Physiology of Blood Flow During Cardiopulmonary Resuscitation
A Transesophageal Echocardiographic Study

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Background. There are two competing theories of the mechanism of blood flow during cardiopulmonary resuscitation. The "cardiac pump" theory postulates that blood flows because the heart is squeezed between the sternum and the spine. The "thoracic pump" theory postulates that blood flows from the thorax because intrathoracic pressure exceeds extrathoracic vascular pressure and that flow is restricted to the venous-to-arterial direction because of venous valves that prevent retrograde flow at the thoracic inlet. To determine which mechanism is operative during actual cardiopulmonary resuscitation, 20 patients were imaged with transesophageal echocardiography during resuscitation.

Methods and Results. Transesophageal two-dimensional and pulse Doppler echocardiography was begun within 7 minutes of initiation of cardiopulmonary resuscitation. In the 18 patients who could be analyzed, the mitral valve opened during the release phase (diastole) and closed during the compression phase (systole) of cardiopulmonary resuscitation. Mitral velocity-time integral measured 8±3 cm during diastole. There was compression of right and left ventricular cavities with significant reduction in measured left ventricular volume during cardiopulmonary resuscitation. In five patients, mitral regurgitation was present.

Conclusions. Transesophageal echocardiography performed during actual cardiopulmonary resuscitation showing mitral valve opening during cardiac release, reduction of ventricular cavity size with compression, and atrioventricular regurgitation support the cardiac pump theory of cardiopulmonary resuscitation. This study demonstrates the feasibility and usefulness of transesophageal echocardiography during cardiopulmonary resuscitation. (Circulation 1993;88:534-542)

KEY WORDS • circulation • echocardiography • cardiopulmonary resuscitation • flow • physiology

Although modern closed-chest methods of cardiopulmonary resuscitation were introduced by Kouwenhoven and colleagues more than 30 years ago,1 the mechanism of blood flow during cardiopulmonary resuscitation remains controversial. Both cardiac pump and thoracic pump models have been proposed. The "cardiac pump" theory postulates that forward blood flow during cardiopulmonary resuscitation is due to compression of the heart between the sternum and the spine. This theory requires that the atrioventricular valves be closed during cardiac compression (systole). The "thoracic pump" theory postulates that external chest compression increases intrathoracic pressure, which forces blood to flow from the thoracic to the systemic circulation, with the heart acting as a passive conduit without having a pump function. This theory requires that the atrioventricular valves be open during cardiac compression.2-4 Because valvular position and the ventricles can be imaged by echocardiography, these theories can be tested by study...

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Francisco on a compassionate-use basis as informed consent was not possible. San Francisco has an advanced life support paramedic system that responds to all cardiac arrests under physician direction; patients are resuscitated according to standard advanced cardiac life support guidelines. All adult patients in a nontraumatic cardiopulmonary arrest who were transported to the emergency department as well as those in the coronary care unit at the University of California San Francisco Moffitt Hospital and those transported to the emergency department at San Francisco General Hospital between June and October 1991 were eligible for enrollment in the study. Patients had to be in full cardiopulmonary arrest for entry in the study. They were excluded if there was known or suspected esophageal injury or disease.

In the emergency department or coronary care unit, an arrest team trained according to advanced cardiac life support guidelines9,10 responded to all arrests and used manual closed-chest cardiopulmonary resuscitation. Standard resuscitation efforts were continued in all patients during transesophageal echocardiographic imaging, including during administration of drugs per American Heart Association guidelines and during cardiopulmonary resuscitation. All patients in this study had been endotracheally intubated before esophageal intubation for transesophageal echocardiography. An ultrasound machine with a 5-MHz monoplane transesophageal echocardiographic probe (Sono 500 ultrasound system and 21362A ultrasound transducer; Hewlett-Packard, Andover, Mass) was dedicated to each hospital’s emergency department to facilitate rapid imaging. One of the investigators (R.F.R. or K.J.T.), who was trained in the performance of transesophageal echocardiography according to the guidelines of the American Society of Echocardiography,11 was notified by telephone or beeper on a 24-hour basis before arrival of the arrest victim in the emergency department.

Cardiopulmonary Resuscitation Protocol

Resuscitation was performed according to advanced cardiac life support guidelines. Compression rate was 80 min-1 (metronome synchronized) with a 50% duty cycle.12 Compression depth was 4 to 5 cm. Cardiopulmonary resuscitation was performed by a team of three or four people, who alternated doing cardiac compression. Patients continued to receive epinephrine and other routine drugs as indicated by advanced cardiac life support guidelines during imaging.

Ten patients in this study were also enrolled in a study of new methods of resuscitation. These patients had femoral line monitoring of arterial pressures and end-tidal CO2 measurements every 30 seconds, as well as arterial blood gas measurements, as has been described.13 Arterial pressure was recorded using an 18F right femoral artery fluid-filled catheter with a Statham-Gould pressure P23 transducer (Gould, Inc, Oxnard, Calif). End-tidal CO2 was recorded with a Nellcor P1000 Capnometer (Nellcor Inc, Hayward, Calif). Transesophageal echocardiographic imaging was started before initiation of the resuscitation study protocol. Five of these 10 patients also had chest compression performed using a programmable cardiopulmonary resuscitator (Thumper; 1005; Michigan Instrument, Grand Rapids, Mich), which is routinely used to assist with compression in the Moffitt Hospital Emergency Department. This device delivered 80 compressions per minute with a 50% duty cycle and a force of compression of 70 lb, which achieves a depth of compression similar to that of manual compression.

Echocardiographic Protocol

Blind esophageal intubation was attempted in all patients followed by laryngoscopic esophageal intubation, if necessary. The following echocardiographic views were obtained: (1) basal short-axis scan to visualize the aortic root and valve, coronary arteries, pulmonary venous inlet, and atrial appendages; (2) apical four-chamber scan to visualize the mitral valve apparatus, left and right ventricles, and left ventricular outflow tract; (3) transgastric short-axis scan to visualize the left and right ventricles; and (4) posterior mediastinal scan to visualize the thoracic aorta, aortic arch, and pulmonary arteries. The probe was maintained in the four-chamber view during cardiopulmonary resuscitation for optimal monitoring of aortoventricular valve motion and flows. Doppler interrogation of the mitral valve was performed at the tips of the mitral leaflet in the pulse wave and color flow modes from the apical four-chamber view, using a sweep speed of 75 mm/s. All studies were recorded continuously and stored on super VHS videotape; random-number sequences for each patient and method of resuscitation were used to code videotapes so that data analysis could be blindly performed at a later time. During cardiac compression, the microphone on the ultrasound machine was used to narrate the timing of compression and release.

All tapes were carefully reviewed by two cardiologists who were expert in transesophageal echocardiography and were blinded to clinical scenario or method of resuscitation. Left and right ventricular compression and cavity areas, valvular motion, and flow patterns were recorded. The appearance of spontaneous echocardiographic contrast was monitored and was defined as smokelike clouds of echoes curling up slowly in a circular or spiral shape.14 These echoes can be distinguished from white noise artifact by their swirling and more specific appearance. Compression and decompression (release) phases of chest massage were determined by echocardiographic visualization and confirmed by using the online voice narration. Changes in left ventricular and right ventricular internal dimensions during compression and decompression were measured from the four-chamber view using a commercially available software analysis package (Freeland Systems, Broomfield, Colo). The volume of the left ventricle at end compression and end decompression in the four-chamber view was measured by two-dimensional planimetry and calculated by the area-length method.15,16 The difference between these two volumes is expressed as the stroke volume. The positions of the mitral, tricuspid, and aortic valves during compression-and-decompression phases were noted. After videotape review of all Doppler signals, quantitative measurements of the forward mitral velocity time integral (stroke distance) were done by integrating the forward Doppler velocity profile over time using the Freeland system. Stroke distance is a measure of the distance an
**Table 1. Clinical Characteristics of Patients Studied by Transesophageal Echocardiography During Cardiopulmonary Resuscitation**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Medical history</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 M.W.</td>
<td>67</td>
<td>Female</td>
<td>Unknown</td>
</tr>
<tr>
<td>2. L.H.</td>
<td>94</td>
<td>Male</td>
<td>MI, dementia</td>
</tr>
<tr>
<td>3 J.C.</td>
<td>75</td>
<td>Male</td>
<td>Diabetic ketoacidosis</td>
</tr>
<tr>
<td>4 C.W.</td>
<td>37</td>
<td>Male</td>
<td>Heart failure, ESRD S/P AVR, MVR, drug abuse</td>
</tr>
<tr>
<td>5 C.E.F.</td>
<td>37</td>
<td>Male</td>
<td>Seizure</td>
</tr>
<tr>
<td>6 R.B.</td>
<td>88</td>
<td>Male</td>
<td>Supraventricular tachycardia</td>
</tr>
<tr>
<td>7 J.D.</td>
<td>72</td>
<td>Male</td>
<td>Coma</td>
</tr>
<tr>
<td>8 R.R.</td>
<td>67</td>
<td>Male</td>
<td>Cardiac arrest</td>
</tr>
<tr>
<td>9 S.B.</td>
<td>30</td>
<td>Female</td>
<td>Drug overdose</td>
</tr>
<tr>
<td>10 A.S.</td>
<td>59</td>
<td>Male</td>
<td>Cardiomyopathy, recent digoxin/theophylline toxicity</td>
</tr>
<tr>
<td>11 K.S.</td>
<td>32</td>
<td>Male</td>
<td>No past medical history</td>
</tr>
<tr>
<td>12 B.J.</td>
<td>48</td>
<td>Female</td>
<td>S/P CABG, ESRD pulmonary disease</td>
</tr>
<tr>
<td>13 J.P.</td>
<td>76</td>
<td>Male</td>
<td>Unknown</td>
</tr>
<tr>
<td>14 P.J.</td>
<td>57</td>
<td>Female</td>
<td>Unknown</td>
</tr>
<tr>
<td>15 E.P.</td>
<td>70</td>
<td>Female</td>
<td>Unknown</td>
</tr>
<tr>
<td>16 L.E.</td>
<td>80</td>
<td>Male</td>
<td>Heart failure, arrhythmia</td>
</tr>
<tr>
<td>17 J.B.</td>
<td>80</td>
<td>Male</td>
<td>Cerebrovascular accident</td>
</tr>
<tr>
<td>18 C.C.</td>
<td>73</td>
<td>Male</td>
<td>Pulmonary disease</td>
</tr>
<tr>
<td>19 S.C.</td>
<td>21</td>
<td>Female</td>
<td>No past medical history</td>
</tr>
<tr>
<td>20 R.L.</td>
<td>70</td>
<td>Female</td>
<td>Aortic stenosis</td>
</tr>
</tbody>
</table>

MI indicates myocardial infarction; ESRD, end-stage renal disease; AVR, aortic valve replacement; MVR, mitral valve replacement; and CABG, coronary artery bypass grafting.

average red blood cell travels during each compression cycle.17-19 Doppler signals were considered technically adequate for tracing if they had complete envelopes with well-defined borders. After calibration, at least three mitral inflow signals were measured and averaged to determine stroke distance during left ventricular filling. The extent and rate of appearance and disappearance of spontaneous echocardiographic contrast also were noted for each patient.

**Statistical Analysis**

Continuous variables are expressed as mean±1 SD. Student's t test was used to compare the significance of differences between groups for continuous variables. Significance was assumed if P<.05.

**Results**

Twenty cardiopulmonary arrest patients (15 men and 5 women; age, 62±20 years) were studied by transesophageal echocardiography. They had a wide variety of diagnoses (Table 1). In 5 of 20 patients (25%), the arrest was witnessed. From collapse to start of cardiopulmonary resuscitation was estimated to be 12.6±8.0 minutes. In all cases, echocardiographic imaging was begun within 7 minutes of initiation of cardiopulmonary resuscitation in the hospital. The initial rhythm at the arrest was ventricular fibrillation in 5, asystole in 4, ventricular tachycardia in 3, electromechanical dissociation in 2, bradycardia in 2, and not recorded in 4 patients. Five patients were successfully resuscitated, and two patients lived until hospital discharge. Blind esophageal intubation was accomplished in 15 of 20 patients. Cardiopulmonary resuscitation efforts lasted from 25 to 70 minutes.

Two studies could not be analyzed because of technical reasons—one patient was not imaged during standard cardiopulmonary resuscitation, and one patient had two mechanical prosthetic valves. In all of the remaining 18 studies, the mitral valve closed during compression and opened only during the release phase of cardiopulmonary resuscitation (Fig 1). There was some variability observed in the degree of mitral valve opening. The valve appeared to open more fully with more forceful compression as determined by the degree of ventricular displacement that was observed. Group mean mitral stroke distance measured by pulse-wave Doppler was 8±3 cm. The mitral stroke distance appeared to correlate with extent of mitral valve opening, with higher flows in valves that opened more fully. In 4 patients, we were able to simultaneously image the mitral and tricuspid valves and found that right heart valvular dynamics paralleled left heart valvular dynamics. In 10 patients, we withdrew the probe from the four-chamber view during monitoring to visualize the aortic valve, and, we observed opening of the aortic valve during compression (Fig 2). In all patients, reduction in the area of the right and left ventricular cavities was observed during the compression phase of cardiopulmonary resuscitation. Minimum left ventricle internal diameter measured from the four-chamber view was 2.48±0.5 cm during the compression phase and 1.66±0.4 cm during the release phase (P<.001). Displacement of the right ventricle along the long axis from its position in the release phase to the compression phase of cardiopulmonary resuscitation was 3.6±1.5 cm (n=8). Right ventricular internal diameter could not be measured because the ventricle was oblique to any measurement plane. Adequate views of the left ventricle for measurement of single-plane area-length volumes were obtained in 5 patients. The end-compression volume was 49.7±9.3 mL and the end-release volume was 69.4±10.8 mL (P<.05) with a stroke volume of 17.6±5.2 mL measured from the four-chamber view (Table 2).

In 5 patients, we noted mitral regurgitation, and 2 patients had tricuspid regurgitation of varying severity during the compression phase of resuscitation. This indicates a positive ventriculoatrial pressure gradient (Fig 3).

We also noted the appearance of spontaneous echocardiographic contrast in all four cardiac chambers in patients with times from collapse to initiation of cardiopulmonary resuscitation as short as 5 minutes and occasionally appearing during pauses in cardiopulmonary resuscitation as brief as 30 seconds. Spontaneous contrast appeared to clear immediately with more forceful (greater displacement measured on echocardiography) compressions and clear more slowly with less forceful compression (Fig 4).

As in other studies of human cardiopulmonary resuscitation, we were not able to directly measure force of compression for standard cardiopulmonary resuscitation in this study. However, in 5 patients, chest compression also was performed using a programmable cardiopulmonary resuscitator, with a
known force of compression (70 lb), at the same rate of compression and duty cycle as standard cardiopulmonary resuscitation. Flow data, pressures, and valvular motion during mechanical cardiopulmonary resuscitation were similar to data from standard cardiopulmonary resuscitation (Table 3), so we can estimate that force of compression was similar with each technique.
In the subset of 10 patients with capnometry data, end-tidal CO₂ was 4.7±3.8 mm Hg (range, 0 to 11 mm Hg), which is typical of previous results in the cardiac arrest population.²⁰,²¹ Systolic and diastolic arterial pressures in this subset, measured by femoral arterial line, were 52.5±14.0 and 17.6±9.1 mm Hg, respectively. There was no relation among end-tidal CO₂, arterial blood pressure, valvular motion, chamber size, and survival.

**Discussion**

There have been several excellent experimental studies concerning the mechanism of blood flow in cardiopulmonary resuscitation since it was first described more than 30 years ago. According to the original description of the technique, Kouwenhove and colleagues believed that blood flow occurred because of compression of the heart between the sternum and the vertebral bodies and the pericardium. “Pressure on the sternum compresses the heart between it and the spine, forcing out blood. Relaxation of the pressure allows the heart to fill.”¹ Thus, chest compression squeezed the left ventricle, causing the aortic valve to open and forward blood flow to leave the heart. The mitral valve would close during compression to prevent retrograde flow. The model of Kouwenhoven and colleagues has come to be known as the cardiac pump theory of cardiopulmonary resuscitation.

This was the generally accepted theory until the late 1970s. In 1976, Criley and colleagues described “cough cardiopulmonary resuscitation” based on observations in the cardiac catheterization laboratory that repetitive coughing alone could maintain the conscious state for as long as 40 seconds during ventricular fibrillation without chest compression. They postulated that forward flow occurred secondary to increased intrathoracic pressure during coughing. Other investigators had noted previously that there were equal increases in arterial and venous pressures with chest compression.²³-²⁵ These observations, along with observations that emphysematous patients with increased anteroposterior diameter and relatively small hearts, which would make direct cardiac compression difficult, are still able to be resuscitated and that patients with a flail sternum, which would readily allow cardiac compression, did not have an increase in blood pressure unless they had a belt placed around the thoracic cage,² sparked renewed investigation into the mechanism of blood flow during cardiopulmonary resuscitation. In 1980, experimental research in dogs²⁶ and in humans suggested a different mechanism for flow during cardiopulmonary resuscitation. Rudikoff and colleagues² found that during chest compression, there were equal pressures in the left ventricle, aorta, right atrium, pulmonary artery, and esophagus and that these also were equal to intrathoracic pressure. Increasing intrathoracic pressure by maintaining the lungs fully inflated during compression or by abdominal binding to prevent paradoxical dia-

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**TABLE 2. Transesophageal Echocardiographic Quantitation of Left Ventricular Volumes (Area–Length Method)**

<table>
<thead>
<tr>
<th>Patient</th>
<th>ECV (mL)</th>
<th>EDV (mL)</th>
<th>SV (mL)</th>
<th>VTI (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 J.C.</td>
<td>55.0</td>
<td>68.2</td>
<td>13.2</td>
<td>5.2</td>
</tr>
<tr>
<td>2 C.W.</td>
<td>34.2</td>
<td>53.0</td>
<td>18.8</td>
<td>5.3</td>
</tr>
<tr>
<td>3 S.B.</td>
<td>57.3</td>
<td>83.4</td>
<td>25.9</td>
<td>9.5</td>
</tr>
<tr>
<td>4 K.S.</td>
<td>54.0</td>
<td>71.2</td>
<td>17.2</td>
<td>9.5</td>
</tr>
<tr>
<td>5 B.J.</td>
<td>48.2</td>
<td>71.4</td>
<td>23.2</td>
<td>9.6</td>
</tr>
<tr>
<td>Mean</td>
<td>49.7</td>
<td>69.4</td>
<td>19.7</td>
<td>7.8</td>
</tr>
</tbody>
</table>

ECV indicates end-compression volume; EDV, end-decompression volume; SV, stroke volume; and VTI, velocity-time integral.

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**FIG 2. Transesophageal echo of long-axis view of left ventricle and left ventricular outflow tract during cardiopulmonary resuscitation showing open aortic valve during compression.**
Concurrently, two transthoracic echocardiographic studies of a combined total of nine patients during cardiopulmonary resuscitation reported that left ventricular dimensions did not change appreciably and that the mitral valve remained open during the entire cycle, whereas the aortic valve opened during the compression phase and closed during the release phase. These findings suggested that blood flow depended on an increase in intrathoracic pressure and that the forward direction of blood flow is favored by venous valves at the thoracic outlet that prevent retrograde flow. These venous valves were reported to be visualized angiographically. These investigators also concluded that the heart is a passive conduit for blood flow during cardiopulmonary resuscitation. This is the thoracic pump theory.

Further experimental research showed that the mechanism of blood flow during cardiopulmonary resuscitation actually depended on the momentum of compression, and the thoracic pump model was questioned. In a highly instrumented dog model in studies at Duke University in 1984, compression force and rate were varied during cardiopulmonary resuscitation. Using high-fidelity micromanometer catheters to measure intrathoracic pressure and coronary blood flow, the researchers found that increased compression force did not increase stroke volume and concluded that direct cardiac compression was the major determinant of stroke volume during cardiopulmonary resuscitation. In 1987, the Duke University investigators used transthoracic and transthoracic echocardiography in nine dogs to investigate mitral valve motion during cardiopulmonary resuscitation. They found that the mitral valve closed during compression phase and reopened during release phase unless a low-impulse compression technique was used. This work offered new evidence for direct cardiac compression as the mechanism of blood flow and suggested that the momentum of compression would influence mitral valve motion. The Duke University group performed additional investigations to show that increasing the compression rate to 120 min⁻¹ significantly improves survival in dogs when standard manual cardiopulmonary resuscitation is used compared with a compression rate of 60 min⁻¹. Support for the idea that the mechanism of cardiopulmonary resuscitation also depends on the force of compression came from later experimental work in pigs. In a model that varied force of compression, it was found that when compressive force was 200 N (44 lb), the mitral valve closed in 16% of cardiac cycles, and that when force was increased to 500 N (110 lb), the mitral valve closed in 95% of cardiac cycles. Increased rates of mitral valve closure were associated with increased diastolic myocardial perfusion pressures, systolic cerebral pressures, and cardiac output. These findings are also consistent with the cardiac compression model of cardiopulmonary resuscitation.
Our results confirm the findings of two previous reports of a total of 3 patients who had transepophageal echocardiographic studies during cardiopulmonary resuscitation. In another recent study of 17 patients by Porter and colleagues in which transepophageal echocardiography was used during cardiopulmonary resuscitation, the mitral valve during compression closed in 12 patients and opened wider in 5 patients. Valve behavior, however, did not correlate with left ventricular fractional shortening. This study was done using a mechanical piston device at a force of compression higher than that recommended by the manufacturer to comply with American Heart Association resuscitation guidelines. These investigators found no distinguishing factors between these two groups, but they did not attempt comparisons of rhythm, time from arrest to echocardiographic study, clinical characteristics, or outcome. We speculate that our findings did not show the variability of valve behavior seen in the study of Porter and colleagues because mean arterial pressures and mitral forward flow in patients receiving Thumper cardiopulmonary resuscitation are lower and have a wider range compared with standard manual cardiopulmonary resuscitation.

Our protocol also differed from the reports of Higano and colleagues, Kuhn and Juchems, and Porter and colleagues in several respects. First, cardiopulmonary resuscitation methodology and hemodynamics were carefully monitored in our study. We measured end-tidal CO2, which has been shown to correlate with coronary perfusion pressure and predict the likelihood of successful resuscitation. End-tidal CO2 has a high linear correlation to cardiac output. In our study, these measurements show that cardiopulmonary resuscitation was being performed effectively and serve as an indirect measure of coronary perfusion pressure. We also invasively monitored arterial pressure, which was not reported previously.

Second, other studies had long or unrecorded intervals between cardiac arrest and echocardiography; the expected increase in left ventricular stiffness may affect hemodynamic findings. In a report of two patients during cardiopulmonary resuscitation who had a time interval from arrest to transepophageal echocardiography of 5 minutes, Higano and colleagues had findings similar to ours. Desmukh and colleagues have shown that valve motion diminishes after 5 minutes of cardiac arrest in minipigs. Our data are consistent with this work, in which cardiopulmonary resuscitation was started immediately and showed a 24% reduction in left ventricular area during compression.

In addition, although we closely monitored resuscitative efforts, this study was conducted during actual

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**TABLE 3. Arterial Pressure and Mitral Stroke Distance in Standard versus Thumper Cardiopulmonary Resuscitation**

<table>
<thead>
<tr>
<th></th>
<th>Mean SAP (mm Hg)</th>
<th>Mean DAP (mm Hg)</th>
<th>Mean MAP (mm Hg)</th>
<th>Mean VTI (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standard (n=8)</td>
<td>56.23</td>
<td>18.79</td>
<td>31.18</td>
<td>9.17</td>
</tr>
<tr>
<td>Thumper (n=5)</td>
<td>39.27</td>
<td>13.97</td>
<td>21.13</td>
<td>8.4</td>
</tr>
<tr>
<td><em>P</em></td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

SAP indicates systolic arterial pressure; DAP, diastolic arterial pressure; MAP, mean arterial pressure; and VTI, velocity-time integral (stroke distance).
"real life" cardiopulmonary resuscitation as opposed to a laboratory environment and thus better reflects actual cardiopulmonary resuscitation conditions. The use of the dog model for human cardiopulmonary resuscitation has been criticized because of differences in chest configuration between dogs and humans, which make comparison difficult. Dogs also lack a mediastinal web, which limits stability of the heart necessary for direct cardiac compression.

Finally, we used transesophageal echocardiography, whereas most previous studies used transthoracic echocardiography. There are inherent technical limitations in obtaining images by transthoracic echocardiography during actual cardiac compression. The improved resolution of transesophageal echocardiography facilitates cardiac measurements. Another advantage of transesophageal echocardiography over transthoracic echocardiography is transducer stability; because transesophageal echocardiography does not interfere with cardiac compression, it can be started immediately and performed continuously without interrupting cardiopulmonary resuscitation. This may explain the difference in this study and previous transthoracic echocardiographic studies that reported changes in left ventricular internal diameter.

Our findings of decreased right ventricular and left ventricular cavity size and left ventricular volume with compression, mitral valve closure with compression and opening with release, aortic valve opening with compression, and atrioventricular regurgitation during compression systole support cardiac compression as the predominant mechanism for blood flow during cardiopulmonary resuscitation. Although Halperin and colleagues found that in a canine model during certain situations mitral valve closure can occur with cyclic elevation of intrathoracic pressure alone, our concomitant findings detailed above and the observation that forward transmitial flow is detected only during release favor the cardiac pump mechanism.

A limitation of the present study is the lack of intrathoracic pressure monitoring. Future studies are planned to directly measure intrathoracic pressure. In addition, as stated earlier, we did not measure coronary perfusion pressure, although we were able to use endtidal CO₂ as an indicator in a subset of patients.

We also were not able to directly measure or vary the force of compression used in this study. During mechanical compression, the force of compression was held constant, and during manual compression, we attempted to keep it constant by careful monitoring with advanced cardiac life support—trained personnel. Because the depth of chest compression and ventricular compression were similar during manual and standard compression, as measured by echocardiography, we estimated that force of compression using standard and mechanical cardiopulmonary resuscitation, which was set at 70 lb, was similar. As this study sought to evaluate the physiology of blood flow during actual cardiopulmonary resuscitation, we used the Thumper device according to American Heart Association guidelines and did not attempt to vary the force of compression. This comparison has been made in previous work, which has shown that a lower force of compression favors the thoracic pump theory, whereas a higher force favors the cardiac pump theory.

The status of the airway may be an important variable in determining which mechanism of cardiopulmonary resuscitation is operative. In this study, the airway tended to be open, whereas in past studies that supported the thoracic pump theory in humans, the lungs were fully inflated. It will be most important to measure intrathoracic pressure in future studies to determine the significance of this variable. Current evidence suggests that both cardiac compression and thoracic compression may operate at different times during cardiopulmonary resuscitation, and the mechanism that is operative depends on the force and rate of compression, body habitus, airway pressure, and, perhaps, rhythm. The type of thoracic compression used also influences the mechanism of blood flow, and, undoubtedly, cough cardiopulmonary resuscitation and vest cardiopulmonary resuscitation would favor a thoracic pump mechanism.

The mechanism of blood flow during standard methods of cardiopulmonary resuscitation in humans has a direct impact on our current search for methods to improve survival after cardiopulmonary resuscitation. The use of mitral valvular opening and measurement of mitral flow by transesophageal echocardiography Doppler may prove a valuable way to monitor and improve resuscitative techniques during cardiopulmonary resuscitation. We believe transesophageal echocardiographic measurement of cardiac flow should be the standard for evaluation of proposed new methods of resuscitation. Further investigation is warranted to determine if improved cardiac flow is correlated to improved cerebral perfusion and to survival.

Conclusions

We have shown that transesophageal echocardiography can be performed during ongoing cardiopulmonary resuscitation to directly visualize the mechanism and blood flow during resuscitative efforts without interruption of resuscitation. Data derived from transesophageal echocardiography support the cardiac pump theory of cardiopulmonary resuscitation. Further studies are needed to determine the relation of valvular motion and flows to cerebral perfusion and to survival.

References


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