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Left Ventricular Diastolic Filling is Related to the Atrioventricular Plane Displacement in Patients with Coronary Artery Disease

Erik Rydberg, Ronnie Willenheimer, Björn Brand and Leif R. Erhardt

Objective—Left atrioventricular plane displacement (AVPD) is often decreased and abnormalities in left ventricular diastolic filling are common in patients with coronary artery disease (CAD). This study was designed to assess the relationship between AVPD and diastolic filling in patients with CAD.
Design—AVPD was assessed by echocardiography and diastolic filling by transmitral and pulmonary venous pulsed Doppler in 170 consecutive patients (66 ± 11 years) with proven CAD at coronary angiography. Diastolic filling was grouped as normal, mildly impaired and moderately to severely impaired.
Results—A simple linear regression analysis showed that AVPD decreased in relation to increased severity of diastolic filling impairment ($r = -0.36$, $p < 0.0001$). In a multiple regression analysis, ejection fraction, diastolic filling, age and body surface were independently correlated with AVPD. Each millimetre of decrease in AVPD increased the probability of impaired diastolic filling by 28%.
Conclusion—AVPD was independently correlated with both left ventricular systolic function and diastolic filling in patients with CAD. Thus, given the same degree of ejection fraction, it was found that the greater the impairment in diastolic filling, the lower the AVPD.

Key Words: atrioventricular plane displacement, coronary artery disease, diastolic filling

Patients with coronary artery disease (CAD) often have decreased mean left atrioventricular plane displacement (AVPD) (1, 2), and AVPD impairment is greater with more widespread coronary atherosclerosis, irrespective of previous myocardial infarction (MI) (3). AVPD is a strong prognostic marker in patients with chronic heart failure (4, 5). AVPD has been suggested to be mainly dependent on the activity of the subendocardial, longitudinal myocardial fibres, whereas ejection fraction is considered to be related primarily to subepicardial fibre performance (6). Because subendocardial fibres are more susceptible to develop ischaemia than subepicardial fibres, AVPD may be decreased already in mild CAD when ejection fraction is preserved (3).

Irrespective of systolic function, abnormalities in left ventricular (LV) diastolic performance are common in patients with CAD, as assessed by Doppler and also seen as left atrial enlargement (3, 7–9). LV relaxation, compliance, and filling pressures have been shown to be related to the clinical status and prognosis of patients with various heart diseases (8, 10–18). Combined transmitral and pulmonary venous Doppler echocardiography provides a clinically useful measure of LV diastolic filling, evaluating the ratio of the early transmitral flow peak velocity to the peak velocity of the late transmitral flow caused by the atrial contraction (E/A), and the ratio of the systolic and diastolic filling of the left atrium from the pulmonary veins (S/D) (10). Using this method, we recently showed that diastolic filling is significantly more impaired with more widespread CAD (unpublished data from our group).

We have reported that AVPD is independently related to Doppler variables of LV diastolic filling in heart failure patients (19). Thus, AVPD reflects systolic function as well as diastolic filling in patients with heart failure. The purpose of the present study was to examine whether AVPD is also independently related to LV diastolic filling, assessed by both Doppler methods, in patients with CAD.

MATERIAL AND METHODS

Patients

Patients who were referred to our department for elective clinical coronary angiography for a period of 6 months and who were examined by echocardiography were eligible. All patients had to have sinus rhythm. Exclusion criteria were coronary bypass grafting and percutaneous transluminal coronary angioplasty within 12 months prior to this study ($n = 37$). Patients who had no significant stenoses were excluded from all

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Table I. *Mean AVPD in patients with normal, mildly impaired and moderately/severely impaired diastolic filling, divided by one-, two-and three-vessel disease at coronary angiography*

	Normal diastolic filling ($n = 91$)	Mild diastolic filling impairment ($n = 55$)	Severe diastolic filling impairment ($n = 14$)	p-value
One-vessel disease	12.4 ± 2.0	12.0 ± 1.3	6.8 ± 1.1	0.02
Two-vessel disease	12.1 ± 2.3	11.0 ± 1.5	9.7 ± 1.9	0.03
Three-vessel disease	11.7 ± 2.5	11.0 ± 1.6	8.4 ± 2.7	0.01
All	12.0 ± 2.3	11.1 ± 1.6	8.5 ± 2.3	<0.0001

AVPD = atrioventricular plane displacement.

analyses ($n = 124$). We consecutively included the remaining 170 patients (age 66 ± 11 years, 121 males) who had significant stenoses at coronary angiography.

Echocardiographic examination

Two-dimensional echocardiography and Doppler examinations were performed blind using a Hewlett-Packard (Andover, Mass, 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 transducer. Parasternal and apical views were obtained with the patient in a left lateral recumbent position. Measurements were acquired during silent respiration or end-expiratory apnoea.

Left AVPD was determined in two-dimensionally-guided M-mode in the four- and two-chamber views, as described previously (4, 5). The regional displacement (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 post-ejection shortening), i.e. the full extent of the displacement. The displacement was measured in the septal, lateral, posterior and anterior regions, and was calculated from an average of four cardiac cycles. The mean of the displacement in the four regions was calculated. The mean AVPD in 15 controls (mean age 65 years, range 54–77 years, 8 (53%) females) was 13.5 ± 1.1 mm, and a mean AVPD of 10.0 mm was considered normal (20). In our laboratory the mean interindividual variability between two investigators (examining each patient immediately after one another) was 4.8% (AVPD difference range 0–1.2 mm) in a series of 53 consecutive patients with a mean AVPD of 7.8 mm (range 3.3–15.5 mm) (4). The mean intraindividual variability of the determination of AVPD was 2.0% (range 0–6%) corresponding to 0.23 mm (range 0–0.95 mm) (20).

LV diastolic filling was evaluated by transmitral and pulmonary venous pulsed Doppler echocardiography. The transmitral Doppler examination was performed as described previously (21) by determination of the E/A-ratio (22). Pulmonary venous flow was examined with the cursor placed perpendicular to the flow and the sample volume 0.5 to 1.0 cm into the right upper pulmonary vein.

The variables of diastolic filling included E/A and S/D. LV diastolic filling was considered as normal (grade 1) when E/A was > 0.75 and ≤1.0 or when E/A was >1.0 combined with S/D > 1.0. Diastolic filling was considered mildly impaired (grade 2) when E/A was ≤0.75, and moderately to severely impaired (grade 3) when E/A was >1.0 and S/D ≤ 1.0 (pseudonormalized or abnormally high E/A). The lower E/A limit (0.75) of the normal diastolic filling interval chosen was based on the study by Mantero et al. (23), showing that the E/A in healthy individuals of an age similar to the mean age of our patients was 1.0 ± 0.2. In healthy middle-aged individuals the maximum flow velocity of the systolic component (S) is higher than that of the diastolic inflow (D) (14, 24–26). Therefore S < D was considered as abnormal.

Left ventricular ejection fraction (LVEF) was determined by visual quantification, and the evaluation was done irrespective of AVPD, based on regional and global wall motion assessment. The LVEF determinations were always carried out by two investigators. When there was a discrepancy between the investigators regarding the LVEF, the examination was re-evaluated until agreement was reached. The variability between the investigators performing the LVEF evaluations in the present study was tested in a series of 26 patients with a mean LVEF of 0.48 (range 0.17–0.70). The mean difference between the LVEF determinations was 0.02 (range 0.00–0.07), corresponding to a 5.6% mean difference (range 0–19%).

Cardiac dimensions were measured in the parasternal long axis view in two-dimensional mode (27). Semi-quantitative grading of valvular regurgitation was based on colour Doppler signal area, continuous Doppler signal density and continuous Doppler pressure half-time (aortic regurgitation only) in the parasternal and apical views. The different degrees were: none (0), trace (0.5), mild (1), mild–moderate (1.5), moderate (2), moderate–severe (2.5), and severe (3).

Coronary angiography

Coronary angiography was performed in all patients within 30 days after echocardiography. The evaluation of the examinations was done by visual assessment by physicians blinded to the results of the echocardiographic examination. Occlusions and significant stenoses, defined as at least 50% reduction of the coronary artery cross-sectional lumen area, were registered. The most severe stenoses in each of the three vessel areas constituted the respective degree of stenosis. Left main coronary artery stenosis was regarded as two-vessel disease. The angular artery was considered as a branch of the circumflex artery.

Table II. *Mean AVPD in patients with normal, mildly impaired and moderately/severely impaired diastolic filling divided into those with a history of MI and those without*

	Normal diastolic filling ($n = 91$)	Mild diastolic filling impairment ($n = 55$)	Severe diastolic filling impairment ($n = 14$)	p-value
Prior MI ($n = 101$)	12.1 ± 2.5	10.6 ± 1.44	8.7 ± 2.5	<0.0001
No Prior MI ($n = 59$)	11.7 ± 2.1	11.6 ± 1.6	7.7 ± 1.8	0.02
P	0.50	0.01	0.51	

AVPD = atrioventricular plane displacement; MI = myocardial infarction.

Table III. *Left ventricular ejection fraction (LVEF) in patients with normal, mildly impaired and moderately/severely impaired diastolic filling divided according to one-, two-and three-vessel disease at coronary angiography*

	Degree of diastolic filling			
	Normal ($n = 94$)	Mildly impaired ($n = 54$)	Moderately/severely impaired ($n = 15$)	p-value
One-vessel disease	58 ± 12	60 ± 4	35 ± 9	0.056
Two-vessel disease	60 ± 10	59 ± 10	46 ± 18	0.121
Three-vessel disease	58 ± 10	54 ± 10	48 ± 19	0.109
All	59 ± 10	56 ± 10	45 ± 17	0.001

Electrocardiogram and assessment of myocardial infarction

A patient was judged to have had a myocardial infarction (MI) if this diagnosis had been validated at hospitalization and/or if the electrocardiogram showed signs of an MI. Changes in electrocardiogram results suggestive of MI were assessed by standard clinical criteria based on the Minnesota code.

Statistics

To test for differences between two groups regarding continuous variables, the unpaired *t*-test was applied, and for multiple group comparisons the Kruskal–Wallis test was performed. For assessment of correlation's between continuous variables simple and multiple linear regression analyses were used. Correlation between a "yes or no" dependent variable and nominal or continuous independent variables was examined by logistic regression analysis. Differences between groups with respect to nominal variables were tested a χ^2 test. Data are expressed as mean ± SD, and a p-value <0.05 was considered significant.

RESULTS

AVPD was significantly decreased when diastolic filling impairment was more severe (Table I). Furthermore, when patients with one-, two- and three-vessel disease were analysed separately, the same significant trend was evident (Table I). The relation between AVPD and diastolic filling was also evident when patients were divided into two groups, those with and those without prior MI (Table II). Among patients with normal diastolic filling, no significant difference in AVPD was found between patients with and those without a prior MI, whereas a significant difference was shown among those with mildly impaired diastolic dysfunction. Among the few patients with moderately/severely impaired diastolic filling, there was no significant difference in AVPD between patients with and those without a prior MI.

AVPD was significantly lower in patients with S/D ≤ 1 than in those with S/D $>$ 1:8.8 ± 2.4 vs 11.8 ± 2.1, $p < 0.0001$. In a linear regression analysis, AVPD was negatively correlated with E/A ($r = -0.18$, $p = 0.02$). However, when patients with pseudonormalized/abnormally high E/A ($n = 14$) were excluded from the analysis there was a positive correlation ($r = 0.34$, $p < 0.0001$). In a logistic regression analysis AVPD was significantly lower in patients with any degree of impaired diastolic filling than in those with normal diastolic filling. The probability of having any degree of impaired diastolic filling increased by 28% for each millimetre of AVPD decrease ($p < 0.0001$). A significant difference in LVEF was found between patients with normal, mildly impaired and moderately/severely impaired diastolic filling (Table III). When patients with one-, two- and three-vessel disease were analysed individually, LVEF did not differ significantly between the three diastolic filling groups (Table III).

Variables correlating with AVPD showing a p-value <0.15 in a univariate linear regression analysis were included in a multiple linear regression analysis. These were age, body surface, left atrial diameter, LV end diastolic diameter, interventricular septum diameter, LV posterior wall diameter, degree of mitral regurgitation, degree of aortic regurgitation, degree of tricuspid regurgitation, LVEF and diastolic filling group (Table IV). These 11 variables explained 68% of the variation in AVPD. LVEF ($p < 0.0001$), diastolic filling ($p = 0.016$), body surface ($p = 0.043$) and age ($p = 0.045$) correlated independently with AVPD. These four variables alone explained 65% of the variation in AVPD.

Table IV. *Simple linear regression analysis comparing different variables with mean left atrioventricular plane displacement (AVPD)*

	r	p-value
Age	−0.19	0.016
Body surface	0.15	0.061
LA	−0.33	<0.0001
LVEDD	−0.39	<0.0001
IVSD	−0.12	0.142
LVPWD	−0.18	0.023
Degree of mitral regurgitation	−0.36	<0.0001
Degree of aortic regurgitation	−0.17	0.027
Degree of tricuspid regurgitation	−0.21	0.008
LVEF	0.76	<0.0001
Diastolic filling group (1–3)	−0.36	<0.0001

LA = left atrial diameter at end systole; LVEDD = left ventricular internal diameter at end diastole; IVSD = interventricular septum diameter at end diastole; LVPWD = left ventricular posterior wall diameter at end diastole; LVEF = left ventricular ejection fraction.

DISCUSSION

Patients with CAD often have impaired myocardial relaxation because of reduced myocardial blood flow (28, 29). In these patients usually a low E/A as assessed by Doppler is seen (7, 31). Chronic ischaemia, myocardial hibernation and stunning and infarction cause myocardial remodelling with fibrosis, leading to decreased myocardial compliance, which further impairs the diastolic performance (28–30). The stiff LV is characterized by disturbed LV filling during late diastole, reduced active left atrial emptying, and increased pulmonary venous and LV filling pressures (24, 32, 33). The elevated filling pressure causes a filling pattern with a pseudonormal E/A. With further progress, rapid early filling leads to a restrictive filling pattern with an abnormally high E/A (17, 18). A pseudonormal/abnormally high E/A can be separated from a normal pattern by Doppler evaluation of the pulmonary venous inflow to the left atrium. Thus, impaired diastolic filling as assessed by Doppler can be found either as a low E/A (a pseudonormalized E/A with low S/D) or as a restrictive pattern with abnormally high E/A and low S/D.

We have previously shown that the prevalence of diastolic filling abnormalities is increased in relation to an increasing number of affected coronary arteries (unpublished data from our group), and that, furthermore, the more widespread the CAD, the greater the decrease in AVPD (3). Prior studies have shown that AVPD is related to LV diastolic filling as assessed by transmitral Doppler in patients with atrial fibrillation and in patients with heart failure (19, 34). However, up until now there has been no investigation into whether AVPD is related to LV diastolic filling in patients with CAD.

In the present study we found that the more pronounced the diastolic filling impairment in patients with CAD, the greater the decrease in AVPD. This relationship was found independently in patients with one-, two- and three-vessel disease, and also irrespective of prior MI. In a simple regression analysis a weak negative correlation was found between AVPD and E/A, which is in accordance with prior findings in patients with heart failure (19). When the patients with pseudonormalized and restrictive E/A were excluded, there was a stronger and more positive correlation between AVPD and E/A, suggesting that patients with a pseudonormalized/restrictive E/A had the most decreased AVPD, as might be expected. This was also shown in the three-group comparison between patients with different degrees of diastolic filling abnormalities (Table I). AVPD was also significantly lower in patients with decreased S/D. This is in line with the other findings, since S/D is decreased when E/A is pseudonormalized or abnormally high. Thus, the combined assessment of E/A and S/D in the evaluation of diastolic filling allowed for identification of patients with more pronounced diastolic filling impairment, making a relationship between diastolic filling and AVPD evident. Indeed, an increase in AVPD of one millimetre increased the probability of having any degree of impaired diastolic filling by 28%.

The variables included in the multiple regression analysis explained 68% of the variation in AVPD. Diastolic filling and LVEF were both independently correlated to AVPD and explained almost 59% of the variation in AVPD. In an earlier study from our group we showed that AVPD was related to both systolic and diastolic performance in patients with heart failure (19). The diastolic filling was estimated with E/A and the deceleration time of the early transmitral diastolic flow (E_{dt}), and E_{dt} and fractional shortening were the only variables independently correlated with AVPD. S/D was not measured in that study and therefore patients with pseudonormalized E/A might not always have been detected. Nevertheless, the results of the two studies are quite similar.

There are a number of pitfalls when using the Doppler method to evaluate LV diastolic function. The Doppler evaluation is affected by age, heart rate, filling pressures, peripheral resistance, gender, systolic blood pressure, left atrial diameter and LV wall thickness (35) . All these factors render the Doppler method somewhat precarious when evaluating diastolic performance. Therefore, our findings might not be related to LV diastolic function per se, but rather to LV diastolic filling as a result of all the factors influencing it. In the present study E_{dt} was not measured.

STUDY LIMITATIONS

The use of visual quantification for the assessment of left ventricular ejection fraction might seem imprecise and somewhat unreliable compared with an actual measurement of ejection fraction based on endocardial border tracing in two-dimensional mode. However, several studies have shown that the former approach provides an excellent assessment of ejection fraction, superior to endocardial border tracing for the measurement of ejection fraction, using radionuclide measurement, or ventriculography as reference (36–39).

CONCLUSION

In conclusion, AVPD was independently correlated with both LV systolic function and diastolic filling in patients with CAD, irrespective of a history of prior MI. Thus, for any given LVEF, the more pronounced the

diastolic filling impairment, the lower the AVPD. This observation further underscores that AVPD and LVEF are different measures of LV function.

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