Mechanisms of Blood Flow During Cardiopulmonary Resuscitation

MICHAEL T. RUDIKOFF, M.D., W. LOWELL MAUGHAN, M.D., MARK EFFRON, M.D., PAUL FREUND, AND MYRON L. WEISFELDT, M.D.

SUMMARY Despite the widespread clinical application of cardiopulmonary resuscitation (CPR), the mechanism responsible for blood flow during this maneuver remains undefined, although it has been assumed that blood is squeezed from the heart by direct compression of the sternum. We studied the hemodynamics of CPR in 15 arrested dogs. During chest compression, pressures in the left ventricle, aorta, right atrium and pulmonary artery were essentially identical. These pressures were also equal to the intrathoracic pressure as estimated by an esophageal balloon catheter. Unequal transmission of pressures to the extrathoracic arterial and venous system resulted from collapse of the great veins at the thoracic outlet as intrathoracic pressures rose. This phenomenon gave rise to a peripheral arteriovenous pressure gradient and antegrade flow. When intrathoracic pressure was increased by maintaining the lungs fully inflated during chest compression, aortic systolic pressure rose from 27.3 ± 4.0 mm Hg to 58.4 ± 7.9 mm Hg (p < 0.001) and carotid blood flow increased from 9.0 ± 2.2 ml/min to 28.6 ± 5.9 ml/min (p < 0.001). Increasing the intrathoracic pressure by tightly binding the abdomen to prevent paradoxical diaphragmatic motion during chest compression also resulted in a rise in aortic systolic pressure from 29.4 ± 3.2 to 57.7 ± 7.7 mm Hg (p < 0.001), and an increase in carotid blood flow, from 14.5 ± 8.1 ml/min to 32.3 ± 9.7 ml/min (p < 0.005). It appears that pressure generation and blood flow during CPR in the dog result from a generalized rise in intrathoracic pressure, not from direct cardiac compression. Maneuvers that raise the intrathoracic pressure can dramatically increase carotid blood flow during CPR.

THE CONCEPT that blood moves during cardiopulmonary resuscitation (CPR) as a result of direct compression of the heart between the sternum and the vertebral column was originally proposed by Kouwenhoven, Jude and Knickerbocker in 1960. Although contradictory views have been expressed, this concept remains widely accepted despite the lack of supporting scientific data. Recently, Taylor and associates reported that prolonged compression is vital in optimizing carotid flow in man during CPR. At a constant compression force, we repeatedly observed that the compression initiated at end-inspiration was characterized by augmented arterial pressure and blood flow. Because the lung is inflated, this compression is one in which the sternum is farther from the vertebral column and there should be less direct compression of the heart. In addition, emphysematous patients with increased antero-posterior chest dimension and relatively small heart size are readily resuscitated despite a thoracic anatomy that makes direct cardiac compression unlikely. We also observed two patients with flail sternums as a result of previous chest trauma who suffered cardiac arrest and noted that their arterial blood pressure did not rise during chest compression, although a flail sternum should allow ready compression of the heart. It was only when the paradoxical motion of the rest of the chest wall was restricted by placing a belt around the thoracic cage that sternal compression resulted in a detectable rise in arterial pressure. All of these observations suggested to us that the common denominator for movement of blood during CPR in man was an increase in intrathoracic pressure, not direct cardiac compression.

This notion was further supported by the observation of Criley and associates, who demonstrated that repeated coughing alone without chest compression can produce effective arterial pressure and sufficient cerebral blood flow to maintain the conscious state in patients during ventricular fibrillation.

In this study, we explored the hypothesis that a rise in the intrathoracic pressure results in movement of blood during chest compression and tried to clarify how intrathoracic pressure might be transmitted unequally to the peripheral arterial and venous tree to produce the peripheral arteriovenous pressure gradient necessary for blood flow. A preliminary report has been presented.

Methods

Studies were performed in 15 mongrel dogs that weighed 20–45 kg. After sodium pentobarbital anesthesia (25 mg/kg i.v.), vessels were exposed in the neck and inguinal areas. All dogs were intubated with a cuffed endotracheal tube. Millar micromanometer catheters were placed via the right femoral artery and vein into the descending thoracic aorta and right atrium, respectively. Fluid-filled catheters were placed in the left ventricle and pulmonary artery via the left femoral vessels and in the left external jugular vein and carotid artery via side branches. In addition, a balloon-tipped, fluid-filled catheter was passed via the oral cavity into the thoracic portion of the esophagus.
Pressures in the fluid-filled catheters were measured with Statham P23Db transducers. Catheter positions were confirmed by characteristic pressure recordings and position at the termination of the study. Mean systemic pressure was determined periodically by stopping the chest compression and ventilation and allowing 20 seconds for equilibration of intravascular pressures. Millar catheters were calibrated against mercury in vitro and adjusted for baseline drift in vivo by comparison with pressure recorded through the lumen of the catheter. After administration of heparin (300 units/kg i.v.), cannulating electromagnetic flow probes were placed in the right carotid artery and external jugular vein and flow was measured with a Biotronix BL 613 sine-wave flow meter. All data were recorded on a Brush Mark 600 recorder. Minute flows were subsequently calculated by integration of the instantaneous flow trace using a Hewlett Packard 9810A calculator and 9468A digitizer. In expressing these results, antegrade flow refers to flow in the jugular vein toward the chest and in the carotid artery away from the chest.

After recording of control pressures and flows, ventricular fibrillation was induced with a transthoracic electrical current. Anteroposterior external chest compression was initiated after 15 seconds by means of a pneumatic chest compression device set to compress the chest once per second with a compression duration of 0.5 seconds and a compression force of 140 pounds. These procedures were not changed during the course of the study. Every fifth compression, diastole was prolonged by 0.5 second and the lungs were inflated to an inspiratory pressure of 45 cm H₂O by a synchronized, pressure-limited ventilator. The beat after ventilation was arbitrarily defined as beat 1 of the five-beat cycle.

Additional Protocols

Airway Occlusion at End-inspiration

Pressures and flows were recorded during standard CPR, i.e., with the airway open to atmosphere during chest compression. The airway was then occluded at end-inspiration by clamping the endotracheal tube. With the lungs held inflated, pressures and flows were recorded during the next five beats.

Abdominal Binding

An inflatable bladder was placed over the abdomen and attached securely by wrapping the area between the xiphoid and iliac crest with adhesive tape. Firm abdominal pressure was then produced by inflating the bladder. This pressure was maintained constant during subsequent chest compressions.

Airway Occlusion After Binding

After binding the abdomen, the protocol for airway occlusion at end-inspiration was repeated. All observations were completed within 15 minutes of the onset of ventricular fibrillation, but usually within 10 minutes. Carotid flow during standard CPR before and after each additional protocol differed by less than 15%, falling slightly in most dogs. Arterial Po₂ was >300 mm Hg in all dogs and Pco₂ was 22-35 mm Hg.

Results

Pressure and Flow Pattern During CPR

Each chest compression produced positive pressure pulses of similar magnitude in the right atrium, left ventricle, aorta and carotid artery (fig. 1) and antegrade carotid blood flow (12.3 ± 2.7 ml/min) (mean ± SEM for five-beat cycle in 13 dogs). When compression was released, there was significant retrograde carotid blood flow (3.3 ± 1.0 ml/min), as well as antegrade jugular flow (fig. 2). There was negligible retrograde jugular flow during chest compression despite a large pressure gradient between the right atrium and the jugular vein outside the chest (table 1). Jugular pressure rose slowly during systole, probably as a result of antegrade filling.

The systolic aortic pressure was dependent on the state of lung inflation. Beat 1, the first chest compression after lung inflation, produced higher systolic aortic pressures than the subsequent beats of the compression cycle that occurred with the lungs deflated (table 1). This augmentation of aortic pressure was produced by lung inflation rather than by the associated prolonged diastolic pause, because the

![Figure 1](http://circ.ahajournals.org/)

**Figure 1.** Intravascular and intracardiac pressures and carotid flow during cardiopulmonary resuscitation. With each chest compression there is a positive pressure pulse in each of the cardiac chambers and arterial vessels and antegrade carotid blood flow. Pressures are essentially identical during compression in the left ventricle (LV), right atrium (RA) and thoracic aorta (Ao). The beat after ventilation (beat 1) has augmented pressure and carotid blood flow.
augmentation did not occur when the ventilator was turned off during the pause.

Systolic pressures were similar in each of the cardiac chambers and intrathoracic great vessels for each beat of the compression cycle (table 1). In nine dogs in which aortic and esophageal pressures were recorded simultaneously, these systolic pressures were also nearly identical (fig. 3). Corresponding aortic and jugular pressures show that the jugular venous pressure was consistently lower than the aortic pressure (table 1). Carotid pressures were also slightly lower than aortic pressure for each beat of the compression cycle, but higher than jugular venous pressure (table 2).

The near-equality of systolic pressures in each of the cardiac chambers, the intrathoracic great vessels and the intrathoracic pressure as reflected in the esophagus, and the variation of all these pressures with the state of lung inflation, strongly suggests that the rise in intravascular pressures during chest compression is produced by a generalized rise in intrathoracic pressure, not by direct cardiac compression. The unequal transmission of these pressures to the carotid artery and jugular vein results in an extrathoracic peripheral gradient for blood flow.

Airway Occlusion at End-inspiration

In 13 dogs we sought to improve blood flow through increases in systolic intrathoracic pressure. Occluding the airway at peak inspiration raised systolic aortic pressure during subsequent chest compressions (fig. 4). Airway occlusion resulted in increased aortic and carotid pressures for beats 3 and 5 (fig. 5). Beat 1 showed little increase; even without airway occlusion the lungs were inflated during beat 1 as a result of ven-

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**Table 1. Systolic Pressure During Standard Cardiopulmonary Resuscitation**

<table>
<thead>
<tr>
<th>Chamber</th>
<th>Beat 1 (mm Hg)</th>
<th>Beat 3 (mm Hg)</th>
<th>Beat 5 (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aorta</td>
<td>45.0 ± 7.1</td>
<td>22.0 ± 4.7†</td>
<td>22.7 ± 4.4†</td>
</tr>
<tr>
<td>Right atrium</td>
<td>42.0 ± 4.6</td>
<td>21.4 ± 1.5</td>
<td>22.1 ± 1.5</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>47.0 ± 6.4</td>
<td>25.1 ± 4.0</td>
<td>24.6 ± 3.9</td>
</tr>
<tr>
<td>Left ventricle</td>
<td>47.6 ± 6.0</td>
<td>24.6 ± 4.1</td>
<td>25.4 ± 4.1</td>
</tr>
<tr>
<td>Jugular vein</td>
<td>20.7 ± 4.3*</td>
<td>12.6 ± 1.8*</td>
<td>12.4 ± 1.7*</td>
</tr>
</tbody>
</table>

All values are mean ± SEM; n = 7.
* p < 0.01 vs aorta.
† p < 0.001 vs aorta beat 1.

**Table 2. Systolic Pressure During Standard Cardiopulmonary Resuscitation**

<table>
<thead>
<tr>
<th>Chamber</th>
<th>Beat 1 (mm Hg)</th>
<th>Beat 3 (mm Hg)</th>
<th>Beat 5 (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aorta</td>
<td>73.0 ± 4.3</td>
<td>34.2 ± 4.4</td>
<td>33.8 ± 4.4</td>
</tr>
<tr>
<td>Carotid artery</td>
<td>47.3 ± 5.6*</td>
<td>25.8 ± 2.4</td>
<td>23.5 ± 2.2</td>
</tr>
<tr>
<td>Jugular vein</td>
<td>29.0 ± 4.3†</td>
<td>16.8 ± 1.8‡</td>
<td>16.8 ± 1.9‡</td>
</tr>
</tbody>
</table>

All values are mean ± SEM; n = 10.
* p < 0.005 vs aorta.
† p < 0.02 vs carotid artery.
‡ p < 0.001 vs carotid artery.
tilation immediately before beat 1. The increased systolic pressures were accompanied by an increase in carotid blood flow in 12 of the 13 dogs studied (fig. 6). Chest compression with the lung held inflated again produced nearly identical systolic aortic and esophageal pressures of 69.1 ± 8.4 and 66.9 ± 7.2 mm Hg for beat 1 and 58.4 ± 7.9 and 60.3 ± 6.2 mm Hg for beat 3. Pressures in the right atrium, ventricle and pulmonary artery were also nearly identical to aortic pressure. These data confirm the dependence of intravascular pressures on intrathoracic pressure and indicate the ability to effect changes in blood flow and pressure through manipulation of intrathoracic pressure.

Abdominal Binding

Tightly binding the abdomen in 15 dogs had two major effects (fig. 7). First, mean systemic pressure rose from 10.7 to 20.0 mm Hg \((p < 0.001)\). Only 2.6 ± 1.1 mm Hg of this rise could be accounted for by a rise in diastolic intrathoracic pressure. The remaining change in mean systemic pressure reflected increased transmural filling pressure probably related to an increase in effective intravascular volume. The second major effect was a rise in systolic pressure in excess of that accounted for by the diastolic rise produced nearly identical systolic aortic and esophageal pressures of 69.1 ± 8.4 and 66.9 ± 7.2 mm Hg. The difference between systolic aortic and esophageal pressure was only 2.0 ± 1.8 mm Hg. Systolic carotid pressure increased for each beat (fig. 7). Carotid blood flow increased in 13 of 15 dogs studied (fig. 8).

The relative importance of the volume shift vs the increased systolic pressure in the augmentation of carotid blood flow was assessed by intravenous infusion 1000–2000 ml of normal saline in six dogs. Mean systemic pressure increased 6.7 ± 2.0 mm Hg (compared with 9.5 mm Hg with abdominal binding). However, carotid blood flow did not increase significantly. These data suggest that the rise in systolic pressure accounts for most of the increased

Figure 4. Airway occlusion at end-inspiration. All pressures and carotid blood flow increased during subsequent chest compressions. Aortic (Ao), right atrial (RA), left ventricular (LV) and esophageal (ESOPH) pressures are shown.

Figure 5. Aortic and carotid arterial systolic pressure during cardiopulmonary resuscitation before and after airway occlusion at peak inspiration. After airway occlusion, aortic pressures for beats 3 and 5 and carotid pressure for beats 1, 3 and 5 were higher.
blood flow produced by abdominal binding. In addition, abdominal binding may decrease the size of the perfused arterial bed.

**Airway Occlusion After Abdominal Binding**

In 14 dogs the airway was occluded at peak inspiration after the abdomen was bound. Aortic pressure rose for each beat (table 3). In seven dogs there was an increase in carotid flow, from 18.3 ± 9.1 to 33.9 ± 14.1 ml/min. However, carotid collapse occurred in the remaining seven dogs and there was a fall in carotid blood flow, from 54.6 ± 21.0 to 30.1 ± 21.7 ml/min (fig. 9). In the group as a whole, carotid blood flow changed little, from 31.9 ± 11.6 to 32.0 ± 12.5 ml/min (NS).

**Discussion**

Although blood flow during CPR has been presumed to result from direct cardiac compression between the sternum and vertebral column, the present canine studies suggest an alternative mechanism. The first event is a rise in intrathoracic pressure produced by chest compression alone or in combination with other maneuvers, such as airway occlusion.

**Table 3. Effect of Airway Occlusion on Systolic Pressure (mm Hg) After Abdominal Binding**

<table>
<thead>
<tr>
<th></th>
<th>Abdominal binding alone</th>
<th>Binding and airway occlusion</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aorta</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beat 1</td>
<td>82.9 ± 6.1</td>
<td>95.6 ± 7.8*</td>
</tr>
<tr>
<td>Beat 3</td>
<td>49.4 ± 5.7</td>
<td>91.4 ± 7.5*</td>
</tr>
<tr>
<td>Beat 5</td>
<td>50.6 ± 5.8</td>
<td>90.0 ± 7.6*</td>
</tr>
<tr>
<td><strong>Carotid</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beat 1</td>
<td>50.8 ± 9.9</td>
<td>53.0 ± 9.0</td>
</tr>
<tr>
<td>Beat 3</td>
<td>39.6 ± 6.9</td>
<td>50.1 ± 7.9†</td>
</tr>
<tr>
<td>Beat 5</td>
<td>40.1 ± 7.5</td>
<td>49.0 ± 8.1</td>
</tr>
</tbody>
</table>

All values are mean ± SEM.

* *p < 0.001 vs binding alone.
† *p < 0.05 vs binding alone.
This rise in intrathoracic pressure is reflected as an equal rise in pressure in each of the cardiac chambers and intrathoracic great vessels. The relatively small gradient between the aorta and carotid artery indicates that there is relatively direct transmission of the pressure from the arterial system within the chest to the arterial system outside the chest. Despite a large apparent pressure gradient, there is minimal flow of blood from the intrathoracic venous system to the extrathoracic veins. This results in a smaller systolic pressure rise in the extrathoracic veins with chest compression (fig. 2). This unequal transmission of intrathoracic pressure to the extrathoracic arterial and venous systems results in a peripheral systolic arteriovenous pressure gradient with antegrade blood flow. When chest compression is released, intrathoracic pressure falls below venous pressure and blood from the extrathoracic venous system flows into the chest.

The absence of larger-volume retrograde flow into the jugular vein during chest compression is critical to this proposed mechanism. We are accustomed to considering flow as proportional to the gradient between the high-pressure, or inflow, and low-pressure, or outflow, ends of a conduit. In a very collapsible tube, such as a systemic vein, this relationship applies only if the pressure surrounding the tube is less than the outflow pressure. When surrounding pressure exceeds outflow pressure, flow will depend instead on the gradient between inflow and surrounding pressures. In these studies on the venous side, outflow pressure (jugular) is low and inflow pressure (right atrial) and outside pressure (intrathoracic) are similar during compression. Thus, there is little gradient for retrograde flow. A small gradient is probably present at the onset of compression, because filling of the right heart and central veins causes right atrial pressure to exceed intrathoracic pressure slightly. The large pressure gradient between the right atrium and the extrathoracic jugular vein during compression probably results from venous collapse. These conclusions are supported by the minimal measured retrograde flow in the jugular vein after the initial brief retrograde jet (fig. 2). In isolated systems, collapse occurs just inside the region with high outside pressure; that is, during chest compression, collapse would be expected to occur just inside the thorax. The high resistance of the collapsed segment of vessel and negligible driving gradient result in little or no flow.

Our data indicate that this collapse phenomenon is less marked on the arterial side. The arterial system is intrinsically somewhat stiffer than the venous system and may thereby be less subject to collapse from the high surrounding intrathoracic pressure. In addition, because the capacity of the extrathoracic arterial system is less than that of the venous system, any blood leaving the thorax will produce a relatively greater extrathoracic intravascular pressure, which will tend to prevent vascular collapse with rapid movement of blood from the thoracic arteries at the onset of compression. Clearly, complete collapse of arterial structures does occur under conditions of extremely high intrathoracic pressure (fig. 9).

The dependence of the aortic pressure and thereby, carotid arterial pressure on intrathoracic pressure suggested that blood pressure and flow would be improved by maneuvers that raise intrathoracic pressure. When the airway was occluded at the end-inspiration, the lungs remained fully inflated during subsequent chest compressions. Aortic pressure increased and remained similar to intrathoracic pressure measured in the esophagus. The increase in aortic and carotid pressures was associated with a more than doubling of carotid blood flow. Abdominal binding, first suggested by Harris et al., proved to be a strikingly effective technique for augmenting carotid blood flow during external chest compression. By restricting paradoxical diaphragmatic movement during chest compression, abdominal binding increased intrathoracic pressure and thereby, aortic systolic pressure. Also, there may be some benefit in terms of carotid flow from limitation of abdominal flow with vascular compression. Volume infusion alone did not have a consistently beneficial effect. This suggests that the favorable effect of binding is not mainly due to blood volume shifts. Liver lacerations from the abdominal compression were sought routinely but not found in any of the dogs studied.

By maintaining the lungs inflated after the abdomen had been bound, we were able to achieve very high systolic aortic pressures, frequently greater than 100 mm Hg. Despite the high aortic pressure, carotid
pulse pressure and flow frequently fell. The development of a large systolic pressure gradient from the aorta to the carotid artery indicates arterial collapse. The mechanism of the sudden complete arterial collapse seen in the first beat after airway occlusion (fig. 9) probably results from the mechanism of venous collapse discussed above.

Recently, Mashiro and associates\textsuperscript{12} measured intracardiac pressures and dimensions after ventricular fibrillation induced by coronary embolism or by electrolyte changes. Evidence of left ventricular contracture was found with left ventricular pressures exceeding right ventricular pressures for 15 seconds. To ensure sustained ventricular fibrillation, we did not begin chest compression for 15 seconds. At the onset of compression, pressure and flows were as presented. Thirty seconds after the onset of fibrillation, diastolic pressure in the aorta was $13 \pm 1$ mm Hg in seven dogs. Aortic and right atrial systolic pressures were similar, with a maximal difference of 3 mm Hg and a mean systolic aortic pressure of $57 \pm 3$ mm Hg for beat 1. During the study, the diastolic pressure fell slightly.

The obvious differences in thoracic anatomy dictate caution in the extrapolation of these data to man. Confirmation of a gradient in the jugular vein at the thoracic outlet in man (fig. 10) indicates that venous collapse occurs during human CPR and suggests that the mechanism of flow identified in the dog at least contributes to flow in man. With differences in chest configuration, direct cardiac compression may also occur in some humans. The anecdotal observations described in the introduction, however, could not easily be explained in terms of direct cardiac compression in man. The critical data in man would be demonstration of the equality of intrathoracic and aortic pressures. We have not been able to perform aortic catheterization because appropriate consent cannot be obtained. Mean radial artery pressures during resuscitation in man define two groups: 1) a high-pressure group ($n = 11$, or 30\%), beat 1 = $79 \pm 30$ mm Hg), in which direct cardiac compression may play a major role, and 2) a group in which mean radial artery pressure ($n = 26$, or 70\%), beat 1 = $40.4 \pm 2.0$ mm Hg) approximates the mean carotid pressures in the canine study group (table 2). After completion of the reported studies, a 12-kg dog with a flat sternum was resuscitated, and a higher aortic than right atrial systolic pressure was observed. Direct cardiac compression probably occurred here, as with 30\% of the humans studied. Mean radial pressure was selected because vasoconstriction markedly damps radial artery pressure during CPR in man. It has not escaped our attention that clinical CPR\textsuperscript{13} and, in fact, other circulatory support systems, might well be substantially improved by use of the principle of increased blood flow through increases in intrathoracic pressure in many patients. Expansion of blood volume and/or augmentation of pulmonary blood flow might limit the possibility of arterial collapse and the strategy might provide very substantial overall increases in blood flow to the carotid and other arterial beds. Under all conditions studied to date the aortic diastolic pressure relative to atmosphere is extremely low: 35 mm Hg. The low aortic pressure must indicate a very small volume of blood in the central arterial vessels and raises concern regarding the adequacy of coronary perfusion in any life-support system using intrathoracic pressure to move blood into the peripheral tissues.

\section*{Addendum}

At the 52nd Scientific Sessions of the American Heart Association in Anaheim, California, November 1979, J.T. Niemann, D. Garner, J. Rosborough, and J.M. Criley presented convincing evidence that there is an anatomic and functional venous valve near the thoracic outlet that contributes to the establishment of the low extrathoracic venous pressure under conditions of high intrathoracic pressure noted in these studies. (With permission of Dr. Criley and associates.)

\section*{Acknowledgment}

We are indebted to the Emergency Care Research Institute (Waltham, Massachusetts) for providing the card-programmable cardiopulmonary-resuscitation equipment used in this study. This equipment was engineered and manufactured for Emergency Care Research Institute, and to specifications for use in their resuscitation physiology research program by Michigan Instruments, Inc., Ann Arbor, Michigan.

\section*{References}

7. Permutt S, Riley RL: Hemodynamics of collapsible vessels

\begin{figure}[h]
\centering
\includegraphics[width=0.5\textwidth]{figure10}
\caption{During cardiopulmonary resuscitation in a patient with a central venous catheter inserted percutaneously via the internal jugular vein, the catheter was slowly withdrawn. When the catheter tip reached the thoracic outlet, systolic pressure suddenly fell. The paper speed is 5 mm/sec.}
\end{figure}
The Effect of the Pericardium on Ventricular Systolic Function in Man

DENNIS T. MANGANO, PH.D., M.D.

SUMMARY To determine the role of the pericardium in man, ventricular function was studied in 20 patients who underwent pericardiotomy during coronary artery bypass surgery. Left and right ventricular function curves were generated before and after pericardiotomy 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.

Pericardiotomy 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, administration of sodium nitroprusside, or obstruction to right and left ventricular outflow. Coupling between right and left ventricular filling pressures is enhanced by the pericardium, especially at markedly elevated filling pressures. Right ventricular stroke work is limited when the left ventricle is differentially stressed in dogs with elevated filling pressures. 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 pericardiotomy 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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