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Left atrioventricular plane displacement but not left ventricular ejection fraction is influenced by the degree of aortic stenosis

E Rydberg, P Gudmundsson, L Kennedy, L Erhardt, R Willenheimer

Methods and results: Cardiac dimensions, LV filling, left AVPD, LV ejection fraction (LVEF), and valve function were assessed by echocardiography/Doppler in 182 patients with various cardiac diseases (mean (SD) age 69 (12) years, 36% women), 49 consecutive with and 133 consecutive without aortic stenosis. In an analysis of covariance, neither left AVPD nor LVEF was independently correlated with the presence of aortic stenosis. However, looking separately at patients with aortic stenosis, left AVPD (p = 0.03) but not LVEF correlated independently with degree of aortic stenosis in multiple linear regression analysis. In patients with aortic stenosis, an abnormal left AVPD had 94% sensitivity and 90% negative predictive value with regard to severe aortic stenosis, compared with 56% and 62%, respectively, for LVEF.

Conclusion: In patients with cardiac disease, neither left AVPD nor LVEF correlated independently with presence of aortic stenosis. However, in patients with aortic stenosis, left AVPD but not LVEF correlated with the degree of aortic valve obstruction and left AVPD but not LVEF had high sensitivity and negative predictive value with regard to severe aortic stenosis. Compared with LVEF, left AVPD is an earlier and more sensitive marker of LV haemodynamic load in patients with aortic stenosis.

METHODS

Patients

We enrolled 49 consecutive patients with and 133 consecutive patients without aortic stenosis during 14 and four months, respectively, referred to our echocardiography laboratory for a diagnostic examination at the department of cardiology in Malmö University Hospital, with a primary catchment area of 250 000 inhabitants. The reasons for referral were mainly assessment of LV and valve function in patients with coronary artery disease, heart failure, and valve disease, but no referral reason excluded the patient. Since atrial fibrillation has been shown to decrease left AVPD and since LV diastolic performance cannot be adequately assessed by echocardiography/Doppler, we excluded patients with atrial fibrillation.

Abbreviations: AVPD, atrioventricular plane displacement; E/A, ratio of early to atrial peak velocity of diastolic transmitral flow; LV, left ventricular; LVEF, left ventricular ejection fraction
Doppler in patients with atrial fibrillation, only patients in sinus rhythm were included. The definition of coronary artery disease was prior or current myocardial infarction, angina verified with exercise test, stress echocardiography, radionuclide examination, or coronary angiography, and prior percutaneous transluminal coronary angioplasty or coronary bypass grafting.

**Echocardiographic examination**

Two dimensional echocardiography and Doppler examinations were performed by either of two investigators with a Hewlett Packard (Andover, Massachusetts, USA) Sonos 2000 or 2500 echocardiography system and a 2.5 MHz transducer. Pulsed, continuous, and colour flow Doppler examinations were performed with the same transducers. Parasternal and apical views were obtained with the patient in a left lateral decubitus position. Measurements were acquired during silent respiration or end expiratory apnoea.

Left AVPD was determined by two dimensionally guided M mode in the four and two chamber views, as described previously. The regional AVPD (mm) was the distance covered by the atrioventricular plane between the position most remote from the apex (corresponding to the onset of contraction) and the location closest to the apex (corresponding to the end of contraction, including any postejiction shortening)—that is, the full extent of the displacement. It was measured in the septal, lateral, posterior, and anterior regions and was calculated from an average of two to four heartbeats. The mean of the AVPD in the four regions was calculated. The mean interobserver variability in our laboratory was immediately after one another was 4.8% (AVPD difference range 0–1.2 mm) in a series of 53 consecutive patients with a mean left AVPD of 7.8 mm (range 3.3–15.5 mm). The intraobserver variability of the determination of left AVPD was mean 2.0% (range 0–6%), corresponding to 0.23 mm (range 0–0.95 mm), in 39 randomly examined patients with a mean left AVPD of 11.2 mm (range 5.6–17.5 mm).

LVEF was determined by visual quantification and was evaluated independently of left AVPD, based on regional and continuous Doppler pressure half time (aortic regurgitation only) in the parasternal and apical views. The grades were none (0), mild (1), mild to moderate (2), moderate (3), moderate to severe (4), and severe (5). With this method, interobserver and intraobserver variability was a maximum of one grade in our laboratory.

**Statistical analysis**

The t test was used to test differences between two groups. For assessment of correlations between variables, univariate and multivariate linear regression analyses, covariance analysis, and the Spearman rank correlation test were used. Data are expressed as mean (SD). Two tailed p values < 0.05 were considered significant. Variables that in univariate regression analysis were significantly correlated (p < 0.05) with left AVPD and LVEF were considered for inclusion in multivariate linear regression analysis. However, interventricular septum diameter was excluded, since it is highly correlated with LV posterior wall diameter and since LV posterior wall diameter is a better marker of LV mass than interventricular septum diameter. We wanted to explore the relation between LV diameter and left AVPD and, since LV mass is a function of LV wall thickness and LV diameter, LV mass was also excluded. E:A was excluded, since it was part of the “LV diastolic function” variable.

**Ethics**

The study is approved by the ethics committee of Lund University.

**RESULTS**

The mean (SD) age of all patients was 69 (12) years; 65 (36%) were women. Table 1 shows the most important variables analysed in the present study for patients with and patients without aortic stenosis. Twenty four (49%) of the patients with aortic stenosis and 110 (83%) of the patients without aortic stenosis had coronary artery disease (p < 0.0001). The presence of coronary artery disease did not correlate with left AVPD or LVEF in any patient group. Among patients with aortic stenosis, 26 (53%) had angina pectoris, 25 (51%) had congestive heart failure, 8 (16%) had a history of syncope, and 8 (16%) had a history of dizziness. Left AVPD, but not LVEF, was significantly lower in patients with aortic stenosis than in those without.
Left AVPD

Tables 2, 3, and 4 show variables that correlated significantly with left AVPD in univariate analysis in the entire patient group, in patients with aortic stenosis, and in patients without aortic stenosis. In the entire patient group, AVPD correlated significantly with the presence of aortic stenosis. In patients with aortic stenosis, the velocity–time integral ratio correlated significantly with AVPD. This correlation was closer in patients without coronary artery disease ($r = 0.58$, $p = 0.004$) than in patients with coronary artery disease ($r = 0.39$, $p = 0.07$). Variables that correlated independently with left AVPD in multivariate analysis in the entire patient group were age ($p < 0.0001$), LVPWD ($p < 0.0001$), LV size ($p < 0.0001$), and LV diastolic filling ($p = 0.0001$). Among patients with aortic stenosis, the variables that qualified for the multivariate linear regression analysis explained 52% of the left AVPD variability. Age ($p = 0.041$), velocity–time integral ratio ($p = 0.026$), and LV diastolic filling ($p = 0.023$) correlated independently with left AVPD. The variables that qualified for the multivariate linear regression analysis in patients without aortic stenosis explained 48% of the left AVPD variability. Age ($p = 0.002$), LV size ($p = 0.0001$), LVPWD ($p = 0.029$), and LV diastolic filling ($p = 0.002$) correlated independently with left AVPD. Among patients with aortic stenosis, an abnormal left AVPD—defined as $<11.0$ mm, which corresponds to an LVEF $<55\%$—had a negative predictive value of 90% and a sensitivity of 94% with regard to severe aortic stenosis (table 5). Of the 17 patients with depressed left AVPD despite non-severe aortic stenosis, 11 had coronary artery disease.

**LVEF**

In univariate analysis in the entire patient group, age ($p < 0.0001$), LV size ($p < 0.0001$), left atrial diameter ($p < 0.0001$), mitral regurgitation ($p < 0.0001$), LV diastolic filling ($p < 0.0001$), but not presence of aortic stenosis ($p = 0.54$) correlated with LVEF. In multivariate analysis, LV size ($p < 0.0001$) and LV diastolic filling ($p = 0.0004$) remained independent correlates. Table 6 shows significant correlates of LVEF in univariate analysis among patients with aortic stenosis. The velocity–time integral ratio did not correlate with LVEF, neither in patients without coronary artery disease ($r = 0.40$, $p = 0.06$) nor in patients with coronary artery disease ($r = 0.23$, $p = 0.30$). In multivariate regression analysis, explaining 47% of the LVEF variability in patients with aortic stenosis, only LV size ($p = 0.012$) correlated independently with LVEF. Among patients with aortic stenosis, an abnormal LVEF ($<55\%$) had a negative predictive value of 62% and a sensitivity of 56% with regard to severe aortic stenosis (table 5). Of the 13 patients with depressed LVEF despite non-severe aortic stenosis, 10 had coronary artery disease.

**DISCUSSION**

Aortic stenosis imposes increased afterload and increased wall stress on the LV. The increased afterload is offset by the development of concentric LV hypertrophy by the process of sarcomere replication. Compensated aortic stenosis is

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**Table 1** Age, body surface area, and echocardiographic and Doppler variables in patients with and without aortic stenosis

<table>
<thead>
<tr>
<th>Variable</th>
<th>Aortic stenosis (n = 49)</th>
<th>No aortic stenosis (n = 133)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean (SD)</strong></td>
<td><strong>Mean (SD)</strong></td>
<td><strong>p Value</strong></td>
</tr>
<tr>
<td>Age (years)</td>
<td>77 (8)</td>
<td>67 (12)</td>
</tr>
<tr>
<td>Body surface (m$^2$)</td>
<td>1.77 (0.18)</td>
<td>1.92 (0.20)</td>
</tr>
<tr>
<td>IVSDd/m$^2$ (mm)</td>
<td>7.4 (1.9)</td>
<td>5.6 (1.0)</td>
</tr>
<tr>
<td>LVPWd/m$^2$ (mm)</td>
<td>7.2 (1.5)</td>
<td>5.9 (0.8)</td>
</tr>
<tr>
<td>LVd/m$^2$ (mm)</td>
<td>27.3 (3.4)</td>
<td>26.6 (4.2)</td>
</tr>
<tr>
<td>LVM/m$^2$ (g)</td>
<td>137 (43)</td>
<td>112 (32)</td>
</tr>
<tr>
<td>LAs/m$^2$ (mm)</td>
<td>24.7 (3.4)</td>
<td>22.1 (2.9)</td>
</tr>
<tr>
<td>AVPD (mm)</td>
<td>9.2 (2.3)</td>
<td>10.7 (2.8)</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>50 (12)</td>
<td>52 (14)</td>
</tr>
<tr>
<td>E:A</td>
<td>1.08 (0.89)</td>
<td>1.08 (0.60)</td>
</tr>
<tr>
<td>Diastolic performance (grade 1–3)</td>
<td>2.1 (0.57)</td>
<td>1.9 (0.67)</td>
</tr>
<tr>
<td>MR</td>
<td>1.9 (1.0)</td>
<td>1.5 (0.9)</td>
</tr>
<tr>
<td>VTI ratio</td>
<td>0.25 (0.07)</td>
<td>0.12 (0.39)</td>
</tr>
<tr>
<td>Aortic V-max (m/s)</td>
<td>3.8 (1.0)</td>
<td>2.1 (1.6)</td>
</tr>
</tbody>
</table>

*aortic V-max, maximum aortic flow velocity; AVPD, atrioventricular plane displacement; E:A, ratio of early to atrial peak velocity of diastolic transmitral flow; IVSDd, interventricular septum diameter at end diastole; LAs, left atrial diameter at end systole; LVEF, left ventricular ejection fraction; LVd, left ventricular internal diameter at end diastole; LVM, left ventricular mass; LVPWD, left ventricular posterior wall diameter at end diastole; MR, mitral regurgitation; NA, not applicable; VTI ratio, velocity–time integral ratio of left ventricular outflow tract flow to aortic flow.

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**Table 2** Variables that significantly correlated with AVPD in univariate linear regression analyses in the entire patient group

<table>
<thead>
<tr>
<th>Variable</th>
<th>$r$</th>
<th>$p$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>-0.52</td>
<td>$&lt;0.0001$</td>
</tr>
<tr>
<td>Body surface (m$^2$)</td>
<td>0.32</td>
<td>$&lt;0.0001$</td>
</tr>
<tr>
<td>IVSDd/m$^2$ (mm)</td>
<td>-0.46</td>
<td>$&lt;0.0001$</td>
</tr>
<tr>
<td>LVd/m$^2$ (mm)</td>
<td>-0.41</td>
<td>$&lt;0.0001$</td>
</tr>
<tr>
<td>LVM/m$^2$</td>
<td>-0.30</td>
<td>$&lt;0.0001$</td>
</tr>
<tr>
<td>LVPWd/m$^2$ (mm)</td>
<td>-0.45</td>
<td>$&lt;0.0001$</td>
</tr>
<tr>
<td>Presence/absence of aortic stenosis</td>
<td>-0.24</td>
<td>0.0012</td>
</tr>
<tr>
<td>Diastolic performance (grade 1–3)</td>
<td>-0.36*</td>
<td>$&lt;0.0001^*$</td>
</tr>
<tr>
<td>MR</td>
<td>-0.38</td>
<td>$&lt;0.0001^*$</td>
</tr>
</tbody>
</table>

*Spearman rank correlation test.

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**Table 3** Variables that correlated significantly with AVPD in univariate linear regression analyses in patients with aortic stenosis

<table>
<thead>
<tr>
<th>Variable</th>
<th>$r$</th>
<th>$p$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>-0.45</td>
<td>0.001</td>
</tr>
<tr>
<td>Body surface (m$^2$)</td>
<td>0.31</td>
<td>0.030</td>
</tr>
<tr>
<td>IVSDd/m$^2$ (mm)</td>
<td>-0.39</td>
<td>0.006</td>
</tr>
<tr>
<td>LVPWd/m$^2$ (mm)</td>
<td>-0.32</td>
<td>0.024</td>
</tr>
<tr>
<td>LVd/m$^2$ (mm)</td>
<td>-0.32</td>
<td>0.028</td>
</tr>
<tr>
<td>Diastolic performance (grade 1–3)</td>
<td>-0.37*</td>
<td>0.0009*</td>
</tr>
<tr>
<td>VTI ratio</td>
<td>0.43</td>
<td>0.003</td>
</tr>
<tr>
<td>VTI ratio (in patients with VTI ratio $\geq 0.23$)</td>
<td>0.20</td>
<td>0.055</td>
</tr>
<tr>
<td>VTI ratio (in patients with VTI ratio $&lt; 0.23$)</td>
<td>0.12</td>
<td>0.54</td>
</tr>
<tr>
<td>MR</td>
<td>-0.28</td>
<td>0.049</td>
</tr>
</tbody>
</table>

*Spearman rank correlation test.
characterised by concentric hypertrophy and a normal or supranormal LVEF. With longstanding hypertrophy LV contractility may be depressed somewhat, but LVEF is relatively preserved by the Frank-Starling mechanism. LV contractility may be depressed somewhat, but LVEF is supranormal. With longstanding hypertrophy LV is characterised by concentric hypertrophy and a normal or supranormal LVEF.

Prior studies have shown that left AVPD is decreased in patients with LV hypertrophy, which was also the case in the present study. However, our results indicate that the decrease of left AVPD in relation to the degree of valve obstruction is independent of LV hypertrophy, suggesting a direct relation with the increased intraventricular pressure resulting from aortic stenosis.

Conclusion

This study shows that left AVPD, as opposed to LVEF, correlated significantly with the presence of aortic stenosis in univariate analysis, although not independently in multivariate analysis. In patients with aortic stenosis, left AVPD correlated independently with the degree of aortic valve obstruction, whereas LVEF did not. This was especially evident among patients without coronary artery disease. An abnormal left AVPD and degree of aortic stenosis: Patients with severe aortic stenosis are very likely to have an abnormal left AVPD, whereas a normal left AVPD precludes severe aortic stenosis with high probability. Our results indicate that, compared with LVEF, left AVPD is an earlier and more sensitive marker of LV haemodynamic load among patients with aortic stenosis.

Among patients with aortic stenosis, both left AVPD and LVEF had poor specificity and positive predictive value with regard to severe aortic stenosis. This was mainly because some patients with coronary artery disease had depressed left AVPD and LVEF without having severe aortic stenosis.

A possible explanation for the result of the present study may be that left AVPD is more sensitive to ischaemia caused by increased wall stress because left AVPD reflects subendocardial fibre function. The result may also be explained by the relation between LV diastolic performance and left AVPD, as opposed to LVEF, since diastolic dysfunction occurs before systolic dysfunction in aortic stenosis.

Prior studies have shown that left AVPD is decreased in patients with LV hypertrophy, which was also the case in the present study. However, our results indicate that the decrease of left AVPD in relation to the degree of valve obstruction is independent of LV hypertrophy, suggesting a direct relation with the increased intraventricular pressure resulting from aortic stenosis.

ACKNOWLEDGEMENTS

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Atrioventricular plane displacement in valve dysfunction


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