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Diastolic properties of hypertrophied hearts in essential hypertension: Classification by left ventricular wall stress

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Summary

Hypertensive cardiac hypertrophy of 20 patients was classified as inappropriate hypertrophy (HH-I) and appropriate hypertrophy (HH-II) according to their end-systolic wall stress, as measured by echocardiography. The differences in systolic and diastolic performances among the HH-I and HH-II subjects and 10 normal controls (NC) before and during isoproterenol infusion were investigated. Eight patients had subnormal end-systolic wall stress (inappropriate hypertrophy) and 12, normal end-systolic wall stress (appropriate hypertrophy). Before isoproterenol infusion, normalized peak rate of a change in left ventricular diameter during systole was significantly greater in HH-I (3.5 ± 0.8/s) than in NC (2.3 ± 0.5/s) and HH-II (2.6 ± 0.6/s) ($p<0.01$ and $p<0.005$), but there was no significant difference between HH-II and NC. There was no significant difference in normalized peak rate of a change of left ventricular diameter during the rapid filling phase among the three groups (4.5 ± 1.2/s in HH-I, 4.0 ± 1.6/s in HH-II, and 4.2 ± 0.8/s in NC). During isoproterenol infusion, normalized peak rate of a change of left ventricular diameter during systole was significantly greater in HH-I (7.0 ± 1.9/s) than in HH-II (4.8 ± 1.7/s) and NC (4.8 ± 0.8/s) ($p<0.05$ and $p<0.01$, respectively), but there was no significant difference between HH-II and NC. Normalized peak rate of a change of left ventricular diameter during rapid filling was significantly less in HH-II (4.8 ± 1.7/s) than in HH-I (7.3 ± 1.3/s) and NC (6.5 ± 0.8/s) ($p<0.005$ and $p<0.005$, respectively), but there was no significant difference between HH-I and NC.

These results suggest that hypertensive patients with inappropriate hypertrophy have relatively diminished diastolic velocity (supernormal systolic velocity and normal diastolic velocity) before and during isoproterenol infusion, and that hypertensive patients with appropriate hypertrophy have absolutely diminished diastolic velocity during isoproterenol infusion, in spite of normal diastolic velocity before the infusion.

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Introduction

That chronic hypertension leads to left ventricular hypertrophy in an effort to normalize left ventricular systolic wall stress is generally recognized. Hypertrophy of the left ventricle may result in left ventricular systolic and diastolic dysfunction. We reported that the left ventricular systolic response to beta-adrenergic stimulation increases in patients with hypertensive hypertrophy with *subnormal* end-systolic wall stress (inappropriate hypertrophy) and that the response was normal or becomes reduced in hypertrophy with *normal* end-systolic wall stress[1] (appropriate hypertrophy). It is well known that left ventricular hypertrophy affects systolic performance, which in turn affects diastolic performance. Therefore, the difference between inappropriate and appropriate hypertrophy can lead to different diastolic performances. Echocardiographic and radionuclide angiographic studies may provide much important information about left ventricular diastolic function. However, to our knowledge, the difference in diastolic function between inappropriate and appropriate hypertrophy has not been investigated in patients with hypertension.

Diastolic function should be evaluated not only at rest but also under loading condition such as exercise or isoproterenol infusion because rapid filling becomes increasingly important as heart rate increases. We reported that augmentation of left ventricular filling during exercise impaired in patients with hypertrophic cardiomyopathy[2]. The present study was designed to determine whether there is any difference in peak left ventricular filling velocity between inappropriate and appropriate hypertrophy in patients with hypertension before and during isoproterenol infusion.

Patients and methods

Patients

Twenty patients with left ventricular hypertrophy who had hypertension lasting more than five years (15 men and five women ranging in age from 31 to 64 years), and ten normal controls (NC, eight men and two women, 30 to 55 years of age) were studied. Blood pressure was measured by cuff sphygmomanometers, using 1 and 5 of the Korotkoff sounds. Arterial hypertension was defined as persistent systolic arterial pressure higher than 95 mmHg before instituting antihypertensive treatment, or four weeks after discontinuing antihypertensive drugs. Essential hypertension was diagnosed in all 20 patients, all of whom had echocardiographic evidence of left ventricular hypertrophy, consisting of 1) interventricular septal wall thickness $\geqq$12 mm at end-diastole, and 2) left ventricular end-diastolic posterior wall thickness $\geqq$12 mm. In order to restrict the study to patients with pure hypertrophy, we studied only those with left ventricular end-diastolic diameter $<$55 mm. None of these patients had evidence of other heart disease from their cardiovascular histories, physical examinations, electrocardiography and echocardiography; and all were in sinus rhythm without signs of heart failure. Coronary arteriography was not justified in these patients; therefore, concomitant coronary artery disease was necessarily excluded on clinical grounds alone. Thus, the patients in this study had arterial hypertension without clinical evidence of coronary artery disease.

The normal controls had no cardiovascular or pulmonary disease and gave their written consents before participating in this study. They were judged as normal by detailed physical examination including blood pressure, chest radiography, electrocardiography, exercise stress tests, and echocardiography.

Methods

1. Echocardiographic studies

M-mode echocardiograms were recorded using a Toshiba SSH-11A ultrasonoscope with a 2.25 MHz transducer and a Honeywell 1219 strip chart recorder, operating at a paper speed

of 50 mm/s. The transducer was placed over the left third or fourth intercostal space at the sternal margin. Left ventricular echocardiograms were recorded at the level of the chordae tendineae just below the tips of the mitral leaflets under monitoring of two-dimensional echocardiograms. An electrocardiogram (ECG) and a phonocardiogram (PCG) with a contact microphone over the left second intercostal space at the sternal margin were recorded simultaneously with the echocardiogram. Each echocardiogram was obtained during expiration. The thicknesses of the interventricular septum and the posterior left ventricular wall were measured at the time of the R wave on the ECG. End-systolic wall stress was calculated using the equation:

$$\frac{\text{peak arterial pressure} \times \text{LVDs}^2}{4(\text{LV wall thickness}) \times (\text{LVDs} + \text{LV wall thickness})^4}$$

LVDs=left ventricular end-systolic diameter; wall thickness=(thickness of the septum+thickness of the posterior wall)/2 at end-systole.

This is an expression of the average meridional wall stress, defined as the force per unit area acting at the equatorial plane of the ventricle in the direction of the apex-to-base axis. Calculating end-systolic wall stress from these measurements has been validated.

The patients were categorized as: group 1, consisting of eight patients with end-systolic wall stress <2SD below the normal mean (<36.4 g/cm^2) (HH-I); and group 2, consisting of 12 patients with end-systolic wall stress within 2SD of the normal mean (HH-II).

2. Digitized echocardiographic analysis

Left ventricular echocardiograms were analyzed using a digitizer (Tektronix 4953) which had interfaced with a minicomputer (Yokogawa Hewlett Packard 2108). This technique has been described previously[3,4]. The coordinates of the points representing the interventricular septum were subtracted from those of the posterior left ventricular wall echoes to provide instantaneous left ventricular diameters (D). The first derivatives (dD/dt) and (dD/dt)/D (dividing dD/dt by instantaneous D) were obtained. From the digitized data, the following values were calculated: 1) left ventricular end-diastolic diameter (Dd) as determined at the time of the R wave on the ECG and end-systolic diameter (Ds) as determined at the time of the onset of the second heart sound on the PCG. Fractional shortening (FS, %) was calculated as $(Dd-Ds)/Dd \times 100$. 2) Normalized peak rate of a change of left ventricular diameter: Normalized peak rate of a change of the diameter during systole (pVs) and that during the rapid filling phase (pVd) were obtained as the minimum and maximum (dD/dt)/D, respectively. To estimate the reproducibility of the digitized echocardiographic analysis, pVs and pVd were measured three times from the echocardiogram by the same observer.

3. Isoproterenol infusion

The study was performed in the afternoon in all patients. Simultaneous echocardiographic and electrocardiographic baseline recordings, and blood pressure measurements were performed with the patients in the supine position immediately before intravenous infusion of isoproterenol (0.02 μg/kg/min) by means of a calibrated infusion pump. After 5-min infusion, the echocardiogram and electrocardiogram were recorded and the blood pressure was measured using a cuff and mercury column sphygmomanometer. The transducer was fixed to the same part of the chest wall throughout the examination. To standardize the technique among the patients, and to monitor the same part of the left ventricle (just below the tip of the anterior mitral leaflet) before and after the infusion of isoproterenol, the beams of M-mode echocardiography were aligned perpendicular to the posterior wall of the left ventricle and to the wall of the interventricular septum in all patients both before and after the isoproterenol infusion.

The data were analyzed using a one-way layout analysis of variance. Differences were considered statistically significant when probability (p) was less than 0.05. All data were given as means$\pm$SD.

Table 1. Hemodynamic and echocardiographic data at baseline

	HR (beats/min)	SBP (mmHg)	DBP (mmHg)	Dd (mm)	Ds (mm)	FS (%)
HH-I	68±12	147± 9	102± 7	45±4	24±3	46±4
HH-II	69±14	171±16	106±15	46±5	28±5	38±9
NC	69± 6	127±14	81± 9	49±3	32±3	36±3
HH-I vs HH-II	ns	***	ns	ns	ns	*
HH-I vs NC	ns	***	****	*	****	***
HH-II vs NC	ns	***	****	ns	ns	ns

HH-I=inappropriate hypertrophy (subnormal end-systolic wall stress); HH-II=appropriate hypertrophy (normal end-systolic wall stress); NC=normal controls; HR=heart rate; SBP=systolic blood pressure; DBP=diastolic blood pressure; Dd=left ventricular end-diastolic diameter; Ds=left ventricular end-systolic diameter; FS=fractional shortening.

*=$p<0.05$, ***=$p<0.005$, ****=$p<0.001$.

Results

Table 1 shows the hemodynamic values and echocardiographic variables before the isoproterenol infusion. There was no significant difference in heart rate among the groups. Systolic blood pressure was significantly higher in HH-II than in HH-I and NC ($p<0.005$ and $p<0.005$, respectively), and was also significantly higher in HH-I than in NC ($p<0.005$ and $p<0.005$, respectively). Diastolic pressure was significantly higher in HH-I and HH-II than in NC ($p<0.001$ and $p<0.001$, respectively), but there was no significant difference between HH-I and HH-II. Left ventricular end-diastolic diameter (LVDd) was significantly less in HH-I than in NC ($p<0.05$) but there was no significant difference between HH-I and HH-II. Left ventricular end-systolic diameter (LVDs) was significantly less in HH-I than in NC ($p<0.001$), but there was no significant difference between HH-I and HH-II. FS was significantly greater in HH-I than in NC and HH-II ($p<0.005$ and $p<0.05$, respectively). Normalized peak rate of a change of left ventricular diameter during systole was significantly greater in HH-I (3.5±0.8/s) than in NC (2.3±0.5/s) and HH-II (2.6±0.6/s) ($p<0.01$ and $p<0.005$), but there was no significant difference between HH-II and NC (**Fig. 1**). There was no significant difference in pVd among the three groups (4.5±1.2/s in HH-I, 4.0±1.6/s in HH-II, 4.2±0.8/s in NC) (**Fig. 1**). Supernormal systolic velocity and normal diastolic velocity in HH-I represent relatively diminished diastolic velocity. There was no significant difference in the thickness of the interventricular septum and posterior wall between HH-I and HH-II (**Fig. 2**). There was asymmetric septal hyper-

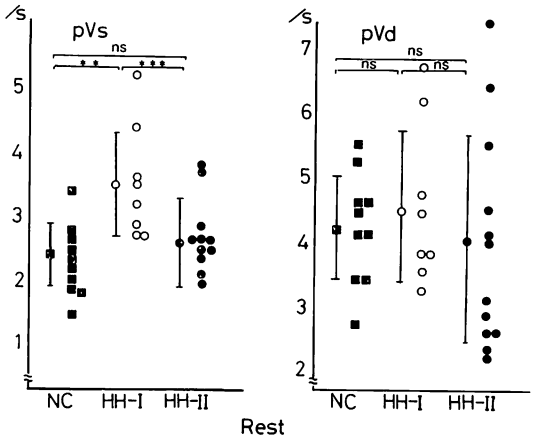

Fig. 1. Comparison of normalized peak rate of the diameter during systole (pVs) **and normalized peak rate of a change of the diameter during the rapid filling phase** (pVd).

Abbreviations are as in Table 1.

**=$p<0.01$, ***=$p<0.005$.

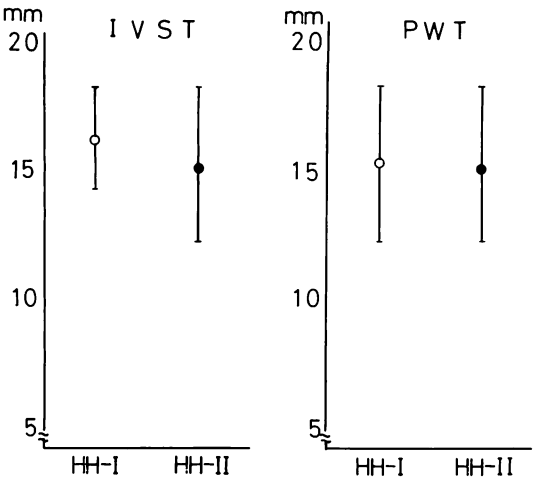

Fig. 2. Left ventricular wall thicknesses in hypertensive groups.

There is no significant difference in IVST and PWT between HH-I and HH-II.

IVST=interventricular septal thickness; PWT=posterior wall thickness.

trophy (interventricular septal thickness / posterior wall thickness $\geqq 1.3$) in three patients (38%) with HH-I and in three patients (25%) with HH-II.

Isoproterenol infusion

Fig. 3 shows echocardiograms before and during isoproterenol infusion. The endocardial echo of the posterior left ventricular wall and the left side of the interventricular septum can be seen clearly in both of the echocardiograms. Echoes of the chordae tendineae indicate that these echocardiograms are from the same part of the left ventricle.

Table 2 shows the hemodynamic and echocardiographic variables during isoproterenol infusion. Infusion of isoproterenol increased heart rate in the three groups. There was no significant difference in heart rate during isoproterenol infusion among the three groups. Systolic blood pressure was significantly higher in HH-II than in HH-I and NC ($p<0.01$ and $p<0.005$, respectively). Diastolic blood pressure was significantly higher in HH-I and HH-II than in NC ($p<0.005$ and $p<0.005$, respectively), but there was no significant difference between HH-I and HH-II. LVDd was significantly less in HH-II than in NC ($p<0.05$). LVDs was significantly less in HH-I than in HH-II and NC ($p<0.01$ and $p<0.005$). FS was significantly greater in HH-I than in HH-II and NC ($p<0.01$ and $p<0.005$, respectively) and there was no significant difference between

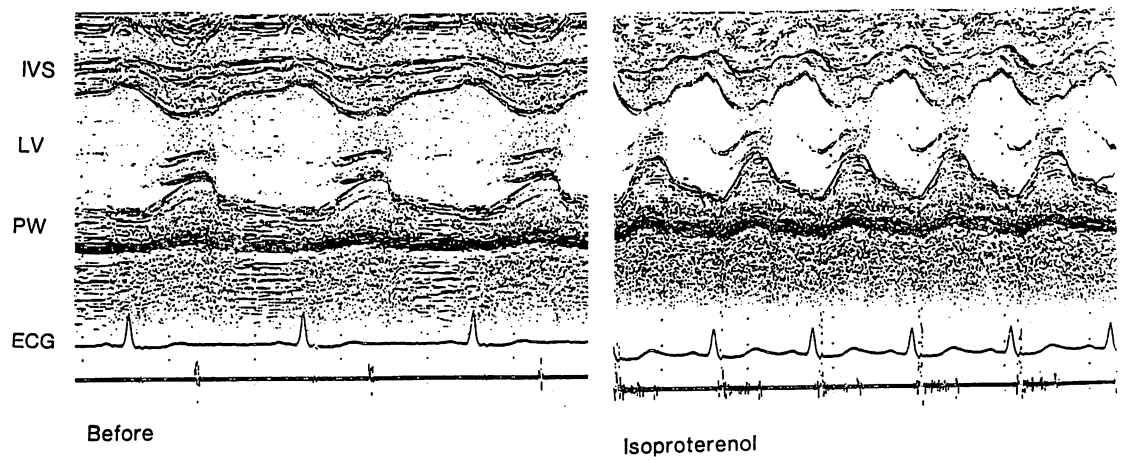

Fig. 3. Echocardiograms of a patient in HH-II before and during isoproterenol infusion.

IVS=interventricular septum; LV=left ventricular cavity; PW=left ventricular posterior wall.

Table 2. Hemodynamic and echocardiographic data during isoproterenol infusion

	HR (beats/min)	SBP (mmHg)	DBP (mmHg)	Dd (mmHg)	Ds (mm)	FS (%)
HH-I	101±18	160±14	90±12	45±4	18±3	60±6
HH-II	104± 9	184±19	94±19	44±5	23±4	47±8
NC	99±12	139±19	66±17	49±3	25±4	49±5
HH-I vs HH-II	ns	***	ns	ns	ns	*
HH-I vs NC	ns	*	****	ns	****	****
HH-II vs NC	ns	****	***	*	ns	ns

Abbreviations: See Table 1.

HH-II and NC. Normalized peak rate of change of left ventricular diameter during systole was significantly larger in HH-I (7.0±1.9/s) than in HH-II (4.8±1.7/s) and NC (4.8±0.8/s) ($p<0.05$ and $p<0.01$, respectively), but there was no significant difference between HH-II and NC (**Fig. 4**). Normalized peak rate of a change of left ventricular diameter during the rapid filling phase was significantly less in HH-II (4.8±1.7/s) than in HH-I (7.3±1.3/s) and NC (6.5±0.8/s) ($p<0.005$ and $p<0.005$, respectively) (**Fig. 4**). The supernormal systolic velocity and normal diastolic velocity in HH-I represent relatively diminished diastolic velocity and normal systolic velocity, and the decreased diastolic velocity in HH-II represents an absolutely decreased diastolic velocity.

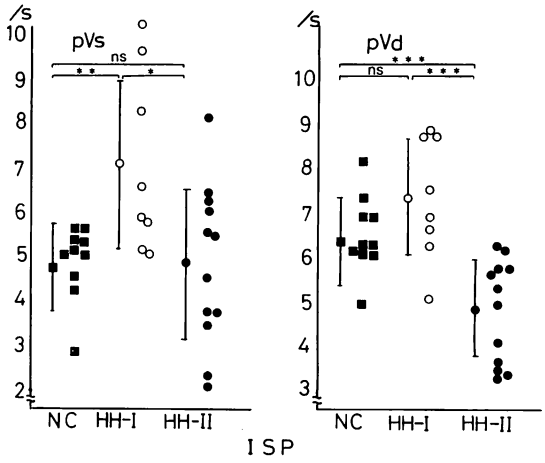

Fig. 4. Comparison of pVs and pVd in the three groups.

ISP=isoproterenol. Other abbreviations: see Fig. 1. *=$p<0.05$, **=$p<0.01$, ***=$p<0.005$.

Discussion

This study demonstrated differences in left ventricular diastolic function between patients with hypertensive hypertrophy with subnormal end-systolic wall stress (inappropriate hypertrophy: HH-I) and patients with hypertensive hypertrophy with normal end-systolic wall stress (appropriate hypertrophy: HH-II). There was no significant difference in pVd between HH-I and HH-II before isoproterenol infusion; however, pVd was significantly lower in HH-II than in HH-I during isoproterenol infusion. Echocardiographic systolic parameters (FS and pVs) were significantly higher in HH-I than in HH-II, as reported previously[1].

Method of studying ventricular filling

Left ventricular function can be evaluated by several methods. Left ventricular filling velocity and pressure-volume relations can be determined by cardiac catheterization and cineangiography. Although these measurements may be the most accurate for evaluating diastolic function, they require an invasive technique[5], which is not justified in patients with hypertension who presumably have no other heart disease, such as coronary artery disease. Diastolic performance can be evaluated using several noninvasive techniques. The left ventricular

filling rate can be obtained by radionuclide angiography and by digitized echocardiography. Digitized echocardiography can be used to evaluate left ventricular filling performance without incurring ionizing radiation exposure. Another echocardiographic parameter for evaluating left ventricular diastolic function rather than pVd is isovolumic relaxation time (IRT). The duration of this interval is determined not only by the rate of a left ventricular pressure decline (negative dP/dt), but also by the magnitude of a pressure drop[6,7]. The pressure drop is determined by the pressure at the time of the aortic component of the second heart sound and the pressure at the time of left ventricular/left atrial pressure crossover (mitral valve opening). As a result, blood pressure will affect the absolute value of IRT. Therefore, this is not an appropriate means for evaluating the difference in filling performance between normal controls and patients with hypertension.

Inappropriate hypertrophy (subnormal end-systolic wall stress: HH-I)

The present study demonstrated that systolic indices such as FS and pVs were significantly higher in patients with inappropriate hypertrophy than in patients with appropriate hypertrophy or normal controls before and during isoproterenol infusion. Several previous studies demonstrated that systolic function was nearly normal or slightly depressed in patients with hypertension[8~15]. However, some studies have shown that systolic function was increased in patients with mild or labile hypertension[16~18]. In patients with borderline hypertension, Safar et al. reported a reduced pre-ejection period and increased cardiac output, which suggests increased cardioadrenergic drive[19]. We previously reported that the beta adrenergic response of the left ventricle is increased in patients with hypertensive hypertrophy with inappropriate hypertrophy and suggested that, in addition to mechanical factors such as blood pressure, neurohormonal influences can play an important causative role in cardiac hypertrophy of patients with hypertension[1]. Experimentally, small doses of isoproterenol or other catecholamines have been reported to cause cardiac hypertrophy in animals without changing their blood pressure or heart rate[20,21]. The hypersensitivity (increased response to isoproterenol) supposedly produces the same effect (left ventricular hypertrophy) as does catecholamine infusion.

In the present study, diastolic function as expressed by pVd was normal before and during isoproterenol infusions in patients with inappropriate hypertrophy. Left ventricular diastolic function in hypertensive patients has been evaluated using echocardiographic, Doppler echocardiographic and radionuclide angiographic techniques. Abnormal left ventricular diastolic function has been reported in patients with cardiac hypertrophy[10,11,22,23]. However, this is still controversial. Hanrath demonstrated an abnormal prolongation of the left ventricular relaxation time, as measured from the aortic component of the second heart sound to the mitral valve opening in echocardiograms[22]. Gibson et al. reported that digitized echocardiograms showed slowing of pVd in patients with hypertension[10]. Inoue et al. used gamma camera-derived left ventricular volume curves to assess three indexes of left ventricular filling (first one-third filling fraction, peak filling rate and time-to-peak filling rate) in patients with mild-to-moderate hypertension. These variables were abnormal in hypertensive patients as compared with controls, and significant correlations were present between posterior wall thickness and the first third filling fraction, and between posterior wall thickness and left ventricular mass and time-to-peak filling rate[23]. Fouad et al. showed that pVd was decreased in hypertensive patients using radionuclide-derived ventricular volume curves and that pVd correlated inversely with left ventricular mass[11]. Smith et al. used a non-imaging nuclear probe time-activity curve to assess left ventricular filling in hypertensive patients. Their patients had a markedly lower average of the rapid left ventricular filling rate and there was a modest inverse relation between the echocardiographic left ventricular mass index and average filling rate[13]. However, several studies have demonstrated that pVd does

not significantly differ from that of normal controls. A study by digitized echocardiography demonstrated that pVd was similar between normal subjects and patients with hypertension, though the peak rate of early relaxation of the posterior left ventricular wall was significantly lower[12]. Dianzumba et al., using Doppler echocardiography, reported no significant difference in early diastolic mitral peak flow velocities between patients with untreated mild hypertension and normotensive controls[14]. Gardin et al. reported that transmitral early diastolic peak flow velocities in mild hypertensive patients, evaluated by Doppler echocardiography, did not differ significantly from values predicted on the basis of these normal regression equations[24]. The velocity correlated neither with echocardiographic left ventricular mass nor with casual or 24-hour average systolic or diastolic blood pressures.

The systolic and diastolic characteristics in our patients with inappropriate hypertrophy were very similar to those in hypertrophic cardiomyopathy[25]. Though the etiology of hypertrophic cardiomyopathy is yet unknown, a number of clinical and experimental clues suggest the link with catecholamine function. We previously reported hypersensitivity of the beta-adrenergic system in hypertrophic cardiomyopathy with asymmetric septal hypertrophy and suggested that this hypersensitivity plays an important role in producing cardiac hypertrophy[26]. It is not clear from our study whether these patients with inappropriate hypertrophy have genetic disorders, such as hypersensitivity of the beta-adrenergic system producing a substrate to that, and develop typical features of hypertrophic cardiomyopathy when exposed to long-standing hypertension. Furthermore, it is not certain whether this hypertrophy simply manifests a coincidence of hypertrophic cardiomyopathy and hypertension.

Appropriate hypertrophy (normal end-systolic wall stress: HH-II)

The present study showed that there was no significant difference in pVs and pVd between HH-II and normal controls before isoproterenol infusion. During isoproterenol infusion, pVd was significantly lower in HH-II than in normal controls, though there was no significant difference in pVs between HH-II and normal controls.

We previously reported that the systolic response of the left ventricle to beta-adrenergic stimulation was normal in hypertensive patients with appropriate hypertrophy, and proposed it might have been introduced by a mechanical factor; for example, high blood pressure[1]. To our knowledge, pVd during isoproterenol infusion has not been investigated in patients with hypertension. As mentioned above, the change of pVd at rest is still controversial among hypertensive patients. The present study showed that the diastolic response of the left ventricle to beta-adrenergic stimulation deteriorated in these patients. Fouad et al. demonstrated that there was a highly significant negative correlation between pVd and left ventricular end-systolic wall stress[11]. By definition, end-systolic wall stress is related to each of the values entering into the calculation of stress; namely systolic pressure, LVDd and left ventricular wall thickness in end-systole. LVDs during isoproterenol infusion was greater in HH-II than in HH-I, indicating that FS was smaller in HH-II than in HH-I. Fouad et al. and Inouye et al. reported that there were significant correlations between indexes of overall systolic function or left ventricular contractility and the left ventricular filling rate[11,23]. These smaller FS and pVs during isoproterenol infusion may partially account for the decreased pVd in patients with appropriate hypertrophy.

Significance of abnormal left ventricular filling

Left ventricular diastolic filling consists of three phases. Rapid filling of the ventricle occurs in early diastole. It is followed by mid-diastolic slow filling and filling due to atrial contraction. As heart rate increases, mid-diastolic slow filling steadily decreases in duration until passive rapid filling and active filling due to atrial contraction[27] are superimposed. The reduced dimensional increase during the rapid filling phase

is compensated by a more vigorous atrial kick, which is associated with elevated atrial pressure. The resultant effect on pulmonary pressure supposedly provides major limitations on the ability to exercise, even though systolic function is normal. Although pVd was normal before and during isoproterenol infusion in hypertensive patients with inappropriate hypertrophy, supernormal systolic function suggests that diastolic function is relatively diminished in those patients. We previously reported that the ratio of pVd to pVs showed no significant increment during exercise in patients with hypertrophic cardiomyopathy though that ratio was significantly increased by exercise in normal controls[2]. The relatively low pVd in inappropriate hypertrophy and absolutely low pVd in appropriate hypertrophy may decrease exercise capacity.

Mechanism of abnormal left ventricular filling

From the present study, the exact cause of abnormal diastolic function is not clear. There are possible explanations for abnormal left ventricular filling, once cardiac hypertrophy is established. Myocardial ischemia, often present in left ventricular hypertrophy, has been shown to impair left ventricular diastolic function[28,29]. However, the present study demonstrated that systolic function was supernormal in inappropriate hypertrophy and that it was normal in appropriate hypertrophy before and during isoproterenol infusion. Though there was no significant difference in the left ventricular thickness between appropriate and inappropriate hypertrophy, diastolic performance was different between them. These results do not support the argument that myocardial ischemia alone plays an etiologic role in abnormal diastolic function.

Experimentally, Morgan et al. demonstrated an imbalance in intracellular calcium regulation, relating to diastolic abnormalities in the hypertensive hypertrophied heart[30]. We showed that nifedipine increased pVd in patients with hypertrophic cardiomyopathy[4], and Betocchi et al. reported that verapamil increased the peak filling rate in hypertensive patients[31]. These observations suggest that abnormal calcium metabolism may impair left ventricular diastolic function in hypertensive patients with hypertrophy.

Clinical implications

There are numerous types of antihypertensive medications. Some, like calcium antagonists, not only lower elevated blood pressure, but ameliorate diastolic function; others, like beta-blockers, lower blood pressure and decrease systolic function. The present study demonstrated the difference in diastolic performance between inappropriate and appropriate hypertrophy, and suggested the importance in considering diastolic function when prescribing medications for hypertensive patients. Further studies to delineate the differences between these two groups as to the influence of therapy on cardiac function and left ventricular hypertrophy, may not only assist our understanding of the hypertensive hypertrophied heart, but lead to new approaches in the management of the hypertensive heart, as well.

要　約

高血圧心における収縮速度ならびに拡張速度の肥大様式による差異

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高血圧性肥大心 20 例を，心エコー図にて求めた収縮終期左室応力により，収縮期血圧に対し過剰な心肥大 inappropriate hypertrophy (HH-I) 8 例および収縮期血圧に見合った心肥大 appropriate hypertrophy (HH-II) 12 例に分類し，正常対象群 10 例および各肥大群での isoproterenol 負荷前後における収縮ならびに拡張動態を検討した．

Isoproterenol 負荷前において HH-I の左室収縮最大速度 (3.5±0.8/s) は，HH-II (2.6±0.6/s) および NC (2.3±0.5/s) より有意に高値であった

が(それぞれ $p<0.01$, $p<0.005$), HH-II と NC 間には有意差を認めなかった. 左室拡張最大速度には3群間で有意差を認めなかった (HH-I: 4.5±1.2/s, HH-II: 4.0±1.6/s, NC: 4.2±0.8/s). Isoproterenol 負荷において HH-I の左室収縮最大速度 (7.0±1.9/s) は HH-II (4.8±1.7/s) および NC (4.8±0.8/s) より有意に高値であった(それぞれ $p<0.05$, $p<0.01$) が, HH-II と NC 間に有意差を認めなかった. HH-II の左室拡張最大速度 (4.8±1.7/s) は HH-I (7.3±1.3/s) および NC (6.5±0.8/s) より有意に低値であったが(それぞれ $p<0.005$, $p<0.005$), HH-I と NC 間に有意差を認めなかった.

以上の結果より, inappropriate hypertrophy 群における収縮速度は isoproterenol 負荷前後において正常より高値であるが, 拡張速度は正常域に留まり, 相対的拡張機能不全が示された. 一方 appropriate hypertrophy では, 拡張速度は isoproterenol 負荷後において正常より低下しており, isoproterenol 負荷後における絶対的拡張機能が示された.

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