Pulmonary venous flow pattern — its relationship to cardiac dynamics

A pulsed Doppler echocardiographic study

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ABSTRACT We studied the physiology of pulmonary venous flow in 13 normal subjects and five patients with atrial rhythm disorders and atrioventricular conduction disturbances with pulsed Doppler and two-dimensional echocardiography. The left atrium, mitral valve, and pulmonary venous ostia were visualized through the apical four-chamber view. Mitral and pulmonary venous flows were obtained by placing the Doppler sample volume at the appropriate orifice. Pulmonary venous flow was biphasic: a rapid filling wave was observed during systole when the mitral valve was closed; a second wave was observed in diastole during the rapid ventricular filling phase of mitral flow, but was significantly delayed. In patients without atrial contraction (atrial fibrillation and sinoatrial standstill), the initial rapid filling was greatly diminished and only the second diastolic wave appeared to contribute to left atrial filling. In patients with high-grade atrioventricular block, each atrial contraction was followed by a surge in flow from the pulmonary veins. These results are consistent with data obtained from invasive measurements in both dogs and man, and confirm the validity of the use of pulsed Doppler echocardiography in the study of pulmonary venous flow. We suggest that pulmonary venous flow is influenced by dynamic changes in left atrial pressure created by contraction and relaxation of the atrium and ventricle. The initial peak in pulmonary venous flow occurs with atrial relaxation simultaneously with the reduction of left atrial pressure, and the second peak occurs with left ventricular relaxation and rapid transmural filling of the ventricle.


THE PATTERN OF FLOW in the large extraparenchymal pulmonary veins is pulsatile in both dog and man.1-11 The pulsatile nature of pulmonary venous flow has been suggested by several investigators to result from changes in left atrial pressure occurring throughout the cardiac cycle.1-6 Others have attributed pulmonary flow to forward transmission of pressure pulses from the right ventricle through the pulmonary circulation7-10 or to a combination of the propulsive force of the right ventricle and suction created by the left heart.8-11 Simultaneous invasive measurements of pulmonary vein flow and left atrial pressure in a canine preparation and in patients with aortic stenosis revealed that pulmonary venous flow velocity was maximal during the troughs and minimal during the peaks of left atrial pressure.1

Using pulsed Doppler echocardiography, we studied the pattern of the pulmonary venous flow in normal subjects and in patients with atrial fibrillation, atrial standstill, and high-grade atrioventricular block to attempt to elucidate the relationship between pulmonary venous flow, atrial systole, and other events occurring during the cardiac cycle.

Materials and methods

Subjects. Eighteen individuals were recruited to participate in the study and data from all are reported. Eleven subjects were normal student volunteers; one was a normal baby and another a normal octogenarian. Two patients had "lone" atrial fibrillation, one patient had isolated sinoatrial standstill, one patient had 2:1 atrioventricular block, and another patient had complete atrioventricular block. The latter patients had primary conduction disorders and presented for pacemaker implantation. Only patients clinically free of structural heart disease were selected for study.

All patients underwent thorough clinical evaluation before the study; relevant findings are given in table 1. M mode and two-dimensional echocardiography as well as pulsed Doppler cardiographic studies of the mitral, pulmonary venous, and pulmonary arterial flows were performed.

Echocardiographic studies. An Electronics for Medicine/
Honeywell ultramager was used for both imaging and Doppler echocardiographic flow studies. The instrument has a mechanical transducer that oscillates through an angle of 30 to 75 degrees. A movable Doppler cursor allows sampling along a line within the image when the oscillating transducer system is stopped and set into the Doppler mode.

**M mode echocardiography.** M mode echocardiograms were spatially oriented from the two-dimensional image, preferably from the short-axis view. The transducer was held perpendicular to the chest wall in the third or fourth left intercostal space near the left sternal border, with the patient in the left lateral decubitus position. Special attention was directed to the display of mitral valve opening, leaflet motion, and coaptation. The transducer was then rotated inferolaterally to obtain a two-dimensional short-axis view of the heart at the level of the chordae tendineae. Care was taken to ensure proper transducer angulation so that the left ventricle had a circular rather than an oval or elliptical appearance. M mode echocardiograms of the interventricular septum and of the posterior wall were recorded. M mode echocardiograms of the aorta, aortic valve, and left atrium were obtained from the short-axis view at the level of the great vessels by orienting the transducer medially and craniad.

**Two-dimensional echocardiography.** Two-dimensional echocardiograms were recorded with the use of mechanical 2.25 and 3.5 MHz transducers. The study included several cross-sectional views of the heart. The long-axis view was obtained by orienting the sector plane parallel to the longitudinal axis of the left ventricle. Short-axis views were obtained by orienting the sector plane perpendicular to the long axis. The apical four-chamber view that best demonstrated the left atrium and pulmonary veins was obtained with the transducer placed at the cardiac apex and the tomographic plane directed perpendicular to the ventricular and atrial septa and through the plane of the mitral and tricuspid valves. Occasionally, to define the orifices of the pulmonary veins, the transducer had to be rotated slightly from this position.

**Pulsed Doppler cardiological studies.** The depth of the sample volume in this instrument is variable to a maximum of 16 cm from the transducer and the sample volume length is adjustable from 2 mm to 2 cm. The Doppler shift is detected in the region specified within the sample volume. Sampling position is automatically updated after every fifth cardiac cycle to permit maintenance of the sampling volume in the desired position, and also by switching the instrument from the Doppler to real-time imaging mode. In addition to the audio output, the frequency shifts are processed through a fast-Fourier transform spectral analyzer and expressed graphically as flow velocity (V) by solving the Doppler formula V = fVm/200 cosO, where Vm = the speed of the sound in the medium (1540 cm/sec); f0 = emitting frequency of the transducer; O = the angle of incidence between the sound waves and the flow. When solving the Doppler equation, the instrument assumes that O = 0 (cosO = 1). To obtain pulmonary venous flow, the sample volume was placed at the orifice of the pulmonary vein. In most instances, we were confined to recording flow from one pulmonary vein, since other veins, although sometimes well visualized, were at an angle relatively perpendicular to the sampling direction. The best vein for registering flow was usually the right upper paraseptal vein, although sometimes other veins were used (figure 1). The angle of the Doppler sampling direction of flow (f) was estimated by an angle cursor on the sample line and the view was adjusted so that the angle was less than 20 degrees.

Although the direction of flow in the plane perpendicular to the image, the azimuthal plane, could not be determined, we attempted to maximize the Doppler shift by small positional

### Table 1

**Clinical and echocardiographic characteristics of study patients**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>LVID (mm)</th>
<th>IVS (mm)</th>
<th>LVPW (mm)</th>
<th>AO (mm)</th>
<th>LA (mm)</th>
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<tr>
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<td>SA</td>
<td>45</td>
<td>11</td>
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<td>50</td>
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</tbody>
</table>

Echocardiographic measurements are from M mode study.

N = normal; AF = atrial fibrillation; LVID = left ventricular internal diameter; IVS = interventricular septum; LVPW = left ventricular posterior wall; AO = aorta; LA = left atrium; SA = sinoatrial; AV = atrioventricular.
changes in order to be as closely parallel to the flow as possible. Sample volume was usually set at 0.5 to 1 cm, depending on the size of the pulmonary vein orifice. The M mode, two-dimensional, and Doppler echocardiographic studies were recorded with a black and white hard-copy recorder.

**Analysis of data.** The time relationship between phases of mitral flow, pulmonary venous flow, and M mode echocardiographic correlates of mitral valve motion were analyzed in all patients. Pulmonary arterial flow was studied in 11 patients and was analyzed separately. All time measurements were related to the beginning of the QRS complex on the electrocardiogram. 

**Analysis of mitral flow (figure 2).** From each record 5 beats were chosen for analysis. The following parameters were measured for each beat: (1) Time from the beginning of the QRS complex of the electrocardiogram to the beginning of mitral flow (Q→D), to the peak of the rapid filling wave (Q→E), to the peak of atrial contribution (Q→A), and to the end of mitral flow (Q→C). (2) Peak mitral flow velocity at the rapid filling wave (E) and atrial contribution (A) wave.

**Analysis of pulmonary venous flow (figure 2).** The following measurements were made: (1) Time from beginning of the QRS complex to the beginning of pulmonary venous flow (Q→BJ), to the peak of the first phase of pulmonary venous flow (Q→J), to the peak of the second phase (Q→K), and to the end of pulmonary venous flow (Q→EK). (2) Peak velocity of the first (J) and second (K) phases of pulmonary venous flow. (3) Analysis of the area under the curve of both filling phases with an ultrasonic digitizer (Science Accessories Corporation) coupled to a PDP 11/24 digital computer and calculation of the ratio between the phases.

**Analysis of mitral valve echocardiograms.** The mitral valve echocardiogram was assessed with respect to the time intervals and the extent of leaflet separation with the use of the following measurements: (1) Time from onset of the QRS complex to opening of the mitral valve (Q→D), to the peak opening of the valve at the rapid filling wave (Q→E), to the peak opening of the valve at atrial contraction (Q→A), and to closure of the mitral valve (Q→C). (2) Maximal separation of the mitral leaflets at the E and A points. (3) In patients in whom an intermediate motion of mitral valve leaflets in diastole between the E and A waves was clearly observed, the time from onset of QRS to peak of the intermediate motion.

It should be emphasized that the present study is qualitative in nature. To provide quantitative data, a correlation with pressure measurements in the pulmonary veins, pulmonary artery, and left atrium is required. Quantification of Doppler echocardiographic data requires accurate estimation of pulmonary vein size and confirmation by invasive measurements that the velocities measured by Doppler cardiography in the assumed direction of

**FIGURE 1.** Four-chamber view of the heart showing the sampling volume of the Doppler beam placed at the orifice of the right upper pulmonary vein (RUPV, A) and left upper pulmonary vein (LUPV, B). LV = left ventricle; LA = left atrium. Small positional changes enable orientation of the sampling volume almost parallel to the flow.

**FIGURE 2.** Schematic illustration of the pulmonary venous flow (PVF), mitral valve flow (MVF), and pulmonary arterial flow (PAF) and their relative timing in the cardiac cycle. D = onset of mitral flow; C = cessation of mitral flow; Js = area under the curve of the first phase; Ks = area under the curve for the second phase of PVF. For accurate explanation of each interval, see text.
flow are truly peak velocities. The setting of the present study precluded acquisition of such data. The results are expressed as the mean ± SD. Statistical analyses were performed with Student’s t test.

Results

Normal subjects (table 2). Figure 3 illustrates the characteristics of flow through the pulmonary veins in normal subjects as it is related to the mitral valve echocardiogram and flow through the mitral valve. The tracings were not registered simultaneously but taken a few minutes apart and synchronized at the same heart rate with the electrocardiogram as a marker. In normal subjects, pulmonary venous flow has two phases (figures 2 and 3). The first or J phase occurs during ventricular systole, shortly after the QRS (77 ± 28 msec; table 2). In our normal subjects this phase of flow reached a peak at 229 ± 57 msec, about 180 msec after mitral valve closure (Q→J). The second or K phase, corresponding to ventricular diastole, reached its maximum (Q→K) 558 ± 72 msec after the QRS complex. The second phase ended before the end of the cardiac cycle (703 ± 136 msec). This pattern of flow was consistent in all 13 normal subjects. In subject 10 a very small and continuous low second phase of flow was observed and a peak could not be demonstrated. The mean peak flow velocity of the first phase (J) was 44.5 ± 10.3 cm/sec and that of the second phase was 53 ± 15 cm/sec.

The second phase of flow (K), during ventricular diastole, occurs consistently after the rapid filling wave of the mitral flow (E wave). In our normal subjects mitral flow started 397 ± 43 msec after the QRS complex, and the peak of the rapid filling wave was measured at 496 ± 56 msec. The maximum velocity of mitral flow occurred significantly earlier (p < .001) than the peak of the K phase of the pulmonary venous flow. The atrial contribution to ventricular filling (A wave) peaked at 836 ± 215 msec, long after the end of the pulmonary venous flow (135 msec). The cessation of mitral flow was observed at 875 ± 270 msec, corresponding to the final closure of the mitral valve. After a short delay, the next J wave of pulmonary venous flow began. It should be noted that during atrial contraction, the flow from the pulmonary veins into the left atrium was either very slow or did not occur at all. No regurgitant flow from the atrium into the large extraparenchymal pulmonary veins was observed during left atrial contraction. Peak flow velocity of the rapid filling wave (E) was 79.1 ± 21.3 cm/sec and that of the A wave was 45.5 ± 16.4 cm/sec.

The opening of the mitral valve on the M mode echocardiogram occurred 388 ± 40 msec after the QRS in our subjects and corresponded to the beginning of transmitral flow. However, maximal opening of the mitral valve (E point) on the M mode echocardiogram occurred about 30 msec before peak mitral flow (E

TABLE 2
Pulmonary venous and mitral valve flow and echocardiographic measurements in normal subjects

<table>
<thead>
<tr>
<th>Interval</th>
<th>RR (msec)</th>
<th>Q→BJ (msec)</th>
<th>Q→J (msec)</th>
<th>Q→K (msec)</th>
<th>Q→KE (msec)</th>
<th>J (cm/sec)</th>
<th>K (cm/sec)</th>
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<tr>
<td>PVF 44.5</td>
<td>871</td>
<td>77</td>
<td>229</td>
<td>558</td>
<td>703</td>
<td>44.5</td>
<td>53</td>
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<tr>
<td>± SD</td>
<td>229</td>
<td>28</td>
<td>57</td>
<td>72</td>
<td>136</td>
<td>10.3</td>
<td>15</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Interval</th>
<th>RR (msec)</th>
<th>Q→D (msec)</th>
<th>Q→E (msec)</th>
<th>Q→A (msec)</th>
<th>Q→C (msec)</th>
<th>E (cm/sec)</th>
<th>A (cm/sec)</th>
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<tbody>
<tr>
<td>MVF</td>
<td>867</td>
<td>397</td>
<td>496</td>
<td>836</td>
<td>875</td>
<td>79.1</td>
<td>45.5</td>
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<td>± SD</td>
<td>220</td>
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<td>56</td>
<td>215</td>
<td>270</td>
<td>21.3</td>
<td>16.4</td>
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<tr>
<td>MVE</td>
<td>866</td>
<td>388</td>
<td>465</td>
<td>824</td>
<td>907</td>
<td>26.8</td>
<td>21.4</td>
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<tr>
<td>± SD</td>
<td>212</td>
<td>40</td>
<td>42</td>
<td>206</td>
<td>214</td>
<td>7.3</td>
<td>4.6</td>
</tr>
</tbody>
</table>

For explanation of different intervals (Q→BJ, Q→J, Q→K, Q→KE, RR, Q→D, Q→E, Q→A, Q→C) see figure 2 and text.

Comparison between RR intervals indicated no significant differences.

PVF = pulmonary venous flow; MVF = mitral valve flow; MVE = mitral valve echocardiogram.

+p < .001 compared with Q→E (nonpaired t test).

*Measured in cm/sec for MVF and mm for MVE.
point of the Doppler echocardiogram). This difference was significant (p < .01). Maximal opening of the mitral valve during atrial contraction occurred at 824 ± 206 msec, about 12 msec before the occurrence of peak flow.

The area under the curve for the J phase and the K phase were digitized separately for each beat and the ratio between the flow pulses was calculated (figure 2). The mean (± SD) J/K ratio for the normal subjects was 1.19 ± 0.53, demonstrating that both phases are important determinants of left heart filling. In many people, and mainly during bradycardia (prolonged RR intervals), a third phase of motion can be demonstrated between the two conventional E and A waves. Individuals with longer RR intervals (subjects 2, 3, 5, 8, and 11) in many beats demonstrated three phases of mitral valve motion. The intermediate phase always occurred after the K wave of pulmonary venous flow. The mean delay in 25 beats from five patients was 127 ± 22 msec.

**Pulmonary venous flow in patients without atrial contraction.** The data suggest that atrial contraction and relaxation are important determinants of the J phase of pulmonary venous flow (table 3, figure 4). To verify this conclusion we studied three patients without effective atrial contraction; two patients had atrial fibrillation (Nos. 14 and 15) and one had sinoatrial standstill with an atrioventricular nodal escape rhythm (No. 16). These patients were compared with two others, the first with 2:1 atrioventricular block and the second with complete atrioventricular block (patients 17 and 18). In patients without atrial contraction (Nos. 14, 15

**TABLE 3**

Pulmonary venous flow in patients without atrial kick (atrial fibrillation and sinoatrial standstill; mean ± SD)

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>RR (msec)</th>
<th>Q→BJ (msec)</th>
<th>Q→J (msec)</th>
<th>Q→BK (msec)</th>
<th>Q→K (msec)</th>
<th>Q→EK (msec)</th>
<th>J (cm/sec)</th>
<th>K (cm/sec)</th>
<th>RR (msec)</th>
<th>Q→D (msec)</th>
<th>Q→E (msec)</th>
<th>Q→A (msec)</th>
<th>Q→C (msec)</th>
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<tr>
<td>14</td>
<td>746 ± 83</td>
<td>80 ± 7</td>
<td>216 ± 11</td>
<td>435 ± 13</td>
<td>574 ± 27</td>
<td>802 ± 31</td>
<td>13 ± 2</td>
<td>43 ± 3</td>
<td>722 ± 55</td>
<td>402 ± 4</td>
<td>498 ± 10</td>
<td>—</td>
<td>800 ± 48</td>
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<tr>
<td>15</td>
<td>634 ± 38</td>
<td>62 ± 3</td>
<td>275 ± 20</td>
<td>448 ± 35</td>
<td>546 ± 38</td>
<td>746 ± 47</td>
<td>7 ± 0</td>
<td>54 ± 4</td>
<td>632 ± 39</td>
<td>416 ± 5</td>
<td>464 ± 5</td>
<td>—</td>
<td>690 ± 34</td>
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<tr>
<td>16</td>
<td>1390 ± 25</td>
<td>104 ± 9</td>
<td>346 ± 5</td>
<td>540 ± 0</td>
<td>692 ± 8</td>
<td>1250 ± 56</td>
<td>7.3 ± 1</td>
<td>46 ± 4</td>
<td>1382 ± 5</td>
<td>439 ± 7</td>
<td>543 ± 4</td>
<td>—</td>
<td>1451 ± 5</td>
<td>52 ± 1</td>
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</table>

PVF = pulmonary venous flow; MVF = mitral valve flow; Q→BK = interval from onset of QRS to beginning of K wave; for explanation of other intervals see text and figure 2.

*p < .01 (Q→K vs Q→E by t test).
and 16) there was only one major phase of flow through the pulmonary veins into the left atrium (K phase). During ventricular systole, the first flow phase (J) was small. After opening of the mitral valve, the K phase of flow from the pulmonary vein into the left atrium was readily recorded. The peak of the K phase (the only significant phase of flow in these patients) occurred in all three patients after the peak of the mitral E wave.

**Pulmonary flow in patients with high-grade atrioventricular block (figure 5).** In patients with high-grade atrioventricular block (Nos. 17 and 18), significant surges of pulmonary venous flow followed each atrial contraction, including contractions caused by blocked P waves (J2 in figure 5). Pulmonary venous flow varied with changes in the temporal relationship between atrial and ventricular systole. When ventricular systole occurred within 200 msec after atrial contraction, augmented pulmonary venous flow was observed (J1 in figure 5). In patient 17 (2:1 atrioventricular block), conducted P waves were associated with a large normal J phase of flow during ventricular systole.

**Discussion**

The pulsatile nature of pulmonary venous flow has been established in previous studies and confirmed by the present observations. Two mechanisms have been suggested for the control of pulmonary venous flow. One group of investigators holds that pulmonary venous flow should be attributed to forward transmission of pressure from the right ventricle through the pulmonary vasculature. A second group suggests that pulmonary flow is determined by a "suctioning effect" created by the left atrium and left ventricle throughout the cardiac cycle.

Rajagopalan et al. observed, in a canine preparation, that flow in the large extraparenchymal pulmonary veins is dominated by left atrial events. Thus, when the left atrial pressure was high, flow through the pulmonary veins was reduced, and when the pressure was low, flow into the left atrium increased. Dixon et al. and Morgan et al. argue that the mechanical events in the left heart define the phasic characteristics of pulmonary venous flow and that the suction effect on blood by the heart, in their view, is more important in determining venous flow pattern than the influence of the transmitted forward pulse.

Our data support the views of Rajagopalan and others and suggest that the pattern of flow from the pulmonary veins is determined mainly by events occurring on the left side of the heart. Each of the pulmonary venous flow pulses, the first of which occurs during ventricular systole and the second of which occurs during diastole, contributed significantly to left ventricular filling. This was documented by digitizing the area under the curve for each flow pulse. Since our
study was noninvasive, we could not relate our findings to pressure events in the left atrium and pulmonary artery. However, since left ventricular filling is adequately reflected by mitral flow, it is reasonable to relate flow through the mitral valve to the pulses of flow observed in the pulmonary veins. The J phase occurs while the mitral valve is closed and is a reflection of the reduced pressure in the left atrium that results from relaxation of the atrium at the end of diastole. Moreover, during the early phase of ventricular systole, the mitral valve descends slightly as a result of the contraction and shortening of the long diameter of the left ventricle. This movement contributes to the reduction in left atrial pressure that promotes flow from the pulmonary veins into the left atrium. After the rapid filling wave across the mitral valve, the pressure in the left atrium is reduced and flow from the pulmonary veins into the left atrium resumes in a second phase. This K phase occurs during ventricular diastole, reaches a peak after the rapid filling wave, and diminishes before the atrial contribution to mitral flow. During this phase, the atrium acts as an open conduit through which blood flows directly from the pulmonary veins through the mitral valve into the left ventricle.

Experimental support for this hypothesis was provided by Morkin et al., who demonstrated that pulmonary venous flow and opening of the mitral valve are synchronized such that on cineangiography, contrast material is literally injected from the pulmonary veins into the left ventricle. In individuals with bradycardia, this K phase of pulmonary venous flow may explain the intermediate motion of the mitral valve as observed on the M mode echocardiogram. The subsequent atrial contraction results in a rise in left atrial pressure, which prevents further flow from the large pulmonary veins into the left atrium. Thus, the left atrium and ventricle appear to be actively involved in their own filling from the pulmonary venous circulation. It is reasonable to suggest that this active filling might involve some form of suction.

If pulmonary venous flow were the result of transmission of a right ventricular systolic pressure pulse, then there should have been only one wave of pulmonary venous flow occurring at a specific time after right ventricular systole. However, the flow in all of our normal subjects was double peaked and each phase occurred after the reduction in left atrial pressure associated with mitral flow.

A study of patients in whom atrial and ventricular
phenomena are not synchronized constitutes a setting in which the relative contributions of atrial and ventricular events to pulmonary venous flow can be assessed. If right ventricular events were the main determinants of pulmonary venous flow, these patients should have had a grossly normal pulmonary venous flow pattern. This was not the case. In patients without atrial contraction (atrial fibrillation and sinoatrial standstill) the J phase, which corresponds in time to ventricular systole, was of minimal magnitude whereas the K phase, which followed mitral rapid filling, was recorded as a significant pulse of flow. Moreover, the phenomena observed in patients with high-grade atrioventricular block support the view that left-sided forces are the main determinants of pulmonary venous flow. Had right ventricular systole been the dominant factor in pulmonary venous flow, no flow would have been expected following a blocked P wave. However, significant flow was observed after atrial contraction both after conducted and nonconducted P waves (figure 5). Thus, it appears that the reduction in left atrial pressure is responsible for the J phase of the pulmonary venous pulse, while left ventricular relaxation and rapid emptying of the left atrium are responsible for the second or K wave.

Rajagopalan et al. suggested that since the pulmonary veins are thin walled and collapsible, they function as compliant structures, and because of their large reservoir capacity, the left ventricular stroke volume can be maintained relatively unaffected by beat-to-beat changes in right ventricular stroke output. The dimensional changes that occur in the large pulmonary veins during each cardiac cycle provide the major component of the overall compliance of the pulmonary vein system. According to Rajagopalan et al., this property enables pressure and flow in the pulmonary capillaries to take place in virtual isolation from pressure changes in the left atrium. Naito et al. could demonstrate retrograde flow from the atrium into the pulmonary veins only when atrial contraction occurred during ventricular systole. In the present study, no retrograde flow was observed from the left atrium into the pulmonary venous system during atrial systole.

In conclusion, our results confirm that the pulsatile nature of pulmonary venous flow is primarily influenced by events occurring on the left side of the heart. Reduction in left atrial pressure, secondary to left atrial relaxation and augmented by the descent of the base of the heart at the beginning of systole, results in the initial flow of blood from the pulmonary veins. This J phase corresponds in time to ventricular systole but is not initiated by it. The K phase of pulmonary venous flow corresponds in time to rapid flow across the mitral valve, and is terminated by the rise in left atrial pressure during atrial systole.

References
Pulmonary venous flow pattern--its relationship to cardiac dynamics. A pulsed Doppler echocardiographic study.
G Keren, J Sherez, R Megidish, B Levitt and S Laniado

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