Velocity vector imaging in assessing myocardial systolic function of hypertensive patients with left ventricular hypertrophy

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BACKGROUND: To date, most studies about strain and strain rate (SR) are based on Doppler tissue imaging (DTI), which is dependent on the angle between ultrasonic scan line and tissue. Velocity vector imaging (VVI) is a new echocardiographic method based on two-dimensional gray scale imaging, which is angle-independent and can provide more information about cardiac function than DTI.

OBJECTIVES: To assess regional myocardial SR in hypertensive patients with left ventricular hypertrophy (LVH) but normal global ejection fraction (GEF) and fractional shortening (FS) using VVI.

METHODS: Using VVI, two-dimensional images were performed in 20 hypertensive patients with LVH and 20 normal control subjects. The segmental systolic peak SR (SRs) in the short-axis view and the apical SRs in the long-axis view were analyzed by offline software.

RESULTS: The segmental SRs in the long-axis and short-axis views were significantly lower in the LVH group than in the corresponding segments of the control group. There was no significant difference between the circumferential SRs of different segments in the short-axis view in the LVH and control groups. The circumferential SRs decreased significantly from the endocardium to the middle layer of the myocardium in the short-axis view in the LVH group and in the control group.

CONCLUSIONS: Hypertensive patients with LVH may have regional LV systolic function impairment despite having normal GEF and FS. The GEF and FS were not the decisive factors of myocardial systolic function in the present study. There was an obvious systolic gradient from the endocardium to the middle layer of myocardium in circumferential SRs in the short-axis view. VVI can be used to accurately recognize and quantify abnormalities of regional myocardial deformation.

Key Words: Left ventricular hypertrophy; Systolic peak strain rate; Velocity vector imaging

In recent years, strain and strain rate (SR) have been shown to be a useful tool in quantifying cardiac function (1-3). SR imaging based on Doppler tissue imaging (DTI) can only quantify the axial component of motion, so it is angle-dependent; once the angle between the ultrasonic beam and the tissue are beyond a certain range, often more than 20°, the results lose validity (4,5). Velocity vector imaging (VVI) is a novel echocardiographic imaging technique based on routine two-dimensional gray scale echocardiographic images; therefore, it is angle-independent in

Imagerie vectorielle de vitesse dans l'évaluation du fonctionnement systolique myocardique chez des patients hypertendus, atteints d'hypertrophie ventriculaire gauche

CONTEXTE : La plupart des examens sur la déformation et la vitesse de déformation (VD) reposent, jusqu'à maintenant, sur l'imagerie Doppler tissulaire (IDT), qui est dépendante de l'angle formé par la ligne de balayage ultrasonore et les tissus. L'imagerie vectorielle de vitesse (IVV) est une nouvelle technique d'échocardiographie qui repose sur une échelle bidimensionnelle de gris, qui est indépendante de l'angle et qui fournit plus de renseignements sur le fonctionnement du cœur que l'IDT.

BUT : L'étude avait pour but d'évaluer, au moyen de l'IVV, la vitesse de déformation myocardique régionale chez des patients hypertendus présentant une hypertrophie ventriculaire gauche (HVG) mais ayant une fraction d'éjection globale (FEG) et une fraction de raccourcissement (FR) normales.

MÉTHODE : Nous avons procédé à la prise d'images bidimensionnelles à l'aide de l'IVV chez 20 patients hypertendus présentant une HVG et chez 20 sujets témoins normaux; nous avons ensuite analysé la VD systolique segmentaire de pointe sur les coupes transversales et la VD apicale sur les coupes longitudinales à l'aide d'un logiciel utilisé en mode autonome.

RÉSULTATS : Les VD segmentaires sur les coupes longitudinales et les coupes transversales étaient significativement plus faibles dans le groupe de patients atteints d'HVG que dans le groupe de sujets témoins. Par contre, il n'y avait pas de différence importante en ce qui concerne les VD circonférentielles de différents segments sur les coupes transversales entre les deux groupes. Une diminution significative des VD circonférentielles depuis l'endocarde jusqu'à la couche moyenne du myocarde a été observée sur les coupes transversales dans le groupe de patients atteints d'HVG et dans le groupe de sujets témoins.

CONCLUSIONS : Les patients hypertendus, atteints d'une HVG peuvent présenter un dysfonctionnement systolique ventriculaire gauche régional malgré une FEG et une FR normales. Ces deux derniers paramètres ne constituaient pas des facteurs décisifs de fonctionnement systolique myocardique dans la présente étude. Un gradient systolique évident des VD circonférentielles, depuis l'endocarde jusqu'à la couche moyenne du myocarde, a été observé sur les coupes transversales. L'IVV peut aider à détecter et à quantifier précisément les anomalies liées à une déformation myocardique régionale.

VVI can be used to study cardiac mechanics and quantify global and regional cardiac function with abundant parameters. It quantifies longitudinal and circumferential myocardial deformation, and the latter is important in understanding myocardial motion in the short-axis direction.

Left ventricular hypertrophy (LVH) is an independent risk factor and can result in severe cardiovascular events such as heart failure, sudden death and others (6). Researchers have reported significantly decreased longitudinal LV systolic function in patients

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TABLE 1
Patient and echocardiographic characteristics in the control group and the left ventricular hypertrophy (LVH) group

Characteristic	Control group (n=20)	LVH group (n=20)	P
Age (years)	55±8	53±6	NS
Men/women (n)	10/10	12/8	NS
Systolic blood pressure (mmHg)	120±8	161±14*	0.000
Diastolic blood pressure (mmHg)	74±4	99±9*	0.000
Septum wall thickness (cm)	0.8±0.1	1.4±0.2*	0.000
LV posterior wall thickness (cm)	0.8±0.1	1.4±0.1*	0.000
LV end-diastolic diameter (cm)	4.6±0.3	4.7±0.4	NS
LV end-systolic diameter (cm)	2.9±0.4	2.8±0.4	NS
LV mass index (g/m ²)	92±15	146±30*	0.000
Left atrial diameter (cm)	3.2±0.2	4.3±0.3*	0.000
Global ejection fraction (%)	65±8	68±7	NS
Fractional shortening (%)	39±6	40±5	NS
E/A ratio	1.20±0.30	0.78±0.25*	0.000

Data are presented as mean ± SD. *P<0.001 LVH group versus the control group. E/A Early diastolic velocity to late diastolic velocity; NS Not significant

with hypertension, most pronounced in patients with concentric and eccentric hypertrophy (7). We used angle-independent VVI to study the segmental systolic SR (SRs) from longitudinal and circumferential directions to observe whether hypertensive patients with LVH but normal global ejection fraction (GEF) and fractional shortening (FS) had regional systolic abnormalities.

METHODS

Patients

Twenty hypertensive patients with LVH and 20 age- and sex-matched normal control subjects were included in the present study. Eight patients in the LVH group had undergone coronary angiography. No significant differences were found between the two groups in terms of age or sex (Table 1). The diagnosis of hypertension was based on the standards outlined in the Seventh Report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (8). Patients with secondary hypertension, valvular disease, hypertrophic cardiomyopathy, previous myocardial infarction, or history of angina pectoris or diabetes mellitus were excluded. Each study participant gave written, informed consent. The study was approved by the local scientific ethics committee.

Conventional echocardiography

M-mode echocardiography was performed with an ultrasound machine (3.0 MHz transducer; Sequoia 512, Siemens, USA). The following conventional echocardiographic parameters were measured to determine the difference between the LVH group and control group subjects: the sample volume was placed between the tips of the mitral leaflets to acquire the mitral early diastolic velocity to late diastolic velocity (E/A) ratio by pulsed Doppler, and FS, interventricular septum thickness, LV posterior wall thickness, LV end-diastolic diameter and LV end-systolic diameter (LVESD) were measured according to the standards of the American Society of Echocardiography (9). LV mass was calculated with a validated anatomical formula, and relative wall thickness was calculated as the following ratio: (2 × posterior wall thickness in diastole)/LV diameter (10). LVH was considered to be present if the LV mass index was greater than 134 g/m² in men or greater than 110 g/m² in women (11). Increased relative wall thickness was indicated by a

Figure 1 A Original endocardial regions of interest in the short-axis view. B Velocity vectors in the short-axis view after the calculation process

ratio greater than 0.43. Increased LV mass index and relative wall thickness identified concentric LVH (12). GEF was estimated by the modified biplane Simpson's method (13), and all the patients had normal GEF and FS (Table 1).

VVI

Two-dimensional images of apical four- and two-chamber views, as well as the apical long-axis view and the LV parasternal short-axis view (at the root of papillary muscle), were recorded with the same ultrasound machine. Three consecutive cardiac cycle loops were recorded at end expiration. Images were recorded with an acoustic method that provides information frame by frame. The frame rate was kept between 70 Hz and 100 Hz. The imaging sector was optimized to complete visualization of the LV. The 16-segment LV model of the American Society of Echocardiography (13) was used to analyze regional cardiac function. The long-axis ventricular walls were divided into three levels: base, middle and apical. In the long-axis view, only the apical level was analyzed, which has rarely been studied before. VVI data were analyzed by Research-Arena program (TomTec Imaging Systems, Germany).

Using a point-click approach, a number of points were placed along the endocardium or the middle layer of myocardium. The number of points was decided depending on the radius of the endocardium. After performing the calculation process, three new points were inserted between every two former points, and velocity vectors with different directions and sizes were produced automatically (Figure 1). Each point was called a region of interest (ROI), which was identified and tracked frame by frame. The geometric shift of each ROI represents local tissue motion. Local velocity was calculated as the shift of the ROI divided by time between frames, which equals the two-dimensional velocity vector. When the ROI moves with the extending or shortening of myocardium throughout the cardiac cycle, the displacements of every ROI and the velocity of deformation in every frame are the strain and SR, which were calculated automatically. Two points were selected in each segment of the short-axis view or at the apical levels of the long-axis view; their averaged SRs value was taken as the SRs of each segment (Figure 2). In the VVI analysis, a relatively static reference point was placed at the apical location in the long-axis view and at the centre of ventricle in the short-axis view.

Statistics

All results are expressed as mean ± SD, and differences between groups were tested by independent samples *t* tests. SPSS software (version 10.0, SAS Institute, USA) was used for statistical analysis. P<0.05 was considered to be significant.

Figure 2 A Colour M-mode echocardiography map showing velocity (Vel), strain and strain rate (SR) in the short-axis view. B Time curves of velocity, strain and strain rate

RESULTS

Interventricular septum thickness, LV posterior wall thickness, LV mass index and the E/A ratio were significantly different between the two groups, but there was no significant difference in GEF and FS between the two groups (Table 1). The circumferential SRs acquired in the short-axis view of the LVH group was significantly lower than in the control group, and circumferential SRs decreased significantly from the endocardium to the middle layer of myocardium in the short-axis view in the LVH group and in the control group (Table 2). The longitudinal apical SRs acquired in the long-axis view was also significantly lower than in the corresponding segments in the control group (Table 3).

DISCUSSION

SR imaging based on one-dimensional DTI has only been used to study the myocardial motion of the base and middle levels in the long-axis view and the motion of the anterior septum and posterior wall in the short-axis view because of the angle dependence of the Doppler technique (14). Early two-dimensional strain and SR images based on the speckle tracking echocardiographic (STE) technique were developed to overcome the angle dependence of DTI (15,16). In the past, DTI and STE have been time consuming, and the high variability in DTI, as well as the computational complexity and immature algorithm presented by early STE, limited their application to the human heart (17-19). Recently, the STE technique has been improved and used effectively in the

study of cardiac function. Kaluzynski et al (20) initially demonstrated how speckle tracking can generate regional strain maps from ultrasound images using a phantom model. Reiser et al (21) used strain measurements by speckle tracking to calculate the global strain in myocardial infarction patients, and the results showed a close correlation to a wall motion score index. Amundsen (22) validated that STE was not angle-independent and had good correlation with sonomicrometry, and tagged magnetic resonance imaging (MRI) in myocardial strain measurement.

VVI is an advanced echocardiographic method that is based on STE. In gray scale images, interference by backscattered ultrasound from neighbouring structures results in a random, speckled pattern. This gives each small area a unique pattern that remains relatively constant from one frame to the next (20). With the optimized pattern-matching algorithm, VVI can accurately track these speckles frame by frame, and through reconstructing the deformation and motion, the motion of flow and tissue can be analyzed. The advantage of VVI is that it is self-updating. Special reference settings are applied, including valvular annulus, chamber borders and tissue motion, as well as the relatively static reference point provided by the software. All of the above make the tracking process more precise. VVI is faster than conventional STE, and obtaining each patient's parameters may take approximately 5 min, which is less time than in routine STE study. The velocity, strain and SR of every frame can be exported manually or automatically with Excel (Microsoft, USA). The segmental EF contribution of each segment

TABLE 2

The difference of circumferential systolic strain rate of each segment between the endocardium and the middle layer in the short-axis view (at the root of papillary muscle level)

Group	Location	Inferior septum	Lateral	Anterior septum	Posterior	Anterior	Inferior
Control group	Endocardium	2.23±0.36	2.21±0.30	2.36±0.32	2.15±0.40	2.22±0.40	2.15±0.45
	Middle layer	1.46±0.29*	1.39±0.24*	1.35±0.27*	1.41±0.22*	1.45±0.31*	1.36±0.27*
Left ventricular hypertrophy group	Endocardium	1.80±0.25**	1.68±0.25**	1.46±0.31**	1.66±0.25**	1.30±0.28**	1.67±0.33**
	Middle layer	0.79±0.18***	0.78±0.16***	0.75±0.21***	0.74±0.13***	0.75±0.20***	0.78±0.20***

* $P<0.001$ Endocardium versus the middle layer in the control group; ** $P<0.001$ Endocardium in the left ventricular hypertrophy group versus the control group;

*** $P<0.001$ Endocardium versus the middle layer in the left ventricular hypertrophy group

TABLE 3

Comparison of longitudinal systolic strain rate at the apical level of the long-axis view

Group	Inferior septum	Lateral	Anterior septum	Posterior	Anterior	Inferior
Control group	1.49±0.29	1.55±0.25	1.60±0.36	1.47±0.31	1.66±0.24	1.56±0.32
Left ventricular hypertrophy group	0.85±0.28*	0.80±0.25*	0.83±0.20*	0.96±0.27*	0.86±0.24*	0.87±0.27*

* $P<0.001$ Control group versus left ventricular hypertrophy group in each segment

may be obtained with VVI, which may not be acquired with the conventional echocardiographic method. Time to peak velocity, time to peak strain and SR, especially time to 75% and 50% strain and SR, enable the abnormalities in different systolic phases to be recognized more easily.

It has been proven that LVH is a severe complication of essential hypertension and an important risk factor for heart failure, acute myocardial infarction and sudden death. Stimulated by pressure and volume overload, as well as by neuroendocrine factors, the proliferation of cardiac fibroblasts and the hypertrophy of myocardial cells comprises the vital pathophysiological basis that leads to LVH (23,24). Abnormal diastolic LV filling is frequently found in patients with hypertension, while LV systolic function is commonly considered normal if the GEF and FS are normal. But the GEF and FS only reflect the global cardiac contractile function and do not take the regional systolic abnormality into consideration. With this new echocardiographic method (ie, VVI), we found that regional systolic dysfunction does exist in hypertensive patients with LVH, even if their global systolic parameters were normal. The longitudinal apical SRs were lower in the LVH group with normal GEF and FS than the corresponding segments in the control group, a finding that was consistent with the DTI results of Poulsen et al (7). In the short-axis view, decreased circumferential segmental SRs was also found in these LVH patients. LV geometry is closely related to LV performance, especially in hypertension. In hypertensive patients, coronary flow reserve has been shown to be impaired with changes in LV geometry (25). The presence of increased perivascular and interstitial fibrosis has previously been demonstrated in patients with hypertension (26,27). The derangement of the myocardial fibre array and fibrosis may interfere with the contractile ability of myocardial fibres, and the degree of myocardial fibrosis can predict diastolic and systolic dysfunction. With the progression of LVH, myocardial mass and wall thickness increase to maintain ejection volume, but the amount of blood ejected per unit of myocardial mass may be lower, potentially resulting in abnormalities in regional systolic function. Dumesnil and Shoucri (28) used a cylindrical model to validate that wall shortening and thickening, which depend on the changes in LV geometry, contribute to LV ejection. They found that patients with concentric LVH had an increase in wall thickness and a decrease in ejection efficiency, even though they had preserved GEF. They thought that this decrease might have been due to an increase in subendocardial wall stress and radial stress.

Subendocardial fibres have decreased shortening and thickening, and the rest of the wall needs to compensate for this insufficiency; therefore, greater muscle mass is needed to eject the same quantity of blood. Manaka et al (29) found that myocardial systolic impairment in hypertensive LVH may originate at the endocardial side and significantly move to the epicardium compared with control, and the impairment may progress with increased LVH, but this study was limited to the longitudinal direction. With VVI, we found that the SRs decreased significantly from the endocardium to the middle layer of myocardium in the circumferential direction in the LVH group and in the control group. Naturally, we suspected that the shortening of the myocardium increased from the epicardium to the endocardium, because the LV wall is circular in the short axis-view; therefore, the shortening effects of the layers from the outside toward the inside should be additive because the myocardium is incompressible. This assumption, however, should be further confirmed with mathematical or other methods.

Vannan et al (30) used VVI to assess LV dynamics in a patient with heart failure who did not respond to cardiac resynchronization therapy (CRT). The investigators found that persistent heterogeneity of circumferential strain can account for the nonsustained improvement in LV systolic performance after CRT, even though the longitudinal and radial velocities of LV were synchronized. The difference of time to peak strain and SR between the opposite ventricular walls shown by VVI can predict immediate and long-term response to CRT. The motion of the myocardium is complicated and LV torsional deformation is sensitive to changes in both regional and global LV function. DTI can measure LV torsion, but for severely deformed LVs, a method completely free from the angle dependency of the ultrasound beam line is needed to overcome these problems (31). Thus far, MRI tagging has been the only clinically available method, and implementation has therefore been limited by complexity and cost (32). Helle-Valle et al (33) demonstrated that magnitude, timing and dynamics of regional LV rotation and torsion can be measured accurately by STE. VVI based on STE can measure LV torsion independently of angle, but it still needs to be validated with another efficient method, such as MRI.

Using the angle-independent characteristics of VVI to study regional systolic function from different directions in hypertensive patients with LVH will improve our understanding of hypertensive hypertrophy mechanisms from the aspect of mechanics. Our results also showed that the circumferential SRs in the short-axis view was higher than that in the long-axis view in the LVH

group and in the control group. We did not test this difference statistically, but our results may imply that myocardial deformation in the short-axis direction contributes more than in the long-axis direction in myocardial contraction. It is well known that the LV wall consists of three layers of myocardial fibres that run in different directions, and the middle layer is nearly circumferential. We believe that the force component of myocardial contraction in the circumferential direction may be the dominant force that makes myocardial circumferential shortening greater than in longitudinal shortening.

Limitations

Because VVI is based on two-dimensional gray scale imagery, a high-quality image is needed, and special care must be taken to draw the outline of an ROI. The deformation of myocardium is three-dimensional, but currently, VVI only offers a two-dimensional plane. Because angiography was not performed in every participant, the presence of coronary artery disease cannot be ruled out completely. The sample of patients in the present study was small, so further investigation is needed to confidently elucidate the precise mechanism of myocardial contraction.

CONCLUSION

VVI is an effective angle-independent echocardiographic method that can be used to assess regional myocardial dysfunction more conveniently and accurately. With VVI, regional systolic dysfunction can be found in hypertensive LVH patients who have normal GEF and FS.

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