Simultaneous LV and RV volumes by conductance catheter: effects of lung insufflation on parallel conductance

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Szwarc, Richard S., and Howard A. Ball. Simultaneous LV and RV volumes by conductance catheter: effects of lung insufflation on parallel conductance. Am. J. Physiol. 275 (Heart Circ. Physiol. 44): H653–H661, 1998.—One aspect in the measurement of ventricular volume using the conductance catheter technique is the assessment of parallel electrical conductivity of structures extrinsic to the ventricular blood pool. Because it is sometimes necessary to make volume measurements during ventilation or spontaneous respiration, the extent to which parallel conductance may vary with lung insufflation was investigated. Anesthetized pigs (11–15 kg) were ventilated and instrumented with both left (LV) and right ventricular (RV) conductance and pressure-tip catheters and end-hole catheters for injection of hypertonic saline into the inferior vena cava and pulmonary artery. Data were recorded during ventilation with tidal volumes of 10 and 20 ml/kg, and the associated fluctuations to LV and RV end-diastolic (EDV) and stroke (SV) volumes were measured. With the use of a saline dilution technique, parallel conductance (V_c) was determined for each ventricle with the ventilator off and lungs insufflated to 0, 10, and 20 ml/kg. Whereas ventilation caused marked oscillations in LV and RV EDV and SV, these variations could not be attributed to V_c, which remained statistically unchanged from their baseline values of 34.1 ± 3.1 in the LV and 31.1 ± 4.4 in the RV. These results indicate that the fluctuations that occur in conductance catheter-derived LV and RV volume signals with ventilation are not caused by any significant changes to parallel conductance.

conductance catheter; ventricular volume; ventricular interaction; parallel conductance

THE CONDUCTANCE CATHETER method of measuring ventricular volume has become an important advance in the assessment of left ventricular (LV) (3, 4, 10, 20, 21) and, more recently, right ventricular (RV) (7, 11, 18, 23) function. The multielectrode catheter emits a low current electrical field within the ventricle, allowing the measurement of a time-varying, intracavity electrical conductance assumed to be proportional to changes in ventricular blood volume. In practice, however, in order to obtain absolute volume measurements, both a gain and an offset must first be applied to the conductance signal.

The conductance offset term is required because the electrical field is not contained entirely within the ventricular cavity but extends through the myocardium, contralateral ventricle, and surrounding structures. Thus whereas the conductance catheter measures conductivity of the ventricular blood pool, it also detects that of extrinsic structures. This parallel conductance is manifested as a positive offset of the conductance-derived volume signal and is usually assumed to be a constant in any given subject.

Normally, conductance catheter measurements are made in the anesthetized patient with ventilation temporarily suspended at end expiration or in the awake patient during shallow or held respiration. However, because the conductance catheter is used in a wider variety of situations, one component of parallel conductance that must be addressed is the lungs, especially if the method is employed in spontaneously breathing subjects, during Muller and Valsalva maneuvers or to assess the actual effects of ventilation on ventricular volume and function. It is well known that both RV and LV volumes change in response to breathing and ventilation (1, 6, 8, 12–14, 22), but the effects of changing lung volume on parallel conductance are unknown.

The present study was undertaken to establish whether LV and RV parallel conductances are affected by lung insufflation volume and, if so, to what extent such changes are manifested in the volume signal fluctuations observed during ventilation in the anesthetized minipig. Fluctuations in conductance-derived LV and RV end-diastolic and stroke volumes associated with ventilation with various tidal volumes were recorded. Multiple determinations of parallel conductance were made, using a saline dilution technique, at end expiration and during steady-state lung insufflation at different lung volumes.

This paper also introduces simultaneous biventricular conductance volumetry, a method that may prove to be a valuable technique for the assessment of ventricular interaction.

METHODS

Instrumentation. The study was conducted on eight anesthetized Göttiinger minipigs (11–15 kg). Anesthesia was induced with pentobarbital sodium (75 mg/kg ip), and pigs were intubated and ventilated with 40% O_2 in air. The left femoral artery was cannulated (5-Fr single lumen) for taking blood samples and monitoring arterial blood pressure, and the right femoral vein was cannulated (7-Fr triple lumen) for administration of anesthetic and hypertonic saline. Anesthesia was maintained with a continuous intravenous pentobarbital infusion (0.2 mg·kg·min^(-1)) and adjusted to a level sufficient to suppress spontaneous breathing. Ventilation was adjusted to achieve normal blood gases. A Fleisch tube pneumotachogram was connected in series with the endotracheal tube, which was also connected via a sidearm to a pressure transducer. An eight-electrode, 5-Fr conductance catheter (Webster Laboratories, Irvine, CA) was inserted through a left carotid artery cutdown and advanced to the LV apex, and a 5-Fr micromanometer-tip catheter (Millar, Houston, TX) was advanced from the right carotid artery into the LV. A second 5-Fr micromanometer tip catheter was advanced into the RV through a right jugular vein cutdown. A 5-Fr, flow-directed, end-hole catheter was advanced from the left jugular vein into the pulmonary artery, through which an
0.018-in. exchange wire (Cordis, Miami, FL) was passed into a distal pulmonary artery. The flow-directed catheter was exchanged for a 6-Fr eight-electrode conductance catheter (Webster), which was advanced into the pulmonary artery. Interplay between the conductance catheter and exchange wire was used to form a curve in the catheter such that the proximal electrode was positioned at the RV apex with the distal electrode remaining in the pulmonary artery just above the pulmonary valve. A 5-Fr, flow-directed thermodilution catheter (Baxter, Irvine, CA) was advanced to the pulmonary artery from the left femoral vein. All catheters were positioned under fluoroscopic guidance. The final positions of the catheters within the heart are shown in Fig. 1.

Conductance catheter method. The conductance catheters with outer electrode separations ranging from 4.9 to 5.6 cm were connected to separate volume signal-conditioning units (BioMetrics, Las Vegas, NV and Cardiodynamics, Oegstgeest, The Netherlands) and computer data acquisition systems. The signal-conditioning units were set to different excitation frequencies (20.0 and 23.5 kHz) allowing simultaneous operation without interfering with one another. For both left and right ventricles, the five time-varying, segmental conductance (G) signals were combined to yield total volume signals (V) according to

\[ V(t) = \frac{1}{\alpha} L^2 \cdot \rho \cdot \sum_{i=1}^{5} G_i(t) - V_c \]

where \( L \) is the conductance catheter interelectrode separation, \( \rho \) is the measured specific electrical resistance of blood, \( \alpha \) is the conductance gain factor, and \( V_c \) is the offset term required due to parallel conductance. Correct conductance catheter positions and electrode separations were ascertained both fluoroscopically and by monitoring the pressure-volume loops of the segments in the LV and RV outflow tracts. If, with one excitation electrode at the ventricular apex, advancing the catheter slightly into the pulmonary artery (in the case of the RV catheter) or withdrawing it slightly into the aorta (in the case of the LV catheter) caused an outflow tract segmental volume-phase shift to occur at end systole (Fig. 2), then electrode separation and position were deemed to be correct. Synchronization of the two systems was achieved by issuing an electronic pulse transmitted simultaneously to both ventricular pressure channels at the beginning of each data acquisition episode. Data were digitally acquired at 250 Hz and 12-bit resolution with one system recording RV segmental volumes, RV pressure, right atrial pressure, pulmonary artery pressure, and electrocardiogram. The other system recorded LV segmental volumes, LV pressure, arterial blood pressure, electrocardiogram, and tracheal flow and pressure.

Protocol. After a 1-h stabilization period, specific electrical resistance of blood was measured, and baseline LV and RV pressure-volume data were acquired with the ventilator off at end expiration. Cardiac output was measured (Baxter COM1) by thermodilution (3 determinations using 1 ml of 0–5°C normal saline injections) and used to compute independent LV and RV conductance gain factors (\( \alpha \)). These values for \( \alpha \) were assumed to be constant throughout the protocol.

Parallel conductance was assessed using a saline dilution technique whereby the conductivity of blood was transiently increased by injection of a saturated saline solution (0.02–0.04 ml/kg). The injection was administered into the femoral vein for assessment of RV parallel conductance and into the

Fig. 1. Anteroposterior view showing position of catheters within the heart. RV, right ventricular; LV, left ventricular.

Fig. 2. A: left ventricular pressure segment 5 volume loops from one animal with segment 5 (proximal segment) completely in LV outflow tract. B: temporary, very slight withdrawal of catheter (on the order of 1–2 mm), results in a figure 8-shaped loop. This is most likely the result of the proximal segment being forced into aorta near end of systole by ventricular long-axis shortening. With catheter properly positioned, its tip at ventricular apex, formation of such a segment 5 pressure-volume loop suggests that the most proximal sensing electrode resides just below the aortic valve. A similar loop is formed by RV segment 1 when conductance catheter is advanced slightly into the pulmonary artery.
pulmonary artery for LV assessment. If ectopic beats occurred during saline washin or if heart rate or systolic ventricular pressure changed noticeably, the injection was repeated with a smaller volume. Analysis of the volume signal transient used to determine Vc, was by nonlinear regression of apparent stroke volume against maximum and minimum volumes, a method previously described for both the LV (21) and RV (18). Briefly, the method combines both maximum and minimum volume values in a single regression equation. Regressing maximum and subsequent minimum volumes of each beat during the washin period against the corresponding stroke volume yields two lines, the intersection of which occurs at the y-axis (stroke volume = 0 ml) at some intercept Vc (Fig. 3). A nonlinear regression technique is applied to solve for the parameters in a single regression equation combining minimum, maximum, and stroke volumes.

\[
VV = [m_{\text{min}} (SV - SV_0) + V_c] (1 - C) + [m_{\text{max}} (SV - SV_0) + V_c] C
\]

where VV is the ventricular volume (uncorrected for gain or offset), m\text{max} and m\text{min} are the slopes of the two regression lines, SV is stroke volume defined as maximum volume less minimum volume, and C is a coding variable set to 0 for minimum volumes and 1 for maximum volumes. The regression yields estimates for the parameters m\text{max} and m\text{min}, as well as SV_0 and V_c, the coordinate pair defining the intersection point of the two regression lines. Saline injection and Vc calculation were repeated three times, and an average value was computed for each ventricle at each step of the protocol.

Synchronized biventricular pressure-volume data were recorded during ventilation with tidal volumes of 10 and 20 ml/kg at 16 and 12 breaths/min, respectively. Data were also acquired under steady-state conditions and during hypertonic saline washin, with the ventilator off, at three lung insufflation volumes: first at ambient pressure, the endotracheal tube vented to the atmosphere; next with lungs insufflated to 10 ml/kg; and finally with an insufflation volume of 20 ml/kg. These recordings were ~20 s long, with onset of steady state occurring several cardiac cycles after the lungs were inflated and persisting for another 7–10 cardiac cycles (Fig. 4). A 5- to 10-min pause, during which normal ventilation was applied, was allowed between each lung volume maneuver.

Data analysis. To investigate the effects of ventilation tidal volume on changes to conductance-derived ventricular volume signals, end-diastolic and stroke volumes of each cardiac cycle over three contiguous ventilation cycles were calculated. Direct analysis of the relationship between tidal volumes and the position of the cardiac cycle within the ventilation cycle was not possible because of interanimal heart rate differences and relative timing of cardiac and ventilation cycles. Thus the three ventilation cycles were divided into 24 sections, each \(\pi/4\) radians wide (1/8 of a ventilation cycle). End-diastolic and stroke volumes that would occur at each \(\pi/4\) radian division were extrapolated based on a linear relationship between values falling immediately on either side of the division. A 2 \(\times\) 8 repeated-measures analysis of variance was then applied to test for significant effects of tidal volume (2 levels) and phase of the ventilation cycle (8 levels) on both LV and RV end-diastolic and stroke volumes. The changes in end-diastolic and stroke volumes that occurred during ventilation at both tidal volumes were also expressed as a percentage of the mean values. The maximum positive and negative deviations from the mean of both parameters were calculated. The maximum end-diastolic and stroke volumes during ventilation were compared using a paired sample \(t\)-test.

During steady-state lung insufflation, ventricular volume data are based on an average of 7–10 consecutive cardiac cycles. An analysis of variance for repeated measures (3 levels of lung inflation) was used to test for a significant effect on LV and RV V_c, end-diastolic volume, and stroke volume. A paired sample \(t\)-test was then used to establish whether the mean LV stroke volume was different from the mean RV stroke volume at the three steady-state lung insufflation volumes.

Hemodynamic stability during the measurement of parallel conductance at each insufflation volume was established by computing the coefficients of variation of heart rate and maximum rate of change of ventricular pressure (dP/dt max) over the cardiac cycles starting three beats before the onset of saline washin and ending with the beat at the peak volume transient. If either coefficient of variation for any given saline injection episode was >5%, that run was deemed hemodynamically unstable and was excluded from subsequent analysis. The precision of V_c measurement was assessed by computing the coefficient of variation of the three measurements of parallel conductance used to determine V_c for each ventricle at each lung insufflation volume. Analysis of variance for repeated measures was used to establish if V_c was dependent on lung insufflation volume. A paired sample \(t\)-test was used.
to determine whether $LV_V_c$ was significantly different from $RV_V_c$.

**RESULTS**

Unless otherwise stated, all data are expressed as means ± SD. The coefficient of variation of thermodilution measurements was $4.72 ± 2.13\%$. At baseline, with the ventilator off at end expiration, the $LV_a$ was $0.64 ± 0.12$ and $RV_a$ was $0.61 ± 0.18$.

Ventilation with tidal volumes of 10 and 20 ml/kg caused cyclic changes to both $LV$ and $RV$ volumes and pressures (Fig. 5). Analysis of variance revealed that both $LV$ and $RV$ end-diastolic and stroke volumes were dependent on the position of the cardiac cycle within the ventilation cycle ($P < 0.0001$ for all) with minimum values occurring near end inspiration (Fig. 6), the minimum $LV$ stroke volume lagging that of the $RV$ by one beat. Furthermore, the magnitude of the cyclic variation of both $LV$ and $RV$ end-diastolic and stroke volumes was related to ventilation tidal volume (Table 1). The mean $LV$ stroke volume of $10.31 ± 2.05$ ml during ventilation with a 10 ml/kg tidal volume was not significantly different from the $RV$ stroke volume $10.09 ± 1.73$. With a tidal volume of 20 ml/kg, the $LV$ stroke volume was $9.80 ± 2.17$ ml, also not different from the $RV$ value of $9.86 ± 2.07$ ml.

Steady-state lung insufflation at 10 and 20 ml/kg caused shifts in both $LV$ and $RV$ pressure and volume (Fig. 7). Lung insufflation volume-dependent decreases in both $LV$ stroke volume ($P < 0.002$) and $RV$ stroke...
volume (P < 0.0001) were observed. However, only the LV end-diastolic volume decreased (P < 0.0001) with steady-state lung insufflation, with RV end-diastolic volume showing no significant change (Table 2).

Of the combined total of 144 saline runs recorded for this study, 1 was omitted from LV analysis and 2 from RV analysis due to not meeting the hemodynamic stability test. Analysis of variance did not reveal any relationship between LV $V_c$ or RV $V_c$ and the degree of lung insufflation. Values of $V_c$ are presented in Table 3 as are the mean coefficients of variation of the repeated $V_c$ determinations made at each state of lung insufflation, which also were not related to lung insufflation volume. Parallel conductance for the RV was significantly, albeit slightly, lower than that measured for the LV (P < 0.05).

**DISCUSSION**

The conductance catheter technique is being increasingly applied to measure LV and RV volumes in both animals and humans. Whereas most studies may ben-
efit from the absence of hemodynamic perturbations associated with breathing or ventilation, suspending respiration during conductance measurements may not always be possible or even desirable (16). In those instances, it will be important to know the extent to which volume signal artifact is present. One possible component of that artifact would be parallel conductance associated with the degree of insufflation of the lungs. This study documented the changes to both LV and RV conductance-derived volumes during ventilation and investigated the effects of lung insufflation volume on LV and RV parallel conductances in an anesthetized minipig model.

Theoretically, the volume of air, an electrical insulator, within the lungs could affect conductance catheter parallel conductance. Changes in lung volume associated with respiration and ventilation could possibly be manifested as changes to conductance-derived volume signals. Specifically, if the electrical conductivity of the lungs were decreased during inspiration, a decrease in LV and RV parallel conductances would be expected.

Table 1. Maximum, minimum, and maximum fluctuation of LV and RV end-diastolic and stroke volumes observed during ventilation with 10 and 20 ml/kg tidal volumes

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<tr>
<th></th>
<th>10 ml/kg Tidal Volume</th>
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<th>20 ml/kg Tidal Volume</th>
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<tr>
<td></td>
<td>Maximum</td>
<td>Minimum</td>
<td>Difference</td>
<td>Maximum</td>
<td>Minimum</td>
<td>Difference</td>
</tr>
<tr>
<td>LV end-diastolic volume, ml</td>
<td>25.7 ± 6.1</td>
<td>24.7 ± 6.3</td>
<td>0.96 ± 0.32</td>
<td>26.5 ± 6.1</td>
<td>24.2 ± 6.4</td>
<td>2.26 ± 0.52</td>
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<tr>
<td>RV end-diastolic volume, ml</td>
<td>26.5 ± 7.4</td>
<td>24.8 ± 7.0</td>
<td>1.65 ± 1.26</td>
<td>28.3 ± 9.1</td>
<td>24.1 ± 9.7</td>
<td>4.15 ± 1.89</td>
</tr>
<tr>
<td>LV stroke volume, ml</td>
<td>10.8 ± 2.2</td>
<td>9.8 ± 2.0</td>
<td>1.03 ± 0.40</td>
<td>10.6 ± 2.6</td>
<td>8.8 ± 1.9</td>
<td>1.86 ± 0.76</td>
</tr>
<tr>
<td>RV stroke volume, ml</td>
<td>10.8 ± 1.7</td>
<td>8.7 ± 1.8</td>
<td>2.08 ± 0.41</td>
<td>10.9 ± 1.7</td>
<td>7.4 ± 2.0</td>
<td>3.47 ± 1.00</td>
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Data are means ± SD of 8 pigs over 3 contiguous ventilation cycles (n = 24 sections). LV, left ventricular; RV, right ventricular.
parallel conductance may follow, resulting in underestimation of absolute ventricular volumes. Another mechanism by which parallel conductance could be affected by lung insufflation is the degree to which the lungs are engorged with blood during the respiratory cycle. Whereas significant changes to both LV and RV conductance-derived stroke and end-diastolic volumes were observed with ventilation, assessment of parallel conductance during steady-state lung insufflation did not reveal any relationship between insufflation volume and parallel conductance, which remained essentially unchanged throughout the protocol. This can be explained by the relatively low conductance of air. Even a small amount of air, the residual capacity of the lungs after expiration, can provide sufficient electrical separation that increasing the lung volume further has little effect on $V_c$. It may require complete collapse of the lungs for this effect to have an impact on parallel conductance.

An indirect mechanism of affecting parallel conductance by lung insufflation would be that of changing volume of the contralateral ventricle, the blood pool of the opposite ventricle offering a source of parallel conductance. However, the effects of RV volume on LV parallel conductance are reportedly minimal (9, 21). Furthermore, some studies have indicated $V_c$ is volume dependent, LV $V_c$ being somewhat dependent on LV volume itself (2, 5). However, it has also been suggested that such volume dependency is more related to the method of achieving volume reduction, specifically, inferior vena cava occlusion (21). When LV parallel conductance was assessed at different ventricular volumes achieved by volume loading and depletion (which would necessarily have also affected RV volume), it was found to be relatively unchanged by the procedures (21). In the present study, parallel conductance was assessed under relatively steady-state conditions. It is possible that, under the dynamic conditions of respiration, $V_c$ may respond differently. However, because the effect of ventilation on conductance-derived RV volume was double that of the LV, one would expect that if $V_c$ were being affected to any extent by associated changes in actual LV or RV volumes, the mean conductance-derived ventricular outputs would have been different. Because this was not the case, we can assume that either there is little effect or the result is masked because the effects on $V_c$ are similar in both ventricles. In any case, if some ventricular volume-dependent effect on $V_c$ were present, at a normal tidal volume of 10 ml/kg, end-diastolic volumes only changed by ~5%, thus offering little cause for concern regarding this possible mechanism.

Ventilation caused significant fluctuations in LV and RV stroke volumes, the magnitude of which were related to tidal volume. Such cyclic changes in both LV and RV output during the respiratory cycle are well known (1, 6, 8, 12–14, 22). RV stroke volume decreases with inspiration as intrathoracic and intrapulmonary pressures increase, while decreases in LV stroke volume are smaller and lag those of the RV by a few beats. On expiration, RV stroke volume recovers almost immediately while that of the LV increases gradually. Given that ventilation causes larger changes to RV stroke volume, those changes must be more abrupt with higher maximum and lower minimum values than the LV in order to maintain equal mean RV and LV outputs.

Table 2. LV and RV stroke and end-diastolic volumes measured during steady-state lung insufflation with volumes of 0, 10, and 20 mg/kg

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<th>LV</th>
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<th>RV</th>
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<td></td>
<td>0 ml/kg</td>
<td>10 ml/kg</td>
<td>20 ml/kg</td>
<td>0 ml/kg</td>
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<tr>
<td>Stroke volume, ml</td>
<td>10.3 ± 2.0</td>
<td>9.9 ± 2.1</td>
<td>8.2 ± 1.5</td>
<td>10.3 ± 1.8</td>
</tr>
<tr>
<td>End-diastolic volume, ml</td>
<td>23.8 ± 2.1</td>
<td>22.3 ± 2.7</td>
<td>18.1 ± 1.9</td>
<td>25.8 ± 9.0</td>
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</table>

Data are means ± SD; n = 8 pigs. The relatively large RV end-diastolic volume standard deviation (SD) was due to one animal’s unusually large RV, the SDs falling below 4.0 ml when that animal was excluded.
Such events have led the RV to be described as a buffer of changes in systemic venous return, effectively dampening respiratory-induced variations of LV stroke volume (17). Observations made in the present study are in keeping with the cited work as can be seen in Fig. 5; fluctuations in RV stroke volume almost double those of the LV with maximum decreases to both occurring near end inspiration, the LV effect lagging the RV by at least a beat.

Some of the observed changes to stroke volume could have been a result of changes to \( \alpha \); however, because of the design of the protocol, possible effects on \( \alpha \) can only be speculated on, which is a limitation of this study. Whereas it is well established that both LV (15, 16) and RV (7, 18) \( \alpha \) values are somewhat inversely related to ventricular volume, there is no reason to suspect that changes in lung volume would directly affect this parameter. Rather, an indirect effect could again be postulated in that lung insufflation does alter absolute ventricular volumes and thereby could also change \( \alpha \). As ventricular volumes decrease during inspiration, a concomitant increase in \( \alpha \) may be expected. In the present study, for each ventricle \( \alpha \) was determined at the beginning of the protocol and thereafter was assumed to be constant. Thus both stroke and end-diastolic volumes may be increasingly overestimated with increasing lung insufflation. Whereas such overestimation would not affect the conclusions drawn by this study regarding \( V_c \), changes to LV and RV stroke and end-diastolic volumes with ventilation may have been underestimated. If \( \alpha \) were affected by ventilation, the change must have been similar in both LV and RV because there was no significant difference between the mean LV and RV stroke volumes during ventilation with 10 and 20 ml/kg tidal volumes. Steady-state lung insufflation at 0, 10, and 20 ml/kg did result in insufflation volume-related decreases to both LV and RV stroke volume with that of the RV decreasing more than the LV, although not significantly. Because during steady state one would expect both ventricles to have the same stroke volume, this could suggest that the LV \( \alpha \) was affected more than that of the RV. However, because the excursion of end-diastolic volume with ventilation with a normal tidal volume of 10 ml/kg was relatively small, on the order of 5%, substantial changes to \( \alpha \) do not seem likely. The changes in absolute ventricular volumes associated with the normal ejection (stroke volume) of both ventricles, which are greater than those due to normal lung inflation, have previously been shown to have a minimal effect on \( \alpha \) (18, 20).

Ventilation also resulted in fluctuations in both LV and RV end-diastolic volume. Again, the effect on the RV was double that of the LV, with minimum end-diastolic volumes occurring at end inspiration. As with stroke volume, recovery of RV end-diastolic volume occurred almost immediately on expiration, whereas that of the LV was more gradual, not reaching a maximum value plateau before onset of the next inspiratory phase. Because this may be the first study to record absolute ventricular volumes during respiration, there is little in the literature with which to compare these findings. In the context of this study, though, the most important observation regarding end-diastolic volume is that absolute ventricular volumes only changed minimally (relative to stroke volume for example) with insufflation, thus limiting the impact that ventricular volume changes may have had on either \( \alpha \) or \( V_c \).

When continuous lung insufflation was applied, following a transient of a few cardiac cycles (Fig. 6), RV end-diastolic volume returned to its preinsufflation value, whereas stroke volume remained decreased. RV end-diastolic volume assumed a value falling between the maximum and minimum observed during ventilation, whereas LV end-diastolic volume reached a plateau at a value even less than the minimum observed during ventilation. During this sustained insufflation, hemodynamic signals remained stable sufficiently long enough to assess parallel conductance, with only three saline runs being rejected from analysis due to hemodynamic instability. Inflation of the lungs was also stable during this period as confirmed by both elevated, yet constant, end-diastolic pressures and stable intrathoracic pressure. We are, therefore, confident that parallel conductance measurements were reliable and were, in fact, made at different lung volumes.

This paper introduces the use of the conductance catheter technique to measure pressure-volume data of both ventricles, simultaneously offering an exciting prospect for a novel method of studying ventricular interaction. No technical obstacles were apparent in using the technique once it was ascertained that the two volume signal-conditioning units did not interfere electrically with one another. The method simply combines LV and RV techniques already described with the addition of an electrical synchronizing pulse recorded by both systems. The synchronizing signal would not have been necessary if data were recorded on a single computer. The use of double bolus injections of hypertonic saline for assessment of LV and RV parallel conductances also may not be required because the femoral vein injection used for RV \( V_c \) also resulted in a

### Table 3. LV and RV \( V_c \), measured at insufflation volumes of 0, 10, and 20 mg/kg

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<th>LV</th>
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<tr>
<td></td>
<td>0 ml/kg</td>
<td>10 ml/kg</td>
<td>20 ml/kg</td>
</tr>
<tr>
<td>( V_c, \text{ ml} )</td>
<td>34.1 ± 3.1</td>
<td>34.1 ± 4.1</td>
<td>35.3 ± 3.1</td>
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<tr>
<td>Coefficient of variation, %</td>
<td>4.4 ± 1.8</td>
<td>4.8 ± 2.4</td>
<td>2.1 ± 1.2</td>
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<th>LV</th>
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<td></td>
<td>0 ml/kg</td>
<td>10 ml/kg</td>
<td>20 ml/kg</td>
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<tr>
<td>( V_c, \text{ ml} )</td>
<td>31.1 ± 4.4</td>
<td>31.2 ± 3.7</td>
<td>30.7 ± 4.9</td>
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<tr>
<td>Coefficient of variation, %</td>
<td>5.8 ± 3.7</td>
<td>5.0 ± 4.6</td>
<td>7.1 ± 3.6</td>
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Data are means ± SD; \( n = 8 \) pigs. Each value of parallel conductance \( (V_c) \) is the mean of 3 independent measurements made at each insufflation volume and the coefficient of variation is the mean coefficient of variation of those 3 measurements.
transient in the LV signal, which could be used for computation of LV $V_c$.

In conclusion, in this study, there was no evidence that the degree of lung insufflation affects either LV or RV conductance catheter parallel conductance. The possibility that $\alpha$ may have changed as a result of the effects of lung insufflation on absolute ventricular volumes should not preclude use of the technique during normal ventilation or respiration, since the absolute volume of either ventricle changes a great deal under those circumstances. It should be noted that these results may not be valid under conditions of spontaneous respiration, during which the diaaphragm may move in and out of the conductance catheter excitation field, thus altering parallel conductance. Furthermore, pericardial or pleural effusions may increase both mechanical and electrical coupling of both ventricles with the lungs and other extracardiac structures, thus allowing the possibility of changes to both $\alpha$ and $V_c$ with ventilation.

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