Echocardiographic Assessment of Ventricular Performance Following Induction with Two Anesthetics


Echocardiographic studies were made of 20 healthy patients scheduled for minor surgical procedures to determine whether this technique could be used routinely in the operating room and to evaluate the effects of halothane and enflurane on left ventricular performance. Thirteen minutes following induction of anesthesia with halothane in ten patients (mean end-tidal halothane concentration 0.93 per cent), mean arterial blood pressure, left ventricular (LV) diastolic dimension, LV fractional shortening, mean velocity of circumferential fiber shortening and systolic thickening of the posterior LV wall were significantly decreased. LV systolic dimension was increased significantly. These data indicate that halothane caused decreased contractility in the presence of a decreased afterload. Twelve minutes following induction of anesthesia with enflurane in ten patients (mean delivered enflurane concentration 2.4 per cent), mean arterial blood pressure, LV systolic and diastolic dimensions were decreased, while heart rate was increased significantly, indicating that enflurane caused vasodilatation and may have had some depressant effect on contractility. Echocardiography is a non-invasive, safe and relatively rapid method that can be used in the perioperative period to assess cardiac function and to evaluate the effects of pharmacologic agents on the heart. (Key words: Anesthetics; volatile; halothane; enflurane; Heart: contractility; echocardiography; myocardial function; vascular pressures.)

Echocardiography, a promising non-invasive technique for assessing cardiac performance, employs high-frequency sound waves that are transmitted and received by means of a piezoelectric crystal in the frequency range of 1 to 10 MHz. The crystal and lens system are the key components of the manually held transducer which, when placed in the appropriate intercostal space, opens an "echocardiographic window" to the heart. From reflected sound waves an electrical signal that can be displayed and recorded is generated. Upon sweeping the transducer in a cephalo-caudal direction across the heart with continuous recording (M or motion mode), valvular and chamber landmarks can be identified and recorded for later analysis (fig. 1). Such a display allows for the semiquantitative derivation of many indices of left ventricular performance.

In an attempt to provide information about the usefulness of echocardiography as a monitor in the operating room, we evaluated the cardiac effects of two commonly used inhalational anesthetics by this method. The anesthetic drugs used in this study were halothane, a known depressant of myocardial contractility, and enflurane, about which there is some dispute as to its effects on the heart.

Methods

Twenty essentially healthy patients (ASA physical status I or II) scheduled for minor elective extrathoracic operations were studied. The investigation was reviewed and approved by the hospital's human experimentation committee. During the preoperative visit an electrocardiogram was taken and a routine cardiac examination was performed. Obese patients, those showing chest-wall deformities, and patients manifesting clinical evidence of cardiovascular disease were omitted from the study. All patients had fasted for a minimum of seven hours. They were properly

**Abbreviations**

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
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<tbody>
<tr>
<td>D1</td>
<td>left ventricular diastolic dimension</td>
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<tr>
<td>D2</td>
<td>left ventricular systolic dimension</td>
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<td>FS%</td>
<td>fractional shortening of the left ventricle</td>
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<td>ST%</td>
<td>percentage systolic thickening of the left ventricular posterior wall</td>
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<tr>
<td>MVS</td>
<td>early diastolic slope of the anterior mitral valve leaflet</td>
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<td>Vt</td>
<td>mean velocity of circumferential fiber shortening</td>
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<td>LVEF</td>
<td>left ventricular ejection fraction</td>
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<td>LAD</td>
<td>left atrial dimension</td>
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<td>MAP</td>
<td>mean systemic arterial blood pressure</td>
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<tr>
<td>HR</td>
<td>heart rate</td>
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<tr>
<td>CVP</td>
<td>central venous pressure</td>
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<tr>
<td>LV</td>
<td>left ventricular</td>
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informed of their forthcoming anesthetic and surgical experience, and premedication was omitted. Following arrival in the operating room, an intravenous catheter was placed and an electrocardiographic recording was started. An arterial catheter was placed percutaneously in a radial artery for pressure monitoring and blood-gas sampling. A central venous catheter was inserted via an antecubital vein. Arterial and central venous blood samples were drawn for analysis just before beginning each scan. The preinduction echocardiographic scan was then performed and the position on the chest from which it was taken was marked.

Anesthesia was induced and maintained with halothane in oxygen in ten patients (six male, four female), whose mean age was 26 years (range: 17 to 35 years). In another group of ten patients (five male, five female), whose mean age was 34 years (range: 23 to 53 years), anesthesia was induced and maintained with enflurane in oxygen. An oral airway was inserted after induction, and respiration was assisted to maintain arterial blood-gas and pH values within normal limits for all conditions (Pao2, 279 ± 25 torr; Paco2, 42 ± 1 torr; pH 7.39 ± 0.01). End-tidal halothane levels were measured by means of a mass spectrometer (Perkin-Elmer MGA100A) calibrated for a linear response for halothane concentrations from 0 to 4 per cent. The end-tidal halothane concentration obtained at the time of the postinduction echogram (18 ± 2 min following induction) was 0.93 ± 0.07 per cent. Since our mass spectrometer did not allow end-tidal enflurane measurements, a calibrated vaporizer (Drager S110) setting was used as an indicator of the enflurane concentration. Induction was accomplished with enflurane, 4 per cent. The delivered enflurane concentration at the postinduction echogram (12 ± 1 min after induction) had a mean value of 2.4 ± 0.2 per cent. Surgical stimulation was avoided during the entire period of observation.

The pre- and postinduction echograms were performed in a manner described previously.8 Both scans were taken from the same marked area. This procedure allows accurate comparisons of values obtained at different times from individual patients.8 Quality recordings were obtained for all patients both prior to induction and after the period of stabilization with both anesthetic agents. The echograms were taken using a Smith Kline Ultrasonoscope with either a 2.25-MHz or a 1.9-MHz transducer. Recording was accomplished on a Honeywell 1856A Visicorder.

At the level of the chordae tendineae (fig. 2/1), the left ventricular end-diastolic dimension was measured at the R wave of the electrocardiogram. The end-systolic dimension was measured at the peak of the anterior movement of the left ventricular endocardium. Fractional shortening, or the percentage change in dimension during systole, was calculated by the formula:

\[ FS\% = \frac{D_d - D_s}{D_d} \times 100 \]

The mean velocity of circumferential fiber shorte-
ing was determined by the modified method of Cooper et al.1,8 according to the formula:

\[ V_e = \frac{(D_d - D_a)}{(D_a \times LVET)} \]

LVET was determined from the aortic valve echoogram.6 The percentage systolic thickening of the left ventricular posterior wall (fig. 2A) was taken as the difference of the left ventricular thicknesses at end-systole and end-diastole divided by the end-diastolic thickness \( \times 100 \).

Two indications of preload, the early diastolic slope of the anterior mitral valve leaflet16 and the left atrial dimension (fig. 2B) were measured by standard techniques.1,7,8

All echocardiographic dimensions were measured in at least three sequential beats and averaged for further data analysis. The echocardiographic measurements were made independently by two individuals blinded to the procedure. Statistical evaluation of the data was accomplished through the use of the t test for paired data (awake versus anesthetized for each patient). A P value of 5 per cent of less was considered significant. All values were presented as means ± standard errors of the mean.

Results

Halothane induction (table 1) resulted in a decrease in mean arterial blood pressure from 96.1 ± 5.9 torr to 75.3 ± 4.9 torr \( (P < 0.05) \). Heart rate, central venous pressure, MVS and LAD did not change in this group of patients. The left ventricular \( D_a \) decreased from 4.9 ± 0.11 cm to 4.49 ± 0.21 cm \( (P < 0.01) \). Concurrently, \( D_s \) increased from 3.34 ± 0.15 cm before induction to 3.47 ± 0.15 cm \( (P < 0.01) \) following halothane induction. The FS% computation derived from these dimensional changes therefore showed a highly significant decrease from 38.1 ± 2.2 per cent to 22.2 ± 2.5 per cent \( (P < 0.005) \) following halothane. Mean \( V_{et} \) was also decreased, from 1.29 ± 0.09 circumferences/second to 0.85 ± 0.09 circumferences/second \( (P < 0.05) \) and \( ST_p \) % was halved \( (P < 0.01) \).

Enflurane induction (table 2) resulted in a mean decrease in MAP of 19 torr \( (P < 0.05) \), coupled with a 10 beat/min increase in HR \( (P < 0.05) \). CVP, MVS and LAD were essentially unchanged. There was a significant decrease in \( D_a \) from 4.91 ± 0.17 cm before induction to 4.64 ± 0.19 cm after induction \( (P < 0.05) \). A similar decrease in \( D_s \) from 3.50 ± 0.16 cm to 3.22 ± 0.15 cm \( (P < 0.05) \) occurred. Consequently, there was no change in FS%. Neither \( V_{et} \) nor \( ST_p \) % showed any decrease following enflurane induction.

Discussion

Several considerations must be taken into account when analyzing cardiac performance through the use of single-plane echocardiography, as was done in this study. For example, the left ventricular chamber is considered to be a prolate ellipse, the minor axis of which is measured and taken to be the diameter of the chamber.6 Volume of the ventricle can then be cal-
culated during both diastole and systole by appropriate geometric formulas. Volume determinations arrived at in this manner are subject to a variety of criticisms, one of which is that errors in minor-axis dimensional measurement are amplified for volume calculations. Additionally, the assumption that the left ventricular chamber is a prolate ellipse fails in large or dilated hearts. Thus, conditions that change ventricular geometry require that caution be used in interpreting the echograms, particularly when volume calculations have been made. For such reasons, we have chosen to employ dimensional measurements and fractional shortening determinations derived directly from the dimensions. This provides a more conservative, but more accurate, representation of myocardial changes. It must be stressed that the entire ventricular motion is being assessed from only one or two points of measurement, and that the ventricle is assumed to be contracting without abnormal segmental wall motion.

The data derived from this study confirm previous reports that halothane causes a significant decrease in myocardial contractility in the initial stages of anesthesia. All of the indices of left ventricular ejection were significantly decreased despite a decrease in MAP. The decrease in $D_a$ could reflect a decrease in afterload in conjunction with the decreased MAP, or it may signal a decrease in LV compliance. The first of these two explanations seems more likely, since MVS was unchanged in this group of patients. Ventricular compliance is known to affect MVS. The left ventricular filling rate could be decreased by decreased ventricular compliance. This decreased filling rate could then be reflected by decreased MVS.

The increase in $D_s$ is indicative of diminished systolic emptying of the LV chamber. Combining the decreased $D_a$ with the increased $D_s$, a highly significant decrease in FS% occurred. It may be relevant to point out the similarity between FS% and ejection fraction (as determined angiographically). The major difference is that calculations of FS% involve circumferential measurements taken from only one angle. This decrease in FS%, combined with the lowered MAP, can be interpreted as a diminished overall muscle shortening.

This decreased shortening was directly reflected in the ST₆% measurement, which showed that the systolic thickening of the LV posterior wall was greatly diminished. Although it would be difficult to gauge overall ventricular performance by looking at this isolated area of the ventricle, several reports have suggested that pharmacologic interventions can be adequately assessed by the technique of wall-thickness measurements. Therefore, the 45 per cent decrease in ST₆% observed following halothane induction, in the presence of a lesser afterload, would be strong evidence that the strength of ventricular contraction had decreased. The overall rate of muscle shortening is also decreased following halothane induction, a point that can be inferred from the $V_e$ data.

Echocardiographic (MVS and LAD) and hemodynamic (CVP) variables related to preload did not show any change following halothane induction. The early diastolic filling slope of the mitral valve was constant (at a steady heart rate). That MVS is a measure of left ventricular filling, or preload, can be assessed from the work of DeMaria et al. They showed a decrease in blood flow across the mitral valve when the diastolic compliance of the ventricle was decreased. This decreased flow correlated well with a decreased MVS during the first third of ventricular diastole. Similarly, the size of the left atrium can be used as a measure of preload. Subject to the integrity of the mitral valve and to the relative compliances of both left atrium and ventricle, any increase in LAD would indicate a greater filling pressure.

The effects of enflurane induction on cardiac performance remain controversial. Some reports suggested decreased cardiac contractility in animals, while others indicated no change in cardiac performance. Similarly, interpretation of data obtained in man following enflurane remains uncertain. In this study, there was no decrease in the ejection-phase indices in the enflurane group. All of the determined measures of contractility, in both pump performance
and muscle function, were maintained. Even though $D_p$ decreased significantly, there was an equivalent decrease in $D_s$. As a result, there was no change in FS%. The rate of fiber shortening and total systolic wall thickening both remained at control values during enfurane anesthesia. There was no discernible change in preload-related variables.

These findings indicate that left ventricular function (systolic emptying) is either improved or, at least, not diminished with enfurane induction. Three general mechanisms could account for this result. Since preload was constant, contractility was increased, afterload was decreased, or some contractility alteration was present in combination with the decreased systemic pressure. Increased contractility can be dismissed from consideration, since none of the variables relating to muscle function was increased. This unchanged status is of particular significance since an increased heart rate and a decreased afterload could well be expected to result in increased values for indices of contractility.\(^{25,26}\) It was apparent that the afterload was decreased, a finding consistent with the hypotensive effects of enfurane.\(^{27}\) Therefore, we cannot exclude the possibility that some left ventricular hypokinesis was masked by the decreased MAP.

From this investigation, it became apparent that echocardiography can be utilized with ease in the operating theater. The technique is noninvasive, safe, and can be performed relatively rapidly. Although proper instrumentation and skilled personnel are essential for acquisition and analysis of data, most hospital centers already have ongoing ultrasound programs. Additionally, rapid data retrieval through the use of computerization techniques\(^{27}\) may well extend the research and diagnostic capabilities of echocardiography into the area of monitoring the surgical patient.

References