Original Article

Impaired Left Ventricular Systolic Synchronicity in Hypertensive Patients with Ventricular Arrhythmias

Hong-Wei TAN[1),2)], Li LI[1),2)], Zhi-Hao WANG[1),2)], Shan-Shan XING[1),2)], Xiu-Ping BI[1),2)], Ming ZHONG[1),2)], and Wei ZHANG[1),2)]

Left ventricular (LV) systolic synchronicity is impaired in hypertensive patients. Ventricular arrhythmias often co-exist in hypertensive patients; hypertension and ventricular arrhythmias have an adverse impact on cardiac function. However, the influence of ventricular arrhythmias on LV synchronicity was not clear. The objective of the present study was to investigate the influence of ventricular arrhythmias on LV synchronicity in hypertensive patients. Tissue Doppler imaging (TDI) was performed in 136 subjects. Group 1 consisted of 74 hypertensives without any arrhythmias; group 2 consisted of 30 hypertensive patients with ventricular arrhythmias; and the control group consisted of 32 normal subjects. Using three apical views, LV synchronicity was assessed by the maximal differences in time to peak myocardial systolic contraction (T_s) and early diastolic relaxation (T_e) between any two of the LV segments (T_s-max, T_e-max) and the standard deviation of T_s (T_s-SD) and T_e (T_e-SD) of all 12 segments. T_s-max was significantly prolonged in group 2 compared with group 1 and the control group (93.70±20.97 ms *vs.* 79.48±25.46 ms [$p<0.01$] or 53.83±15.42 ms [$p<0.001$], respectively). T_s-SD was also significantly prolonged in group 2 compared with group 1 and the control group (38.16±5.82 ms *vs.* 33.37±6.04 ms [$p<0.05$] or 24.01±3.58 ms [$p<0.001$], respectively). In conclusion, LV systolic synchronicity was impaired in hypertensive patients with ventricular arrhythmias, and TDI was shown to be useful for the detection of myocardial abnormalities in such patients. (*Hypertens Res* 2007; 30: 759–766)

***Key Words*: hypertension, synchronicity, tissue Doppler imaging, ventricular arrhythmias**

Introduction

Several lines of evidence from experimental and clinical studies suggest that hypertension has an adverse impact on the heart (*1–3*). The presence of hypertension may affect cardiac function not only by impairing myocardial function (*4*, *5*), but also by disturbing the coordination between regions of the ventricle, resulting in ineffective contractions (*6*). Hypertension and ventricular arrhythmias coexist in many patients (*7*). It is well known that left ventricular (LV) hypertrophy is associated with an increased incidence of ventricular arrhythmia and sudden cardiac death (*8*). Many studies have demonstrated that ventricular arrhythmias may significantly reduce LV performance (*9*, *10*).

LV systolic synchronicity is an important factor in the determination of LV systolic function. Correcting the asynchrony by biventricular pacing can improve LV function in patients with congestive heart failure (*11*). Recently, Kosmala *et al*. (*6*), based on the apical views used to assess the long-axis movement, found that LV systolic and diastolic synchronicity were impaired in hypertensive patients. LV hypertro-

From the [1)]Key Laboratory of Cardiovascular Remodeling and Function Research, Chinese Ministry of Education and Chinese Ministry of Health, Jinan, P.R. China; and [2)]Department of Cardiology, QiLu Hospital, Shandong University, Jinan, P.R. China.

Address for Reprints: Wei Zhang, M.D., Ming Zhong, M.D., #107 West Wenhua Road, Department of Cardiology, QiLu Hospital, Shandong University, Jinan 250012, P.R. China. E-mail: zhangwei7@medmail.com.cn; zhongming2@medmail.com.cn

Received November 17, 2006; Accepted in revised form March 16, 2007.

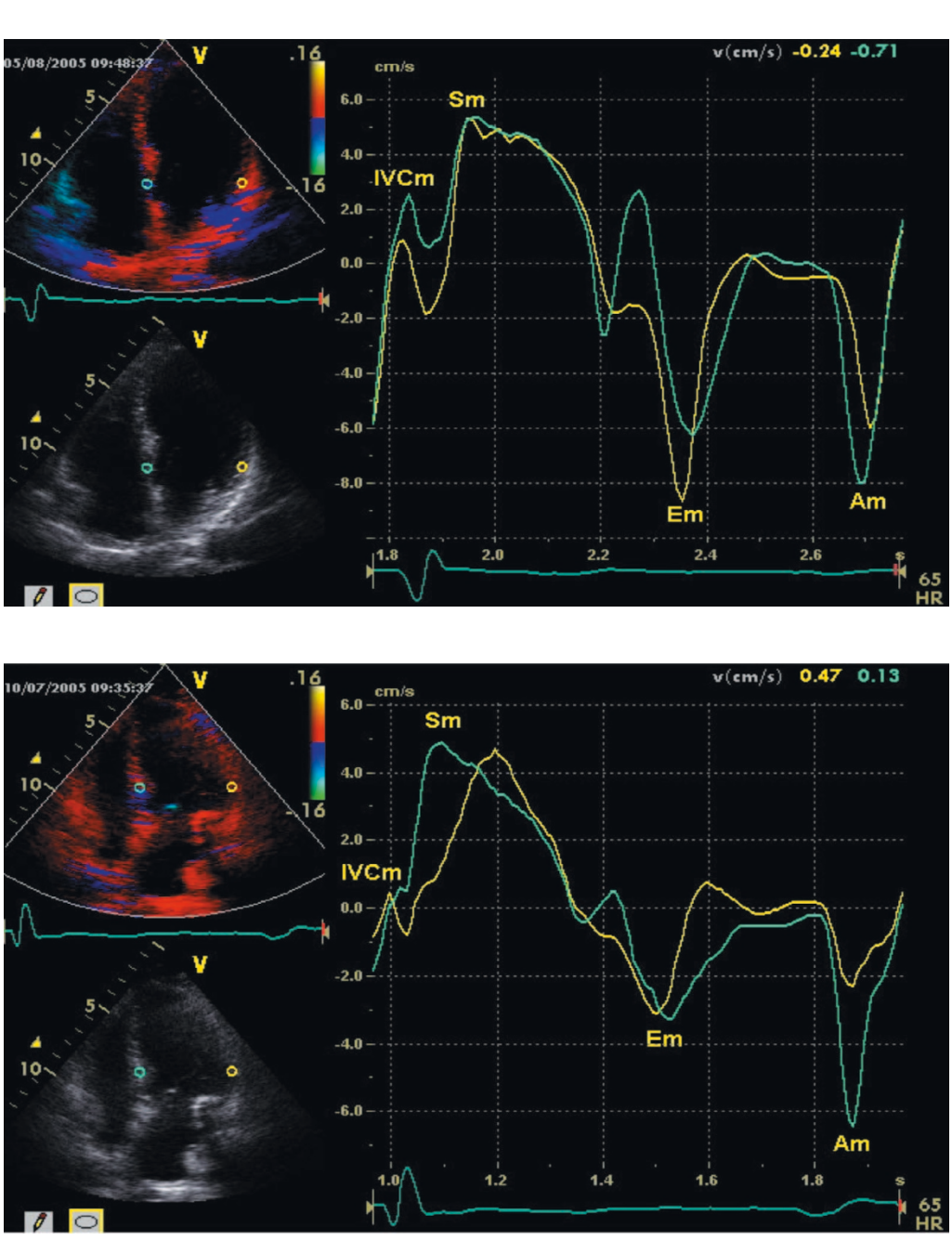

Fig. 1. *A: Tissue Doppler myocardial velocity curves obtained from the basal septal (green line) and basal lateral (yellow line) segments at the apical four chamber view in a healthy subject. Note that systolic motions in the left ventricular basal segments are highly synchronized, which is reflected by the exact overlap of the two curves. B: Tissue Doppler myocardial velocity curves obtained from the basal septal (green line) and basal lateral (yellow line) segments at the apical four chamber view in a hypertensive patient with ventricular arrhythmias. Note that there is an obvious delay in T_s in the lateral segment; T_s-max is 115 ms.*

phy is very common in hypertensives and is associated with the occurrence of ventricular arrhythmia. We therefore considered that it would be of great interest to study LV synchronicity in hypertensive patients with ventricular arrhythmias.

Tissue Doppler imaging (TDI) is a new echocardiographic technique that uses high amplitude, low frequency ultrasound signals reflected from the myocardium. TDI allows the simultaneous examination of several regional cardiac structure movements with an excellent time resolution of approximately 10 ms (*12*), and has been used to evaluate ventricular synchronicity in both healthy (*13*) and diseased (*6*, *12*, *14*) hearts.

Table 1. Clinical Characteristics of Studied Population

	Control group (n=32)	Group 1 (n=74)	Group 2 (n=30)
Age (years)	46±10	49±10	53±8
Men (%)	50	59	70
HR (beats/min)	71±11	73±16	71±14
QRS duration (ms)	99±10	102±13	100±14
SBP (mmHg)	116±11	148±23***	142±17***
DBP (mmHg)	75±5	93±16***	87±11***,†
Duration of hypertension (months)	—	93±90	92±88
BMI (kg/m^2)	24.18±2.64	25.60±3.38*	26.12±2.98*
LVMI (g/m^2)	94.88±19.44	117.09±32.17**	128.09±41.47**
Hypertension treatment (n (%))			
Diuretics	—	25 (34)	11 (34)
β-Blockers	—	14 (19)	10 (33)
CCBs	—	19 (26)	9 (30)
ACEI	—	23 (31)	14 (47)
ARB	—	5 (7)	5 (17)
Duration of ventricular arrhythmias (months)	—	—	20±11
Numbers of ventricular arrhythmias in Holter monitor (beats)	—	—	4,676±586
Anti-arrhythmia therapy (n (%))	—	—	4 (13)

HR, heart rate; BMI, body mass index, LVMI, left ventricular mass index; CCBs, calcium channel blockers; ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blockers. *p<0.05 compared with control group; **p<0.01 compared with control group; ***p<0.001 compared with control group; †p<0.05 compared with group 1.

Table 2. Two-Dimensional, M-Mode, Pulsed Doppler Echocardiographic Parameters in All Patients Groups and the Controls

	Control group	Group 1	Group 2
LVDd (mm)	46.04±4.12	43.42±8.01	47.38±7.52
LVPWd (mm)	9.88±0.83	11.19±2.26	11.46±2.46
IVSd (mm)	10.36±1.14	11.96±2.50*	12.53±3.34*
EF (%)	69.58±5.11	69.11±10.52	68.28±14.31
E (cm/s)	78.86±15.69	70.38±25.11	77.31±24.83
A (cm/s)	61.54±14.39	78.99±26.75**	83.26±23.84**
E/*A*	1.34±0.38	0.93±0.31***	0.96±0.36***

LVDd, left ventricular diameter in end diastole; LVPWd, left ventricular posterior wall thickness in end diastole; IVSd, interventricular septum; EF, ejection fraction; *E*, maximum amplitudes of the early systolic wave; *A*, maximum amplitudes of the late diastolic wave. *p<0.05 compared with control group; **p<0.01 compared with control group; ***p<0.001 compared with control group.

In the present study, we hypothesized that LV synchronicity is impaired in hypertensive patients with ventricular arrhythmias, and tested this hypothesis by using TDI to study the regional synchronicity of LV.

Methods

Patients

One-hundred and four consecutive patients with mild-to-moderate hypertension (60% males) who visited our hospital from April 2005 to November 2005 were enrolled in this study. Inclusion criteria were as follows: outpatients of either sex, aged from 18 to 65 years, with mild-to-moderate hypertension (diastolic blood pressure [DBP] >90 mmHg and <110 mmHg; systolic blood pressure [SBP] >140 mmHg and <180 mmHg). Patients with secondary hypertension, dilated cardiomyopathy, left bundle branch block, right bundle branch block, atrioventricular block or pre-excitation syndrome on ECG, evident myocardial ischemia (positive findings on treadmill exercise testing), major cardiovascular complications (myocardial infarction or unstable angina within 6 months, congestive heart failure), diabetes, liver or kidney disease, and cancer were excluded from the study. The selected patients were divided into two groups: group 1 consisted of 74 patients (44 males and 30 females) who had hypertension without any arrhythmias (as proved by ECG or Holter monitor), and group 2 consisted of 30 patients (21

Table 3. Comparison of TDI Parameters in All Patients Groups and the Controls

	S_m (cm/s)			E_m (cm/s)		
	Controls	Group 1	Group 2	Controls	Group 1	Group 2
Basal septum	5.42±0.90	5.16±1.42	5.36±1.40	6.89±1.79	4.87±2.02***	4.88±1.66***
Mid septum	3.57±2.78	3.46±1.21	4.30±1.75*,††	5.52±1.83	4.24±1.82***	4.54±1.49*
Basal lateral	6.68±1.82	5.83±2.30*	4.94±1.88**,†	8.47±1.89	6.71±3.18**	5.47±2.22***,†
Mid lateral	4.76±2.27	4.43±2.17	3.37±1.68**,†	5.16±2.22	4.19±2.30*	3.04±1.41***,†
Basal anteroseptal	4.59±1.60	4.43±1.69	4.41±2.00	5.19±1.69	4.01±1.84**	3.76±2.05**
Mid anteroseptal	2.82±1.27	2.67±1.45	3.13±1.78	3.93±1.91	2.84±1.73**	2.63±1.48**
Basal posterior	6.56±1.68	6.03±2.20	5.07±2.50**,†	8.34±2.85	5.75±3.07***	4.74±2.98***
Mid posterior	4.32±1.81	3.82±2.15	3.11±1.73*	5.10±2.15	3.69±2.10**	2.72±1.80***,†
Basal anterior	5.36±2.22	4.85±2.26	4.66±1.58	6.14±2.49	4.68±2.70**	4.29±1.71**
Mid anterior	3.16±2.01	2.97±1.66	2.94±1.36	3.67±2.38	3.07±1.93	2.89±1.56
Basal inferior	5.84±1.94	5.50±1.77	5.74±1.87	7.47±2.94	5.05±2.76***	5.16±2.70**
Mid inferior	3.69±1.29	3.11±1.22*	3.47±1.22	4.98±1.99	3.33±1.82***	3.55±1.56**

TDI, tissue Doppler imaging; S_m, peak velocity during sustained systole; E_m, peak velocity during early diastole. *$p<0.05$, **$p<0.01$, ***$p<0.001$ compared with control group. †$p<0.05$, ††$p<0.01$ compared with group 1.

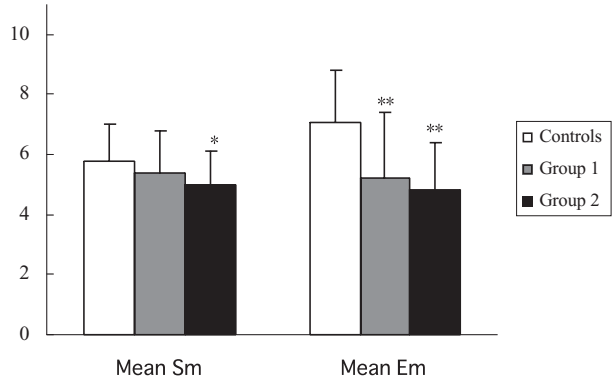

Fig. 2. *Mean S_m (cm/s) and mean E_m (cm/s) of six basal segments in the patient groups and controls. *$p<0.05$ compared with the control group; **$p<0.001$ compared with the control group.*

males and 9 females) who had hypertension and ventricular arrhythmias. In order to evaluate the influence of ventricular arrhythmias on LV synchronicity, we used the following criteria: frequent premature ventricular contractions (>10/min on ECG (*9*) or >1,440/24 h by Holter monitor (*15*)) or non-sustained ventricular tachycardia as proved by ECG or Holter monitoring; a history of ventricular arrhythmias of more than 3 months; and an absence of arrhythmias at the time of echocardiographic examination. If ventricular arrhythmias were found on the ECG, then an additional ECG was performed on another day to confirm them. Ventricular arrhythmias were identified in 8 patients by ECG and in 22 patients by Holter monitoring. The control group consisted of 32 healthy subjects (16 males and 16 females) without a history of cardiac disease or systemic hypertension and having normal findings on physical examination, chest roentgenography, Holter monitor, and echocardiography (Holter monitoring was performed in all control subjects to exclude arrhythmias). Informed consent was obtained from all subjects based on a protocol approved by the Ethics Committee of QiLu Hospital, Shandong University.

Two-Dimensional, M-mode, Pulsed Doppler Echocardiography

All the subjects were examined in the left lateral decubitus using a System Seven digital ultrasound machine (GE Vingemed Ultrasound, Horten, Norway) with a 2.5- or 3.5-MHz phased array transducer. One technician operated the machine with no prior information about the study. Measurements of LV diameter in end diastole (LVDd), LV diameter in end systole (LVDs), interventricular septum (IVSd) and LV posterior wall thickness in end diastole (LVPWd) were made from M-mode readings according to the recommendations of the American Society of Echocardiography (*16*). LV mass (LVM) was calculated using the Devereux-modified American Society of Echocardiography (ASE) cube formula (*17*), and the LV mass index (LVMI) was obtained by dividing the LVM by the body surface area. The LV ejection fraction (EF) was estimated by a modified version of Simpson's biplane method. For the pulsed Doppler echocardiographic studies, the sample volume was placed between the anterior and posterior mitral leaflet tips to record the LV inflow patterns, and the maximum amplitudes of the early diastolic wave (*E*) and late diastolic wave (*A*) were estimated; the *E*/*A* ratios were then calculated.

Tissue Doppler Echocardiography

TDI was performed to assess the long-axis function of the LV using the apical four-chamber, two-chamber, and long-axis

Table 4. Comparison of LV Synchronicity in All Patients Groups and the Controls

	T_s (ms)			T_e (ms)		
	Controls	Group 1	Group 2	Controls	Group 1	Group 2
Basal septum	142±22	148±24	152±38	517±39	523±42	535±40
Mid septum	142±23	151±30	150±38	535±41	534±44	549±41
Basal lateral	139±23	162±42**	181±43***,†	513±39	511±44	527±39
Mid lateral	139±27	158±45*	178±48***,†	531±42	523±45	535±36
Basal anteroseptal	136±24	143±26	148±34	527±38	532±36	539±40
Mid anteroseptal	135±26	144±33	153±35*	538±41	535±40	536±42
Basal posterior	152±28	176±35**	186±41***	511±35	514±45	545±39
Mid posterior	152±29	173±37**	186±44**	535±39	534±41	544±40
Basal anterior	130±16	143±28	143±26	524±34	526±40	543±42
Mid anterior	131±21	144±34	146±32	533±45	534±39	547±42
Basal inferior	156±24	176±31**	172±38*	510±38	519±37	526±39
Mid inferior	159±25	176±34*	177±40*	532±41	539±37	549±50

LV, left ventricular; T_s, time to peak myocardial systolic contraction; T_e, time to peak early diastolic relaxation. *$p<0.05$, **$p<0.01$, ***$p<0.001$ compared with control group; †$p<0.05$ compared with group 1.

views (*13*, *18*). A 12-segement model was used to assess regional LV function. This included the anterior, anteroseptal, septum, inferior, posterior, and lateral segments at the basal level and mid-level of the ventricle, respectively (*6*, *13*, *18*). Tissue Doppler velocity data were recorded at frame rate close to 100–140 Hz. Gain settings, filters and pulse repetitive frequency were adjusted to optimize the color saturation. At least three consecutive beats were stored and analyzed offline.

From the recorded images, we measured peak velocities during isovolumic contraction (IVCm), sustained systole (S_m), early diastole (E_m) and late diastole (A_m). For the measurement of timing, the beginning of the QRS complex obtained from the surface ECG was used as the reference point, and the times to IVCm (T_{ivc}), S_m (T_s), E_m (T_e), and A_m (T_a) were quantified (*13*, *18*). For assessment of the systolic and diastolic synchronicity of longitudinal myocardial function, the standard deviation of T_s (T_s-SD) and T_e (T_e-SD) of all 12 segments and the maximal difference in T_s and T_e between any two of the LV segments (T_s-max, T_e-max) were calculated. To assess global cardiac function, the mean S_m and mean E_m from the six basal segments were calculated (*6*, *18*). For the six basal segments, the E_m/A_m ratio was also calculated (Fig. 1).

Statistical Analysis

Values are expressed as the mean±SD. Student's two-tailed *t*-test, χ^2 test, and analysis of variance were used where appropriate. Pearson correlation coefficients were used to test correlations between the parameters of LV systolic synchronicity and age, heart rate, blood pressure, number and duration of ventricular arrhythmias, LV hypertrophy and color Doppler parameters. A receiver operating characteristic (ROC) curve was used to assess the diagnostic efficacy of T_s-max. All statistical analysis were performed using software from SPSS Inc., and the differences were considered significant at $p<0.05$.

Results

The mean duration of ventricular arrhythmia in group 2 was 20 months. In group 2, 24 patients had frequent premature ventricular contractions, and 6 had nonsustained ventricular tachycardia. The mean number of ventricular arrhythmias in patients who underwent Holter monitoring was 4,676 beats.

There were no significant differences in age, gender, heart rate among the disease groups and controls. Patients in group 1 and group 2 had significantly higher SBP and DBP, a higher prevalence of LV hypertrophy, and a higher rate of antihypertensive drug use than the control group, but there were no significant differences in any of these parameters between groups 1 and 2 (Table 1).

As to the conventional Doppler parameters, IVSd was significantly higher in group 1 and group 2 than in the controls, whereas EF, fractional shortening (FS) and other parameters of LV dimension showed no difference among the three groups. *E* and *E*/*A* were significantly lower in group 1 and group 2 than the control group, but there was no significant difference in these parameters between group 1 and group 2 (Table 2).

Impairment of longitudinal systolic myocardial function as evidenced by decreased S_m and mean S_m was seen in both patient groups, and was most advanced in group 2 (mean S_m: 5.02±1.07 cm/s in group 2 compared with 5.76±1.28 cm/s in the controls; $p<0.05$). Like systolic dysfunction, longitudinal diastolic dysfunction was most prevalent in group 2, as expressed by decreased values of TDI E_m and mean E_m (mean E_m: 4.81±1.60 cm/s in group 2 and 5.24±1.25 cm/s in group 1 compared with 7.08±1.74 cm/s in the controls, all

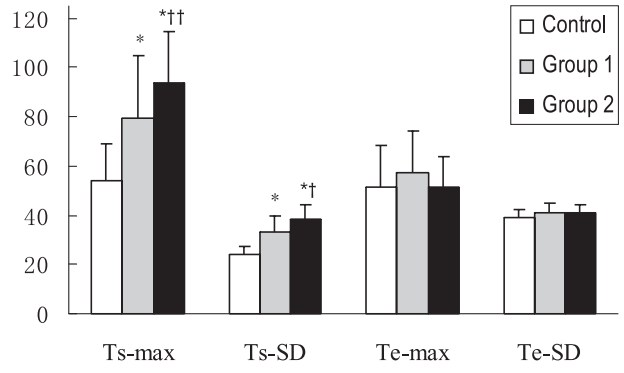

Fig. 3. *T_s-max (ms), T_e-max (ms), T_s-SD (ms) and T_e-SD (ms) in the patient groups and controls. *$p<0.001$ compared with the control group; †$p<0.05$, ††$p<0.01$ compared with group 1.*

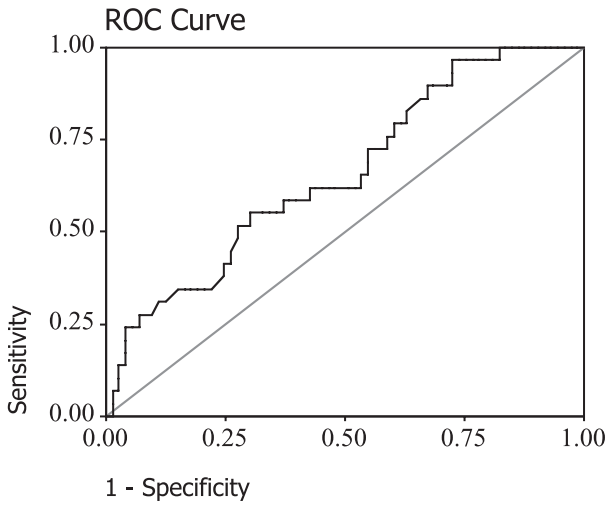

Fig. 4. *ROC curve used for the definition of the cut value of T_s-max that best characterizes the hypertensive patients with ventricular arrhythmias.*

$p<0.001$) (Table 3, Fig. 2). E_m/A_m was significantly decreased in group 1 and group 2 compared with the controls (0.92±0.44 for group 1 and 0.87±0.45 for group 2 *vs*. 1.41±0.68 for the controls; $p<0.0001$ for both comparisons.)

Systolic synchronicity, as measured by T_s, T_s-SD, and T_s-max, was impaired in the patient groups compared with the controls, and the impairment was more serious in group 2 (T_s-SD: 38.16±5.82 ms in group 2 compared with 33.37±6.04 ms in group 1 and 24.01±3.58 ms in the control group, $p<0.05$, $p<0.001$, respectively, and 33.37±6.04 ms in group 1 compared with 24.01±3.58 ms in the control group, $p<0.001$; T_s-max: 93.70±20.97 ms in group 2 compared with 79.48±25.46 ms in group 1 and 53.83±15.42 ms in the control group, $p<0.01$, $p<0.001$, respectively, and 79.48±25.46 ms in group 1 compared with 53.83±15.42 ms in the control group, $p<0.001$). There were no significant difference in the T_e, T_e-max and T_e-SD among the three groups (Table 4, Fig. 3).

In group 2 patients, T_s-max correlated weakly with the duration of ventricular arrhythmia ($r=0.43$, $p<0.05$) and LVMI ($r=0.37$, $p<0.05$). In a sub-analysis of patients who underwent Holter monitoring, T_s-max was correlated with the number of ventricular arrhythmias ($r=0.59$, $p<0.01$). There were no other significant correlations between parameters of LV systolic synchronicity and age, heart rate, blood pressure, and color Doppler parameters.

The ability of T_s-max to detect hypertensive patients with ventricular arrhythmias was assessed by using an ROC curve. The area under the ROC curve was 0.658 (95% confidential interval, 0.543–0.772). A T_s-max value of 70 ms had 90% sensitivity and 33% specificity for detecting hypertensive patients with ventricular arrhythmia (Fig. 4).

Discussion

This study demonstrated that hypertensive patients with ventricular arrhythmias show clear evidence of impaired LV systolic synchronicity as demonstrated by TDI. In these patients, T_s in lateral LV segments, the maximal difference in T_s between any two of the LV segments, and T_s-SD were significantly prolonged compared to those of the control group and the isolated hypertensives. This indicates that TDI is a sensitive method of detection and evaluation of myocardial impairment in hypertensive patients with ventricular arrhythmias. To the best of our knowledge, this is the first study focused on ventricular synchronicity in such patients.

LV systolic function is sustained not only by contraction of the myocardium, but also by the synchronicity of LV. This has been proven by clinical studies in which correction of the LV asynchrony improved LV systolic function in heart failure patients (*11*, *19*). LV synchronicity is influenced by many heart diseases, including dilated cardiomyopathy, coronary heart disease (*14*), and diabetes mellitus (*6*). In a recent study, Kosmala *et al*. (*6*) demonstrated that LV synchronicity was impaired in hypertensive patients, and the impairment was more serious in the hypertensive patients with diabetes mellitus. With the improvements in echocardiographic technology, it has become possible to assess LV synchronicity in diseased hearts using TDI (*6*, *12*, *14*, *18*). TDI has been shown to be more reliable than three-dimensional tagged magnetic resonance imaging (*20*) and myocardial scintigraphy (*21*). Even in patients with normal QRS duration, the prevalence of LV asynchrony is relatively higher (*18*).

Clinical studies have indicated that depressed LV systolic function and impaired diastolic function often co-exist in hypertensive patients (*1*, *4*, *22*), and LV synchronicity is impaired in hypertensive patients (*6*). Hypertension and cardiac arrhythmias co-exist in many patients, and the most important promoter of ventricular arrhythmogenesis in hyper-

tensives is LV hypertrophy (*7*, *8*, *23*). Frequent premature ventricular contractions can reduce LV systolic function (*9*), and reducing the premature ventricular contractions can improve the symptoms of patients (*24*). We therefore considered that it would be of great interest to study the effect of ventricular arrhythmias on LV synchronicity.

The present study demonstrated that LV systolic function was impaired in hypertensive patients with ventricular arrhythmias, as demonstrated by the decreased mean S_m and S_m in the lateral and posterior segments of the LV. In the isolated hypertensive group, mean S_m was not significantly different from that of the control group, but S_m in the basal lateral and mid-inferior segments of the LV was significantly difference when compared with that of the control group, which indicated systolic function was impaired in hypertensives, in accordance with the previous studies (*6*, *25*). In this study, EF showed no difference among the three groups, which may have indicated that EF is not as sensitive for reflecting LV systolic dysfunction as S_m is. Support for such a conclusion comes from previous studies which showed that although EF was similar among different patient groups, S_m was different (*6*, *25*, *26*).

T_s-max and T_s-SD were also prolonged in hypertensive patients without ventricular arrhythmias when compared with the control group, indicated that LV synchronicity was impaired in such patients. However, the most valuable finding of this study was that LV synchronicity was impaired in hypertensive patients with ventricular arrhythmias, when compared with controls and isolated hypertensive patients. This is similar to the result of Kosmala's study (*6*). As to the diastolic function and diastolic synchronicity, it seems that ventricular arrhythmias only influence diastolic function, as indicated by the finding that E_m in LV segments was significantly different among the three groups, but there was no difference in LV diastolic synchronicity among the three groups.

It is well known that LV performance is depressed during premature ventricular contractions by loss of atrioventricular and ventricular synchronicity (*27*). Ventricular arrhythmias are not only associated with marked impairment of LV systolic function but also diastolic filling and relaxation (*10*). In hypertensive hearts, cardiac myocytes could change the width and morphology of the action potential, resulting in ventricular arrhythmias (*7*). In a more advanced stage, myocardial fibrosis is evident (*2*, *25*, *28*); these electrical and structural changes may be associates with impaired LV synchronicity. Other factors are thought to be responsible for the impaired LV synchronicity in hypertensive patients with ventricular arrhythmias. These include altered myocardial glucose metabolism (*29*), amplified transmural repolarization dispersion caused by LV hypertrophy (*30*), and long-term cardiac memory (*31*). The present study showed that T_s-max was positively correlated with the number and the duration of ventricular arrhythmias and LVMI, indicating the noxious effect of hypertension and ventricular arrhythmias on myocardial function, but the exact mechanism remains unclear, and requires further investigation. In our study, the value of T_s-max of 70 ms had 90% sensitivity and 33% specificity for detecting hypertensive patients with ventricular arrhythmia from those without ventricular arrhythmias; the relatively lower specificity may have been due to the small study population.

The information obtained from the apical views allows comparison of nearly all the segments of LV quantitatively in the same manner, except the apical segments where long-axis movement is minimal (*14*). The time to 'peak' rather than 'onset' of sustained systolic contraction was used to assess cardiac systolic synchronicity, because S_m indicates the moment of the maximal endocardial excursion, and it more truly reflects the degree of synchronous movement of various regions than the onset of contraction (*13*).

In conclusion, LV systolic synchronicity was impaired in hypertensive patients with ventricular arrhythmias. TDI is a sensitive and feasible method for the detection and evaluation of myocardial impairment in such patients. Future studies should focus on the influence of antihypertensive drugs and anti-arrhythmia therapy on the LV synchronicity in such patients.

References

1. Bella JN, Palmieri V, Liu JE, *et al*: Relationship between left ventricular diastolic relaxation and systolic function in hypertension: the Hypertension Genetic Epidemiology Network (HyperGEN) Study. *Hypertension* 2001; **38**: 424–428.
2. Maceira AM, Barba J, Varo N, Beloqui O, Diez J: Ultrasonic backscatter and serum marker of cardiac fibrosis in hypertensives. *Hypertension* 2002; **39**: 923–928.
3. Tsunoda S, Kawano Y, Horio T, Okuda N, Takishita S: Relationship between home blood pressure and longitudinal changes in target organ damage in treated hypertensive patients. *Hypertens Res* 2002; **25**: 167–173.
4. Tan HW, Li L, Zhang W, Ma ZY, Zhong XZ, Zhang, Y: Effect of cilnidipine on left ventricular function in hypertensive patients as assessed by tissue Doppler Tei index. *J Hum Hypertens* 2006; **20**: 618–624.
5. Lin M, Sumimoto T, Hiwada K: Left ventricular geometry and cardiac function in mild to moderate essential hypertension. *Hypertens Res* 1995; **18**: 151–157.
6. Kosmala W, Kucharski W, Przewlocka-Kosmala M, Mazurek W: Comparison of left ventricular function by tissue Doppler imaging in patients with diabetes mellitus without systemic hypertension *versus* diabetes mellitus with systemic hypertension. *Am J Cardiol* 2004; **94**: 395–399.
7. Lombardi F, Terranova P: Hypertension and concurrent arrhythmias. *Curr Pharm Des* 2003; **9**: 1703–1713.
8. Le Heuzey JY, Guize L: Cardiac prognosis in hypertensive patients. Incidence of sudden death and ventricular arrhythmias. *Am J Med* 1988; **84**: 65–68.
9. Sun Y, Blom NA, Yu Y, *et al*: The influence of premature ventricular contractions on left ventricular function in asymptomatic children without structural heart disease: an echocardiographic evaluation. *Int J Cardiovasc Imaging* 2003; **19**: 295–299.

10. Stoddard MF, Pearson AC, Kern MJ, Labovitz AJ: The effect of premature ventricular contraction on left ventricular relaxation, chamber stiffness, and filling in humans. *Am Heart J* 1989; **118**: 725–733.
11. Schuster P, Faerestrand S, Ohm OJ: Colour tissue velocity imaging can show resynchronisation of longitudinal left ventricular contraction pattern by biventricular pacing in patients with severe heart failure. *Heart* 2003; **89**: 859–864.
12. Schuster P, Faerestrand S, Ohm OJ: Color Doppler tissue velocity imaging can disclose systolic left ventricular asynchrony independent of the QRS morphology in patients with severe heart failure. *Pacing Clin Electrophysiol* 2004; **27**: 460–467.
13. Yu CM, Lin H, Ho PC, Yang H: Assessment of left and right ventricular systolic and diastolic synchronicity in normal subjects by tissue Doppler echocardiography and the effects of age and heart rate. *Echocardiography* 2003; **20**: 19–27.
14. Yu CM, Yang H, Lau CP, *et al*: Regional left ventricle mechanical asynchrony in patients with heart disease and normal QRS duration: implication for biventricular pacing therapy. *Pacing Clin Electrophysiol* 2003; **26**: 562–570.
15. Geelen A, Brouwer IA, Schouten EG, Maan AC, Katan MB, Zock PL: Effects of *n*-3 fatty acids from fish on premature ventricular complexes and heart rate in humans. *Am J Clin Nutr* 2005; **81**: 416–420.
16. Sahn DJ, DeMaria A, Kisslo J, Weyman A: Recommendations regarding quantitation in M-mode echocardiography: results of a survey of echocardiographic measurements. *Circulation* 1978; **58**: 1072–1083.
17. Devereux RB, Alonso DR, Lutas EM, *et al*: Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. *Am J Cardiol* 1986; **57**: 450–458.
18. Yu CM, Lin H, Zhang Q, Sanderson JE: High prevalence of left ventricular systolic and diastolic asynchrony in patients with congestive heart failure and normal QRS duration. *Heart* 2003; **89**: 54–60.
19. Penicka M, Bartunek J, De Bruyne B, *et al*: Improvement of left ventricular function after cardiac resynchronization therapy is predicted by tissue Doppler imaging echocardiography. *Circulation* 2004; **109**: 978–983.
20. Curry CW, Nelson GS, Wyman BT, *et al*: Mechanical dyssynchrony in dilated cardiomyopathy with intraventricular conduction delay as depicted by 3D tagged magnetic resonance imaging. *Circulation* 2000; **101**: E2.
21. Kerwin WF, Botvinick EH, O'Connell JW, *et al*: Ventricular contraction abnormalities in dilated cardiomyopathy: effect of biventricular pacing to correct interventricular dyssynchrony. *J Am Coll Cardiol* 2000; **35**: 1221–1227.
22. de Simone G, Greco R, Mureddu G, *et al*: Relation of left ventricular diastolic properties to systolic function in arterial hypertension. *Circulation* 2000; **101**: 152–157.
23. Hennersdorf MG, Strauer BE: Arterial hypertension and cardiac arrhythmias. *J Hypertens* 2001; **19**: 167–177.
24. Duffee DF, Shen WK, Smith HC: Suppression of frequent premature ventricular contractions and improvement of left ventricular function in patients with presumed idiopathic dilated cardiomyopathy. *Mayo Clin Proc* 1998; **73**: 430–403.
25. Yuda S, Short L, Leano R, Marwick TH: Myocardial abnormalities in hypertensive patients with normal and abnormal left ventricular filling: a study of ultrasound tissue characterization and strain. *Clin Sci* (*Lond*) 2002; **103**: 283–293.
26. Demir M, Paydas S, Cayli M, Akpinar O, Balal M, Acarturk E: Tissue Doppler is a more reliable method in early detection of cardiac dysfunction in patients with AA amyloidosis. *Ren Fail* 2005; **27**: 415–420.
27. Eber LM, Berkovits BV, Matloff JM, Gorlin R: Dynamic characterization of premature ventricular beats and ventricular tachycardias. *Am J Cardiol* 1974; **33**: 378–383.
28. Kai H, Kuwahara F, Tokuda K, Imaizumi T: Diastolic dysfunction in hypertensive hearts: roles of perivascular inflammation and reactive myocardial fibrosis. *Hypertens Res* 2005; **28**: 483–490.
29. Matsumoto K, Takahashi N, Ishikawa T, *et al*: Evaluation of myocardial glucose metabolism before and after recovery of myocardial function in patients with tachycardia-induced cardiomyopathy. *Pacing Clin Electrophysiol* 2006; **29**: 175–180.
30. Yan GX, Rials SJ, Wu Y, *et al*: Ventricular hypertrophy amplifies transmural repolarization dispersion and induces early afterdepolarization. *Am J Physiol Heart Circ Physiol* 2001: **281**: H1968–H1975.
31. Patberg KW, Rosen MR: Molecular determinants of cardiac memory and their regulation. *J Mol Cell Cardiol* 2004; **36**: 195–204.