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PVF Velocity Pattern in Patients with Heart Failure: Transesophageal Echocardiographic Assessment

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Key Words

Transesophageal echocardiography
Congestive heart failure
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Abstract

In order to assess the role of the pulmonary venous flow (PVF) velocity pattern in the evaluation of patients with congestive heart failure (CHF), we studied 41 CHF patients by means of transthoracic echocardiography (TTE) and multiplane transesophageal echocardiography (TEE). The etiology of CHF was idiopathic or ischemic dilated cardiomyopathy in 19 patients and hypertensive heart disease in 22. Sixteen subjects without cardiovascular disease were selected as normal controls. PVF peak systolic and peak early diastolic (D) velocities were recorded by TEE and TTE and the systolic fraction (SF) was measured (i.e., the systolic velocity-time integral – VTI – expressed as a fraction of the sum of systolic and early diastolic VTI). TEE tracings were obtained in all patients and had more laminar-appearing spectral signals, thus were used for analysis. By TTE the mitral flow velocity patterns were also evaluated: peak early diastolic velocity (E), peak velocity at atrial contraction, E velocity normalized for VTI (E/VTI), deceleration time (DT), and left ventricular isovolumic relaxation time (LVIRT). The left ventricular ejection fraction (LVEF) was calculated by two-dimensional echocardiographic images using the modified Simpson method. The SF was lower in CHF patients as compared with normal controls ($p < 0.0001$). The E/VTI ratio was higher, and DT and LVIRT were shorter ($p < 0.0001$) in CHF patients. A significant correlation was observed between SF and LVEF in CHF patients ($r = 0.76$, $p < 0.001$). Two different PVF velocity patterns (type A: SF $\ll 50\%$, $D > 50$ cm/s; type B: SF $\sim 50\%$, $D > 50$ cm/s) were recognized in patients with a low LVEF (type A) and a nearly normal or normal LVEF (type B). Patients with LVEF < 40% showed mean SF values significantly lower than patients with LVEF > 40% (33.26 ± 10.84 vs. $51.00 \pm 4.00\%$, $p < 0.0001$). Mean DT and LVIRT values were not significantly different in patients with LVEF < 40% and > 40%. Thus in CHF patients TEE PVF velocity patterns help in distinguishing patients with systolic dysfunction (low LVEF and SF) from patients with predominant diastolic impairment (normal or nearly normal LVEF, high D velocities).

Introduction

The pulsatile pulmonary venous flow (PVF) is known to be related to the change of the left atrial pressure [1–3]. The role of the PVF velocity pattern in assessing diastolic filling in individual patients has been stressed upon [4–6]. It has been suggested that this pattern may be more sensitive to loading conditions than the mitral flow velocity pattern, since in patients with left ventricular (LV) diastolic dysfunction the abnormal ratio of peak velocity of transmitral flow in early diastole (E) to that during atrial contraction (A) can be normalized when concomitant congestive heart failure occurs [7–13]. Although Doppler

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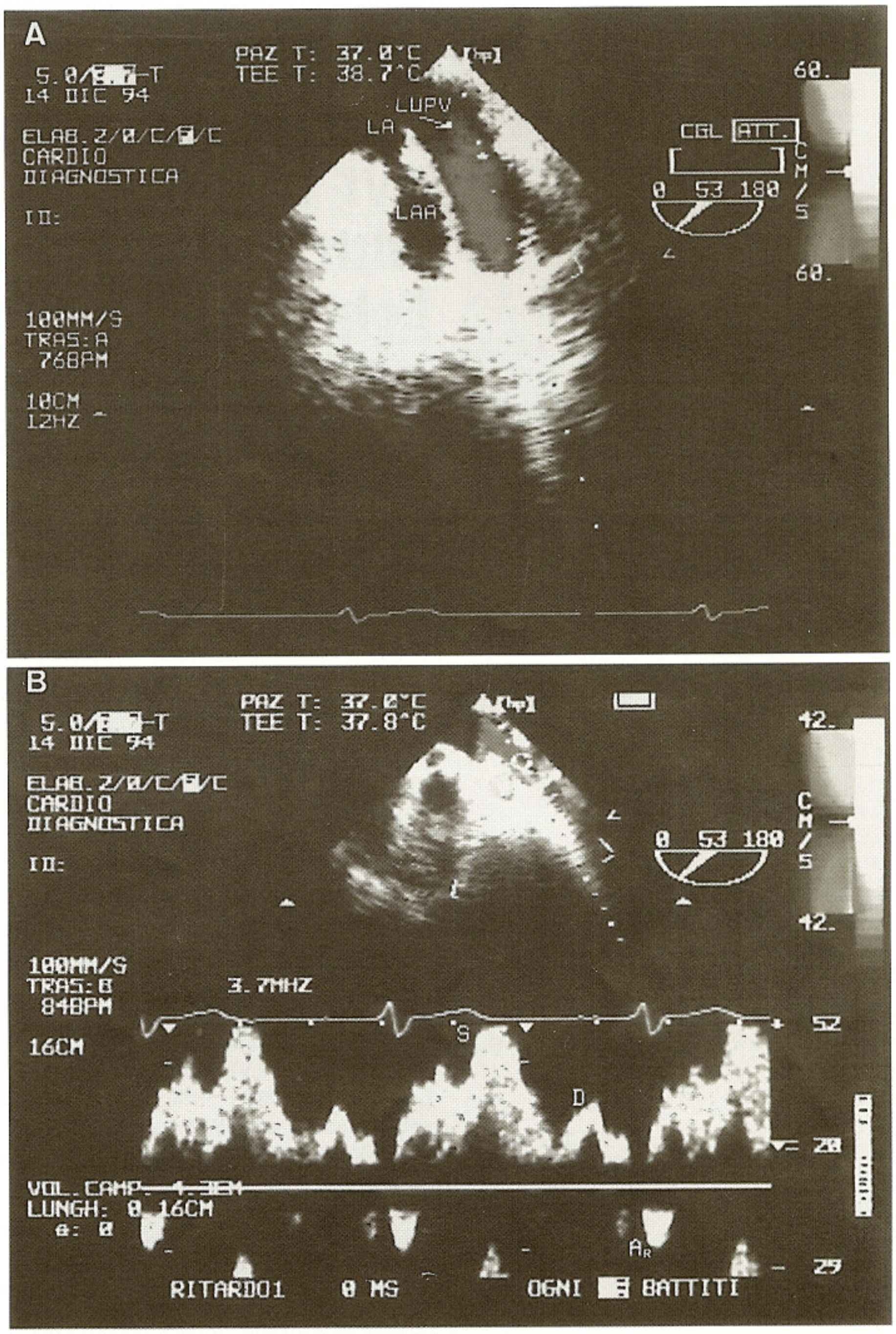

Fig. 1. Multiplane TEE color flow imaging and pulsed Doppler of the left upper pulmonary vein (LUPV) flow in a 47-year-old normal subject. **A** LUPV flow in red. LA = Left atrium; LAA= left atrial appendage. **B** The Doppler beam is aligned as parallel as possible to the long axis of the pulmonary vein, using the multiplane imaging capabilities of the transesophageal probe. The PVF profile is shown with a larger positive peak in S, a smaller peak in D, and a negative peak in atrial systole. A_r = A reversed.

PVF velocity patterns have been previously investigated, some discrepancy exists between transthoracic [14–16] and transesophageal [17, 18] findings. Furthermore, although it has been shown that patterns of diastolic predominance of PVF velocity are associated with increased pulmonary capillary wedge pressure [4], it may be difficult to interpret these findings as a result of impaired systolic function or diastolic dysfunction or both.

The aim of our study was to analyze the PVF velocity pattern by means of color Doppler transesophageal echocardiography (TEE) in patients with congestive heart failure (CHF) for comparing the results with transthoracic

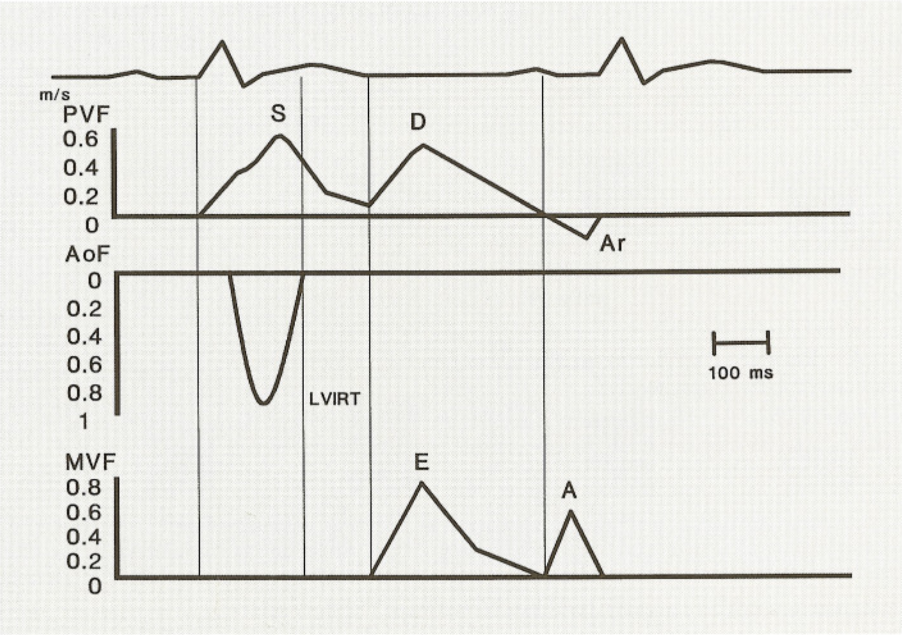

Fig. 2. Diagrams of Doppler echocardiographic tracings of the PVF, aortic flow (AoF), and mitral valve flow (MVF) and their relative timing in the cardiac cycle. A = Late diastolic mitral flow velocity; A_r= atrial reversed PVF velocity. For explanation of the other abbreviations see text.

echocardiography (TTE) findingsand further understanding its value in assessing LV systolic-diastolic dysfunction.

Patients and Methods

Study Group

We studied 41 patients in sinus rhythm with mild to moderate CHF (New York Heart Association functional classes II–III). There were 25 men and 16 women ranging in age from 22 to 70 (mean 55) years. The etiology of CHF was idiopathic or ischemic dilated cardiomyopathy in 19 patients and hypertensive heart disease in 22. Five patients had mild mitral regurgitation as shown by color flow imaging [19] but none had moderate to severe regurgitation. The patients were asked to stop cardiac therapy for 12 h before the study to minimize the possible influence of different loading conditions as a result of medical therapy on Doppler diastolic filling measurements.

Sixteen subjects (ranging in age from 26 to 73 years, mean 50) with normal cardiovascular findings and no evidence of heart failure were selected as controls. Nine subjects were normal volunteers. In the remainder the reason for TEE was a poor transthoracic window in 4 and suspected congenital heart disease in 3.

Transesophageal Echocardiography

Color Doppler TEE and TTE studies were performed with a commercially available equipment (Sonos 1500 or 2500; Hewlett-Packard, Andover, Mass., USA) with a 5/3.5-MHz phased-array multiplane transducer mounted on the tip of a gastroscope. TEE procedures were performed with patients in a mildly sedated fasting state after informed consent was obtained. All patients were in the left decubitus position during examination.

We measured the PVF velocities by positioning the Doppler over the upper left pulmonary vein (fig. 1) approximately 1–1.5 cm proximal to its entrance to the left atrium. The Doppler beam was aligned as parallel as possible to its long axis by using the multiplane imaging capabilities of the transesophageal transducer and observing color Doppler flow indicating maximal forward flow where the audiosignal was maximal. During recording, the wall filter was set as low as possible, and baseline and time resolution settings were adjusted to optimize velocity peaks. PVF velocities were recorded on a strip chart at 50- and 100-mm/s paper speeds with the simultaneous electrocardiographic tracing. Three cardiac cycles were averaged for quantitation.

From the venous flow velocity tracing (fig. 2) we measured peak systolic (S) and peak early diastolic (D) flow velocities, peak velocities of flow reversal at atrial contraction, and velocity-time integrals of the systolic and early diastolic phases. The systolic velocity-time integral (VTI-S) was measured from the onset of forward flow to the onset of the second wave, and the early diastolic velocity-time integral (VTI-D) was measured from the onset of the second wave to its crossover with the zero line. The VTI-S was also expressed as a fraction of the sum of VTI-S and early VTI-D [systolic fraction (SF) = VTI-S/(VTI-S + VTI-D)].

Transthoracic Echocardiography

TTE pulsed-Doppler tracings were obtained with a transducer array of 2.5/2.0 MHz at a paper speed of 50 and 100 mm/s with simultaneous electrocardiographic recording. PVF and mitral flow velocity patterns were examined. PVF velocities were obtained in the apical four-chamber view by placing the sample volume at the orifice of the upper right pulmonary vein. Recordings were made with the patients in a slight left lateral position and during quiet respiration, after minimizing the high-pass filter. The mitral flow velocity pattern was recorded in the apical four-chamber view with the sample volume carefully placed between the tips of the mitral leaflets where the

Table 1. TEE and TTE pulmonary venous velocities in CHF patients

	TEE	TTE
Peak S, cm/s	48±19	37±16*
Peak D, cm/s	70±8	68±9
VTI-S, cm	21±9	15±6*
VTI-D, cm	30±6	27±7
Peak A_r, cm/s	31±12	28±13
SF, %	40±13	35±15*

A_r = Reversed PVF during atrial contraction. For explanation of the other abbreviations see text.
* p<0.05.

Table 2. Matrix of bivariate correlation between pulmonary and mitral flow variables and systolic-diastolic LV function parameters

	LVEF		LVIRT	
	CHF	NRM	CHF	NRM
SF	r=0.755 p<0.001	r=0.557 p=0.025	r=–0.0346 p=0.830	r=–0.496 p=0.050
VTI-D	r=–0.450 p=0.003	r=–0.478 p=0.061	r=–0.031 p=0.847	r=0.174 p=0.517
VTI-S	r=0.694 p=0.001	r=0.045 p=0.867	r=–0.062 p=0.700	r=–0.254 p=0.342
Peak E	r=–0.220 p=0.167	r=–0.243 p=0.364	r=–0.433 p=0.005	r=–0.002 p=0.992
Peak A	r=–0.271 p=0.086	r=–0.082 p=0.761	r=0.295 p=0.061	r=–0.011 p=0.965
DT	r=–0.32 p=0.037	r=–0.144 p=0.595	r=0.794 p<0.001	r=–0.059 p=0.826

CHF = Congestive heart failure; NRM = normal cardiovascular findings. For the other explanations of the abbreviations see text.

maximal flow velocity in early diastole was obtained. The flow velocity measurements at this position were more reproducible than at the mitral annulus level [20].

Along the instantaneous highest velocity spectra we traced the mitral flow velocity profiles to determine peak E filling velocity, peak filling velocity at A, and flow velocity integrals of the E filling wave and the filling wave at A. The flow VTI of the E filling wave was the area under the traced mitral flow velocity profile during the E filling period, and the flow velocity-time integral of the filling wave at the A was the area during the A period. Peak filling rate was expressed [21] as E velocity normalized for VTI (E/VTI ratio). The left ventricular isovolumic relaxation time (LVIRT) was measured [22] from aortic valve closure on the Doppler flow tracing to the start of mitral flow (fig. 2). The deceleration time (DT) was measured [12] from the peak of the E wave to the time when the extrapolated descent of the E wave intercepted the zero baseline. The average values of three consecutive cardiac cycles were used for quantitative analysis.

Standard two-dimensional echocardiographic studies [23] were performed in all patients. The left ventricular ejection fraction (LVEF) was calculated by two-dimensional echocardiography using the modified Simpson rule.

Assessment of Reproducibility

To determine intraobserver and interobserver variability of echocardiographic measurements, parameters were analyzed in a blinded manner in 10 randomly selected patients by one observer on two different occasions (intraobserver variability) and by two independent observers (interobserver variability). For determination of intra- and interobserver variability, the mean values of the absolute differences between the two occasions and those between the two observers were calculated and expressed in percent.

Statistical Analysis

Values are presented as mean ± SD. The statistical significance of the differences between mean Doppler variables in the study group and in the normal group was tested by analysis of variance (Fisher's test). A p<0.05 was considered significant. The correlation between LVEF and pulmonary venous and mitral inflow variables was assessed using simple linear regression analysis.

Results

TEE and TTE PVF Velocity Patterns

Of the 41 patients undergoing TEE, adequate tracings were obtained in all cases. Adequate TTE tracings were recorded in 37 of the 41 patients. The quality of the TTE tracings was usually poorer than that of the TEE tracings, with marked spectral broadening and a low signal-to-noise ratio. In TTE tracings biphasic ventricular systolic forward flow was not observed in any patient, and reversed flow during A was recorded in only 16 (39%). Peak systolic flow velocities (table 1) were significantly lower by the TTE technique (37 ± 16 vs. 48 ± 19 cm/s, p< 0.05). Thus only TEE tracings were used for subsequent analysis.

Comparison of TEE PVF Velocity Pattern in Patients with Heart Failure and in Normal Controls

The peak D flow velocity was significantly higher in patients with heart failure (68.19 ± 8.02 cm/s) than in normal subjects (39.93 ± 11.73 cm/s, p < 0.0001). The flow VTI of diastolic forward flow wave (VTI-D) was significantly higher in patients with heart failure (29.33 ±

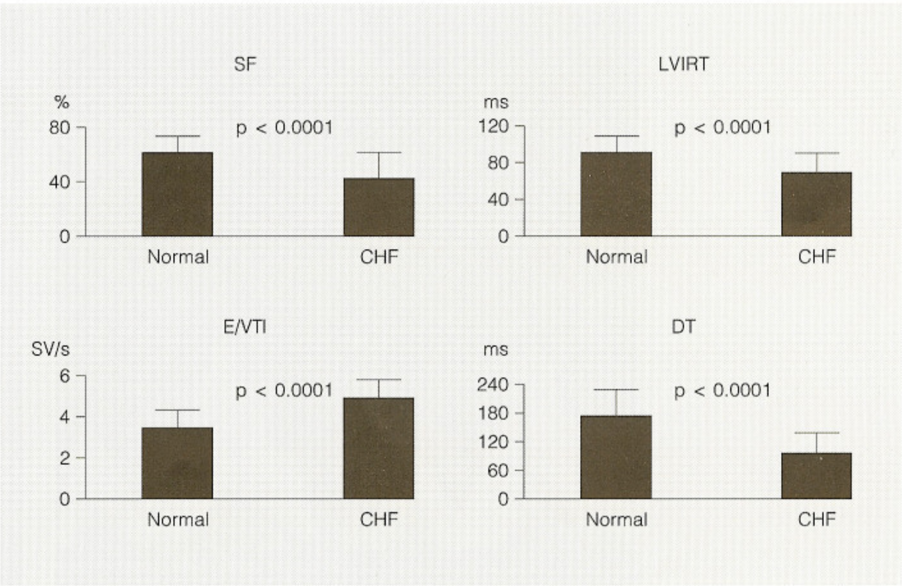

Fig. 3. Bar charts showing a significant difference in mean values of SF of PVF, peak filling rate (E/VTI), DT, and LVIRT between CHF patients and normal controls. SV = Mitral stroke volume.

6.60 cm) than in normal subjects (13.93 ± 3.74 cm, $p < 0.0001$). The ratio of the flow VTI of systolic-to-diastolic forward flow wave and the SF (fig. 3) were lower in CHF patients as compared with normal subjects (0.82 ± 0.55 vs. 1.38 0.71, and 41.04 ± 12.28 vs. 62.12 ± 5.92, respectively, $p < 0.0001$).

Comparison of Mitral Flow Velocity Pattern in Patients with Heart Failure and in Normal Controls

Isovolumic relaxation time, DT, E, E/A ratio, and E/VTI ratio differed in patients with heart failure as compared with normal subjects (fig. 3). E/A ratio (1.63 ± 0.38 vs. 1.18 ± 0.27, $p < 0.0001$) and E/VTI ratio (5.14 ± 1.08 vs. 3.49 ± 0.93 SV/s, $p < 0.0001$) were higher, and DT (97.14 ± 15.41 vs. 172.87 ± 37.85 ms, $p < 0.0001$) and isovolumic relaxation time (70.95 ± 12.29 vs. 92.31 ± 14.62 ms, $p < 0.0001$) were shorter in CHF patients. There was no significant difference in A between the two groups.

Relation between TEE PVF Velocity Pattern and Systolic-Diastolic Ventricular Parameters

A significant correlation (fig. 4, table 2) was shown between SF and LVEF in patients with heart failure ($r = 0.76$, $p < 0.001$). When a cutoff value of 50 cm/s was chosen in advance to judge decreased S and increased D velocities, two distinct PVF patterns (fig. 5) were observed in CHF patients: (A) SF ⩽ 50% with decreased S velocity (<50 cm/s) and increased D velocity (>50 cm/s)

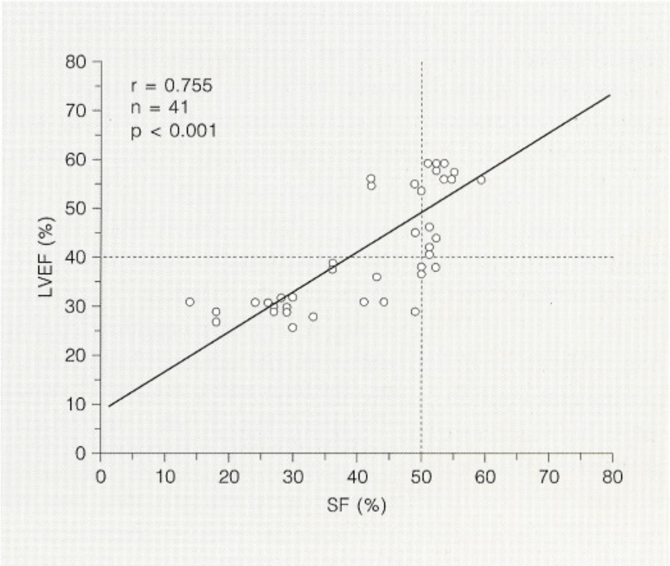

Fig. 4. Scatterplots of linear regression between SF of PVF and LVEF in CHF patients. The horizontal dotted line at 40% marks the arbitrary limit between low LVEF and nearly normal or normal LVEF. The vertical dotted line marks equal systolic and diastolic flow velocity integrals.

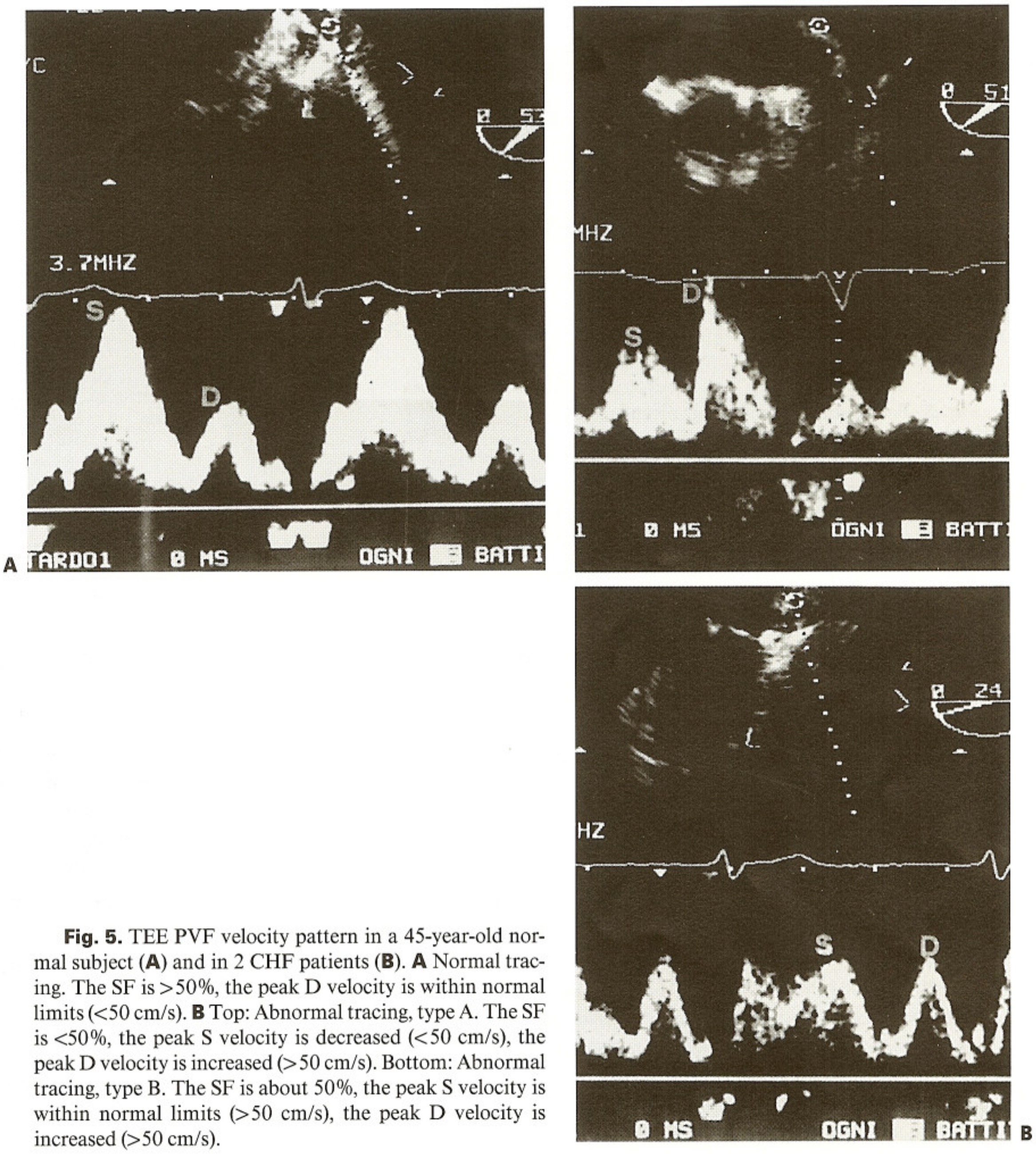

Fig. 5. TEE PVF velocity pattern in a 45-year-old normal subject (**A**) and in 2 CHF patients (**B**). **A** Normal tracing. The SF is >50%, the peak D velocity is within normal limits (<50 cm/s). **B** Top: Abnormal tracing, type A. The SF is <50%, the peak S velocity is decreased (<50 cm/s), the peak D velocity is increased (>50 cm/s). Bottom: Abnormal tracing, type B. The SF is about 50%, the peak S velocity is within normal limits (>50 cm/s), the peak D velocity is increased (>50 cm/s).

and (B) SF about 50% with normal S velocity (>50 cm/s) and increased D velocity (>50 cm/s). Pattern A was observed in 21 patients with a low LVEF (<40%), whereas 18 patients with pattern B had LVEF >40%. Two patients had an undetermined pattern relative to LVEF. Patients with LVEF < 40% showed significantly lower (fig. 6) mean SF values than patients with LVEF > 40% (33.26 ± 10.84% vs. 51.00 ± 4.00%, $p < 0.0001$). Mean DT and LVIRT values were not significantly different in patients with LVEF <40 and >40%.

Relation between Heart Rate and Echo-Doppler Variables

The peak VTI and SF of PVF did not correlate significantly with the heart rate. No significant correlation was shown between LVEF and heart rate. There was a weak

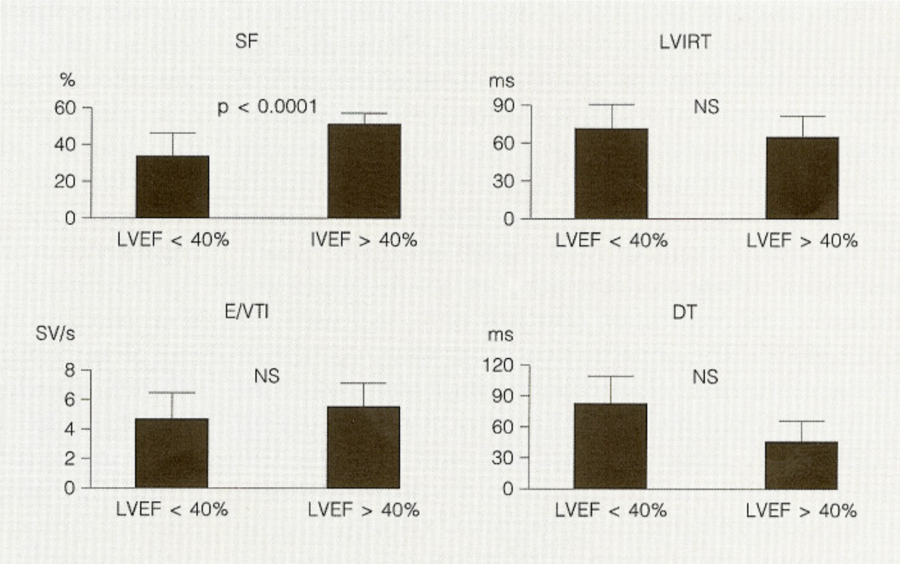

Fig. 6. Bar charts showing mean values of SF of PVF, peak filling rate (E/VTI), DT, and LVIRT in CHF patients with LVEF<40 and >40%. NS = Not significant; SV = mitral stroke volume.

correlation between peak A of mitral inflow and heart rate ($r = 0.39$, $p < 0.05$). No significant difference was observed between CHF patients and normal controls in systolic blood pressure (121 ± 17 vs. 126 ± 11 mm Hg) and diastolic blood pressure (74 ± 10 vs. 73 ± 7 mm Hg).

Reproducibility of Measurements

For TEE PVF velocities, the interobserver variability was 1.8 ± 3.9% for peak systolic velocities and 1.9 ± 4.5% for the SF. The corresponding values for intraobserver variability were 0.9 ± 4.9% and 0.4 ± 2.4%, respectively. For mitral inflow variables, interobserver variability was 0.8 ± 1.8% for peak early diastolic mitral inflow velocities, 2.1 ± 3.3% for peak late diastolic velocities, 0.8 ± 4.7 for E/A ratio, 0.9 ± 5.5 for E/VTI ratio, 23 ± 12 for DT, and 8.9 ± 5.3% for LVIRT. The intraobserver variability values were 0.6 ± 1.7, 2.8 ± 3.9, 0.6 ± 5.1%, 0.7 ± 4.8, 19 ± 11, and 8.1 ± 5.4%, respectively. Inter- and intraobserver variabilities for LVEF were <5%.

Discussion

The present observations suggest that the PVF velocity pattern, obtained by TEE in a cohort of patients with CHF, provides valuable informations in distinguishing systolic-diastolic from diastolic ventricular dysfunction.

Relation of PVF Velocity Pattern to LV Function

It has been shown that the PVF velocity patterns may serve as an 'eyeball index' of mean left atrial pressure and that the level of mean left atrial pressure or pulmonary capillary wedge pressure is related to the SF of PVF [2, 3, 24–27]. The respective influences on this relationship of factors such as left atrial expansion [28], descent of the mitral annulus [29], and left ventricular contractile function [4, 24], as well as the relation of PVF to mitral inflow [17], have been tested.

Despite its limitations in assessing left ventricular performance, the LVEF is a widely used clinical measure of overall systolic function and provides prognostic information in several clinical settings. The significant correlation that we obtained between SF of PVF and LVEF may be explained by the fact that patients with a low LVEF may have higher atrial pressure and, therefore, lower systolic flow velocity of PVF or less systolic displacement of mitral annulus [29]. These findings are consistent with previous studies that showed that the systolic PVF increases directly with cardiac output [17, 30] and that the left ventricular end-systolic dimension is an independent determinant of the S/D ratio [24] when all determining factors are analyzed with stepwise multiple linear regression. We can rule out mitral regurgitation as a factor that may decrease systolic PVF [31–33], since patients with more severe mitral regurgitation were excluded from this study.

The present study also documents the relationship of PVF velocity patterns to some left ventricular diastolic variables. It has been shown that the abnormal mitral flow velocity pattern that is seen in patients with left ventricular diastolic dysfunction can be normalized in association with elevated left atrial filling pressures [8, 13, 34–36]. In patients with impaired ventricular relaxation but with relatively normal chamber stiffness, the reduction in early ventricular filling produces a higher atrial preload and forceful atrial contraction (low E/A ratio, increased DT and LVIRT). The opposite may occur when ventricular filling pressures are elevated, imposing a higher afterload on the left atrium and a shift in ventricular filling toward E (high E/A ratio, decreased DT and LVIRT). Our patients showed an increased mitral E velocity as well as an increased pulmonary venous peak D velocity. Diastolic parameters derived from mitral inflow and isovolumetric relaxation time (E/VTI, DT, LVIRT) were different as compared with normals with a poor correlation with LVEF changes, whereas a significant correlation was shown between SF and LVEF changes. The mean SF values were significantly lower in patients with a low LVEF as compared with patients with a higher or normal LVEF. Patients with a nearly normal LVEF showed an increased pulmonary venous systolic fraction due to a relative increase of peak S velocity compared to increased peak D velocity. Thus in the spectrum of left ventricular dysfunction, a combined assessment of venous SF, E/VTI, and LVIRT is useful in distinguishing those patients with predominant diastolic impairment (normal or nearly normal LVEF, high E and D velocities) from patients with systolic impairment (low LVEF, low systolic fraction).

Caution must be exercised not to falsely diagnose a 'pathologic' PVF pattern on the basis of peak velocity ratio alone. Previous studies [15] showed that the systolic flow velocity can be even <30 cm/s in young normal subjects, suggesting that a low systolic flow does not necessarily indicate abnormal hemodynamics. The correlations between ages and PVF parameters may be explained by the changes in LV diastolic filling associated with aging because of the demonstrated close relation between PVF and LV diastolic filling pattern.

TEE and TTE in Obtaining PVF

TEE examination is more accurate than TTE examination in evaluating PVF because the transducer can be located nearer to the pulmonary vein; there are few obstacles between the transducer and pulmonary vein by TEE approach. The difficulty of obtaining good narrow-band Doppler signals of PVF by the TTE approach may be a possible explanation for the discrepancy in the findings between TTE and TEE studies [3, 15, 18]. Even if it has been shown recently [37] that TTE tracings provide reliable quantitation of the PVF pattern in patients with cardiac disorders, the TEE approach was considered to be better than the TTE in terms of the detection rate.

Some of the velocities may be underestimated if the angle between the vector of the PVF and the ultrasound beam cannot be determined. Both systolic and diastolic flow velocities can be equally affected by the angle. The use of multiplane TEE minimized this effect in our patients, allowing the appropriate selection of the most correct angulation. Unlike the monoplane TEE probe that allows one to study the PVF only in the basal short-axis plane [18], the biplane probe has already been used [37] to improve the alignment of the Doppler beam in the longitudinal scan. The use of the multiplane transducer in multiple intermediate scan planes further improves the possibility to optimize the Doppler incident angle and obtain the best Doppler recordings of the left upper or right upper PVF.

Study Limitations

Some potential limitations of the study should be considered. First, diastolic dysfunction has not been substantiated by invasive hemodynamic monitoring; however, the echo Doppler parameters we used have been extensively validated in previous studies. Second, left atrial compliance or functions were not measured in this study. Third, when using PVF velocities as an indicator of PVF, it is assumed that the cross-sectional area in the pulmonary veins is relatively constant throughout the cardiac cycle. The cross-sectional area in extraparenchymal pulmonary veins decreases during ventricular systole by approximately 20% [38], thus PVF velocities may not have a linear relation to flow volume. However, it has been shown [24] that when the S/D flow VTI is used to assess the pattern of PVF, changes in the pulmonary vein cross-sectional area during the cardiac cycle can be ignored. Finally, only the flow velocity pattern of the left upper pulmonary vein was examined, and this flow does not directly reflect the flow volume filling into the left atrium because of the difference among the four pulmonary veins.

Concluding Remarks

Despite these limitations, this study provides evidence that in patients with heart failure syndrome PVF velocity patterns as determined by TEE are abnormal in those patients with a low as well as in those with a normal

LVEF. PVF velocity patterns may serve not only as an eyeball index of mean left atrial pressure but also to give insight into abnormal LV systolic or diastolic impairment. A combined assessment of the systolic filling fraction of pulmonary venous forward flow velocity, the mitral inflow indexes, and isovolumic relaxation time may improve the capability in distinguishing systolic (low LVEF) from diastolic or systolic-diastolic dysfunction (normal or nearly normal LVEF).

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