Deceleration time of systolic pulmonary venous flow: a new clinical marker of left atrial pressure and compliance

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Hunderi, J. O., C. R. Thompson, and O. A. Smiseth. Deceleration time of systolic pulmonary venous flow: a new clinical marker of left atrial pressure and compliance. J Appl Physiol 100: 685–689, 2006. First published October 20, 2005; doi:10.1152/japplphysiol.00705.2005.—The curvilinearity of the atrial pressure-volume curve implies that atrial compliance decreases progressively with increasing left atrial (LA) pressure (LAP). We predicted that reduced LA compliance leads to more rapid deceleration of systolic pulmonary venous (PV) flow. With this rationale, we investigated whether the deceleration time (t_{dec}) of PV systolic flow velocity reflects mean LAP. In eight patients during coronary surgery, before extracorporeal circulation, PV flow by ultrasonic transit time and invasive LAP were recorded during stepwise volume loading. The t_{dec} was calculated using two methods: by drawing a tangent through peak deceleration and by drawing a line from peak systolic flow through the nadir between the systolic and early diastolic flow waves. LA compliance was calculated as the systolic PV flow integral divided by LAP increment. Volume loading increased mean LAP from 11 ± 3 to 20 ± 5 mmHg (P < 0.001) (n = 40), reduced LA compliance from 1.16 ± 0.42 to 0.72 ± 0.40 ml/mmHg (P < 0.004) (n = 40), and reduced t_{dec} from 320 ± 50 to 170 ± 40 ms (P < 0.0005) (n = 40). Mean LAP correlated well with t_{dec} (r = 0.84, P < 0.0005) (n = 40) and LA compliance (r = 0.79, P < 0.0005) (n = 40). Elevated LAP caused a decrease in LA compliance and therefore more rapid deceleration of systolic PV flow. The t_{dec} has potential to become a semiquantitative marker of LAP and an index of LA passive elastic properties.

left atrial compliance; left ventricular end-diastolic pressure; pulmonary venous flow

HEART FAILURE IS A MAJOR CAUSE of disability and death in the Western world today, and there is need for better diagnostic methods. It would be of great clinical value to know left ventricular (LV) end-diastolic pressure, which is typically elevated in untreated patients. Therefore, a number of noninvasive Doppler echocardiographic approaches has been suggested to provide noninvasive estimates of LV end-diastolic pressure (5–10, 12, 13). Most of these methods measure hemodynamic variables that reflect LV properties, including LV diastolic compliance, and there has been little attention to the mechanical properties of the left atrium (LA). In patients with unrestricted mitral flow, mean LA pressure (LAP) approximates LV end-diastolic pressure.

In the present study, we introduce a new principle for estimating LAP based on a relationship between deceleration time (t_{dec}) of pulmonary venous (PV) flow velocity during LV systole and LA chamber compliance (dV/dP). This principle is analogous to that used to assess LV diastolic compliance from t_{dec} of early transmitral flow (2, 7). The rationale for our proposal is the curvilinearity of the atrial diastolic pressure-volume relationship, which implies that atrial chamber compliance decreases progressively with increasing pressure.

The aim of the study was to investigate whether the systolic PV t_{dec} may represent a means to estimate LAP. The study was done in patients undergoing heart surgery, and t_{dec} was calculated from PV flow traces measured by ultrasound transit time. LAP was measured by micromanometry. As an estimate of LA compliance, we used the systolic PV flow integral divided by the systolic pressure increment.

METHODS

Patients

The study population included eight patients who underwent elective coronary artery bypass surgery. There were seven men and one woman, with a mean age of 61 yr (SD 12). The patients had either double- or triple-vessel coronary artery diseases. Mean LV ejection fraction was 52% (SD 12), ranging from 35 to 68%. Patients with unstable angina and patients with hemodynamic instability were excluded. The study was approved by the ethics committees at St. Paul’s Hospital and the University of British Columbia. Informed, written consent was obtained from each patient.

Preoperative medication included β-adrenergic-blocking agents in five patients, nitrates in five patients, calcium channel-blocking agents in four patients, and angiotensin-converting enzyme inhibitors in three patients. Two patients had diabetes mellitus and were on insulin.

Procedure

Patients were anesthetized using a balanced anesthetic technique, including sufentanil, midazolam, isoflurane, and muscle relaxation. Ventilation was adjusted to maintain normal arterial blood gases and end-tidal CO2. Patients were ventilated with volume-controlled ventilators.

The surgical procedure included sternotomy and wide pericardial split. The study was done just before initiation of extracorporeal circulation, after all preparations had been done, including insertion of an aortic cannula. An electrocardiogram was recorded and was fed into the echocardiographic recorder and the computer system.

Pressure Measurements

According to routine surgical procedure during cardiopulmonary bypass, the LV was vented via a PV. Therefore, the right lower PV was prepared with a small incision that could also be used for introduction of the 7-Fr combined micromanometer and fluid velocity sensor. The velocity measurements were not used in the present study. Approximately 4.5 cm from the tip of the catheter, there was a micromanometer (model SSD-827, Millar Instruments, Houston, TX).

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The pressure sensor was placed in the PV <2 cm from its entrance into the LA. For each patient, the pressure sensor was calibrated before catheter sterilization and recalibrated at the end of each study. In each case, the calibration factor (gain) was unchanged.

To serve as an absolute pressure reference for the micromanometers, a 16-gauge fluid-filled cannula was placed in the LA and was connected to an external pressure transducer. Mid-chest level was used as pressure zero. During zero adjustment, mean PV pressure was assumed to equal mean LAP. Because the PV pressure sensor was <2 cm into the vein, the error in this assumption was small.

**Echocardiography**

A Hewlett-Packard 5-MHz transesophageal echocardiographic probe (Hewlett-Packard, Palo Alto, CA) was placed in the esophagus for measuring PV diameter.

**PV Flow Measurements**

An 8- or 10-mm ultrasonic transit time flow probe (Transonic Systems, Ithaca, NY) was placed on the right lower PV close to its entrance into the LA. The probe was connected to a flowmeter (Transonic Systems). Ultrasonic gel was applied to obtain good contact between the vein and the flowprobe.

All electrocardiogram, pressure, and flow data were digitized at 200 Hz and were stored on a computer for later analysis using CVSOFT (Odessa Computers, Calgary, Canada).

**Protocol**

The first set of recordings was taken during baseline conditions. To study the effect of elevation of LAP, saline was infused via the aortic cannula, aiming at a peak mean LAP of ~18 mmHg. An average of 700 ml was infused.

**Calculations**

Flow values were converted to velocity by dividing flow by the cross-section of the vein. Flow deceleration was calculated as the time derivative of velocity. To calculate $t_{dec}$, we used two different methods, based on peak deceleration and mean deceleration, respectively.

**Peak deceleration method.** As illustrated in Fig. 1A, the first step was to identify the timing of peak deceleration. Then a tangent was drawn through this point on the flow-velocity curve, and peak $t_{dec}$ was

![Fig. 1. A: calculation of deceleration time ($t_{dec}$) by the peak deceleration method. A tangent was drawn through the point of peak deceleration on the pulmonary venous (PV) flow-velocity trace and was extrapolated upward to the time of peak systolic flow velocity and downward to zero flow velocity. Bottom: point of peak deceleration was defined at peak negative time derivative of PV flow velocity (dPV flow velocity/dt). Peak $t_{dec}$ ($t_{dec}$) was measured as the time interval between peak systolic flow velocity and the tangent interception with zero flow velocity. B: calculation of $t_{dec}$ by the mean deceleration method. A line was drawn between the point of peak systolic flow velocity to the nadir between systolic and early diastolic flow velocity. Then the line was extrapolated to zero flow velocity. With this method, mean $t_{dec}$ was defined as the time interval between peak systolic flow velocity and the zero intercept.](http://jap.physiology.org/)

![Fig. 2. Traces showing pulmonary venous (PV) flow velocity, the time integral of early systolic flow, and left atrial (LA) pressure (LAP) at baseline and after volume loading. LA compliance was calculated as the time integral of systolic PV flow ($\Delta V$) divided by the LAP increment ($\Delta P$).](http://jap.physiology.org/)

![Fig. 3. Changes in peak $t_{dec}$ with intravenous volume loading. Elevated LAP after volume loading was associated with marked shortening of PV $t_{dec}$.](http://jap.physiology.org/)
measured as the time interval between peak systolic flow and tangent interception with zero flow.

**Mean deceleration method.** As a potentially more practical clinical method, we also calculated $t_{\text{dec}}$ by drawing a line from the point of peak systolic flow to the nadir between systolic and early-diastolic flow and then extrapolating the line to zero flow. With this method, mean $t_{\text{dec}}$ was defined as the time interval between peak systolic flow and the zero intercept, as shown in Fig. 1B.

LA compliance was calculated as the time integral of systolic PV flow divided by the increment in LAP during ventricular systole ($\Delta P$) (Fig. 2). In these calculations, we used flow in a single PV, assuming that this vein represents a constant fraction of flow in all PV. Therefore, our compliance measure is an index rather than true compliance for the entire atrium.

**Statistical Analysis**

All measurements were made by averaging three cardiac cycles. Data are reported as means ± SE. Values before and after volume loading were compared using a two-tailed Student’s $t$-test. Regression analyses were performed using a least squares method and, when appropriate according to Glantz and Slinker (3), a multiple-regression model including dummy variables to account for between-individual differences. A $P$ value of $<0.05$ was considered significant.

**RESULTS**

Figures 2 and 3 illustrate the responses to volume loading in a representative patient. Volume loading caused a 9-mmHg rise in mean LAP ($P < 0.001$) ($n = 40$) and was associated with a reduction in LA compliance from 1.16 ± 0.42 to 0.72 ± 0.40 ml/mmHg ($P < 0.004$) ($n = 40$) and $t_{\text{dec}}$ from 320 ± 50 to 170 ± 40 ms ($P < 0.0005$) ($n = 40$) (Table 1). Figure 4 displays LA pressure-volume relations. After volume loading, there is less increase in LA volume for a given increase in LAP, which means reduced compliance.

Figure 5 illustrates the relationship between $p_{\text{dec}}$ and mean LAP and LA compliance in a representative patient. Individual regression data from all patients are presented in Table 2, confirming good correlation between $p_{\text{dec}}$/LAP and LA compliance, respectively.

Figure 6 displays pooled data for the whole study population. When performing multiple regression in a model that included dummy variables to account for between-individual differences, the relationship between the different parameters correlated well and was statistically significant ($P < 0.0005$, and $r$ values of 0.84 and 0.79 for $t_{\text{dec}}$ vs. LAP and LA compliance, respectively).

The results of the regression analyses using peak $t_{\text{dec}}$ and mean $t_{\text{dec}}$ methods were essentially similar (Tables 2 and 3).

**DISCUSSION**

The present study introduces a new method for estimating LAP that utilizes the $t_{\text{dec}}$ of systolic PV flow velocity and its relationship to LA chamber compliance. As predicted, we observed a curvilinear relationship between LAP and volume. In the stiff or poorly compliant LA, there was a marked rise in LAP during LV systole, which caused rapid deceleration of PV flow. Therefore, at LAP near physiological values, the atrium could accommodate a substantial volume with only a small rise in mean LAP ($P < 0.001$) ($n = 40$) and was associated with a reduction in LA compliance from 1.16 ± 0.42 to 0.72 ± 0.40 ml/mmHg ($P < 0.004$) ($n = 40$) and $t_{\text{dec}}$ from 320 ± 50 to 170 ± 40 ms ($P < 0.0005$) ($n = 40$) (Table 1). Figure 4 displays LA pressure-volume relations. After volume loading, there is less increase in LA volume for a given increase in LAP, which means reduced compliance.

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in pressure, whereas at elevated LAP a small increase in volume caused a marked rise in pressure. The physiological meaning of these relationships is that atrial reservoir function decreased progressively as LAP was increased. This was reflected in faster deceleration of systolic PV flow at elevated LAP and explains the inverse relationship between $t_{\text{dec}}$ and LAP.

**Clinical Implications**

The observed relationship between $t_{\text{dec}}$ and LA compliance suggests a new noninvasive approach for estimating LA passive elastic properties. Measurement of $t_{\text{dec}}$, however, does not differentiate between “true” changes in LA compliance due to structural remodeling of the atrial wall and pressure-related changes in chamber compliance. It has been shown that the LA adapts to chronic overload by changes in passive elastic properties, which is evident as a shift of the pressure-volume relationship (4). The chamber stiffness constant of LA progressively decreases along with the course of chronic volume overload, and an anatomical increase in the reservoir volume occurs. When changes in $t_{\text{dec}}$ occur acutely, however, significant structural remodeling can be excluded, and it is likely that $t_{\text{dec}}$ reflects changes in LAP. The clinical feasibility of using $t_{\text{dec}}$ as marker of LAP and LV end-diastolic pressure remains to be investigated.

We used two different methods to quantify $t_{\text{dec}}$: a peak $t_{\text{dec}}$ method and a mean $t_{\text{dec}}$ method. Both methods correlated well with LAP and LA compliance. The mean $t_{\text{dec}}$ method, however, might be easier to apply in a clinical setting.

**Study Limitations**

The present study was done using implanted flowmeter and not Doppler echocardiography, which is the conventional clinical method to study PV velocities. The highly invasive methodology was used to validate the hypothesis under rigorous methodological conditions. Further studies are needed to investigate whether the relationships between $t_{\text{dec}}$ and atrial pressure and compliance are valid in clinical routine when Doppler echocardiography is used to assess PV velocities.

Our study was done in patients who were in surgical anesthesia, on mechanical ventilation, and with open chest, and each of these factors influence PV flow. The flow tracings, however, were similar to those recorded previously in awake patients. Therefore, we believe the principles that were tested and the conclusions in this study are likely to be valid for awake patients as well. Importantly, chronic LV pressure or volume overload may cause atrial remodelling, which may lead to changes in atrial passive elastic properties and modify the relationship between $t_{\text{dec}}$ and LA compliance. This may limit the ability of $t_{\text{dec}}$ to serve as a marker of LA function in chronic studies.

The number of measurements was limited in the individual patients. This probably explains why the relationships between $t_{\text{dec}}$ and LA compliance and LAP did not reach statistical significance in some patients. This interpretation is supported by the pooled data analysis, which showed highly significant correlations.

**Comparison to Other Methods**

The PV $t_{\text{dec}}$ is one of several Doppler echocardiographic methods that has been proposed to estimate LAP (1, 2, 4–6, 8–11). Other approaches that are based on PV flow velocities include magnitude and duration of reversed PV flow during atrial systole (11) and the ratio between systolic and diastolic PVF velocities (6). The measurement of reversed PV flow during atrial systole is limited by problems with signal quality.

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**Table 2. Correlation between deceleration time of systolic pulmonary venous flow velocity and left atrial compliance**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>$r$ Value (Peak Deceleration Method)</th>
<th>$r$ Value (Mean Deceleration Method)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.90*</td>
<td>0.80†</td>
</tr>
<tr>
<td>2</td>
<td>0.98*</td>
<td>0.98*</td>
</tr>
<tr>
<td>3</td>
<td>0.97*</td>
<td>0.95*</td>
</tr>
<tr>
<td>4</td>
<td>0.93*</td>
<td>0.95*</td>
</tr>
<tr>
<td>5</td>
<td>0.87‡</td>
<td>0.80‡</td>
</tr>
<tr>
<td>6</td>
<td>0.84*</td>
<td>0.78†</td>
</tr>
<tr>
<td>7</td>
<td>0.91*</td>
<td>0.92*</td>
</tr>
<tr>
<td>8</td>
<td>0.84†</td>
<td>0.85†</td>
</tr>
</tbody>
</table>

*P < 0.05; †P < 0.07; ‡P > 0.07.

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**Table 3. Correlation between deceleration time of systolic pulmonary venous flow velocity and mean left atrial pressure**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>$r$ Value (Peak Deceleration Method)</th>
<th>$r$ Value (Mean Deceleration Method)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.79†</td>
<td>0.77‡</td>
</tr>
<tr>
<td>2</td>
<td>0.95*</td>
<td>0.96*</td>
</tr>
<tr>
<td>3</td>
<td>0.98*</td>
<td>0.97*</td>
</tr>
<tr>
<td>4</td>
<td>0.79*</td>
<td>0.84*</td>
</tr>
<tr>
<td>5</td>
<td>0.69‡</td>
<td>0.59‡</td>
</tr>
<tr>
<td>6</td>
<td>0.91*</td>
<td>0.79‡</td>
</tr>
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<td>7</td>
<td>0.92*</td>
<td>0.92‡</td>
</tr>
<tr>
<td>8</td>
<td>0.87†</td>
<td>0.84‡</td>
</tr>
</tbody>
</table>

*P < 0.05; †P < 0.07; ‡P > 0.07.
and by a strong dependence on heart rate (15, 16), whereas the ratio between systolic and diastolic velocity does not reflect acute changes in LAP (14). Kinnaird et al. (5) proposed to use the $t_{\text{dec}}$ of PV diastolic flow to estimate LAP. Whereas this method is quite promising, it may be limited by tachycardia, which may markedly abbreviate or abolish the PV diastolic flow wave. Furthermore, $t_{\text{dec}}$ measured from diastolic venous flow is determined by elastic properties of the LV as well as the LA, whereas systolic $t_{\text{dec}}$ is determined predominantly by LA elastic properties. In patients with mitral regurgitation, however, LV function may have an impact on $t_{\text{dec}}$. The systolic flow wave may still be used to calculate systolic $t_{\text{dec}}$, except when heart rate causes fusion of the systolic and the diastolic flow waves.

In conclusion, the present intraoperative study demonstrates that $t_{\text{dec}}$ can be used as an index of LAP and LA compliance. The clinical feasibility of this principle should be tested in a clinical trial where $t_{\text{dec}}$ is measured by transthoracic Doppler echocardiography.

ACKNOWLEDGMENTS

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REFERENCES


