One versus Two MAC Halothane Anesthesia Does Not Alter the Left Ventricular Diastolic Pressure–Volume Relationship

Elliott S. Greene, M.D.,* John I. Gerson, M.D.†

Previous studies on halothane's effect on left ventricular diastolic compliance (LVDC) not only have had conflicting results, but are not directly applicable to most intraoperative settings. Therefore, the authors examined in dogs whether the depth of halothane anesthesia alters LVDC under surgical conditions over a wide range of hemodynamic stresses with the cardiovascular reflexes intact. The left ventricular diastolic pressure–volume relation was examined at 1 MAC and 2 MAC halothane in seven dogs over wide ranges of preload and afterload during left thoracotomy. Pulmonary capillary wedge pressure (PCWP), left ventricular end-diastolic pressure (LVEDP), and echocardiographic left ventricular end-diastolic volume (LVEDV) were analyzed with the exponential pressure–volume relation \( P = A e^{BV} \) (where \( P = \) pressure, \( V = \) volume, and \( A \) and \( B \) are empirically derived coefficients). Multivariate analysis showed no significant differences for diastolic pressure–volume relations, comparing both levels of halothane using either PCWP or LVEDP for pressure. The authors conclude that in the intact cardiovascular system in the healthy open-chest dog: 1) LVEDV does not change with the depth of halothane between 1 and 2 MAC (it is still possible LVEDV changed between 0 and 1 MAC) and 2) PCWP does reflect the LVEDV during halothane anesthesia (between 1 and 2 MAC) under surgical conditions over a wide range of cardiovascular stresses. (Key words: Anesthetics, volatile: halothane. Blood pressure: pulmonary capillary wedge; left ventricular end-diastolic. Heart: compliance; preload; volume.)

HALOTHANE HAS BEEN shown to decrease left ventricular contractility in numerous studies. However, investigations of halothane's effect on left ventricular diastolic compliance have produced conflicting results. Several studies showed decreased compliance, whereas others reported no change. These studies are not directly comparable to most intraoperative situations because they did not examine left ventricular diastolic compliance under surgical conditions with the cardiovascular system intact. Pulmonary capillary wedge pressure (PCWP) is the most widely used left ventricular preload measurement. As the depth of halothane anesthesia increases, we need to know whether PCWP still reflects left ventricular end-diastolic volume (LVEDV) or whether compliance changes, which would necessarily alter the pressure–volume relationship. This information is needed to determine if changes in left ventricular filling pressure (i.e., PCWP) are due to altered left ventricular contractility or due to changes in left ventricular diastolic compliance. This ability to distinguish changes of contractility from changes of diastolic compliance is also important when we use PCWP as preload in Starling curves to assess cardiac function.

The purpose of this study was to investigate the effect of halothane on left ventricular diastolic compliance under surgical conditions with the cardiovascular system intact. Pharmacologically induced cardiovascular stresses were used to produce wide pressure ranges of preload and afterload that may have been seen during surgical stimulation. We investigated left ventricular diastolic compliance by examining the PCWP–LVEDV relationship and the left ventricular end-diastolic pressure (LVEDP)–LVEDV relationship.

Methods

Seven mongrel dogs, weighing 21.0 to 29.8 kg, were anesthetized with halothane and oxygen via a face mask and intubated. Ventilation was controlled using a Harvard respiration pump delivering 15 ml/kg tidal volume with 5 cm H2O positive end-expiratory pressure. Anesthesia was maintained with halothane, oxygen, and air. Metocurine was given in 6- to 8-mg increments for muscular relaxation to prevent diaphragmatic movement (typically every 45–60 min). The following catheters were inserted: femoral arterial and venous catheters, a 7F pulmonary artery catheter via the external jugular vein (American Edwards), and a left ventricular 7F Millar Mikro-Tip Catheter via the carotid artery. Arterial blood gases (ABGs) were checked on average every half-hour to maintain \( \text{PaO}_2 > 100 \text{ mmHg, normocarbia, and normal pH. ABGs were checked more frequently, as indicated, to maintain a stable preparation. Metabolic acidosis was corrected with intravenous sodium bicarbonate when the base excess was less than –5 meq/l. To maintain the dog's temperature at 36–38° C (measured with the pulmonary artery catheter thermistor), we used a humidifier warmer in the breathing circuit, warmed the laboratory room to 75–80° F, and minimized convective heat losses. Lactated Ringer's solution was infused at 6–8 ml · kg\(^{-1}\) · h\(^{-1}\).

Following a left thoracotomy in the lateral position and rib spreader insertion, the pulmonary artery catheter was confirmed to be in the right pulmonary artery. An M-
mode echocardiographic transducer (Smith Kline Instruments® model E20A/S, 2 MHz frequency) was positioned on the intact pericardium overlying the right ventricle in the same direction as the standard left parasternal view defined by the American Society of Echocardiography (ASE) (fig. 1).9 The echocardiographic transducer direction was held constant by using external and internal reference points. The external position was fixed by marker clips on the pericardium. In each echocardiographic recording, the proper transducer direction to measure the left ventricular internal dimension in the minor axis was ensured by identifying the point of maximum excursion of the anterior and posterior mitral valve leaflets. All recordings were made at 100 mm/s. Left ventricular internal end-diastolic diameter (LVID) was measured at the time of the electrocardiographic "Q" wave (according to the criteria of the ASE). Using the prolate ellipsoid model,9,10 LVEDV equals LVID3. LVID3 provided the volume measurement of left ventricular preload. The pressure measurements of left ventricular preload were mean PCWP and LVEDP (measured at the "Z" point prior to ventricular contraction.11)

The left ventricular pressure (PCWP and LVEDP) volume (LVID3) relationships were studied at 1 MAC and 2 MAC halothane (1 MAC halothane in dogs12 equals 0.87%). A steady-state, end-tidal halothane (Beckman® LB-2 infrared analyzer) was maintained for at least 20 min prior to data measurements. At each anesthetic dose, PCWP was varied randomly over a range of approximately 5 to 25 mmHg using constant-rate infusions of sodium nitroprusside (typical PCWP range: 5–10 mmHg) and phenylephrine (typical PCWP range: 10–25 mmHg) via a Harvard Pump® infusion apparatus. A constant PCWP was maintained for 10 min to ensure steady-state hemodynamics prior to each set of pressure and echocardiographic measurements. Drug infusion rates depended on the dose required to maintain a desired PCWP level. Dose ranges used were: sodium nitroprusside 1.4–25 μg · kg⁻¹ · min⁻¹ and phenylephrine 0.7–8.8 μg · kg⁻¹ · min⁻¹. Simultaneous PCWP and LVID data were averaged over two respiratory cycles to equal one pair of pressure–volume data. An average of 12 pairs of pressure–volume data were obtained at each level of halothane in each dog. Mean arterial blood pressure (BP), PCWP, central venous pressure (CVP), and electrocardiogram (modified V₅ lead) were recorded at 25 mm/s on a Hewlett-Packard® multichannel recorder. Pressure was measured electronically. Recordings were not used if the heat was not in normal sinus rhythm or if electrocardiographic evidence of ischemia was present.

PCWP and LVEDP were each used as pressure (P), and LVID3 was used as volume (V) in the exponential pressure–volume relation P = AeBV, where A and B are the empirical coefficients that define the pressure–volume relationship.

### TABLE 1. Range of Cardiovascular Variables for All Dogs Combined (minimum and maximum values presented as mean ± SD)

<table>
<thead>
<tr>
<th>Halothane (MAC)</th>
<th>PCWP (mmHg)</th>
<th>LVEDP (mmHg)</th>
<th>LVID3 m⁻³ (cm³ · m⁻³)</th>
<th>SVR (dyn · s · cm⁻⁵)</th>
<th>CI (l · min⁻¹ · m⁻²)</th>
<th>HR (beat/min)</th>
<th>CVP (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4.9 ± 1.5</td>
<td>6.8 ± 1.2</td>
<td>75 ± 24 − 144 ± 44</td>
<td>1527 ± 657 − 444 ± 1712</td>
<td>2.5 ± 0.88 − 5.35 ± 1.70</td>
<td>2.3 ± 1.3 − 14.0 ± 5.7</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>6.8 ± 1.2</td>
<td>5.8 ± 2.0</td>
<td>89 ± 26 − 151 ± 59</td>
<td>1460 ± 694 − 4697 ± 1036</td>
<td>1.97 ± 0.51 − 2.94 ± 0.75</td>
<td>9 ± 13 − 121 ± 17</td>
<td>3.1 ± 2.1 − 12.9 ± 3.5</td>
</tr>
</tbody>
</table>

Abbreviations: PCWP = pulmonary capillary wedge pressure; LVEDP = left ventricular end-diastolic pressure; LVID = left ventricular internal end-diastolic diameter; BP = mean arterial blood pressure; SVR = systemic vascular resistance; CI = cardiac index; HR = heart rate; CVP = central venous pressure.
curves for each dog at each level of halothane anesthesia. The exponential equation was converted to the logarithmic form: \( \ln(P) = \ln(A) + B \cdot V \) for linear regression analysis at each anesthetic dose.\(^{13}\) The intercepts and regression coefficients for both doses of halothane were compared with multivariate analysis using Hotelling’s \( t^2 \) test with a significant level at \( P < 0.05.\(^{14}\)

**Results**

The minimum and maximum per cent end-tidal halothane concentrations in all seven dogs were, at 1 MAC: 0.82 ± 0.05 to 0.87 ± 0.07 (mean ± SD) and, at 2 MAC: 1.68 ± 0.10 to 1.77 ± 0.10 (mean ± SD). Wide ranges of cardiovascular stress were produced during the experiments (table 1). We achieved a wide range of PCWP consistent with the experimental design. The mean PCWP range was 4.9–21.0 mmHg and 6.6–23.0 mmHg at 1 MAC and 2 MAC halothane, respectively. Mean LVEDP varied from 4.3 to 21.7 mmHg at 1 MAC halothane and 5.8 to 26.0 mmHg at 2 MAC halothane. The mean CVP was 2.3–14.0 mmHg at 1 MAC halothane and 3.1–12.9 mmHg at 2 MAC halothane. The mean heart rate was less extreme in its ranges: from 74.9 to 129.6 beats/min at 1 MAC halothane and from 90.9 to 121.0 beats/min at 2 MAC halothane.

The pressure–volume data fit the logarithmic pressure–volume relationship well for each dog at each level of halothane. Using PCWP for pressure, the mean correlation coefficient for all data was \( r = 0.83 ± 0.12 \) (SD); all correlations in the individual dogs are significant with \( P < 0.05 \) (table 2). Figure 2 is an example of actual pressure–volume data from dog 5. Using LVEDP for pressure, the mean correlation coefficient for all data was \( r = 0.85 ± 0.10 \) (SD); for all correlations in each dog, \( P < 0.05 \) (table 3). Analysis of \( \ln(A) \) and \( B \) for the logarithmic PCWP–volume relationship (table 2), using Hotelling’s \( t^2 \) test, revealed no significant difference between 1 MAC and 2 MAC halothane (\( P > 0.05 \)). Analysis of \( \ln(A) \) and \( B \) for the logarithmic LVEDP–volume relationship (table 3), using Hotelling’s \( t^2 \) test, revealed no significant difference between 1 MAC and 2 MAC halothane (\( P > 0.05 \)).

**Discussion**

Our study indicates that the left ventricular diastolic pressure–volume relationship was unchanged, comparing 1 MAC to 2 MAC halothane anesthesia during surgical and pharmacologic stresses in the intact cardiovascular system. This result holds true whether PCWP or LVEDP is used for pressure. Therefore, left ventricular diastolic compliance did not change at these different levels of hal-
HALOTHANE DOES NOT ALTER LEFT VENTRICULAR COMPLIANCE

othane. Thus, PCWP reflects LVEDV over a wide range of hemodynamic stresses, despite changes in the depth of halothane anesthesia from 1 to 2 MAC.

Previous studies on the effect of halothane on left ventricular diastolic compliance have had conflicting results. This may be the result of different experimental models used and different methods for measuring compliance. Experimental design varied, including in vitro testing on ventricular muscle, right heart bypass, and relatively intact animal preparations not undergoing surgery. Their direct relevance to most clinical situations is limited because these studies did not examine left ventricular diastolic compliance during surgical and pharmacologic stresses with the autonomic reflexes intact. In contrast, our study was designed using these considerations.

The experimental model was designed to be closely comparable to the anesthetized patient undergoing surgery. Surgical stress cannot be titrated as reproducibly as can pharmacologic stress. Therefore, we used a pharmacologic vasoconstrictor and vasodilator to reproduce the hemodynamic conditions of vasoconstriction and vasodilation which occur during surgery. An acute preparation used a thoracotomy with the cardiovascular system left anatomically intact. This provided two factors we wanted: 1) a baseline level of acute surgical stimulation from the rib spreader, and 2) since the left chest was opened to the atmosphere, intrapleural pressure did not alter intravascular pressure readings, nor did it act as a potential external mechanical constraint on the left ventricle that would shift the diastolic pressure-volume relationship. Rather than pacing the heart, we left the heart rate dependent on autonomic tone, humoral effects, cardiac and baroreceptor reflexes, and anesthetic influences that occur in the anesthetized patient. This also left intact any potential direct or reflex changes in the pressure-volume relationship that might hypothetically depend on the depth of halothane anesthesia. By intentionally allowing heart rate to vary, we believe this enhanced the relevance of our study to most clinical situations. There is no effect of increasing heart rate on ventricular diastolic compliance until the rate averages 178-208 beats/min or greater. As seen in table 1, the heart rates in our study were well below this range. The ranges of heart rate were quite comparable at both levels of halothane (table 1). There was less range of heart rate at 2 MAC than 1 MAC halothane, as might be expected from greater depression of baroreceptor reflexes.

Vasoactive drug infusions were used to induce cardiovascular stresses for several reasons. They varied the volume (preload) returning to the heart and reproduced the wide range of systemic vasoconstriction, vasodilation, and blood pressure changes (afterload) that can occur during surgery. While we were able to obtain desired end results (hemodynamic changes), we cannot claim that the entire cascade of neurologic and metabolic events accompanying pure surgical stimulation also took place in our model, which used pharmacologic therapy as well as surgery (thoracotomy). However, experimentally-induced surgical stimulation cannot be quantified to produce the steady-state hemodynamic changes we required for data acquisition and might cause marked tachycardias as well. Vasoactive drug infusions often are used during surgery. However, no agent is known to affect the passive elasticity of normally oxygenated papillary muscle. Neither nitroprusside nor angiotensin is known to affect the passive elasticity of normally oxygenated cardiac muscle. Proper arterial oxygenation was maintained throughout our experiments, and there was no electrocardiographic evidence of myocardial ischemia for the data used. It is clearly supported by the previously cited study that nitroprusside, "of itself," should not have altered the results of our study.

Brodie et al. studied the effects of nitroprusside on left ventricular diastolic pressure-volume relations in patients with clinical evidence of congestive heart failure. In agreement with Alderman and Glantz, they demonstrated a downward displacement of the pressure-volume curve with nitroprusside. Brodie et al. felt the most likely explanations for this downward shift were that, "Nitroprusside either improves left ventricular relaxation, affects left ventricular viscous properties, or alters one or more of the external constraints acting upon the left ventricle. However, in their discussion, Brodie et al. discount altered ventricular relaxation as a factor in the nonhypoxic model. Brodie et al. leave open the possibility of autonomic-nervous-system-mediated relaxation effects from nitroprusside and cannot entirely eliminate a direct relaxing effect of nitroprusside. While Alderman and Glantz cannot eliminate the possibility that nitroprusside

![Graph showing pressure-volume relationship](Image)
alters intrinsic myocardial compliance, and they also state that shifts in the pressure–volume curve may be due to effects on left ventricular viscoelastic properties, they emphasize that these shifts are due "more likely, to external mechanical loading on the left ventricle produced by changes in right ventricular pressure and . . . by restrictive action of the pericardium."\(^{15}\) Further on in this discussion, we address why we feel it unlikely that viscoelastic effects or external mechanical constraints influenced our results.

We must cite indirect but, we feel, compelling, evidence that phenylephrine either does not "of itself" affect left ventricular diastolic compliance or, at least, does not affect the conclusion that there is no change in ventricular diastolic compliance between 1 and 2 MAC halothane. We can find nothing in the literature concerning phenylephrine directly, but we have found data on another agonist, methoxamine, which is a vasoconstrictor, like angiotensin cited above. Methoxamine was found to affect left ventricular compliance only in that it increased left ventricular filling rate.\(^{18}\) Noble's paper postulated that methoxamine affected left ventricular compliance by increasing diastolic filling rate in the rapid filling phases of early and late diastole; middiastolic compliance was not affected by methoxamine. Viscoelastic properties of cardiac muscle, which are sensitive to the rate of volume change, may influence the pressure–volume relationship, particularly during atrial systole.\(^{15}\) We examined our data to look for such an effect. Specifically, we investigated the relationship of LVEDP to PCWP to see if late diastolic filling or ventricular compliance was altered at 1 MAC versus 2 MAC halothane.

There is normally a close correlation between LVEDP and PCWP,\(^{19,20}\) Furthermore, there is a good correlation of PCWP with LVPD pre-"a" wave.\(^{20,21}\) However, Rahimtoola demonstrated that PCWP did not accurately reflect LVEDP in patients with acute myocardial infarction.\(^{21}\) They showed a larger difference between LVEDP and PCWP when the LVEDP was greater than 12 mmHg.\(^{21}\) Thus, LVEDP is expected to be greater than PCWP when there is either a significant atrial contribution to ventricular filling or with reduced LV compliance.\(^{21,22}\)

If either altered ventricular filling in late diastole or altered left ventricular compliance occurred in our experimental model using phenylephrine at 1 MAC compared to 2 MAC halothane, we would expect to see a difference in the relation between LVEDP and PCWP. To investigate this we used the relationship LVEDP = A + B (PCWP) for linear regression analysis in each dog at each level of halothane. The mean correlation coefficient

### Table 4. Left Ventricular End-diastolic Pressure Versus Pulmonary Capillary Wedge Pressure Data where: LVEDP = A + B (PCWP)

<table>
<thead>
<tr>
<th>Dog*</th>
<th>1 MAC Halothane</th>
<th>2 MAC Halothane</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
<td>n†</td>
</tr>
<tr>
<td>2</td>
<td>0.988‡</td>
<td>14</td>
</tr>
<tr>
<td>3</td>
<td>0.983‡</td>
<td>12</td>
</tr>
<tr>
<td>4</td>
<td>0.971‡</td>
<td>11</td>
</tr>
<tr>
<td>5</td>
<td>0.980‡</td>
<td>12</td>
</tr>
<tr>
<td>6</td>
<td>0.990‡</td>
<td>12</td>
</tr>
<tr>
<td>7</td>
<td>0.955‡</td>
<td>12</td>
</tr>
</tbody>
</table>

* No LVEDP data were obtained in dog 1.
† n = Number of LVEDP versus PCWP measurement pairs.
‡ P < 0.001.
HALOTHANE DOES NOT ALTER LEFT VENTRICULAR COMPLIANCE

for all data was $r = 0.974 \pm 0.025$ (SD); for all correlations in each dog, $P$ was less than 0.001. Analysis of A and B for this relationship (table 4), using Hotelling’s $t^2$ test, revealed no significant difference between 1 MAC and 2 MAC halothane ($P > 0.05$). This means that either halothane and phenylephrine each had no effect on ventricular compliance and late diastolic ventricular filling, or that each drug had precisely opposing and cancelling effects. We think the former possibility is more likely, but we cannot exclude the latter.

Even if the foregoing evidence is discounted, the use of phenylephrine does not affect the conclusion that halothane does not alter ventricular compliance (between 1 and 2 MAC) because we used phenylephrine at both 1 MAC and 2 MAC halothane under similar conditions and in the same range of filling pressures by experimental design. The most that could be said against the conclusion is that it is limited (in the high range of LVEDP only) to the concurrent effects of halothane and phenylephrine.

In addition, phenylephrine was used as the vasopressor to avoid the positive inotropic effects and tachycardia that can occur with other vasopressor agents. In some circumstances, vasoactive drugs may affect left ventricular diastolic compliance via ventricular septal shift due to changes in right ventricular filling pressures with the pericardium intact. Alderman and Glantz state that, “External mechanical constraints on the left ventricle such as the right ventricular pressure and the pericardium and perhaps viscoelastic effects related to changes in filling rate account for the pressure–volume curve shifts with their pharmacologic interventions using sodium nitroprusside and angiotensin.” They indicate that mechanical changes in right ventricular diastolic filling pressure with the pericardium intact, and not pharmacologic factors, are felt to be most likely responsible for shifts in their left ventricular pressure–volume curves. However, mechanical changes in right ventricular diastolic filling pressure should not have altered our results because the right ventricular filling pressures (CVP) were similar at both levels of halothane (table 1). Any effect of the pericardium on the pressure–volume curve should have been the same at 1 MAC and 2 MAC halothane anesthesia, since the pericardium was left intact throughout the experiment. As previously explained, there was no evidence to suggest that halothane and phenylephrine altered left ventricular compliance by changing the left ventricular diastolic filling rate. Thus, there was no evidence of viscoelastic effects related to potential changes in the ventricular diastolic filling rate resulting from phenylephrine administration.

Therefore, we do not have any evidence that suggests the vasoactive agents used in this study altered myocardial muscle stiffness intrinsically or otherwise directly or indirectly altered the diastolic pressure–volume relationship comparing 1 MAC to 2 MAC halothane. This supports the use of these agents in our experimental model.

Finally, the intent of our study was to examine the left ventricular diastolic pressure–volume relationship under conditions of pharmacologic stress added to baseline surgical stress rather than under conditions of volume loading. Previous workers who have looked at halothane’s effects on ventricular compliance in intact hearts have employed either volume loading or no pharmacologic stress aside from the anesthetic itself.

The echocardiographic measurements have a good resolution (1 mm), which is about 2% of a typical LVID of 4.5 cm. Esophageal and external echocardiographic recordings were attempted initially but technically were unfeasible. Direct echocardiographic recordings on the pericardial surface permitted repeated measurements of dimension data while maintaining a stable acute preparation. The prolite ellipsoid model used for left ventricular volume calculations, where volume equals LVID$^3$, is a standard model used for approximating left ventricular geometry. The mean correlation coefficient ($r$) equals 0.83 for the pressure–volume relations using our echocardiographic dimension data and the prolite ellipsoid model with PCWP data, and $r$ equals 0.85 using LVEDP data. This compares extremely well with pressure–volume relation correlation coefficients found in studies using angiography for volume measurements where $r$ equals 0.89. This agreement supports both our use of echocardiographic dimension measurements to estimate ventricular volume in the acute preparation and the use of the mathematical model employed (see following).

An awake control state was not obtained due to the experimental methodology. Therefore, we cannot say how awake left ventricular pressure–volume relations change from 0 to 1 MAC. However, we can say that a change in the depth of halothane anesthesia from 1 to 2 MAC does not affect left ventricular diastolic compliance.

The mathematical model $P = Ae^{BV}$ describes the exponential pressure–volume relationship seen in the ventricle in diastole. The coefficients A and B are empirical constants that describe the pressure–volume relationship. A and B cannot be related to intrinsic myocardial muscle stiffness, nor was that the object of this study. Since the diastolic pressure–volume relationship is exponential for pressures from 2–5 mmHg up to 30 mmHg, the model should be valid over the clinical range from 5–25 mmHg we examined.

A recent study (appearing in abstract form) in humans used transesophageal 2-D echocardiography for left ventricular short-axis cross-sectional area measurements and compared them to PCWP. They found poor correlations...
(r < 0.30) for 77% of patients when comparing echocardiographic left ventricular end-diastolic area with PCWP. The patients were undergoing renal transplantation, aortic reconstruction, or coronary revascularization; however, the type of anesthesia used was not specified, so a direct comparison to our study is not possible. They conclude the poor correlations they saw indicate that alterations in left ventricular compliance are commonplace during these cardiovascular procedures. However, they did not provide supporting data indicating what factors were etiologic for changes in ventricular compliance in their patients. Numerous reasons for changes in ventricular compliance have been well documented, including myocardial ischemia, marked tachycardia, heart failure, hypothermia, changes in intrapleural pressure, or altered right ventricular loading with septal shift due to either positive end-expiratory pressure or altered right ventricular diastolic pressure from drug therapy or pulmonary hypertension. Alderman and Glantz concluded that the diastolic pressure-volume curve shifts they observed were due to use of end-diastolic pressure interchangeably with end-diastolic fiber length when interpreting experimental or clinical results in terms of the Frank-Starling mechanism. Inherent, left ventricular end-diastolic pressure and PCWP are not adequate measurements or predictors of left ventricular end-diastolic volume under conditions known to change left ventricular diastolic compliance.

In conclusion, we found no change in left ventricular diastolic compliance at 1 MAC compared to 2 MAC halothane anesthesia. Our study not only confirms previous work by using a completely different experimental model and method for measuring compliance, but also extends the results to include the surgical period over a wide range of hemodynamic stresses with cardiovascular reflexes intact. The conclusions drawn from this study apply to the healthy open-chest dog. In the results of our study and the results of the three previous studies that support our finding can be applied to anesthesia in humans, then we conclude that PCWP can be used to estimate LVEDV during halothane anesthesia between 1 and 2 MAC in the intact cardiovascular system when no changes in left ventricular compliance occur due to well-documented causes previously mentioned. Thus, during surgery with halothane anesthesia (1 to 2 MAC), left ventricular diastolic compliance can be measured and changes in compliance occur, they are not due to changes in the depth of halothane anesthesia. Rather, other causes must be considered and treated appropriately.

The authors gratefully acknowledge Frances Kriese for technical assistance, Paul R. Sheeehe, Sc.D., for assistance in statistical analysis, and Florence Starr for typing the manuscript.

References


---


