Simultaneous Conductance Catheter and Dimension Assessment of Left Ventricle Volume in the Intact Animal

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We compared left ventricle (LV) volume (V) simultaneously measured using the conductance catheter (V<sub>m</sub>) with volume calculated from three LV dimensions (V<sub>d</sub>) determined ultrasonically from endocardial crystals. Seven adult mongrel dogs (20–30 kg) were anesthetized and instrumented to measure micromanometer LV pressure and V. Three pairs of crystals were placed orthogonally in subendocardial positions and a conductance catheter was placed in the LV retrograde across the aortic valve. Under steady-state conditions, over the range of a single cardiac cycle, the relation between V<sub>m</sub> and V<sub>d</sub> was well described by a straight line. There was an excellent correlation of conductance and dimension volumes with r equal to 0.97±0.04 and SEE 0.8±0.5 ml. The gain (1/α) and parallel conductance volume (αV<sub>c</sub>) were constant. At lower volumes obtained during bicaval occlusion, however, the relation between V<sub>m</sub> and V<sub>d</sub> was curvilinear. 1/α and αV<sub>c</sub> both decreased as LVV fell. Thus, determination of absolute volume using the conductance catheter depended on the conditions under which the data were obtained. Under steady-state conditions, αV<sub>c</sub> calculated by both the saline method (mean±SD, 50±15 ml) and by regression of V<sub>m</sub> and V<sub>d</sub> (45±21 ml) were similar. Consequently, absolute LV end-diastolic volumes and end-systolic volumes by the conductance and dimension methods were similar (53±14 ml and 38±14 ml vs. 56±17 ml and 44±16 ml, respectively, p=NS). When volume decreased during bicaval occlusion, there was a progressively greater decrease in V<sub>m</sub> as compared with V<sub>d</sub>. The absolute slope (E<sub>ES</sub>) of the end-systolic pressure-volume relation (ESPVR) was consistently higher by the dimension method, group average, 16.3±7.6, than by the catheter, 8.5±5.9, p<0.05. The direction and magnitude of the change in E<sub>ES</sub> at different inotropic states (autonomic blockade; dobutamine), however, was similarly measured by both the conductance catheter and dimension method. We conclude that the gain and offset of the conductance catheter are relatively constant at steady state but vary when volume is reduced by caval occlusion. Thus, the conductance catheter accurately measures absolute volumes at steady state but can underestimate the slope and position of the ESPVR when it is determined by caval occlusion. The conductance catheter does, however, accurately measure the directions and magnitude of change in contractile state. (Circulation 1990;81:638–648)

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alysis of the left ventricle (LV) in the pressure-volume plane has provided insights into LV performance.1–5 Clinical application of pressure-volume analysis, however, has been limited because of the difficulty in measuring LV volume. Recent refinement of a conductance catheter has provided a tool capable of continuously measuring LV volume without disturbing the LV and its surrounding tissues.6–9 The signal from the conductance catheter reflects LV conductance as well as parallel conductance because of current paths outside the LV. Measurement of absolute LV volume using the conductance catheter, thus, depends on accurate calculation of the volume attributable to parallel conductance. Validation of conductance catheter measurement of absolute LV volume has been obtained in the postmortem canine heart9 and in an isolated ejecting left heart preparation.10 Limited data are available comparing conductance cath-

See p 703

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eter measurement of LV volume to angiographic LV volumes under steady-state conditions in intact animals and patients, and after balloon occlusion of the inferior venae cavae, aorta, and pulmonary artery. No previous study, however, has evaluated conductance catheter measurement of absolute LV volume at steady state and over a range of volumes and inotropic states in an intact animal using an independent method of continuously determining LV volume. Accordingly, the purpose of this study was to compare measurements of absolute LV size and pressure-volume analysis performed with the conductance catheter to sonomicrometry measurement of LV dimensions in the intact animal. LV sonomicrometry provides continuous measurements of LV volume in an intact animal model. This technique has been used extensively and validated in our laboratory.

Methods

Instrumentation

Seven mongrel dogs (20–30 kg) were premedicated with xylazine (0.1 mg/kg), anesthetized with sodium pentobarbital (10–15 mg/kg), and maintained on an intravenous pentobarbital drip (1–2 mg/kg/hr). Ventilation was provided through an endotracheal tube by a volume respirator. Blood gasses were maintained within the physiologic range by adjusting ventilation and by oxygen supplementation as necessary. The animals were placed on their right side and the thorax was opened in the left fourth or fifth intercostal space. The lungs were retracted, and a periocardial cradle was constructed to fully expose the heart. Three ultrasonic crystal pairs (5 mm, 3 MHz) were implanted in the endocardium to measure the anteroposterior (AP), septal-lateral (SL), and base-apex long-axis (LA) dimensions. Umbilical tape snares were placed around the inferior and superior venae cavae to allow bicaval occlusion. Vascular sheaths were then placed through local cutdown in peripheral arterial and venous sites for catheter placement under fluoroscopic guidance. A balloon-tipped fluid-filled tube was positioned in the pulmonary artery. A micromanometer catheter (Millar Instruments, Inc., Houston, Texas) was passed retrograde across the aortic valve into the LV. Finally, a conductance catheter (Webster, Inc., Baldwin Park, California) was passed retrograde across the aortic valve and the tip positioned in the apex of the LV.

Volume Measurements

Conductance Catheter

Using the electrode catheter inserted into the LV retrograde across the aortic valve, an electrical field was generated (20 KHz, 0.03 mA RMS current) in the LV from electrodes at the apex and at the aortic valve. Sensing electrodes evenly distributed along the catheter measure conductances between electrode pairs located within the LV. The conductances are summed and converted to volume using a signal conditioner (Sigma 5, Leycom, The Netherlands). The volume (V) of the ventricle at any time t is calculated as:

\[ V(t) = (1/\alpha) L^2 \rho [G_M(t) - G_P] \]  

where, \( \alpha \) equals unitless constant, L equals distance between sensing electrodes, \( \rho \) equals resistivity of blood, which is inversely related to conductivity, \( G_M(t) \) is sum of conductances measured at any time (t), and \( G_P \) is parallel conductance. Because L and \( \rho \) are known and 1/\( \alpha \) remains essentially constant, \( V(t) \) is proportional to \( G_M(t) \). In using the catheter system, the values for L and \( \rho \) are measured and entered into the signal conditioner, which computes an uncorrected conductance volume (\( V_M(t) \)):

\[ V_M(t) = L^2 \rho G_P \]  

At the frequency used to generate the current, LV tissue, RV tissue and fluid, and the juxtapercardial tissues also contribute to the total measured conductance. The volume offset, \( V_C \), caused by the parallel conductance, \( G_P \), is equal to:

\[ V_C = (1/\alpha) V_M(t) - \alpha V_C \]  

Substituting Equations 2 and 3 into Equation 1:

\[ V_G(t) = (1/\alpha) (V_M(t) - \alpha V_C) \]  

where, \( V_G \) is the conductance catheter volume corrected for parallel conductance volume, and \( \alpha V_C \) is the volume correction because of parallel conductance. Baan et al have developed a technique to calculate \( \alpha V_C \) by transiently altering blood conductivity within the LV without actually changing LV volume or ejection fraction. Hypertonic saline (5 ml of 20% NaCl) is injected into the pulmonary artery, causing a transient increase in measured volume, \( V_M \). The calculation of parallel conductance by the saline method assumes that \( G_P \) remains constant, and that ejection fraction does not change. The line characterizing the relation between the end-diastolic (ED) and end-systolic (ES) \( V_M \) is computed. This line is extrapolated to the point where conductance within the LV is zero, that is, \( V_M \) ED = \( V_M \) ES. This point is equal to \( \alpha V_C \) because any \( V_M \) arises from parallel conductance outside of the LV. Baan et al have evaluated this approach in the isolated heart and open-chest anesthetized dogs and have found that the saline method accurately measured \( \alpha V_C \).

Dimension Method

Left ventricular volume was also calculated from the three endocardial LV dimensions (D) measured by the sonomicrometry crystals according to the equation:

\[ V_D = (\pi/6) D_{AP} D_{SL} D_{LA} \]

This method of LV volume measurement from ultrasonic dimensions has been evaluated in our laboratory and found to provide a consistent measure of LV volume during caval occlusions despite changes in LV size, configuration, and synchrony. The con-
duction catheter and dimension volumes were obtained simultaneously.

Protocol

Adequate placement of the conductance catheter was confirmed by two methods. First, under fluoroscopic guidance, the tip of the catheter was placed in the LV apex, and the proximal most electrode was positioned just above the aortic valve. Second, the waveform of the proximal most sensing pair was compared with the next most proximal pair. The catheter position was considered acceptable if the proximal and next most proximal sensing waveforms were in phase. Blood resistivity, \( \rho \), was then measured and entered into the signal conditioner along with the interelectrode sensing distance. The parallel conductance volume (\( \alpha V_C \)) was calculated as the average value obtained from at least three hypertonic saline injections. Steady-state recordings were made after the hemodynamics had stabilized. Transient bicaval occlusions were then performed at least twice under each condition resulting in a 30–40 mm Hg drop in LV systolic pressure. The cavae were released before the LV systolic pressure dropped lower than 60 mm Hg to prevent global LV ischemia. Autonomic blockade was then induced with hexamethonium bromide (5–10 mg/kg) and atropine (0.1 mg/kg). Steady-state and bicaval occlusion recordings were then obtained. A continuous infusion of dobutamine hydrochloride (2.5–10 \( \mu \)g/kg/min) was then administered to increase \( \frac{dP}{dt} \) by approximately 1,000 mm Hg/sec. Steady-state and bicaval occlusion recordings were obtained during the dobutamine infusion.

Data Analysis

Data were collected for 20 seconds and digitized at 200 Hz. All collections were obtained during end expiration with the respirator off (open to atmospheric pressure). Data were stored on a computer (PCs Limited 286 AT, Austin, Texas) and analyzed using software developed in our laboratory. End diastole was defined as the relative minima of LV pressure after the A wave. End systole was defined as the upper left corner of the pressure-volume (P-V) loop identified using the iterative technique of Kono et al.\(^{26}\)

\( \alpha V_C \) was calculated at steady state by two methods. The first technique was the saline method as described. The variability of repeated saline measures of \( \alpha V_C \) was evaluated by the coefficient of variation, defined as the standard deviation divided by the mean \( \times 100\% \). \( \alpha \) and \( \alpha V_C \) were also calculated by comparing LV volume obtained by using the conductance catheter and the dimension method. \( V_M \) was plotted versus \( V_D \) from the ejection phase of a steady-state cardiac cycle. If \( V_D \) and \( V_0 \) both represent true LV volume, from Equation 4: \( V_M(t) = \left(1/\alpha\right)(V_M(t) - \alpha V_C) \). Linear regression of \( V_M \) and \( V_D \) yields a slope equal to \( \alpha \) and \( V_M \) intercept equal to \( \alpha V_C \):

\[
V_M = \alpha V_D + \alpha V_C
\]  

Ejection phase points were chosen to examine a phase of the cardiac cycle when volumes were rapidly changing and to provide a manageable number of points for analysis. \( \alpha V_C \) determined by the two methods were then compared.

\( \alpha \) and \( \alpha V_C \) were also examined by comparing conductance catheter and dimension volumes of variably loaded beats during bicaval occlusion. Ejection phase \( V_M - V_D \) points from each beat of a bicaval occlusion were fit to a linear regression as previously described. \( \alpha \) and \( \alpha V_C \) of the first, middle, and last beat of the bicaval occlusion were compared to assess volume-dependent changes in these variables at each inotropic state.

The slope of the end-systolic pressure-volume relation (ESPVR), \( E_{ES} \), was calculated by linear regression of the ES pressure-volume points obtained during the bicaval occlusion. The first five beats of the occlusion were excluded to minimize the effect of changes in RV volume on parallel conductance.\(^{25}\) Points below an LV systolic pressure of 60 mm Hg, premature beats, and postpremature beats were also excluded from the analysis. Similarly, the slopes of the stroke work–end-diastolic volume (SW-EDV) relation and the \( \frac{dP}{dt_{\text{MAX}}} \)-end-diastolic volume (\( \frac{dP}{dt_{\text{MAX}}}-\text{EDV} \)) relation\(^{28}\) were calculated using the same points as were used in calculating \( E_{ES} \).\(^ {29}\) Pressure-volume analyses were compared using end-systolic volumes from both the conductance catheter corrected for parallel conductance (\( V_D \)) using the saline method and the three orthogonal dimensions (\( V_D \)). The relative position of each pressure-volume relation can be described by the volume-axis position at a point in the midrange of the y-axis values generated during the caval occlusion.\(^ {29}\) This point is not subject to errors in extrapolation that can influence the extrapolated volume-axis intercepts, \( V_0 \).

For the ESPVR relation, \( V_0 \) was defined as the volume associated with an LV end-systolic pressure (ESP) of 100 mm Hg. This value was chosen because it corresponded to the midrange of end-systolic pressures obtained during most of the caval occlusions. For each caval occlusion, this point was determined by calculating the volume associated with an LVESP of 100 mm Hg using the line describing \( E_{ES} = E_{ES}(V_{ES} - V_0) \). The relative positions of both the SW-EDV and \( \frac{dP}{dt_{\text{MAX}}}-\text{EDV} \) relations (\( V_{MID} \)) were characterized in a similar manner, and defined as the volumes associated with a stroke work and \( \frac{dP}{dt_{\text{MAX}}} \) in the midrange of values obtained during the caval occlusion. The ability of the ESPVR to evaluate alterations in inotropic state was also assessed. To quantify the magnitude of the measured change in contractile state, we compared the ratio of \( E_{ES} \) obtained during caval occlusion at different inotropic states using both the conductance catheter and the dimensions. We also examined the percentage of change in \( E_{ES} \) at different inotropic states for both the conductance catheter and dimensions.
of the $V_M$ curve after injection of the saline. Parallel conductance volume ($\alpha V_C$) calculated by the saline method was 50±15 ml, whereas $\alpha V_C$ estimated by regression of $V_M$ versus $V_D$ was 48±18 ml, $p=NS$ versus saline. $\alpha$ was 1.0±0.2. The coefficient of variation for repeated saline measures of $\alpha V_C$ was 22±13%. Comparison of parallel conductance volume obtained by the saline method ($\alpha V_C$, SAL) and by the dimension method ($\alpha V_C$ D) in all of the animals resulted in the regression equation: $\alpha V_C$ D=1.1 $\alpha V_C$ SAL−9.8 ml, with $r=0.78$ and SEE=15.7 ml.

Simultaneous $V_M$ and $V_D$ obtained every 5 msec under basal steady-state conditions from a representative animal are shown in Figure 2. In all animals, the relation was well defined by a straight line. For the group, the correlation between $V_M$ and $V_D$ was 0.970±0.04 with a range within the group of 0.877–0.996. The SEE for the group was 0.80±0.5 ml. An example of LV volume during a steady-state cardiac cycle is shown in Figure 3 for both $V_G$ and for $V_D$. Phasic changes in the cardiac cycle were recorded comparably by $V_G$ and $V_D$. Simultaneous $V_G$ and $V_D$ measurements obtained at steady state for the group are shown in Table 1. End-diastolic and end-systolic conductance volumes corrected by the
saline method were 53±14 and 38±14 ml, and 55±16 and 41±15 ml, respectively, for the regression corrected conductance catheter volumes. These were both similar to the end-diastolic and end-systolic \(V_D\) of 56±17 and 44±16 ml, respectively. Stroke volume determined by the two methods were also similar. Dimension crystal-derived stroke volume (\(SV_D\)) averaged 12±4 ml, whereas conductance catheter stroke volume (\(SV_G\)) was 15±4 ml, \(p=NS\).

**Variably Loaded Beats (Caval Occlusion)**

A representative analog recording of simultaneous conductance catheter volumes and dimension measurements during transient bicaval occlusion is shown in Figure 4. A representative example of a comparison of \(V_M\) versus \(V_G\) using points obtained from beats during bicaval occlusion is shown in Figure 5. Over the volume range encompassed by one cardiac cycle, the \(V_M-V_D\) relation was linear, similar to the data obtained at steady state. Additionally, the regression of \(V_M\) and \(V_D\) was well approximated by a straight line for each beat throughout the caval occlusion. Over the range of the entire caval occlusion, however, the relation was curvilinear and concave toward the \(V_D\) axis. Consequently, there was a progressive increase in the slope, \(\alpha\), of the relation and a decrease in the \(V_M\) intercept, \(\alpha V_C\). This was consistently found in all of the animals studied. To quantify this effect, we compared \(\alpha\) and \(\alpha V_C\) for the first, middle, and last beats of each bicaval occlusion. The results are shown in Table 2 along with the corresponding LVEDV and LVESV obtained during the bicaval occlusion. For the first beat, at an LVEDV=63±20 ml and LVESV=49±17 ml, \(\alpha\) was 1.0±0.2, and \(\alpha V_C\) was 46±21 ml. \(\alpha\) progressively increased to 1.5±0.3 for the last beat ( \(p<0.05\) vs. first and middle beats), and \(\alpha V_C\) fell to 19±26 ml for the last beat ( \(p<0.05\) vs. first and middle beats), with a corresponding LVEDV=49±18 ml, and LVESV=43±17 ml. Thus, using the conductance catheter, there was a progressive decrease in \(1/\alpha\) and \(\alpha V_C\). Additionally, \(1/\alpha\) and \(\alpha V_C\) fell from the first to the last beat of the bicaval occlusion at each inotropic state. Moreover, there was no significant difference between the change in \(1/\alpha\) and \(\alpha V_C\) among the different inotropic states.

An example of SV determined by both techniques during the course of a bicaval occlusion is shown in Figure 6. There was a progressive fall in \(SV_D\), which was paralleled by a fall in \(SV_G\). Regression of \(SV_D\) versus \(SV_G\) during bicaval occlusion was obtained for each animal, with group average \(r=0.90±0.15\) (range, 0.71–0.99). For the group \(SV_G=1.1\) (±0.4) \(SV_D=3.4\) (±3.9) ml. Thus, the catheter accurately measured SV over the range of volumes obtained during a bicaval occlusion despite changes in \(\alpha\) and \(\alpha V_C\).

LVPV loops generated from a bicaval occlusion are shown in Figure 7 using both \(V_D\) and \(V_G\).
Although dimension and conductance LV volumes were similar at the start of the caval occlusion, there was a progressively smaller $V_0$ and $V_D$ over the course of the bivacal occlusion. As a result, $E_{ES}$, the slope of the ESPVR, was greater when determined by the dimension method ($E_{ESD}$) as compared with the conductance catheter method ($E_{ESG}$). This was consistently seen in all of the animals. Both ESPVRs were well described by straight lines with correlations of 0.98. For the group under basal conditions, $E_{ESD}$ was approximately twice $E_{ESG}$, 16.3±7.6 vs 8.5±5.9 mm Hg/ml, *p<0.05.

Changes in Inotropic State

To assess the ability of the conductance catheter to measure changes in inotropic state, we evaluated the ESPVR under several different inotropic conditions. Hemodynamic variables obtained under steady-state conditions for each of the different inotropic states are listed in Table 3. An example of simultaneous LVPV loops during bivacal occlusion using both $V_0$ and $V_D$ from each of the three inotropic states is shown in Figure 8. Throughout the bivacal occlusion at each of the inotropic states, $V_{0}$ $E_{ES}$ was consistently lower than $V_{D}$ $E_{ES}$. The direction and magnitude of changes in $E_{ESG}$, however, was similar to changes in $E_{ESD}$. Results of the quantitative ESPVR assessment are shown in Table 4. The percentage of difference in $E_{ES}$ between dobutamine and autonomic blockade was similar for the conductance catheter (59.0±28.4%) and for the crystals (61.0±19.0%), *p=NS. Thus, $E_{ESG}$ consistently underestimated $E_{ESD}$ but measured the relative change in inotropic states similarly.

### Table 2. Regression of $V_0$ Versus $V_M$ During Bivacal Occlusion

<table>
<thead>
<tr>
<th></th>
<th>$a$</th>
<th>$aV_C$ (ml)</th>
<th>$r$</th>
<th>EDV (ml)</th>
<th>ESV (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>First beat</td>
<td>1.0±0.2</td>
<td>46±21</td>
<td>0.98±0.03</td>
<td>63±20</td>
<td>49±18</td>
</tr>
<tr>
<td>Middle beat</td>
<td>1.2±0.2*</td>
<td>30±24*</td>
<td>0.98±0.02</td>
<td>54±18*</td>
<td>45±17*</td>
</tr>
<tr>
<td>Last beat</td>
<td>1.5±0.3*†</td>
<td>19±26*†</td>
<td>0.98±0.01</td>
<td>49±18*†</td>
<td>43±17*†</td>
</tr>
</tbody>
</table>

$V_0$, left ventricle volume, dimension method; $V_M$, conductance catheter left ventricle volume not corrected for parallel conductance volume; $a$, unitless constant; $aV_C$, parallel conductance volume; $r$, correlation coefficient; EDV, end-diastolic volume, dimension method; ESV, end-systolic volume, dimension method.

*p<0.05 vs. first beat; †*p<0.05 vs. middle beat.

The ability of the conductance catheter to assess changes in contractile performance in the PV plane was also assessed using the SW-EDV and dP/dtMAX-EDV relations. A representative example of simultaneous conductance catheter and dimension SW-EDV points obtained during a bivacal occlusion is seen in Figure 9. Both relations were linear, and the slopes were similar. Quantitative analysis of the SW-EDV relation for the group at all three inotropic states is shown in Table 4. The slope of the relation was similar at each inotropic state for the conductance catheter and dimension method, and reflected equivalent changes in the inotropic state. The ratio of the slopes of the SW-EDV relation determined using the two methods of volume measurement was approximately 1. $V_{MID}$ was consistently lower for the conductance catheter compared with the dimension method. An example of simultaneous conductance catheter and dimension method dP/dtMAX-EDV points during a basal-state caval occlusion is also shown in Figure 9. Similar to the SW-EDV relation, the dP/dtMAX-EDV relation was linear. Results of quantitative analysis of the dP/dtMAX-EDV relation for the group at all three inotropic states is presented in Table 4. Comparable with the results obtained using the SW-EDV analysis, the slopes from the conductance catheter and dimension method were similar at each inotropic state, and the ratio of the slopes was not significantly different from 1. Moreover, the slopes of the dP/dtMAX-EDV relation changed appropriate to the inotropic state. $V_{MID}$ also tended to be lower for the conductance catheter compared with the dimension method but the differ-

**Figure 5.** Scatterplot showing simultaneous conductance catheter volume (not corrected for parallel conductance volume), $V_M$, and dimension derived volumes, $V_D$, obtained every 5 msec during ejection phase over course of bivacal occlusion.

**Figure 6.** Scatterplot showing conductance catheter and dimension-derived stroke volume during typical bivacal occlusion from one animal. SV(G), conductance catheter stroke volume; SV(D), dimension-method stroke volume.
ences were not seen as consistently as with the SW-EDV relation.

Discussion

In this study, we compare LV volume obtained from the conductance catheter with LV volume calculated from three orthogonal dimensions using ultrasonic crystals. Dimensional analysis of LV volume using sonomicrometry provides continuous measurement of LV volume over a wide range of physiologically relevant volumes. Sonomicrometry has been extensively evaluated and validated by several investigators,13–22 It is reproducible, capable of accurately reflecting small changes in LV volume, and is relatively unaffected by conformational changes of the LV at volumes within the physiologic range. Sonomicrometry is, therefore, well suited for assessing the parallel conductance volume component of the conductance catheter signal. At steady state, over the volume range encompasses by one cardiac cycle, the relation of uncorrected conductance catheter (V_m) and dimension volumes (V_D) was well approximated by a straight line. Thus, both parallel conductance and the gain (1/α) were essentially constant over the range of a normal cardiac cycle. Furthermore, the volume attributable to the parallel conductance (αV_c) could be accurately estimated using the saline method.

Unlike the findings under steady-state conditions, both 1/α and αV_c were found to vary when variably loaded beats were examined. Over the volumes obtained during bivacal occlusion, the relation between conductance and dimension volumes was curvilinear and concave toward the dimension-volume axis. This was apparent as a progressive decrease in 1/α and αV_c from the first to the last beat of the bivacal occlusion. Because the range of volumes obtained during the bivacal occlusion overlapped considerably with the volumes obtained at steady state, significant changes in 1/α and αV_c may occur only when a critical reduction in volume has been surpassed. Our data suggest that this critical volume might be close to steady-state end-systolic volume in the anesthetized dog. Similar changes in 1/α and αV_c were also observed at markedly different inotropic states during caval occlusion (over comparable volume ranges), indicating that alterations in 1/α and αV_c were reproducible and were volume dependent and not dependent on the contractile state of the LV. Thus, determination of absolute LV volume using the conductance catheter appear to be influenced by the volume range over which the data are examined.

Current understanding of measurement of absolute LV volume using the conductance catheter is based on studies demonstrating a linear relation

Table 3. Hemodynamics During Assessment of End-Systolic Pressure-Volume Relation at Different Inotropic States

<table>
<thead>
<tr>
<th></th>
<th>HR (beats/min)</th>
<th>LVESP (mm Hg)</th>
<th>LVEDP (mm Hg)</th>
<th>dP/dt_max (mm Hg/sec)</th>
<th>LVEDV (ml)</th>
<th>LVESV (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Autonomic blockade</td>
<td>114±13</td>
<td>121±13</td>
<td>11±3</td>
<td>1,054±276</td>
<td>53±13</td>
<td>35±13</td>
</tr>
<tr>
<td>Basal state</td>
<td>146±20*</td>
<td>130±14</td>
<td>5±3*</td>
<td>1,549±533*</td>
<td>43±18*</td>
<td>30±13*</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>140±13*</td>
<td>147±16*†</td>
<td>8±3</td>
<td>2,048±695*†</td>
<td>46±17</td>
<td>30±13</td>
</tr>
</tbody>
</table>

HR, heart rate; LVESP, left ventricle end-systolic pressure; LVEDP, left ventricle end-diastolic pressure; dP/dt_max, positive maximal derivative of left ventricle pressure; LVEDV, left ventricle end-diastolic volume; LVESV, left ventricle end-systolic volume.

*p<0.05 vs. autonomic blockade; †p<0.05 vs. basal state.
between conductance and actual volume.\textsuperscript{6–10} Baan et al.\textsuperscript{8} evaluated measurement of conductance volume in postmortem canine hearts. They emptied the LV by suction and found a linear relation between absolute LV volume and LV conductance down to zero volume. Subsequently, they demonstrated a linear relation between SV measured by the conductance catheter and an electromagnetic flow probe in intact animals.\textsuperscript{6–8} As seen in this study, however, changes in SV might not be significantly affected by volume-dependent changes in $1/\alpha$ and $\alpha V_C$. Thus, the effect of changes in $1/\alpha$ and $\alpha V_C$ might be minimized by this technique. Baan et al.\textsuperscript{8} also compared LV volume measured by conductance and angiography in patients. They were able to accurately measure parallel conductance volume by the saline method and, thereby, obtain accurate absolute LV volume measurements. The linearity of conductance and angiographic volumes in these patients, however, was assessed under steady-state conditions when $1/\alpha$ and $\alpha V_C$ can be relatively constant. Burkhoff et al.\textsuperscript{10} compared conductance catheter LV volume corrected for parallel conductance volume by the saline method to LV volume in the isolated heart. They found that conductance catheter accurately measured LV volume. Burkhoff et al.\textsuperscript{10} placed the catheter in the LV across the aortic valve. Thus, changes in parallel conductance might be related to differences in the catheter position within the LV. Additionally, there are significant differences in the structures contributing to parallel conductance in the isolated heart preparation compared with the intact animal. For this reason, observations of the behavior of parallel conductances obtained in the isolated heart preparation might not be comparable with findings in the intact animal.

Several more recent reports suggest that parallel conductance might not be constant under all conditions. Boltwood et al.\textsuperscript{12} recently evaluated conductance catheter and angiographic LV volume determinations in anesthetized dogs. They assessed $1/\alpha$ and $\alpha V_C$ at steady state, during pulmonary and aortic occlusions, and during limited caval occlusions. They observed that parallel conductance volume was not constant but varied as a direct function of LVES volume. Regression of conductance catheter volumes uncorrected for parallel conductance volume and dimension volumes during caval occlusion (Figure 5, Table 2) from our study are consistent with their observations. Tjon-A-Meeuw et al.\textsuperscript{11} compared steady-state conductance catheter LV volume to single-plane angiographic LV volume in anesthetized dogs. They observed that the conductance catheter overestimated LVES angiographic volume but accu-
rately estimated LVED angiographic volume. This suggests that parallel conductance might not be constant at smaller LV volumes. The data from these studies and the present study, together, indicate that the gain and parallel conductance volume vary when volume is reduced.

Mur and Baan noted that the theoretical relation between conductance catheter volume and absolute volume was not perfectly linear, probably because of changes in the electrical field distribution within the LV. Conductance volumes underestimated actual volumes at the lower range of ranges, which is consistent with the data observed in this study, and the observations of Boltwood et al. The changes in the electrical field might be more pronounced at smaller volumes and explain small differences in parallel conductance within a cardiac cycle. Newer algorithms for determining conductance catheter LV volume may minimize the effect of volume-dependent changes in parallel conductance and allow accurate determination of the absolute ESPVR using the conductance catheter.

Other possibilities for the apparent change in parallel conductance during the caval occlusion should be examined. Because caval occlusion was used to generate the variably loaded beats, it is possible that parallel conductance progressively fell because of continuous reductions in right ventricle (RV) and pulmonary vascular contributions to parallel conductance although we excluded the initial beats after caval occlusion when RV volume is rapidly changing. It is also possible that movement of the catheter out of the LV during caval occlusion could affect conductance catheter volumes. We continuously assessed the position of the catheter fluoroscopically and did not see a change in its position within the LV. Thus, this did not likely affect our results. It is also possible that the dimension method progressively overestimated LV volume. At steady state, the gain ($1/\alpha$) relating conductance volume to dimension volume was $1.0\pm0.2$. This value is consistent with previously reported values of $\alpha$ ranging from 0.8 to 1.1$^{6-9}$ with other methods of volume determination. Although configuration changes in the LV can affect ultrasonic measurement of LV volume at extremely low volumes, data from this laboratory and others have shown that sonomicrometry using endocardial crystals, reliably and consistently measures LV volume over the range used in this study.$^{13-22}$

Clinical Implications

Our study and previous studies indicate that the conductance catheter can be used to determine SV over a wide range. Determination of absolute LV volumes under steady-state conditions, when $1/\alpha$ and $\alpha V_C$ are essentially constant, are also feasible using the conductance catheter and the saline method of calculating parallel conductance volume. Because of the volume-dependent changes in $1/\alpha$ and $\alpha V_C$, however, use of the conductance catheter in non–steady-state conditions might have limitations.
Particularly, analysis of ESPVRs over a range of volumes using the conductance catheter might underestimate the absolute ESPVR because of volume-dependent changes in $1/\alpha$ and $\alpha V_c$. During the course of a bicaval occlusion, in this study, there was a progressively greater reduction in LV volume measured by the conductance catheter compared with dimension volume. As a consequence, the absolute slope of the ESPVR, $E_{ES}$, was consistently lower at all inotropic states for the conductance catheter than for the dimension method. This observation is in close agreement with the prediction of Boltwood et al.\textsuperscript{12} Although the conductance catheter underestimated the absolute ESPVR, the direction and magnitude of changes in inotropic state were similarly measured by the conductance catheter and dimension method. Thus, the catheter might not determine the absolute position of the ESPVR. In a single subject, however, the effect of maneuvers or pharmacologic interventions on the ESPVR measured over similar volume ranges could be accurately compared. Comparisons over markedly different volume ranges in a subject can be problematic because of the effect of changes in $1/\alpha$ and $\alpha V_c$ on the absolute ESPVR, independent of changes in inotropic state. Furthermore, use of the conductance catheter to compare the absolute ESPVR between subjects can be complicated by the potential variations in volume-dependent changes in the gain and parallel conductance volume shown in this and other studies.

In contrast to the differences in the ESPVR measured by the two techniques, we found the SW-EDV relation and the $dP/dt_{\text{MAX}}$-EDV relation were similar, regardless of whether LV volume was measured by the conductance catheter or calculated from three LV dimensions. This can occur because the range of ED volumes obtained during a caval occlusion are similar to the range of volumes encountered in a steady-state beat (see Figures 2 and 5). Over this range, $V_D$ and $V_M$ are linearly related. At smaller volumes, however, the $V_D^*V_M$ relation is curvilinear and ES conductance volumes underestimate actual LV volume. Thus, EDPV analyses might have advantages in assessing LV performance with the conductance catheter.

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