
The Effect of the Pericardium on Ventricular Systolic Function in Man

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SUMMARY To determine the role of the pericardium in man, ventricular function was studied in 20 patients who underwent pericardiectomy during coronary artery bypass surgery. Left and right ventricular function curves were generated before and after pericardiectomy by changing body position to alter venous pressure. Central venous (0–14 mm Hg) and pulmonary wedge pressures (1–25 mm Hg) ranged over normal and moderately elevated values.

Pericardiectomy did not alter the relationship between left ventricular stroke work index and pulmonary wedge pressure, between right ventricular stroke work index and central venous pressure, or between pulmonary wedge pressure and central venous pressure.

These data suggest that in patients with normal and moderately elevated filling pressures, the pericardium does not influence right or left ventricular systolic function or the coupling between the right and left ventricles.

SEVERAL STUDIES IN ANIMALS indicate that the pericardium has significant effects on ventricular diastolic and systolic function, especially at elevated filling pressures. The pericardium affects left ventricular diastolic pressure-volume and pressure-segment length relationships in dogs subjected to changes in filling pressures by acute volume loading,1,2 administration of sodium nitroprusside,1 or obstruction to right and left ventricular outflow.4 Coupling between right and left ventricular filling pressures is enhanced by the pericardium, especially at markedly elevated filling pressures.5 Right ventricular stroke work is limited when the left ventricle is differentially stressed in dogs with elevated filling pressures.6 Although the pericardium influences ventricular function at markedly elevated filling pressures, its role at normal or moderately elevated filling pressures is controversial.

Opportunities to measure the effect of the pericardium on ventricular systolic and diastolic function in man are relatively rare. Patients who undergo cardiac surgery are unique in this regard in that they undergo pericardiectomy before cardiopulmonary bypass. In this study, we investigated the effects of the pericardium on right and left ventricular function curves (stroke work vs central venous [CVP] or pulmonary wedge pressure [PCW]) and on right and left ventricular pressure coupling (relationship between CVP and PCW) over a range of normal and moderately elevated filling pressures.

Methods

Twenty patients admitted for coronary artery surgery were studied. No patient had valvular disease, a history of right- or left-heart failure, or evidence of right or left ventricular hypertrophy or dilatation. Cardiac catheterization revealed 90% stenosis of two or more coronary arteries, ejection fractions ranging from 0.41–0.76 (normal 0.66 ± 0.06 [sd]), left ventricular end-diastolic volume indices from 54–81 ml/m² (normal assumed to be 70 ± 20 ml/m²), PCWs from 4–16 mm Hg, and CVPs from 1–10 mm Hg. Medications included isosorbide and nitroglycerin in all patients and propranolol (40–160 mg p.o. q.d.) in 14 patients. Medications were continued until 8 hours before surgery.

All patients were premedicated with morphine sulfate (10 mg i.m.) and oral diazepam (10 mg). Anesthesia consisted of morphine sulfate (1–1.5 mg/kg i.v.) and diazepam (0.25 to 0.50 mg/kg i.v.). Pancuronium (0.05–0.15 mg/kg i.v.) provided muscle relaxation, and ventilation (with 100% oxygen) was controlled. Hemodynamics were monitored by means

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of radial artery and triple-lumen thermodilution pulmonary artery catheters. All pressure measurements were recorded from equisensitive Bell and Howell transducers calibrated with mercury. Before each set of measurements, zero reference point was measured as 5 cm posterior to the sternal angle in a direction perpendicular to the frontal plane of the chest.

After median sternotomy, the sternum was retracted and the pericardium exposed. No surgical dissection was necessary to further expose the pericardium before the study. The pericardium remained fully intact and its mediastinal tethering was not disturbed. The sternal retractor remained in this position throughout the study. Surgical stimulation was stopped, systemic blood pressure and heart rate were monitored, and a 2-minute period of stability was established (systolic blood pressure varying less than 10 mm Hg and heart rate less than 5 beats/min). With the patient in the supine position (first control), we recorded these values at end-expiration: radial artery systolic and diastolic pressure, heart rate, pulmonary artery systolic and diastolic pressures, PCV, CVP and two cardiac outputs (within 10%) determined by thermodilution. Next, the lower extremities were elevated to a 45° position relative to the chest. After another 2-minute period of stabilization, the above measurements were recorded at end-expiration. The lower extremities then were elevated to 90° (perpendicular to the chest) and the measurements repeated. The fourth set of measurements was made with lower extremities returned to the 0° position (second control).

The anterior pericardium was then incised from apex to base and sutured laterally to the sternal edges to expose the heart fully. Surgery was again stopped, and a 2-minute period of hemodynamic stability established. Recordings were made in the 0°, 45°, 90° and 0° positions as previously outlined.

No pharmacologic agent or intravenous volume transfusion was given and no ventilatory adjustment was made before (15 minutes) or during the measurement period.

**Results**

No significant difference was found by paired-sample t test between the pre- and postpericardiotomy control values for heart rate (p > 0.10), systemic blood pressure (p > 0.05), systemic vascular resistance (p > 0.10), pulmonary artery pressure (p > 0.05) or pulmonary vascular resistance (p > 0.10) (table 1). Similarly, no significant (p > 0.10) difference was found between the pre- and postpericardiotomy values of systemic vascular resistance or pulmonary vascular resistance at each position (0°, 45° and 90°).

| Table 1. Control Hemodynamic Variables Before and After Pericardiotomy |
|-----------------------------|-----------------------------|-----------------------------|
|                             | Before pericardi- | After pericardi- | Average difference† |
|                             | otomy*           | otomy*           |                |
| Heart rate (beats/min)      | 64.5 ± 3.3       | 67.3 ± 3.1       | 2.3 ± 0.5      |
| Systolic pressure (mm Hg)   | 122.7 ± 4.3      | 117.8 ± 4.1      | 4.6 ± 0.4      |
| Diastolic pressure (mm Hg)  | 72.8 ± 3.9       | 70.2 ± 3.3       | 2.1 ± 0.3      |
| Systemic vascular resistance (dyn-sec-cm⁻⁵) | 1584.9 ± 121.2 | 1630.1 ± 141.8 | 81.1 ± 20.2 |
| Cardiac index (l·min⁻¹·BSA⁻¹) | 2.47 ± 0.13 | 2.38 ± 0.18 | 0.19 ± 0.08 |
| Pulmonary artery pressure (mm Hg) | 22.3 ± 2.4 | 24.1 ± 2.2 | 2.1 ± 0.3 |
| Diastolic pressure (mm Hg)   | 10.2 ± 1.2       | 10.9 ± 1.3       | 1.0 ± 0.2      |
| Pulmonary vascular resistance (dyn-sec-cm⁻¹) | 93.2 ± 12.1 | 97.3 ± 14.3 | 7.1 ± 0.4 |

*Values averaged over 20 patients. All values are mean ± SEM.
†For each patient the difference between the pre- and postpericardiotomy values was calculated. Average difference is the mean value (over 20 patients) of the individual differences. All values are mean ± SEM.

diotomy and three postpericardiotomy data points. The three points are derived from the measurements made in the 0° (first control), 45° and 90° positions. A statistical analysis of this data was performed using a paired-sample test. For each patient the pre- and postpericardiotomy data were paired at each lower extremity position (0°, 45° and 90°). There are three pairs of data points for each patient, or 60 pairs for the 20 patients. The statistical analyses were performed on these 60 pairs. Pericardiotomy did not significantly change the left ventricular stroke work (p > 0.10) or the right ventricular stroke work (p > 0.10) at any lower-extremity position. The data associated with normal filling pressures were compared with those associated with abnormal filling pressures. The 60 pairs for the left ventricular function curve were divided into two groups: those with a normal PCW (≤ 12 mm Hg) and those with an abnormal PCW (> 12 mm Hg). Neither group demonstrated a significant (p > 0.05) difference between the pre- and postpericardiotomy data. Right ventricular function curve data were also subgrouped (CVP ≤ 6 mm Hg, CVP > 6 mm Hg), and no difference (p > 0.05) was found.

Combining data for all patients, analysis of variance revealed no difference in the slope (− 0.50 vs − 0.54, p > 0.10) or intercept (54.1 vs 55.8, p > 0.10) of the regression of left ventricular stroke
work index on PCW before and after pericardiotomy, nor did pericardiotomy affect the slope (−0.03 vs 0.03, p > 0.10) or intercept (7.43 vs 6.26, p > 0.05) of right ventricular stroke work index on CVP.

The relationship between CVP and PCW for each of the 20 patients before and after pericardiotomy is shown in figure 3. Grouping the data from all patients (60 pairs), a paired t test comparison revealed no significant (p > 0.05) difference in the relationship between CVP and PCW. Similarly, regression line analysis showed no difference with pericardiotomy.

We attempted to correlate the shape of the ventricular functions with preoperative ejection fraction, extent of dyskinesis, age and distribution of coronary disease, and found no correlation.

Discussion

Our results support the hypothesis that in patients with normal or moderately elevated filling pressures, the pericardium has little effect on ventricular function. Ventricular systolic function, assessed by means of ventricular function curves, and ventricular pressure-coupling, assessed by means of the CVP-PCW relationship, were unaffected by pericardiotomy.

Our approach to the assessment of ventricular systolic function merits discussion. PCW and CVP may not be exact estimates of their respective ventricular end-diastolic pressures. For example, in patients with acute myocardial infarction, neither PCW nor left ventricular mean diastolic pressure accurately reflect left ventricular end-diastolic pressure because of the contribution of the left atrial "kick." Although no patient had evidence of myocardial infarction (by ECG, enzymes or pyrophosphate scan) during or after our study, it has not been established that PCW and CVP are accurate estimates of their respective filling pressures in this class of patients. Thus, the ventricular function curves used in our study are only estimates of stroke work vs end-diastolic
volume curves. Nevertheless, one clinically important effect of changes in left ventricular compliance is on PCW, and our results show that at the levels of venous return evaluated, pericardiectomy did not alter ventricular stroke work or estimated diastolic pressure (PCW and CVP). Further, vascular resistance was not statistically different in any patient before and after pericardiectomy at any level of PCW (CVP). Thus, comparison of the pre- and postpericardiectomy curves reflects only the relationship between stroke work and PCW (CVP), and does not reflect alterations in outflow resistance.

The effects of the pericardium on ventricular systolic function have been studied in dogs. Berglund et al. found that the pericardium limited right ventricular expansion and right ventricular stroke work when the left ventricle was differentially stressed by increasing outflow resistance. These effects were most marked at high filling pressures. Moulopoulos et al. studied left ventricular function curves during bypass and distension of the right ventricle with the pericardium open. They found impairment of left ventricular function only at relatively high right ventricular end-diastolic pressure. In man, Bartle and Hermann studied ventricular pressure contours 5 days after the appearance of acute mitral regurgitation and found...
that these contours resembled those observed in constrictive pericarditis. These studies suggest that the pericardium can substantially affect ventricular systolic function when filling pressures are markedly elevated. Neither the left nor right ventricular function curves were affected by pericardiectomy over normal values of estimated filling pressures in any of the 20 patients. In five of these patients the filling pressures were moderately elevated, and pericardiectomy produced no change.

Although the results of this study may differ from those for intact unanesthetized man, these differences have been minimized. The anesthetic used, morphine sulfate with oxygen, does not depress ventricular contractility at the doses used in this study. Also, all measurements were made at end-expiration, when the difference between pleural pressures with the chest open vs closed is minimal.

The effects of the pericardium on ventricular diastolic function have been studied in dogs by using volume loading and occlusion of ventricular outflow tracts. These studies suggest that the pericardium may restrict ventricular filling at high end-diastolic pressure. However, the level of filling pressure at which the pericardium begins to assume an important role is not well delineated. Recently, Mirsky and Rankin found in dogs that shifts in ventricular intracavity pressure-volume relationships are due to pericardial pressure; however, the transmural pressure-volume relationships were not markedly altered. They concluded that no alteration in the intrinsic ventricular compliance occurred. In our study ventricular diastolic function was not assessed directly, but the coupling between estimates of ventricular diastolic pressures was studied. This indirect measure of the effect of the pericardium on relative
diastolic filling was also used by Glantz et al. in dogs. They found that with the pericardium closed, coupling between left and right end-diastolic pressures was tighter than with it open and that these effects were more marked at high end-diastolic pressures. They suggested that this coupling is determined by the ventricular muscle as well as by the stiffer pericardium. In our study, removal of the pericardium did not change the coupling relationship between CVP and PCW over normal and moderately elevated ranges. Our results in man suggest that the effects of the pericardium on this right-left filling pressure relationship are minimal when pressures are normal to moderately elevated. The ventricular muscle itself, and not the stiffer pericardium, appears to be the major determinant of the relationship between these filling pressures.

The results of this study support the hypothesis that the pericardium does not have a significant effect on ventricular systolic function or pressure-coupling in patients with normal to moderately elevated filling pressures.

References

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