Characterization of Regional Diastolic Pressure Gradients in the Right Ventricle

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Regional intraventricular pressure gradients exist in the left ventricle (LV) during both the early and late filling phases of diastole. These regional pressure gradients comprise a fundamental component of the mechanism of normal LV filling. To determine whether similar diastolic pressure gradients also occur in the right ventricle (RV), we measured right atrial (RA) and RV regional pressures with use of micromanometers in six anesthetized, closed-chest dogs. Tricuspid flow velocity was recorded with use of transesophageal Doppler echocardiography, and right ventriculograms were obtained with contrast angiography. As in the LV, the maximum RA-RV pressure gradient during early diastole was consistently greater if RV pressure was measured near the apex than in the inflow tract (1.6±0.5 versus 0.8±0.4 mm Hg). The area of reversed pressure was also found to be significantly greater in the apex than in the inflow tract (72±43 versus 8±6 mm Hg·msec). However, unlike the LV, the lowest minimum pressure was usually recorded in the RV outflow tract, resulting in a significantly increased RA-RV outflow tract pressure gradient compared with the RA-RV apex pressure gradient (2.5±0.8 versus 1.6±0.5 mm Hg). Analysis of right ventriculograms indicates marked narrowing of the RV outflow tract at end systole in all six animals, suggesting that an end-systolic deformation in this region is the likely mechanism for production of low early diastolic pressure in this region. During atrial contraction the RV regional pressure gradient pattern was similar to the LV pattern: the RV a-wave ascent occurred earlier in the inflow tract and later in the apex. A-wave ascent appeared to occur almost simultaneously in the apex and outflow tract. In the six animals, Doppler-derived peak tricuspid flow velocity during early diastole was 35±6 cm/sec. Early tricuspid flow acceleration (393±101 cm/sec²) was found to be significantly greater than deceleration of flow (182±59 cm/sec²). Comparison of tricuspid pressure-flow data with mitral pressure-flow data previously obtained in our laboratory indicates that the driving pressure gradient across the tricuspid valve is significantly less than across the mitral. This pressure difference corresponds to differences in acceleration and peak flow found across the two valves. Consideration of these physiological patterns of RV diastolic intraventricular pressure and their relation to filling has important implications with regard to the development of indexes that characterize diastolic pressure-flow relations and provides physiological insight relating to the location of ventricular restoring forces. (Circulation 1990;82:1413–1423)

Regional intraventricular pressure gradients exist in the left ventricle (LV) during both the early rapid filling and late atrial filling phases of diastole.¹² These regional pressure gradients comprise a fundamental property of the mech-

anism of normal LV filling³ and must be taken into account when developing pressure-derived indexes or constructing theoretical models of cardiac diastolic function. Accordingly, the present study was designed to determine whether significant diastolic pressure gradients also occur in the right ventricle and to characterize their relation to RV filling.

Methods
Six mongrel dogs of either gender weighing 27–33 kg (31±2) were sedated with morphine (1 mg/kg s.c.) 30 minutes before induction of general anesthesia with sodium pentothal (12.5 mg/kg i.v.) and α-chloralose (90 mg/kg i.v.). Each dog was intubated and ventilated with room air using a Harvard respi-
The right and left jugular veins, the right and left femoral veins, and the left femoral artery were isolated and a valved sheath (USCI; Hemaquet 8F) was placed in each. A bolus injection of heparin sodium (5,000 USP units) was then administered intravenously. A Swan-Ganz thermodilution catheter (model 93A-131-7F, American Edwards) was directed under fluoroscopy from the left femoral vein to the inferior vena cava. A micromanometer-tipped catheter (model PC-482-8F, Millar Instruments) was advanced from the right femoral vein into the pulmonary artery. This catheter was then pulled slowly back across the pulmonary valve and positioned in the right ventricular outflow tract such that a smooth, artifact-free RV pressure contour was obtained throughout systole and diastole. A second micromanometer-tipped catheter (model PC-482-8F, Millar Instruments) was advanced from the right external jugular vein to a position as close as possible to the RV apex. Because of the degree of trabeculation in the right ventricle, it was often difficult to obtain an artifact-free pressure recording in the apical region. In two dogs, we were unable to obtain adequate apical pressure recordings, and these animals were excluded from the data analysis (for apical pressures; all other data from these animals are included). After the recording of RV apical pressures, this catheter was pulled back to a position just inside the tricuspid valve to obtain RV inflow tract pressures. A third micromanometer-tipped catheter (model 484A-8F, Millar Instruments) was advanced from the left external jugular vein into the midnight atrium. RV inflow pressure recordings were also obtained with this catheter by advancing it across the tricuspid valve. Thus, by altering the position of these three catheters we obtained simultaneous recordings of pressure in the RA and RV outflow tract, the RA and RV apex, the RA and RV inflow tract, the outflow tract and apex, the outflow and inflow tracts, and the apex and inflow tract. This allowed us to examine differences in pressure between any combination of the three regions by either sequential or simultaneous recordings. Thus, for example, we were able to verify the reliability of differences obtained by sequential measurement between the RV apex and inflow tract by the method of simultaneous pressure measurement. A fourth micromanometric catheter (model 484A-8F, Millar Instruments) was directed from the right femoral artery to the aortic arch to obtain aortic pressure. To optimize the stability of the micromanometric signals, catheters were immersed in saline for a minimum of 12 hours before use. To record “absolute” RV pressure, referenced solely to atmospheric pressure rather than to an external fluid-filled transducer signal which is highly dependent on the height of the external transducer relative to the height of the heart, the right atrial catheter was placed in a dry graduated cylinder that was immersed in a water bath warmed between 36.5° and 38°C, depending on the temperature of the animal. Because micromanometric sensors have been shown to be light sensitive, the room lights were extinguished prior to “zeroing.” The RA pressure signal was used as the “standard” throughout the experiment, and all other pressure signals were aligned with this signal during the late diastatic filling period. In the presence of rapid heart rates, the alignment of pressures was accomplished during the long diastatic period occurring after premature ventricular contractions that occurred either spontaneously or were stimulated mechanically. At the conclusion of the experiment the RA catheter was replaced in the graduated cylinder, the room lights were extinguished, and the zero baseline was checked. In no case did the signal drift more than 1.0 mm Hg from the original zero baseline.

A low-gain pressure signal from the aortic micromanometric catheter (100 mm Hg =10.0 cm) and three high-gain pressure signals from the RV outflow, RV apex, and RA micromanometers (20 mm Hg =15 cm) were transmitted to a photographic recorder (model 1508B, Honeywell Visicorder). The RV outflow high-pressure signal was also transmitted to a heat-sensitive recorder (model 77500B, Hewlett-Packard) interfaced with an ultrasound imaging system. Transticuspid Doppler recordings were made with an esophageal two-dimensional phased-array echocardiographic 5-MHz transducer with pulsed Doppler capabilities (model 77020A Ultrasound System, Hewlett-Packard) positioned to allow for maximum inspection of the tricuspid valve. The Doppler sample volume was placed at the level of the tricuspid valve annulus. All pressure and Doppler data were recorded at a chart speed of 100 mm/sec. At the conclusion of pressure recording, a right ventriculogram was recorded in the right anterior oblique projection on 35-mm cine film at a rate of 30 frames/sec by injecting 25 ml nonionic contrast medium (Omnipaque 350, Winthrop) through a pigtail catheter (7F, Cordis) positioned in the RV apex through the left jugular vein.

All hemodynamic, flow velocity, and ventriculographic measurements were recorded during brief apnea with the animal in the supine position. Core body temperature was maintained with use of a circulating water heating pad, and was monitored by the Swan-Ganz catheter thermistor. In no case did the temperature drop below 36.0°C. Blood gases were measured repeatedly, and ventilator respiratory rate and volume adjusted accordingly.

Analysis of Data

The following pressure measurements were obtained from the micromanometric pressure transducer recordings: aortic systolic and diastolic pressure, peak RV apical pressure, minimum pressure in the RV apex and inflow and outflow tracts, time to reach minimum pressure after the first crossover of atrial and ventricular pressures in the three different RV cavitary regions, the maximum forward (RA pressure>RV pressure) tricuspid pressure gradient recorded between the RA transducer and the
three regionally placed transducers; the upslope of the rapid filling pressure wave after minimum pressure, and the area of reversed pressure (where RV pressure > RA pressure) between the second and third crossover points of atrial and ventricular pressures. Acceleration and deceleration rates of early transtricuspid flow were derived from Doppler time-velocity profiles by visual construction of a tangent with the use of a straightedge from three consecutive normal sinus beats.\(^5\)

Group data were analyzed with a one-way ANOVA. Differences between groups were detected with Fisher’s least-significant difference test.\(^6\) Significance level was set at \(p<0.05\). Data are expressed as mean±SD.

Results

Heart Rate and Aortic and Right Ventricular Pressures

Mean heart rate for the six animals was 83±10 beats/min. Mean aortic systolic and diastolic pressure was 115±20 and 80±10 mm Hg, respectively. For the six animals, mean peak pressure measured in the RV apex was 22±5 mm Hg. As has been previously described for the left,\(^7,8\) and right\(^9\) ventricles, systolic regional pressure gradients were also found to exist. The highest peak systolic pressure was found in the apex, consistently exceeding peak systolic pressure in the inflow tract by approximately 1 mm Hg. The peak systolic inflow tract pressure in turn consistently exceeded that of the outflow tract, also by about 1 mm Hg.

Regional Differences in Right Ventricular Early Diastolic Pressures

Figures 1A and 1B, showing sequential measurement of RA and RV apical pressures, and RA and RV inflow tract pressures, illustrate the characteristic pressure differences between these two regions during early diastole. As in the LV apex,\(^1,2\) the RV apical pressure recording is marked by a sharp, relatively deep decline in pressure during early diastole, followed by an early, sharp upturn, followed by the characteristic oscillatory “F wave.” Also observed is a reversal of the atrioventricular pressure gradient, where RVP exceeds RAP between the second \((X_2)\) and third \((X_3)\) crossover points of RA and RV pressures. This contrasts with the pressure signal recorded in the RV inflow tract, which is similar to the pressure pattern recorded in the LV basal region. The pressure signal in the RV inflow tract is characterized by a relatively shallow decline that reaches its nadir relatively late compared with the corresponding apical signal. Pressure then begins to gradually increase while exhibiting little or no oscillatory behavior and little or no pressure reversal.

In addition to the characteristic pressure waveform pattern recorded in the apical and inflow regions of both ventricles (Figure 2), we also consistently recorded a third characteristic pressure waveform in the RV outflow tract of all six dogs. This waveform featured a sharp, deep descent with early nadir similar to an apical recording but also featured a gentle upslope without oscillatory behavior similar to an inflow tract recording. Figure 3 shows a representative simultaneous recording of pressures in the RV apex and outflow tract. As can be seen, a lower minimum pressure is reached in the outflow tract than in the apex. This was the case in five of the six animals. In the sixth animal minimum pressure was equal in the two locations.

Regional and transtricuspid pressure differences between the inflow and outflow tracts and RV apex are presented quantitatively in Table 1. Significant differences were found between the regions during early diastole for time to reach minimum pressure after the first crossover of RA-RV pressures, the maximum early forward (RAP > RVP) transtricuspid pressure gradient, the upslope of the rapid filling
pressure wave after minimum pressure, and the area of the reversed (RVP>RAP) pressure gradient between the second and third points of RA-RV pressure crossover. All parameters except for minimum pressure during early diastole differed significantly between the regions. Although the ANOVA failed to indicate significant differences between the three RV regions for minimum early diastolic pressure, this probably relates to the relatively large standard deviations associated with this variable and to the conservative nature of the ANOVA. Thus, the lack of significance for this variable may well represent a type 2 error. Applying the more robust paired t test to the same data indicates highly significant differences in minimum pressure between all three groups.

**Regional Pressure Differences During the Atrial Filling Phase**

Pressure gradients were also observed in the RV during the atrial filling phase of diastole. Comparing Figures 1A and 1B, it can be observed that during atrial contraction the upward deflection of the RV apical a wave is seen to occur slightly later than the upslope of the RV inflow a wave. This difference is even more obvious in Figure 4. The simultaneous recording of pressures in the apex and inflow tracts clearly shows that the RV inflow pressure a wave precedes the apical pressure a wave.

As seen in Figure 3, the apical a and the outflow tract a wave generally begin to rise at approximately the same point in time. However, in some instances the outflow tract a wave appeared to rise slightly later than the apical a wave.

**Pulsed Doppler Indexes of Transtricuspid Flow**

Peak transtricuspid velocity for the six animals was 35±6 cm/sec. Early tricuspid flow acceleration (393±101 cm/sec²) was found to be greater than deceleration of flow (182±59 cm/sec²). This difference was significant at the 0.001 level (paired t test).

**Right Ventriculography**

Figure 5 shows representative RV end-diastolic and end-systolic cine film frames. A marked narrowing of the RV outflow tract is clearly evident at end systole. Some degree of outflow tract deformation at end systole was seen in all six animals.

**Discussion**

**Right Ventricular Apex-Inflow Tract Pressure Gradients**

The present study indicates that a pattern of diastolic apex to inflow pressure gradients, similar to that recorded in the LV, also exists in the RV during both early and late diastole. We have also shown that in the RV, unlike in the LV, the lowest early diastolic pressures are usually recorded in the outflow tract.

With the logic used in our previous paper, because early diastolic RV minimum pressure begins to rise first in the apex and last in the inflow tract, this implies that blood undergoes deceleration first in the apex and last in the inflow tract. Further, our observation that the RV inflow pressure a wave during late diastole rises first in the inflow tract and last in the apex...
suggests that during late diastole the ventricle is filled passively by blood transferred from the right atrium by atrial contraction. Thus, some finite time is required for the atrial filling wave to travel the length of the ventricular cavity. The fact that the regional pressure gradient pattern during early diastole is precisely opposite to the pattern recorded during late diastole suggests that the RV, like the LV, contributes to early diastolic filling by some mechanism other than passive filling. The form and timing of the regional ventricular pressure gradients during early diastole suggest that this other mechanism is the mechanical suction of blood into the ventricular cavity.

The present study also clearly demonstrates for the first time that reversed pressure gradients are present during the latter portion of rapid filling across the tricuspid valve. As we have observed elsewhere,5,10 because blood accelerated during early diastole returns to near zero velocity before systolic ejection, a force of comparable magnitude must be exerted to decelerate flow. Thus, reversed gradients are a physiological event that may be observed during deceleration of flow across all four cardiac valves.2-11-13

**Diastolic Right Ventricular Outflow Tract Pressure**

The low minimum pressure in the RV outflow tract is probably related to the existence of “restoring forces.” As recently speculated by Nicolic et al.,14 the occurrence of such restoring forces is “due to a ventricular shape change that occurs after sarcomere shortening has ended.” They noted that “this is most evident in the RV outflow tract, which is frequently seen to invaginate late in systole,” and speculated that the primary mechanism responsible for this deformation is the momentum associated with ventricular ejection. According to this concept, blood continues to leave the ventricle after muscle contraction has ceased, thereby causing the shape of the outflow tract to distort. This causes elastic energy to be stored in the myocardium, leading to recoil of the outflow tract walls during early diastole. This phenomenon would account for the low early diastolic pressures recorded during early diastole in this region.

In addition, storage of elastic energy in the RV outflow tract may also result from the active contraction of muscle fibers to lengths below equilibrium for that region of the heart.15 Parenthetically, the importance of active contraction of the RV outflow tract is exemplified by the presence of infundibular hypertrophy under conditions of pressure overload such as pulmonary valve stenosis.

Finally, a third possible mechanism contributing to the generation of low early diastolic pressure in the RV outflow tract may relate to the motion of the heart during systole. During contraction, as blood is propelled upward, the heart is propelled downward, stretching the great vessels.16 Subsequently, during early diastole, as the heart returns toward its pre-systolic position, the upward motion of the closed pulmonary valves may act to cause a local drop in pressure in the region behind the valve leaflets. Thus, it may be that factors other than the unique architecture of the RV outflow tract contribute to the generation of low early diastolic pressures in this region. This is consistent with recent measurements.
made in our laboratory in the canine LV using a dual sensor micromanometric catheter with 4 cm spacing between the sensors. In at least some dogs (three of nine), the proximal sensor, positioned in the LV just inside the aortic valve, recorded a minimum pressure as low as or lower than that recorded in the LV apex.

Although the largest relative RV-RA early diastolic pressure gradient is usually recorded in the outflow tract, because the dimensions of this region are small compared with the overall size of the RV, its contribution to RV filling may be minor. However, the presence of low early diastolic pressure in the outflow tract of the RV, in conjunction with substantial narrowing of this region at end systole, strongly supports the concept of restoring forces as a useful explanatory device for accounting for the magnitude of regional diastolic intraventricular pressure gradients in this region.

**Transtricuspid Pressure-Flow During Early Diastole**

Other investigators have demonstrated that in the normal heart the magnitude of indexes of transtricuspid flow measured by pulsed Doppler echocardiography are significantly less than similar measurements of flow made across the mitral valve. The present study confirms this observation. As shown in Figure 6A, early diastolic flow across the tricuspid valve is characterized by relatively slow acceleration, low peak velocity, and a slow rate of deceleration. Early flow across the mitral valve, as demonstrated by Figure 6B, is characterized by rapid acceleration, high peak velocity, and rapid deceleration. As shown in Figure 7, indexes of peak flow, acceleration, and deceleration of early tricuspid flow are significantly less than similarly obtained indexes of mitral flow measured in our laboratory. In addition, the present study indicates that the magnitude of the pressure gradients driving transtricuspid flow is also significantly less than the magnitude of those driving transmitral flow. This is demonstrated in Figure 8, where the maximum pressure gradients recorded across the tricuspid valve during early diastole in the six animals are compared with gradients recorded in anatomically similar positions across the mitral valve in 11 animals from our laboratory. On average, for any given intracavitary position, the atrioventricular pressure gradients on the left side of the heart exceed gradients recorded on the right side by greater than two to one. These relatively small pressure gradients across the tricuspid valve are consistent with the lower acceleration and peak velocity of tricuspid flow compared with mitral flow. Although the above measurements of transmitral and transtricuspid velocities and pressure gradients were obtained in different groups of animals, the experimental protocols used in each study were essentially identical. Because no significant differences existed between the groups in terms of heart rate or mean arterial pressure, we are confident that the reported differences are reliable. In addition, because it is well known that tricuspid valve area exceeds mitral valve area and because for any extended interval of time RV inflow approximates LV inflow, particularly under the stable condition of end-expiratory apnea, flow velocity must be less across the tricuspid valve than across the mitral.

As discussed above, we have shown that deceleration of ventricular early diastolic flow is temporally related to the reversal of atrial and ventricular pressures. Because the velocity of early diastolic flow across the tricuspid valve is much slower than across the mitral valve, it would follow that the magnitude of the reversed pressure gradient needed to decelerate
FIGURE 6. Upper panel: Record of tricuspid flow recorded by transesophageal Doppler echocardiography. Early diastolic flow (E) across the tricuspid valve is characterized by relatively slow acceleration, low peak velocity, and a slow rate of deceleration. Lower panel: Record of mitral flow recorded by transesophageal Doppler echocardiography. Early diastolic flow across the mitral valve is characterized by relatively fast acceleration, high peak velocity, and a rapid rate of deceleration. A, atrial contraction flow; ECG, electrocardiogram; RVP, right ventricular pressure.
this flow would also be less on the right side of the heart. Comparison of the area of the reversed gradient between the second and third crossover points of atrial and ventricular pressures from the present study

with that of corresponding left-sided data from a previously published study from our laboratory confirms this difference (72±43 mm Hg · msec measured from RA to RV apex compared with 101±41 mm Hg · msec measured from LA to LV apex; p<0.05). That early diastolic tricuspid flow deceleration is slower than mitral flow deceleration may be related to the thinner-walled RV being a less effective decelerator than the thicker-walled LV.

A further potential difference between the intraventricular pressure gradient patterns in the RV and LV occurs during the isovolumic contraction and relaxation phases. In two animals from the present study simultaneous pressure recordings in the RV outflow tract and apex demonstrated a delay in the pressure upslope during the isovolumic contraction phase. Pressure was seen to rise first in the apex and last in the outflow tract. In three of the six animals a pressure gradient was noted during the isovolumic relaxation period between simultaneously recorded outflow tract and apical pressures. During this phase, apical pressure is seen to cross over outflow tract pressure at the start of rapid pressure decline. Apical pressure then continues to fall rapidly, preceding the fall in outflow tract pressure. This pressure difference remains until apical and outflow tract pressures cross again at approximately the time of tricuspid valve opening (Figure 9). These data suggest that in some animals the outflow tract may function as a separate chamber, having contraction and relaxation characteristics that differ from those of the remainder of the ventricular cavity. Using a dual sensor microma-

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nometric catheter with 4 cm spacing between the sensors, we have thus far been able to document similar delays in apex to outflow tract pressure during isovolumic contraction and relaxation in the normally contracting LV.

Factors Affecting RV Intraventricular Pressure Gradients

We have previously shown in the LV that the magnitude of the early diastolic apical intracavitary pressure gradient is related to global systolic function, and that extensive anterior myocardial ischemia is associated with attenuation, loss, or possibly even reversal of this regional pressure gradient. It is thus reasonable to assume that the RV early diastolic intraventricular pressure gradients are also related to global RV systolic function and may be significantly altered in the setting of ischemia. Interventions that have been shown to differentially alter regional RV contractile temporal sequences, extent of shortening, and intramyocardial and intracavitary pressures would also be likely to affect the absolute early diastolic pressures in, and the relative early diastolic pressure differences between, the RV conus and sinus regions.

Implications for RV Function

The present study suggests that the RV is capable of producing a suction effect that actively draws in blood during early diastole, a finding that may have important implications for studies concerning RV function. For example, early diastolic shifts of the interventricular septum (IVS) have been reported in a number of cardiac pathological conditions. The mechanism for these shifts has generally been attributed to imbalances between right and left ventricular filling. In the normally functioning heart during early diastole the IVS is convex with respect to the right ventricular cavity. It might be speculated that the ability of the RV to generate low pressure during early diastole by the mechanism of recoil is an important factor in maintaining this normal septal configuration. Thus, early diastolic displacement of the IVS—resulting from RV volume overload, extensive RV ischemia, or RV hypertrophy secondary to primary pulmonary hypertension, where RV recoil may be depressed as a result of increased end-diastolic volume or depressed relaxation rate—may occur, in part, because of the loss of the capacity of the RV to create low pressures during early diastole. Thus, under these conditions, the RV contribution to maintaining normal early diastolic IVS curvature may be reduced or absent, and this may contribute to a shift of the IVS toward the LV cavity. This shift of the septum toward the LV during early diastole would serve to augment RV filling, and return of the septum to a relatively normal configuration during LV systole would serve to augment RV ejection. This pistonlike action of the IVS, drawing blood into the RV as it is displaced toward the LV during diastole, and ejecting it as it moves "paradoxically" during LV systole, may be an important mechanism accounting for, in part, the lack of observable change in right ventricular systolic function reported after massive destruction of the RV free wall.

Consistent with the above speculation are data indicating that in cases of severe mitral stenosis, a condition that markedly lowers LV early diastolic pressure because of LV recoil against restricted inflow, the IVS is shifted toward the LV during early diastole. Thus, despite relatively normal RV function, the presence of unusually low LV early diastolic pressure would also result in the early diastolic shift of the IVS. Similarly, Yellin et al noted in a recent

![Figure 9. Record in one dog of simultaneous right ventricular (RV) apex and outflow tract pressures obtained with micromanometric pressure sensors. A delay is present in the pressure upslope during the isovolumic contraction phase. Pressure rises first in the apex and last in the outflow tract. This phenomenon was observed in two of the six animals. During the isovolumic relaxation phase, the apical pressure was seen in three of six animals to cross over outflow tract pressure at the start of rapid pressure decline (upper arrow). Apical pressure then continues to fall rapidly, preceding the fall in outflow tract pressure. This pressure difference remains until apical and outflow tract pressures cross again at approximately the time of tricuspid valve opening (lower arrow).](http://circ.ahajournals.org/content/1421/10/1421/F9.large.jpg)
review that data obtained using an experimental model that completely blocks early mitral flow often resulted in an increase in RV stroke volume in the three beats after mitral occlusion, despite increased RV afterload. These investigators emphasized the role of ventricular suction and its effect on displacement of the IVS as the probable mechanism. Thus, it is interesting to speculate that the relative magnitudes of early diastolic suction produced by the two ventricles may be an additional factor contributing to ventricular interdependence effects.

Conclusion

The present study demonstrates that patterns of intraventricular diastolic pressure gradients between apex and inflow exist in the RV similar to those recorded in the LV during both early and late diastole. In the RV, unlike in the LV, the lowest diastolic pressures are usually recorded in the outflow tract, and end-systolic deformation of the walls of the outflow tract is the likely mechanism for the production of low early diastolic pressure in this region. We have also demonstrated the presence of a reversed transmitial diastolic pressure gradient during deceleration of early RV diastolic flow and have shown that the magnitudes of the atrial and ventricular pressure gradients and flow across the tricuspid valve are significantly less than similar measurements recorded across the mitral valve. Consideration of these physiological patterns of RV diastolic intraventricular pressure and their relation to filling has important implications with regard to the derivation of formulas and the development of indexes that characterize diastolic pressure-flow relations and provides physiological insight relating to the location of ventricular restoring forces.

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References


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