardless of the systolic contractile state the heart can pump only the blood it receives, this diastolic filling of the LV output. In the presence of LV diastolic dysfunction LV is a primary determinant of cardiac output. In the presence of LV diastolic dysfunction LV filling is output symptom function. It can thus (dyspnoea) impaired and hence a In decreased cardiac our stud of easy fatigue, all the patients with this ability. Accordingly LV had a normal systolic produce diastolic dysfunction symptoms of both backward failure or forward failure (easy fatiguability) as can an impaired systolic function. The bedside 10 sec. post valsalva maneuvre test was positive in 75 percent of the P indicating an elevated left patients atrial filling the pressures hall mark of diastolic dysfunction in the presence of n. normal systolic function. All these patients had LV diastolic dysfunction as assessed by pulsed wave Doppler. About 25% of the hypertensives had a negative test as indicated by disappearance of Korotkoff sounds after 10 sec. of valsalva maneuver. Of this group (25%), 40% had diastolic dysfunction as assessed by PWD. The 10 sec. post valsalva bedside test is a simple test and a very good guide for assessing clinically LV diastolic dysfunction as it is correlating very significantly with the PWD estimate of diastolic function. The cardiac adaptation to combined systolic and diastolic hypertension is an increase in wall thickness leading to concentric left ventricular hypertrophy. This adaptation occurs to maintain Cardiac output in the setting of increased after load. It is a well known fact that LVH is a significant risk factor for cardiovascular myocardiac morbidity and mortality mainly ischemia and sudden death. In an epidemiological sense, HTN is an important risk factor for CHF. Date from the Framingham study have indicated that patients with hypertension have a six fold increase in the likelihood of CHF. In the present study LVH (defined by posterior and interventricular septal thickness > 1.1 cm) was present in 55% of patients and none had LVH in the normotensive group. The prevalence as reported in other studies ranged from 23% to 48% in hypertensive patients and 0% to 10% in normal subjects (10a).

Significance of LVH

LVH delays or slows that active relaxation of the myocardium because of increased calcium load in hypertrophied ventricle leading to slow dissociation of actin and myosin cross bridges during myocardial relaxation [6]. LVH also alters the chamber compliance in addition to the slowing of myocardial relaxation - main parameters affecting LV filling and thus this results in an alteration of the two diastolic function. mitral flow velocity profile in the form of increased 'A peak and decreased ESA ratio - the major indicators of LV diastolic function. The slowed active myocardial relaxation accounts for the prolonged isometric volume relaxation time another sensitive parameter for assessing diastolic function. A significant number (30%) of patients in the current study had evidence of diastolic dysfunction (as assessed by PWD) in the absence of LVH indicating that LVH is but one factor of the many other factors that are known to affect the diastolic properties of the left ventricle.

Table - 4: Pulsed wave Doppler variables - controls and cases.

Parameter	Controls	Hypertensives	P Value
No of subjects	5	20	
Peak Early Velocity 'E' cm/s	80.84 + 16.80	63.68 + 15.87	*
Peak Atrial Velocity 'A' cm/s	38.56 + 14.32	67.67 + 16.59	***
E/A Ratio	2.23 ± 0.53	0.96 ± 0.21	***
Isometric Volume Relaxation time (ms)	76.40 ± 8.17	122.95 + 20.21	***
Decelaration Time(ms)	159.20 ± 35.85	157.15 + 38.15	NS
Peak filling rate	365.62 ± 109.24	274.89 + 79.28	*

(1. Values given are mean ± standard deviation, 2. NS = Not significant, 3 *** = P < 0.005 - Highly significant, 4 * = P < 0.05 – Significant)

The left ventricular mass and mass index (LVMI) as derived following the recommendation of the American Society of Echocardiography (35) have been found in the current study to be statistically highly significant (P value <0.005) - mean value of 64.66 + 10.81 g/m^2 versus 114.19 + 38.09 g/m^2 in the hypertensive cases. But there was no significant correlation between LV Mass Index and the Diastolic or systolic blood pressure in the cases. However when the data from normotensive subjects and 1 in participants were pooled there was a weak but statistically significant correlation for Diastolic, Bp (r = 0.5074). A similar result has been obtained in a study of isolated systolic hypertension in the elderly subjects by Anthony et al. (10a). There was no statistical correlation between LVMI and body Mass index. LVMI also did not correlate\with any Doppler variable of LV filling. The systolic function as assessed by fractional shortening and ejection fraction was well preserved in the hypertensive group. Statistically there was no significant difference between controls and the hypertensive subgroup. But almost 85% of the hypertensive group had a diastolic dysfunction as shown by PWD. Systolic decompensation probably occurs late in the course of the disease or in very severe hypertension cases. This finding has been endorsed by Isao Inouye, et al. (10b) who in their study of assessment of diastolic function in HTN patients found systolic dysfunction to be uncommon. In contrast, diastolic dysfunction as evidenced by abnormalities of diastolic filling was found to be the rule. The left atrial size is an indirect indicator of the LV diastolic function. In the presence of LV diastolic dysfunction elevation of left ventricular filling pressure rises the left atrial mean pressure (> 12 mm Hg). Thus producing LA enlargement. In the present study though LA size was increased in the when compared with the controls the statistically insignificant. Anthony, et al., reported a statistically significant increase in LA size in the hypertensive group. The size of the aortic root (cm) and the rnitral valve area was similar in both the controls and hypertensives as per other studies [4]. Concurring with similar findings in LV diastolic function as measured by PWD mitral flow velocity profile was found to be affected in 85% of the hypertensive subgroup studied. In the current study, diastolic filling was altered in patients with systemic HTN, with an increase in peak atrial velocity 'A' and a reduced E/A ratio. There was also a decrease in mean peak early velocity when compared with the normotensives.

The difference in the early peak velocity (E) in the control group (80.84 16.86 cm/sec.) versus (63.68 ± 15.87 cm/sec.) in the hypertensive group was statistically significant (P value < 0.005 - highly significant). Because of alterations in the LV diastolic properties viz., myocardial relaxation, resistance to there is increased compliance, etc. filling of LV producing a shift in the pattern of LV fillip from early diastole to late diastole. The late g atrial velocity control

(A) in the versus group 67.67 ± 16.59 cm/sec. being 38.56 ± 14.32 a difference of high cm/sec. in the hypertensive group statistical significance. Thus the decrease in E filling velocity and an augmentation of A filling Enhanced may provide velocity. An to indicate a subgroup E/A ratio. measure can be velocityreversusthe normal E/A atrial filling due to elevated LA pressure an additional factor for increased atrial E/A ratio of >1 is taken by many authors normal diastolic function. In our patient ratio was less than 1 in 60%. The isometric volume relaxation time is a of ventricular relaxation. Abnormal relaxation indicated by the presence of prolonged IVRT. It does not provide information of LV filling and therefore is of some what limited volume when assessed as a sole index of diastolic function [7]. Nevertheless, Rick N.Nakamura, et al. [8] from Mayoclinic reported "IVRT is probably the most sensitive measurement as it is the first to become abnormal" and has with findings of cardiac cath. In our there was high statistical correlated well current study significance in the difference between IVRT (ms) of the control group versus the hypertensive group. The deceleration time (the rate of decay of LV flow profile from the peak E to the point of beginning of diastasis) between the two groups- controls [9, 10] (mean of 159.20 + 35.85) versus (mean of 157.15 + 38.15) in hypertensives, was Moreover there was of no statistical significance wide overlap of individual values of the hypertensive and normotensive subjects. The peak filling rate was also found to be of statistical significance between the groups, the peak filling rate being reduced in the hypertensive subgroup.

Conclusion

The major goal of the study was to assess the LV diastolic function noninvasively by pulsed wave Doppler. correlated measures The assessment by this technique has significantly with other standard invasive such as cardiac catheterization. Doppler Echocardiography, an easily available non invasive technique today, can be utilised for identifying hypertensives with diastolic dysfunction and thus treat this group with specific therapy (Beta Blockers) so as to arrest or reverse the pathological changes produced in left ventricle due to hypertension.

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