Effects of Percutaneous Balloon Mitral Valvotomy on Pulmonary Venous Flow in Severe Mitral Stenosis

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Pulsed Doppler Echocardiography has revealed that normal pulmonary venous flow consists of systolic and diastolic phases, which are equal in size and velocity. Systolic flow occurs when the mitral valve is closed, the left atrium is relaxed and the mitral annulus is descending toward the apex. The second component occurs in diastole when the mitral leaflets open and allow blood to enter the left ventricle.[1] The biphasic flow may be disturbed in certain pathologic states. For instance, in dilated cardiomyopathy, mitral annulus motion subject to the degree of systolic dysfunction, may be markedly reduced. There is, consequently, a reduction or absence of the systolic phase of pulmonary venous flow.[1,2] Mitral stenosis due to rheumatic heart disease also results in marked reduction in the mobility of the mitral valve apparatus and leaflets. Moreover, there is considerable fusion of the commissures.[3] Percutaneous balloon mitral valvotomy (PBMV) has rapidly emerged as an alternative to surgical valvotomy in patients with mitral stenosis.[4] Successful PBMV results in rapid reduction of left atrial pressure and transmitral gradient, with significant increase in mitral valve area. There have been no reports demonstrating alteration in pulmonary venous flow with acute reduction in left atrial pressure after PBMV in mitral stenosis.[5] This study was designed to investigate alterations in pulmonary venous flow, using transesophageal echocardiography (TEE) after PBMV in patients with severe mitral stenosis.

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Fifteen patients (6 men and 9 women age range 16 to 32 years, average 24) with severe mitral stenosis were studied. All patients were in New York Heart Association functional class III and had sinus rhythm. No patient with left atrial thrombus, demonstrated by 2-dimensional and TEE, underwent PBMV. All patients had an echo score of ≤8 and none had any degree of mitral regurgitation.

All patients underwent right- and left-sided cardiac catheterization before and after PBMV, which was performed with the Inoue balloon catheter after obtaining informed consent.[6]

Patients had fasted for 4 hours before examination and had given informed consent. TEE was performed 24 hours before PBMV and within 48 hours after the intervention. Dysphagic disease was excluded. No sedation was administered before examination. In all patients 2% lidocaine hydrochloride solution was used for local anesthesia. The patients were asked to gargle with the lidocaine solution until they felt numbness of the tongue. No routine antibiotic prophylaxis was given.

Patients were placed in the supine position, with the head flexed in midline. We used a 5 MHz phased-array ultrasound transducer (model 21362A of Hewlett Packard) for the TEE. The patient was asked to swallow the probe after it was placed on the posterior aspect of the tongue. The probe was advanced about 30 to 35 cm from the incisors, where it was placed behind the left atrium, and standard short-axis and oblique views obtained. The model 2199A TEE adaptor connected the TEE imaging transducer to a SONOS 1000 Hewlett Packard ultra-

TABLE I Clinical, Hemodynamic and Pulmonary Venous Flow Data Before and After Percutaneous Balloon Mitral Valvotomy in 12 Patients

		MVA (cm^2)		MPG (mm Hg)		MR Grade		Heart Rate (beats/min)		SPVF (cm/s)		DPVF (cm/s)	
Pts.	Age (yr) & Sex	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	25F	0.6	1.9	13	4	0	1	78	76	15	54	18	50
2	23F	0.7	1.8	16	2	0	0	100	86	21	48	24	46
3	17M	0.7	1.9	26	4	0	0	74	80	18	64	22	63
4	32F	0.8	2.0	12	2	0	0	72	111	23	49	38	45
5	18M	0.7	2.2	15	3	0	0	116	74	27	48	38	42
6	25F	0.8	2.1	11	0	0	1	90	86	24	68	34	66
7	28F	0.9	1.9	10	0	0	1	78	86	16	72	28	70
8	22M	0.7	1.8	9	1	0	1	74	86	20	60	30	58
9	28F	0.6	1.7	18	3	0	0	88	84	16	64	24	42
10	25F	0.6	2.3	24	4	0	0	90	96	25	46	38	42
11	21F	0.8	2.3	16	2	0	0	94	98	23	52	38	46
12	23M	0.7	2.2	22	3	0	0	88	78	15	52	25	38
Mean ± SD	24 ± 4	0.7 ± 0.1	2 ± 0.2*	16 ± 6	23 ± 1*			87 ± 13	86 ± 11	20 ± 4	56 ± 9*	30 ± 7	51 ± 10*

*$p < 0.005$.
DPVF = diastolic pulmonary vein flow; MPG = mean transmitral gradient; MR = mitral regurgitation; MVA = mitral valve area; SPVF = systolic pulmonary vein flow.

sound system. After evaluating standardized scan planes, the best color flow signals from the left upper pulmonary vein was obtained. The Pulse Doppler volume sample was positioned to obtain the best spectral display of the systolic and diastolic waveforms of pulmonary venous flow. In all patients the color flow Doppler gain/reject was adjusted just to the level where background noise was seen.

Conventional echocardiography and Doppler examinations were performed routinely before TEE procedure using a 3.5 MHz phased-array transducer.

Data were expressed as mean ± SD. Statistical comparisons were obtained by paired student's t *test. A probability value <0.05 was considered statistically significant.*

The mitral valve area, evaluated by 2-dimensional echocardiography increased from 0.7 ± 0.1 to 2.0 ± 0.2 cm² after PBMV in all 12 patients who had repeat TEE (p <0.005). Two patients refused assessment by TEE after PBMV, and 1 patient was excluded because she developed moderately severe mitral regurgitation. Mild

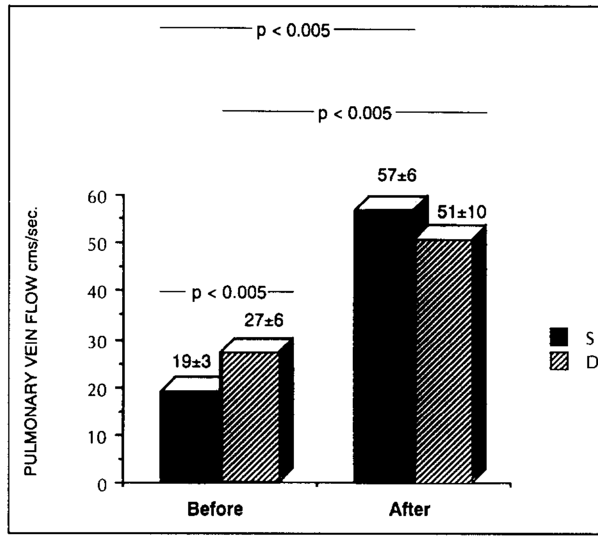

FIGURE 1. Alterations in systolic (S) and diastolic (D) pulmonary venous flow after percutaneous balloon mitral valvotomy. Values are expressed as mean ± standard deviation.

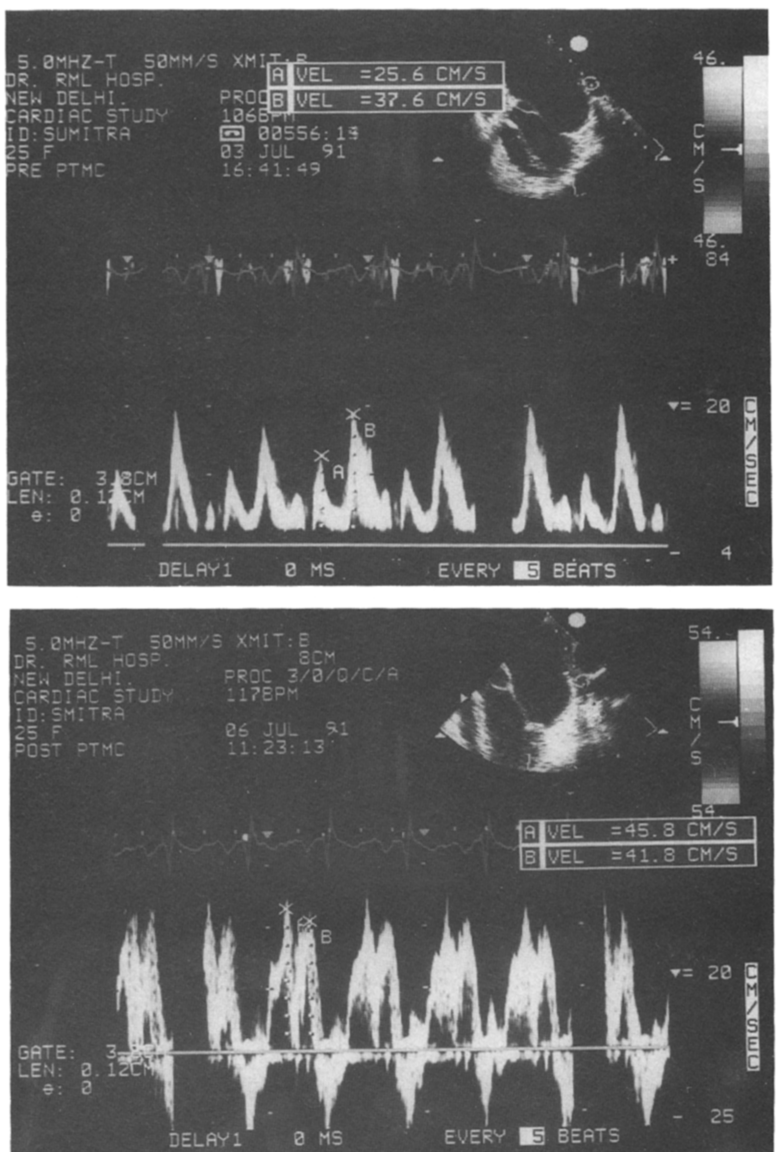

FIGURE 2. ***Top,*** **transesophageal Doppler echocardiogram showing pulmonary venous flow that is reduced in systole (A) before percutaneous balloon mitral valvotomy.** ***Bottom,*** **transesophageal Doppler echocardiogram of pulmonary venous flow after percutaneous balloon mitral valvotomy. Both waveforms are greater than those before percutaneous balloon mitral valvotomy** ***(top),*** **and the systolic wave (A) is slightly greater than the diastolic flow (B).**

mitral regurgitation was produced in 4 of 12 patients. Mean transmitral gradient decreased from 16 ± 5.6 mm Hg to 2.3 ± 1.5 mm Hg ($p < 0.005$) (Table I). There was no significant change in heart rate before or after PBMV.

All 12 patients were in sinus rhythm, and all exhibited reduced flow in systole compared with diastole before PBMV. The systolic velocity was measured at 19 ± 3.2 cm/s and diastolic flow at 27 ± 6 cm/s. After PBMV systolic flow was greater than diastolic flow, and measured 57 ± 8 compared with a diastolic flow of 51 ± 10 cm/s. There was significant increase in both phases of pulmonary venous flow subsequent to PBMV ($p < 0.005$), but after PBMV the systolic flow was not significantly greater than the diastolic flow (Figures 1 and 2). There were no complications during PBMV and TEE.

This study demonstrates that systolic pulmonary venous flow is markedly less than diastolic flow in patients with severe mitral stenosis. After PBMV there was significant increase in the velocity of the systolic and diastolic waves of pulmonary venous flow into the left atrium. There was significant increase in mitral valve area accompanied by substantial reduction in the mean transmitral pressure gradient. The improved hemodynamics consequent to PBMV were accompanied by the restoration of the systolic pulmonary vein flow to its usual size in relation to the diastolic flow. It was now slightly greater than diastolic flow.

The present study strongly suggests that the mitral valve apparatus greatly influences pulmonary venous flow. There is increase in systolic flow with the mechanical splitting of mitral commissures after PBMV. All our patients were young with minimal subvalvular pathology or calcification. The main obstacle to left atrial blood flow was contributed by the thickened, fused but pliant leaflets of the stenosed mitral valve. The critical changes in systolic flow of pulmonary veins imply that stenosed mitral leaflets also impair the descent of the atrioventricular ring during systole. It is quite likely that as the mitral leaflets are in continuity with the annulus, restricted mobility of the leaflets during diastole also compromise systolic motion of the atrioventricular ring.

The other mechanism, to explain the increase in systolic flow after PBMV, could be the substantial decrease in left atrial pressure. With the reduction of mean left atrial pressure to near-normal values, both systolic and diastolic flow of the pulmonary veins accelerate. With the sudden removal of obstruction by PBMV, net flow into the left atrium and then into the left ventricle is bound to increase.

Pulsed Doppler echocardiography of pulmonary veins obtained at their entry into the left atrium by conventional transthoracic 4-chamber apical window displays biphasic pulmonary vein flow in normal hearts. However, there often is difficulty in getting adequate color doppler flow signals in patients with dilated ventricles and in patients with severe mitral stenosis because of rotation of the heart plus chest wall deformity. We therefore used TEE to evaluate pulmonary venous flow. Besides obtaining excellent color Doppler imaging of pulmonary vein flow, TEE is extremely sensitive in excluding left atrial appendage clots before PBMV,[7] and documenting the creation of atrial septal defects consequent to PBMV.[8] The advantages of TEE in image quality and improved sensitivity of color flow Doppler imaging are particularly useful in evaluating patients with mitral stenosis before and after PBMV.

Before PBMV, color flow from the left pulmonary vein is a continuous red flame. After PBMV, not only does the flame become enlarged, but it also gets a tinge of green blue, suggesting increased velocity.

This study emphasizes the feasibility of using TEE in evaluating the alterations of pulmonary venous flow after PBMV. Before PBMV, systolic flow is less than diastolic flow. Subsequent to PBMV there is significant reduction in left atrial pressure and transmitral gradient, inducing substantial increase in both systolic and diastolic flow, with the systolic wave greater than diastolic wave.

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