LEFT VENTRICULAR PUMP FUNCTION IN ESSENTIAL HYPERTENSION

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ABSTRACT

Forty six hypertensive patients and forty normotensive controls were assessed echocardiographically for left ventricular (LV) stroke volume (SV), cardiac output (CO), and heart rate (HR). Severe hypertensives showed significant decrease in heart rate than the mild hypertensives. Similarly, severe hypertensives showed significant decrease in stroke volume and cardiac output as compared to moderate hypertensives. Highly significant decrease was seen when compared to normotensive controls and mild hypertensives. However, mild hypertensives showed non-significant increase in stroke volume and cardiac output than controls.

INTRODUCTION

Role of hypertension in the causation of various cardiovascular complications is now well established. various studies conducted in Pakistan and abroad have shown that hypertension is a significant public health problem[1,2]. essential hypertension is considered to be one of the most common cause of cardiac failure. Left ventricle is of paramount importance due to its pumping capability, but the raised systemic blood pressure badly affects this function. Therefore, multi-directional and thorough information about the left ventricular function at appropriate time is considered imperative in assessing progress of cardiac lesions[33]. Initially there is a short period of left ventricular hypertension, followed by a prolonged period of stable hypertension. Normal resting cardiac output is maintained in these patients until left ventricular hypertrophy (LVH) is demonstrated[4]. The importance of systolic and diastolic properties of the left ventricle in patients with systemic arterial hypertension have been well described. The abnormalities in both of these functions contribute significantly to signs and symptoms in individuals with a variety of cardiac disorders[5].

Pressure load exerts a chronic mechanical burden on the left ventricle causing an increase in the thickness of its wall, the mechanism of this important adaptation is unknown[6]. Although heart rate is slightly increased, but stroke volume and cardiac output are reduced in patients with essential hypertension[7].

Although the function of left ventricular myocardium was difficult to be assessed, intensive investigations were carried out in clinical laboratories during the past few years[8]. Invasive methods like cardiac catheterization and angiography lacked reliability and easy performance[9], thus echocardiography being non-invasive, more sensitively elaborate, and easily repeatable than ECG and X-Ray chest, paramount importance in measuring left ventricular wall and septal thickness and motion alongwith related pump function[10]. This study was designed to assess the left ventricular pump function in patients with controlled and uncontrolled essential hypertension and to provide a comparison with normotensive controls.

MATERIALS AND METHODS

Forty, age and sex matched normotensive controls and forty-six patients of essential hypertension (age 20-70 years) were selected for this study by systematic randomization from the Hypertension Clinic of Mayo Hospital, Lahore.

Patients suffering from cardiac failure or any valvular and congenital anomaly were excluded from the study. The hypertensives were divided into:

GROUP I:
(Controlled Hypertensives)
Twenty labelled cases of essential hypertension whose diastolic blood pressure was below 95 mmHg and was controlled by medicaion.

GROUP II:
(Uncontrolled hypertensives)
Twenty six cases of uncontrolled essential hypertension who were either not on medication or on inadequate medication.

Uncontrolled hypertensives (Group II) were further sub-grouped according to the following diastolic blood pressure level[11]

(a) Mild hypertensives
91-104 mmHg (10 patients)
(b) Moderate hypertensives
105-115 mmHg (10 patients)
(c) Severe hypertensives
116-mmHg and above (6 patients)

Mercury sphygmomanometer with 12.5 cm cuff was used to record blood pressure in each individual in setting position after 15 minutes rest. Stroke volume and cardiac output were evaluated in all the subjects by echocardiography (Toshiba, SDS/20 1980). Heart rate was determined by counting the number of beats per minute. Student's "t" test was applied for statistical analysis of results.

RESULTS

The mean % SD values of heart rate (HR) of all subjects are shown in Table 1.

Group I (Controlled Hypertensives) shows a slight non-significant rise in heart rate (73.02 beats per minute) as compared to the controls (71.56). Although there is a tendency towards increased HR in mild hypertensives (80.45), but the increase is statistically not significant when compared with controls. Contrary to this, moderate hypertensives (68.13) and severe hypertensives (62.58) show a non-significant ($P > 0.05$) decrease in heart rate when compared to the normotensive controls. However, the heart rate in severe hypertensives registered a significant decrease when compared to the mild hypertensives. Both controlled and uncontrolled hypertensive groups did not show any significant change in heart rate.

Table 1. Comparison of Heart Rate (H R) in Normotensive and Hypertensive Groups

Groups	H R (Beats/min Mean % S D	P Value
Normotensives n = 40	71.56 % 8.69	P > 0.05
Controlled hypertensives n = 20	73.02 % 6.70	(N S)
Normotensives n = 40	71.56 % 8.69	P > 0.05
Mild hypertensives n = 10	80.45 % 7.89	(N S)
Normotensives n = 40	71.56 % 8.69	P > 0.05
Moderate hypertensives n = 10	68.13 % 5.09	(N S)
Normotensives n = 40	71.56 % 8.69	P > 0.05
Severe hypertensives n = 6	62.58 % 6.77	(N S)
Mild hypertensives n = 10	80.45 % 7.89	P < 0.05
Severe hypertensives n = 6	62.58 % 6.77	(S)
Moderate hypertensives n = 10	68.13 % 5.09	P > 0.05
Severe hypertensives n = 6	62.58 % 6.77	(N S)

S = Statistically significant ($P < 0.05$)
N S = Non significant ($P > 0.05$)

Table 2 shows mean % SD values of stroke volume (SV) and cardiac output (CO) in all the subjects. Controlled hypertensives did not show any significant difference in SV (68.65 ± 17.19 ml) and CO (5.09 ± 1.19 L) as compared to normotensive controls (SV = 70.22 ± 14.12 ml and CO = 5.01 ± 0.97 L). Mild hypertensives showed slight rise in SV (77.00 ± 19.16 ml) and CO (5.83 ± 1.32 L) but it was statistically non-significant as compared to controls. Similarly, moderate hypertensives also did not show any significant difference in both SV (66.10 % 19.37 ml) and CO (5.20 % 1.32 L). However, severe hypertensives showed a significant decrease in both SV (48.83 % 13.55 ml) and CO (4.30 % 1.81 L).

Table 2. Comparison of Stroke Volume (S V) and Cardiac Output (CO) in Normotensive and Hypertensive Groups

Groups	S V (ml) Mean % S D	P Value	C O (L) Mean % S D	P Value
Normotensives n = 40	70.22 % 14.12	P > 0.05	5.01 % 0.97	P > 0.05
Controlled hypertensives n = 20	68.65 % 17.19	(N S)	5.09 % 1.19	(N S)
Normotensives n = 40	70.22 % 14.12	P < 0.05	5.01 % 0.97	P<0.05
Mild hypertensives n = 10	77.00 % 19.16	(N S)	5.83 % 1.32	(N S)
Normotensives n = 40	70.22 % 14.12	P < 0.05	5.01 % 0.97	P > 0.05
Moderate hypertensives n = 10	66.10 % 19.37	(N S)	5.20 % 1.32	(N S)
Normotensives n = 40	70.22 % 14.12	P < 0.01	5.01 % 0.97	P < 0.05
Severe hypertensives n = 6	48.83 % 13.55	(H S)	4.30 % 81	(S)
Mild hypertensives n = 10	77.00 % 19.16	P < 0.05	5.83 % 1.32	P < 0.05
Severe hypertensives n = 6	48.83 % 13.55	(S)	4.30 % 1.81	(S)
Moderate hypertensives n = 10	66.10 % 19.37	P < 0.05	P < 0.05	
Severe hypertensiving n = 6	48.83 % 13.55	(S)	4.30 % 1.81	(S)

S = Statistically significant ($P < 0.05$)
H S = High significant ($P < 0.01$)
N S = Non significant ($P > 0.05$)

Comparison of SV and CO values of mild and moderate hypertensive groups with severe hypertensive group values show a statistically significant ($P < 0.05$) decrease in the latter group. Severe hypertensives also show a significant decrease in SV and CO as compared to controlled hypertensives.

DISCUSSION

In this study we observed significant decrease in stroke volume (48.83 % 1.55 ml) and cardiac output (4.30 % 1.81 L) of severe hypertensive patients than those of normotensives (SV = 70.22 % 14.12 ml and CO = 5.01 % 0.97 L) and controlled hypertensives (SV = 68.65 % 17.19 ml; CO = 5.09 % 1.19 L). But mild hypertensives showed non-significant increase in both the parameters (SV and CO). Devereux et al[12] reported a high cardiac output and relatively normal peripheral resistance in the early stages of essential hypertension and low cardiac output in moderate and severe hypertensives. They suggested that increased left ventricular chamber stiffness due to increased wall thickness might impair diastolic filling thus causing a secondary reduction in stroke volume and cardiac output. In early hypertension, increased stroke volume and cardiac output could be due to venoconstriction leading to increased venous return[4] Diastolic dysfunction could be the earliest sign of hypertensive cardiomyopathy prior to left ventricular hypertrophy[13]. Our results are in agreement with Nunez et al[14] who suggested decreased and delayed diastolic filling of left ventricle in most of the severe hypertensives with left ventricular hypertrophy resulting in decreased stroke volume and cardiac output. Therefore, it may be beneficial to assess the left ventricular diastolic functions in detail to detect any impairment.

Study of the heart rate in all the groups showed slight non-significant ($P > 0.05$) increase in heart rate in mild hypertensives (80.45 % 7.89) compared to all other groups. However, statistically significant ($P < 0.05$) fall in HR was observed in severe hypertensives (62.58 % 6.77) as compared to mild hypertensives. This could be attributed to sympathetic over-stimulation in the early phases of essential hypertension. Moreover, some naturally occuring humoral agents like catecholamines, angiotensin II, increased Ca^{++} and vasopressin may also participate in increasing the inotropic and

chronotropic function of heart in the initial phase of disease to overcome the ventricular after load[4,15]. However, the decrease or lack of these effects with progression of disease could lead to a decrease in HR as seen in severe hypertensives. It has also been reported that left ventricular wall motion is also decreased highly significantly in severe hyertensives[11,16].

It is thus concluded that moderate and severe hypertensives indicate hypofunction in their ability to pump adequate quantity of blood resulting in low stroke volume and cardiac output. It is suggested that periodic echocardiographic evaluation of left ventricular functions in essential hypertensives should be carried out to detect any deterioration.

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