Visualization of Cardiac Valve Motion in Man During External Chest Compression Using Two-dimensional Echocardiography

Implications Regarding the Mechanism of Blood Flow

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SUMMARY Five patients who underwent cardiopulmonary resuscitation (CPR) were studied by two-dimensional echocardiography to assess valve motion. The mitral valve remained open throughout the entire compression-release cycle of CPR. The aortic valve opened during the compression phase of CPR and closed during the release phase. The pulmonic valve moved toward the closed position during the compression phase and the valve leaflets opened during release. Tricuspid valve leaflets never completely apposed, even during maximum chest compression, and they were widely open during release. Left ventricular dimensions did not change appreciably during CPR.

These findings support the theory that forward blood flow during CPR depends on a generalized increase in intrathoracic pressure and not on direct compression of the heart itself. The left heart appears to act as a conduit for passage of blood, and mitral valve closure is not necessary for forward blood flow during CPR.

EXTERNAL CHEST COMPRESSION and airway ventilation are effective in maintaining some perfusion and oxygenation in victims of cardiac arrest until definitive treatment, defibrillation and/or cardiovascular drugs can be administered. However, questions still exist regarding the actual mechanism of forward blood flow during cardiopulmonary resuscitation (CPR) as conventionally applied. The central issue in this controversy is whether forward blood flow occurs primarily as a result of direct mechanical compression of the heart between the sternum and vertebrae or, as more recently argued, that the left heart in particular is acting passively as a conduit for blood forced out of the lungs along a pressure gradient primarily generated by increasing intrathoracic pressure. The latter theory of blood flow in CPR (the “chest-pump” theory) recently received considerable physiologic support from Rudikoff et al.1

Fundamental to this hypothesis in man is the demonstration of cardiac valve motion during conventional external chest compression. In an attempt to demonstrate these changes directly, we performed two-dimensional echocardiography during CPR by manual external chest compression in five patients.

Methods

Five patients were studied during CPR in the Coronary and Medical Intensive Care Units at Harborview Medical Center between February and June 1980. All patients were ventilated by endotracheal tubes. Pulmonary artery catheters and indwelling radial artery catheters had been inserted previously for continuous pressure measurements in four of five patients. In one patient without intraarterial monitoring, femoral artery pulses were monitored manually and by transcutaneous Doppler external flow probe during chest compressions.

Two-dimensional echocardiography was performed during CPR using an Advanced Technology Laboratories Mark III mechanical sector scanner and recorded on videotape, simultaneous with audio track. Parasternal, apical and subcostal approaches were attempted in all patients. In four of the five patients M-mode and two-dimensional echocardiograms had been performed for clinical reasons during spontaneous cardiac rhythm in the 24 hours before the CPR study, allowing for selection of the best acoustic windows in a given patient. Having prior echocardiograms also permitted comparison of chamber sizes and valve appearance during external chest compression with those in spontaneous rhythm. The long-axis and short-axis parasternal positions were the most useful during external chest compression. Informed consent was obtained from immediate relatives before the studies in four of five cases. The fifth patient had no living relatives or legal custodians who could be identified before death.

Results

Patients

Three males and two females were studied. The mean age was 45 years (range 28–58 years). Three of four patients reported prior heart disease (table 1). No patient had a prolonged or complicated hospital course. No patient was thought to have severe acute or chronic lung disease. No patient had known primary valvular heart disease.
TABLE 1. Patients

<table>
<thead>
<tr>
<th>Pt</th>
<th>Cardiac diagnosis</th>
<th>Cause of death</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Old myocardial infarction</td>
<td>Subarachnoid hemorrhage</td>
</tr>
<tr>
<td>2</td>
<td>s/p ventricular fibrillation</td>
<td>Circulatory shock</td>
</tr>
<tr>
<td>3</td>
<td>Cardiomyopathy</td>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>4</td>
<td>s/p ventricular fibrillation</td>
<td>Alcoholism, aspiration</td>
</tr>
<tr>
<td></td>
<td></td>
<td>pneumonia, multiple metabolic abnormalities</td>
</tr>
<tr>
<td>5</td>
<td>Hypertension, LVII,</td>
<td>Acute and chronic</td>
</tr>
<tr>
<td></td>
<td>s/p ventricular</td>
<td>subdural hematomas</td>
</tr>
</tbody>
</table>

Abbreviations: s/p = status post; LVII = left ventricular hypotrophy.

Hemodynamics

CPR was performed in the usual fashion. Peak pulmonary artery pressures observed varied between 35–60 mm Hg during chest compression. Arterial pressures were almost identical. In one patient, external Doppler flow probe over the right femoral artery confirmed antegrade flow in the femoral artery simultaneous with external chest compression. Effective peak pressures decreased with time, in part because of physician fatigue. One patient had broken ribs due to earlier CPR before the study. No clinical complications from CPR occurred during the study. Postmortem examination was performed on one patient and revealed no evidence of hemopneumothorax or liver laceration.

Echocardiograms

Left-heart Findings

Mitral Valve. During the release phase of CPR, the mitral valve was in midposition, with both leaflets separated. During maximum deep chest compression, the mitral leaflets moved farther apart from one another, toward the left ventricular walls and then rebounded during release toward the midposition. At no time during a compression or release cycle did the mitral leaflets move toward or appose one another. Both the long- and short-axis parasternal views confirmed the lack of apposition of anterior and posterior mitral valve leaflets during the compression cycle in all patients (fig. 1).

Aortic Valve. As would be expected during chest compression, aortic valve leaflets opened widely (fig. 2) during chest compression and moved to the closed position during release. Maximum opening of aortic valve leaflets coincided with the presence of an easily palpable pulse in the carotid and femoral arteries and antegrade femoral artery flow over the femoral artery as documented by Doppler flow probe. Hence, in all cases, simultaneous opening of aortic and mitral valves during maximum chest compression (fig. 3) resulted in the production of either a palpable, audible or flow-confirmed pulse.

Left Ventricle. Conclusions regarding the changes in the size of the left ventricle during external chest compression must be interpreted with caution because true volumes cannot be measured using the two-dimensional echocardiographic technique necessary for this study. Some degree of left- and/or right-heart shape change probably occurs during chest compression. However, we could not see a significant diminution in overall left ventricular chamber size in the two patients in whom endocardial definition was adequate for examination in at least two tomographic planes. Indeed, a planimetered area using the short-axis view in one patient during a compression and release cycle demonstrated little change, with an area of 1.11 units during release and 1.28 units during compression.

![Figure 1. Two-dimensional echocardiogram of the left ventricle, short-axis view, parasternal position. One continuous compression and release cycle is represented. The cycle begins (top left) during release. Compression begins (top middle) and continues to maximum compression (bottom left). Release continues (bottom middle and right). The anterior and posterior mitral valve leaflets (ALMV and PLMV) are seen throughout the compression cycle. The leaflets, indicated by the arrows, remain widely open throughout the compression and release sequence. Arrows indicate the ALMV and PLMV during the cycle. IVS = interventricular septum; LV = left ventricular cavity.](image-url)
Figure 2. Short-axis view, parasternal position, aortic valve. (left) Appearance of aortic valve during release. (right) Aortic valve leaflets (AO) opening during chest compression. The tricuspid valve (TV) is also seen during compression and is moving toward the closed position, although apposition of leaflets is not demonstrated. RVOT = right ventricular outflow tract; RA = right atrium.

Figure 3. Three frames from one release-compression-release sequence during cardiopulmonary resuscitation. The long-axis view of the ventricle and aorta from the parasternal position are shown. (top left) The mitral valve leaflets are in the midposition and aortic valve leaflets (AO) are closed. (top right) During compression, aortic valve leaflets are widely opened. The mitral valve leaflets, indicated by the arrows, remain widely open during compression. (bottom) During complete release, the aortic valve leaflets are reapposed and mitral valve leaflets still in the midposition in the body of the ventricle. RV = right ventricle; LV = left ventricular cavity; ALMV = anterior leaflet mitral valve; PLMV = posterior leaflet mitral valve; LA = left atrium.
Right-heart Findings

The right heart was considerably more difficult to visualize than the left heart during chest compression; it was visualized in only two of the five patients.

Tricuspid Valve. Of the four cardiac valves, the tricuspid valve seemed the most inconstant in its motion during external chest compression and release. The tricuspid leaflets appeared to move toward the closed position, but never completely apposed, even during maximum chest compression. During some chest compressions, the tricuspid valve moved only slightly toward the closed position. Saline contrast injections into the proximal portion of a thermodilution pulmonary artery catheter confirmed the presence of swirling antegrade and retrograde flow across the tricuspid valve during maximum chest compression. In fact, some reflux of contrast material toward the inferior vena cava was identified during chest compression. During release, the tricuspid valve opened in all cases and a brief but rapid acceleration of blood occurred in an antegrade fashion from atria to ventricles and toward the pulmonary outflow tract.

Pulmonic Valve. The pulmonic valve was observed during artificial ventilation alone, during external chest compression and during external chest compression together with mechanical ventilation. During all three of these mechanical states, the pulmonic valve moved to the closed position during either mechanical ventilation (without positive end-expiratory pressure) and during manual external chest compression with and without mechanical ventilation (fig. 4). Complete apposition of the two visualizable pulmonic valve leaflets seemed to occur. Very early valve movement during the onset of chest compression was jerky, but during maximum chest compression the leaflets clearly moved to the closed position. During release and at rest the pulmonic valve leaflets were widely open.

Discussion

The mechanism underlying effective circulatory support is still unclear. The modern concepts, mechanisms and theory of CPR are generally attributed to Kouwenhoven et al., who reported that the circulation could be supported through the rhythmical application of firm pressure to the lower sternum. They hypothesized that forward blood flow during external chest compression occurred primarily as a result of compression of the ventricles between the sternum and spine, with normal competence of the heart valves. Despite the general acceptance of this concept of CPR, other investigators argued that closed-chest compression was successful for other reasons. In 1964, MacKenzie et al. placed catheters and measured cardiac outputs in three patients treated with external chest compression. They demonstrated that right atrial and aortic root pressures were elevated equally during compression and argued that this maneuver caused a generalized increase in intrathoracic pressure. Criley et al. later reported that patients sustaining ventricular fibrillation during coronary angiography could be kept conscious and alert for up to 40 seconds by following instructions to cough every 1–3 seconds. The mean aortic systolic pressure

![Figure 4](http://circ.ahajournals.org/lookup/doi/10.1161/01.CIR.63.6.1420)
was higher during coughing than during external CPR. These observations could be interpreted to mean that a generalized increase in intrathoracic pressure causes systolic aortic pressure and forward blood flow during cardiac arrest.

The first direct evidence for the mechanism of forward blood flow during CPR came from investigators at The Johns Hopkins University, who studied the hemodynamics of CPR in 15 dogs with cardiac arrest. 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 estimated by an esophageal balloon catheter. Rudikoff and colleagues further demonstrated that unequal transmission of intrathoracic pressure to the extrathoracic arterial and venous system resulted from collapse of the great veins at the thoracic outlet as intrathoracic pressures rose. Hence, the collapse of the intrathoracic venous system gave rise to a lower extrathoracic venous pressure and ultimately the arterial–venous pressure gradient necessary for forward flow across peripheral beds. The presence of an anatomic or functional venous valve reduced venous pressure at the thoracic inlet and established an important additional clue to the mechanism of continuous forward blood flow. These authors demonstrated that increasing intrathoracic pressure further by maintaining the lungs fully inflated during chest compression or by tightly binding the abdomen to prevent paradoxical diaphragmatic motion further increased peak aortic pressure and carotid blood flow.

More recently, Niemann and colleagues presented cineangiographic evidence in dogs that ventricular volume did not decrease during closed-chest compression. Additionally, the cineangiograms showed blood flow through both left heart valves during the latter phase of mechanical compression during ventricular fibrillation. They also demonstrated evidence for an anatomic and functional venous valve at the thoracic outlet, which could maintain an intrathoracic–extrathoracic venous pressure gradient. These data seem to support the chest-pump theory of CPR, which hypothesizes a generalized increase in intrathoracic pressure as the primary driving force that moves blood out of the lungs through the left heart and into the periphery due to the intrathoracic–extrathoracic pressure gradient during chest compression.

Clearly, an appreciation of valve motion and chamber size during CPR is fundamental to the understanding of the physiologic mechanism of blood flow. If, as suggested by Kouwenhoven et al., forward blood flow during CPR occurs primarily through cardiac compression, one would expect the left and right ventricles to become smaller and the mitral valve to close and aortic valve to open during "mechanical systole." However, if the primary force moving blood is an increase in intrathoracic pressure and expulsion of blood from the lungs through a left-heart conduit, then aortic and mitral valves should be open simultaneously during chest compression and the left ventricle should remain essentially the same size or possibly even increase in response to the increased blood volume. Two-dimensional echocardiography seemed the ideal tool to demonstrate these valve motions and mechanisms.

Our findings support the chest-pump theory of CPR. In our five patients, aortic and mitral valves opened simultaneously during each external chest compression performed manually in the conventional manner. Indeed, deeper and more prolonged chest compression appear to result in an even wider separation of mitral valve leaflets, suggesting augmentation of forward blood flow through the mitral valve. The findings in the right heart were more difficult to obtain but appear to demonstrate closure of the pulmonic valve during chest compression and opening during release. This latter finding is consistent with the flow of blood from the right heart into the lungs during release and forward blood flow across the pulmonary capillaries into the left heart during compression. Backward blood flow appears to be prevented by the closed or near-closed pulmonic valve during compression. The tricuspid valve appears to remain relatively incompetent (although moving toward the closed position) during chest compression and opens widely during release, further suggesting transient inflow of blood during the release phase of the cycle.

This study thus confirms the concept that forward blood flow during CPR occurs by a mechanism that does not rely on closing of the mitral valve. Rather, the left heart acts simply as a conduit during CPR, and these findings in man are consistent with the theory that blood flow during CPR results from a generalized rise in intrathoracic pressure and not from direct cardiac compression. Even if the correct theory of the mechanism of blood flow during CPR is at odds with some of the concepts that led to the development of standard CPR techniques, the standard techniques are effective, can be performed for several minutes by one person alone and should continue to be used until further studies demonstrate improved survival by a different technique.

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References
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