Relation of Pulmonary Vein to Mitral Flow Velocities by Transesophageal Doppler Echocardiography

Effect of Different Loading Conditions

Rick A. Nishimura, MD, Martin D. Abel, MD, Liv K. Hatle, MD, and A. Jamil Tajik, MD

It has previously been demonstrated that predictable changes occur in mitral flow velocities under different loading conditions. The purpose of this study was to relate changes in pulmonary venous and mitral flow velocities during different loading conditions as assessed by transesophageal echocardiography in the operating room. Nineteen patients had measurements of hemodynamics, that is, mitral and pulmonary vein flow velocities during the control situation, a decrease in preload by administration of nitroglycerin, an increase in preload by administration of fluids, and an increase in afterload by infusion of phenylephrine. There was a direct correlation between the changes in the mitral E velocity and the early peak diastolic velocity in the pulmonary vein curves ($r=0.61$) as well as a direct correlation between the deceleration time of the mitral and pulmonary venous flow velocities in early diastole ($r=0.84$). This indicates that diastolic flow velocity in the pulmonary vein is determined by the same factors that influence the mitral flow velocity curves. A decrease in preload caused a significant reduction in the initial E velocity and prolongation of deceleration time, and an increase in preload caused an increase in E velocity and shortening of deceleration time. An increase in afterload produced a variable effect on the initial E velocity and deceleration time and was dependent on the left ventricular filling pressure. The change in systolic forward flow velocity in the pulmonary vein was directly proportional to the change in cardiac output ($r=0.60$). The pulmonary capillary wedge pressure correlated best with the flow velocity reversal in the pulmonary vein at atrial contraction ($r=0.81$). Use of pulmonary vein velocities in conjunction with mitral flow velocities can help in understanding left ventricular filling. (Circulation 1990;81:1488–1497)

Abnormalities of diastolic function of the heart play an integral role in many disease entities.1,2 Because of the complexity of the multiple, interrelated processes contributing to diastolic function, there is no clinical tool available for its assessment.3 It has been proposed that Doppler echocardiography might provide information concerning diastole because of its ability to noninvasively measure blood flow velocities across the mitral valve.4,5 We, as well as others,6–9 previously demonstrated that predictable changes occur in the mitral flow velocities under different loading conditions. This has resulted in understanding the significance of changes in these velocities and has allowed the development of a conceptual framework for interpretation of the various velocity curves.10

Pulmonary vein velocities have recently been used in conjunction with mitral flow velocities to increase our understanding of ventricular filling.11–13 However, because of the location of the pulmonary vein in the far field from a precordial apical window, it can be difficult to place the sample volume within the pulmonary vein to obtain the true velocities through the pulmonary vein. The advent of transesophageal echocardiography with pulsed Doppler imaging capability has provided a method by which both the mitral valve and pulmonary vein velocities can be easily recorded because of the posterior approach providing unimpeded interrogation of cardiac structures.14

During our initial studies assessing the changes in mitral flow velocities by transesophageal echocardiography during different loading conditions,9,10 predictable changes were also observed in pulmonary veins. Because recording both mitral and pulmonary
vein velocities can improve our understanding of left ventricular filling, the purpose of this study was to systematically compare the changes in pulmonary venous and mitral flow velocities during different loading conditions. To allow safe manipulation of the loading conditions in a controlled, systematic manner, this study was performed in the anesthetized patient before open-heart surgery.

Methods

Patient Population

The patient population consisted of 19 patients undergoing coronary artery bypass graft surgery. There were 16 men and three women with a mean age of 66±9 years. All patients had ejection fractions greater than 40% in the absence of valvular heart disease. The average ejection fraction was 62±10% (mean±SD). Patients were excluded for symptoms of unstable angina or unstable hemodynamics or the presence of significant left main coronary artery disease. This study was approved by the Institutional Review Board of the Mayo Clinic, and informed, written consent was obtained from each patient.

Six patients had a history of mild hypertension. Preoperative medications included β-blockers in 11 patients, nitrates in 14 patients, and calcium channel blockers in 12 patients. Sinus rhythm was present in 17 patients, and atrial fibrillation with controlled ventricular response was present in two patients.

Procedure

After induction of anesthesia and intubation, a Swan-Ganz catheter was placed in the pulmonary artery through the right internal jugular vein. A radial artery catheter was placed for continuous arterial pressure monitoring. A Hewlett-Packard 5-MHz transesophageal echocardiographic probe (Hewlett-Packard Co., Palo Alto, California) was inserted into the esophagus. The transducer was manipulated to obtain a four-chamber view, placing the sample volume at the tip of the mitral valve leaflets to obtain the highest velocity.10,15 The scope was then slightly withdrawn, and the tip was flexed and turned to the left to obtain a clear view of the left upper pulmonary vein as it emptied into the left atrium. A sample volume was then placed 1–2 cm into the pulmonary vein from its junction with the left atrium. Color flow imaging was used to obtain a beam direction as parallel as possible to pulmonary vein flow. The flow velocities were recorded on a strip chart at a recorder speed of 50 mm/sec with simultaneous radial artery pressure and on an electrocardiogram. For convention, the mitral valve velocities were inverted so that the flow across the mitral valve was depicted as a positive velocity.

Baseline measurements that were obtained included mitral flow velocities, pulmonary vein velocities, heart rate, blood pressure, pulmonary capillary wedge pressure, and cardiac output. Cardiac output measurements were made by the thermodilution tech-

ique (i.e., the average of three consecutive injections). All measurements were made before the pericardium was opened and with the ventilator at end-tidal volume to minimize the effect of respiration.

After baseline measurements, intravenous nitroglycerin was administered to decrease systolic blood pressure and wedge pressure by at least 20%. When hemodynamic values were stabilized, repeat Doppler and hemodynamic measurements were obtained. The nitroglycerin was discontinued, and the hemodynamic values were allowed to return to the baseline state. Intravenous phenylephrine was infused to increase the blood pressure by at least 20%, followed by repeat Doppler and hemodynamic measurements. After the hemodynamic values again were allowed to return to baseline after the discontinuation of phenylephrine, 500–1,000 ml crystalloid was rapidly infused to increase the wedge pressure by at least 20% above the baseline. Subsequent repeat hemodynamic and Doppler measurements were made. Single-lead electrocardiographic monitoring was performed throughout the study, and there were no significant ST segment abnormalities.

Measurements

Three consecutive mitral flow velocity curves at each loading condition were analyzed for the following variables (Figure 1).10,15 The E velocity was the highest initial velocity in early diastole, and the A velocity was the velocity at atrial contraction. The mean percentage of interobserver and intraobserver variability for measurement of these velocities is 6.0% and 3.3%, respectively.9 A line was drawn from the peak E velocity along the fall in initial velocity and extrapolated to the baseline. The deceleration time was measured from the peak E velocity to when the extrapolated line intersected the baseline. The mean percentage of interobserver and intraobserver variability for measurement of deceleration time is 8.8% and 6.8%, respectively.9

Three consecutive pulmonary vein velocity curves were analyzed (Figure 1). The height of the retrograde velocity at atrial contraction and the peak forward flow velocity during ventricular systole and diastole were measured. The deceleration time of early diastolic filling was measured in a manner similar to that of the deceleration time for the mitral velocities.

Mitral velocities were obtained in all 19 patients at the four different loading conditions (76 total). Adequate pulmonary vein forward velocities were available for analysis in all 19 patients at the four different loading conditions. The two patients in atrial fibrillation did not have retrograde velocities at atrial contraction. Of the 17 patients in normal sinus rhythm, the retrograde velocity at atrial contraction was not visualized adequately for analysis in the afterload and fluid-loaded conditions in one patient; in the afterload state in a second patient; and in the control, preload, and afterload condi-
tions in a third patient. Therefore, at atrial contraction, 65 velocities were analyzed.

Statistical Analysis

Changes in the hemodynamic and Doppler values at the control state and at each interventional state were compared by using repeated-measures analysis of variance. To analyze the overall relation between two variables, normalized values (intervention divided by control) were used to consider baseline differences in mitral valve impedance, left atrial compliance, and pulmonary vein size. These normalized values were then compared by linear regression analysis.

Results

Relation Between Pulmonary Vein and Mitral Valve Velocities

The temporal relation of the pulmonary vein and mitral valve velocities is shown in Figure 1. At the onset of atrial contraction, there is forward flow through the mitral valve as well as reverse flow into the pulmonary veins. Following atrial contraction and after mitral valve closure, there is forward flow in the pulmonary vein throughout systole with a decrease toward zero (baseline) at end systole. At mitral valve opening, there is rapid early forward flow across the mitral valve followed by deceleration of flow. In the pulmonary vein, there is a nearly simultaneous but slightly slower increase in forward flow velocity with deceleration of flow velocity soon after the deceleration of mitral flow. The contours of the diastolic velocities were similar in both the mitral and pulmonary vein tracings; this resulted in similar deceleration times (Figure 2A) as well as changes in the height of the peak early velocities during the hemodynamic interventions (Figure 2B).

In the two patients with atrial fibrillation, the reverse flow into the pulmonary vein occurring during atrial contraction in patients with sinus rhythm is absent. After mitral valve closure, forward flow is present in the pulmonary vein, although it is a lower velocity than the diastolic flow. Similar to patients in sinus rhythm at mitral valve opening, there is rapid early forward flow velocity followed by deceleration of flow that is similar in the pulmonary vein and across the mitral valve (Figure 3).

Preload Changes

Preload reduction resulted in a significant decrease in the pulmonary wedge pressure (p<0.05) and systolic blood pressure (p<0.005) with no significant change in heart rate or cardiac output (Table 1). During preload reduction, there was a decrease in the height and duration of the atrial reversal in the pulmonary vein (p<0.05) (Figure 4). During systole, the forward flow signal became biphasic in 12 patients. There was a trend toward a lower amplitude
of the diastolic forward velocity as compared with the control state \((p<0.06)\), and there was a significant prolongation of the deceleration time \((p<0.05)\) (Table 2). The mitral velocity curve demonstrated a significant decrease in E velocity \((p<0.05)\) and prolongation of deceleration time \((p<0.05)\).

The increase in preload by fluid administration resulted in a significant increase in blood pressure \((p<0.05)\), pulmonary capillary wedge pressure \((p<0.005)\), and cardiac output \((p<0.05)\) with no significant change in heart rate. The atrial reversal in the pulmonary vein became significantly higher and of longer duration \((p<0.05)\). Both the systolic and diastolic forward flow velocities increased \((p<0.05)\) with a shortening of the deceleration time \((p<0.05)\). The mitral flow velocity demonstrated a significant increase in E velocity \((p<0.05)\), increase in A velocity \((p<0.05)\), and decrease in deceleration time \((p<0.05)\) compared with the control state (Figure 5).

Afterload Changes

Infusion of phenylephrine resulted in an increase in blood pressure \((p<0.001)\) with an overall increase in pulmonary capillary wedge pressure \((p<0.05)\) (Table 1). There was a decrease in heart rate \((p<0.05)\) with no change in cardiac output. There was no consistent change in the pulmonary vein or mitral flow velocity values compared with the control state (Table 2). However, in 13 patients in whom there was a less than 50% increase in wedge pressure, there was a consistent decrease in diastolic pulmonary vein peak velocity, a decrease in mitral flow E velocity, and a prolongation of deceleration times (Figure 6). In the six patients with a pronounced rise in wedge pressure, the Doppler values changed in a manner similar to what occurred with
an increase in preload (i.e., increase in diastolic pulmonary vein velocity, increase in mitral flow E velocity, and shortening of deceleration time). In these six patients, a mid-diastolic flow appeared in both the pulmonary vein and mitral flow velocity curves (Figure 7).

**Overall Relation of Hemodynamics to Doppler Velocities**

Changes in the Doppler values were compared with changes in the hemodynamic variables. The change in systolic flow velocity in the pulmonary vein correlated directly with the change in cardiac output \((r=0.60)\) (Figure 8). As the cardiac output increased, there was a higher systolic velocity.

There was poor correlation between the change in the A velocity of the mitral flow and the change in pulmonary wedge pressure \((r=0.43)\) (Figure 9A). However, there was a better correlation between the change in A velocity of the pulmonary vein flow and the change in pulmonary capillary wedge pressure \((r=0.81)\) (Figure 9B). There was an inverse relation between the change in deceleration time and the change in pulmonary capillary wedge pressure \((r=-0.68)\). A direct correlation was present between the change in mitral flow E velocity and the change in pulmonary capillary wedge pressure \((r=0.69)\).

**Discussion**

Until recently, evaluation of diastolic function by Doppler echocardiography has been limited to measurement of transmitral velocities.\(^5\) Blood flow from the pulmonary circulation into the left ventricle, however, also involves pulmonary venous flow, left atrial contraction, and relaxation as well as flow across the mitral valve. By examining pulmonary vein velocities in conjunction with mitral velocities, a more complete assessment can be made of the filling characteristics of the left side of the heart. The use of transesophageal echocardiography has provided an excellent window for obtaining both the pulmonary vein and mitral velocities by pulsed-wave Doppler echocardiography. By altering the loading conditions in the operating room setting that uses transesophageal echocardiography, information can be obtained to correlate these velocity curves with changing hemodynamic values.

![Figure 4](image-url)  
**Figure 4.** Recordings of a mitral flow velocity curve (top) and pulmonary vein velocity curve (bottom) are shown during control state (left) and after preload reduction with nitroglycerin (NTG) (right). With infusion of nitroglycerin, there was prolongation of deceleration time \((DT)\) with reduction in initial E velocity in the mitral flow. Similarly, in pulmonary vein velocities, there was also a reduction in initial diastolic velocity as well as prolongation of deceleration time. After infusion of nitroglycerin, there was biphasic systolic flow in pulmonary vein velocities. Reversal at atrial contraction \((A)\) in pulmonary vein velocity curve demonstrated decrease in amplitude as well as in duration of this velocity. \(W\), wedge pressure (mm Hg); \(CO\), cardiac output (l/min). Also shown are simultaneous radial artery pressures, with peak systolic pressure shown in parentheses.

---

**Table 1. Hemodynamic Variables**

<table>
<thead>
<tr>
<th>Condition</th>
<th>SBP (mm Hg)</th>
<th>PCWP (mm Hg)</th>
<th>CO (l/min)</th>
<th>HR (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>115±14</td>
<td>13±4</td>
<td>4.2±1.7</td>
<td>59±8</td>
</tr>
<tr>
<td>NTG</td>
<td>90±17*</td>
<td>9±3†</td>
<td>4.0±1.6</td>
<td>61±9</td>
</tr>
<tr>
<td>PE</td>
<td>153±8*</td>
<td>16±5†</td>
<td>4.0±1.6</td>
<td>56±8†</td>
</tr>
<tr>
<td>Fluids</td>
<td>138±12†</td>
<td>21±4*</td>
<td>5.2±2.1†</td>
<td>60±8</td>
</tr>
</tbody>
</table>

SBP, systolic blood pressure; PCWP, pulmonary capillary wedge pressure; CO, cardiac output; HR, heart rate; NTG, intravenous nitroglycerin; PE, intravenous phenylephrine.

\(^*p<0.005\) as compared with control; \(^†p<0.05\) as compared with control.
Table 2. Doppler Variables

<table>
<thead>
<tr>
<th>Condition</th>
<th>S (m/sec)</th>
<th>D (m/sec)</th>
<th>A (m/sec)</th>
<th>A dur (msec)</th>
<th>DT (msec)</th>
<th>E (m/sec)</th>
<th>A (m/sec)</th>
<th>DT (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>0.41±0.13</td>
<td>0.33±0.14</td>
<td>0.18±0.07</td>
<td>110±60</td>
<td>218±39</td>
<td>0.54±0.13</td>
<td>0.44±0.21</td>
<td>225±39</td>
</tr>
<tr>
<td>NTG</td>
<td>0.37±0.14</td>
<td>0.29±0.16</td>
<td>0.12±0.06*</td>
<td>75±44</td>
<td>273±89*</td>
<td>0.46±0.12*</td>
<td>0.41±0.19</td>
<td>301±80*</td>
</tr>
<tr>
<td>PE</td>
<td>0.37±0.16</td>
<td>0.30±0.14</td>
<td>0.20±0.07</td>
<td>105±72</td>
<td>261±88</td>
<td>0.49±0.14</td>
<td>0.44±0.24</td>
<td>264±106</td>
</tr>
<tr>
<td>Fluids</td>
<td>0.52±0.20*</td>
<td>0.44±0.18*</td>
<td>0.29±0.08*</td>
<td>163±65*</td>
<td>150±26*</td>
<td>0.69±0.13*</td>
<td>0.52±0.25*</td>
<td>150±30*</td>
</tr>
</tbody>
</table>

S, maximum systolic antegrade velocity; D, maximum diastolic antegrade velocity; A, velocity at atrial contraction; A dur, duration of velocity at atrial contraction; DT, deceleration time; E, peak velocity at early diastole; NTG, intravenous nitroglycerin; PE, intravenous phenylephrine.

*p<0.05 as compared with control.

Determinants of Pulmonary Vein Flow

Pulmonary vein flow has biphasic forward flow in both animal models and humans.16–21 The pulsatility of flow has been attributed to different factors, including forward transmission of the pressure pulse from the right ventricle through the pulmonary circulation,18,19 changes in left atrial pressure throughout the cardiac cycle,16,17 and a combination of propulsive force of the right ventricle and suction of the left ventricle.20 Recent studies have shown, however, that there is little contribution from the right side of the heart to flow in the large extraparenchymal pulmonary vein and that it is influenced by dynamic changes in left atrial pressure created by contraction and relaxation of both the left atrium and left ventricle.11,13,22

Systolic Forward Flow

Atrial relaxation decreases left atrial pressure, causing systolic forward flow in patients with sinus rhythm.21 In the absence of atrial contraction in the two patients in atrial fibrillation, however, forward systolic flow was still present although of lower velocity than diastolic forward flow. This indicates that downward movement of the mitral valve annulus also contributes to systolic forward flow.11,13 During left ventricular contraction, the annulus moves toward the apex in a rightward direction,23–25 causing an increase in the left atrial area. This left atrial area increase results in a drop in left atrial pressure with a subsequent increase in flow from the pulmonary veins into the left atrium. The magnitude of the annulus excursion and left atrial area change is related to the degree of ventricular emptying.23,26 In the present study, there was a linear correlation between cardiac output and the velocity of systolic forward flow.

In instances of low filling pressures, the systolic forward flow became biphasic. This is probably because of a separation of the atrial relaxation contribution from the annulus movement contribution. With lower atrial pressure, the systolic filling from annular movement will continue longer and peak later than that due to atrial relaxation. Other variables that can affect pulmonary vein systolic flow are the level of left atrial pressure, left atrial compliance, and coexistent mitral regurgitation. In the canine model, as the degree of left atrial pressure is increased, there is less forward flow in late systole.20 Severe mitral regurgitation, if acute, can result in complete reversal of systolic flow.20

Figure 5. Recordings of a mitral flow velocity curve (top) and a pulmonary vein velocity curve (bottom) before (left) and after (right) administration of fluids to increase preload. Mitral flow velocity demonstrates increase in initial E velocity and shortening of deceleration time (DT). Similarly, pulmonary vein velocity demonstrates increase in early diastolic velocity and shortening of deceleration time after administration of fluids. There is also increase in systolic forward flow (S) in pulmonary vein as well as increase in height and duration of atrial reversal (A) after increasing preload and cardiac output. W, wedge pressure (mm Hg); CO, cardiac output (l/min).
These changes are similar to what has been demonstrated in the jugular venous flow pattern for the right side of the heart.27

**Diastolic Forward Flow**

The diastolic forward flow in the pulmonary veins reflects the transmitral filling pattern. The peak of the pulmonary vein diastolic flow occurs approximately 50 msec after the peak of the transmitral flow.13 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.11,18 Both the peak diastolic velocity and deceleration time of the pulmonary vein in this study were similar to the E velocity and deceleration time of the mitral flow. Therefore, the pulmonary vein diastolic flow is dependent on the same factors that influence the mitral valve E velocity and deceleration time; these include left atrial pressure, left ventricular relaxation, and viscoelastic forces of the myocardium.

Previous investigations examined the various factors that influence the velocity of flow across the mitral valve.7,8,28–31 These velocities can be understood if considered in terms of the driving pressure between the left atrium and left ventricle rather than as representing volumetric flow. The mitral flow velocity begins as the left ventricular pressure drops below the left atrial pressure because of continued myocardial relaxation and diastolic suction of the left ventricle.22–24 Acceleration of flow is then established, whereas the left ventricular pressure continues to decline despite filling.29 After a certain time, the effect of filling the ventricle exceeds the effect of relaxation, and the left ventricular pressure begins to rise, whereas the left atrial pressure decreases. The left ventricular pressure then approaches or even exceeds left atrial pressure. With a decreasing pressure gradient, there will be deceleration of forward flow velocity.7,29,32

Changes in mitral flow are measured by the peak E velocity and deceleration time. The peak E velocity is mainly dependent on the initial driving pressure across the mitral valve.7,8,28–31 which is determined by left atrial pressure and the rate of ventricular relaxation. The deceleration time, or rate of deceleration of flow, is dependent on continued ventricular relaxation and ventricular compliance. In this study, as the filling pressure increased, there was a direct increase in the E velocity and decrease in deceleration time. In the presence of increased afterload and the absence of a significant elevation of filling pressure, there was a decrease in E velocity and prolongation of deceleration time.

**FIGURE 6.** Recordings of a mitral flow velocity curve (top) and a pulmonary vein velocity curve (bottom) before (left) and after (right) administration of phenylephrine to increase afterload. After increase in afterload, there is lowering of initial E velocity and prolongation of deceleration time on mitral flow velocity. Similarly, on pulmonary vein velocity, there is decrease in initial diastolic velocity and prolongation of deceleration time (DT)(arrows). There is increase in A velocity on mitral flow velocity curve after administration of phenylephrine. W, wedge pressure (mm Hg); BP, blood pressure (mm Hg).
There was an increase in middiastolic flow that occurred in some patients with early rapid mitral flow deceleration and high afterload.\textsuperscript{13,30} It has been proposed that in the presence of a rapid mitral flow deceleration, the pulmonary veins will refill the atrium, thus restoring a positive-pressure gradient between the veins and atrium. This will cause the mitral valve to reopen and reaccelerate mitral flow.\textsuperscript{13,30} Alternatively, the increase in middiastolic flow can be caused by a change in left ventricular dynamics because of continued ventricular relaxation. It is probably a combination of both factors that contributes to the middiastolic flow because it only occurred in patients with both high filling pressures and high afterload.

**Atrial Reversal**

Atrial contraction results in forward flow across the mitral valve and retrograde flow into the pulmonary veins.\textsuperscript{35} The ventricular filling with atrial contraction has been shown to be dependent on the left ventricular pressure at which contraction begins, with less filling occurring in patients with higher left ventricular diastolic pressures.\textsuperscript{36} There are other factors, however, that influence the velocities at atrial contraction, that is, left ventricular compliance and left atrial contractility. It is only by examining both the mitral and the pulmonary vein velocities together that a better understanding of these factors can be gained.

One cannot directly compare the peak velocity of the pulmonary vein reversal and forward mitral flow velocities at atrial contraction as a measurement of forward and reverse flow because of the different sizes of the orifices through which flow occurs. Examination of both velocity curves, however, can demonstrate what effect atrial contraction has on left atrial and left ventricular pressure. When there is a decrease in the compliance of the left ventricle, there will be a larger increase in left atrial pressure during atrial contraction. Thus, the atrial reversal in the pulmonary vein can become relatively more pronounced than atrial forward flow through the mitral valve. Conversely, an increase in left ventricular compliance results in a reduction in the rise of left atrial pressure during atrial contraction, and there is a lower pulmonary vein atrial reversal and a comparatively higher mitral inflow A wave. When there is a higher atrial preload as occurred with fluid loading, atrial contractility increases, and both the mitral forward flow and pulmonary venous reversal velocities can demonstrate an increase during atrial contraction.\textsuperscript{31,37}
Figure 9. Scatterplots showing relation between change (intervention divided by control [I/C]) in pulmonary capillary wedge pressure (PCWP) and velocities at atrial contraction is shown. Panel A: Comparison of change in pulmonary capillary wedge pressure and peak velocity at atrial contraction on mitral flow velocity. There is poor relation between these two variables. Panel B: Change in pulmonary capillary wedge pressure as compared with change in height of A velocity in pulmonary vein. There is significant linear correlation between these two variables. NTG, administration of nitroglycerin; PE, administration of phenylephrine.

Previous studies of the pulmonary vein in humans have not consistently indicated the presence of atrial reversal.11-13,30 These studies, however, used transthoracic echocardiography, in which the sample volume might not have been placed within the pulmonary vein but rather at the orifice of the pulmonary vein.22 With transesophageal echocardiography, the sample volume can be placed 1-2 cm proximal to the pulmonary vein–left atrial junction. Placing the sample volume in this area revealed the presence of an atrial reversal in most adult patients with sinus rhythm.

Limitations

The results of this study demonstrate the relation of the pulmonary vein velocities to mitral flow velocities at different loading conditions to help understand the significance of the various velocity curves in relation to filling of the left side of the heart. More sophisticated measurements of diastolic values such as the time constant of relaxation (r) or stress-strain relations would have been ideal to further correlate with the Doppler velocities. It was not believed ethically correct, however, to perform these invasive measurements in the prebypass patients. Previous studies in the canine model provide a background for the diastolic variables that are affected by different loading conditions, which can be extrapolated to the current study.7,8,28,29,31 Both afterload and preload increase prolong the time constant of relaxation, which is a marker of myocardial relaxation. In the absence of a direct affect on the myocardium, there should be little change in the module of chamber stiffness during different loading conditions. However, because the diastolic portion of the pressure-volume curve is curvilinear,38 there should be a decrease in the “effective compliance” with increases in both afterload and preload.

There was no attempt made to control the filling pressures of the heart during afterload manipulation or to control blood pressure during preload manipulation. Thus, the results represent not necessarily the sole effect of changing one loading condition but rather a complex interaction of several loading variables. This was particularly evident in the afterload increase state, in which the measured variables were affected by the resultant change in wedge pressure.

All of the patients entered into the study had ischemic heart disease. Although continuous electrocardiographic monitoring demonstrated no ST segment abnormalities, it is still possible that the presence or absence of myocardial ischemia might have contributed to the changes observed in the diastolic flow parameters. Patients with valvular heart disease and no significant coronary disease, however, were not studied because the valvular abnormalities themselves can affect the mitral flow velocity curves. It was not ethically possible to perform this study on patients with normal hearts who were not undergoing open-heart surgery.

Complete autonomic blockade also was not used in this study, and reflex autonomic changes might have affected the filling variables of the heart. Nonetheless, the anesthesia provided some sympatholytic effect, as reflected in the relatively constant heart rate throughout the study.

Conclusion

Examination of pulmonary vein velocities with mitral flow velocities provides additional information regarding diastolic function. This information cannot be obtained from the mitral flow velocities alone. The changes in both of these velocity curves during different loading conditions are helpful in understanding the main determinants and their effects on the various velocity curves.
References


**Key Words**: echocardiography, transesophageal • diastolic filling • echocardiography, Doppler
Relation of pulmonary vein to mitral flow velocities by transesophageal Doppler echocardiography. Effect of different loading conditions.
R A Nishimura, M D Abel, L K Hatle and A J Tajik

Circulation. 1990;81:1488-1497
do: 10.1161/01.CIR.81.5.1488
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1990 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/81/5/1488

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/