The Transmitral Pressure–Flow Velocity Relation

Effect of Abrupt Preload Reduction

Michael Courtois, MA, Zvi Vered, MD, Benico Barzilai, MD, Nancy A. Ricciotti, MSN, Julio E. Pérez, MD, and Philip A. Ludbrook, MB, BS, FRACP

Although recent animal and clinical studies suggest that Doppler-derived indexes may be useful for the characterization of ventricular diastolic behavior, the hemodynamic basis for the preload dependency of these indexes has not previously been fully elucidated. Accordingly, effects of reduction of left atrial load on the pressure–flow velocity relation were characterized in 10 anesthetized, closed-chest dogs during transient inferior vena caval occlusion by means of simultaneously recorded left atrial and left ventricular micromanometric pressure measurement and transesophageal Doppler echocardiograms. Within four or five beats after inferior vena caval balloon occlusion, left atrial loading was reduced as evidenced by a decrease in the slope of the left atrial v wave from 21±4 to 13±4 mm Hg/sec (p<0.001) and by a decrease in the first crossover point of left atrial and left ventricular pressures from 5.6±1.1 to 2.9±1.5 mm Hg (p<0.001). This decrease in left atrial loading resulted in reductions during early diastole of minimum left ventricular pressure (from 1.0±0.8 to −0.4±1.2 mm Hg, p<0.001), the maximum early forward (i.e., left atrial pressure>left ventricular pressure) transmitral pressure gradient (from 2.8±0.8 to 2.4±0.5 mm Hg, p<0.01); the slope of the rapid filling pressure wave (from 44±11 to 38±10 mm Hg/sec, p<0.025); and the area of the reversed (i.e., left ventricular pressure>left atrial pressure) transmitral pressure gradient (from 79±42 to 53±33 mm Hg·msec, p<0.05). During late diastole, both the heights and slopes of the left atrial and left ventricular a waves fell, resulting in a decrease in the maximum late transmitral pressure gradient (from 1.2±0.7 to 0.9±0.5 mm Hg, p<0.05). Vena caval occlusion also altered Doppler transmitral velocity profiles during both the early and late phases of diastole. Peak velocity of the E wave decreased (from 50±11 to 41±7 cm/sec, p<0.01) as did acceleration (from 880±222 to 757±258 cm/sec², p<0.025) and deceleration (from 597±260 to 429±197 cm/sec², p<0.025). Peak velocity of the A wave also fell (from 29±9 to 22±5 cm/sec, p<0.005). Abrupt inferior vena caval occlusion did not significantly change heart rate or mean aortic pressure. Significant correlations were obtained between peak flow velocity during early diastole and the peak forward pressure gradient during early diastole (r=0.67, p<0.05), between peak flow velocity during late diastole and the peak pressure gradient during late diastole (r=0.80, p<0.01), and between deceleration rate of the Doppler E wave and the magnitude of the reversed transmitral pressure gradient (r=0.60, p<0.01). These results indicate that an acute reduction in left atrial loading conditions significantly alters the diastolic transmitral pressure relation and thus profoundly affects the Doppler flow velocity profile.

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Pulsed Doppler–derived indexes of left ventricular diastolic function have been used to characterize diastolic behavior in various cardiac diseases and age-related conditions. However, recent evidence has indicated that the Doppler time-velocity profile is affected by reductions in left ventricular preload. Because Doppler echocardiography is gaining wide acceptance as a clinical tool, it is crucial that the alterations in cardiac hemodynamics that underlie these preload-
related alterations in the Doppler velocity profile be fully elucidated and understood. Accordingly, this study was designed to define the effects of a sudden reduction in preload on the transmitral pressure–flow velocity relation.

Materials and Methods

Mongrel dogs weighing 27–41 kg were sedated with morphine sulfate (1 mg/kg s.c.) 30 minutes before induction of general anesthesia with sodium pentothal (12.5 mg/kg i.v.) and α-chloralose (70 mg/kg i.v.). Each dog was intubated and ventilated with room air with a Harvard respirator. The right jugular vein, left common carotid artery, right femoral artery and vein, and left femoral vein were isolated and a valved sheath (Hemoquet 8F, USCI, Billerica, Massachusetts) placed in each. A transesophageal two-dimensional phased-array echocardiographic 5-MHz transducer with pulsed Doppler capabilities (Model 77020A Ultrasound System, Hewlett-Packard, Andover, Massachusetts) was positioned so that the interatrial septum was clearly visualized. A Mullins transseptal catheter introducer set (8F, USCI) and Brockenbrough needle (18 gauge, USCI) were directed under fluoroscopy into the right atrium, and aided by two-dimensional echocardiographic targeting, the entire assembly was pressed against the interatrial septum and advanced through it into the left atrium. After successful puncture was verified by injection and visualization of contrast in the left atrium and left ventricle with two-dimensional echocardiography, the dilator and needle were removed and a bolus of heparin sodium (4,000 USP units) administered. Under fluoroscopy, micromanometric-angiographic catheters (8F; Model 484A, Millar Instruments, Houston, Texas) were directed into both the left atrium via the transseptal sheath and the left ventricle retrogradely across the aortic valve via the left carotid artery. A Swan-Ganz thermodilution catheter (Model 93-131-7F, American Edwards, Santa Ana, California) was directed from the jugular vein to the superior vena cava; a fluid-filled pigtail catheter (7F, Cordis) was directed from the femoral artery to the arch of the aorta, and an atrioseptostomy balloon catheter (Model 83-051-5F, American Edwards) was directed from the left femoral vein to the inferior vena cava. Before introduction into the animal, the micromanometers were calibrated relative to atmospheric pressure. Before each experimental recording, the micromanometric signal obtained from the left atrial catheter was aligned with the pressure measured by its fluid-filled lumen connected to a transducer positioned at the midthoracic level. Left ventricular micromanometric pressure was then aligned with left atrial micromanometric pressure during long diastatic periods associated with slow heart rates or compensatory pauses after premature ventricular contractions (Figure 1). A low-gain signal (100 mm Hg=12.5 cm) from the aortic fluid-filled catheter and a high-gain signal (20 mm Hg=10 cm) from the

FIGURE 1. Record of measurements designed to characterize the transmitral pressure relation. Measurements included slope of the left atrial v wave, first transmitral pressure crossover point (X1), minimum left ventricular pressure, maximum early forward pressure gradient where left atrial pressure exceeds left ventricular pressure, the slope of the left ventricular rapid-filling pressure wave, the area of the reversed gradient between the second (X2) and third (X3) points of transmitral pressure crossover where left ventricular pressure exceeds left atrial pressure, slopes and heights of both the left atrial and left ventricular a waves, and maximum pressure gradient during atrial contraction.
left atrial micromanometric catheter were transmitted to a photographic recorder (visicorder Model 1508B, Honeywell, Denver, Colorado), and a high-gain signal from the left ventricular micromanometric catheter was transmitted to both the photographic recorder and a heat-sensitive recorder (Model 77500B, Hewlett-Packard) interfaced with the ultrasound imaging system. At the start of each experimental recording, a square wave was delivered to both recorders via the left ventricular transducer control unit (Model TCB 100, Millar Instruments, Houston, Texas), facilitating precise alignment of the transmitral pressures and the Doppler time-velocity profiles.

Because regional pressure gradients have been shown to exist in the left ventricle during both the early and the late phases of diastole, the position of the micromanometer in the left ventricle was standardized at 4 cm from the apex. This was accomplished by advancing the left ventricular micromanometric-angiographic catheter under fluoroscopy to the cardiac apex. Once contact with the apex was made, the catheter was pulled back 2.5 cm. Because the measured distance of the micromanometer from the leading edge of the pigtail was 1.5 cm, this maneuver positioned the transducer approximately 4 cm from the ventricular apex. Because previous experience in our laboratory with dogs of the size used in this study indicates that the major chord is in the range of 7–8 cm, this position corresponds to a midventricular level.

All hemodynamic and echocardiographic measurements were made with the animal in the supine position. Transmitral time-velocity profiles were obtained by adjusting the transesophageal probe to obtain a modified four-chamber view, with the sample volume positioned at the level of the mitral anulus. Electrocardiographic activity was monitored via a precordial (V4) electrode. Core temperature was monitored throughout the study and maintained with a circulating water (38°C) heating pad. At no time did any of the animals’ core temperature fall below 36.6°C. Arterial blood gases were measured periodically, and respiratory rate and volume adjusted accordingly.

**Analysis of Data**

All pressure and time-velocity measurements were made during the end-expiratory portion of the respiratory cycle within four or five beats after balloon inflation, before any increase in heart rate. Hemodynamic measurements included (Figure 1) the slope of the left atrial v wave, the point of first crossover of the left atrial and ventricular pressures (X.), minimum left ventricular pressure, the maximum early forward (left atrial pressure>left ventricular

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**FIGURE 2. Record of measurements designed to characterize the Doppler time-velocity profile. Recordings were obtained from a transesophageal view. In this position, the flow of blood during diastolic filling is away from the transducer; hence, the time-velocity waveforms are negative. Measurements included acceleration and deceleration rate of the early (E) diastolic filling wave and peak velocity of both the early and late (A) diastolic filling waves.**
FIGURE 3. Top panel: Record of transmitral pressures (mm Hg) at baseline, before inferior vena caval occlusion. Bottom panel: Record of transmitral pressures (mm Hg) during inferior vena caval occlusion. Within four or five beats after balloon occlusion of the inferior vena cava, filling of the left atrium is greatly reduced as evidenced by a decrease in the slope and height of the left atrial v wave. This decrease in left atrial loading leads to a reduction in minimum left ventricular pressure, a decrease in the early forward maximum pressure gradient during early diastolic filling, and a reduction in the slope of the left ventricular rapid filling pressure wave and subsequent overshoot of left atrial pressures in the form of a reversed pressure gradient. Decreases also occur in both the heights and slopes of the left atrial and left ventricular pressure a waves and in the maximum pressure gradient generated during atrial contraction.

pressure) transmitral pressure gradient, the slope of the left ventricular rapid filling pressure wave, the area of the reversed (left ventricular pressure>left atrial pressure) transmitral pressure gradient circumscribed by the atrial and ventricular pressure signals between the second (X₂) and third (X₃) points of crossover, the heights and slopes of both the left ventricular and left atrial a waves, and the maximum pressure gradient during atrial contraction. To facilitate precise measurement of these gradients and areas, hemodynamic recordings were magnified with a high-quality copier.

Transesophageal Doppler time-velocity profile measurements included (Figure 2) acceleration and deceleration rate of the early (E) diastolic filling wave and peak velocity of both the early and late (A) diastolic filling waves.

Five to eight occlusions of the inferior vena cava were performed on each animal. Subsequent analyses of the pressure and Doppler waveforms were
carried out on recordings that were free of premature ventricular contractions, demonstrated well-defined Doppler profiles, showed minimal (<5%) changes in heart rate and aortic pressure, and exhibited an end-expiratory respiratory phase in the beats immediately after inferior vena caval occlusion. Data from recordings meeting these criteria were averaged; this was generally two recordings for each animal.

Differences between baseline and intervention conditions were detected with the paired t test. All data are reported as mean±SD.

**Results**

**Early Diastolic Transmural Pressures and Doppler Flow Velocity During Inferior Vena Caval Occlusion**

Within four to five beats after balloon occlusion of the inferior vena cava, pressure in the left atrium was greatly reduced (Figure 3) as evidenced by a prominent reduction in the slope and height of the left atrial v wave. This reduction in left atrial loading was associated with a decline in minimum left ventricular pressure, a decrease in the early maximum forward pressure gradient during early filling, and a reduction in the slope of the left ventricular rapid filling pressure wave and subsequent overshoot of left atrial pressures in the form of a reversed pressure gradient. Group data derived from transmural pressures for the baseline and occlusion conditions are presented in Table 1. All measured parameters except heart rate and mean aortic pressure changed significantly after vena caval occlusion. These changes in early transmural pressures greatly affected the configuration of the early Doppler time-velocity profile (Figure 4), manifested by significant reductions in acceleration, maximum velocity, and deceleration. Group data derived from the Doppler E wave is presented in Table 2. Individual data points for the maximum early pressure gradient and peak E wave velocity are presented in Figures 5 and 6, respectively.

**Late Diastolic Transmural Pressures and Doppler Flow Velocity During Inferior Vena Caval Occlusion**

After reduction of left atrial loading, significant changes in transmural pressure and flow velocity also occurred during the late phase of diastolic filling associated with atrial contraction. Significant decreases were noted in the heights and slopes of both the left atrial and left ventricular pressure and wave, resulting in a decline in the maximum pressure gradient generated during atrial contraction (Figure 3). This reduction in the pressure gradient across the mitral valve during late diastole was associated with a reduction in the maximum velocity of the Doppler A wave (Figure 4). Group data derived from left atrial and left ventricular pressures and the Doppler time-velocity profiles during atrial contraction are presented in Tables 1 and 2, respectively. Significant changes were observed in all variables after vena caval occlusion. Individual data points for the maximum pressure gradient during late diastole and peak velocity of the Doppler A wave are presented in Figures 7 and 8, respectively.

**Correlation Between Doppler Flow Velocity Measurements and Transmural Pressure Gradients**

To normalize for differences in mitral valve resistance, the ratios of the paired data (baseline divided by occlusion) were used. With this technique, peak velocity of the Doppler E wave and the maximum forward pressure gradient during early diastolic filling exhibited a moderate correlation ($r=0.67, p<0.05$) (Figure 9), whereas peak velocity of the Doppler A wave and the maximum pressure gradient during atrial contraction correlated somewhat more strongly ($r=0.80, p<0.01$) (Figure 10). In addition, a significant, albeit weak, correlation was found between deceleration of the early filling wave and the magnitude of the reversed transmural pressure gradient ($r=0.60, p<0.01$) (Figure 11).

**Discussion**

The present study confirms recent reports that reductions in left ventricular preload significantly alter the Doppler transmural inflow time-velocity profile during early diastolic filling. We also noted a significant change in the late diastolic filling wave.

### Table 1. Changes in Hemodynamics During Inferior Vena Cava Occlusion

<table>
<thead>
<tr>
<th></th>
<th>Baseline (n=10)</th>
<th>IVCO (n=10)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats/min)</td>
<td>84±16</td>
<td>85±18</td>
<td>NS</td>
</tr>
<tr>
<td>AoM (mm Hg)</td>
<td>99±14</td>
<td>96±15</td>
<td>NS</td>
</tr>
<tr>
<td>X1 (mm Hg)</td>
<td>5.6±1.1</td>
<td>2.9±1.5</td>
<td>0.001</td>
</tr>
<tr>
<td>v wave slope (mm Hg/sec)</td>
<td>21±4</td>
<td>13±4</td>
<td>0.001</td>
</tr>
<tr>
<td>LVP max (mm Hg)</td>
<td>1.0±0.8</td>
<td>-0.4±1.2</td>
<td>0.001</td>
</tr>
<tr>
<td>ETP max (mm Hg)</td>
<td>2.8±0.8</td>
<td>2.4±0.5</td>
<td>0.008</td>
</tr>
<tr>
<td>RFW slope (mm Hg/sec)</td>
<td>44±11</td>
<td>38±10</td>
<td>0.023</td>
</tr>
<tr>
<td>ARP (mm Hg * msec)</td>
<td>79±42</td>
<td>53±33</td>
<td>0.033</td>
</tr>
<tr>
<td>LA a wave slope (mm Hg/sec)</td>
<td>60±24</td>
<td>37±14</td>
<td>0.002</td>
</tr>
<tr>
<td>LA a wave max (mm Hg)</td>
<td>6.6±1.1</td>
<td>4.3±1.2</td>
<td>0.001</td>
</tr>
<tr>
<td>LV a wave slope (mm Hg/sec)</td>
<td>27±12</td>
<td>18±8</td>
<td>0.001</td>
</tr>
<tr>
<td>LV a wave max (mm Hg)</td>
<td>6.2±1.3</td>
<td>3.5±1.6</td>
<td>0.001</td>
</tr>
<tr>
<td>LTP max (mm Hg)</td>
<td>1.2±0.7</td>
<td>0.9±0.5</td>
<td>0.049</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>6.5±1.8</td>
<td>3.8±1.5</td>
<td>0.001</td>
</tr>
</tbody>
</table>

IVCO, inferior vena caval occlusion; HR, heart rate; AoM, mean aortic pressure; X1, first crossover of transmural pressures; v wave slope, upslope of the left atrial v wave; LVP max, minimum left ventricular pressure; ETP max, maximum early diastolic transmural pressure gradient; RFW slope, slope of the rapid filling pressure wave; ARP, area of reversed pressure gradient where left ventricular pressure exceeds left atrial pressure; LA a wave slope, upslope of the a wave; a wave max, peak of a wave; LV, left ventricular; LTF max, maximum late diastolic transmural pressure gradient; LVEDP, left ventricular end-diastolic pressure.
More important, the present study describes in detail the underlying transmural pressure changes that accompany and are responsible for these alterations in flow velocity.

Other investigators have reported that a decrease in preload is accompanied by significant changes in the early filling wave, but only small, generally statistically nonsignificant changes in the late filling wave. Choong et al\(^6\) effected a reduction in preload with nitroglycerin infusion in humans undergoing cardiac catheterization and noted reductions in acceleration, deceleration, and maximum velocity of the early filling wave but no change in maximum A wave velocity. Using lower body negative and positive pressure to alter preload by altering venous return in humans with coronary artery disease, Takanashi et al\(^8\) showed the maximum velocity of the early diastolic filling wave to be very sensitive to changes in left atrial pressure, whereas significant differences in A wave maximum velocity were seen only at the extreme of applied negative pressure. Bhatia et al\(^9\) decreased left ventricular preload in normal humans with nitroglycerin and reported a significant difference in maximum E wave velocity but no change in A wave velocity.

The present study confirms that the early diastolic Doppler flow velocity waveform is substantially altered by changes in left ventricular preload. Significant changes were found in acceleration, deceleration, and peak velocity of flow as measured from the Doppler E wave. However, contrary to the results of other studies, we also found a significant difference in the maximum velocity of the late filling wave associated with atrial contraction. This discrepancy may be due in part to modest increases in heart rate that accompanied preload reduction in these other studies. Choong et al\(^6\) reported no change in peak A wave velocity and noted a statistically significant increase in heart rate (from 60 to 65 beats/min). Likewise, Bhatia et al\(^9\) reported a significant increase in heart rate (from 65 to 73 beats/min) but noted no changes in the Doppler A wave. Increases in heart rate are well known to affect the late phase of diastolic filling.\(^1^4\) Two recent preliminary reports of the effects of heart rate on transmural Doppler profiles concluded that late mitral inflow patterns are highly sensitive to heart rate changes. In humans, Parker et al\(^1^5\) noted a

![Graphical representation of Doppler time-velocity profiles](image)

**Figure 4.** Left panel: Record of the Doppler time-velocity profile at baseline before inferior vena caval occlusion. Right panel: Record of the Doppler time-velocity profile during balloon occlusion of the inferior vena cava. Compared with baseline, decreases are noted in the acceleration and deceleration rates of the E wave and in the heights of both the E and A Doppler time-velocity waveforms.

<table>
<thead>
<tr>
<th>TABLE 2. Changes in Doppler Time-Velocity Profile During Inferior Vena Caval Occlusion</th>
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</thead>
<tbody>
<tr>
<td>Baseline</td>
</tr>
<tr>
<td>(n=10)</td>
</tr>
<tr>
<td>E acceleration (cm/sec(^2))</td>
</tr>
<tr>
<td>Maximum E velocity (cm/sec)</td>
</tr>
<tr>
<td>E deceleration (cm/sec(^2))</td>
</tr>
<tr>
<td>Maximum A velocity (cm/sec)</td>
</tr>
</tbody>
</table>

IVCO, inferior vena caval occlusion; E, early Doppler time-velocity filling wave; A, late Doppler time-velocity filling wave.
uniform rise in peak A wave velocity with increasing heart rate, with an excellent correlation ($r = -0.97$) between heart rate and the ratio of maximum E to maximum A wave velocity. Likewise, Gillam et al. measured mitral inflow profiles with Doppler in patients with programmable pacemakers and reported that changes in heart rate greatly influenced late inflow patterns. An increase in heart rate from 60 to 70 beats/min was associated with a statistically significant 10% increase in peak velocity of the A wave. Thus, small increases in heart rate may negate any decrease in A wave peak velocity that might otherwise accompany a decrease in preload. In the present study in which hemodynamics and Doppler data were recorded within four or five beats of the onset of vena caval occlusion, no increase in heart rate was noted (from 84 to 85 beats/min). Hence, the results of the preceding reports may represent changes associated with preload reduction in a clinical setting where a number of uncontrollable factors, including heart rate, may be expected to vary, whereas the results of the present study may represent more nearly pure preload reduction.

A second possible explanation for the discrepancy between our report and those of others might be the degree of preload reduction produced by inferior vena caval occlusion compared to that, for example, of nitroglycerin. However, Choong et al. who found only a small, statistically nonsignificant decrease in peak A wave velocity (from 61 to 59 cm/sec), reported a 58% decrease in the peak of the pulmonary wedge v wave (from 12 to 5 mm Hg) with nitroglycerin, which would seem to compare reasonably with the $X_t$ data reported in this study (a 48% decrease from 5.6 to 2.9 mm Hg). Still, the idea that changes in the A wave are related to the degree of preload reduction is reinforced by the report of Takahashi et al. who noted a statistically significant 17% decrease in peak A wave velocity at the extreme of applied negative lower-body pressure (−40 mm Hg) but found statistically nonsignificant changes at higher pressures.

A third possible explanation for this discrepancy may be found on close examination of the individual data points presented in Figure 8. As can be seen, the greatest changes in A wave peak velocity are found in animals exhibiting the largest A waves at baseline. Mean change for the four animals with the largest A waves at baseline is −35% (from 38 to 25 cm/sec; peak E: peak A ratio at baseline, 1.20). Mean change for the remaining six animals is −17% (from 24 to 20 cm/sec; peak E: peak A ratio at baseline, 2.32). Thus, the substantial (−25%) decrease in the A wave reported in this study may have been influenced by a
heart is less capable of accelerating blood as measured by Doppler echocardiography. The reduction in acceleration and maximum flow velocity during early diastole appears to be directly due to the reduction in the pressure gradient across the mitral valve. This loss of pressure gradient due to the sudden reduction in left atrial loading cannot be compensated for by elastic recoil alone despite a possible reduction in end-systolic volume that is known to accompany reductions in preload and leads to augmented suction. In this study, the maximum transmitral pressure gradient during early diastole was found to correlate significantly with the peak velocity of the Doppler E wave. This is in keeping with a recent report by Ishida et al., who described a significant linear correlation between early peak transmitral flow measured by electromagnetic flow probe and the peak transmitral pressure difference during early diastolic filling.

The results of the present study are consistent with a model of the heart that views early diastolic filling as resulting from two processes: elastic potential energy developed in the left atrial wall during the previous right ventricular contraction and elastic potential energy stored in the left ventricular wall during the previous left ventricular contraction. If, as in this study, atrial loading is reduced dramatically, forces developed in the atrium that normally contribute to diastolic filling during early diastole are attenuated and subsequent rapid ventricular filling may not be completed as effectively. Although the right side of the heart was not evaluated in this experiment, we assume that diastolic filling of the right ventricle might be similarly influenced by abrupt preload reduction.

During late diastolic filling, a significant decrease in height and upslope of the left atrial a wave during inferior vena caval occlusion. This probably relates to the biphasic nature of pulmonary blood flow and is the result of reduced refilling of the atrium after ventricular rapid filling phase, resulting in unloading of the atrium before atrial contraction. Thus, the velocity of the blood subsequently injected into the ventricle during left atrial contraction is reduced. This is also reflected in a reduction in the upslope and
height of the left ventricular a wave as less impact is produced in the ventricle. As expected, a significant correlation was also found between the maximum pressure gradient and Doppler-derived peak flow velocity during this phase of diastole.

Deceleration of Early Flow Velocity and the Reversed Transmitral Pressure Gradient

Because blood accelerated during early diastole returns to near zero velocity before systolic ejection, a force comparable in magnitude and opposite in direction to the accelerative force should be present during deceleration of flow. The downslope of the early time-velocity transmitral filling wave has previously been shown to be temporally related to the occurrence of a reversed transmitral pressure gradient. The weak, though significant, correlation between E wave deceleration and degree of transmitral pressure reversal obtained in the present study also supports the idea that these phenomena are related. The low correlation is most probably attributable to variation in factors not specifically measured in this study, such as relative intraventricular catheter position and ventricular compliance.

The mechanism for this observed reduction in the downslope of the E wave in association with preload reduction is unknown, but it may be speculated that it might be related to changes in the process of elastic recoil. During normal early diastolic filling, the ventricular wall, augmented by elastic potential energy stored in and then released by the left atrial wall, presumably recoils promptly to some "equilibrium" configuration. Consequently, blood decelerating by collision with the ventricular wall will be impacting with an essentially stationary structure and will therefore decelerate rapidly. If left atrial loading is substantially reduced, forces ordinarily provided by normal atrial filling are reduced, and the rate of ventricular recoil might tend to be attenuated. Thus, blood decelerating by impaction against the ventricular wall will collide with a structure that is still in motion, moving in the same direction as the flow of blood. Hence, the impact of collision is reduced, and deceleration, as evidenced by the downslope of the Doppler E wave, is less abrupt. Clinical pathological examples of the extremes of this scenario are represented by restrictive-constrictive physiology and mitral stenosis. In the case of restrictive ventricular physiology, wall expansion is halted abruptly, and shortened deceleration times for the E wave are noted across both the mitral and tricuspid valves. At the other extreme, mitral stenosis represents a situation in which wall expansion occurs over a prolonged period because of restricted mitral inflow, and E wave deceleration times are markedly prolonged. If it is conceded that the upstroke of the left ventricular rapid filling pressure wave results from the collision of blood with the ventricular wall during early diastolic filling, the decrease in the slope of the rapid filling pressure wave and extent of subsequent transmural pressure reversal after preload reduction may also be due in part to this phenomenon and in part to the reduction in peak velocity reached by the blood during the rapid filling phase.

Another possible explanation for the slower E wave deceleration rate recorded after preload reduction relates to factors that contribute to the functional compliance of the left ventricle. Because reductions in left ventricular volume accompany reductions in preload, the ventricle may be operating on a lower portion of the pressure-volume curve, and it is conceivable that the functionally more compliant ventricle may be less effective at rapidly decelerating blood. In addition, substantial decreases in right ventricular volume that accompany vena caval occlusion may also contribute to shifting the left ventricle to a lower portion of the pressure-volume curve or displacing the entire curve downward.

Implications and Conclusions

The present study demonstrates that in this preparation, a sudden reduction of left atrial pressure by inferior vena caval occlusion significantly alters the transmitral pressure relation and, thus, profoundly affects the transmital Doppler flow velocity profile during both the early and late phases of diastole. Hence, experimental or clinical studies that use parameters derived from Doppler time-velocity plots to characterize left ventricular diastolic function must rigorously control or make allowances for left atrial loading conditions.

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References

6. Choong CY, Herrmann HC, Weyman AE, Fifer MA: Preload dependence of Doppler-derived indexes of left ventric-


8. Takahashi T, Sato H, Iizuka M, Serizawa T, Ohya T, Takatomi M, Sakamoto T: Left ventricular early filling flow is sensitive to changes in left atrial pressure: A study using lower body negative and positive pressure (abstract). *Circulation* 1986;74(suppl II):II-477


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