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ORIGINAL ARTICLE

Assessment of Regional Atrial Function in Patients with Hypertrophic Cardiomyopathies Using Tissue Doppler Imaging

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Abstract
Background This study applied tissue Doppler imaging and color tissue Doppler imaging to study atrial function changes in patients with hypertrophic cardiomyopathy (HCM). The profile of the segmental atrial velocities and the strain rate were determined and compared with those of normal matched control subjects.
Methods This study investigated 20 patients with HCM and 20 age-matched healthy control subjects. In a four-chamber apical view, tissue Doppler imaging was used to measure the lateral left and right atrial (LA and RA) and interatrial septal (IAS) wall systolic, early, and late diastolic velocities. Similarly, the atrial strain rate during ventricular systole (SR_S) and the early (SR_E) and late (SR_A) diastolic phases in patients and control subjects were measured. The interventricular septal tissue Doppler-derived isovolumic relaxation time was calculated.
Results Only the IAS annular and middle segments showed a significant reduction in the early diastolic velocity (mean, 4.01 ± 2.2 vs 8.7 ± 1.1, $p = 0.001$; 3.23 ± 2 vs 6.01 ± 1.9, $p = 0.001$, respectively) for the patients with HCM in comparison with the control subjects. Generally, the atrial strain rate was clearly reduced. The systolic strain rate (SR_S) was significantly reduced in the LA wall in the annular ($p = 0.007$) and middle ($p = 0.001$) segments and in the IAS middle segment ($p = 0.007$). Similarly, there was a reduction of the early diastolic strain rate (SR_E) in the LA annular ($p = 0.001$) and middle ($p = 0.01$) segments and in the IAS annular ($p = 0.05$) and middle ($p = 0.001$) segments, as well as in the RA annular segment ($p = 0.02$). The RA middle segments showed insignificant changes.
Conclusion Atrial function may be affected by HCM due to impairment of myocardial diastolic function. Strain rate imaging is reproducible, yields readily obtained parameters that provide unique data about global and longitudinal segmental atrial contraction, and can quantify the atrial dysfunction in patients with HCM.

Keywords Atrial performance · Cardiomyopathy · Doppler · Strain · Tissue velocity

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In addition to evaluation of ventricular function, assessment of atrial function in patients with hypertrophic cardiomyopathy (HCM) may be of diagnostic and prognostic value. Physiologically, the atria serve both as a reservoir and as a conduit for the passage of blood from the pulmonary veins to the left ventricle (LV) and from the superior and inferior caval veins to the right ventricle (RV).

Previous studies indicate that the left atrium contributes up to 30% of LV filling and cardiac output and has an important role, particularly in the setting of impaired LV function [8]. Traditionally, blood flow velocity during atrial contraction, peak atrioventricular inflow A-wave velocity [6, 9], and its velocity time integral and atrial emptying fraction have been used as surrogate markers of active atrial contraction. Atrial ejection force, based on classic Newtonian principles and derived as the force

exerted by the left atrium to accelerate blood into the LV, also has been used as a marker of atrial function [7].

Tissue Doppler imaging (TDI) is a recently developed technique for the quantitative assessment of myocardial contraction and relaxation in the LV using low-velocity pulsed-wave Doppler interrogation of the myocardium [3, 12]. The TDI technique enables simultaneous acquisition of myocardium in the same image view, with measurements performed offline. Earlier work examined differences between the contraction velocity of the left atrial appendage and body [5] and differences in blood flow velocities within the atrium [14].

To our knowledge, comprehensive segmental contraction of the atria body has not been studied previously, nor have atrial functions in patients with HCM been evaluated. Our study aimed to assess the value of TD-derived velocity and strain rate for quantitative analysis of regional atrial performance in asymptomatic patients with HCM.

Materials and Methods

Study Population

Our study population consisted of 20 pediatric patients with the diagnosis of hypertrophic obstructive cardiomyopathy. The 9 girls and 11 boys, ages 2 to 18 years, were evaluated in terms of their medical history, initial symptoms and signs, and noninvasive procedures (echocardiography, Doppler, electrocardiogram [ECG]). The diagnosis of HCM was based on two-dimensional (2D) echocardiography, Doppler, and cardiac catheter findings of hypertrophy of cardiac muscle as well as significant intraventricular obstruction and pressure gradients exceeding 30 mmHg in the LV outlet tract (LVOT).

The novel noninvasive tissue Doppler imaging of the ventricles and both atria was performed to evaluate the diastolic dysfunction of the LV (prolonged isovolumic relaxation time) as well as the extent to which atrial function was affected. The control group consisted of 20 age-matched healthy subjects (10 girls). Both patients and control subjects were in New York Heart Association (NYHA) Class I-II. Their weight and height at examination, birth weight, blood pressure, and other patient characteristics are summarized in Table 1.

Table 1 Patient characteristics (mean ± SD)

Parameter	HCM patients	Control subjects	*p* Value
Age (years)	18.60 ± 8.80	19.4 ± 9.30	0.22
Male gender	11	10	NS
Heart rate	79.8 ± 17	74.7 ± 10	0.22
Body weight (kg)	56.98 ± 23.90	61.6 ± 19.9	0.6
Height (cm)	160 ± 29.50	159 ± 34.60	0.37
Systolic blood pressure	113 ± 13.00	111 + 10.34	0.29
Diastolic blood pressure	57 ± 10.30	56 ± 9.37	0.62
Age at time of initial diagnosis (mos)	64.30 ± 81.40	—	—

SD, standard deviation; NS, not significant

Echocardiography

Examination was performed using a 3.5-MHz transducer interfaced with a Vingmed System V ultrasound system (GE-Vingmed, Horten, Norway). All patients underwent standard transthoracic imaging of the heart and great vessels at rest, performed with the patient in the left lateral decubitus position. Initial routine diagnostic imaging included M-mode, color-flow mapping, and pulsed- and continuous-wave Doppler. Simultaneous ECG recording was done for all the patients during the echocardiographic examination.

Tissue Doppler Imaging Data Acquisition

After completion of the diagnostic standard transthoracic echocardiography examination, the echocardiographic settings were changed to the application of TDI with the patient in the same position using a simultaneous ECG recording. A four-chamber view was obtained with the color-coded 2D method, including both ventricles and both atria. We imaged the left and right atrium using standard apical four-chamber views and a magnified atrial view. The ultrasonic beam was aligned parallel with the walls of each myocardial segment, thus minimizing the angle-dependent effect (<30°), and a single cine loop of 2D image each for the left atrial (LA) free wall, right atrial (RA) free wall, interatrial septal (IAS) wall, and interventricular septal (IVS) wall was obtained. This technique measures mean velocities (not peak) with both a high temporal frame rate (130 frames/s) and a high spatial resolution in the axial direction [17]. Special attention was paid to the Doppler velocity range to avoid aliasing. The cine loop with three heart cycles was transferred to EchoPac 6.3.6 (Vingmed, Horton, Norway) and recorded digitaly for offline processing. The same procedure was performed with the control subjects.

Postprocessing 2D Myocardial Color Doppler Velocity Data

At the special workstation designed for offline measurement of the collected tissue Doppler data, we measured

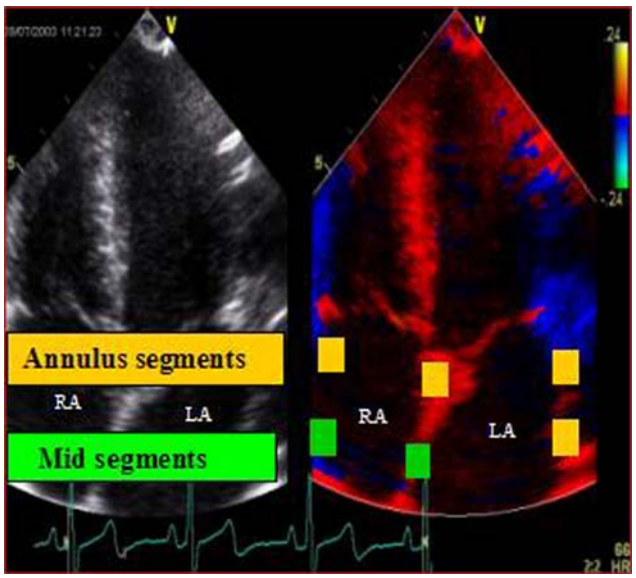

Fig. 1 Sites of velocity and strain rate samples at the annular and middle segments of the atrial walls (region of interest)

systolic and diastolic TD-derived velocities and strain rate by applying the published techniques [16]. Using unprocessed IVS velocity curves and anatomic grayscale M-mode, it was possible to calculate the different cardiac cycle intervals. We measured TD-derived isovolumic relaxation time (IVRT) in both the patients and the control subjects. Regional atrial function was evaluated using velocity and strain rate curves.

All analysis was performed at a workstation (GE/Vingmed EchoPac) with customized software. Regions of interest were placed at the corresponding points of the atrial walls: annular and middle segments of the RA, LA, and IAS wall. From the apical four-chamber view, measurements were made from four segments of the left atrium (IAS annular segment, IAS middle segment, lateral middle segment, and lateral annular segment) and two segments of the right atrium (lateral annular segment and lateral middle RA segment) (Fig. 1). A 9×9 pixel size was used, and the tissue velocity profile throughout the cardiac cycle was displayed in each sample location. A tagging technique was used to control the position of the sample volume within the ventricular wall, and particularly the atrial wall. The maximal and minimal values at the time velocity and strain rate curves were tagged, and the simultaneous position of the sample volume was controlled to remain within the myocardial wall region.

The mean peak velocity of atrial contraction was measured in each segment as an average of three cardiac cycles. Furthermore, because the atrium is thin walled, we measured segmental velocities using the 9×9 pixel size.

Atrial Strain Rate

At the regions of interest, the atrial regional strain rate was measured, and the S (systole), E (early diastole), and A (atrial contraction) waves were recorded, (Fig. 2). Values were compared with those of the control subjects.

Statistical Methods

The statistical software used was SPSS 10.0 (SPSS, Inc., Chicago, IL, USA). Data were expressed as mean ± standard deviation. The nonparametric Mann–Whitney test was used to assess the differences between two unpaired groups. For analysis of correlations, nonparametric Spearman rank correlation was performed. We considered a p value less than 0.05 to be statistically significant.

Results

Standard Echocardiographic Findings

The echocardiographic examination showed IVS hypertrophy and intraventricular obstruction, with turbulent flow and a significant pressure gradient across the LVOT in the patients. Doppler examination showed that 90% of patients have turbulent flow in the LVOT. Pulsed- and continuous-

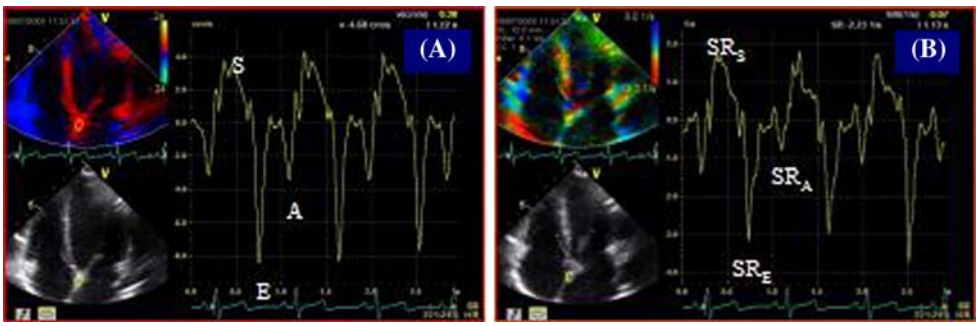

Fig. 2 Pattern of atrial regional velocity (**A**) and strain rate at interatrial septal (IAS) annular segments (**B**) in control subjects. S, systolic velocity; E, early diastolic velocity; A, late diastolic velocity; SR_S: systolic strain rate; SR_E, diastolic strain rate in early diastolic phase; SR_A, diastolic strain rate in late diastolic phase

Table 2 Echocardiographic findings (mean ± SD)

	HCM (n = 20)	Controls (n = 20)	p Value
EF (%)	73.80 ± 17.50	72.90 ± 14	0.220
FS (%)	48.60 ± 12.60	45.70 ± 10	0.410
LVOT Vmax (m/s)	3.10 ± 01.10	1.20 ± 0.40	0.001
LVOT gradient (mmHg)	64.60 ± 42.00	9.40 ± 0.30	0.001
RVOT gradient (mmHg)	46.00 ± 27.80	7.40 ± 5.30	0.021
IVS_s (mm)	20 ± 0.98	9.70 ± 0.80	0.001
IVS_d (mm)	16 ± 0.50	15.21 ± 0.45	0.001
$LVED_d$ (mm)	37.30 ± 3.40	39.80 ± 2.9	0.32
$LVED_s$ (mm)	26.10 ± 0.91	27.21 ± 0.72	0.31
PW_s (cm)	1.60 ± 00.50	4.00 ± 0.50	0.010
IVS_s/PW_s	2.00 ± 00.98	1.01 ± 0.11	0.010
Mitral Doppler inflow E-wave (m/s)	1.00 ± 00.30	0.93 ± 0.16	0.080
Mitral Doppler inflow A-wave (m/s)	0.60 ± 00.20	0.62 ± 0.14	0.090
E/A	1.65 ± 06.30	1.50 ± 0.20	0.120
Tricuspid Doppler inflow E-wave (m/s)	0.60 ± 00.13	0.55 ± 0.10	0.080
Tricuspid Doppler inflow A-wave (m/s)	0.50 ± 00.13	0.49 ± 0.10	0.070
E/A	1.23 ± 01.30	1.12 ± 1.03	0.320
Isovolumic relaxation time (ms) by TDI	84.1 ± 27.8	60 ± 13.30	0.004
Cycle length (ms)	801.8 ± 246	817.3 ± 113	0.220

SD, standard deviation; EF, ejection fraction; FS, fractional shortening; LVOT/RVOT, left/right ventricular outflow tract; IVS, interventricular septum; LVED, left ventricular end dimension; PW, posterior wall; TDI, tissue Doppler imaging

wave measurements of the maximal velocity value in the LVOT ranged from 1 to 4.7 m/s, and the calculated mean pressure gradient across the LVOT was 64.6 ± 42 mmHg. The findings showed that 10% of the patients also had right ventricular outlet tract (RVOT) obstruction, with a mean gradient of 46 ± 28 mmHg. Furthermore, 63% of the patients had mild to moderate mitral valve insufficiency, and 50% of the cases had systolic anterior motion of the mitral valve leaflet [systolic anterior motion (SAM)]. Normal systolic global cardiac function of the hypertrophied cardiac muscle was seen with an ejection fraction of 73% ± 17.5% and a fractional shortening of 48% ± 12% (Table 2).

Regional Atrial Velocities and Strain Rates in HCM Patients Compared with Control Subjects

We found a general slight reduction of the systolic and diastolic regional longitudinal velocities at the annular and middle segments of the LA, IAS, and RA wall in the HCM patients compared with the control subjects. A significant reduction in the mean regional velocities during early filling in patients with HCM, compared with healthy subjects, was found at the annular and middle segments of the IAS, respectively (4.01 ± 2.2 vs 8.77 ± 1.09, p = 0.001; 3.23 ± 2.0 vs 6.01 ± 1.9, p = 0.001).

There was no significant difference between the systolic or diastolic velocities at other atrial wall segments between the patients with HCM and the healthy control subjects (Table 3). The pressure gradient on the LVOT was negatively correlated with the velocity values: early diastolic velocity at the IAS annular segment (r = –0.7; p = 0.01), RA annular segment (systolic velocity: r = –0.6; p = 0.02), and RA annular segment (late velocity: r = –0.5; p = 0.03).

In the patients with HCM, the systolic and diastolic strain rate values reflecting the deformation rate and regional contractile function in the studied regions were reduced in the annular and middle segments of the LA lateral atrial wall, the IAS middle segment, and the RA annular segment compared with those of the healthy control subjects, as shown in Table 4.

The atrial velocities and strain rate values were not associated with the thickness of the IVS septum or heart rate. The pressure gradient at the LVOT was negatively correlated with the systolic and early diastolic deformation rate at the IAS wall (systole [SR_S]: r = –0.6, p = 0.03; annular early diastolic phase [SR_E]: r = –0.6, p = 0.02; middle SR_E: r = –0.6, p = 0.04, respectively). These findings may indicate that an adaptive change in the regional deformation of the left atrium appears related to pressure overload rather than myocardial thickness of the IVS in patients with HCM. The early peak strain rate (SR_E) at the annular segment of the LA wall is negatively correlated

Table 3 Comparison between the regional atrial velocity (cm/s) in patients with hypertrophic cardiomyopathy (HCM) (n = 20) and control subjects (n = 20)

Parameters	Velocity (mean ± SD)								
	S			E			A		
	HCM	Controls	P Value	HCM	Controls	P Value	HCM	Controls	p Value
LA annular seg	5.03 ± 2.2	6.70 ± 1.6	0.07	5.74 ± 3.0	8.69 ± 3.9	0.1	3.77 ± 1.3	5.14 ± 2.4	0.2
LA middle seg	2.94 ± 1.5	4.29 ± 2.5	0.2	1.87 ± 2.2	3.72 ± 2.5	0.09	2.29 ± 1.7	3.12 ± 2.2	0.4
IAS annular seg	4.65 ± 1.5	5.23 ± 1.1	0.4	4.01 ± 2.2	8.77 ± 1.1	0.001	4.45 ± 1.9	5.36 ± 1.8	0.3
IAS middle seg	3.60 ± 1.8	4.28 ± 2.0	0.3	3.23 ± 2.0	6.01 ± 1.9	0.001	2.75 ± 1.6	3.93 ± 2.1	0.2
RA annular seg	6.17 ± 2.9	7.59 ± 2.0	0.2	4.96 ± 3.4	5.79 ± 2.8	0.3	5.87 ± 2.7	5.30 ± 2.9	0.6
RA middle seg	2.65 ± 2.2	2.95 ± 3	0.9	1.75 ± 1.9	2.18 ± 2.7	1.00	2.60 ± 2.4	2.10 ± 2.6	0.3

SD, standard deviation; S, systolic; E, early diastolic velocity; A, late diastolic velocity; LA, left atrial; seg, segment; IAS, interatrial septum; RA, right atrial

Table 4 Comparison between the atrial strain rate (1/s) in patients with hypertrophic cardiomyopathy (HCM) (n = 20) and control subjects (n = 20)

Parameters	Strain rate (mean ± SD)								
	SR_S			SR_E			SR_A		
	HCM	Controls	P Value	HCM	Controls	p Value	HCM	Controls	p Value
LA annular seg	1.59 ± 1.2	2.58 ± 0.8	<0.01	1.77 ± 1.0	4.37 ± 1.9	<0.01	1.26 ± 1.1	1.35 ± 0.7	0.3
LA middle seg	1.87 ± 0.9	3.71 ± 1.4	0.01	1.55 ± 1.2	4.46 ± 3.2	0.01	1.69 ± 1.7	2.94 ± 1.8	0.07
IAS annular seg	1.59 ± 0.9	2.38 ± 1.1	0.3	1.49 ± 1.1	2.38 ± 1.1	0.05	0.82 ± 0.8	1.91 ± 0.7	0.7
IAS middle seg	2.73 ± 1.5	4.60 ± 1.4	<0.01	2.16 ± 1.3	5.39 ± 1.9	<0.01	2.15 ± 1.1	3.54 ± 1.14	0.01
RA annular seg	3.67 ± 2.6	4.42 ± 1.4	0.3	3.67 ± 2.9	6.06 ± 2.6	0.02	3.99 ± 3.0	2.99 ± 2.0	0.4
RA middle seg	3.40 ± 2.1	4.25 ± 2.7	0.5	2.51 ± 1.9	3.71 ± 2.9	0.3	3.07 ± 2.1	3.11 ± 2.0	0.8

SD, standard deviation; SR_S, systolic; SR_E, early diastolic velocity; SR_A, late diastolic velocity; LA, left atrial; seg, segment; IAS, interatrial septum; RA, right atrial

with the TD-derived IVRT of the IVS excursion (r = –0.6; p = 0.03) (Fig. 3).

Discussion

As a genetic disease of the myocardium, HCM is associated with diastolic ventricular dysfunction and characterized by cardiac hypertrophy, myocyte disarray, interstitial fibrosis, and LV dysfunction, with LV and/or RV hypertrophy. If asymmetric, the hypertrophy usually is greatest within the IVS [19]. The atrial function in patients with HCM has not been extensively studied, and little is known about the characteristics of regional atrial contractile function. The atrial wall contractile function is complex, containing as it does both active and passive components of wall motion. It also is markedly influenced by overall heart motion [15] and different for each atrium, varying with atrial size, loading conditions, atrial rhythm, and atrial contractility and compliance.

The velocity profiles for longitudinal regional atrial motion could be recorded from the walls of both atria using tissue Doppler imaging. In addition to the annular systolic and diastolic wall velocities, analysis of the deformation rate (strain rate) for the atrial wall through the three phases of the heart cycle may provide more information on the atrial contractile function.

Longitudinal Atrial Segmental Velocity Profiles

The atrial velocity curve is characterized by the three main waves S, E, A, which coincide, respectively, with the ventricular systolic, early diastolic, and late diastolic periods. The normal pattern of atrial longitudinal regional velocities (Table 5) is characterized by the finding that the annular segments of the left and right atrial and IAS walls have higher velocities than those of the corresponding middle segments in control subjects and patients with HCM (Fig. 4). The additive translation from cardiac

Table 5 Normal distribution of the longitudinal atrial segmental velocities (cm/s)

Parameters	Velocity in controls (mean ± SD)		
	S	E	A
LA annular seg	6.71 ± 1.67	8.69 ± 3.94	5.14 ± 2.46
LA middle seg	4.29 ± 2.53	3.72 ± 2.58	3.12 ± 2.29
IAS annular seg	5.23 ± 1.07	8.77 ± 1.09	8.77 ± 1.09
IAS middle seg	4.28 ± 2.03	6.01 ± 1.96	5.36 ± 1.84
RA annular seg	7.59 ± 2.04	5.79 ± 2.81	5.30 ± 2.97
RA middle seg	2.95 ± 3.13	2.18 ± 2.73	2.07 ± 2.58

SD, standard deviation; S, systolic; E, early diastolic velocity; A, late diastolic velocity; LA, left atrial; seg, segment; IAS, interatrial septum; RA, right atrial

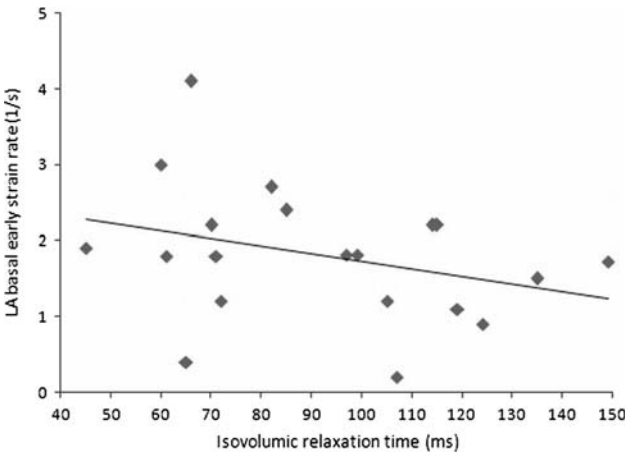

Fig. 3 Correlation between the early strain rate in left atrial (LA) and isovolumic relaxation time (IVRT) of the interventricular septum (IVS) in patients with hypertrophic cardiomyopathy (HCM)

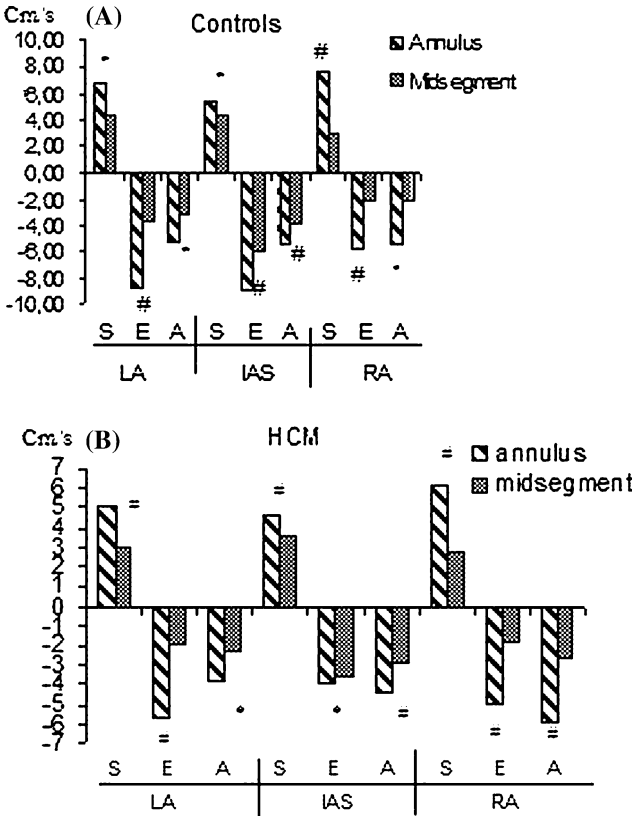

Fig. 4 Differences in atrial velocities between annular and middle segments for each wall in control subjects (**A**) and patients with hypertrophic cardiomyopathy (HCM) (**B**). LA, left atrial; IAS, interatrial septum; RA, right atrial; S, systolic; E, early diastolic peak velocity; A, late diastolic peak velocity $*p < 0.05$, $\#p < 0.01$

motion may contribute to increased velocities in the atrial segment adjacent to the annulus. The apical atrial segments are rather stationary and make no contribution to the atrial motion and active contraction, and there is an inhomogeneous velocity distribution between the different segments and between the lateral atria walls and IAS consistent with the findings of Thomas et al. [15].

In patients with HCM, the lateral left and right atrial walls showed no changes in the regional wall systolic and diastolic velocities compared with control subjects. The only remarkable velocity changes we found were in peak early velocity of the IAS at the annular ($p = 0.001$) and middle segments ($p = 0.001$) of the IAS wall in the patients with HCM compared with the healthy control subjects (Table 3). Because the atrial walls contribute no motion during the ventricular early diastolic phase, the reduction of the early IAS velocity may reflect the impaired and delayed early diastolic relaxation of the IVS, as shown by the prolongation of the IVRT of the IVS. The late velocity, which correlates with atrial pumping, was not reduced in the patients with HCM, which may reflect a compensatory capacity by the late active atrial contraction. In addition, the changes in regional velocities may not reflect the active contractility of the myocardium because the point velocity of the specific atrial region does not differentiate between active contraction and passive motion related to cardiac translation [15]. Thus, strain rate patterns, which are not influenced by the whole heart movements, may provide more information on the intrinsic regional atrial wall contractile function.

The Pattern of the Strain Rates

Strain rate, the velocity of deformation, calculates spatial differences in tissue velocities between adjacent myocardial regions and represents the regional contractility [1, 4]. The quality of regional strain rate is highly dependent on both the ultrasonic methodology and the acquisition frame rate. The atrial strain rate curve was characterized by the three main waves, SR_S, SR_E, SR_A, which coincide,

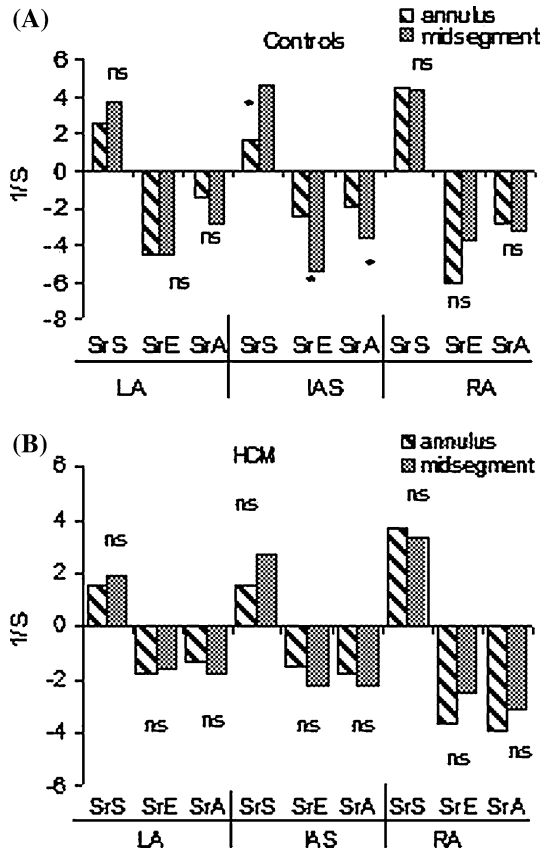

Fig. 5 Significant differences in the distribution of strain rate in wall segments of the interatrial septum (IAS) in control subjects (**A**) and a similar distribution in patients with hypertrophic cardiomyopathy (HCM) (**B**). SrS, peak systolic strain rate; SrE, peak early diastolic strain rate; SrA, peak late diastolic strain rate; LA, left atrial; RA, right atrial; NS, insignificant $*p < 0.05$

respectively, with the ventricular systolic, early diastolic, and late diastolic periods (Fig. 2).

In our studies, the strain rate profile in the control subjects was distributed in a homogeneous pattern in the LA and RA walls, but the IAS strain rate showed inhomogeneous distribution. The annular segment of IAS had lower systolic and diastolic strain rate values than the middle segment (Fig. 5A), whereas velocities decreased from the annulus to the middle segment in both the control subjects and the patients with HCM. The pattern of strain rate distribution was opposite that of the velocity in the IAS of the healthy control subjects. However, the systolic, early, and late diastolic strain rate values showed a loss of these strain rate differences between the interatrial annular and the middle segments in the patients with HCM (Fig. 5 B).The apical segments, as mentioned earlier, are less contractile and may not have contributed significantly to the actual deformations, in either the control subjects or the patients with HCM.

The Regional Atrial Performance in Patients With HCM

A pattern of impaired ventricular relaxation was previously evidenced by TDI-derived M-mode in patients with HCM [2]. The TDI technique is a reliable noninvasive tool for evaluating regional ventricular function, and the regional longitudinal function of LV is reduced in patients with HCM, as reported in previous studies [4, 10, 11, 13, 18]. In our study, TDI of the left atrium showed a slight reduction of myocardial velocity at the annular and middle segments of the free left and right atrial and IAS walls. The main reduction in tissue velocities occurred at the annular and middle segments of the IAS wall and in the free left atrial wall, where a significant reduction in the atrial strain rates also were found.

Our findings show for the first time that regional atrial function impairment is inhomogeneous in patients with HCM. The reduced strain rate SR_S and SR_E peaks may reflect the decreased LV function and increased atrial stiffness because the left atrium has no active motion in the ventricular systolic and early diastolic periods. The atrial contraction (SR_A) did not show any impairment in the patients with HCM compared with the control subjects, which may indicate normal or enhanced atrial pumping contraction in patients with HCM to compensate for the reduced ventricular diastolic capacity.

The delayed ventricular relaxation in patients with HCM represented by prolongation of the IVS IVRT is negatively correlated with the deformation in the left atrial wall. The reduced regional atrial function is not significantly related to the degree of the IVS thickness in patients with HCM. However, the pressure load of the LV represented by the pressure gradient across the LVOT is negatively correlated with the regional atrial strain rate and early atrial wall velocities. This may indicate that increased pressure might lead to more impairment of the LA regional atrial function in patients with HCM. Nevertheless, the grade of the IVS thickness certainly influences the ventricular and atrial diastolic function.

Our data showed new findings on the restricted function of the atria, mainly the LA, as a consequence of the LV diastolic impairment due to hypertrophic changes involving the IVS, which lead to pressure overload in the LV of patients with HCM. Whether these findings are related to hemodynamically dependent changes or to pathophysiologic and morphologic processes of the HCM involving the atrial walls needs further clinical study.

Conclusion

According to the findings of this study, myocardial ventricular hypertrophy and altered longitudinal ventricular function seem to cause significant reduction in the regional

function of the atria, mainly the left atrium. The early velocity and systolic and early strain rate of the IAS and lateral LA walls may be suitable parameters for assessing the regional LA function in patients with HCM. Further studies to compare these findings with other methods are needed to evaluate the clinical utility of these results.

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