Left Ventricular and Left Atrial Function in Patients with Borderline and Established Hypertension

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To evaluate left ventricular (LV) and left atrial (LA) function in patients with borderline and established hypertension, 7 indexes of LV systolic function (cardiac index, end-diastolic volume index, ejection fraction, LV network, mean VCF, Vmax and peak dp/dt), 7 indexes of LV diastolic properties (specific compliance, LV mass index, LV wall thickness, stiffness constant, time constant T, end-diastolic pressure and peak negative dp/dt) and 3 indexes of LA function (volume index, chamber stiffness constant and muscle stiffness constant), all obtained from the data of cardiac catheterization and cineangiogram, were analyzed in patients with normotension (N, n = 12), borderline hypertension (BH, n = 13) and established hypertension (EH, n = 17). LV systolic function was not impaired in the BH and EH groups. In the BH group, LV diastolic function was not affected, but LV wall thickness and stiffness constant were significantly greater in the EH group than in the N group. There was no significant difference in LA volume index among the 3 groups, but a significant increase of LA chamber stiffness constant was found only in the EH group as compared to the values of the N group. On the other hand, LA muscle stiffness constant was significantly greater in the BH group than in the N group, and significantly greater in the EH group than in the BH group.

From these results it seemed that LV systolic and diastolic functions were not impaired in patients with borderline hypertension, and that LV systolic function was not affected, but LV diastolic function was possibly impaired in patients with established hypertension. Moreover, it was suggested that LA compliance was impaired already not only in patients with established hypertension, but also in patients with borderline hypertension.

Essential hypertension is the most frequent form of chronic left ventricular (LV) pressure overload in man. Hypertensive hearts are considered to compensate for increased chronic afterload in accordance with Frank-Starling's law, an increase in contractility by the regulation of neuro-humoral factors and the left ventricular hypertrophy. Left ventricular hypertrophy is the most prominent of the compensation mechanisms. Thus, evaluations of cardiac function in hypertensive hearts are very important.

There have been relatively few reports on the left ventricular function in hypertensive hearts, in which LV pressure and left ventriculogram were analyzed. Most of these reports indicate that the cardiac consequences of hypertension
TABLE I SEX, AGE AND ARTERIAL BLOOD PRESSURE IN PATIENTS WITH NORMOTENSION (N), BORDERLINE HYPERTENSION (BH) AND ESTABLISHED HYPERTENSION (EH)

<table>
<thead>
<tr>
<th>Group</th>
<th>n (M, F)</th>
<th>Age (years)</th>
<th>Arterial blood pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Systolic</td>
</tr>
<tr>
<td>N</td>
<td>12 (9, 3)</td>
<td>47 ± 2.9</td>
<td>124 ± 2.6</td>
</tr>
<tr>
<td>BH</td>
<td>13 (9, 4)</td>
<td>49 ± 2.3</td>
<td>149 ± 1.6***</td>
</tr>
<tr>
<td>EH</td>
<td>17 (11, 6)</td>
<td>53 ± 1.7</td>
<td>178 ± 6.7***</td>
</tr>
</tbody>
</table>

Results are expressed as mean ± SEM.
*** = p < 0.001 as compared to values of the N group.
++ = p < 0.01 and +++ = p < 0.001 as compared to values of the BH group.

depend on the degree of myocardial hypertrophy and the progression of coronary arterial sclerosis. LV systolic function was presented to be impaired by a few of these reports and to be unaffected by the others. Thus, evaluations of LV systolic function in hypertensive hearts have not been definitely established.

As diastolic properties have been reported to be affected by LV hypertrophy, hypertensive hearts with LV hypertrophy are considered to have a decreased diastolic function. In fact, there have been many reports on the hypertrophy of hypertensive hearts. However, estimations of diastolic function in hypertensive hearts without LV hypertrophy have not been established definitely.

Furthermore, there have been very few reports on the LV function in patients with borderline hypertension. Although echocardiographic or hemodynamic studies on a borderline hypertensive circulatory state have been done, evaluations of their LV mechanical function have not been established definitely.

Possibilities of a reduction of left atrial (LA) function in hypertensive hearts, have been reported from the point of view of P waves on an electrocardiogram and an echocardiogram. Dreslinski et al. have suggested a decrease in LV diastolic compliance in cases with uncomplicated hypertension based on echocardiographic studies. However, no report related to the studies on LA mechanical function with hypertensive hearts has been found.

We therefore analyzed and evaluated LV systolic function and LV diastolic properties in patients with borderline hypertension and established hypertension. We will also report for the first time the analyses and evaluations of LA function in borderline and established hypertension in man.

METHODS

Patients

The results reported here were based on 42 patients who complained of "chest pain". These patients were divided into the following 3 groups: a normotensive group (N group, n = 12), a borderline hypertensive group (BH group, n = 13) and an established hypertensive group (EH group, n = 17), according to arterial blood pressure based on the WHO Classification. Patients with secondary hypertension were excluded from this study. The blood pressure was obtained as an average of 3 values measured in the sitting position, on 3 different days in the outpatient clinic, when the condition of patients was stable. Sex, age and arterial blood pressures in each group are shown in Table I.

All patients in this study gave no history of myocardial infarction or congestive heart failure, and patients with significant coronary arterial stenosis or coronary arterial spasm, shown by coronary arteriogram, were excluded from the study. An electrocardiogram revealed normal sinus rhythm in all patients. Furthermore, no patient had taken any medication for more than 4 weeks before examinations.

All patients gave informed written consent before this study. There were no complications attributable to the investigative procedures.

Cardiac Catheterization and Angiography

All catheterizations were performed according to the femoral approach in the non-sedated and fasting state of the patients. A 5F high-fidelity
catheter-tip micromanometer (Millar Instruments) was inserted into the LV retrogradely through the femoral artery. Then, a Brockenbrough catheter was inserted transeptally into the LA, monitoring images of the right atrium and the LA bidirectionally in antero-posterior (A-P) and lateral views. A 4F or 5F catheter-tip manometer was inserted into the LA through the Brockenbrough catheter.

LV and LA pressures in each patient were recorded photographically at paper speeds of 50 and 100 mm/sec with an eight-channel optical recording system (Model 7758 C, Hewlett-Packard). Following the recording of pressures, cardiac output was determined 3 times using a thermo-dilution method and the values were averaged.

LV cineangiograms were performed in 30° right anterior oblique (RAO) view after the measurement of cardiac output, and LV pressure was simultaneously recorded with a 5F tip-manometer at a paper speed of 100 mm/sec. Forty ml of contrast medium (76% Urografin) was injected for 2 or 3 sec into the LA through the Brockenbrough catheter using a power injector (Contract-4T, Siemens). Films were exposed at a rate of 60 frames/sec using a 35 mm cine camera and a 5 inch image intensifier (Phillips).

In the same way as for the LV pressure-volume measurement, LA cineangiograms were performed bidirectionally in A-P and lateral views, and LA pressure was recorded with a 4F or 5F tip-manometer inserted into the LA through the Brockenbrough catheter. Forty-five ml of contrast material was injected for 2.5 sec with a 8F NIH catheter inserted into the main pulmonary artery.

Finally, selective diagnostic coronary arteriography and a provocation test with 0.2 mg of ergonovine maleate, for evaluating coronary arterial spasm, were performed with 8F Judkins catheter in multiple projections.

Data Analysis

Comprehensive hemodynamic, volume and pressure-volume analyses were performed using the first available and adequately opacified sinus beat, avoiding ectopic beats and the first or second postectopic beat.

and by using a computerized system. Mean VCF was calculated as (EDL-ESL)/EDL × ET, where
EDL is end-diastolic inner diameter, ESL is end-
systolic inner diameter and ET is LV ejection
time.17 Vmax was obtained from the dp/dt/TP—
TP relation, where TP is total pressure.18 Further-
more LV Net-Work was determined from the LV
pressure-volume curve, which was obtained from
LV volume and the corresponding LV pressure
recorded simultaneously.

As parameters of the LV diastolic properties,
LV mass index (LVMI), LV end-diastolic wall
thickness (LVWTh), LV specific compliance (Compliance), LV stiffness constant (K1), time
constant (T), end-diastolic pressure (EDP) and
peak negative dp/dt (—dp/dt) were calculated
and analyzed.

LV mass was determined from measurements
of wall thickness and volume. Calculations
were based on determinations of the volume obtained
by subtracting the adjusted LV volume from the
total volume in which lateral wall thickness
included. Compliance was calculated as ΔV/ΔP,
where V is end-diastolic volume.19 Then, K1
proposed by Mirsky20 was calculated as follows:

\[ k = 3(1 + \log 2.33 \text{Ped}) \left[ \frac{1}{\frac{v + v1/3}{(1 + v)^2/3}} \right] \]

where k, Ped, VEd and Vw are stiffness constant,
end-diastolic pressure, end-diastolic volume and
LV mass, respectively, based on v = VEd/Vw. T
was calculated from the high speed recordings
of LV pressure and dp/dt according to the method
of Weiss et al.21

Furthermore, left atrial maximal volume
(LAV1), left atrial chamber stiffness constant
(K2) and left atrial muscle stiffness constant
(K3) were calculated and analyzed as indexes of
the LA function.23,24

LA volume was measured using the integra-
tion method of Chapman et al.22 Then, a pres-
sure-volume curve of LA was obtained as shown
in Fig. 1. This curve is composed of an “A” loop
and a “V” loop. The “A” loop represents LA
pumping function, and the “V” loop, reservoir
function. Then, the upward curve in the “V”
loop as shown in Fig. 2 is fitted to the formula

\[ P = aeK1V + c \]

where s is intrathoracic pressure. In this formula K2 represents the grade of
slope and chamber stiffness in the LA.

Furthermore, if it is supposed that the LA is a
thin walled sphere, wall stress is obtained from
“PR/2h” according to Laplace’s law, where h
is assumed to be 3 mm from the average of the

As parameters of the LV systolic function,
cardiac index (CI), left ventricular end-diastolic
volume index (LVEDVI), ejection fraction (EF),
left ventricular net-work (LV Net-Work), mean
ventricular circumferential fiber shortening
(m-VCF), Vmax and peak dp/dt (dp/dt) were
calculated and analyzed.

LV volume was obtained in all 60 frames by
the area-length method of Sandler and Dodge et
al; by tracing the silhouette with a sonic pen

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TABLE II  LEFT VENTRICULAR SYSTOLIC FUNCTION IN PATIENTS WITH NORMOTENSION (N), BORDERLINE HYPERTENSION (BH) AND ESTABLISHED HYPERTENSION (EH)

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>CI (L/min/m²)</th>
<th>LVEDVI (ml/m²)</th>
<th>EF (%)</th>
<th>LV Net Work (g·cm)</th>
<th>m-VCF (circ/sec)</th>
<th>Vmax (circ/sec)</th>
<th>Peak dp/dt (mmHg/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>12</td>
<td>3.55 ± 0.25</td>
<td>75 ± 5.7</td>
<td>67 ± 1.5</td>
<td>9050 ± 969</td>
<td>1.94 ± 0.16</td>
<td>1.71 ± 0.14</td>
<td>1308 ± 118</td>
</tr>
<tr>
<td>BH</td>
<td>13</td>
<td>4.09 ± 0.34</td>
<td>73 ± 3.9</td>
<td>67 ± 1.5</td>
<td>12508 ± 1742*</td>
<td>1.69 ± 0.17</td>
<td>1.82 ± 0.07</td>
<td>1520 ± 173</td>
</tr>
<tr>
<td>EH</td>
<td>17</td>
<td>3.65 ± 0.20</td>
<td>73 ± 4.1</td>
<td>66 ± 1.3</td>
<td>11145 ± 1495</td>
<td>1.64 ± 0.12</td>
<td>1.61 ± 0.07+</td>
<td>1493 ± 74</td>
</tr>
</tbody>
</table>

CI = cardiac index, LVEDVI = left ventricular end-diastolic volume index, EF = ejection fraction, LV Net Work = left ventricular net-work, m-VCF = mean ventricular circumferential fiber shortening.
Values are represented as mean ± SEM.
* = p < 0.05 as compared to values of the N group, + = p < 0.05 as compared to values of the BH group.

Fig.3. Correlations of left ventricular stiffness constant (Kₚ) with systolic (SBP), diastolic (DBP) and mean blood pressure (MBP) in 42 patients. Cases with normotension, borderline hypertension and established hypertension are expressed as (○), (×) and (●), respectively.

Fig.4. Correlations of left ventricular end-diastolic wall thickness (Thickness) with systolic (SBP), diastolic (DBP) and mean blood pressure (MBP) in 42 patients. Their correlations are not close. Cases with normotension, borderline hypertension and established hypertension are expressed as (○), (×) and (●), respectively.

LA wall thickness examined anatomically, because the LA wall thickness is difficult to measure clinically. Then wall strain is fitted to “P = a'eKₚV + c”, where P', V' and Kₚ are stress, strain and muscle stiffness constant, respectively. Hence, “K₂” and “Kₚ” represent the LA chamber stiffness constant and the LA muscle stiffness constant, respectively.

Statistical Analysis
The results are presented as mean ± SEM. Analyses of correlations between any of systolic.
TABLE III  LEFT VENTRICULAR DIASTOLIC PROPERTIES IN PATIENTS WITH NORMOTENSION (N), BORDERLINE HYPERTENSION (BH) AND ESTABLISHED HYPERTENSION (EH)

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>LVMI (g/m²)</th>
<th>LVWTh (mm)</th>
<th>Compliance (mmHg⁻¹)</th>
<th>K₁ (g/cm³)</th>
<th>T (msec)</th>
<th>EDP (mmHg)</th>
<th>Peak – dp/dt (mmHg/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>12</td>
<td>88 ± 8.0</td>
<td>11.1 ± 0.8</td>
<td>0.11 ± 0.04</td>
<td>37.3 ± 1.0</td>
<td>49 ± 3.4</td>
<td>9 ± 1.2</td>
<td>-1797 ± 137</td>
</tr>
<tr>
<td>BH</td>
<td>13</td>
<td>112 ± 14.5</td>
<td>13.0 ± 1.1</td>
<td>0.22 ± 0.03*</td>
<td>39.2 ± 1.6</td>
<td>54 ± 4.1</td>
<td>7 ± 1.3</td>
<td>-1971 ± 207</td>
</tr>
<tr>
<td>EH</td>
<td>17</td>
<td>142 ± 21.7</td>
<td>15.5 ± 1.3*</td>
<td>0.13 ± 0.01**</td>
<td>42.5 ± 1.5*</td>
<td>49 ± 1.7</td>
<td>9 ± 0.7</td>
<td>-2046 ± 209</td>
</tr>
</tbody>
</table>

LVMI = left ventricular mass index, LVWTh = left ventricular wall thickness, K₁ = Mirsky’s stiffness constant, T = Weiss’s time constant, EDP = left ventricular end-diastolic pressure. Results are expressed as mean ± SEM. * = p < 0.05 as compared to values of the N group, ++ = p < 0.01 as compared to values of the BH group.

Fig.5. Correlations of left ventricular specific compliance with systolic (SBP), diastolic (DBP) and mean blood pressure (MBP) in 42 patients. Cases with normotension, borderline hypertension and established hypertension are expressed as (○), (×) and (●), respectively.

Fig.6. Correlations of the left atrial (LA) volume index with systolic (SBP), diastolic (DBP) and mean blood pressure (MBP) in 18 patients. The numbers of cases with normotension (○), borderline hypertension (×) and established hypertension (●), are 6, 5 and 7, respectively. These correlations are statistically not significant (n.s.).

diastolic, mean blood pressure and any of the indexes of the LV and the LA functions, were made using standard methods of correlation coefficient. Group comparisons were assessed using the t-test for differences between group means. Statistical significance was considered at the p < 0.05 level.

RESULTS

LV Systolic Function

Seven parameters of LV systolic function
TABLE IV  LEFT ATRIAL VOLUME AND FUNCTION IN PATIENTS WITH NORMOTENSION (N), BORDERLINE HYPERTENSION (BH) AND ESTABLISHED HYPERTENSION (EH)

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>(K_2) (mmHg/ml)</th>
<th>(K_3) (g/cm^2)</th>
<th>LAVI (ml/m^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>6</td>
<td>25.5 ± 1.1</td>
<td>9.4 ± 0.6</td>
<td>52 ± 4.3</td>
</tr>
<tr>
<td>BH</td>
<td>5</td>
<td>29.2 ± 2.1</td>
<td>11.8 ± 0.5*</td>
<td>61 ± 3.5</td>
</tr>
<tr>
<td>EH</td>
<td>7</td>
<td>34.7 ± 1.5***</td>
<td>12.8 ± 0.5**</td>
<td>64 ± 4.4</td>
</tr>
</tbody>
</table>

\(K_2\) = left atrial chamber stiffness constant, \(K_3\) = left atrial muscle stiffness constant, LAVI = left atrial volume index.

Values are expressed as mean ± SEM.

* = \(p < 0.05\), ** = \(p < 0.01\) and *** = \(p < 0.001\) as compared to values of the N group.

Fig. 7. Correlations of left atrial chamber stiffness constant (\(K_2\)) with systolic (SBP), diastolic (DBP) and mean blood pressure (MBP) in 18 patients. The number of cases with normotension (○), borderline hypertension (×) and established hypertension (●) are 6, 5 and 7, respectively.

were measured and analyzed. Any of these parameters were not significantly correlated with systolic, diastolic or mean blood pressure. Correlation coefficients of blood pressure were less than 0.280 with CI, LVEDVI, EF, LV Net-Work, m-VCF, Vmax and peak dp/dt.

On the other hand, as shown in Table II, significant differences among indexes of LV systolic function in each group were not found, except for LV Net-Work in the BH group vs the N group, and Vmax in the EH group vs the BH group (\(p < 0.05\)).

LV Diastolic Properties

LVMI, LVWTh, specific compliance, \(K_1\), T, EDP and –dp/dt were determined and analyzed. In all 42 patients, \(K_1\) (Fig. 3). LVMI and LVWTh (Fig. 4) were roughly correlated with systolic and mean blood pressure (\(p < 0.05\), \(r = 0.306\sim 0.353\)). Furthermore, LVWTh was also correlated significantly with diastolic blood pressure, as shown in Fig. 4. However, no significant correlations were found between blood pressure and any of specific compliance, T, EDP and –dp/dt. Correlation coefficients of blood pressure were \(r = -0.008\sim -0.047\) with specific compliance, as shown in Fig. 5, \(r = 0.100\sim 0.132\) with T, \(r = 0.038\sim 0.151\) with EDP and \(r = 0.094\sim 0.229\) with –dp/dt.

There were significant differences in LVWTh and \(K_1\) between the N and the EH groups. There was a greater tendency towards an increase in LVMI, LVWTh, \(K_1\) and –dp/dt in the EH than in the BH group, and in the BH than in the N group, even when this was not significant. These results are indicated in Table III. Nevertheless, in specific compliance, the BH group presented the greatest increase among the 3 groups and it was significant.

LA Function

LAVI was not correlated significantly with blood pressure (\(r = 0.308\sim 0.384\), as shown in Fig. 6. Among the N, the BH and the EH groups,
in man. Its cardiac consequences have been reported to depend closely on the degree of myocardial hypertrophy, and on the progression of coronary artery disease. Strauer has pointed out that coronary blood flow and coronary resistance were increased, and that LV contractile performance was not affected in patients with established hypertension without significant coronary arterial stenosis. Moreover, he indicated that hypertrophied LV in essential hypertension, when it was compensated, did not reveal a reduction of LV contractile performance. Although Kailner et al. have reported that LV systolic function was not impaired in hypertensive hearts regardless of LV hypertrophy, Mehmel et al. have indicated, on the contrary, that LV systolic function was reduced in patients with hypertension.

From the foregoing results obtained from 7 parameters inducing LV systolic function, except for $V_{max}$, we may conclude that LV systolic function is not reduced in patients with established hypertension without a significant coronary stenosis and a history of congestive heart failure. $V_{max}$ in the EH group was lower than in the BH and the N groups. However, $V_{max}$ may have some weak points in assessing systolic function when applied to the whole heart because it was a parameter proposed in the experiment of a single myocardial fiber.

There have been especially few reports on the LV function in patients with borderline hypertension, partly because such patients are often asymptomatic and are not thought to justify LV catheterization. Therefore, LV systolic function in patients with borderline hypertension has not been evaluated at all.

DISCUSSION

Cardiac function in patients with essential hypertension has been studied as the most frequent model of chronic LV pressure overload

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Frohlich et al. have proposed a concept of "Hyperdynamic Circulatory State", supposedly by other echocardiographic or hemodynamic studies in borderline or labile hypertension. This condition was defined as a mild elevation of blood pressure, resulting from an increase of cardiac output and a decrease in total peripheral resistance. It has been suggested that LV systolic function in borderline hypertension may increase more than in normotensive controls. Our hemodynamic results were similar to theirs, and indicated an increase of CI in the BH group, but no difference of total peripheral resistance between the BH and the N group, though significantly less than in the EH group, as shown in Fig. 9. However, 7 parameters of LV systolic function, except for LV Net-Work, presented no significant increase in patients with borderline hypertension. CI, LV Net-Work, V_max and Peak dp/dt were greater in the BH group than in the N group, but they were not statistically significant, except for LV Net-Work.

From these results, it is suggested that LV systolic function in patients with borderline hypertension neither increase nor decrease as compared to LV systolic function in patients with normotension.

LV diastolic properties are a major determinant of cardiac function. Cardiac hypertrophy in hypertensive hearts is one of the fundamental compensating mechanisms for chronic pressure overload.

From our results, LVMI, LVTh and Mirsky's stiffness constant K_s revealed a greater increase in the EH group than in the BH and the N group. However, in regard to Weiss's time constant T, LVEDP and peak negative dp/dt, significant differences were not found among 3 groups. Furthermore, in relation to LV specific compliance, the greatest increase was found in patients with borderline hypertension. Hence, specific compliance determined in the whole heart is not so sensitive, and may possibly be influenced by high cardiac output and low LVEDP in the BH group.

Therefore, it is suggested that LV diastolic properties are not reduced in patients with borderline hypertension. On the other hand, LV diastolic properties in patients with established hypertension are possibly impaired due to the progression of the LV hypertrophy.

Left atrial function contributes to the passive filling of the LV. Therefore, it is of particular importance to analyze the LA function in patients with the LV hypertrophy or other conditions of reduced ventricular compliance, in which the LV filling is impeded. Tarazi et al. have reported electrocardiographic abnormalities in hypertension. They described that a left atrial P wave was to be found before the development of the LV hypertrophy. Many echocardiographic studies have indicated that an enlargement of the LA size was demonstrated in patients with hypertension, prior to the development of the LV hypertrophy. Dreslinski et al. have proposed "Atrial Emptying Index" as an indicator of LV diastolic properties. They demonstrated that atrial emptying index in patients with essential hypertension were smaller than in normotensive patients, without roentgenographic, electrocardiographic or echocardiographic evidence of the LV enlargement or abnormality.

The LA function consists of pumping and reservoir function. Analyses of LA pumping function were made in animal experiments by Mitchell et al. and Kalmanson et al. Then, left atrial compliance was evaluated quantitatively in the canine left atrium by Suga. He described that the LA compliance was particularly important for the LV performance. However, evaluations of the LA have not been definitely established.

Nakajima et al. have determined first analyses of the LA compliance in man, applying a method of the analysis of the LV stiffness to the LA, as described in Methods. In patients with hypertrophied hypertensive heart or mitral stenosis, both chamber stiffness constant and muscle stiffness constant in the LA were increased significantly. On the other hand, in patients with mitral regurgitation, muscle stiffness constant increased, but chamber stiffness constant decreased because of the enlargement of the LA to compensate for the volume overload. From our present data of hypertensive hearts, although both chamber stiffness constant and muscle stiffness constant increased in patients with established hypertension, muscle stiffness constant was impaired, and chamber stiffness constant tended to increase in patients with borderline hypertension. These results demonstrate that the LA compliance may be affected already both in patients with borderline hypertension and in patients with established hypertension.

From our present results, we conclude finally that the LV systolic function is not impaired in...
patients with borderline and established hypertension, and that the LV diastolic function seems to be reduced by the progression of the LV hypertrophy in patients with established hypertension, and not to be affected in patients with borderline hypertension. However, it seems that the LA function is reduced already in patients with borderline hypertension and also in patients with established hypertension.

REFERENCES


