Left Ventricular End-systolic Cavity Obliteration as an Estimate of Intraoperative Hypovolemia

Jacqueline M. Leung, M.D.,* Edward H. Levine, B.S.†

Background: Transesophageal echocardiography is increasingly used intraoperatively as a monitor of ventricular function and volume. Although obliteration of the left ventricular (LV) cavity at end-systole is interpreted as indicative of intraoperative hypovolemia, this relation has not been demonstrated directly.

Methods: We continuously monitored the LV short axis by using transesophageal echocardiography and determined the relation between acute changes in LV area and hemodynamic variables in 139 patients undergoing elective coronary artery bypass graft surgery. The end-diastolic areas (EDA) and end-systolic areas were calculated during the control state (after anesthetic induction) and during LV end-systolic cavity obliteration.

Results: Thirty-nine of 139 patients had episodes of LV cavity obliteration. Mean LV end-systolic area decreased significantly from the control to obliterated state (7.29 ± 2.56 to 4.60 ± 1.46 cm², P = 0.0001). The corresponding mean LV EDA also significantly decreased from the control to obliterated state (18.18 ± 4.36 to 12.92 ± 3.74 cm², P = 0.0001). Mean ejection fraction area increased from 0.609 ± 0.095 (control) to 0.692 ± 0.083 (obliteration) (P < 0.0001). Of these 39 episodes, 31 (80%) were associated with a greater than 10% decrease in EDA relative to the initial value after induction of anesthesia and tracheal intubation; 4 (10%) with increases in ejection fraction area only; and an additional 4 (10%) with no substantial change in either the EDA or ejection fraction area. Overall, LV cavity obliteration was not associated with hemodynamic changes.

Conclusions: Our study demonstrates that LV cavity obliteration is rarely preceded by any acute alteration in hemodynamic parameters. Although end-systolic cavity obliteration detected by intraoperative transesophageal echocardiography is frequently associated with decreases in EDA, not every instance of end-systolic cavity obliteration is indicative of decreased left ventricular filling. (Key words: Heart: hemodynamics; preload. Measurements techniques: transesophageal echocardiography. Surgery, cardiac: coronary artery bypass graft.)

The search for an accurate, noninvasive technique to measure left ventricular (LV) volume is an important clinical pursuit. One such technique is two-dimensional echocardiography, which has been used to calculate LV volume and mass.1–4 To estimate cardiac volume, empiric formulas are used in the calculations, based on certain assumptions. For example, Simpson’s rule is based on dividing the object into slices of known thickness, assuming that the object is regular in shape;5 the volume of the object is then equal to the sum of the volumes of the slices. Because two-dimensional echocardiography can obtain multiple slices through the LV, echocardiographic data could be applied using Simpson’s rule to estimate ventricular volume. However, the measurement and calculation are tedious because multiple views must be obtained.

Because transesophageal echocardiography (TEE) is increasingly used intraoperatively as a monitor of ventricular function and volume, physicians have been interested in whether a single plane of the LV may provide a reasonable estimate of LV volume. One study conducted in patients undergoing vascular surgery reported a good correlation between the LV end-diastolic volume measured by first-pass radionuclide angiography and the LV end-diastolic area (EDA) (short-axis) measured by two-dimensional TEE.6 Because the end-diastolic distending force of the ventricle (preload) correlates better with LV end-diastolic volume or dimension than with end-diastolic pressure,7 monitoring the LV at the level of the short axis may be useful in estimating LV preload.

Use of TEE as a monitor of preload during the intraoperative period, however, requires accurate detection of real-time changes. Although online quantitative measurement of LV area has become available and may be beneficial,8–10 the technique can be too cumbersome.

* Assistant Professor of Anesthesiology.
† Medical student.

Received from the Department of Anesthesiology, University of California, San Francisco, Mount Zion Medical Center, San Francisco, California. Accepted for publication July 5, 1994. Supported in part by a grant from the Anesthesiology Young Investigator Award from the Foundation for Anesthesia Education and the Burroughs Wellcome Fund. Presented in part at the annual meeting of the American Society of Anesthesiologists, New Orleans, Louisiana, October 1992.

Address reprint requests to Dr. Leung: Department of Anesthesiology, University of California, San Francisco, Mount Zion Medical Center, 1600 Divisadero, San Francisco, California 94115.

Anesthesiology, Vol 81, No 5, Nov 1994
to apply quickly in real intraoperative time. In addition, in 20–28% of cases image quality is not adequate to allow real-time quantitation.\textsuperscript{9,11} In contrast, LV end-systolic cavity obliteration is easily visualized by TEE and frequently used as an indicator of intraoperative hypovolemia. However, no study has yet assessed whether intraoperative LV end-systolic cavity obliteration reflects a true decrease in EDA or other factors. Although one of the determinants of end-systolic volume is preload (end-diastolic volume), other factors that may influence end-systolic volume include LV contractility and afterload.\textsuperscript{12} Thus, a decrease in end-systolic volume as represented by end-systolic cavity obliteration may not necessarily reflect a decrease in preload.

Therefore, we continuously monitored the LV using TEE to determine the relation between LV end-systolic cavity obliteration and the corresponding LV EDA (an estimate of intraoperative LV preload) in patients undergoing elective coronary artery bypass graft surgery. We also related acute changes in LV area to hemodynamic changes.

Materials and Methods

After receiving institutional approval and written informed consent, we studied 139 men scheduled for elective coronary artery bypass graft surgery. Patients were excluded from the study if they had esophageal disease that precluded the insertion of the echocardiographic transducer. Anesthesia consisted of either high-dose sufentanil or fentanyl, or isoflurane supplemented with fentanyl. After tracheal intubation, we inserted a 9-mm transesophageal probe with a 3.5-MHz (Diasonic, Milpitas, CA) or 5-MHz (General Electric, Milwaukee, WI) transducer to obtain a short-axis view of the LV at the midpapillary muscle level. TEE images were continuously recorded onto 0.5-inch VHS videotapes. Routine clinical monitors included a seven-lead electrocardiograph and radial artery and pulmonary artery catheters. All patients received 100% inspired O\textsubscript{2}. Ventilation was controlled to maintain arterial O\textsubscript{2} tension > 70 mmHg and arterial CO\textsubscript{2} tension between 35–45 mmHg.

**Determination of Left Ventricular End-systolic Cavity Obliteration**

During off-line analysis, videotapes were scanned both in real-time and stop-frame for end-systolic cavity obliteration of the LV. The criterion for end-systolic cavity obliteration was defined as apposition of the papillary muscles during end-systole. When such an episode was identified, the videotape was backtracked in 5-min increments to identify the onset time of obliteration.

**Digitization and Measurement**

At the onset of LV end-systolic cavity obliteration, we digitized one cardiac cycle as 16 video frames (Data-Vue system, Microsonics, Indianapolis, IN). Only normal beats that followed normal beats were chosen. End-diastole was defined by the frame corresponding to the largest LV cavity and end-systole by the frame with the smallest cavity. We traced the endocardium at end-diastole and end-systole using a light-pen pointer. We included both the anterolateral and posteromedial papillary muscles in our area tracings as is routine.\textsuperscript{13} Areas of the tracing were calculated by the Data-Vue system. In patients with obliteration episodes, we also digitized one “control” cardiac cycle after anesthet induction and proper placement of the TEE probe. We similarly measured EDA and end-systolic area (ESA) for these control cycles. We calculated the percent EDA and ESA change from control to obliterated state for all patients.

To determine the intra- and interobserver variabilities, two investigators repeated each measurement of EDA and ESA (during control and obliterated states) in ten randomly chosen patients.

**Hemodynamic Calculations**

Systolic (SBP), diastolic (DBP), mean radial arterial and pulmonary artery diastolic (PAD) pressures, and heart rate (HR) were measured continuously on entry into the operating room and stored in a microcomputer (T1000, Toshiba Corporation, Tokyo, Japan) using a digital interface system. All hemodynamic data also were recorded continuously onto hard copy at 1 mm/s with a four-channel strip-chart recorder (4-Inch Direct Digital Writer, Marquette) linked to the hemodynamic monitor (frequency response 0.05–120 Hz; 7010, Marquette). Hemodynamic data were averaged every 60 s and entered into the microcomputer. To determine the relation between hemodynamics and preload changes as assessed by TEE, hemodynamic parameters recorded at the onset of LV end-systolic cavity obliteration and at 5 and 10 min after onset were compared with those recorded at 10 min before onset (baseline). A significant change in hemodynamics was defined as
correction was applied to categorical data. $P$ values $\leq 0.05$ were considered significant. Data were expressed as means $\pm$ SD.

**Results**

Thirty-nine (28%) of the 139 study patients had 62 episodes of LV end-systolic cavity obliteration. For patients with more than one obliterated episode, the first episode was chosen for analysis. Thus, a total of 39 episodes was selected for the following analysis. Shown in figure 1 are four frames from 1 patient demonstrating end-diastole and end-systole at control and obliterated states.

*End-systolic Area versus End-diastolic Area*

There was a significant decrease in mean LV ESA from control to obliterated state (from 7.29 $\pm$ 2.56 to 4.00 $\pm$ 1.46 cm$^2$, $P = 0.0001$). A similarly significant decrease in mean LV EDA was found from control to obliterated state (from 18.18 $\pm$ 4.36 to 12.92 $\pm$ 3.74 cm$^2$, $P = 0.0001$).

As ESA became obliterated (compared to control measurements), the EDA was reduced accordingly (fig. 2). Overall, mean EFA increased from 0.609 $\pm$ 0.095 to 0.692 $\pm$ 0.083 from control to the obliterated states, $P < 0.0001$. We also determined whether the obliterated states were primarily related to changes in LV

![Graph showing change in LV end-systolic area compared to end-diastolic area.](http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931291/)

**Statistical Analysis**

Paired Student's $t$ test (two-tailed) was used to determine significant differences between EDA and ESA in the control and obliterated states. The ejection fraction area (EFA) was computed as $\text{EDA} - \text{ESA}/\text{EDA} \times 100\%$ for each patient. Stroke area was calculated as $\text{EDA} - \text{ESA}$. We determined inter- and intraobserver variabilities by comparing the repeated measurements by using single-factor analysis of variance test. Chi-squared analysis or Fisher's exact test with continuity

---

Anesthesiology, V 81, No 5, Nov 1994
EFA, decreases in LV EDA, or a combination. The results are shown in table 1. Of these 39 episodes, 31 (80%) were associated with >10% reduction in EDA, 4 (10%) with increases in EFA only, and an additional 4 (10%) with no substantial change in either the EDA or EFA.

The reduction in stroke area from control to obliterated states correlated better with the reduction in EDA than with decreased ESA ($r = 0.89$, $P = 0.0001$ vs. $r = 0.35$; fig. 3).

**Hemodynamic Measurements versus End-diastolic Area**

There was poor correlation between any of the hemodynamic parameters and the LV EDA obtained immediately after induction (control) and at obliterated states (fig. 4). In addition, a substantial (>10%) decrease in EDA was not always associated with increases in HR, or decreases in SBP, DBP, PAD, or central venous pressure (CVP), the conventional parameters used to estimate preload. Of the 31 episodes with >10% reduction in EDA, 23 (74%) were associated with increased HR; 7 (23%) with decreased SBP; 7 (23%) with decreased DBP; 17 (55%) with reduced PAD; and, 19 with reduced CVP (61%). Of the 8 episodes with no substantial decrease in EDA, 5 (63%) actually were associated with increases in HR; 1 (13%) with reduced SBP; 1 (13%) with decreased DBP; 2 (25%) with decreased PAD; and 2 (25%) with reduced CVP. Similarly, hemodynamic parameters correlated poorly with LV ESA measured at control vs. obliterated states.

We also determined whether hemodynamics changed acutely at the time of cavity obliteration. No significant changes from baseline occurred in HR, SBP, DBP, PAD, or CVP at the onset of LV end-systolic cavity obliteration, or at 5 or 10 min after onset (table 2). At onset of LV cavity obliteration, only 20% of episodes were associated with a >10% increase in HR, 21% with decreased SBP, 25% with decreased DBP, 16% with decreased PAD, and 12% with decreased CVP (fig. 5). A similar pattern existed at 5 or 10 min after the onset of obliteration episodes (fig. 5). In addition, 35% of HR values actually decreased, whereas 44% of SBP, 46% of DBP, 16% of PAD, and 18% of CVP increased during obliterated states.

### Table 1. Combination of Changes in End-diastolic Area and Ejection Fraction Area

<table>
<thead>
<tr>
<th></th>
<th>EDA (decreases)</th>
<th>EDA (no change)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EFA (increases)</td>
<td>16</td>
<td>4</td>
</tr>
<tr>
<td>EFA (no change)</td>
<td>12</td>
<td>4</td>
</tr>
<tr>
<td>EFA (decreases)</td>
<td>3</td>
<td>0</td>
</tr>
</tbody>
</table>

EDA = end-diastolic area; EFA = ejection fraction area.
The number in each cell represents the number of episodes of end-systolic cavity obliteration. Decreases refer to >10% change from baseline. No change indicates <10% change from baseline measurement. For example, 18 episodes of end-systolic cavity obliteration were associated with increases (>10%) in EFA and >10% decreases in EDA.

Quantification of Observer Variability

The measurements of EDA and ESA from ten randomly chosen patients were repeated to determine inter- and

Anesthesiology, V 81, No 5, Nov 1994
intraobserver variabilities. The linear relations obtained between the two measurements performed by observer 1 showed a good correlation ($r = 0.99$). Similar correlation existed for observer 2 ($r = 0.93$) and between observers 1 and 2 ($r = 0.96$).

**Discussion**

Our study demonstrates that the appearance of end-systolic cavity obliteration is frequently associated with decreases in EDA, decreases in stroke area, and mild increases in EFA. The appearance of end-systolic LV cavity obliteration and associated decreases in EDA, however, is rarely preceded by any acute alteration in hemodynamic parameters.

**Comparison with Previous Studies**

Our finding that EDA correlates with ESA is consistent with the close relation that exists between systolic and diastolic events. Although the principal factor influencing LV filling is the atrioventricular pressure gradient, diastolic ventricular suction may be another physiologic mechanism to augment dia-

**Table 2. Hemodynamic Parameters Measured before, after, and at the Onset of End-systolic Cavity Obliteration**

<table>
<thead>
<tr>
<th></th>
<th>−10 min</th>
<th>−5 min</th>
<th>Onset</th>
<th>+5 min</th>
<th>+10 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats/min)</td>
<td>64 ± 22</td>
<td>66 ± 22</td>
<td>71 ± 14</td>
<td>70 ± 22</td>
<td>68 ± 21</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>112 ± 34</td>
<td>117 ± 37</td>
<td>115 ± 26</td>
<td>114 ± 36</td>
<td>112 ± 35</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>58 ± 19</td>
<td>59 ± 20</td>
<td>61 ± 16</td>
<td>59 ± 17</td>
<td>58 ± 18</td>
</tr>
<tr>
<td>PAD (mmHg)</td>
<td>11 ± 6</td>
<td>11 ± 5</td>
<td>11 ± 4</td>
<td>11 ± 5</td>
<td>11 ± 4</td>
</tr>
<tr>
<td>CVP (mmHg)</td>
<td>6 ± 7</td>
<td>6 ± 6</td>
<td>6 ± 5</td>
<td>6 ± 5</td>
<td>7 ± 7</td>
</tr>
</tbody>
</table>

Values are mean ± SD. The mean hemodynamic variables (HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure; PAD = pulmonary artery diastolic pressure; CVP = central venous pressure) at the onset, 5 and 10 min before (indicated by −5 min and −10 min), and 5 and 10 min after (indicated by +5 min and +10 min) the onset of end-systolic cavity obliteration are listed in each cell.

Anesthesiology, V 81, No 5, Nov 1994
The frequency with which hemodynamic variables change (>10%) at the onset and at 5 or 10 min after the onset of end-systolic cavity obliteration. The hemodynamic variables examined were heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), pulmonary artery diastolic pressure (PAD), and central venous pressure (CVP). For example, at the onset of end-systolic cavity obliteration, only 20% of the episodes were associated with increased (>10%) HR (solid bars), and within 10 min of the onset, only 18% of the episodes were associated with increases in HR (open bars).

The negative diastolic LV pressure may be generated by a decrease in end-systolic volume and an increase in contractility. Thus, when there is a decrease in LV end-diastolic volume, a compensatory mechanism exists to increase contractility by decreasing end-systolic volume to augment LV filling. As EDA decreased, stroke area decreased accordingly in our patients. This finding is consistent with reports that changes in preload directly influence stroke volume in the intact heart.

We found no correlation between decreases in LV EDA and hemodynamic parameters. Several possibilities may account for this. Traditionally, preload is defined as the load imposed on the muscle fiber before contraction. In the intact heart, preload usually is referred to as end-diastolic stress, pressure or volume. The assumption is that a good correlation exists between stress, pressure, volume or sarcomere length. However, this relation may not hold true in some clinical situations. For example, pulmonary capillary wedge pressure has been used to determine LV preload and volume status since 1970. When pulmonary vascular resistance is low, there is diastolic pressure equalization between the pulmonary capillaries, pulmonary artery, left atrium, and LV. However, various conditions may disrupt this DBP continuity. Several clinical investigations demonstrate a poor correlation between pulmonary capillary wedge pressure and LV sizes, as assessed by either two-dimensional echocardiography or gated blood pool scintigraphy during and after cardiac surgery. The poor correlation between pulmonary capillary wedge pressure and LV volume is thought to be due to alteration in the Frank-Starling pressure-volume relation. At normal levels of end-diastolic pressure, large changes in volume produce minimal changes in pressure. However, at higher levels of end-diastolic pressure, such as decreased ventricular compliance during myocardial ischemia, LV hypertrophy or congestive heart failure, small increases in volume will produce substantial pressure changes. Because it is difficult to assess the patient's ventricular compliance clinically, measurement of LV pressure or pulmonary capillary wedge pressure alone to assess LV volume can be inaccurate. Thus, our results agree with those of previous studies suggesting that monitoring LV dimension may provide more sensitive measurement of changes in preload than conventional hemodynamic monitoring such as pulmonary artery and central venous pressure catheterization.

Another explanation for the poor correlation between EDA and hemodynamic changes is that during surgery, alteration in preload, as assessed by LV end-diastolic volume, is primarily due to blood loss and or distribution of blood between the intra- and extra-thoracic compartments. The compensatory mechanisms mediated by the sympathetic nervous system in response to a decrease in circulatory intravascular volume include tachycardia, increased systemic vascular resistance and vasoconstriction. Greater volume deficits may lead to hypotension. In contrast to the awake state, reflexes typically occurring during the hypovolemic state, e.g., tachycardia, may be masked by anesthesia.

Clinical Implications

Two-dimensional echocardiography has been found to be a relatively accurate method of measuring LV volume and correlates well with other measurement techniques such as cineangiography and radionuclide scintigraphy. However, absolute volume calculations have been found to be less accurate. Intraoperatively, knowing the absolute volume may not be as useful as having the ability to detect changes in volume. The use of LV end-systolic cavity obliteration is a potentially useful "alarm" suggesting the development...
of hypovolemia. Caution, however, must be taken not to interpret all cavity obliteration as a decrease in preload because in many cases, cavity obliteration also reflects increased inotropic activity of the heart. In the present study, we found that 10% of episodes of LV end-systolic cavity obliteration is related to increase in EFA only, suggesting that using LV end-systolic cavity obliteration alone to predict hypovolemia is not entirely appropriate.

**Potential Limitations**

In this study, we used echocardiographic areas to estimate preload changes but did not use a third independent method to validate the findings. Thus, we have no information regarding the incidence of false-negative results. Our study, however, did not aim to determine how often "true hypovolemia" or "low-preload states" were detected by TEE monitoring. Rather, we sought to determine the relation between LV end-systolic cavity obliteration and EDA and hemodynamic measurements. In fact, the sensitivity of decreases in ESA for predicting similar decreases in EDA is high (100%), but the specificity of such changes is low (25%–38% when 10%–30% decreases in area measurement are used as a cutoff). The specificity is low because of a high incidence of false-positive results, suggesting that many instances of decreases in ESA are not necessarily associated with decreases in EDA.

None of the conventional "gold standards" is clinically feasible techniques to continuously measure preload. Conventional gold standards used in measuring LV end-diastolic volume include biplane angiography and radionuclide angiography. Biplane angiography assumes that the LV chamber is ellipsoid in shape, ignores the volume of the papillary muscle, and requires invasive cardiac catheterization. Radionuclide angiography has limited anatomic resolution, uses data averaged from multiple beats and therefore has reduced temporal resolution, and involves ionizing radiation. Moreover, none of these techniques allow serial or continuous measurement. In contrast, two-dimensional echocardiography is amenable to repetitive measurement.

The value of TEE in determining LV volumes and ejection fraction has been compared with cineventriculography. The results indicate that standard two-dimensional echocardiographic models could be applied to transesophageal views to predict the angiographic volumes. However, transesophageal views underestimate angiographic end-diastolic and end-systolic LV volumes because of foreshortening of the apex in the four-chamber esophageal planes. During surgery and anesthesia, the measurement of a change in LV preload is more important than the measurement of absolute LV volume because intraoperative clinical intervention is more likely based on a change in the hemodynamic status rather than on an absolute number. Thus, although echocardiographic measurements underestimate absolute LV volumes, the qualitative changes in echocardiographic areas nevertheless remain useful in identifying changes in LV preload.

In summary, our study demonstrates that although end-systolic cavity obliteration detected by intraoperative TEE is frequently associated with decreases in EDA, not all end-systolic cavity obliteration is indicative of decreases in LV filling. Thus, although this simple estimate of LV filling may be attractive as an initial rapid evaluation of cardiac performance in critical situations, additional evaluation of EDA should be performed. Our study also demonstrates that reliance on routine hemodynamic monitoring, such as HR or blood pressure measurement alone, in assessing preload may be misleading because a substantial number of episodes involving a reduction in preload (manifested by decreases in EDA) produce hemodynamic changes opposite of those expected.

The authors thank Winifred Von Ehrenburg for editorial assistance.

**References**


INTRAOPERATIVE PRELOAD ESTIMATION

elakis N, Geiger S. Bethesda, American Physiological Society, 1979, pp 553–580


Anesthesiology, V 81, No 5, Nov 1994