Assessment of right ventricular diastolic suction in dogs with the use of wave intensity analysis

Yichun Sun, Israel Belenkie, Jiun-Jr Wang, and John V. Tyberg

Departments of Cardiac Sciences and Physiology and Biophysics and The Libin Cardiovascular Institute of Alberta, University of Calgary, Calgary, Alberta, Canada

Submitted 10 August 2005; accepted in final form 6 July 2006

The purpose of this study was to measure the energy associated with RV relaxation (IW−) and to test the hypothesis that IW− depends on both the rate at which elastance decreases (τr, an index of the exponential time constant of RV relaxation) and the completeness of RV emptying [RV end-systolic volume (VRVES)]. In particular, the energy associated with RV DS was identified as that component of IW− that remains after the opening of the tricuspid valve [IW−(DS)].

METHODS

Experimental preparation and protocol. The experimental protocol was approved by the Institutional Animal Care Committee and met the standards of the American Physiological Society. Anesthesia was initiated with 10–15 mg/kg iv thiopental sodium in six mongrel dogs weighing 23 ± 5 (SD) kg, which were then intubated and ventilated (70% nitrous oxide-30% oxygen mixture) with a constant-volume ventilator (model 607; Harvard Apparatus, Natick, MA) using a tidal volume of 17–23 ml/kg at 15–20 cycles/min, adjusted to maintain normal blood gases and pH (9.25). A catheter was introduced into the femoral vein for administration of drugs. Fentanyl citrate (50–100 µg/kg) was administered initially over 5 min in 50 ml of normal saline, and anesthesia was maintained with an intravenous infusion of 20–50 µg·kg−1·h−1, adjusted as necessary. The efficacy of anesthesia was monitored by blood pressure, heart rate, and body reflexes. A single ECG lead was used to monitor the heart rate. Body temperature was maintained at 37°C with a warming blanket and heat lamp. A large-bore catheter in the external jugular vein was used to infuse a 2% albumin-normal saline solution and to remove blood for adjustment of intravascular volume.

The animals were instrumented through a midline sternotomy (14, 16). The pericardium was incised from base to apex, along the interventricular sulcus. Sonomicrometry crystals (Sonometrics, London, ON, Canada) were implanted on the RV endocardium to estimate RV volume (37). RV pressure was measured with an 8-F micromanometer-tipped catheter with a reference lumen (model SPC-471; Millar Instruments, Houston, TX) introduced through the RV free wall. Pulmonary arterial (PA) and right atrial (RA) pressures were measured with 3-F micromanometer-tipped catheters (model SPR-524; Millar Instruments) introduced retrogradely through a small pulmonary artery branch and a jugular vein, respectively. Aortic pressure was measured with an 8-F fluid-filled catheter (Cordis, Miami, FL) introduced through a femoral artery. Percardial pressure was measured with a flat liquid-containing balloon loosely sutured to the surface of the mid-RV free wall (16, 39). PA flow was measured with an ultrasonic flowmeter (Transonic Systems, Ithaca, NY) implanted around the main PA. A pneumatic cuff/constrictor (In Vivo Metrics, Healdsburg, CA) was implanted around the inferior vena cava (IVC) above the diaphragm. Isoproterenol and esmolol were administered through a short polyvinyl tube into the jugular vein.

THE UNIQUE CAPABILITIES of wave intensity analysis (WIA) could provide information regarding the dynamics and mechanisms of early right ventricular (RV) filling. Conceptually, diastolic suction (DS) is that property of the ventricle by which it tends to refill itself during early diastole, independent of any force from the atrium. Two apparently different types of mechanistic explanations have emerged. One is decreasing elastance; the other is the degree to which the ventricle empties (i.e., end-systolic volume) (11, 12, 19, 38, 42). Sabbah and colleagues (31, 32) indicated that RV early diastolic pressure is negative in both humans and dogs (perhaps reflecting RV DS) and that the RV can create a sucking effect during early diastole that may contribute to the filling process. Recently, our group clarified the mechanisms of left ventricular (LV) DS using WIA (41).

Address for reprint requests and other correspondence: J. V. Tyberg, Univ. of Calgary Health Sciences Centre, 3330 Hospital Dr. NW, Calgary, Alberta, Canada T2N 4N1 (e-mail: jtyberg@ucalgary.ca).
using a variable-speed pump (Harvard Apparatus). The heart was returned to the pericardial sac, and its margins were loosely reapproximated with individual sutures, with care taken not to compromise pericardial volume (35). Tricuspid blood flow velocity was measured by Doppler echocardiography (Sonos 5500; Agilent Technologies, Andover, MA) with a handheld 5-MHz transesophageal probe applied gently on the surface of the heart; an off-axis view was used to obtain optimal flow-velocity profiles with minimal spectral dispersion. We also measured the time between pulmonary valve closure and tricuspid valve opening in three experiments using M-mode echocardiography.

The rationale of our protocol was to acquire a “two-dimensional” matrix of data by systematically manipulating two independent variables, \( \tau \) and RV end-diastolic pressure (PEVED, an experimental surrogate for manipulating VrVES), over wide ranges. Starting at \( \tau = 30 \) ms and PEVED = 5 mmHg, to determine the value of the zero-pressure intercept (Vd), we constricted the IVC transiently with a pneumatic cuff until PEVED decreased to approximately zero. We then increased PEVED to 10 mmHg and, finally, to 15 mmHg by infusing volume. (By withdrawing blood, we lowered PEVED back to 5 mmHg.) To decrease \( \tau \) (~15 ms), we then began an infusion of isoproterenol (0.1 \( \mu \)g·kg\(^{-1}\)·min\(^{-1}\)) and repeated the PEVED manipulation. Finally, to increase \( \tau \) (~45 ms), we began an infusion of esmolol (0.1 mg·kg\(^{-1}\)·min\(^{-1}\)) and repeated the PEVED manipulation once more. These procedures provided a range of \( \tau \) and VrVES values, thus providing a matrix of data. All the data were collected with the ventilator stopped in the end-expiratory position. Each data-acquisition period lasted ~30 s. Between each intervention, time was allowed for hemodynamic stability to be reestablished. Just before the end of the experiment, PA circumference was measured by passing a fine suture around it twice and tying it snugly, to calculate the cross-sectional area of the PA.

**Data recording and analysis.** Hemodynamic and sonomicrometry data were conditioned via a multichannel recorder (model VR16m; Electronics for Medicine/Honeywell, Pleasantville, NY). Analog signals were passed through antialiasing, low-pass filters with a cutoff frequency of 100 Hz and were then sampled at a frequency of 200 Hz using acquisition software (Sonometrics). Doppler echocardiographic data were recorded on a videocassette recorder and were captured using graphic software (Ulead 5.0; Ulead Systems, Taipei, Taiwan); these data were later digitized using Matlab 6.1 (The MathWorks, Natick, MA). The hemodynamic data were subsequently analyzed with locally developed specialized software (CVWorks; Advanced Measurements, Calgary, AB, Canada). End diastole was defined as the minimum RV pressure following the “a” wave on the RV pressure tracing. End systole was defined as the minimum value of RV dP/dt. A locally built frame counter was used to match the Doppler frames with the simultaneously acquired hemodynamic data.

As discussed below, the duration of RV isovolumic relaxation was very short or negligible, so a conventional exponential time constant (\( \tau \)) could not be determined reliably. Consequently and completely empirically, we took all the data between peak RV systolic pressure and the estimated time of tricuspid valve opening (as guided by the onset of tricuspid flow and the RA-RV pressure crossover) and fit them to the following equation to obtain an index of relaxation, \( \tau \): \[ P(t) = P_{rVES} \cdot e^{-t/\tau} + P_s, \]

where \( t \) is the time from peak systole and \( P_s \) is the theoretical asymptotic value of RV pressure when \( t \rightarrow \infty \).

RV volume was calculated using the multiple tetrahedron method (37). To compare data from different experiments, we normalized VrVES by the RV end-diastolic volume (VrVED) when PEVED was equal to 5 mmHg:

\[ V_{rVES}(\%) = \left( \frac{V_{rVES}}{V_{rVED}} \right) \times 100 \]

For each dog, the tetrahedron VRVED vs. different levels of PEVED were plotted (Fig. 1); from the linear regression equation, the tetrahedron VRVED at PEVED = 5 mmHg could be calculated.

Wave speed was calculated continuously throughout the cardiac cycle using the following equation (37):

\[ c = 0.0089 \cdot E^{0.31} \]

where \( E = P/(V - V_d) \), where \( P \) and \( V \) are RV pressure and volume and \( V_d \) is the extrapolated RV volume when RV pressure is equal to zero. We compared these values with that estimated as \( c = dP/dU \) (23), particularly examining the value at the beginning of RV filling, when reflections can be expected to be minimal, and found excellent agreement. We plotted \( c = dP/dU \) vs. \( c = 0.0089 \cdot E^{0.31} \) and performed linear regression; the slopes equaled 1.02 ± 0.5 (SE), and the relations were linear as indicated by the fact that \( r^2 \) values exceeded 0.85 for each dog.

The intensity of the RV relaxation-related wave was calculated using the formula

\[ d_{W} = \frac{-dP}{dU} \left( \frac{4pc}{1-c} \right) \]

where \( p \) is the density of blood (kg/m\(^3\)), \( c \) is the wave speed (m/s), \( dP \) is the incremental difference in RV pressure (1 mmHg = 133 N/m\(^2\)), and \( dU \) is the difference in tricuspid velocity (m/s) during a 5-ms sampling interval (24).

For this and our group’s previous LV DS study (41), to simplify the analysis of RV relaxation-related phenomena, we adopted the convention that RV inflow velocities (i.e., the tricuspid E and A waves) should be given positive values and RV outflow velocity (i.e., PA flow divided by estimated cross-sectional area) should be given a negative value (see Fig. 2). By this convention, the RV-generated waves propagating into the PA are seen as backward waves. Thus the \( d_{W} \) waveform (W/m\(^2\), power per unit cross-sectional area) described the instantaneous “aspirating power” associated with RV relaxation (42). [As discussed in greater detail previously (41), Wiggers suggested that an “aspirating force” was generated throughout all of ventricular relaxation; to represent the dynamics of that entire interval most straightforwardly, we adopted the convention whereby systolic events also appear with a negative sign indicating a backward-going wave.] We calculated the total time integral under (i.e., between the waveform and zero) the \( d_{W} \) waveform (I\(_{W\rightarrow D_{S}}\) (J/m\(^2\)), energy per unit cross-sectional area) to calculate total “aspirating energy” (42). To measure DS, we also calculated that portion of the total area described after the beginning of tricuspid flow (I\(_{W\rightarrow D_{S}}\) (J/m\(^2\))). I\(_{W\rightarrow D_{S}}\) and I\(_{W\rightarrow D_{S}}\) were plotted against \( \tau \) and/or VrVES (%). Linear regression was used to determine the percentage relation of I\(_{W\rightarrow D_{S}}\) to I\(_{W\rightarrow D_{S}}\).
RESULTS

Figure 2 shows WIA evaluation of one typical cardiac cycle. The top panel shows PA, RV, and RA pressures and PA (calculated from flow) and tricuspid (Doppler) velocities. The bottom panel shows the intensity of forward (dI\textsubscript{W\textendash}) and backward-going (dI\textsubscript{W\textendash}) waves. During relaxation, the RV generated a dominant backward expansion wave that began the moment RV pressure began to decrease and then increased (in absolute value) rapidly and reached a peak when RV dP/dt reached its greatest negative value. dI\textsubscript{W\textendash} decreased during isovolumic relaxation and early diastolic filling. This backward expansion wave ended when dI\textsubscript{W\textendash} reached zero. The total time integral under the dI\textsubscript{W\textendash} waveform represented the total aspirating energy (42). Most of the total energy of the expansion wave was dissipated before the tricuspid valve opened; after it opened, only \textasciitilde10\% (see below) remained to accelerate the tricuspid column of blood.

Figure 3 shows typical tracings obtained during a representative cardiac cycle and demonstrates that RV volume reached a minimum and began to increase before PA flow reached zero, indicating that the RV dimensions began to increase before PA flow stopped. We observed that the isovolumic relaxation interval was negligible in most experiments; in some, the tricuspid valve opened immediately after the pulmonary valve closed (see Fig. 3), and in other experiments, the tricuspid valve opened even before the pulmonary valve closed. The maximum interval between closure of the pulmonary valve and the opening of the tricuspid valve (estimated by M-mode echocardiography) was 14 ms.

Our analysis indicates that \(\tau\) and \(V\text{\textsubscript{RVES}}\) each independently predicted DS (I\textsubscript{W\textendash}). Analysis of each dog's data (Fig. 4), in addition to analysis of the pooled data (Fig. 5), shows an inverse relationship between \(\tau\) and I\textsubscript{W\textendash}: as \(\tau\) decreased, I\textsubscript{W\textendash} increased. We used a three-parameter exponential decay equation [I\textsubscript{W\textendash} = \(a\ \text{e}^{b\tau} + (I\textsubscript{W\textendash})_o \text{e}^{-c\tau}\)]. A similar inverse relationship [I\textsubscript{W\textendash} = \(a\ \text{e}^{b\%\text{RVES}} + (I\textsubscript{W\textendash})_o \text{e}^{-c\%\text{RVES}}\)] was found between I\textsubscript{W\textendash} and \%RVES: as \%RVES decreased, I\textsubscript{W\textendash} increased (Figs. 6 and 7). The summary of the parameters of the two equations is shown in Table 1. The standard errors of \(b\) did not include zero for either of the two equations, indicating that the exponential term was significant.

Our analysis also demonstrated that, together, \(\tau\) and \(V\text{\textsubscript{RVES}}\) predicted I\textsubscript{W\textendash} better than either did individually. A three-dimensional mesh plot shows the nonlinear relations between I\textsubscript{W\textendash} with \(\tau\) and \(V\text{\textsubscript{RVES}}\) in Fig. 8. The equation was

\[
I_{\text{W\textendash}} = -8.85 \times \text{e}^{-0.042\tau} - 0.067\%\text{RVES}.
\]

After the tricuspid valve opened, \textasciitilde10\% of I\textsubscript{W\textendash} [i.e., 10\% was the slope of the pooled data plot of I\textsubscript{W\textendash\textsubscript{DS}}} vs. I\textsubscript{W\textendash}; \(r^2 = 0.58\) ]
the total aspirating energy, remained to accelerate early diastolic tricuspid flow. In each individual experiment, I\textsubscript{W\textendash\textsubscript{DS}}}
was proportional to $I_{W-}$; slopes ranged from 6 to 27% and $r^2$ values, from 0.53 to 0.95.

**DISCUSSION**

Using WIA, we have shown that the energy of the expansion wave generated by the RV during early diastole is inversely related to both the relaxation rate and $V_{RVES}$, reconciling the two apparently unrelated mechanisms of DS that have been proposed previously (11, 12, 19, 22, 38, 41, 42). The total time integral under the $dI_{W-}$ waveform represents the total aspirating energy that decelerates, stops, and reverses the column of blood in the RV outflow tract and then accelerates RV inflow through the tricuspid valve (42).

The relations between $dI_{W-}$ vs. $\tau'$ and $V_{RVES}$ data appeared to be nonlinear, so we used three-parameter exponential decay equations to model these relations. From a physiological point of view, fitting the data with an exponential equation seemed more appropriate than a linear equation. With linear regression, extrapolation suggests that $I_{W-}$ would change its sign at high values of $\tau'$ or $V_{RVES}$, and this seemed illogical a priori. Conversely, extrapolation of an exponential decay relationship suggested that $I_{W-}$ would plateau and reach asymptotic levels at high values of $\tau'$ or $V_{RVES}$.

DS is related to decreasing elastance. Pressure decreases during rapid inflow while the still-relaxing ventricle is filling, which indicates that elastance is decreasing and the ventricle is relaxing faster than it can fill (19, 33, 42). Our results also indicate that DS is related to $V_{RVES}$. Since RV transmural pressure may be negative at small volumes during diastole (31,
(32), this implies that the RV will tend to refill itself until transmural pressure is zero and that the smaller the end-systolic volume, the greater the DS. The concept of an equilibrium volume (8) or elastic recoil (4, 7) implies that restoring forces in the ventricle promote filling when RV volume is less than the equilibrium volume. Thus factors that augment RV emptying will tend to increase the gradient for filling during the subsequent diastole (40). It has been suggested the myocardial fibers of different layers pull against one another and stretch the connections between them, exerting a force that Rushmer called “interfascicular tension” (30). This would create potential energy that might facilitate rapid filling. These restoring forces also may be related to distortion of the intramural structure of the wall or may be derived in part from compression of the sarcomeres (5, 17). The relationship between RV suction and end-systolic volume also may be due, in part, to septal effects (1, 3), which could be important in responding to the demands of increased contractility. Thus, despite the nonspherical shape of the RV, where recoil might not be expected to be important, we nevertheless observed significant DS. Presumably, these mechanisms are advantageous during exercise, when tachycardia limits RV filling time but when contractility is increased. Increased contractility not only increases the rate of relaxation (i.e., decreases $\tau'$) but decreases end-systolic volume (10).

The backward expansion wave that is responsible for RV DS, the energy of which we calculate as $I_{W-\text{(DS)}}$, begins when the tricuspid valve opens and ends when RV pressure reaches a minimum value. This agrees totally and precisely with Katz’s conclusion that the ventricle tends to fill itself until the nadir in pressure (19). During this time, the ventricle is relaxing faster than it can fill (its elastance is still decreasing) and so is responsible for its own filling. After tricuspid valve opening, only a small portion (~10%) of the backward expansion wave remains to accelerate the tricuspid column of blood, a value similar to that for the LV (41). This also is consistent with Wiggers’s anticipation that although the aspirating force aids ventricular filling, only a small fraction of that force remains available for diastolic filling because ventricle pressure approaches its nadir before the valve opens (42). $I_{W-\text{(DS)}}$ is a fraction of $I_{W-}$; accordingly, it can be anticipated that $I_{W-\text{(DS)}}$ will behave in the same manner as $I_{W-}$. $I_{W-\text{(DS)}}$ was found to be dependent on both $\tau'$ and $\%V_{RVES}$, suggesting that tricuspid filling was augmented when $\tau'$ or $V_{RVES}$ were decreased (Fig. 9).

We observed that the duration of RV isovolumic relaxation was negligible in most experiments (Fig. 3); in some, the tricuspid valve opened immediately after the pulmonary valve closed, and in other experiments, the tricuspid valve opened before the pulmonary valve closed (the maximum isovolumic interval we observed was 14 ms). This is consistent with the observations by Myhre et al. (21), who found that peak negative RV dP/dt occurs an average of 60 ms before end ejection and that there is no RV isovolumic relaxation period. In humans, RV isovolumic relaxation is shorter than that in the LV: the pulmonary valve closes after the aortic valve (6, 34, 36) and the tricuspid valve opens before the mitral valve (15). Our results are consistent with the “hangout phenomenon” (measured by the time between the dicrotic notch values of RV and PA pressures), which is believed to occur because PA pressure is low and blood can flow easily into the pulmonary vascular bed because of its low resistance and high capacitance (36). This allows RV ejection to continue after LV ejection has stopped.

With respect to the observations of Myhre et al. (21), Pasipoularides et al. (28) suggested that the RV isovolumic relaxation period appeared negligible only because those dogs were anesthetized and their chests were open. Citing previous observations on LV relaxation (18), they noted that halothane anesthesia increased ejection time and decreased systolic...
stress, thus tending to shorten isovolumic relaxation, and suggested that these effects would be similar on the right side. We have not studied unanesthetized intact dogs and so cannot absolutely exclude this possibility. On the other hand, in the human RV data shown by Shaver et al. (36) (their Fig. 3), the pulmonic component of the second sound (simultaneous with the PA dicrotic notch) occurred only after RV pressure had declined nearly to its minimum value, approximately at the level at which the tricuspid valve would have opened. However, it must also be noted that Condos et al. (13) reported that, as registered by their catheter-mounted electromagnetic velocity sensor, human PA flow stopped before the dicrotic notch.

In principle, pressure and velocity should be recorded from the same cross section to calculate wave intensity. RV pressure was measured as close to the tricuspid orifice as possible and, therefore, a short distance from the pulmonary valve. Thus using this pressure to calculate wave intensity during systolic ejection may be slightly inaccurate.

The applicability of one-dimensional WIA to RV diastolic filling is more problematic than it is in large arteries or the LV (20). However, flow through the tricuspid valve can be described as a one-dimensional wave system. Tricuspid velocity was measured near the midpoint of the tricuspid annulus using Doppler echocardiography. This velocity should be representative of the entire cross section in that the velocity profile is blunt in early diastole (26, 27).

Data are regression values ± SE of the estimate. $I_w$, energy of backward extension wave; $\tau^r$, index of right ventricular relaxation; $\%VRVES$, normalized right ventricular end-systolic volume.

### Table 1. Nonlinear regression parameters

<table>
<thead>
<tr>
<th>Dog</th>
<th>$a$, J/m²</th>
<th>$b$, ms⁻¹</th>
<th>$(I_w-\tau^r)$, J/m²</th>
<th>$r^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>-7.5±5.4</td>
<td>0.07±0.05</td>
<td>-0.03±0.62</td>
<td>0.62</td>
</tr>
<tr>
<td>2</td>
<td>-32.7±19.5</td>
<td>0.12±0.04</td>
<td>-0.34±0.52</td>
<td>0.86</td>
</tr>
<tr>
<td>3</td>
<td>-37.1±43.1</td>
<td>0.09±0.06</td>
<td>-0.11±1.25</td>
<td>0.64</td>
</tr>
<tr>
<td>4</td>
<td>-1535.3±615.6</td>
<td>0.23±0.02</td>
<td>-0.44±0.01</td>
<td>0.97</td>
</tr>
<tr>
<td>5</td>
<td>-6.8±1.0</td>
<td>0.06±0.01</td>
<td>-0.2±0.13</td>
<td>0.87</td>
</tr>
<tr>
<td>6</td>
<td>-256.4±81.2</td>
<td>0.22±0.02</td>
<td>-0.67±0.06</td>
<td>0.90</td>
</tr>
<tr>
<td>Pooled data</td>
<td>-19.3</td>
<td>0.09</td>
<td>-0.20</td>
<td>0.51</td>
</tr>
</tbody>
</table>

$I_w = ae^{b\tau^r} + (I_w-\tau^r)$

<table>
<thead>
<tr>
<th>Dog</th>
<th>$a$, J/m²</th>
<th>$b$, %⁻¹</th>
<th>$(I_w-%VRVES)$, J/m²</th>
<th>$r^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>-34.5±62.5</td>
<td>0.08±0.06</td>
<td>0.09±0.79</td>
<td>0.54</td>
</tr>
<tr>
<td>2</td>
<td>-86.9±178.0</td>
<td>0.09±0.06</td>
<td>-0.12±0.99</td>
<td>0.67</td>
</tr>
<tr>
<td>3</td>
<td>-30.5±8.2</td>
<td>0.04±0.02</td>
<td>3.16±2.58</td>
<td>0.82</td>
</tr>
<tr>
<td>4</td>
<td>-1107.4±965.5</td>
<td>0.17±0.03</td>
<td>-0.16±0.34</td>
<td>0.81</td>
</tr>
<tr>
<td>5</td>
<td>-10.1±4.6</td>
<td>0.05±0.01</td>
<td>-0.41±0.2</td>
<td>0.72</td>
</tr>
<tr>
<td>6</td>
<td>-389.1±397.2</td>
<td>0.15±0.03</td>
<td>-0.63±0.14</td>
<td>0.73</td>
</tr>
<tr>
<td>Pooled data</td>
<td>-482.1</td>
<td>0.15</td>
<td>-0.68</td>
<td>0.59</td>
</tr>
</tbody>
</table>

$I_w = ae^{b(\%VRVES)} + (I_w-\%VRVES)$

Fig. 8. Three-dimensional mesh plot showing the nonlinear relations between $I_w$ with $\tau^r$ and normalized $VRVES$ ($\%VRVES$). $I_w = -8.85\cdot e^{-0.042\tau^r} + 0.067(\%VRVES)$; $r^2 = 0.61$. Data are pooled from 6 dogs.

Fig. 9. Top: energy of the backward extension wave after opening of the tricuspid valve ($I_w-\text{DS}$) plotted against an index of RV relaxation ($\tau^r$). Data are pooled from 6 dogs. Bottom: energy of the backward extension wave after opening of the tricuspid valve ($I_w-\text{DS}$) plotted against normalized $VRVES$ ($\%VRVES$). Data are pooled from 6 dogs.
The two variables studied, $\tau'$ and $V_{RVES}$, are not ideally independent. Isoproterenol increased cardiac contractility and markedly increased cardiac output; both $\tau'$ and $V_{RVES}$ decreased. Esmolol, which decreases cardiac contractility, increased both $\tau'$ and $V_{RVES}$. Volume infusion increased venous pressure and ventricular preload, which increased cardiac output (2), and both $V_{RVES}$ and $\tau'$ increased. These reasons may partially explain why variance was reduced when both $\tau'$ and $V_{RVES}$ were related to $dI_W-$ (Fig. 8) compared with either one alone (Figs. 5 and 7).

It would have been desirable to know the value of RV equilibrium volume (the RV volume when RV transmural pressure is equal to zero) in that we would have used the equilibrium volume to relate DS to the presumably sigmoidal nature of the diastolic transmural pressure-volume relation (7). However, because the RV diastolic pressure-volume relation is nearly flat, small errors in measuring RV and pericardial pressures precluded a confident estimation of the equilibrium volume.

Measurement of velocity of blood flowing through the tricuspid valve using Doppler echocardiography is angle dependent. The angle cannot be known precisely, and it could change during different conditions and at different times. In particular, during volume loading, the RV expanded, which may have resulted in changes in the orientation of the ultrasound transducer. Before each measurement, we adjusted the angle of the transducer and the position of the sample volume to maximize the flow-velocity profile and minimize this error.

In conclusion, $I_W-$, the wave energy associated with RV relaxation, is inversely related to an index of relaxation ($\tau'$, a measure of the rate of decrease of elastance) and the completeness of RV emptying ($V_{RVES}$): $I_W-(DS)$, which represents RV diastolic suction, was ~10% of $I_W-$, despite its thin wall and nonspherical shape, diastolic suction appears to contribute to RV filling.

ACKNOWLEDGMENTS

We acknowledge the excellent technical support provided by Cheryl Meek and Rozsa Sas. J. V. Tyberg is a Medical Scientist of the Alberta Heritage Foundation for Medical Research (Edmonton).

GRANTS

This study was supported by Grant-in-Aid 022961 from the Canadian Institution for Health Region of Ottawa (to J. V. Tyberg).

REFERENCES


42. Wiggers CJ. Cardiac mechanisms that limit operation of ventricular suction. Science 126: 1237, 1957.