Pulmonary Venous Flow by Doppler Echocardiography: Revisited 12 Years Later
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In 2003, pulmonary venous flow (PVF) evaluation by Doppler echocardiography is being used daily in clinical practice. Twelve years ago, we reviewed the potential uses of PVF in various conditions. Some of its important uses in cardiology have materialized, while others have not and have been supplanted by newer approaches. Current applications of measuring PVF have included: differentiating constrictive pericarditis from restriction, estimation of left ventricular (LV) filling pressures, evaluation of LV diastolic dysfunction and left atrial (LA) function, and grading the severity of mitral regurgitation (MR). However, there have been a number of controversies raised in the use of PVF profiles. Using transthoracic echocardiography, there may be technical issues in measuring the atrial reversal flow velocity. The use of PVF in the evaluation of the severity of MR is not always specific and can be affected by atrial fibrillation (AF) and elevated mean LA pressure. Mitral valvuloplasty and radiofrequency ablation for AF, which are the newer applications of PVF in monitoring invasive procedures, are mentioned. This article reviews the important clinical role of Doppler evaluation of PVF, discusses its limitations and pitfalls, and highlights its newer applications. (J Am Coll Cardiol 2003;41:1243–50) © 2003 by the American College of Cardiology Foundation

In 2003, evaluation of pulmonary venous flow (PVF) by Doppler echocardiography is being performed daily. Since the 1970s, PVF was measured invasively using flow meters and was closely related to the pulmonary capillary and left atrial (LA) pressures (1) (Table 1). Pulmonary venous flow was recorded as forward flow during ventricular systole and early diastole with a reversed flow during atrial systole. These flow waves were noted to be reciprocal to the LA pressure waves (2). Noninvasive assessment of the PVF was first reported by Keren et al. (3,4) using pulsed-wave Doppler transthoracic echocardiography (TTE). However, PVF by TTE could be recorded with only systolic and diastolic waves.

Using transesophageal echocardiography (TEE), a complete PVF profile could be clearly recorded because of the posterior approach providing unimpeded interrogation of cardiac structures (5). In 1991, we reviewed the physiology and technique of measuring PVF and described its potential utility in various disease states (6). The PVF profile was proposed as being useful for: differentiating constrictive pericarditis from restrictive cardiomyopathy (7,8); estimating left ventricular (LV) filling pressures (9–11); and evaluating LV diastolic dysfunction (12) and LA function (13,14), severity of mitral regurgitation (MR) (15,16), and stenosis (17,18). Some of its important uses in cardiology have materialized, while others have not.

The purpose of this article, therefore, is to review the place of PVF using Doppler echocardiography, discuss its limitations and pitfalls, as well as mention its newer applications.

Anatomy and physiology of PVF. Between the lung capillaries and the LA, there are the intra- and extraparenchymal pulmonary veins. There are usually four pulmonary veins including the right and left upper and lower veins. The right and left pulmonary veins connect, respectively, medially and laterally to the superior and posterior LA walls (19). The lower veins run below the inferior border of the right and left bronchi, and the upper veins run anterior to their bronchi. The right pulmonary veins run behind the superior vena cava and right atrium and join the LA adjacent to the atrial septum (19).

Recently, the relationship between LA pressure and Doppler-derived PVF has been carefully evaluated (20,21). Pulmonary venous pressure varies according to its proximity to the pulmonary arteries and LA. It resembles the pulmonary artery pressure closer to the pulmonary capillaries and the LA pressure closer to the venoatrial junction (20). The flow in the pulmonary veins is pulsatile, and its waveform shows an inverse relationship to LA pressure (20,21).

IMAGING TECHNIQUE
Characteristics of the normal PVF by TEE. The right pulmonary veins can best be seen at a 45° to 60° angle, and the transducer should be rotated clockwise. In this view, the right upper and lower pulmonary veins appear as a “y” shape. To obtain the left upper and lower veins, the angle should be set at 110°, and the transducer should be rotated counterclockwise. The left lower veins can be visualized by advancing the probe from the position used for the left upper veins (22).
The pulsed-wave Doppler PVF velocity pattern can be recorded by placing the sample volume 1 to 2 cm into the orifice of the pulmonary veins. The normal PVF usually shows a tri- or quadrifascial pattern consisting of a pulmonary venous first systolic wave (S1), pulmonary venous second systolic wave (S2), pulmonary venous early diastolic wave (D), and pulmonary venous atrial reversed flow wave (AR) (16,23) (Fig. 1). Table 2 shows the LA and ventricular factors that influence PVF (6,20,21,24,25). The S1 occurs during LA pressure “a” to “c” and “c” to “x” descent, and the S2 occurs during LA pressure increase between the “x” pressure nadir and the “v” pressure peak (6). There is a direct correlation between the mitral inflow E-wave velocity and the D wave velocity (6).

Characteristics of the normal PVF by TTE. There have been attempts to obtain better quality recordings of PVF by TTE (26,27). One study reported that the measurement of PVF by TTE was feasible and accurate compared with TEE recordings (26). Another study suggested that it was possible to obtain high-quality recordings of PVF in 90% of the patients by TTE with current machine technology, sonographer education, and daily practice (27). Contrast injection may improve the PVF profile (28).

It is the authors’ opinion that the TTE recordings of PVF, especially the atrial reversal, may be limited even with the improvement of transducers. In contrast, TEE can provide clear PVF tracings in most of the patients with more laminar-appearing spectral signals (6). However, TEE may be limited due to its semi-invasive approach, but would be recommended in patients with complex diastolic dysfunction and in assessing hemodynamics.

**Physiologic factors influencing normal PVF velocities.** There are many physiologic variables that will affect PVF including age, preload, LV function, atrioventricular (AV) conduction, and heart rate (6,20,29,30–34). The aging process will influence PVF, and there are published normal values with 95% confidence intervals (29). Increased or decreased preload may change the S2 and AR velocities reflecting the Frank-Starling mechanism (30). Thus, PVF can provide a relatively noninvasive means to assess directional changes in LV preload. There is a significant correlation between S2 velocity and LA pressure in patients with a normal cardiac index (31). The change induced by volume loading in the S2/D ratio positively correlates with the change in LA pressure in normal LV function. This indicates that the S2/D ratio can estimate the LA reservoir function (32,33). In the absence of LV dysfunction, PVF can provide an estimate of mean LA pressure and is determined largely by atrial function (32,34).

**Evaluation of LV diastolic function.** Diastolic dysfunction has been evaluated noninvasively using the Doppler mitral inflow velocity (6); PVF was the first to provide additional information for differentiating pseudonormal from normal LV filling (6,9,35).

**Normal PVF.** The effects of age on PVF in normal subjects have been described previously (29). In healthy older subjects, the PVF shows a greater systolic than diastolic flow, and there are increased atrial reversals compared with younger normal subjects.

**Abnormal PVF. RELAXATION ABNORMALITY.** In patients with impaired LV relaxation, the mitral inflow E velocity decreases with a longer deceleration time, reflecting a decreased early diastolic LV filling rate. The mitral inflow A velocity increases because of the complementary mechanisms. Corresponding to these changes, the pulmonary venous systolic fraction and the S2/D ratio increases, and the deceleration time of the D wave prolongs so that the LA pressure peak (6). There is a direct correlation between the mitral inflow E-wave velocity and the D wave velocity (6).

**Table 1.** History of the Clinical Application of PVF

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Description</th>
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<tbody>
<tr>
<td>Skagseth (1), Rajagopalan et al. (2)</td>
<td>1982</td>
<td>Invasive assessment of PVF</td>
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<tr>
<td>Keren et al. (3,4)</td>
<td>1984</td>
<td>Noninvasive assessment of PVF by TTE</td>
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<tr>
<td>Seward et al. (5)</td>
<td>1985</td>
<td>Technique for recording PVF by TEE</td>
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<tr>
<td>Schiavone et al. (7), Klein et al. (8), Klein et al. (43)</td>
<td>1986</td>
<td>Differentiation of constrictive pericarditis from restrictive cardiomyopathy</td>
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<tr>
<td>Kuecherer et al. (9), Appleton et al. (35)</td>
<td>1987</td>
<td>Assessment of the LV filling pressures</td>
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<td>Rossov et al. (10), Yama moto et al. (11), Dini et al. (28)</td>
<td>1988</td>
<td>Estimation of the LV end-diastolic pressure</td>
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<tr>
<td>Klein et al. (6)</td>
<td>1989</td>
<td>Evaluation of the diastolic dysfunction</td>
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<tr>
<td>Oki et al. (13,14)</td>
<td>1990</td>
<td>Evaluation of the LA function</td>
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<tr>
<td>Klein et al. (46), Castello et al. (15)</td>
<td>1991</td>
<td>Assessment of mitral regurgitation severity</td>
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<td>Klein et al. (17), Tahata et al. (18), Stojnic et al. (56)</td>
<td>1992</td>
<td>Evaluation of mitral stenosis</td>
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<tr>
<td>Klein et al. (6,46)</td>
<td>1993</td>
<td>Monitoring mitral valve procedures</td>
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<tr>
<td>Robbins et al. (63), Scanavacca et al. (64), Sohn et al. (66)</td>
<td>1994</td>
<td>Pulmonary vein stenosis after catheter ablation</td>
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LA = left atrial; LV = left ventricular; PVF = pulmonary venous flow; TEE = transesophageal echocardiography; TTE = transthoracic echocardiography.
reservoir volume during ventricular systole could compensate for the impaired early LV filling (36,37) (Fig. 2A).

**PSEUDONORMALIZATION.** The mitral inflow pattern changes in relation to myocardial function and hemodynamic status, such as preload. An increase in LA pressure normalizes the abnormal mitral inflow pattern and masks the LV relaxation abnormality (6). The mitral inflow E-wave velocity increases, and the A-wave velocity decreases. There are a number of methods to differentiate “pseudonormalization” from a normal mitral inflow pattern (37,38). The classic way was by observing a normal or decreased S2 (“blunted” systolic pattern) and increased D velocities resulting in decreased systolic fraction and S2/D ratio and with a large atrial reversal >35 cm/s (6,38) (Fig. 2B). Another method was by decreasing preload with the Valsalva maneuver (38). The main limitation using PVF in assessing the pseudonormal pattern is the difficulty of accurately recording the atrial reversal velocity.

**RESTRICTIVE PHYSIOLOGY.** The primary abnormality in patients with restrictive cardiomyopathy, such as in advanced cardiac amyloidosis, is increased chamber stiffness. In patients with a restrictive mitral inflow pattern (a deceleration time <150 ms), the PVF shows a lower S2 and higher D velocities (severely blunted systolic flow) and increased atrial reversals (unless atrial systolic failure), suggesting decreased LV operating compliance (12) (Fig. 2C).

**ESTIMATION OF LV FILLING PRESSURES.** Pulmonary venous flows have been used to clinically estimate mean LA pressure; LA pressure has been shown to have a negative correlation with pulmonary venous systolic fraction and S2/D ratio in those patients with pseudonormal and restrictive physiology (9,39). A systolic fraction ≤55% was found to be 91% sensitive and 87% specific in predicting a mean LA pressure >15 mm Hg (40). However, S2 velocity is not only affected by LA pressure, but also by LV contractility (41). There is a negative correlation between the S2 velocity and the LA pressure in patients with a low cardiac index because of the decrease in the systolic descent of the mitral annulus (31,32,41).

On the other hand, the difference between the PVF-AR wave duration and the mitral inflow atrial-wave duration has been reported to correlate with an increase in LV pressure during atrial contraction and LV end-diastolic pressure (10,28). The PVF-AR wave duration (exceeding mitral inflow A-wave duration by 30 ms) is reported to provide high sensitivity (82%) and specificity (92%) for the detection of LV end-diastolic pressure >20 mm Hg (11,28).

**PVF in pericardial disease.** **CONSTRUCTIVE PERICARDITIS.** The hemodynamic characteristics of constrictions show markedly elevated atrial and ventricular pressures and an early diastolic “dip-and-plateau” pattern (42). The respiratory variation of the Doppler flow velocities has been reported in the differentiation between constriction and restriction (7,43,44). In restrictive cardiomyopathy, PVF

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**Table 2. Left Atrial and Ventricular Factors Influence on Each Wave of PVF**

<table>
<thead>
<tr>
<th>Wave of PVF</th>
<th>Ventricular Function</th>
<th>Atrial Function</th>
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</thead>
<tbody>
<tr>
<td>First systolic wave</td>
<td>LV contraction</td>
<td>Atrial relaxation</td>
</tr>
<tr>
<td>Second systolic wave</td>
<td>RV contraction</td>
<td>Reservoir function</td>
</tr>
<tr>
<td>Early diastolic wave</td>
<td>Ventricular relaxation</td>
<td>Atrial compliance</td>
</tr>
<tr>
<td></td>
<td>Ventricular chamber stiffness</td>
<td>Conduit function</td>
</tr>
<tr>
<td>Atrial reversal wave</td>
<td>Ventricular chamber stiffness</td>
<td>Booster pump function</td>
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LV = left ventricular; PVF = pulmonary venous flow; RV = right ventricular.
shows blunting of the S2 velocity and decreased S2/D ratio throughout the respiratory cycle (Fig. 3A). In contrast, marked respiratory change in PVF was observed in constrictive pericarditis (Fig. 3B). The S2 and D velocities increased, especially the D velocity, during expiration, and decreased during inspiration. This is explained by incomplete transmission of the inspiratory fall of intrathoracic pressure to the LA (44). Those changes were more prominent compared with changes in mitral inflow velocities (44). The combination of the S2/D ratio >0.65 in inspiration and a respiratory variation of D velocity >40% correctly classified 86% of patients with constrictive pericarditis (43). Similar respiratory variation can also be observed even in patients with constrictive pericarditis and atrial fibrillation (AF) regardless of the irregular cycle lengths (45) (Fig. 3C).

**PVF in mitral valve diseases. MITRAL REGURGITATION.** Twelve years ago, PVF was suggested to estimate the severity of MR (15,16,46,47). As the degree of MR increases, the S2 velocity decreases, thus causing systolic blunting and then late systolic flow reversal and, finally, pan-systolic reversal occurs, while the D velocity increases (46) (Fig. 4A). A qualitative grading system for MR was proposed using PVF. Normal systolic flow was seen in patients with 1+ or 2+ MR, whereas blunted and reversed systolic flows were detected in patients with 3+ and 4+ MR, respectively. Reversed systolic flow was seen in 93% of the patients with 4+ MR (46) (Fig. 4B). The sensitivity and specificity of reversed systolic flow for severe MR were reported as 90% to 100% by Castello et al. (15) and 82% and 100%, respectively, by Kamp et al. (47). Mitral regurgitation was the most common cause of large LA pressure “v” wave (48), and the “v” wave size and regurgitant volume showed a significant relationship in determining pulmonary venous reversed systolic flow (49). Furthermore, the changes in D velocity were closely related to changes in the “v” wave in MR under altered loading conditions (50). The best correlation of the S2/D ratio was found with the LA pressure “v” wave (r = −0.76), the “v-y” descent (r = −0.73), and the “a/v” ratio (r = 0.71).

On the other hand, there were significant problems in using PVF in the grading of MR. A large “v” wave is neither highly sensitive nor specific for severe MR (51). Increased LA compliance may be associated with trivial “v” wave in the presence of severe MR (48). In addition, there are a number of other physiologic and technical factors influencing either the S2 or D velocities, such as mitral stenosis, presence of LV dysfunction, and presence of AF (10,23,35). The decrease in the velocity time integral of PVF is more prominent for any given volume of MR at a higher LA pressure (52). Jet directions and jet areas may also influence the effect of MR on PVF patterns (53). There are some patients with discordance between the left and right upper PVF patterns. Both PVF patterns must be evaluated when assessing the severity of MR (54) because the left PVF
usually shows blunted systolic flow, and the right PVF shows reversed systolic flow—depending on jet direction (46). Despite these limitations and pitfalls, reversed systolic flow is a highly specific marker of severe MR, whereas the normal PVF is useful to confirm the presence of mild-to-moderate MR. The blunted PVF pattern must be interpreted cautiously in the clinical practice as a marker for the severity of MR (53,55).

**MITRAL STENOSIS.** The characteristics of the PVF pattern in patients with mitral stenosis and normal sinus rhythm are lower S2, D, and AR velocities (18,56). The pressure

![Figure 3](image-url)  
**Figure 3.** Pulmonary venous flow (PVF) velocity profiles recorded by transesophageal echocardiography with respiratory monitoring. (A) Patient with cardiac amyloidosis shows pseudonormal pattern characterized by slight blunting of pulmonary venous systolic wave (S) throughout the respiratory cycle with a large atrial reversal. (B) Patient with constrictive pericarditis and sinus rhythm shows a marked respiratory variation. Both the pulmonary venous systolic and early diastolic (D) flow velocities decreased from expiration to inspiration. (C) Patient with constrictive pericarditis and atrial fibrillation also shows similar respiratory variation in the PVF. Both the S and D velocities increased at the onset of expiration, even with a short RR interval, and decreased at the onset of inspiration with a long RR interval. Exp = expiration; Insp = inspiration.

![Figure 4](image-url)  
**Figure 4.** (A) Simultaneous recording of the pulmonary venous flow (PVF) using transesophageal echocardiography and left atrial pressure (LAP) in patients with 4+ mitral regurgitation (MR). The pulmonary venous systolic wave (S) was blunted, and late systolic reversal flow (SRF) was observed corresponding to the large LAP "v" wave. (B) Relationship between LAP and PVF in 2+, 3+, and 4+ MR. As MR grade increases, the "v" wave and "v-y" descent increase, and the "a" wave and "a-x" descent decrease, which is consistent with decrease in S wave, increase in D and SRF waves. ECG = electrocardiogram. From Klein AL, Savage RM, Kahan F, et al. Experimental and numerically modeled effects of altered loading conditions on pulmonary venous flow and left atrial pressure in patients with mitral regurgitation. J Am Soc Echocardiogr 1997;10:41–51; reproduced with permission.
half time of the D wave is longer and correlated with that in the mitral inflow E-wave because of the gradual decay of the AV pressure gradient (17,56). We observed the blunted PVF pattern in 61% of the patients with mitral stenosis (17). In patients with severe mitral stenosis, LA filling shows a diastolic preponderance (57). The LA contribution to the LV filling and AR velocity correlates positively with the mitral valve area and negatively with the mean LA pressure in patients with sinus rhythm (58). Patients with mitral stenosis and AF have a predominantly blunted systolic pattern. The S2 velocity markedly decreases in the presence of AF due to loss of timed atrial function, and the early diastolic phase is the main LA filling phase (17,56).

**Effects of rhythm disorder.** Rhythm disorders may definitely influence the use of PVF in clinical practice. The AR and S1 waves are generated by active LA contraction and relaxation, respectively (14,59); because of the loss in effective LA function, both of them disappear in patients with rhythm disorders, such as AF and asynchronous AV conduction (23,60). Those velocities are small immediately after restoration to sinus rhythm from AF due to temporal LA stunning, but subsequently increase over time (59,61). In AF, the onset of the S2 wave is delayed, and the S2 velocity and systolic fraction are reduced with increased D velocity (23,41). The S2 velocity is especially lower in patients with LV dysfunction than in those with lone AF. The S2 velocity and LA pressure “v” wave are relatively constant in patients with lone AF, whereas they change corresponding to the preceding cardiac cycle lengths in patients with LV dysfunction (62).

**PVF as a monitor during invasive procedures.** **MITRAL VALVE DISEASE.** In the operating room, we have assessed residual MR during mitral valve repair and demonstrated the return to normal PVF after a successful procedure (46). Similarly, in patients with severe mitral stenosis, successful mitral valvuloplasty could be associated with an immediate increase in S2 velocity (57).

**RADIOFREQUENCY ABLATION.** A new use of monitoring PVF is the detection of pulmonary vein stenosis after radiofrequency catheter ablation for AF. The focal origin of AF has been recently reported to be mainly inside of the pulmonary veins, and catheter ablation has been demonstrated to interrupt chronic incessant AF. However, the progressive veno-occlusive pulmonary syndrome with pulmonary hypertension as a consequence of pulmonary vein stenosis was reported to be a major complication of this procedure (63). Using TEE, the site of stenosis in all four pulmonary veins could be observed two-dimensionally and the severity estimated by the increased PVF velocities (Fig. 5). This complication should be acutely treated by balloon dilation (64).

**Future status of PVF evaluation.** From the available evidence, there are certain indications for the routine use of PVF by Doppler echocardiography. First, it will be useful in assessing LV diastolic dysfunction using an integrated approach with mitral inflow, as well as estimating LV filling pressures (6,9,10,35) especially in patients with decreased LV systolic function (65). On the other hand, tissue Doppler echocardiography and color M-mode Doppler may be more useful in patients with normal LV systolic function (65). Second, it will continue to be key in differentiating constriction from restriction by noting the enhanced respiratory variation of the diastolic flow (43,45). Third, it will play a major contribution in the evaluation of the severity of MR (66). Finally, evaluation of the orifice of all four pulmonary veins and its flow characteristics by TEE (67) or intracardiac ultrasound (68,69) will play an increasing role in radiofrequency ablation for AF.
Conclusions. Pulmonary venous flow revisited 12 years later is still "alive and well" and will continue to play an important role in clinical practice.

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REFERENCES


