Flow velocity distributions in the left ventricular outflow tract and in the aortic annulus in patients with localized basal septal hypertrophy

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Background The velocity distributions in the left ventricular outflow tract and in the aortic annulus in normal subjects and certain cardiac patients are skewed, with the highest velocity along the anterior wall and septum. An abnormal anatomical structure of the interventricular septum changes the shape of the left ventricular outflow tract, and may consequently change the pattern of velocity distribution.

Methods The cross-sectional velocity distributions in the left ventricular outflow tract and in the aortic annulus were constructed by using Doppler colour flow mapping in nine patients with localized basal septal hypertrophy, and in 10 normal subjects. The apical long axis view was used.

Results In the studied patients, the velocity distributions in the left ventricular outflow tract and in the aortic annulus were skewed in a different way from those in normal subjects. The relative location of the maximal velocity on the cross-sectional diameter of the flow channel changed from one level to another. At the point of maximal basal septal hypertrophy, the velocity distribution was most skewed with the highest velocity along the anterior wall (e.g. basal septum). Distal to this level, the highest velocities of the skewed velocity profiles were gradually located closer to the central part of the flow channel. According to the time-velocity integral profile at the level of the aortic annulus, the pattern of skewness (in terms of the difference of the average time-velocity integrals between the anterior and posterior halves of the diameter) was significantly different between the normal and patient groups (5.51 ± 3.55 cm vs 0.03 ± 2.07 cm; P<0.01), while the extent of skewness (in terms of the ratio of the maximal to the cross-sectional mean time-velocity integrals) was close between two groups (1.36 ± 0.28 vs 1.27 ± 0.13; P>0.05).

Conclusion Localized basal septal hypertrophy significantly affects velocity distributions in the left ventricular outflow tract and in the aortic annulus.

Key Words: Velocity distribution, localized basal septal hypertrophy, Doppler colour flow mapping, pulsed Doppler.

Introduction

It has been found that velocity distributions in the left ventricular outflow tract and in the aortic annulus in normal subjects and certain cardiac patients are skewed, with the highest velocity along the anterior wall and septum. Our previous studies suggested that there were at least two major factors responsible for velocity distributions in the left ventricular outflow tract and in the aortic annulus: the anatomical structure of the left ventricular outflow tract and the pattern of flow convergence in the outflow tract.

Localized basal septal hypertrophy can change the anatomical shape of the left ventricular outflow tract significantly, and probably change the cross-sectional velocity distribution in the outflow tract compared to that in normal subjects. Localized basal septal hypertrophy is usually found in elderly patients with systemic hypertension, or aortic stenosis, but may even be found in normal subjects.

This study was designed to investigate velocity distributions in the left ventricular outflow tract and in the aortic annulus in this special patient category, as compared with those in normal subjects. The effect of velocity distribution on the cross-sectional mean velocity measurement was evaluated. Another objective was to further elucidate the mechanisms responsible for velocity distributions in the left ventricular outflow tract and in the aortic annulus.
Materials and methods

Study subjects

The study was approved by the regional ethics committee, and each study subject gave his or her informed consent.

Ten normal male subjects with a mean age of 28 ± 8 years (range 20 to 42 years) constituted the normal group. No subject had a history of cardiac disease, and blood pressure, electrocardiogram and echocardiogram were normal.

Patients were selected on the basis of: (1) localized basal septal hypertrophy (Fig. 1), in each case this occurred in the basal part of the septum (end-diastolic thickness >1.4 cm), with a dune-like structure protruding into the left ventricular outflow tract[10,11]; (2) absence of subaortic obstruction or subaortic pressure gradient on two-dimensional, M-mode or pulsed Doppler echocardiography, or on Doppler colour flow mapping.

The patient group included four males and five females, with a mean age of 74 ± 13 years (range 47 to 89 years). One patient had no other diagnosis than septal hypertrophy. In others the hypertrophy was accompanied by angina pectoris (n=2), angina pectoris and previous myocardial infarction (n=1), hypertension (n=1), and mild aortic stenosis (n=4). Mitral regurgitation was observed in three patients, and mild aortic regurgitation in one patient. All studied patients had a regular sinus rhythm without any bundle branch block. The cardiac functional classes were grade I (n=5) and II (n=4) according to the standard of the New York Heart Association.

Velocity distribution study by using Doppler colour flow mapping

Data acquisition

A combined two-dimensional echocardiographic and Doppler colour flow mapping instrument (CFM 750, Vingmed Sound A/S, Oslo, Norway) with a 3-25 MHz mechanical annular array transducer was used. The instrumental settings were the same as those in a previous study[11]. With the patient in the left semi-recumbent position, the standard apical long axis view was visualized. A minimal intercept angle between the radial direction of the colour flow sector and the direction of blood flow in the aortic annulus was obtained.

The instrument was set to the ECG-triggered mode, where the two-dimensional tissue image and the Doppler colour flow sector were updated once every cardiac cycle. To cover a full systole, a series of sequentially 20 ms delayed Doppler colour flow maps were recorded. The recording procedure is described in detail elsewhere[2,3]. During data acquisition, patients were asked to hold their breath at the end of expiration.

Figure 1 Typical anatomical shape of the left ventricular outflow tract in normal subjects (A) and in patients with localized basal septal hypertrophy (B). Line a is the longitudinal axis of the aortic annulus; line b is parallel to line a and crosses the point where the aortic cusp attaches to the septal side of the aortic annulus; h is the height of the protruding hypertrophic basal septum from line b; d is the distance from the peak point of the protruding basal septum to the level of the aortic annulus. In normal subjects, the value of h is almost zero. AO=aorta; MV=mitral valve; SE=interventricular septum.

All recorded ultrasound data were transferred from the replay memory of the ultrasound instrument to an external computer (Macintosh llx, Apple Computer Inc., Cupertino, California, U.S.A.) for further processing.

Data processing

A previously described time-interpolation method[12-14] was used to correct for the possible time-distortion of Doppler colour flow mapping. Velocity distribution could then be evaluated. By means of the computer, a line was drawn at the level of interest, perpendicular to the blood flow direction at the measured level, and an angle-corrected velocity profile could be obtained (Fig. 2). The velocities in the colour flow map along the line were extracted as 70 numbers, and saved in a computer work sheet. After all consecutive frames throughout systole had been processed, an array of velocities was obtained. The resulting cross-sectional velocity distribution at the studied level was visualized in three-dimensional plots of velocity vs time in systole and position along the diameter of the flow channel.

The level of the aortic annulus was identified from the tissue image and used as a reference in defining other levels of velocity distribution analysis in the left ventricular outflow tract. To observe the dynamic change of the velocity profile in the direction of flow in the left ventricular outflow tract, peak flow velocity profiles (the velocity profile at peak systole) were measured at levels of 3.0 cm, 2.5 cm, 2.0 cm, 1.5 cm, 1.0 cm, 0.5 cm proximal to the aortic annulus, and at the aortic annulus. To quantify the skewness of the cross-sectional velocity profile, two parameters were calculated: (1) the ratio of maximal velocity to cross-sectional mean velocity (max/mean V) — reflecting the extent of skewness; (2) the difference between the average velocities on the anterior and posterior halves of the diameter of the flow channel (ΔV). It mainly reflected

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Figure 2  Two-dimensional tissue image (A), Doppler colour flow map (B) in the apical long axis view, and the peak flow velocity profiles at 1-5 cm (C) and 0-5 cm (D) proximal to the aortic annulus from patient no. 8. In panel A, the arrow indicates the localized basal septal hypertrophy, which occurs without obstructing the left ventricular outflow tract. In panel B, the colour flow map shows the blood flow in the left ventricular outflow tract at peak systole. The direction of flow is away from the transducer (in blue), and the red indicates flow with velocities higher than the Nyquist limit. In the coordinates of panel C and D, the abscissa represents the diameter (cm) of the flow channel at the measured level, from the anterior side to the posterior side. The ordinate represents flow velocity (cm s⁻¹). AO=aorta; LA=left atrium; LV=left ventricle.

the pattern of skewness (e.g. the symmetry of the velocity profile). If the value of ΔV was positive, the profile was skewed with the highest velocity along the anterior wall, if negative, the profile was skewed with the highest velocity along the posterior wall, and zero indicated a symmetrical profile.

Based on the array of velocities saved in the computer work sheet, the time-velocity integral could be calculated for each of 70 points along the diameter of the flow channel. In this way, the time-velocity integral profile was obtained for three levels: 1-0 cm and 0-5 cm proximal to the aortic annulus, and at the aortic annulus. The ratio of the maximal time-velocity integral to the cross-sectional mean time-velocity integral (max/mean TVI) was calculated. The difference between the average time-velocity integrals on the two sides of the middle point of the diameter (ΔTVI) was calculated as for the peak flow velocity profile.

The beat-to-beat variation and variability of repeated measurements (intra- and inter-observer errors) were evaluated as in previous similar studies[5,8] and are therefore not included in this study.

Velocity sampling by using pulsed Doppler echocardiography in the patient group

In the apical long-axis view, the pulsed Doppler velocity spectrum was obtained from the anterior, middle and posterior areas along the diameter of the flow channel at the level of 0-5 cm proximal to the aortic annulus. A small sample volume (axial dimension <3 cm) was used. At each sampling site, the velocity was angle-corrected by adjusting the intercept angle between the ultrasound beam and the assumed flow direction. For each area, time-velocity integrals were measured from the maximal velocity tracing of the flow spectra in five consecutive beats, and the average value was calculated.

Other measurements

In the patient group, the height of the protruding hypertrophic basal septum (h) and the distance from the peak point of the protruding basal septum to the level of the aortic annulus (d) were measured in a two-dimensional tissue image (Fig. 1B). The thickness of the basal part of the interventricular septum (IVSb) was also measured.

In both normal and patient groups, the following parameters were measured at end-diastole in the M-mode tracing of the left ventricle[15]: (1) thickness of the middle part of the interventricular septum (IVSm); (2) left ventricular diameter (LVEDD); and (3) the thickness of the left ventricular posterior wall (LVPW). Left ventricular ejection fraction (LVEF) was measured according to the modified Simpson's rule in the apical
peak flow velocity profiles in the left ventricular outflow tract were also skewed, but the relative location of the anterior wall at different levels. The anatomical parameters of the interventricular septum in the patient group are presented in Table 2. Normal group

<table>
<thead>
<tr>
<th>HR (beats·min⁻¹)</th>
<th>Normal group (n=10)</th>
<th>Patient group (n=9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>59 ± 7</td>
<td>63 ± 7</td>
<td></td>
</tr>
<tr>
<td>D-AA (mm)</td>
<td>24.1 ± 2.4</td>
<td>23.2 ± 3.3</td>
</tr>
<tr>
<td>IVSm (mm)</td>
<td>9.8 ± 1.4</td>
<td>13.1 ± 1.1**</td>
</tr>
<tr>
<td>LVEDD (mm)</td>
<td>53.2 ± 5.0</td>
<td>50.6 ± 6.2</td>
</tr>
<tr>
<td>LVPW (mm)</td>
<td>10.2 ± 1.1</td>
<td>12.1 ± 2.4*</td>
</tr>
<tr>
<td>EF (%)</td>
<td>72.2 ± 7.0</td>
<td>74.0 ± 12.8</td>
</tr>
</tbody>
</table>

HR = heart rate; D-AA = diameter of the aortic annulus; IVSm = thickness of the middle part of the interventricular septum at the end-diastole; LVEDD = end-diastolic diameter of the left ventricle; LVPW = thickness of the left ventricular posterior wall at the end-diastole; EF = left ventricular ejection fraction.

*P<0.05 compared to the corresponding value in the normal group; **P<0.01 compared to the corresponding value in the normal group.

As Fig. 3 shows, in the proximal part of the left ventricular outflow tract, the velocity distributions had a similar pattern of skewness in both groups, but the extent of skewness in patients is slightly higher than that in normal subjects (P<0.05). After the level of 2 cm proximal to the aortic annulus (which was the same as the distance from the peak point of the maximal protruding basal septum to the aortic annulus: 2.02 ± 0.27 cm), the pattern of skewness in the patient group (in terms of ΔV) started to change dramatically and produced a completely different result from those in the normal group (P<0.01), but the extent of skewness (in terms of max/mean V) was close between two groups (P>0.05).

**Results**

The tissue images and colour flow maps were of good quality in all normal subjects and patients. General information about both groups is provided in Table 1. The anatomical parameters of the interventricular septum in the patient group are presented in Table 2.

Dynamic changes in peak flow velocity profile from the left ventricular outflow tract to the aortic annulus (Figs 2 and 3)

**Normal group**

Peak flow velocity profiles from the proximal left ventricular outflow tract to the aortic annulus were skewed with the highest velocities always located along the anterior wall at different levels.

**Patient group**

Peak flow velocity profiles in the left ventricular outflow tract were also skewed, but the relative location of the maximal velocity on the cross-sectional diameter of the flow channel changed from one level to another. At the point where the septum protruded maximally into the outflow tract, the velocity profile was most skewed with the highest velocity along the anterior wall (e.g. basal septum). Distal to this point, the highest velocities of the skewed velocity profiles were located gradually closer to the central part of the flow channel as the flow approached the aortic annulus. At the level of the aortic annulus, the highest velocity was typically located in the central area, or slightly posterior to the centre.

As Fig. 3 shows, in the proximal part of the left ventricular outflow tract, the velocity distributions had a similar pattern of skewness in both groups, but the extent of skewness in patients is slightly higher than that in normal subjects (P<0.05). After the level of 2 cm proximal to the aortic annulus (which was the same as the distance from the peak point of the maximal protruding basal septum to the aortic annulus: 2.02 ± 0.27 cm), the pattern of skewness in the patient group (in terms of ΔV) started to change dramatically and produced a completely different result from those in the normal group (P<0.01), but the extent of skewness (in terms of max/mean V) was close between two groups (P>0.05).

**Statistical analysis**

Average data are expressed as mean ± SD (standard deviation). The unpaired t-test was used to examine differences in all parameters in the normal and patient groups. In the patient group, correlations between skewness parameters of the velocity distribution and the left ventricular ejection fraction, parameters h and d, were evaluated by using simple linear regression. The pulsed Doppler recordings from three different sites were compared using the analysis of variance (ANOVA), and Scheffe's test was used whenever justified by ANOVA. Statistical significance was chosen as P<0.05.
Table 2 Anatomical parameters of the interventricular septum in patients with localized basal septal hypertrophy

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>End-diastolic thickness of the septum (mm)</th>
<th>Value of h (mm)</th>
<th>Ratio of h to D-AA</th>
<th>Value of d (mm)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>IVSm</td>
<td>IVSB</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>13.3</td>
<td>20.7</td>
<td>8.8</td>
<td>0.48</td>
</tr>
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<td>2</td>
<td>12.8</td>
<td>21.4</td>
<td>7.8</td>
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</tr>
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<td>3</td>
<td>14.3</td>
<td>22.0</td>
<td>11.2</td>
<td>0.42</td>
</tr>
<tr>
<td>4</td>
<td>15.0</td>
<td>21.2</td>
<td>8.9</td>
<td>0.39</td>
</tr>
<tr>
<td>5</td>
<td>11.7</td>
<td>19.3</td>
<td>8.6</td>
<td>0.39</td>
</tr>
<tr>
<td>6</td>
<td>12.3</td>
<td>18.5</td>
<td>9.1</td>
<td>0.35</td>
</tr>
<tr>
<td>7</td>
<td>14.1</td>
<td>19.0</td>
<td>7.3</td>
<td>0.35</td>
</tr>
<tr>
<td>8</td>
<td>12.7</td>
<td>19.4</td>
<td>10.0</td>
<td>0.38</td>
</tr>
<tr>
<td>9</td>
<td>12.0</td>
<td>22.2</td>
<td>10.7</td>
<td>0.39</td>
</tr>
<tr>
<td>Mean</td>
<td>13.1</td>
<td>20.4</td>
<td>9.2</td>
<td>0.40</td>
</tr>
<tr>
<td>SD</td>
<td>1.1</td>
<td>1.4</td>
<td>1.3</td>
<td>0.04</td>
</tr>
</tbody>
</table>

IVSb = thickness of the basal part of the interventricular septum; h = height of the protruding hypertrophic basal septum; d = the distance from the peak point of the protruding hypertrophic basal septum to the level of the aortic annulus (see Fig. 1 for details). SD = standard deviation. (For other abbreviations, see Table 1.)

Figure 3 Dynamic changes in the skewness parameters of the peak flow velocity profiles from the left ventricular outflow tract to the aortic annulus. In panel A, max/mean V represents the ratio of maximal velocity to the cross-sectional mean velocity according to the peak flow velocity profile. In panel B, ΔV represents the difference between the average velocities on the anterior and posterior halves of the diameter of the flow channel. AA = the aortic annulus; the numbers on the abscissa represent the distances (cm) from each level of analysis to the aortic annulus. ● = normal group; ■ = patient group. *P < 0.05 compared to the corresponding value in normal group; **P < 0.01 compared to the corresponding value in normal group.

Pulsed Doppler recording in the distal left ventricular outflow tract

In the patient group, the pulsed Doppler recording from different parts of the flow channel (Fig. 5) confirmed a skewed velocity distribution as measured by Doppler colour flow mapping (Figs 2 and 4). The highest velocity was located in the central area, and the lowest velocity was found in the anterior area of the flow channel.

The time-velocity integrals in the anterior, middle and posterior areas of the flow channel were 20.7 ± 3.4 cm³, 28.4 ± 5.8 cm³ and 23.5 ± 5.4 cm³, respectively. Significant differences were found between
Intracardiac velocity distributions

Anterior Velocity (m.s⁻¹)

A

Posterior

B

Anterior

Posterior

Time

1.5

Figure 4 Typical velocity distribution at 0.5 cm proximal to the aortic annulus in normal subjects (A) and in patients with localized basal septal hypertrophy (B). A: skewed velocity distribution with the highest velocities along the anterior wall from a normal subject; B: skewed velocity distribution with the highest velocities at the centre of the flow channel (from patient no. 5).

Table 3 Comparison of the time-velocity integral profiles in the distal left ventricular outflow tract between normal subjects and patients with localized basal septal hypertrophy

<table>
<thead>
<tr>
<th>Levels</th>
<th>Normal group (n=10)</th>
<th>Patient group (n=9)</th>
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<tbody>
<tr>
<td></td>
<td>Mean TVI (cm)</td>
<td>Max/mean TVI</td>
</tr>
<tr>
<td>AA/1.0</td>
<td>16.0 ± 2.5</td>
<td>1.39 ± 0.23</td>
</tr>
<tr>
<td>AA/0.5</td>
<td>16.9 ± 3.2</td>
<td>1.38 ± 0.30</td>
</tr>
<tr>
<td>AA</td>
<td>17.8 ± 3.7</td>
<td>1.36 ± 0.28</td>
</tr>
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</table>

TVI = time-velocity integral; Mean TVI = the cross-sectional mean TVI according to the TVI profile; Max/mean TVI = the ratio of the maximal TVI to the cross-sectional mean TVI; ΔTVI = the difference between the average TVI on the anterior half and the posterior half of the diameter of the flow channel. AA = aortic annulus; AA/0.5 = 0.5 cm proximal to the aortic annulus; AA/1.0 = 1.0 cm proximal to the aortic annulus.

*p < 0.05 compared to the corresponding value in the normal group; **p < 0.01 compared to the corresponding value in the normal group.

neighbouring areas (P < 0.01). The time–velocity integral obtained from the middle area was higher than the average time–velocity integral of the three sampling sites, by 17.0% ± 5.3% (11.6% to 25.6%), and higher, by 36.8% ± 16.8% (17.9% to 56.5%) than the time–velocity integral from the anterior area.

Discussion

In normal subjects, velocity distributions in the left ventricular outflow tract and in the aortic annulus were skewed with the highest velocities typically located along the anterior parts of the flow channel. In patients with localized basal septal hypertrophy, the velocity distributions at those levels were skewed differently from those in normal subjects, most probably due to the abnormal anatomical shape of the interventricular septum. The relative location of the highest velocity in the flow channel changed from one level to another. In the distal left ventricular outflow tract, the highest velocity was typically located in the central area.

Anatomically, the left ventricular outflow tract is a converging flow channel. According to the theory of fluid dynamics, a flow-converging channel combined with flow acceleration should flatten velocity distribu-
tion in the aortic annulus\textsuperscript{17,18}. However, velocity distribution in the aortic annulus was not flat as previously assumed, but significantly skewed\textsuperscript{17,18}.

Our previous studies\textsuperscript{3,8,9} suggested that, in addition to the anatomical structure of the left ventricular outflow tract, the intraventricular flow pattern was another important determinant of velocity distribution. An experimental study showed that the pattern of flow convergence in the left ventricular outflow tract changed throughout systole, and affected the velocity distributions in the distal left ventricular outflow tract and in the aortic annulus\textsuperscript{9}. When the pattern of flow convergence in the left ventricular outflow tract was asymmetrical in relation to the central axis of the aortic annulus during early systole (with the main volume flow along the anterior wall and the septum), the velocity distribution in the aortic annulus was skewed, with the highest velocity along the anterior wall and septum. When the pattern of flow convergence became axis-symmetrical during peak and late systole, the velocity distribution in the aortic annulus became flat even though a considerable septal angle existed throughout the systolic period\textsuperscript{9}.

In adult humans, there was a typical asymmetrical pattern of flow convergence in the left ventricular outflow tract\textsuperscript{3,8,9}. Forward flow in the left ventricular outflow tract started near the apex, then moved along the anterior wall and septum to the aortic annulus. On the posterior side of the left ventricular outflow tract, converging flow was not so obvious, or was just a very minor part of the forward flow stream. With the central axis of the aortic annulus as a reference, the flow did not follow the anterior wall and the posterior wall of the left ventricular outflow tract equally or symmetrically, but mainly followed the anterior wall and septum. In this way, symmetrical flow convergence could not occur. This feature is often observed in conventional Doppler colour flow mapping.

Because of the asymmetrical pattern of flow convergence in the left ventricular outflow tract, with the outflow mainly moving along the anterior wall and septum, the anatomical shape of the septum can significantly affect velocity distribution. In patients with localized basal septal hypertrophy, the protruding basal septum caused more significant convergence of blood flow along the septum and produced a high-velocity flow stream. Therefore, at the peak of the protruding septum, the highest velocity of the cross-sectional velocity profile was located along the septum. Distal to this point, the high-velocity flow stream was prevented from rounding the curvature to move along the septum, but took the direct route to the aortic ostium (Fig. 2B). Therefore, in the distal left ventricular outflow tract and the aortic annulus, skewed velocity distribution, with the highest velocity in the central part of the flow channel, was produced.

No significant correlation was found between the skewness parameter and the parameters h or d, or left ventricular ejection fraction. This may be because (1) the pattern of skewness is not only affected by the value of h or d, but also by other factors; (2) all these parameters varied within a small range in this patient group. For example, almost all patients had a normal left ventricular ejection fraction.

The present data show that the anatomical structure of the left ventricular outflow tract is an important determinant of velocity distribution. This and previous studies suggest that velocity distributions in the left ventricular outflow tract and in the aortic annulus have
complicated and diverse patterns, rather than a simply flat velocity distribution, as assumed previously.

**Clinical relevance**

In this patient category, the accuracy of the flow volume measurement can also be significantly affected by the pulsed Doppler sampling site in the flow channel. Sampling from the central part of the flow channel will overestimate the cross-sectional mean velocity. Averaging velocities from several pulsed Doppler sampling sites across the flow channel is the only accurate way of obtaining a representative cross-sectional mean velocity.

The findings in patients with localized basal septal hypertrophy may be extended to patients with upper septal angulation without hypertrophy such as 'sigmoid septum' \(^{19}\). Certainly, a similar study needs to be conducted in that patient category.

**Limitations of the study**

One limitation of this study was that only one cross-sectional view (apical long axis view) was used, and the velocity distribution was not studied in other planes. In our experience, the apical long axis view is the best plane from which to obtain velocity information in the left ventricular outflow tract and in the aortic annulus \(^{1,2,3}\). Compared to the four chamber view, it was easier to obtain a longitudinal view of the flow channel in the apical long axis view. The flow from the left ventricular outflow tract, via the aortic annulus, to the proximal part of the ascending aorta could be shown clearly, so the direction of flow could be easily judged, and the velocity angle-corrected. In the four chamber view, an oblique section of the flow channel around the aortic annulus was often obtained, and therefore it was often difficult to obtain an overview of the flow channel. Our previous studies also suggested that the most skewed velocity distribution existed in the apical long axis view \(^{3,5}\). Moreover, the main purpose of this study was to confirm whether the anatomical structure of the interventricular septum could affect the velocity distribution, and the presented data are sufficient to support our conclusion.

In the patient group, both Doppler colour flow mapping and pulsed Doppler showed the same skewness pattern in velocity distribution. However, the cross-sectional mean time–velocity integral obtained from Doppler colour flow mapping was lower than the average time–velocity integral measured from pulsed Doppler recordings of the three sampling sites (21.0 ± 3.0 cm vs 24.2 ± 4.6 cm; \(P<0.01\)). This was anticipated because: (1) the velocity obtained from Doppler colour flow mapping was the mean value from each sample volume \(^{[12]}\), whereas the velocity derived from pulsed Doppler recording was measured from the maximal velocity tracing of the flow spectrum; (2) in the construction of velocity distribution by using Doppler colour flow mapping, the loss of some velocity information at the beginning and the end of systole could result in a slightly lower calculated time–velocity integral than the real time–velocity integral \(^{[2,3]}\). However, this error did not exist in the pulsed Doppler recording.

**Conclusions**

In patients with localized basal septal hypertrophy, the velocity distributions in the left ventricular outflow tract and in the aortic annulus were skewed in a different way from those of normal subjects. The relative location of the maximal velocity in the flow channel changed from one level to another. In the distal part of the left ventricular outflow tract, the highest velocity was typically located in the central part of the flow channel. In conclusion, the anatomical shape of the interventricular septum significantly affects the velocity distributions in the left ventricular outflow tract and in the aortic annulus.

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