Pulmonary Venous Flow: Its Relationship to Left Atrial and Mitral Valve Motion

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The factors affecting pulmonary venous flow were studied in a group of 50 patients divided into four groups. Group 1 consisted of 14 normal subjects. Group 2 consisted of 10 patients with pure mitral stenosis and normal sinus rhythm. Group 3 consisted of 15 patients with pure mitral stenosis and atrial fibrillation. Group 4 consisted of 11 patients with atrial fibrillation alone. Pulmonary venous flow, atrial septal motion, and mitral valve flow were obtained by transesophageal echocardiography. Pulmonary wedge pressure or left atrial pressure was measured invasively in all patients. We observed that pulmonary venous flow had a reversed flow during atrial contraction and a biphasic flow in the ventricular phase. The first phase of flow occurred during ventricular systole, corresponding to the beginning of atrial relaxation. The second phase of flow, during ventricular diastole, occurred consistently after the rapid filling wave of mitral flow. The beginning of the second phase, corresponding to maximal relaxation of the atrial septum, reached a maximum corresponding to the beginning of atrial contraction. Results of the present study also indicate that decrease or absence of reversed atrial flow and decrease in the first phase of ventricular flow were noticeable in patients in Groups 2, 3, and 4. The second phase of pulmonary venous flow was significantly decreased in patients with mitral obstruction (Groups 2 and 3), but was maintained when the mitral valve was normal (Group 4). In six patients undergoing percutaneous mitral valvuloplasty, the ventricular phase of pulmonary venous flow increased. We concluded that the pulmonary venous flow is influenced by dynamic changes in the left atrium and in mitral valve motion. The atrial reversal flow and the first phase in pulmonary venous flow are strongly related to the change in left atrial pressure, atrial contraction, and suction effect of the atrium. The second phase is related to mitral valve motion. (J Am Soc Echocardiogr 1993;6:387-94.)

It was recently demonstrated that transmitral flow velocities recorded by pulsed Doppler echocardiography correlate well with invasive measurements of diastolic left ventricular filling,[1] and that pulmonary venous flow velocities reflect changes in left atrial pressure during the cardiac cycle.[2] Thus by analyzing pulmonary venous and mitral flow and their relation to each other, a more complete evaluation of left ventricular diastolic function may be possible.[3]

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Transesophageal echocardiography with pulsed Doppler and color flow imaging capabilities provides an excellent means to evaluate pulmonary venous flow velocities.[4] The proximity of the pulmonary veins to the transducer by the posterior transesophageal approach offers high-resolution images of these cardiac structures and the opportunity to locate the sample volume precisely in the orifice or farther into the vein. Therefore, we investigate further the relationship between pulmonary venous flow and atrial and mitral valve motion during the cardiac cycle.

MATERIALS AND METHODS

Subjects

Fifty patients were divided into four groups. Group 1 consisted of 14 patients (mean age = 55 ± 24

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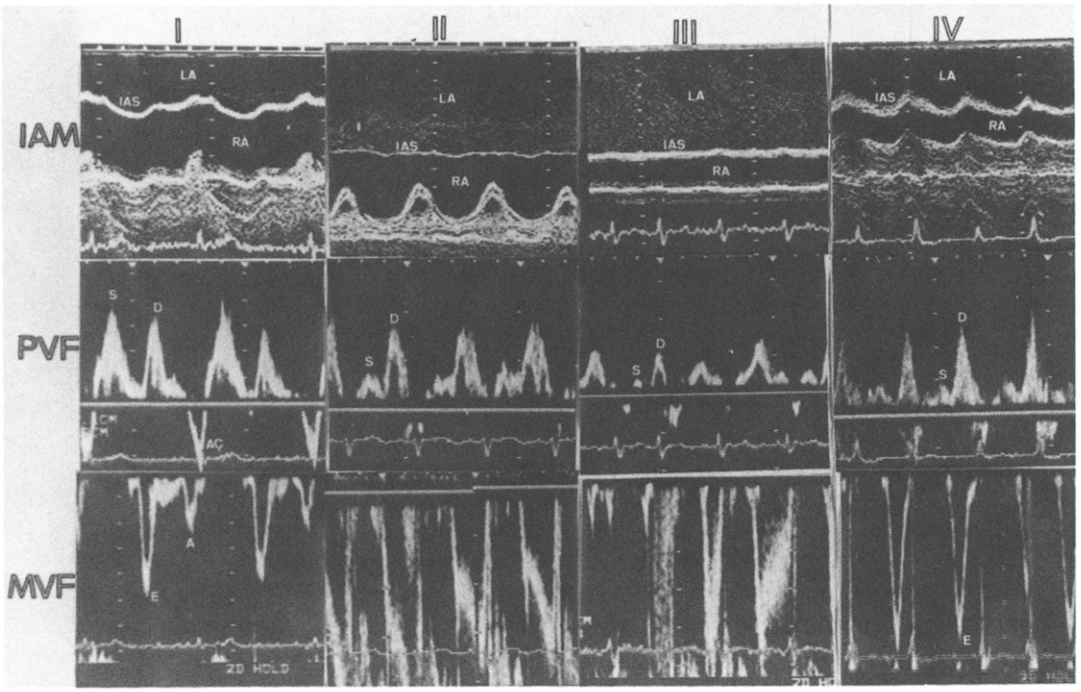

Figure 1 Interatrial septal motion *(IAM),* pulmonary venous flow *(PVF),* and mitral valve flow *(MVF)* in all groups showing the time relationship between the various phases. Group I: normal subjects; group II: pure mitral stenosis with normal sinus rhythm; group III: pure mitral stenosis with atrial fibrillation; group IV: patient with atrial fibrillation alone. *LA,* Left atrium; *IAS,* interatrial septum; *RA,* right atrium; *S,* first peak of pulmonary venous flow; *D,* second peak of pulmonary venous flow; *AC,* reverse flow of atrial contraction; *E,* early peak mitral flow; *A,* late peak mitral flow.

years) with atypical angina pectoris, who served as a control group. These patients had normal coronary arteries and normal left ventricular function documented by cardiac catheterization. Their electrocardiogram showed normal sinus rhythm. Group 2 consisted of 10 patients (mean age = 54 ± 24 years) with pure mitral stenosis. In this group, all patients had normal sinus rhythm undergoing cardiac catheterization. Group 3 consisted of 15 patients (mean age = 53 ± 20 years) with atrial fibrillation and pure mitral stenosis as proved by cardiac catheterization. Group 4 consisted of 11 patients (mean age = 58 ± 4 years) with normal mitral valve and atrial fibrillation. All these patients had idiopathic atrial fibrillation. They underwent cardiac catheterization and had normal coronary arteries and left ventricular function.

Echocardiography

Transthoracic M-mode and two-dimensional echocardiograms were performed with a 2.25 MHz phased array transducer with a Hewlett-Packard cardiac imaging system (Hewlett Packard, Andover, Mass.). Routine echocardiographic views were obtained in all patients. The left atrial diameter was measured by M-mode echocardiography at end systole, according to the recommendation of the American Society of Echocardiography. Mitral flow was obtained with pulsed wave Doppler from the apical four-chamber view of the left ventricle. Mitral flow was obtained by placing the sample volume (5 mm width) at the tips of the mitral valve leaflets. The mitral valvular area was calculated by the pressure half-time from continuous wave Doppler echocardiography.

Transesophageal echocardiography was performed with a 5 MHz phased array probe mounted to the tip of a standard gastroscope, interfaced to a Hewlett-Packard ultrasound imaging system. Patients fasted for at least 6 hours and local pharyngeal anesthesia with 2% lidocaine (Xylocaine) was administered twice before the gastroscope was inserted. The pa-

Table 1 Comparison of hemodynamic and echocardiographic data in 50 patients

Groups	Left atrium		Pulmonary venous flow velocity						Mitral flow		
	Amplitude of IAS (cm)	Dimension (cm)	Atrial reversion flow (cm/sec)	Ventricular phase (cm/sec) S	Ventricular phase (cm/sec) D	Mean PWP (mm Hg)	QD (msec)	DDT (msec)	E (cm/sec)	QE (msec)	EDT (msec)
I. Normal (N = 14)	1.25 ±0.19	2.28*‡§ ±0.17	32.18 ±12.38	64.70 ±10.84	46.78 ±14.04	7.35†‡ ±1.80	578.6 ±24	158.4 ±13	77.6 ±11	542 ±11	164 ±21
II. Mitral stenosis and normal sinus rhythm (N = 10)	0.53*§ ±0.13	4.35‡ ±0.23	13.00* ±3.38	29.50* ±8.26	36.75 ±6.76	22.00 ±3.12	516 ±20	288 ±50	246 ±30	470 ±17	590 ±110
III. Mitral stenosis and atrial fibrillation (N = 15)	0.24*†§ ±0.08	5.71 ±0.94		9.45*†§ ±4.01	23.73*§ ±9.44	23.08 ±2.43	490 ±38	266 ±30	188.3 ±21	466 ±40	571 ±60
IV. Atrial fibrillation alone (N = 11)	0.87* ±0.14	3.47†‡ ±0.44		28.95* ±8.86	49.34 ±6.84	7.80†‡ ±1.40	478 ±38	129 ±11.4	80.9 ±10	428 ±18	139 ±14.3

S, Peak of pulmonary venous flow in ventricular systole; *D*, peak of pulmonary venous flow in ventricular diastole; *E*, early mitral filling velocity; *IAS*, interatrial septum; *QD*, time to peak pulmonary venous diastolic flow; *QE*, time to peak early filling velocity; *DDT*, deceleration time of pulmonary venous diastolic flow velocity; *EDT*, deceleration time of peak early filling velocity; *PWP*, pulmonary wedge pressure.
*Each group's comparison with Group I, $p < 0.05$.
†Each group's comparison with Group II, $p < 0.005$.
‡Each group's comparison with Group III, $p < 0.005$.
§Each group's comparison with Group IV, $p < 0.05$.

tients were studied in the supine or in a slightly lateral position and were monitored by means of continuous electrocardiography. The transesophageal studies usually took less than 10 minutes without any complications.

The transducer was manipulated to obtain a four-chamber view the sample volume was placed at the tip of the mitral valve leaflets to obtain the highest velocity. The scope was then slightly withdrawn and the tip was flexed and turned to the left to obtain a clear view of the left pulmonary vein as it emptied into the left atrium. A sample volume was placed <1 cm into the pulmonary vein from its junction with the left atrium, and color flow imaging was used to obtain a beam direction as parallel as possible to the pulmonary vein flow. An atrial septal motion can be obtained at the atrial septal view with the sound beam directed perpendicular to the atrial septum, and M-mode of the atrial septal motion was recorded throughout the cardiac cycle. We use this atrial septal motion to study the left atrial wall motion.

From pulmonary venous flow velocity tracings we measured peak systolic and peak early diastolic flow velocity and peak velocities of reversal during atrial contraction. The time interval measured was the time from onset of the QRS complex to the peak of diastolic flow. The deceleration time of early diastolic filling was measured from the peak velocity to the extrapolated line intersecting the baseline. From Doppler tracings of mitral flow we measured peak early diastolic flow velocity, and deceleration time of early diastolic velocity was measured in a manner similar to that of the deceleration time for the early diastolic filling velocities. The time interval measured was the time from onset of the QRS complex to the peak of diastolic flow.

The relationship between phases of pulmonary venous flow, mitral flow, and M-mode echocardiographic correlations of atrial septal motion were analyzed in all patients. All time measurements were related to the QRS complex on the electrocardiogram.

Cardiac Catheterization

Complete catheterizations of the left and right sides of the heart were performed in all patients via the femoral artery and vein. All patients provided informed consent. The pressures in all cardiac chambers, including six cases of left atrial pressure, were measured through the transseptal technic. The mean pressure gradient was measured by use of the difference of pulmonary wedge pressure or left atrium and

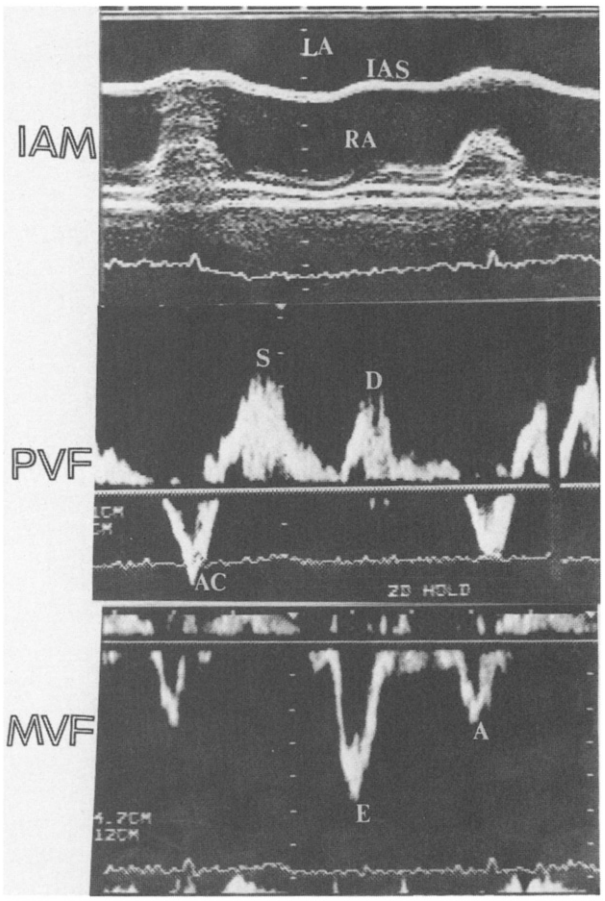

Figure 2 Interatrial septal motion (IAM), pulmonary venous flow *(PVF),* and mitral valve flow *(MVF)* in Figure 1 normal subject with paper speed 100 cm/sec. *LA,* Left atrium; *IAS,* interatrial septum; *RA,* right atrium; *S,* first peak of pulmonary venous flow; *D,* second peak of pulmonary venous flow; *AC,* reverse flow of atrial contraction; *E,* early peak mitral flow; *A,* late peak mitral flow.

left ventricular diastolic pressure. Left ventriculography was performed in right oblique 45-degree projection and lateral projection in all cases. Coronary angiographies were performed in some groups of patients. Repeated measurements of hemodynamic data in six cases were performed after percutaneous mitral valvuloplasty.

Statistical Analysis

The results are expressed as the mean ± SD. Statistical analyses were performed with a one-way ANOVA Scheffe method and Wilcoxon matched-pair signed-rank tests. A p value of less than 0.05 was considered as statistically significant.

RESULTS

Figure 1 shows the characteristic pattern of flow through the pulmonary veins in all groups of patients as related to the atrial septal motion and flow through the mitral valve. The tracings were not recorded simultaneously but taken a few minutes apart and synchronized at the same heart rate with the electrocardiogram as a marker. The pertinent hemodynamic and pulmonary venous flow data were listed in Table 1.

In Group 1 (normal) subjects, the systolic and diastolic phases of pulmonary venous flow and the reversal flow during atrial contraction were recorded (Figure 2). The mean peak flow velocities of the systolic and diastolic phases of pulmonary venous flow were 64.70 ± 10.84 and 46.78 ± 14.04 cm/sec, respectively. The diastolic phase reached its maximum at 542 ± 11 msec after the QRS complex and 36.5 ± 13 msec after the rapid filling peak wave (E wave). On opening the mitral valve, there was rapid early forward flow across the mitral valve followed by deceleration of flow. In the pulmonary vein, there was a nearly simultaneous but slightly slower increase in forward flow velocity with subsequent deceleration soon after the deceleration of mitral flow. The contours of the diastolic velocities were similar in both the mitral and pulmonary vein tracings, resulting in similar deceleration times (164 vs 158.4 msec). The mean pulmonary wedge pressure and the left atrial internal dimension were in the normal range for this group of patients.

In Group 2 (mitral stenosis) patients, the amplitude of the atrial septal motion and the reversal flow of atrial contraction were lower than in those of Group 1. However, the mean pressure gradient between the pulmonary wedge and left ventricle and the left atrial internal dimension were higher than in the normal subjects. Moreover, the deceleration times of the mitral flow and diastolic forward flow in Group 2 were longer and had more proportional increases than those of Group 1.

In Group 3 (atrial fibrillation with mitral stenosis) patients, atrial septal motion became flattened, the left atrial internal dimension was increased, and the reversal flow of atrial contraction was not detectable. However, the systolic phase of pulmonary venous flow was small, and only in the diastolic phase was a significant flow recorded. The pulmonary wedge pressures of Group 3 patients were also higher than those in Group 4. Moreover, the deceleration times of the E wave and pulmonary diastolic flow were

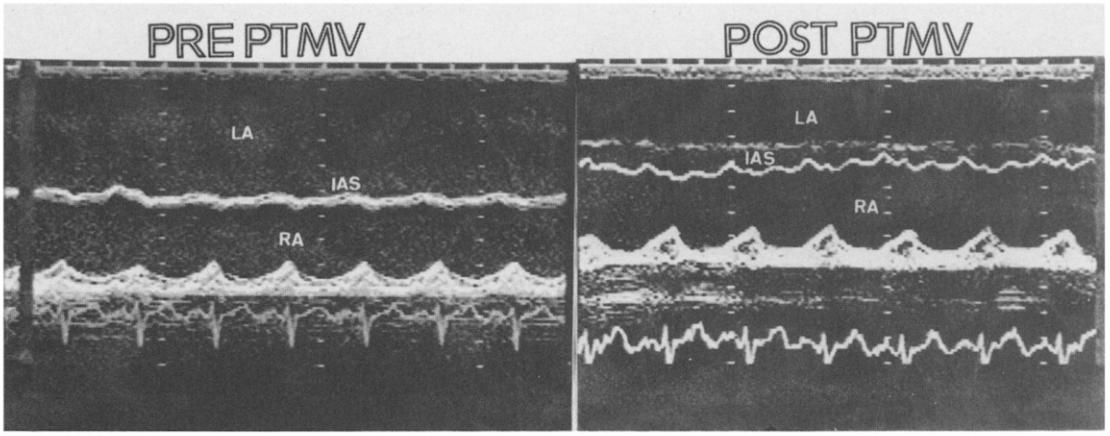

Figure 3 Interatrial septal motion in a patient with rheumatic heart disease with sinus rhythm (Case 5) before *(PRE PTMV)* and after *(POST PTMV)* mitral valvuloplasty. *LA,* Left atrium; *IAS,* interatrial septum; *RA,* right atrium.

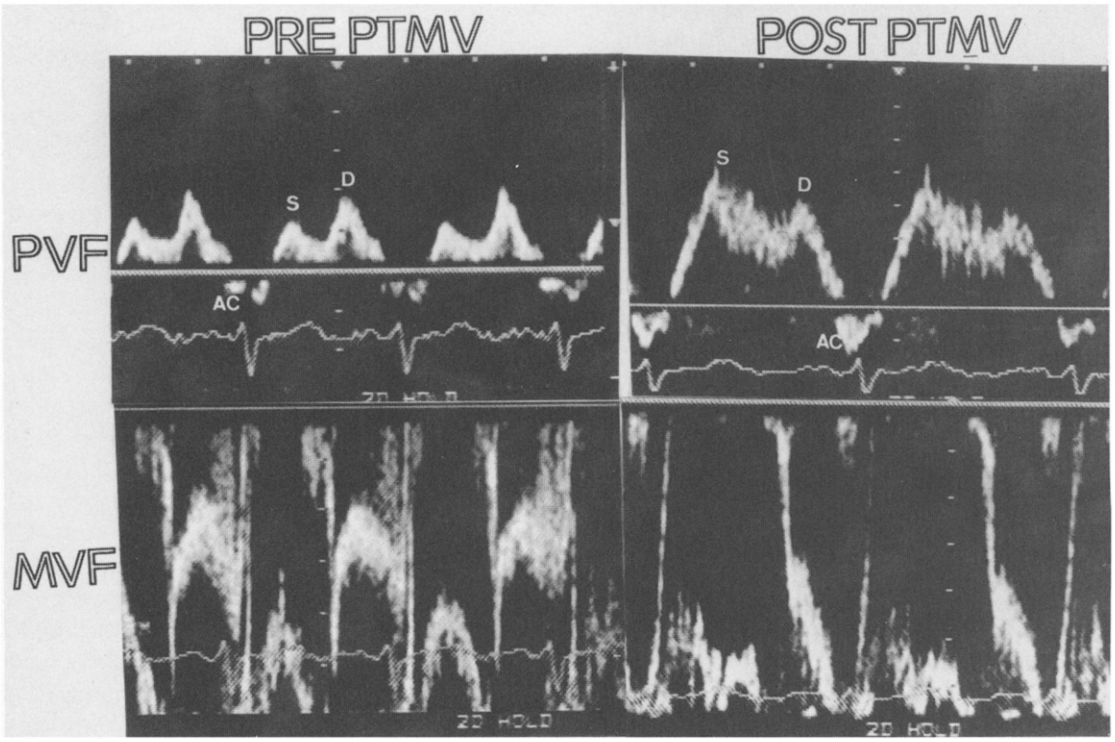

Figure 4 Pulmonary venous flow *(PVF)* and mitral valve flow *(MVF)* in same Case 5, before *(PRE PTMV)* and after *(POST PTMV)* mitral valvuloplasty. *S,* First peak of pulmonary venous flow; *D,* second peak of pulmonary venous flow; *PTMV,* percutaneous transluminal mitral valvuloplasty; *AC,* reverse flow of atrial contraction.

similar to those in Group 2. The above data suggest that atrial contraction and relaxation are important determinants of the systolic phase of pulmonary venous flow.

In Group 4 (lone atrial fibrillation without mitral stenosis) patients, the mean peak flow velocity of the diastolic phase of pulmonary venous flow, the deceleration times of mitral and pulmonary diastolic

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Table 2 Comparison of hemodynamic data and pulmonary venous flow change before and after percutaneous transluminal mitral valvuloplasty in six patients

					Pulmonary venous flow (cm/sec)			
	Amplitude of IAS (cm)		LA diameter (cm)		S		D	
No.	Before	After	Before	After	Before	After	Before	After
1	0.2	0.7	7.0	6.2	8.1	36.2	36.1	44.2
2	0.2	0.7	7.9	7.5	10.2	20.3	30.1	36.4
3	0.5	0.7	4.9	4.1	24.3	34.2	20.1	34.2
4	0.3	0.6	4.4	4.2	30.3	70.2	40.1	60.2
5	0.2	0.5	4.8	4.8	16.3	36.2	36.3	40.4
6	0.2	0.4	7.6	7.5	16.1	20.2	30.1	56.2
Median	0.28 ± 0.13	0.6 ± 0.13	6.1 ± 1.57	5.72 ± 1.57	18.55 ± 10.37	37.88 ± 22.50	32.13 ± 7.06	45.27 ± 10.66
p	<0.05		<0.05		<0.05		<0.05	

IAS, Interatrial septum; *S*, peak of pulmonary venous flow in ventricular systole; *D*, peak of pulmonary venous flow in ventricular diastole; *DDT*, deceleration time of pulmonary diastolic flow velocity; *E*, peak early filling velocity; *EDT*, deceleration time of early filling velocity; statistics with Wilcoxin matched-pair signed-rank test.

forward flow, and the pulmonary wedge pressure were similar and not significantly different from those in Group 1. However, atrial septal motion was decreased, and the reversal flow of atrial contraction was not detectable. Conversely, the peak of ventricular flow velocity was greater than that of Group 3. These findings suggest that left atrial pressure is an important factor influencing the systolic phase of pulmonary venous flow.

Six patients with mitral stenosis (four in Group 2 and two in Group 3) underwent percutaneous transluminal mitral valvuloplasty. The data of hemodynamics, pulmonary venous flow, and mitral flow before and after operation are lited in Table 2. After dilatation, the mitral valvular area was significantly increased, and the pressure gradient between the left atrium and left ventricle at end diastole significantly decreased. The atrial septal motion was improved, and both peak of systolic and diastolic phases of pulmonary venous flow significantly increased (Figures 3 and 4). The deceleration times of the mitral and pulmonary diastolic flows were proportionately and significantly decreased.

DISCUSSION

Transesophageal echocardiography with pulsed Doppler and color flow imaging capabilities provides an excellent means to evaluate pulmonary venous flow velocities because of the transducer location immediately posterior to the left atrium. From this position, the pulmonary veins are usually clearly located in the near field within 2 to 4 cm of the transducer, which enables the analysis of the characteristics of pulmonary venous flow.[5]

Transesophageal Pulmonary Venous Flow Pattern

The pulsatile nature of pulmonary venous flow has been established in previous studies.[6-10] As we have also seen in normal subjects, pulmonary venous flow consists of atrial retrograde flow as well as systolic and diastolic forward flow of ventricular phases. Reversed flow during atrial contraction occurs before the QRS complex and corresponds to the maximal contraction of the atrial septum. The systolic ventricular flow occurs during ventricular systole shortly after the QRS complex, which corresponds to the beginning of atrial relaxation. The peak velocity of this phase corresponds to rapid atrial relaxation. The diastolic ventricular phase of flow during ventricular diastole occurs consistently after the rapid filling wave of the mitral flow E wave. This phase has its maximal flow velocity at the beginning of atrial contraction and ends before the end of the cardiac cycle.

Thus blood flow from the pulmonary circulation into the left ventricle involves pulmonary venous flow and left atrial contraction and relaxation as well as flow across the mitral valve. The pattern of pulmonary venous flow is determined mainly by left atrial and mitral valve motion. In patients with impaired atrial activity (Groups 3 and 4) or increased left atrial pressure (Groups 2 and 3), we found that the atrial retrograde flow and the systolic flow of ventricular phase were decreased or absent. This indicates that

Table 2 (Cont'd)

Mitral area (cm^2)		DDT (msec)		Mitral flow: E (cm/sec)		Mitral flow: EDT (msec)		LA->LV Pressure gradient (mm Hg)	
Before	After	Before	After	Before	After	Before	After	Before	After
0.6	1.72	226	184	260	165	520	342	19	6
0.7	1.10	314	200	180	135	493	238	15	7
0.9	2.04	318	243	200	164	400	220	17	2
1.3	1.47	160	133	186	134	213	160	26	4
1.12	2.46	360	286	199	100	566	310	18	2
0.9	1.47	213	164	248	147	426	280	15	4
0.92 ± 0.26	1.71 ± 0.48	285.1 ± 18	198 ± 21	213 ± 24	140 ± 13	545 ± 15	268.3 ± 20	18.33 ± 4.08	4.17 ± 2.04
<0.05		<0.01		<0.01		<0.01		<0.05	

both of these pulmonary flows were strongly related to the left atrial motion and left atrial pressure. Nishimura et al.[3] have shown that the degree of flow of reversal during atrial contraction correlated strongly with the mean pulmonary capillary pressure in patients undergoing cardiac surgery. They further demonstrated that during preload reduction, there was a decrease in the height and duration of the atrial reversal in the pulmonary vein. Conversely, increase in preload by fluid administration resulted in a significant increase in pulmonary capillary pressure, and the atrial reversal in pulmonary vein became significantly higher.

In this study, there is no significant difference in heart rates between different groups of patients, and we did not find any effects of heart rate on pulmonary venous flow velocity. This is compatible with the results of Kuecherer et al.[11]

Systolic Forward Flow

In this study, we found that the peak of the systolic phase of pulmonary venous flow corresponded to rapid atrial relaxation. Atrial relaxation decreases left atrial pressure causing a "suction effect" to the systolic phase of pulmonary venous flow. In patients with atrial fibrillation (Groups 3 and 4) the suction effect was decreased or even absent, indicating that this suction effect was related to the mean left atrial pressure and size of the left atrium. In Group 4 patients with normal pulmonary wedge pressure, the systolic phase of pulmonary venous flow was larger than in the Group 3 patients who had high pulmonary wedge pressure and left atrial dilatation. In patients with mitral obstruction, the left atrial pressure was decreased after percutaneous transluminal mitral valvuloplasty. These findings were similar to that of Kuecherer et al.,[11] who reported that systolic peak pulmonary venous flow correlated strongly to changing pulmonary wedge pressure with different loading conditions in patients undergoing cardiac surgery. However, this phase with low-flow velocity is still present in patients with high left atrial pressure and left atrial dilatation. We therefore suggest that the suction effect may be one of the factors controlling the systolic phase of pulmonary venous flow. Moreover, the downward movement of the mitral annulus and the propulsive force of the right ventricle may also contribute to this phase of flow.[12] Other factors, such as hyperdynamic left ventricular function (especially in patients with moderate to severe mitral regurgitation), may decrease or reverse the systolic forward flow.[13] In this study, however, we can rule out mitral regurgitation as a factor because patients with mitral regurgitation of greater than mild severity were excluded from the study.

Diastolic Forward Flow

This phase occurs during ventricular diastole. The peak of the pulmonary vein diastolic flow occurs approximately 36.5 ± 13 msec after the rapid filling wave of the mitral valve opening. After mitral valve opening, the left atrial pressure is reduced, and flow from the pulmonary vein passes through the left atrium into the left ventricle because the left atrium acts as an open conduit. In this study, both the peak diastolic velocity and deceleration time of the pulmonary vein were similar to the E velocity and deceleration time of the mitral flow in patients without mitral valve stenosis. Our findings are therefore compatible with that of the study by Nichimera et al.[3] Moreover, in patients with mitral obstruction (Groups 2 and 3), such correlation was not found

and there was still a prolonged deceleration time of peak diastolic venous flow and mitral flow. After percutaneous transluminal mitral valvuloplasty, there was a significant decrease in both phases of time, indicating that the mitral valve motion may influence the pulmonary venous diastolic flow.

CONCLUSION

Our results indicate that the pulsatile nature of pulmonary venous flow is primarily influenced by events occurring on the left side of the heart. The pulmonary venous regurgitant flow velocity and systolic phase of pulmonary venous flow are strongly related to atrial contraction and relaxation and to atrial pressure. The diastolic phase of pulmonary venous flow is affected by mitral valve motion.

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