Left Ventricular Outflow Tract Mean Systolic Acceleration as a Surrogate for the Slope of the Left Ventricular End-Systolic Pressure-Volume Relationship

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OBJECTIVES The goal of this study was to analyze left ventricular outflow tract systolic acceleration (LVOTAcc) during alterations in left ventricular (LV) contractility and LV filling.

BACKGROUND Most indexes described to quantify LV systolic function, such as LV ejection fraction and cardiac output, are dependent on loading conditions.

METHODS In 18 sheep (4 normal, 6 with aortic regurgitation, and 8 with old myocardial infarction), blood flow velocities through the LVOT were recorded using conventional pulsed Doppler. The LVOTAcc was calculated as the aortic peak velocity divided by the time to peak flow; LVOTAcc was compared with LV maximal elastance (Em) acquired by conductance catheter under different loading conditions, including volume and pressure overload during an acute coronary occlusion (n = 10). In addition, a clinically validated lumped-parameter numerical model of the cardiovascular system was used to support our findings.

RESULTS Left ventricular Em and LVOTAcc decreased during ischemia (1.67 ± 0.67 mm Hg·ml⁻¹ before vs. 0.93 ± 0.41 mm Hg·ml⁻¹ during acute coronary occlusion [p < 0.05] and 7.9 ± 3.1 m·s⁻² before vs. 4.4 ± 1.0 m·s⁻² during coronary occlusion [p < 0.05], respectively). Left ventricular outflow tract systolic acceleration showed a strong linear correlation with LV Em (y = 3.84x + 1.87, r = 0.85, p < 0.001). Similar findings were obtained with the numerical modeling, which demonstrated a strong correlation between predicted and actual LV Em (predicted = 0.98 [actual] −0.01, r = 0.86). By analysis of variance, there was no statistically significant difference in LVOTAcc under different loading conditions.

CONCLUSIONS For a variety of hemodynamic conditions, LVOTAcc was linearly related to the LV contractility index LV Em and was independent of loading conditions. These findings were consistent with numerical modeling. Thus, this Doppler index may serve as a good noninvasive index of LV contractility. (J Am Coll Cardiol 2002;40:1320–7) © 2002 by the American College of Cardiology Foundation

The left ventricular (LV) end-systolic pressure-volume relationship, under a variety of loading conditions, has been proposed as an index of LV contractility (1). Because the slope of the LV end-systolic pressure-volume relationship, LV maximal elastance (Em), is almost independent of loading conditions, Em is one of the most reliable indexes of LV contractility (2). However, the complexity of measuring LV Em, requiring a pressure-volume loop recording and at least two different hemodynamic stages, limits seriously its clinical applicability. A load-independent, noninvasive index of LV contractility, which provides reliable results compared with Em, would be ideal. Such a parameter may have considerable clinical value in the diagnosis, prognosis, and management of patients with heart disease.

Ascending aortic blood flow velocities and acceleration have been previously reported to be sensitive to inotropic stimulation and little affected by changes in loading conditions (3,4). Accordingly, this study is aimed at analyzing simultaneously the LV Em and the blood flow acceleration in the left ventricular outflow tract (LVOT) under a variety of hemodynamic conditions, including changes in preload, afterload, and contractility.

METHODS

Preparation. Eighteen juvenile sheep were used in this study. The study protocol was approved by the Animal Care and Use Committee of the National Heart, Lung, and Blood Institute. Details of anesthetic management and surgical procedures have been previously reported (5). Chronic aortic regurgitation had been surgically created six months earlier in six sheep by incising the free edge of the right coronary or the noncoronary cusp. The left anterior descending diagonal coronary artery had been occluded six months earlier, resulting in chronic myocardial infarction (MI) in eight sheep. Four sheep had normal hearts.

Twenty-six weeks later, general anesthesia was induced using intravenous pentobarbital (30 to 50 mg/kg). The...
sheep were intubated and ventilated. Anesthesia was maintained by using isoflurane with oxygen. A median sternotomy was performed. Left ventricular pressure was measured by a catheter-tipped high-fidelity micromanometer (Model SPC-350, Millar Instruments, Houston, Texas) introduced transmurally. Another catheter-tipped micromanometer introduced from the carotid artery measured ascending aortic pressure. The catheters were interfaced with a physiologic paper recorder (ES 2000, Gould Inc., Cleveland, Ohio).

**Conductance catheter.** Left ventricular pressure-volume loops were determined by a conductance catheter (SPC 560, Millar instruments Inc., Houston, Texas) inserted via the LV apex. This catheter was connected to a stimulator-microprocessor (Leycom, CardioDynamics, Zoetermeer, the Netherlands) to display a pressure-volume signal. Electromagnetic flow probes (Model EP455, Carolina Medical Electronics Inc., King, North Carolina) were placed around the aorta and pulmonary artery. The conductance volume signal was calibrated against the stroke volume derived by the aortic electromagnetic flow probe. A snare was placed around the inferior vena cava.

**Protocol.** To analyze the relationship between E\(_m\) and left ventricular outflow tract systolic acceleration (LVOT\(_{\text{Acc}}\)), five different hemodynamic stages were produced for each sheep as follows. Stage 1 was baseline. Five hundred milliliters of whole blood were then transfused over 30 min (stage 2) in order to increase the preload. Angiotensin II was infused to increase the afterload (stage 3), whereas nitroprusside was administered to decrease both preload and the afterload (stage 4). Thereafter, the midpoint of the left anterior descending coronary artery or the proximal left circumflex coronary artery was occluded to induce acute regional LV ischemia (stage 5). For each stage the hemodynamic state was stabilized for 15 min. Ventilation was suspended during each measurement.

**Echocardiography.** Echocardiographic Doppler acquisition was performed on a Toshiba ultrasound machine (PowerVision, Toshiba Medical System, Tokyo, Japan) equipped with a 3.7-MHz transducer. Hearts were scanned from the apical four-chamber window, using a standoff between the epicardium and the surface of the probe. The electrocardiogram was simultaneously acquired and displayed on the screen. The ultrasound beam was positioned in the LVOT, parallel to the aortic flow. To limit measurement errors due to a skewed peak velocity profile (6), the sample volume was placed 1 cm below the aortic valve, in the middle of the LVOT, where the optimal Doppler spectrum for cardiac output is usually recorded. The LVOT was interrogated in the pulsed-Doppler mode. Optimal Doppler gain was adjusted to display a complete blood flow velocity spectral envelope with minimal noise/signal ratio. The Doppler signal was recorded on a high-fidelity videocassette recorder (Model SVO-9500MD, Sony, Tokyo, Japan) interfaced with the ultrasound machine, for offline analysis.

**Mathematical modeling.** Using a previously described mathematical model of the cardiovascular system (7), the relationship between LV E\(_m\) and Doppler LVOT\(_{\text{Acc}}\) was also examined to verify experimental results. Briefly, our model uses 24 first-order differential equations to simulate pressure, volume, and flow throughout the heart and vessels, implemented in the LabView programming environment (National Instruments, Austin, Texas) on a 500 MHz Pentium III based computer. This model has been previously validated clinically in described complex intracardiac fluid dynamics and pressure-volume relationships (8,9). For 45 different permutations modeled by varying LV E\(_m\) (1.0 to 7.0 mm Hg·ml\(^{-1}\)) and independently varying either preload or afterload, instantaneous LV and LVOT pressures, volumes, and velocities were derived in 5-ms intervals for analysis. The LVOT\(_{\text{Acc}}\) was determined from the velocity profiles using methods similar to those previously described for the animal data (Appendix).

**Data measurements.** We measured the peak positive of the first time derivative of the left ventricular pressure (dP/dt) (maximal dP/dt [dP/dt\(_{\max}\)]). We determined LV end-diastolic pressure when positive dP/dt first exceeded 200 mm Hg·ml\(^{-1}\) and LV end-systolic pressure at the upper point of the LV pressure curve. Aortic pressure was measured as the systolic peak and diastolic trough. Left ventricular stroke volume was calculated as the flow-time integral recorded from the aortic electromagnetic flow probe. Left ventricular cardiac output was LV stroke volume × heart rate.

The ventricular end-systolic pressure-volume relationship was derived from a set of multiple and variably loaded pressure-volume loops generated by occlusion of the inferior vena cava. Data were digitally stored for offline analysis. Points of LV end-systolic pressure-volume were recorded during at least 12 loops. Data were fit by linear regression analysis, and the calculated slope was the LV E\(_m\) (Fig. 1).

Peak aortic flow velocity (PV) was measured at the point of maximum blood flow velocity; time to peak velocity (t-PV) was measured as the time from the onset to the peak of the systolic velocity spectrum, and mean LVOT\(_{\text{Acc}}\) was the PV-to-t-PV ratio (Fig. 1). Doppler measurements of PV, t-PV, and LVOT\(_{\text{Acc}}\) were calculated on three consecutive beats and averaged.
Statistical analysis. Data are presented as mean ± SD. Changes in invasive and noninvasive parameters under variable hemodynamic conditions were compared with the use of an analysis of variance (ANOVA). To increase the power of our index, we adjusted LVOTAcc for LV E\(_m\) (covariate) using an analysis of covariance. We initially tested the linear relationship between LVOTAcc and LV E\(_m\) for each stage and in each cardiac group separately by a least-squares method followed by a one-way ANOVA for statistical significance. Then we investigated the homogeneity of slopes, and a standard one-way covariance was indicated. For the overall study, correlation changes in invasive and noninvasive parameters under loading conditions did not affect LV E\(_m\) significantly. However, LV E\(_m\) was significantly decreased during acute ischemia as compared with the other stages (p < 0.05).

Left ventricular outflow tract peak velocity was 63 ± 13 cm s\(^{-1}\). Peak velocity tended to be higher during blood infusion (68 ± 15 cm s\(^{-1}\), p = NS) and angiotensin II infusion (67 ± 17 cm s\(^{-1}\), p = NS) and lower during nitroprusside infusion (59 ± 11 cm s\(^{-1}\), p = NS). Peak velocity was significantly decreased during acute coronary occlusion (44 ± 9 cm s\(^{-1}\), p < 0.05). Time to peak velocity was 91 ± 36 ms at baseline. Time to peak velocity decreased to 83 ± 31 ms and 79 ± 28 ms during angiotensin II infusion and nitroprusside infusion, respectively, and increased to 100 ± 21 ms during LV ischemia.

The LVOTAcc was insensitive to both preload and afterload alteration. However, LVOTAcc showed a tendency

Table 1. Heart Rate, Aortic Pressure, and Left Ventricular Function

<table>
<thead>
<tr>
<th></th>
<th>Normal (n = 4)</th>
<th>Old MI (n = 8)</th>
<th>Aortic Regurgitation (n = 6)</th>
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<tr>
<td>General Data</td>
<td></td>
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<tr>
<td>HR (beats/min(^{-1}))</td>
<td>109 ± 15</td>
<td>93 ± 18*</td>
<td>100 ± 8</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>79 ± 31</td>
<td>84 ± 19</td>
<td>87 ± 17</td>
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<tr>
<td>Systolic Function</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>LV EF (%)</td>
<td>60 ± 9</td>
<td>29 ± 14*</td>
<td>68 ± 10†</td>
</tr>
<tr>
<td>LV ESP (mm Hg)</td>
<td>87 ± 34</td>
<td>94 ± 18</td>
<td>99 ± 19</td>
</tr>
<tr>
<td>LV peak + dP/dt (mm Hg s(^{-1}))</td>
<td>1,346 ± 478</td>
<td>842 ± 191*</td>
<td>1,170 ± 409</td>
</tr>
<tr>
<td>LV E(_m) (mm Hg/ml(^{-1}))</td>
<td>2.71 ± 0.35</td>
<td>1.37 ± 0.47*</td>
<td>1.39 ± 0.20*</td>
</tr>
<tr>
<td>Cardiac output (l/min(^{-1}))</td>
<td>2.0 ± 0.3</td>
<td>1.8 ± 0.2</td>
<td>4.2 ± 0.7†</td>
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</tbody>
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Analysis of variance: *p < 0.05 as compared with normal sheep; †p < 0.05 as compared with old MI.

EF = ejection fraction; E\(_m\) = maximal elastance; ESP = end-systolic pressure; HR = heart rate; LV = left ventricle; MAP = mean aortic pressure; MI = myocardial infarction

Figure 1. An example of left ventricular (LV) pressure-volume relationship recording from an invasive conductance catheter (left) and LV outflow tract pulsed-wave Doppler recording from the apical view. Left ventricular outflow tract acceleration was calculated as peak velocity divided by time to peak velocity (right).
to be higher during angiotensin II infusion. There was a definite change in the LVOTAcc after acute coronary occlusion as compared with the other stages (p < 0.05). When a one-way analysis of covariance was performed, we did not detect any statistical difference between LVOTAcc and LV Em in the four different stages (p = 0.06), indicating that LVOTAcc was load-independent. However, statistical significance was attained for the three different groups (p = 0.002), confirming that LVOTAcc was influenced by different cardiac diseases.

Correlation between LVOTAcc and Em. An example of LVOTAcc and LV Em under different hemodynamic conditions is shown in Figure 2. The LVOTAcc was linearly related to LV Em (y = 3.84x + 1.87, r = 0.85, p < 0.001) (Fig. 3).

The LVOTAcc was calculated from the equation: \( \text{LVOTAcc} = \frac{\text{PV}}{t-PV} \). Because t-PV was significantly prolonged during acute coronary occlusion, we calculated the correct t-PV (the heart rate-corrected time of time to peak velocity [t-PV corr]), obtained by dividing t-PV by the square root of the RR interval (10). The corrected LVOTAcc was then calculated as the ratio of PV/t-PV cor. The correlation between measured LV Em and LVOTAcc corrected for the heart rate was good (y = 8.2x + 6.3, r = 0.84, p < 0.001).

Model of LVOTAcc. For the 45 conditions simulated, LVOTAcc ranged from 4.6 to 28.5 cm/s^2. Similar to the animal data, there was a strong linear relationship between modeled Em and LVOTAcc. LVOTAcc = 3.91[LV Em] + 2.25; r = 0.94, p < 0.001. Furthermore, when the equation relating Em to LVOTAcc was used to predict Em from the animal LVOTAcc measurements, a linear relationship was observed between the observed and expected Em (LV Em expected = 0.98, LV Em observed = -0.01, r = 0.86, p < 0.001) (Fig. 4).

Intraobserver and interobserver variability. A good agreement was found when LVOTAcc and LV Em were measured by the same observer (r = 0.93, mean difference = 0.21 ± 1.16 cm/s^2, r = 0.94, mean difference = 0.23 ± 0.25 mm Hg·ml^(-1), respectively). There was a good agreement between the two independent observers’ measurements for LVOTAcc and for the LV Em (r = 0.89, mean difference = 0.03 ± 1.43 cm/s^2, r = 0.95, mean difference = 0.001 ± 0.42 mm Hg·ml^(-1), respectively).

**DISCUSSION**

This study demonstrates that LVOTAcc, an index of LV contractility independent of loading conditions, predicted alterations in LV systolic function, whereas conventional parameters failed to detect LV systolic impairment.

**Previous studies about LV Em.** A major limitation in assessing LV systolic function is the load-dependence of cardiac output, peak positive dP/dt, and EF. In the late 1970s, Sagawa et al. (1) described the LV end-systolic pressure-volume relationship over a wide range of end-systolic points, demonstrating that the slope of the end-systolic pressure-volume relationship (LV Em) was linear, relatively insensitive to cardiac loading, and varied significantly in response to change in LV contractility. Therefore, LV Em was considered to be the most reliable index of LV contractility. However, this index can be only obtained using sophisticated invasive procedures, including a pressure-volume catheter along with an abrupt change in preload or afterload. Therefore, its clinical use is limited.

Being aware of the advantages of measuring LV Em, several authors attempted to simplify its calculation. The simplest method, reported by Little (11), was to calculate the ratio of end-systolic pressure to end-systolic volume. This simplified equation assumes constant zero pressure and volume intercepts. This is problematic because for a similar LV end-systolic pressure and volume ratio the volume intercepts. This is problematic because for a similar LV end-systolic pressure and volume ratio the volume intercept varies depending on the cardiac abnormality (12). Another approach was to calculate LV Em as LV peak isovolumetric pressure – end-systolic pressure ÷ LV stroke volume. Igarashi et al. (13) and Takeuchi et al. (14) validated this method in normal hearts using the actual or an estimated LV peak isovolumetric pressure. However, when applied to a variety of cardiac abnormalities, the correlation between actual LV Em and estimated LV Em was weak because the cosine function-derived LV peak isovolumetric pressure was not applicable to dilated hearts (15). The last simplified method to calculate LV Em was
described by Senzaki et al (15). During early contraction the normalized time-varying elastance curve of the LV is similar among several underlying cardiac abnormalities and can be integrated in a complex equation in combination with LV end-systolic volume, LV end-diastolic volume, and aortic pressure to determine LV $E_m$. However, despite rigorous efforts to simplify LV $E_m$ measurement, no individual method is applicable to all cardiac conditions and free of complicating factors or invasive procedure. Therefore, they have not seen widespread use in routine clinical settings.

**Aortic blood flow acceleration.** Despite some limitations, Doppler ultrasound recording of blood flow velocity, in the LVOT, ascending aorta, or descending aorta is well-validated to measure and to detect changes in cardiac output (16,17). Additional information such as peak velocity and acceleration can be obtained from aortic blood flow velocity.

Noble et al. (18) first described and demonstrated invasively using a catheter-tipped velocity probe that the maximal acceleration of blood into the ascending aorta was sensitive to inotropic state and relatively insensitive to the loading conditions of the heart. Similar results were reported by Bennett et al. (3) using noninvasive Doppler ultrasound measurement of the ascending blood flow velocities in normals and later by Mehta et al. (19) in patients with chronic MIs. A limitation of measuring the maximal acceleration of the blood flow velocity is the need to calculate the first differential of velocity. This cannot be done routinely. Because a good correlation exists between peak and mean aortic blood flow acceleration (20), Wallmeyer et al. (4) calculated the mean acceleration of the ascending aortic blood flow velocity as the ratio of PV to $t$-PV flow. They reported that ascending aortic blood flow mean acceleration

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**Figure 2.** An example of left ventricular pressure-volume relationship (top), left ventricular outflow tract (LVOT) velocity (middle), and hemodynamic flow velocity data with numerical simulation modeling (bottom) for a sheep with chronic myocardial infarction at baseline, after blood infusion, after angiotensin infusion, after nitroprusside infusion, and during acute coronary occlusion.
was significantly affected by inotropic alteration in dogs. Furthermore, Singer et al. (21) established that aortic blood flow mean acceleration was variably affected by alterations in loading condition.

In the present study, the forward blood flow mean acceleration was measured in a new location, that is, the LVOT. Consistent changes were seen in the Doppler-determined LVOT blood flow mean acceleration with changing LV contractility. However, despite a tendency for the Doppler LVOT blood flow mean acceleration to be higher during blood infusion, angiotensin infusion, and nitroprusside infusion, it was not significantly so. In addition, because there was a linear relationship between LV $E_m$ and LVOT blood flow mean acceleration, LVOT blood flow mean acceleration can be used as a surrogate for LV $E_m$. Further validation of our experimental results was obtained through the numerical simulation of a wide range of physiologic conditions, which confirmed the linear relationship between the LV $E_m$ and LVOT $A_{acc}$. Doppler-determined LVOT blood flow mean acceleration also reflects acute and chronic changes in the LV contractility. Interestingly, in animals with aortic regurgitation, LV systolic dysfunction was present as indicated by both lower LV $E_m$ and lower LVOT blood flow mean acceleration despite good LV EF and cardiac output. Sabbah et al. (22) reported a close correlation between the ascending blood flow maximal acceleration and the LV EF. However, they did not explore this index in animals with potential LV dysfunction and normal LV EF such as found with aortic regurgitation or high-output heart failure.

**Study limitations.** The most important technical problem encountered in measuring the LVOT $A_{acc}$ is difficulty in correctly identifying the onset and the peak of the blood flow velocity spectrum. Due to this technical limitation, Wallmeyer et al. (4) reported an LVOT $A_{acc}$ mean difference between observers of 16.8%. As a second limitation, LVOT $A_{acc}$ was not tested in the presence of turbulent flow in the outflow tract. It is unlikely that LVOT $A_{acc}$ is applicable in patients with aortic stenosis or other types of LVOT obstruction. A third limitation relates to the velocity profile in the LVOT. In the present study, blood flow velocities were interrogated in the center of the LVOT. However, from previous studies we know that the blood flow velocity profile is skewed (6). The highest blood flow velocities are

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**Figure 3.** Comparison between left ventricular (LV) outflow tract acceleration ($LVOT_{A_{acc}}$) (y axis) calculated from peak aortic flow velocity/time to peak velocity and LV maximal elastance ($E_m$) from pressure-volume loops (x-axis) in 82 hemodynamic conditions. **Solid triangles** = during change in loading conditions; **open triangles** = during acute coronary occlusion.

**Figure 4.** Comparison between left ventricular (LV) maximal elastance ($E_m$) calculated from predicted LV outflow tract systolic acceleration (y axis) and actual pressure-volume loops (x axis) in 82 hemodynamic conditions.
recorded near the septum and the anterior wall. As a direct function of velocity, LVOT mean acceleration should be affected by the site of blood flow interrogation, leading to a different relationship between LVOT\textsubscript{Acc} and LV E\textsubscript{m}. For consistency, reproducibility, and repeatability, care must be taken to measure LVOT\textsubscript{Acc} at the same location. By extension, blood flow velocities are also affected by age. However, because the time to peak flow has not been explored in the elderly, age effects on LVOT\textsubscript{Acc} are unknown.

**Clinical implications.** The LVOT\textsubscript{Acc} reflects the LV contractility represented by LV E\textsubscript{m}. It has numerous advantages over the measurement of the LV pressure-volume relationship. It is noninvasive and easily and quickly measurable. It may be preferred when LV EF and cardiac output fail to detect LV dysfunction such as in patients with aortic regurgitation or high-output heart failure. In contrast with studies by Bennett et al. (23), which demonstrated a close relationship between aortic peak flow acceleration and EF in patients with ischemic heart disease, we found a weak correlation between these two parameters (r = 0.43). Similarly, cardiac output was not related to LVOT\textsubscript{Acc} (r = 0.35). However, we found a good correlation between LVOT\textsubscript{Acc} and LV +dP/dt, another index of LV systolic function (r = 0.62).

Measuring the time-course of LVOT\textsubscript{Acc} could be informative for surgical and medical decision-making. To date, LV EF is one of the most employed parameters used to follow patients with heart disease. However, it is well known that LV EF is dependent on loading conditions. Therefore, LVOT\textsubscript{Acc}, which was independent of loading conditions, could be used to detect LV dysfunction in order to initiate therapy and to make serial evaluations of LV function after therapeutic interventions. Last, correlating LVOT\textsubscript{Acc} and the prognosis of disease would certainly be a useful application.

**Conclusions.** Under varied cardiac conditions, LVOT\textsubscript{Acc} was linearly related to LV E\textsubscript{m}, reflected acute and chronic changes in LV E\textsubscript{m} and was independent of loading conditions. Thus, this Doppler-determined index may serve as a good noninvasive index of LV contractility.

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**REFERENCES**


**APPENDIX**

Numerical modeling was based upon previous research describing the mathematical relationships of fluid propagation through the cardiac chambers during both systole and diastole. In the LabView (National Instruments, Austin,
Texas) programming environment we used, the lumped parameter, closed-loop model consisted of 24 first-order differential equations. These iteratively solved equations yield instantaneous (5-ms intervals) pressures (Equation 1), volumes (Equation 2), and flows (Equation 3) through the cardiovascular system (four chambers, pulmonary and systemic arterial and venous systems) across each of the four valves. A linear pressure-volume relationship and a constant compliance is used for the atrial, pulmonary, and systemic systems; for the ventricles, a linear pressure-volume relationship was used for systole, whereas diastole was modeled with a rising monoexponential function above and a negative exponential equation below an equilibrium volume. Experimentally obtained and clinically verified values for left atrial and ventricular systolic and diastolic parameters were used as constants (7). Results of the derived left ventricular hemodynamic data are summarized below.

\[
\begin{align*}
\frac{dP_i}{dt} &= \frac{(Q_{i-1/2} - Q_{i+1/2})}{C_i} \quad [1] \\
\frac{dV}{dt} &= Q_{in} - Q_{out} \quad [2]
\end{align*}
\]

\[
\frac{dQ_j}{dt} = \frac{P_{j-1/2} - P_{j+1/2} - r_j (Q_j)}{m_j} \quad [3]
\]

C = compliance; i = chamber node; j = flow node; m = inertial term; P = pressure; Q = flow; r = resistance term; t = time; V = volume (ml).

Summary of Simulation Data of Hemodynamic Parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Min</th>
<th>Max</th>
<th>Ave</th>
<th>SD</th>
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<tr>
<td>LV end-systolic elastance</td>
<td>1.00</td>
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<tr>
<td>Systemic pressures (mm Hg)</td>
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<td>Stroke volume (ml)</td>
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<td>Ejection fraction</td>
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<tr>
<td>LVOT Acc (cm/s²)</td>
<td>4.01</td>
<td>28.50</td>
<td>16.59</td>
<td>7.94</td>
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Ave = average; EDP = end-diastolic pressure; LV = left ventricular; LVOT Acc = left ventricular outflow tract systolic acceleration.