

Early Detection of Left Ventricular Diastolic Dysfunction in Hypertensive Heart Disease by Color Doppler Myocardial Imaging

Jadranka Šeparović-Hanževački¹, Maja Čikeš¹, Martina Lovrić-Benčić¹, Zdenko Sonicki², Sanja Ceković¹, Aleksander Ernst¹, Nikša Drinković¹, Ivo Čikeš¹

¹Department of Cardiovascular Diseases, Zagreb University Hospital Center; and ²Andrija Štampar School of Public Health, Zagreb University School of Medicine, Zagreb, Croatia

- Aim** To determine if Color Doppler myocardial imaging could provide evidence of diastolic dysfunction in patients with hypertension whose pulse-wave Doppler parameters were normal.
- Method** The study included 33 patients (mean age 48 ± 7.3 years) and a control group of 13 sex- and age-matched healthy individuals. Patients were divided into two groups according to mean blood pressure (BP) values during 24-hour blood pressure monitoring while under antihypertensive therapy: those with uncontrolled hypertension ($n = 22$) and those with controlled hypertension ($n = 11$). All study participants underwent complete standard echocardiography (2D, M-mode, pulsed and continuous Doppler) and a Color Doppler myocardial imaging study.
- Results** Conventional Doppler parameters indicated relaxation disturbances in patients with uncontrolled hypertension, but were within a normal range in patients with controlled hypertension at baseline and follow-up. Parameters of global diastolic function measured by Color Doppler myocardial imaging revealed that E'/A' , the ratio between E' -wave (early filling phase) and A' -wave (late diastolic wave due to atrial contraction), was < 1 in 57% of segments at baseline in patients with uncontrolled hypertension, and did not significantly change at follow-up. In patients with controlled hypertension, $E'/A' < 1$ was noted in 4.7% of segments at baseline and in 28.6% of segments at follow-up.
- Conclusion** Regional diastolic dysfunction measured by Color Doppler myocardial imaging was the first sign of myocardial dysfunction due to arterial hypertension, while the parameters of global diastolic dysfunction measured by conventional Doppler and Color Doppler myocardial imaging were still normal. Furthermore, in patients with uncontrolled hypertension with manifested global diastolic dysfunction, there was a change in late diastolic parameters. Our results point to a potentially important role of Color Doppler myocardial imaging in diagnosing hypertensive heart disease as well as in follow-up of treatment.

Many hemodynamic studies and clinical experience showed that, in addition to systolic heart function, the hemodynamic events during diastole are of exceptional importance (1,2). According to the criteria of the European Society of Cardiology, the diagnosis of primary diastolic heart failure requires evidence of normal left ventricular systolic function and objective measure-

ments confirming abnormal diastolic function in patients with signs and symptoms of heart failure (1). Diastolic dysfunction represents a decrease in left ventricular filling and reduced possibility to maintain stroke volume without a compensatory increase of atrial filling pressures. This condition usually precedes the development of systolic dysfunction and is the main determinant of heart fail-

ure symptoms. Conventional Doppler echocardiography is the method of choice in assessment of diastolic function. The main limiting factor for this method is the dependence of transmitral flow velocity on the time of left ventricular isovolumic relaxation and left atrial pressure. As opposed to conventional Doppler, which evaluates only global myocardial function, color Doppler Myocardial Imaging provides assessment of both global and regional myocardial function, has higher temporal and spatial resolution, and does not depend on left ventricular filling pressure.

Conventional Doppler echocardiography provides parameters of diastolic function on the basis of measurement of transmitral and pulmonary venous flow, so different stages of left ventricular diastolic dysfunction result in various transmitral and pulmonary flow patterns (3). Color Doppler myocardial imaging, on the other hand, objectively quantifies regional and global left ventricular function by the evaluation of myocardial velocity data. This relatively new addition to the echocardiographic examination is similar to conventional Doppler, which evaluates blood flow, but its technological capabilities are oriented toward lower frequency velocities of myocardial tissue motion (2). Color Doppler myocardial imaging, as opposed to conventional Doppler, provides insight into regional myocardial function and is used in detection of diastolic dysfunction, correlating decreased myocardial velocities in early diastole to the time constant of relaxation (τ) (4,5). However, little data are available to show an advantage of strain or strain-rate imaging over velocity recording in assessment of diastolic function (2). The myocardial motion velocity can be imaged by pulse Doppler time-velocity curves at defined segments of the myocardium and by color velocity maps in M-mode or 2-dimensional (2D) mode (6). Another technical advantage of Color Doppler myocardial imaging is high temporal resolution (7). Without the presence of significant shape deformation or regional left ventricular wall motion abnormalities, the velocity of the mitral annulus motion, which can be measured by Color Doppler myocardial imaging, reflects left ventricular volume changes better than the differences in pressure between the left atrium and left ventricle. Myocardial and annular velocities are also less dependent on changes in volume status than on mitral flow velocities (8-10) and, therefore, can be

used to discern pseudonormalization from normal transmitral flow (8,11). Since velocities of mitral annular motion can be different for its different segments, the mean value of the septal, anterior, lateral, and posterior segment velocities is preferred, especially in patients with regional wall motion abnormalities. The diastolic parameters that can be distinguished on the Color Doppler myocardial imaging velocity curves are isovolumic relaxation time, E'-wave (early filling phase), and A'-wave (late diastolic wave due to atrial contraction). While the transmitral E-wave velocity increases with increasing left ventricular filling pressure, the annular velocity during early diastole does not depend on it. Therefore, the ratio of the transmitral E-wave and Color Doppler myocardial imaging E'-wave (E/E') increases with increased filling pressure and is reported to correlate with the pulmonary capillary wedge pressure (12-15). The peak systolic velocity of the mitral annulus motion correlates with the peak temporal derivative of left ventricular pressure (dP/dt) (16).

Since Color Doppler myocardial imaging can detect temporal and spatial inhomogeneity of myocardial motion during diastole, we decided to investigate if it could detect diastolic dysfunction in patients with hypertension whose conventional Doppler parameters were still unchanged.

Participants and Methods

Participants

This prospectively designed study was conducted at the Department of Cardiovascular Diseases, Zagreb University Hospital Center. The participants were recruited from the Outpatient department for arterial hypertension of the hospital within a six-month period. The study included 46 individuals, 33 patients and 13 healthy volunteers as controls. A patient group consisted of 18 men and 15 women with the mean age (\pm standard deviation) of 47 ± 6 years, whereas a control group consisted of 7 men and 6 women with the mean age of 44 ± 5 years. Patient inclusion criteria were as follows: treated arterial hypertension, normal finding on standard 12-lead electrocardiogram, normal stress test (exclusion of significant myocardial ischemia), 1D and 2D echocardiographic findings of normal internal dimension, normal septum, and posterior wall thickness of the left ventricle in end-diastole, and normal ejection fraction by 2D echocardiography. Exclusion criteria

were clinical evidence of cardiac disease found in medical history, on physical examination, or on a routine 2D echocardiographic examination, and other systemic diseases that could alter myocardial function.

The patient group was divided into two subgroups according to the mean blood pressure (BP) values during 24-hour blood pressure monitoring while under antihypertensive therapy. One subgroup comprised 22 patients with uncontrolled hypertension (mean BP value > 140/90 mm Hg) according to European Society for Hypertension and European Society for Cardiology (17). The other subgroup comprised 11 patients with controlled hypertension (mean BP value < 140/90 mm Hg).

Method

All participants underwent a complete standard echocardiographic evaluation (2D, M-mode, pulsed, and continuous Doppler) and Color Doppler myocardial imaging at baseline and after three months of follow-up. During the examinations, the participants lay in the left lateral decubitus position. The echocardiographic data for three complete cardiac cycles during a single end-expiratory breath holding were collected with a 2.5-MHz-phased array transducer (Vivid 7 System, Vingmed GE Horten, Norway) and stored in a cine-loop format. Off-line analysis and data processing were done on a personal computer and TVI tool software (TVI 6.0, GE Vingmed, Horton, Norway).

The study was performed according to the regulations of the Ethics Committee of the Zagreb University Hospital Center and informed consent was obtained from all volunteers and patients.

Standard Echocardiography

Standard echocardiographic examination was performed by a 1D and 2D pulsed-wave and continuous-wave Doppler. Data were obtained from the standard transducer positions (7). For 2D studies, parasternal long and short axis and apical two- and four-chamber views were used. The left atrial size values were obtained by standard 1D and 2D echocardiography. Transmitral flow parameters measured by pulsed-wave Doppler included E-wave velocity, A-wave velocity, E-wave velocity/A-wave velocity ratio, E-wave deceleration time, A-wave duration, and isovolumic relaxation time (Fig. 1). The parameters of pulmo-

nary venous flow measured by pulsed-wave Doppler were pulmonary venous systolic wave, pulmonary venous diastolic wave, pulmonary venous systolic wave to pulmonary venous diastolic wave ratio, pulmonary venous atrial reversal wave, and the duration of pulmonary venous atrial reversal wave. According to the criteria of the Working Group for Diastolic Dysfunction of the European Society of Cardiology (1), the normal values of diastolic function parameters were corrected for age of 41-60 years as follows: E-wave velocity/A-wave velocity ratio, 1.28 ± 0.25 ; E-wave deceleration time, 181 ± 19 ms; A-wave duration, 133 ± 13 ms; and isovolumic relaxation time, 74 ± 7 ms.

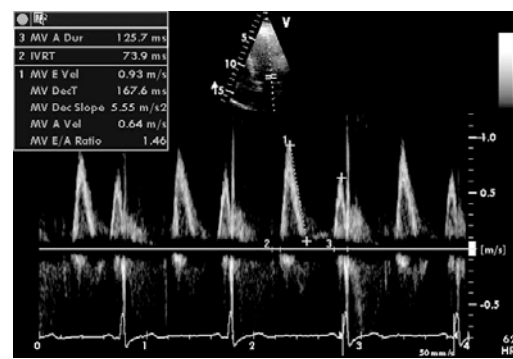


Figure 1. Pulsed wave Doppler transmitral flow. E-wave velocity, A-wave velocity, E-wave velocity/A-wave velocity ratio, E-wave deceleration time, A-wave duration, and isovolumic relaxation time.

Doppler Myocardial Imaging

Color Doppler myocardial imaging was used to measure the velocities of myocardial motion at the annulus, base, and mid-segment of each wall in two-, three-, and four-chamber views. The following parameters were obtained: peak early diastolic velocity E' , peak late diastolic velocity A' , acceleration time A' , deceleration time E' , and E'/A' ratio (Fig. 2). For data acquisition, three complete cardiac cycles during a single end-expiratory breath holding were collected and stored in a cine-loop format. Real-time color Doppler myocardial velocities were acquired as data superimposed on the underlying 2D gray-scale image. To obtain the high frame-rate data, the angle of insonation was reduced to 15 degrees. As a consequence, each segment – septal, lateral, inferior, and anterior – had to be acquired separately at each level. Care was taken that the insonation was parallel to the longitudinal velocity direction. Spe-

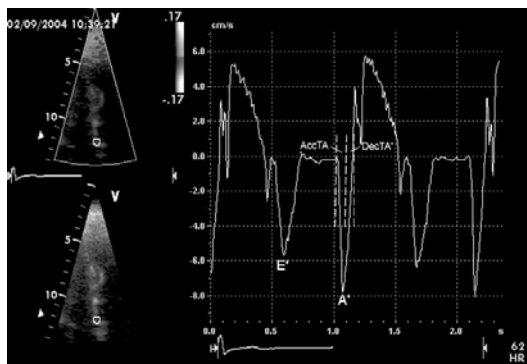


Figure 2. Color Doppler myocardial imaging velocity curve obtained at the septal annulus. E' – peak early-diastolic velocity, A' – peak late-diastolic velocity, AccTA' – acceleration time A', DecTA' – deceleration time A'.

cial attention was paid to avoid aliasing within the image by adjusting the values of pulse repetition frequency (2.0-2.5 kHz).

Statistical Analysis

Measured values were reported as mean value \pm standard deviation (SD). Student t-test was used to compare normally distributed continuous variables. P-values were corrected with Bonferoni correction procedure for multiple comparisons. To compare follow-up data, the paired T-test was applied. P value of <0.05 was considered statistically significant. Statistical analysis was performed

with the SAS System for Windows statistical package (rel. 8.02, SAS Institute Inc., Cary, NC, USA).

Results

Global Diastolic Function

The conventional Doppler parameters of diastolic function – E/A ratio, E-wave deceleration time, A-wave duration, and isovolumic relaxation time – in the patients with controlled hypertension and healthy controls at baseline and follow-up were within the normal range (1). However, relaxation disturbances noted in conventional echocardiography parameters measured in patients with uncontrolled hypertension at baseline were significantly worse from those in the controls and patients with controlled hypertension (Fig. 3). The values of these parameters remained the same at the follow-up.

Left atrial size, dimensions of the left ventricular posterior wall in diastole, and interventricular septum thickness in diastole measured at baseline by M-mode echocardiography did not significantly change after three months of follow-up in any of the groups. Mean left atrial size was 3.43 ± 0.27 cm in the controls, 3.70 ± 0.60 cm in patients with controlled hypertension, and 3.71 ± 0.45 cm in patients with uncontrolled arterial hypertension. Mean left ventricular posterior wall thickness in diastole was 0.96 ± 0.14 cm, 0.99 ± 0.15 cm, and 0.98 ± 0.20 cm in the con-

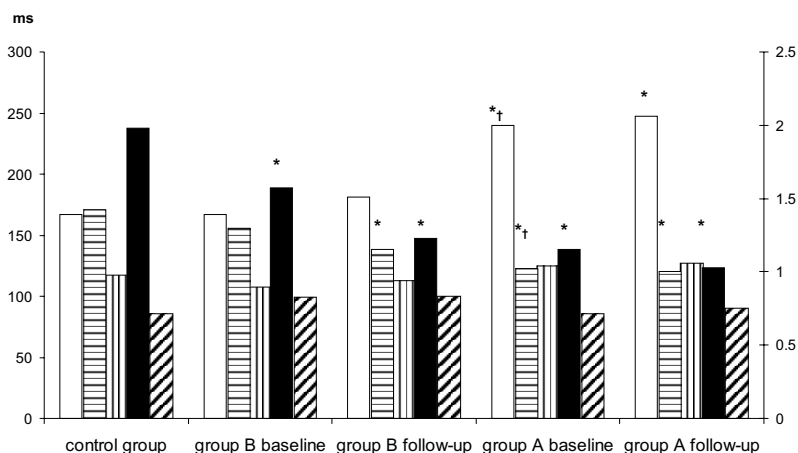


Figure 3. Pulse wave Doppler and Color Doppler myocardial imaging parameters of diastolic function. Open bars – transmitral flow deceleration time; bars with horizontal lines – transmitral flow E-wave velocity to A-wave velocity ratio (pulse wave Doppler); bars with vertical lines – transmitral flow A duration; closed bars – E'-wave velocity to A'-wave velocity ratio (color Doppler); bars with diagonal lines – transmitral flow isovolumic relaxation time; Group B – patients with controlled arterial hypertension; Group A – patients with uncontrolled arterial hypertension; * $P < 0.050$ vs. control group; † $P < 0.050$ in relation to group with controlled hypertension.

trols, patients with controlled hypertension, and patients with uncontrolled hypertension, respectively. The interventricular septum thickness in diastole was 0.87 ± 0.22 cm in the controls, 0.97 ± 0.13 cm in patients with controlled hypertension, and 1.08 ± 0.22 cm in patients with uncontrolled hypertension. According to these values, left ventricular hypertrophy was not present in any of the groups. The mean value of left ventricular systolic function assessed by the Simpson method was normal in both patients and controls.

Color Doppler myocardial imaging parameters of global diastolic function were determined from myocardial tissue velocity curves obtained from the mitral annulus. In the group of patients with controlled hypertension at baseline, a significantly smaller peak early-diastolic velocity E' was noted than in the control group. In the same group at follow-up, E'/A' was statistically smaller than in the control group (1.23 ± 0.31 , $P=0.042$), but did not meet the criteria for diastolic dysfunction ($E'/A' < 1$). In the group of patients with uncontrolled hypertension, both at baseline and follow-up, a significantly smaller peak early-diastolic velocity E' , greater peak late-diastolic velocity A' , and diminished E'/A' were noted in comparison with the control group (Table 1). In the group of patients with controlled hypertension, $E'/A' < 1$

was noted in 2 of 21 segments at baseline and in 6 of 21 segments at the follow-up. In group of patients with uncontrolled hypertension, $E'/A' < 1$ was noted in 12 of 21 segments at baseline and was not significantly different at the follow-up (13 out of 21 segments). In comparison with E/A , E'/A' showed more significant changes in all groups.

Regional Diastolic Function

We analyzed regional myocardial relaxation disturbances manifested as diminished peak early-diastolic velocity E' , increased peak late-diastolic velocity A' , and $E'/A' < 1$. Color Doppler myocardial imaging renders the possibility of measurement of deceleration time E' in early diastole, ie, timing the myocardial deceleration at the time of relaxation, and the acceleration time A' in late diastole, ie, time of myocardial acceleration during atrial systole.

Patients with Controlled Hypertension

By measuring myocardial velocities on the lateral left ventricular wall, a statistically significant reduction of peak early-diastolic velocity E' was noted both in the group of patients with controlled hypertension at baseline and follow-up when compared with control group. At the follow-up, an increase in deceleration time E' and decrease in E'/A' were significant in comparison to

Table 1. Parameters of global diastolic function – septal, lateral, inferior, and posterior region of mitral annulus – measured by Color Doppler myocardial imaging at mitral annulus

Patient group	Global diastolic function (mean±SD)*				
	E' (cm/s)	dt E' (ms)	A' (cm/s)	E'/A'	AccTA' (ms)
Control	-8.82 ± 2.39	0.11 ± 0.03	-6.12 ± 2.88	1.98 ± 0.92	0.06 ± 0.01
Controlled hypertension:					
baseline	$-7.18 \pm 1.37^\dagger$	0.09 ± 0.01	-6.02 ± 2.50	1.57 ± 0.50	0.05 ± 0.01
follow-up	$-7.37 \pm 1.46^\dagger$	0.11 ± 0.03	$-7.34 \pm 2.07^\dagger$	$1.23 \pm 0.31^\dagger$	$0.07 \pm 0.01^\dagger$
Uncontrolled hypertension:					
baseline	$-6.66 \pm 1.16^\dagger$	0.09 ± 0.02	$-6.80 \pm 2.06^\dagger$	$1.15 \pm 0.54^\dagger$	0.06 ± 0.01
follow-up	$-6.34 \pm 1.61^\dagger$	0.12 ± 0.03	$-7.05 \pm 1.96^\dagger$	$1.03 \pm 0.26^\dagger$	$0.07 \pm 0.01^\dagger$

*Abbreviations: E' – peak early-diastolic velocity; dt E' – deceleration time E' ; A' – peak late-diastolic velocity; AccTA' – acceleration time A' .

$^\dagger P < 0.050$ vs. control group.

Table 2. Parameters of regional diastolic function measured by Color Doppler myocardial imaging from lateral left ventricular wall (basal and mid-segment)

Patient group	Regional diastolic function (mean±SD)*				
	E' (cm/s)	dt E' (ms)	A' (cm/s)	E'/A'	AccTA' (ms)
Control	-10.18 ± 2.79	0.07 ± 0.01	-4.41 ± 0.62	2.96 ± 0.36	0.04 ± 0.00
Controlled hypertension:					
baseline	$-6.91 \pm 1.36^\dagger$	0.07 ± 0.02	-5.39 ± 1.47	1.70 ± 0.30	0.04 ± 0.01
follow-up	$-6.05 \pm 2.04^\dagger$	$0.09 \pm 0.03^\ddagger$	-6.98 ± 1.11	$0.96 \pm 0.18^\ddagger$	0.06 ± 0.02
Uncontrolled hypertension:					
baseline	$-6.33 \pm 1.51^\dagger$	0.07 ± 0.01	-6.13 ± 1.62	$1.25 \pm 0.18^\dagger$	0.04 ± 0.01
follow-up	$-4.88 \pm 1.92^\dagger$	$0.09 \pm 0.02^\ddagger$	-5.36 ± 1.57	$1.13 \pm 0.26^\dagger$	$0.06 \pm 0.01^\dagger$

*Abbreviations: E' – peak early-diastolic velocity; dt E' – deceleration time E' ; A' – peak late-diastolic velocity; AccTA' – acceleration time A' .

$^\dagger P < 0.05$ vs control group.

$^\ddagger P < 0.05$ vs baseline values within the group and vs control group.

baseline values and control group. The increase in peak late-diastolic velocity A' and acceleration time A' was not statistically significant (Table 2). The measurements on the anterior wall showed a significant reduction of peak early-diastolic velocity E' both at baseline and follow-up, as well as an increase of peak late-diastolic velocity A' and acceleration time A' at follow-up, when compared with baseline values and control group. The decrease in E'/A' and increase in deceleration time E' were not statistically significant (Table 3). The values obtained at the interventricular septum were not statistically significant when compared with those in the control group (Table 4). At the inferior wall, E'/A' was significantly decreased in both

measurements, whereas peak late-diastolic velocity A' and acceleration time A' were increased at follow-up (Table 5). Velocity measurements on the posterior wall showed an increase in late-diastolic velocity A' and a decrease in both peak early-diastolic velocity E' and E'/A' , when compared with the control group (Table 6).

Patients with Uncontrolled Hypertension

The measurements performed on the lateral wall showed significant reduction of both peak early-diastolic velocity E' and E'/A' at baseline in patients with uncontrolled hypertension when compared with the control group. At follow-up, an increase in deceleration time E' was

Table 3. Parameters of regional diastolic function measured by Color Doppler myocardial imaging from anterior left ventricular wall (basal and mid segment)

Patient group	Regional diastolic function (mean±SD)*				
	E' (cm/s)	dt E' (ms)	A' (cm/s)	E'/A'	AccTA' (ms)
Control	-8.60±2.66	0.09±0.02	-3.38±1.65	2.64±1.15	0.05±0.01
Controlled hypertension:					
baseline	-5.96±1.46†	0.08±0.01	-2.95±1.31	2.50±1.46	0.04±0.00
follow-up	-6.34±0.84†	0.09±0.01	-4.92±0.87†‡	2.17±1.13	0.06±0.01†
Uncontrolled hypertension:					
baseline	-5.92±1.17†	0.08±0.02	-4.72±1.02†	1.37±0.34†	0.05±0.01†
follow-up	-4.37±1.83†‡	0.10±0.01‡	-3.17±1.91‡	1.87±0.75	0.06±0.02†

*Abbreviations: E' – peak early-diastolic velocity; dt E' – deceleration time E' ; A' – peak late-diastolic velocity; AccTA' – acceleration time A' .

† P <0.05 vs control group.

‡ P <0.05 vs baseline values within the group.

Table 4. Parameters of regional diastolic dysfunction measured by Color Doppler myocardial imaging from the left ventricular septum (basal and mid segment)

Patient group	Regional diastolic dysfunction (mean±SD)*				
	E' (cm/s)	dt E' (ms)	A' (cm/s)	E'/A'	AccTA' (ms)
Control	-7.05±0.93	0.09±0.01	-5.36±1.20	1.62±0.27	0.05±0.00
Controlled hypertension:					
baseline	-5.87±1.08	0.09±0.01	-4.75±0.84	1.54±0.07	0.04±0.00
follow-up	-6.51±0.18	0.12±0.03	-6.02±1.40	1.44±0.58	0.07±0.01
Uncontrolled hypertension:					
baseline	-5.44±0.46†	0.08±0.01	-6.58±1.02†	0.87±0.10†	0.05±0.00†
follow-up	-5.68±0.48†	0.13±0.01‡	-7.28±1.47†	0.78±0.11†	0.07±0.01

*Abbreviations: E' – peak early-diastolic velocity; dt E' – deceleration time E' ; A' – peak late-diastolic velocity; AccTA' – acceleration time A' .

† P <0.05 vs control group.

‡ P <0.05 vs baseline values within the group and vs control group.

Table 5. Parameters of regional diastolic dysfunction measured by Color Doppler myocardial imaging from inferior left ventricular wall (basal and mid segment)

Patient group	Regional diastolic dysfunction (mean±SD)*				
	E' (cm/s)	dt E' (ms)	A' (cm/s)	E'/A'	AccTA' (ms)
Control	-7.23±1.85	0.08±0.01	-5.18±1.31	2.05±0.42	0.04±0.00
Controlled hypertension:					
baseline	-6.61±1.43	0.07±0.01	-6.03±0.63	1.25±0.10†	0.04±0.00
follow-up	-5.78±1.93	0.08±0.02	-6.99±1.59†	0.92±0.06†	0.05±0.00†
Uncontrolled hypertension:					
baseline	-5.30±1.38†	0.08±0.00	-6.57±1.05†	0.90±0.06†	0.04±0.00
follow-up	-5.02±1.27†	0.11±0.01‡	-6.91±1.59†	0.83±0.08†	0.06±0.00†

*Abbreviations: E' – peak early-diastolic velocity; dt E' – deceleration time E' ; A' – peak late-diastolic velocity; AccTA' – acceleration time A' .

† P <0.05 vs control group.

‡ P <0.05 vs baseline values within the group and vs control group.

Table 6. Parameters of regional diastolic dysfunction measured by Color Doppler myocardial imaging from posterior left ventricular wall (basal and mid segment)

Patient group	Regional diastolic dysfunction (mean±SD)*				
	E' (cm/s)	dtE' (ms)	A' (cm/s)	E'/A'	AccTA' (ms)
Control	-7.05±1.53	0.09±0.01	-2.67±0.21	3.71±1.24	0.05±0.01
Controlled hypertension:					
baseline	-5.37±0.94†	0.07±0.01	-3.62±0.29†	1.86±0.58†	0.05±0.00
follow-up	-5.89±0.89	0.07±0.02†	-3.59±0.81	2.72±0.79	0.05±0.00
Uncontrolled hypertension:					
baseline	-5.26±0.72†	0.09±0.00	-3.04±0.32	2.35±0.17†	0.06±0.00
follow-up	-3.95±0.59†	0.07±0.01†	-3.04±1.17	1.93±0.66†	0.06±0.01

*Abbreviations: E' – peak early-diastolic velocity; dtE' – deceleration time E'; A' – peak late-diastolic velocity; AccTA' – acceleration time A'.

†P<0.05 vs control group.

significant when compared to baseline values and the control group, and acceleration time A' increased significantly compared with the control group (Table 2). On the anterior wall, a decrease in peak early-diastolic velocity E' at baseline was significant when compared with the control group, but diminished at follow-up. The peak late-diastolic velocity A' wave was notably increased, whereas E'/A' was decreased at baseline in comparison with those in the control group. At follow-up, there was a significant decrease in both peak late-diastolic velocity A' and deceleration time E' was in comparison with baseline values, whereas acceleration time A' increased in comparison with that in the control group (Table 3). The measurements at the septum showed a significant decrease in peak early-diastolic velocity E', increase in peak late-diastolic velocity A', and decrease in E'/A' at baseline and follow-up, when compared with the control group (Table 4). Both measurements on the inferior wall showed a significant increase in the peak late-diastolic velocity A' and decrease in peak early-diastolic velocity E', and E'/A' when compared with the control group. At follow-up, a prolongation of deceleration time E' and acceleration time A' was noted when compared with baseline values and those in the control group (Table 5). Both measurements on the posterior wall segments showed a significant decrease in peak early-diastolic velocity E' and E'/A' ratio in comparison with those in the control group, and a shorter deceleration time E' at follow-up when compared with baseline values and those in the control group (Table 6).

Discussion

To the best of our knowledge, an evaluation of isolated regional diastolic dysfunction determined by Color Doppler myocardial imaging in hypertensive patients has not been previously per-

formed. Doppler myocardial imaging is currently the only available method to measure regional diastolic function. In this study, we showed that Color Doppler myocardial imaging parameters of regional diastolic function reveal significant regional diastolic disturbances in patients with arterial hypertension without signs of left ventricular hypertrophy. In patients with controlled hypertension, these regional disturbances were manifest while conventional Doppler parameters of diastolic function and Color Doppler myocardial imaging parameters for global diastolic function were still within a normal range. Furthermore, a larger number of segments showed relaxation disturbances in patients with uncontrolled hypertension.

Diastolic dysfunction, particularly relaxation disturbances in early diastole, are frequent in patients with pronounced hypertensive heart disease with left ventricular hypertrophy (18,19). In daily clinical practice, global diastolic dysfunction is evaluated by the conventional Doppler method. However, the specificity and sensitivity of existing methods in diagnosing diastolic dysfunction are poor. Color Doppler myocardial imaging has proved an exceptional role in assessment of global diastolic function, particularly in patients with pseudonormalization of transmitral flow by conventional Doppler. Sohn et al (8) reported that the velocity of mitral annular motion measured by Doppler myocardial imaging during diastole was relatively independent on volume overload and efficient in differentiating between pseudonormalization and normal transmitral flow. In hypertensive patients without evidence of left ventricular hypertrophy, the parameters of conventional Doppler often do not show abnormal patterns.

In mild hypertensive disease, Color Doppler myocardial imaging parameters of deranged diastolic function may be the first sign of left ventricular dysfunction (20). In this study, we

showed that the parameters of global diastolic function measured by conventional Doppler in patients with controlled hypertension did not deviate from those in the control group. Furthermore, Color Doppler myocardial imaging parameters of global diastolic dysfunction in this group of patients did not reach the criteria for diagnosis of diastolic dysfunction. Nevertheless, measurements of regional diastolic function showed that the segments with relaxation disturbances accounted for 5% at baseline, which was increased to 30% at follow-up despite well regulated antihypertensive therapy. Pela et al (21) evaluated systolic and diastolic function in 54 patients with controlled arterial hypertension. In long axis view, they recorded a significant decrease in E'-velocity and E'/A' ratio, which were absent in short axis. E'/A' ratio was decreased also in our study, although the values were not < 1. One group of patients in our study had uncontrolled arterial hypertension without left ventricular hypertrophy and met at least one criterium for relaxation disturbances – low grade diastolic dysfunction – among the conventional Doppler parameters. In this group, Color Doppler myocardial imaging revealed many segments with regional diastolic dysfunction (60%). Galderisi et al (19) reported that left ventricular diastolic dysfunction was uniformly distributed among segments in hypertensive patients without left ventricular hypertrophy, while it was more evident at the basal septal level than in other regions in hypertensive patients with left ventricular hypertrophy.

In this study, we also found significant changes in late diastolic parameters, particularly prolongation of acceleration time A' and increase in A' velocity. In impaired relaxation, the left ventricular myocardial velocities, which follow atrial contraction, may be increased. Furthermore, as the left ventricular stroke volume diminishes at high telediastolic pressure, the mentioned velocities diminish as well (8,11,12). Therefore, the stroke volume of the left atrium and ventricle influences the velocity of mitral annulus and the myocardial motion, and the consequence of volume overload is expected to vary depending on the left atrial pressure changes and change of stroke volume (4). Nevertheless, the normal left ventricular function and normal stroke volume without significant increase in telediastolic pressure in our patients could probably not have influenced the change in A' velocity and myocardial acceleration

time during atrial systole. Likewise, the left atria measured in our patients were of normal size or at upper normal limits, which implicated preserved atrial function, ie, atrial contraction. Therefore, it is to be expected that the changes of Color Doppler myocardial imaging parameters in late diastole are most probably not caused by left atrial and left ventricular stroke volume changes. It is most likely that they are due to the influence of initial myocardial fibrosis on left ventricular distensibility, which occurs as a consequence of arterial hypertension. Yamamoto et al (22) showed that myocardial stiffening was enhanced by progressive collagen accumulation rather than compensatory left ventricular hypertrophy in hypertensive hearts.

There were several limitations to our study. The follow-up period was not long enough and the study group was not large enough to obtain more information on diastolic dysfunction in arterial hypertension. To improve our understanding of changes in myocardial velocities during late diastole, pure acceleration in addition to acceleration time, in correlation with left atrial function might be needed for better estimate of left ventricular compliance.

In conclusion, we showed that regional diastolic dysfunction measured by Color Doppler myocardial imaging is the first sign of myocardial dysfunction due to arterial hypertension, while the parameters of global diastolic dysfunction measured by conventional Doppler as well as by Color Doppler myocardial imaging are still unchanged. Furthermore, in patients with uncontrolled arterial hypertension in whom global diastolic dysfunction is already manifested, we have registered a change in late diastolic parameters. Our results point to a potentially important role of Color Doppler myocardial imaging in diagnosing hypertensive heart disease as well as follow-up of treatment in patients with arterial hypertension.

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Correspondence to:

Maja Čikeš
Department of Cardiovascular Diseases
Zagreb University Hospital Center
Kišpatičeva 12
10000 Zagreb, Croatia
maja.cikes@zg.htnet.hr