Original Article

Regional Wall Motion Abnormalities During Early Diastole in Patients with Hypertensive Left Ventricular Hypertrophy: A Doppler Tissue Echocardiographic Study

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To investigate left ventricular wall motion asynchrony in patients with hypertensive heart disease, we measured regional myocardial velocity in hypertensive patients with left ventricular hypertrophy and in normotensive individuals using tissue Doppler imaging. The endocardial velocity and the myocardial velocity gradient were measured in the basal and mid segments of the septal and posterior walls. The dilating velocity of the left ventricular cavity were determined for the basal and mid ventricular segments of left ventricle. The peak myocardial velocity gradient was significantly lower in the hypertensive group than in the control group for all regions. The peak endocardial velocity during early diastole in the mid ventricular septum was significantly lower in the hypertensive group (Hypertensive vs Controls; 3.8 ± 1.3 vs 5.1 ± 1.6 cm/s, P < 0.05), whereas the peak endocardial velocity at the other three sites were similar in the two groups. The peak dilating velocity was significantly lower in the hypertensive group only in the mid portion of the left ventricle (Hypertensive vs Controls; 7.2 ± 2.4 vs 9.8 ± 1.3 s⁻¹, P < 0.005). These results suggest that there were regional wall motion abnormalities and nonuniformity during the early diastolic phase in the hypertensive hearts with left ventricular hypertrophy.

Introduction

Left ventricular diastolic dysfunction plays an important role in the development of congestive heart failure in hypertensive patients.[1–2] Doppler echocardiographic studies have demonstrated impaired diastolic filling of the left ventricle in hypertensive patients at rest[3–4] and during sympathomimetic stress.[5] Recent studies emphasize that diastolic nonuniformity of left ventricular wall motion contributes to diastolic dysfunction.[6–7]

Tissue Doppler imaging (TDI) is a new ultrasonic modality that allows the quantification of myocardial velocities.[8–10] Color-coded M-mode TDI enables the measurement of regional endocardial and epicardial velocities and provides transmural myocardial velocity gradient (MVG) with excellent temporal resolution. Therefore, we determined whether the synchronous left ventricular wall motion occurs in hypertensive hearts with using TDI.

Materials and Methods

Patients

We studied 12 patients (age: 57 ± 6 years, 8 men and 4 women) with essential hypertension (LVH group), who had a systolic blood pressure > 140 mm Hg and a diastolic blood pressure > 90 mm Hg on three consecutive visits over 4-week screening period, and

demonstrated left ventricular hypertrophy (LVH). LVH was defined by both the thickness of the ventricular septum and the posterior wall of the left ventricle > 12 mm at end diastole by way of standards recommended by the American Society of Echocardiography.[11] All patients were in regular sinus rhythm and in New York Heart Association cardiac functional class I. Patients with significant valvular disease, asymmetric septal hypertrophy, episode of congestive heart failure, renal dysfunction, or thyroid disease were excluded from this study. All patients were receiving one or more antihypertensive drugs. Medications included calcium-channel antagonists, angiotensin converting enzyme inhibitors, or alpha-channel blockers, but no patients received any drugs with inotropic properties, such as beta-adrenergic blockers or stimulants, digitalis glycosides, xanthine derivatives, or diuretics at the time of the study. The control group consisted of 12 healthy volunteers (age: 54 ± 6 years, 9 men and 3 women). All were free from illness based on history, physical examination, the standard 12-lead electrocardiogram, and conventional echocardiography. Informed consent was obtained from each participant before the study.

Conventional Echocardiography

Conventional echocardiography was performed with the patients in the left lateral position. A commercially available phased array imaging system (SSA 380A, Toshiba, Tokyo) with Doppler capability was used. The wall thickness and the cavity dimension were measured based on the recommendation of the American Society of Echocardiography.[11] Left ventricular mass was calculated using the formula proposed by Deveroux and Reichek: [left ventricular mass (g)] =1.04 x {([end-diastolic dimension (cm)] + [thickness of the ventricular septum (cm)] + [thickness of the left ventricular posterior wall (cm)])^3-[end-diastolic dimension (cm)]^3}-13.6.[12] Left ventricular mass index (g/m^2) was determined as the left ventricular mass divided by the body surface area (m^2). Fractional shortening (%) was calculated as 100 x {([end-diastolic dimension]-[end-systolic dimension]) / [end-diastolic dimension]}.

The transmitral flow velocity was determined by pulsed-Doppler echocardiography, setting the sample volume at the center of the mitral annulus, using the apical four-chamber image as a guide. The peak early and the peak late filling velocities (E and A) were measured and the E/A ratio calculated. The isovolumic relaxation time was determined as the interval between the aortic valve closure to the onset of trans-

mitral flow based on the continuous wave Doppler recording from the cardiac apex.[13]

Myocardial velocity measurement

After obtaining the conventional echocardiographic recording, the TDI measurements were performed with a 3.75 MHz transducer. We used a velocity range for TDI recording between ±5.8 cm/s and ±14.9 cm/s to set the maximum velocity as low as possible without allowing aliasing to occur. The M-mode echocardiograms for the conventional and TDI recordings were obtained simultaneously. The parasternal recordings were performed with the cursor placed perpendicular to the ventricular wall at the basal segment and the mid-portion of the left ventricle. The lead II electrocardiogram was also recorded simultaneously.

Off-line analysis of the data was performed with analytical software (Heart ver. 1.4.7, Toshiba, Tokyo). The color intensity of the TDI signal was converted to numeric values for one cardiac cycle. The endocardial velocities (EV) of the ventricular septum and the posterior wall during early diastole were measured (Fig. 1AB). The dilating velocity (DV) during early diastole was defined as the value of the septal wall endocardial velocity minus the posterior wall endocardial velocity (Fig. 1C). The myocardial velocity gradient (MVG) was also determined as the slope of the linear regression line calculated from the velocity determined throughout the thickness of the ventricular wall. The peak values of the endocardial velocity (peak EV) and the MVG (peak MVG) were obtained at four sites (basal septum, basal posterior wall, the mid-portion of the septum, and the mid-portion of the posterior wall). The peak DV was determined at the base and the mid-portion of the left ventricle. The measurements are reported as the mean values of three consecutive beats.

Statistical analysis

Values are expressed as the mean ± SD. Differences between two values were compared by the Student's *t* test. A p value < 0.05 was considered statistically significant.

Results

Patient characteristics

The patients characteristics and the values obtained by conventional echocardiography are summarized in Table 1. There were no significant differ-
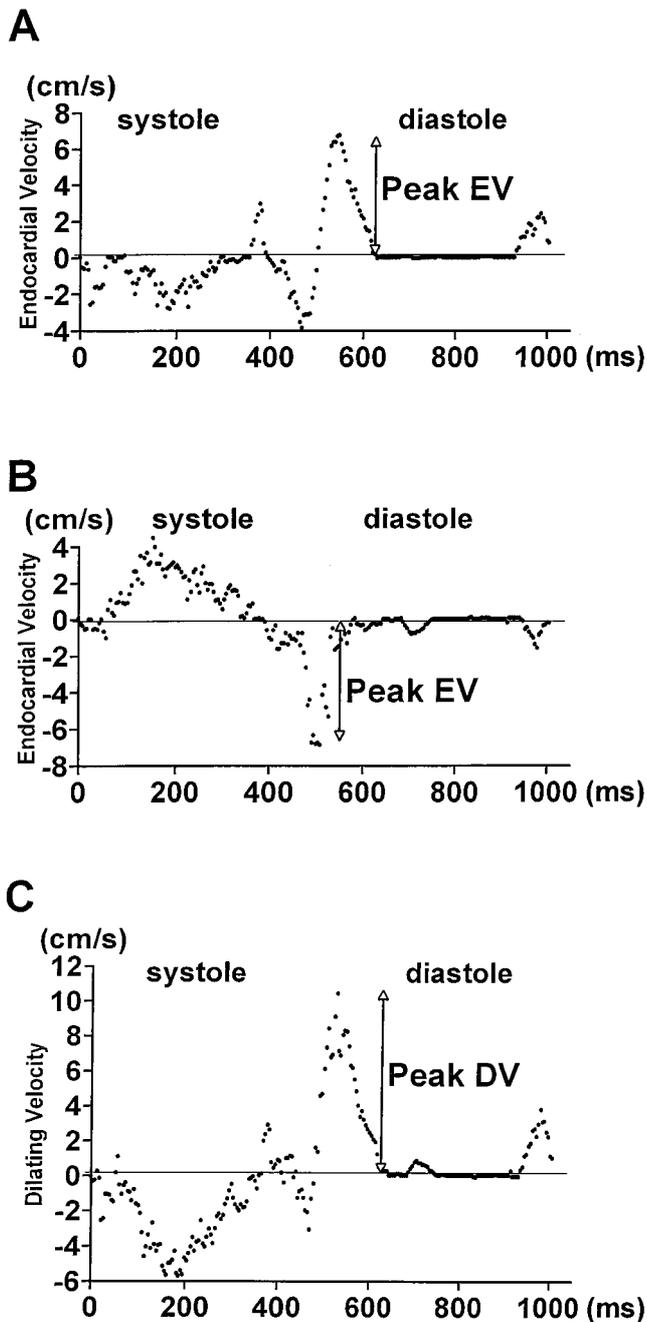
A

B

C

Figure 1. Examples of measurements of peak endocardial velocity (A, septal wall, B, posterior wall) and peak dilating velocity (C) in early diastolic phase. The transverse axis indicates time from onset of electrocardiographic QRS complex.

ences in age, heart rate, left ventricular end-diastolic dimension, or fractional shortening between the groups. All patients had a left ventricular mass index > 150 g/m². The E/A was significantly lower in the LVH group, whereas the isovolumic relaxation time was similar in the two groups.

Table 1. Patients Characteristics and Conventional Echocardiographic Data

	LVH group (n=12)	Control group (n=12)	p value
Age (yr)	57± 6	54± 6	NS
HR (bpm)	65± 8	67± 11	NS
SBP (mm Hg)	152± 11	118± 33	< 0.001
DBP (mm Hg)	90± 8	75± 7	< 0.001
VSth (mm)	16± 2	10± 1	< 0.001
PWth (mm)	16± 2	10± 1	< 0.001
LVMI (g/m2)	200± 33	106± 19	< 0.001
LVDd (mm)	46± 4	48± 4	NS
FS (%)	43± 8	40± 7	NS
E/A	0.8± 0.2	1.0± 0.2	< 0.05
IRT (ms)	91± 14	82± 14	NS

Data are expressed as the mean± SD.
DBP, diastolic blood pressure; E/A, ratio of peak early to late filling velocity; FS, fractional shortening; HR, heart rate; IRT, isovolumic relaxation time; LVDd, end-diastolic left ventricular dimension; LVMI, left ventricular mass index; NS, not significant; Pwth, end-diastolic left ventricular posterior wall thickness; SBP, systolic blood pressure; VSth, end-diastolic ventricular septal wall thickness.

Myocardial velocity measurement

Results of the myocardial velocity measurements are summarized in Table 2. The peak EV at the mid-portion of the ventricular septum in the LVH group was lower than in the control group. However, the peak EV at the other three sites were similar in the two groups. The peak DV at the mid-ventricular level was significantly lower in the LVH group, whereas the peak DV in the basal ventricle was not different from that in the control group (Fig. 2). The peak MVG was significantly lower in the LVH group than in the control group at all four sites.

Discussion

The importance of left ventricular diastolic dysfunction in the genesis of the pathophysiology of congestive heart failure has been recognized.[14–15] Left ventricular hypertrophy is often accompanied by abnormalities in relaxation and filling of the left ventricle. Conventional Doppler echocardiographic indices provide noninvasive measures of diastolic function (e.g., the prolonged isovolumic relaxation time indicates abnormal relaxation and the decreased E/A indicates abnormal early filling in left ventricular hypertrophy).[2,4,16] The decreased E/A in the hypertrophic heart is usually described as a delayed decline in left ventricular pressure during the relaxation phase and decreased chamber compliance.[17] In the present study, patients group consists of hypertensive patients with left ven-
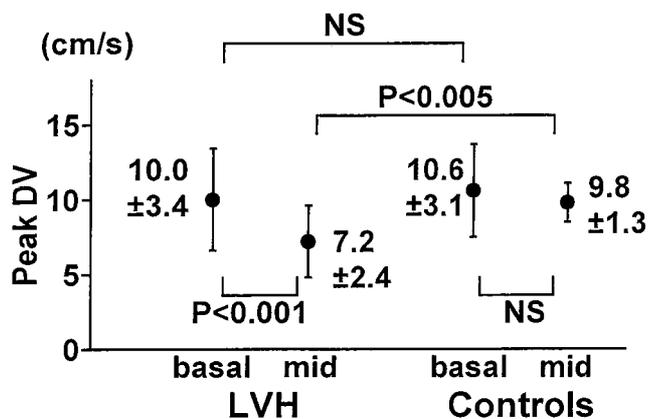
Figure 2. Differences between basal and mid left ventricular segments of peak dilating velocity in LVH group and control group. Data represent the mean value ± SD. NS, not significant.

Table 2. Tissue Doppler Imaging Data of Study Subjects

		LVH group (n=12)	Control group (n=12)	P value
Peak EV (cm/s)	basal VS	3.7± 1.3	3.8± 1.7	NS
	basal PW	7.5± 2.9	8.7± 1.6	NS
	mid VS	3.8± 1.3	5.1± 1.6	<0.05
	mid PW	5.9± 2.5	6.9± 1.0	NS
Peak MVG (S^{-1})	basal VS	1.1± 0.5	1.8± 0.8	<0.05
	basal PW	3.2± 1.8	4.7± 1.2	<0.05
	mid VS	1.2± 0.8	2.4± 1.2	<0.01
	mid PW	2.8± 1.5	4.1± 1.1	<0.05

Data are expressed as mean± SD. NS, not significant; Peak EV, peak endocardial velocity of left ventricular in early diastolic phase; Peak MVG, peak myocardial velocity gradient in early diastolic phase; PW, posterior wall; VS, vetricular septum.

tricular hypertrophy, and shows normal systolic function and abnormal diastolic filling properties.

Although the regional relaxation abnormality is an important determinant of global left ventricular diastolic dysfunction in coronary artery disease[18–20] and hypertrophic cardiomyopathy[7,21–22], the role of regional wall motion asynchrony on global diastolic function is still unclear in hypertensive hearts. In this study, the hypertensive group demonstrated that the peak endocardial velocity at the mid-septum was low and the mid-ventricular peak dilating velocity was attenuated, even in the setting of preserved basal ventricular motion. The regional impairment may also be an important cause of the global filling dysfunction seen in early stage of LVH. In contrast, the MVG decreased at all measured regions in the LVH group. Because endocardial velocities in the LVH group were comparable, or lower, than in the control group, a decreased myocardial velocity gradient could be explained as a reflection of the increased wall thickness. It has not been determined whether the decreased MVG itself plays a primary role in determining diastolic filling.

Although we studied wall motion at four only sites, the nonuniformity of abnormal wall motion in hypertensive patients with left ventricular hypertrophy was demonstrated with TDI. A discrepancy of regional endocardial velocity and TDI derived left ventricular dilating velocity may be new indices for diastolic dysfunction in hypertensive hearts. Although a left ventricular geometric change is considered to be a major determinant of regional wall motion abnormality,[21] no significant correlation demonstrated between the endocardial velocity and the mid-septal wall thickness in our small number of patients (data not indicated).

Further investigations are needed to clarify the cause of attenuated mid-ventricular wall motions in the patients with left ventricular hypertrophy.

References

1. Inoue I, Massie BM, Loge D, et al. Abnormal left ventricular filling: an early finding in moderate systemic hypertension. Am J Cardiol 1984;53:120-6.
2. Phillips RA, Coplan NL, Krakoff LR, et al. Pulsed Doppler echocardiographic analysis of left ventricular filling in treated hypertensive patients. J Am Coll Cardiol 1987;9:317-22.
3. Hartford M, Wikstrand J, Wallentin I, et al. Diastolic function of the heart in untreated primary hypertension. Hypertension 1984;6:329-38.
4. Pearson AC, Labovitz AJ, Mrosek D, et al. Assessment of diastolic function in normal and hypertrophied hearts: Comparison of Doppler echocardiography and M-mode echocardiography. Am Heart J 1987;113:1417-25.
5. Iwakami M, Hashimoto Y, Oniki T, et al. Effects of Dobutamine on left ventricular diastolic performance are attenuated in patients with systemic hypertension. Am J Cardiol 1996;78:369-72.
6. Philippe V, Victor MA, Lynn W, et al. Quantitative evaluation of global and regional left ventricular diastolic function with color kinesis. Circulation 1998;97:1053-61.
7. De Marchi SF, Allemann Y, Seiler C. Relaxation in hypertrophic cardiomyopathy and hypertensive heart disease: relation between hypertrophy and diastolic function. Heart 2000;83:78-84.
8. Sutherland GR, Steward MJ, Grounstroem KWE, et al. Color Doppler myocardial imaging: a new technique for the assessment of myocardial function. J Am Soc Echocardiogr 1994;7:441-58.
9. Miyatake K, Yamagishi M, Tanaka N, et al. A new method for the evaluation of left ventricular wall motion by color-coded tissue Doppler imaging: in vitro and in vivo studies. J Am Coll Cardiol 1995;25:717-24.
10. Trambaiolo P, Tonti G, Salustri A, et al. New insights into regional systolic and diastolic left ventricular function with tissue Doppler echocardiography: From qualitative anylysis to a quantitative approach. J Am Soc Echocardiogr 2001; 14: 85-96.

11. Sahn DJ, DeMaria A, Kisslo J, et al. Recommendations regarding quantitation in M-mode echocardiography: results of a survey of echocardiographic measurements. Circulation 1978;58:1072-83.

12. Devereux RB, Reichek N. Echocardiographic determination of left ventricular mass in man: anatomic validation of the method. Circulation 1977;55:613-8.

13. Lee CH, Vancheri F, Josen MS, et al. Discrepancies in the measurement of isovolumic relaxation time: a study comparing M mode and Doppler echocardiography. Br Heart J 1990;64:214-8

14. Dougherty AH, Maccarelli GV, Gray EL, et al. Congestive heart failure with normal systolic function. Am J Cardiol 1984; 54:778-82.

15. Soufer R, Wohlgelernter D, Vita NA, et al. Intact systolic left ventricular function in congestive heart failure. Circulation 1985; 55:1032-6.

16. Smith VE, White WB, Meeran MK, et al. Improved left ventricular filling accompanies reduced left ventricular mass during therapy of essential hypertension. J Am Coll Cardiol 1986;8:1449-54.

17. Nishimura RA, Abel MD, Hatle LK, et al. Assessment of diastolic function of the heart: Background and current applications of Doppler echocardiography. Part II. Clinical studies. Mayo Clin Proc 1989; 64:181-204.

18. Koh TW, Carr-White GS, Desouza AC, et al. Effect of coronary occulsion on left ventricular function with and without collateral supply during beating heart coronary artery surgery. Heart 1999; 81:285-92.

19. Vignon P, Mor-Avi V, Weinert L, et al. Quantitative evaluation of global and regional left ventricular diastolic function with color kinesis. Circulation 1998; 97: 1053-61.

20. Duval-Moulin AM, Dupouy P, Brun P, et al. Alteration of left ventricular diastolic function during coronary angioplasty-induced ischemia: a color M-mode Doppler study. J Am Coll Cardiol 1997; 29: 1246-55.

21. Oki T, Mishiro Y, Tabata T, et al. Detection of left ventricular regional relaxation abnormalities and asynchrony in patients with hypertrophic cardiomyopathy with the use of tissue Doppler imaging. Am Heart J 2000;139: 497-502.

22. Severino S, Caso P, Galderisi M, et al. Use of pulsed Doppler tissue imaging to assess regional left ventricular diastolic dysfunction in hypertrophic cardiomyopathy. Am J Cardiol 1998; 82: 1394-8.