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# *Poor Correlation Between Pulmonary Arterial Wedge Pressure and Left Ventricular End-diastolic Volume after Coronary Artery Bypass Graft Surgery*

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**The authors studied 12 surgical patients in the intensive care unit post coronary artery bypass graft surgery and ten nonsurgical patients in the coronary care unit with chronic heart failure to determine the usefulness of the pulmonary arterial wedge pressure as an indicator of left ventricular preload. Left ventricular end diastolic volume was derived from concomitant determination of ejection fraction (gated blood pool scintigraphy) and stroke volume (determined from thermodilution cardiac output). In the nonsurgical patients, there was a significant correlation between changes in pulmonary arterial wedge pressure and left ventricular end-diastolic volume ($P < 0.05$, $r = 0.57$). In the 12 patients studied during the first few hours after surgery, there was a poor correlation between changes in pulmonary wedge pressure (range = 4–32 mmHg) and left ventricular end-diastolic volume (range = 25–119 ml/m$^2$), and a poor correlation between pulmonary arterial wedge pressures and stroke work index. In contrast, there was a good correlation between left ventricular end-diastolic volume and stroke work index. The poor correlation between the pulmonary arterial wedge pressure and left ventricular end-diastolic volume was not explained by changes in systemic or pulmonary vascular resistance. The altered ventricular pressure–volume relationship may reflect acute changes in ventricular compliance in the first few hours following coronary artery bypass graft surgery. While measurement of pulmonary arterial wedge pressure remains valuable in clinical management to avoid pulmonary edema, it cannot reliably be used as an index of left ventricular preload while attempting to optimize stroke volume in patients immediately following coronary artery bypass graft surgery. (Key words: Blood pressure: pulmonary arterial wedge pressure; pulmonary artery. Heart: compliance; end-diastolic volume; preload. Surgery: cardiovascular.)**

THE MAINTENANCE OF a normal cardiac output is dependent on adequate ventricular preload (end-diastolic volume). Because ventricular volumes are not measured easily in the clinical setting, the management of critically ill patients is frequently guided by the use of the pulmonary arterial wedge pressure (PAWP) as an approximation of left ventricular end-diastolic pressure. However, in some clinical circumstances left ventricular end-diastolic pressure may not be an accurate reflection of left ventricular end-diastolic volume.[1] Acute changes in ventricular pressure–volume relationships may allow a high end-diastolic pressure to occur in the presence of a normal or even a low end-diastolic volume. Two recent studies from our institution have suggested that acute changes in ventricular distensibility occur immediately following cardiopulmonary bypass while the patient is still in the operating room.[2,3] Therefore, we designed a prospective study to determine the reliability of PAWP measurement as an indicator of left ventricular preload in patients treated in the intensive care unit who had undergone coronary artery bypass graft surgery. For comparison, we also studied a group of patients in the coronary care unit with chronic heart failure.

## Materials and Methods

### PATIENT SELECTION

From December 1983 through April 1984 patients in normal sinus rhythm scheduled for elective coronary artery bypass graft surgery were asked, 1 day prior to surgery, to participate in this study. All patients who provided informed consent and who had radial and pulmonary arterial lines inserted in the operating room were included in the study when they arrived in the intensive care unit. The placement of pulmonary arterial lines was a clinical management decision made jointly by the surgical and anesthesia teams without regard to this study. Among the 15 patients originally entered into the study, two were excluded because of technical difficulties in determining

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TABLE 1. Hemodynamics and Drug Therapy in the 12 Patients Studied After Coronary Bypass Graft Surgery in the Intensive Care Unit

| Patient No. | No. Coronary Arteries Bypassed | Nipride* | Dopamine Dobutamine | Mean/Range PAWP (mmHg) | Mean/Range LVEDVI (ml/m²) | Mean/Range Cardiac Index (l · min⁻¹ · m⁻²) | PEEP ($cmH_2O$) | Range of LV Ejection Fraction |
|---|---|---|---|---|---|---|---|---|
| 1 | 3 | – | + | 19.6/12–23 | 60/51–71 | 2.0/1.2–2.4 | 5 | 0.26–0.47 |
| 2 | 3 | + | – | 10.2/8–13 | 85/72–97 | 2.8/2.5–3.2 | 0–5 | 0.34–0.40 |
| 3 | 3 | – | – | 11.2/10–15 | 49/42–59 | 1.9/1.7–2.3 | 5 | 0.43–0.48 |
| 4 | 4 | – | – | 16.2/13–20 | 52/46–59 | 2.5/2.3–2.8 | 5 | 0.42–0.52 |
| 5 | 3 | – | + | 12.0/4–16 | 32/25–34 | 1.9/1.7–2.2 | 0 | 0.55–0.57 |
| 6 | 4 | + | – | 6.6/5–7 | 43/32–54 | 2.2/1.6–2.6 | 2 | 0.62–0.76 |
| 7 | 3 | + | – | 8.0/5–12 | 49/41–58 | 3.0/2.3–3.8 | 0 | 0.41–0.62 |
| 8 | 3 | – | + | 19.6/15–32 | 111/109–119 | 2.1/1.5–2.5 | 5 | 0.16–0.24 |
| 9 | 3 | – | – | 13.6/10–16 | 66/57–77 | 2.4/2.2–2.8 | 5 | 0.39–0.47 |
| 10 | 2 | – | – | 8.6/8–10 | 59/45–70 | 2.3/1.5–2.5 | 5 | 0.49–0.57 |
| 11 | 4 | – | + | 22.5/21–23 | 56/53–69 | 2.0/1.7–2.3 | 5 | 0.32–0.39 |
| 12 | 2 | + | – | 18.2/12–23 | 35/28–41 | 2.4/2.3–2.8 | 0 | 0.64–0.68 |
| Mean | 3 | NA | NA | 13.8 | 58 | 2.3 | NA | NA |

NA = not applicable. See text for additional abbreviations.

left ventricular volume by radioisotope angiography, and one was excluded because of an early return to the operating room before the study was complete.

The final patient group included 12 patients (11 men and one woman) with an age range of 45 to 71 yr. The patients were transferred directly to the intensive care unit after completion of the surgery. All patients were mechanically ventilated with an intermittent mandatory ventilation mode with a tidal volume of 12–15 ml/kg. Respiratory rate was adjusted to maintain a *p*H of 7.40 to 7.45; the fraction of inspired oxygen was adjusted to maintain a $Pa_{O_2}$ greater than or equal to 70 mmHg. A constant low level ($\leq 5$ $cmH_2O$) of positive end-expiratory pressure was maintained in all patients throughout the study, with the exception of one patient (patient no. 2 had a change from 0 to 5 $cmH_2O$ after the first h; see table 1). Sedation was maintained with intravenous morphine and valium.

Ten additional nonsurgical patients with chronic heart failure (left ventricular ejection fraction range from .13 to .35) from ischemic heart disease (n = 4) and cardiomyopathy (n = 6) were studied in order to provide a comparison group with the 12 patients studied following cardiac surgery. These patients had a mean age of 50 ± 20 yr. They were admitted to the coronary care unit to have their pulmonary and systemic hemodynamics assessed and to undergo a trial of intravenous vasodilator therapy (nitroglycerin or nitroprusside). If the PAWP was less than 18 mmHg, then the patients were given saline infusions to increase the wedge pressure by 3 mmHg (two of ten patients). All these patients were ventilating spontaneously.

## Gated Blood Pool Study Measurements

Radionuclide angiography was performed using established scintigraphic techniques.[4–7] Red blood cells were labeled by injecting 5 mg stannous pyrophosphate 30 min before injection of 15–20 μCi of $^{99m}Tc$ pertectinetate. A mobile gamma camera with a low-energy, all-purpose collimator was positioned in the left anterior oblique (LAO) position and adjusted so that the interventricular septum was clearly visible. ECG-gated equilibrium scintigraphy was performed each h with the patient supine and the camera angle constant. Ejection fraction was calculated in each image from the left ventricular time *versus* activity curve (background subtracted) according to standard techniques.[8,9] Data were stored on a disc system.

Radionuclide scintigraphic calculation of left ventricular ejection fraction correlates closely with those of contrast angiography. Prior studies have shown that ejection fraction by gated blood pool studies correlates well with data from first-pass radionuclide angiocardiography (r = 0.94) and from contrast angiography (r = 0.84).[9] In this institution the 95% confidence interval of left ventricular ejection fraction is 9% and the normal left ventricular ejection fraction is 0.67 ± 0.12, with an intraobserver variability of ±.04.

Simultaneous measurement of cardiac output by thermodilution technique was used to derive left ventricular stroke volume index (SVI) by dividing cardiac output by heart rate and by body surface area ($m^2$). Left ventricular end diastolic volume index (LVEDVI) was then calculated from the relationship between the SVI and left ventricular ejection fraction.

$$\text{LVEDVI (ml/m}^2) = \frac{\text{Stroke Volume Index}}{\text{Left Ventricular Ejection Fraction}}$$

Normal LVEDVI using this technique is 68 ml/$m^2$ (range 54–82 ml/$m^2$).

Cardiac output by thermodilution technique has been found to correlate well with *in vitro* flow data (r = 0.99)[10] and dye dilution,[11] yielding a 95% confidence interval of

TABLE 2. Univariate Regression Analysis of Pulmonary Artery Wedge Pressure (PAWP) *versus* Left Ventricular End Diastolic Volume Index (LVEDVI), and Stroke Work Index (SWI) *versus* either PAWP or LVEDVI in the 12 Patients Studied Following Coronary Artery Bypass Graft Surgery

| | Patient No. | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 |
| PAWP *vs.* LVEDVI | | | | | | | | | | | | |
| r | .67 | .28 | .84 | .76 | .05 | .44 | .44 | .27 | .24 | .80 | .22 | .02 |
| slope | + | + | + | − | − | − | + | + | − | − | + | − |
| SWI *vs.* PAWP | | | | | | | | | | | | |
| r | .40 | .16 | .48 | .90 | .56 | .91 | .51 | .71 | .59 | .67 | .19 | .43 |
| slope | + | + | + | − | − | − | − | + | − | − | − | + |
| SWI *vs.* LVEDVI | | | | | | | | | | | | |
| r | .74 | .78 | .80 | .67 | .84 | .31 | .48 | .52 | .49 | .78 | .87 | .86 |
| slope | + | + | + | + | + | + | + | + | + | + | + | + |
| dF | 3 | 3 | 2 | 3 | 3 | 3 | 3 | 3 | 3 | 3 | 2 | 3 |

Data for each of the 12 patients are displayed with an r value (correlation coefficient), slope of linear regression by least squares designated as positive (+) or negative (−), and degrees of freedom (dF) for each patient.

9%. The validity and reproducibility for determining LVEDVI by this technique has been reported previously.[8,9] The calculated LVEDV has a 95% confidence level of 12.7%. Stroke work index (SWI) ($g - m \cdot m^{-2}$) was calculated as follows:

$$SWI = \frac{0.0136 \times \text{stroke volume} \times (MAP - PAWP)}{m^2 \text{ (body surface area)}}$$

where MAP = mean systemic arterial pressure and PAWP = mean pulmonary arterial wedge pressure.

## Hemodynamic Measurements and Study Protocol

After red blood cell labeling was accomplished, systemic, arterial, right atrial, pulmonary arterial, and pulmonary arterial wedge pressures and cardiac output were determined simultaneously with gated equilibrium radionuclide angiography. Baseline and hourly measurements were made for 4 h, beginning within 90–180 min of arrival in the intensive care unit. The pulmonary arterial, right atrial, and systemic pressures were transduced and recorded on a strip chart recorder. All pressure measurements were then read directly from the strip chart recording at end-expiration. Thus, no meaning device was used. All equipment was recalibrated before each set of measurements. Cardiac outputs in triplicate were determined by thermodilution. Ten milliliters of saline at room temperature was injected, and repeat boluses were injected, if necessary, until three consecutive cardiac-output measurements were within 10% of the mean of three measurements. This approach has been validated for its accuracy in both clinical and experimental studies.[12,13] The study protocol did not influence the use of sedation (morphine, valium), fluid, blood replacement, or the use of vasodilators or vasopressors in the postoperative setting in the intensive care unit.

For the ten nonsurgical patients, hemodynamics and ejection fraction with gated blood pool were determined at baseline and after vasodilator or volume infusion.

## Statistics

For the 12 patients following coronary artery bypass graft surgery, serial changes in PAWP and LVEDVI were compared using chi-square analysis and Spearman rank order test to determine whether the relationship between increases and decreases in these variables was random.[14] For the ten nonsurgical patients, a Spearman rank order test was used to assess for a significant relationship between increases and decreases in PAWP and LVEDVI. A rank test was used rather than chi-square because of fewer data points in the ten nonsurgical patients.[14]

For the 12 surgical patients, univariate least squares regression analysis was applied to assess the correlation between PAWP and LVEDVI, PAWP and SWI, and LVEDVI and SWI. Regression was performed on data for each patient and the correlation coefficient was compared with the critical value of r for the appropriate degrees of freedom (table 2). Similarly, univariate least squares regression analysis was used to correlate log PAWP and LVEDVI, SWI and log PAWP, and SWI and log LVEDVI.

To analyze this further, a change in left ventricular compliance (or distensibility) was strictly defined as: 1) change in PAWP $\geq$ 4 mmHg with an opposite or no change in LVEDVI; or 2) change in LVEDVI $\geq$ 10 ml/$m^2$ with an opposite or no change in PAWP and PAWP $\geq$ 18 mmHg. This definition avoids the problem of misinterpreting data that may lie on the flat portion of the

pressure–volume curve. Increased compliance here was defined as a larger LVEDVI for the same PAWP or a lower PAWP for the same LVEDVI, and decreased compliance was defined as a lower LVEDVI for the same PAWP or a higher PAWP for the same LVEDVI.

Using chi-square analysis, changes in left ventricular compliance were compared with changes in systemic vascular resistance and pulmonary vascular resistance to determine whether the relationships between these changes were random (table 3). Using pooled data with each patient as his or her own control, the paired Student's *t* test was applied to compare "early" (beginning and end of first h) and "late" (beginning and end of last h) PAWP and LVEDVI in the 12 surgical patients.[14]

## Results

For the 12 patients who underwent coronary artery bypass graft surgery, most of the group had a history of either systemic hypertension (3 of 12), prior myocardial infarction (4 of 12), or both (5 of 12). Preoperative ejection fraction ranged from 0.36 to 0.75 and most patients had a normal PAWP, although two patients had values greater than 20 mmHg. Preoperative cardiac index was normal except in four patients who had values between 1.3 and 2.2 $l \cdot min^{-1} \cdot m^{-2}$. Table 1 provides hemodynamic and drug therapy data for the 12 patients studied in the intensive care unit after surgery. Four patients received vasodilators, while four other patients received vasopressors during the course of the study. PAWP ranged from 4 to 32 mmHg while LVEDVI ranged from 25 to 119 $ml/m^2$.

The relationship between changes in PAWP and changes in LVEDVI was random in patients following coronary artery bypass graft surgery (fig. 1). Both concordant and discordant relations were observed. Univariate regression of PAWP and LVEDVI in individual cases showed a poor correlation for most patients (table 2). Only six of 12 patients had the predicted physiologic relationship and a regression line with a positive slope demonstrating concordant changes in PAWP and LVEDVI. Of these six patients, only one had a correlation coefficient greater than the critical value for statistical significance. Regression of SWI and PAWP revealed similar results (table 2).

However, as illustrated in table 2, regression of SWI and LVEDVI showed a much better correlation. All 12 patients had the expected physiologic relationships with a positive slope for the regression line indicating concordant changes. In addition, six of 12 patients had a correlation coefficient greater than or equal to the critical value for statistical significance. Almost identical results occurred using log transformation of these data to account for potential exponential physiologic relationships.

TABLE 3. Relationship between a Change in Ventricular Compliance and an Alteration in Systemic or Pulmonary Vascular Resistance in the 12 Patients Studied Following Coronary Artery Bypass Graft Surgery*

| | Increased Compliance | Decreased Compliance |
|---|---|---|
| Increased systemic vascular resistance | 0 | 3 |
| Decreased systemic vascular resistance | 1 | 4 |
| No significant change | 1 | 2 |
| Total | 2 | 9 |
| Increased pulmonary vascular resistance | 0 | 4 |
| Decreased pulmonary vascular resistance | 1 | 3 |
| No significant change | 1 | 2 |
| Total | 2 | 9 |

* There was a random relationship between changes in ventricular compliance and systemic vascular resistance (chi-square = 1.14; df = 2; $0.75 < P < 0.90$). Also, there was a random relationship between changes in ventricular compliance and pulmonary vascular resistance (chi-square = 1.41; df = 2; $0.50 < P < 0.75$).

Ventricular function curves relating SWI and PAWP in one patient is illustrated in figure 2*A*. Reviewing the curves of all 12 patients demonstrated that upward and downward shifts in the ventricular curves occurred in seven of the 12 patients. However, constructing ventricular function curves by relating LVEDVI to SWI yielded no shifts in any of the 12 patients (see fig. 2*B*).

Using our strict definition for a change in compliance (see "Methods"), we found 11 instances of altered com-

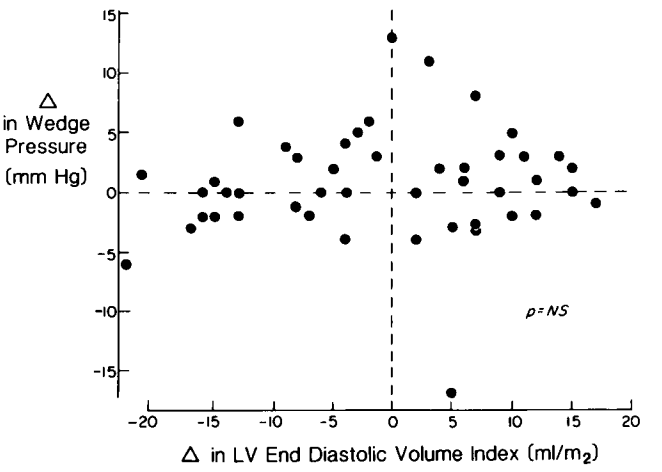


FIG. 1. Each dot (total = 46) represents the hourly change in pulmonary artery wedge pressure (PAWP) and left ventricular end diastolic volume index (LVEDVI) for all 12 patients following coronary artery bypass graft surgery. The relationship is random. Note that two data points are missing as a result of transient malfunction of the nuclear-imaging data-acquisition software system. There was no statistically significant relationship by either chi-square or Spearman rank order test.

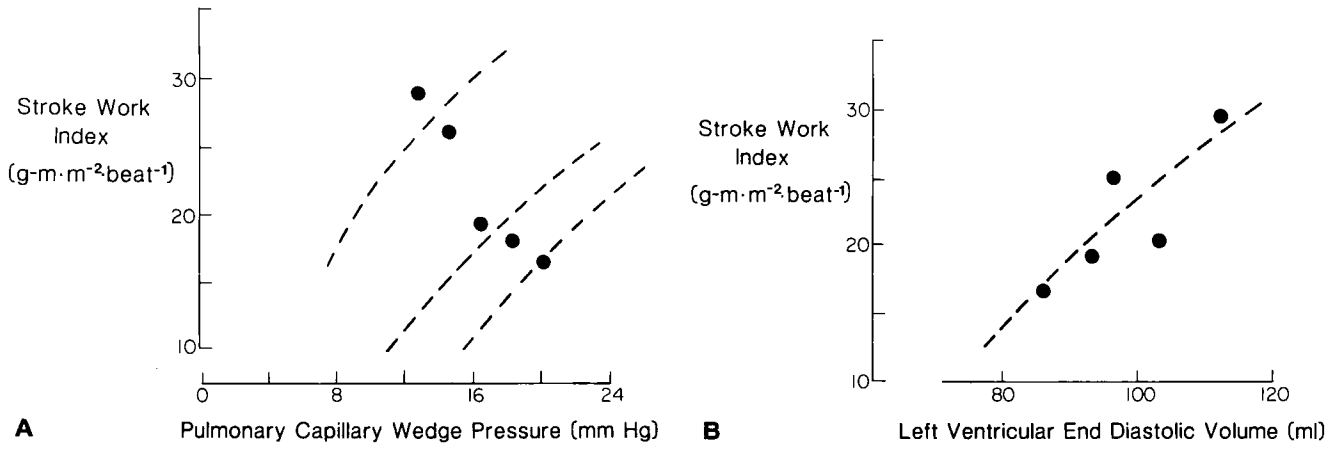


FIG. 2. *A (left).* Relation between stroke work index (SWI) and pulmonary arterial wedge pressure (PAWP) for patient no. 4. Note that least squares regression yields a nonphysiologic, negative slope (table 2). *B (right).* Relation between SWI and left ventricular end diastolic volume (LVEDV) for patient no. 4. In contrast to *A*, *B* shows one ventricular function curve with a physiologic, positive slope (table 2), demonstrating that the apparent shifts in ventricular function in *A* represent shifts in ventricular compliance.

pliance in seven patients. There was a random relationship between these alterations in compliance and changes in systemic or pulmonary vascular resistance (table 3).

In the ten nonsurgical patients, the changes in PAWP and LVEDVI correlated significantly ($P < 0.05$; r = 0.57). Figure 3 shows 14 measurements in the ten patients with only three points indicating a nonphysiologic response.

## Discussion

The results of this study demonstrated that measurements of PAWP did not reliably reflect left ventricular end diastolic volume in the first few h following coronary artery bypass graft surgery. The data for all surgical patients is shown in figure 1, which illustrates the poor correlation between PAWP and left ventricular preload. In addition, analysis of individual patient data points demonstrated that one-half of the patients did not have a positive, physiologic slope when changes in PAWP were plotted against SWI. However, the relationship of SWI to LVEDVI showed a positive slope in all 12 patients with a reasonable correlation coefficient in most (table 2). The nonsurgical patients showed a significant correlation between a change in PAWP and LVEDVI (fig. 3).

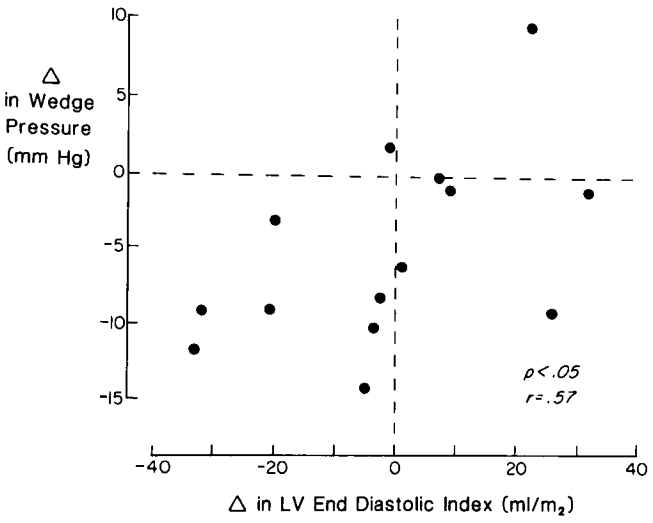


FIG. 3. Each dot (total = 14) represents a change in pulmonary artery wedge pressure (PAWP) and left ventricular end diastolic volume index (LVEDVI) for ten patients studied in the coronary care unit who were being treated with vasodilator therapy for chronic heart failure. There was a statistically significant relationship by Spearman rank order test ($P < 0.05$) with an r = 0.57 for changes in PAWP and LVEDVI.

Our method for measuring ejection fraction and calculating LVEDVI has been found to be accurate both in studies from our institution[8] as well as by other investigators.[9] Our measurements of the PAWP were done with special care to guarantee accurate results. The data were recorded at end-expiration on a strip chart recorder, and the transducers were recalibrated before each set of measurements to be certain that technical factors did not result in inaccurate measurements.[15,16] Prior studies have demonstrated that PAWP reflects left atrial pressure and left ventricular end diastolic pressure accurately in patients with normal hearts, heart disease, and after cardiac operations.[17–19]

The precise explanation for our finding of the poor correlation between PAWP and left ventricular end diastolic volume is uncertain. However, acute changes in left ventricular compliance demonstrated in seven of the 12 patients was almost certainly a contributory mechanism. Acute changes in ventricular compliance during coronary artery bypass graft surgery have been suggested by one group of investigators using similar techniques.[3] However, their data may have represented movement along the "flat portion" of the ventricular pressure–volume curve because the same investigators using identical techniques found concordant changes in PAWP and left

ventricular end-diastolic volume in another study at higher levels of PAWP during coronary artery bypass graft surgery.[20] Others have demonstrated a poor correlation between "left ventricular filling" and PAWP intraoperatively during coronary artery bypass graft surgery, but they measured left ventricular cross-sectional area (not volume) using echocardiographic methods.[2] Finally, changes in ventricular compliance during the rewarming phase after coronary artery bypass graft surgery have recently been described using techniques similar to those used in this study, but relating left atrial pressure to left ventricular end-diastolic volume.[21] In contrast, the patients in this study had already reached the end of the rewarming stage and, as will be discussed later, no difference was found between the "early" and "late" stage of this study.

Previous studies of ventricular function *in vivo* and *in vitro* have demonstrated several mechanisms that may be responsible for acute changes in left ventricular compliance: afterload reduction, use of vasopressors, myocardial ischemia, and ventricular interaction.

Vasodilator agents have been observed to shift the left ventricular pressure volume curve by producing a lower left ventricular end diastolic pressure for the same left ventricular end diastolic volume.[1,22] While four of the 12 postoperative patients were treated with sodium nitroprusside therapy during part or all of the study, apparent shifts in the pressure–volume relationships could not be explained by changes in systemic or pulmonary vascular resistance (table 3). Vasopressor drugs have been noted to increase both left and right ventricular pressures without changing left ventricular end diastolic volume.[23] Only four of the 12 postoperative patients, however, were treated with vasopressors during the course of this study.

Various models of ischemia including atrial pacing,[24,25] coronary occlusion,[26] and hypoxia[27,28] have been used to study both the stress–strain relationship in isolated muscle and the pressure–volume relationship of the heart. It has not been clear how ischemia effects the apparent pressure–volume relationship, *i.e.*, by decreasing systolic performance, altering ventricular distensibility, or both. One of the 12 surgical patients had a documented perioperative myocardial infarction. However, documentation of acute transient ischemia and its possible relationship to changes in the pressure–volume curve during a brief clinical period was not possible in this study.

Both animal and human data support the concept of ventricular interaction with contributions from both the pericardium and the interventricular septum. Some investigators have used excised dog hearts with an intact pericardium to demonstrate that with an increase in right ventricular volume and pressure, the left ventricular pressure–volume relationships change.[29,30] Other investigators[31] have demonstrated significant coupling between the right and left ventricles that may be weakened but still present after opening the pericardium.[32] Glantz *et al.*[32] and Hefner *et al.*[33] have demonstrated an important role for the pericardium in effecting the pressure–volume relationship. Because the pericardium was left open postoperatively in this study, significant ventricular interaction would not be expected. Indeed, no statistically significant relationship was found between the calculated changes in ventricular compliance and mean pulmonary arterial pressure or pulmonary vascular resistance.

The effects of positive-pressure ventilation must be considered. Fewell *et al.*[34] have shown that at positive end-expiratory pressure (PEEP) levels $\leq 12$ $cmH_2O$, pressure–volume curves of the left ventricle are not affected. All postoperative patients in this study had a low level of PEEP ($\leq 5$ $cmH_2O$), which remained constant throughout the study in all but one patient. Also, peak inspiratory pressures and plateau pressures remained stable for each patient throughout the study. As a result, the effects of pleural pressure on transmural pressure and the relationship to measured PAWP should not have altered during the 4 h of this study.

To determine whether the altered pressure–volume relationships were related to the time after bypass, data from the beginning and end of the first study h for each patient were paired with data from the beginning and end of the last study h. There was no significant difference between "early" and "late" PAWP or LVEDVI ($P = 0.64$, $P = 0.81$, respectively). Thus, the pressure–volume relationship was not a function of the 4-h time period in this study, although we did not study patients for longer time intervals.

In summary, this study demonstrated a poor relationship between a change in PAWP and left ventricular end diastolic volume in patients recovering in the intensive care unit from coronary artery bypass graft surgery. Although measurement of PAWP remains useful in clinical management to avoid pulmonary edema, it cannot be reliably used as an index of left ventricular preload while attempting to optimize stroke volume in patients immediately after coronary artery bypass graft surgery. We speculate that this is due to acute changes in the ventricular pressure–volume relationship. Further studies are needed to identify the pressure–volume relationships of the left ventricle over longer time periods following coronary artery bypass graft surgery.

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