Hemodynamics in Humans During Conventional and Experimental Methods of Cardiopulmonary Resuscitation

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High-fidelity hemodynamic recordings of aortic and right atrial pressures and the coronary perfusion gradient (the difference between aortic and atrial pressure) were made in nine patients during cardiopulmonary resuscitation (CPR). Findings during conventional manual CPR were compared with those during high-impulse CPR (rate, 120 cycles/min with a shorter compression:relaxation ratio) as well as during pneumatic vest CPR with and without simultaneous ventilation and abdominal binding. Aortic peak pressure during conventional CPR averaged 61±29 mm Hg but varied widely (range, 39–126 mm Hg) among patients. Although the magnitude of improvement was modest, the high-impulse method was the only technique tested that significantly elevated both aortic peak pressure and the coronary perfusion gradient during cardiac arrest. During conventional CPR, aortic pressure rose from 61±29 to 80±39 mm Hg during high-impulse CPR, and the gradient rose from 9±11 to 14±15 mm Hg, respectively; p<0.01. The pneumatic vest method significantly improved peak aortic pressure but not the coronary perfusion gradient. Simultaneous ventilation and chest compression created high end-expiratory pressures and lowered the coronary perfusion gradient. Abdominal binding had no significant hemodynamic effects. This evaluation of experimental resuscitation methods in humans shows that the high-impulse chest compression method augments aortic pressure over levels achieved during conventional CPR methods; however, the improvement in pressure is modest and may not be clinically important. Simultaneous ventilation as well as abdominal binding during CPR were associated with no benefit; in fact, simultaneous ventilation appears to adversely affect cardiac perfusion and, therefore, should not be used during clinical resuscitation. (Circulation 1988;78:630–639)

Hemodynamic recordings during cardiopulmonary resuscitation (CPR) have been reported for only a few human subjects.1–12 Most recordings have been made with either peripheral arterial lines or fluid-filled transducer systems, conditions that make interpretation of the results difficult. There are known differences between peripheral and central vascular pressures; also, the inherent limitations of fluid-filled catheter systems preclude accurate pressure measurements during the rapid phase shifts associated with chest compression and relaxation. Experimental CPR studies in animals have shown that aortic and right atrial pressures and the instantaneous pressure differences are directly related to myocardial and cerebral blood flow.13,14 The magnitude of the coronary perfusion gradient (aortic minus right atrial pressure) has also been shown in experimental studies to accurately predict the likelihood of survival from attempted resuscitation.14,15

The hemodynamic findings associated with alternative methods of chest compression in animals are provocative, showing that both aortic pressure and flow can be augmented substantially over the low levels obtained during conventional CPR. These alternative techniques include simultaneous ventilation and chest compression, abdominal binding, and “high-impulse” manual chest compression.8,16–22 The relevance of these experimental findings to the clinical setting is uncertain. There are major differences in chest and abdominal anatomy among species; in addition, the cause of arrest (electrical fibrillation), use of healthy animals, and...
general anesthetic agents are quite different from conditions present in patients with coronary heart disease who develop cardiac arrest. Any one of these factors might influence the hemodynamic results.23

In this prospective study, we evaluated the hemodynamics of CPR in humans by making high-fidelity recordings of aortic and right atrial pressures during conventional and several other investigational CPR methods. We compared the hemodynamic findings during conventional CPR, during high-impulse CPR, and when a pneumatic vest was used to compress the chest. Last, we determined what, if any, influence simultaneous ventilation and abdominal binding had on intravascular pressures during CPR.

Patients and Methods

Any patient admitted to Harborview Medical Center after out-of-hospital cardiac arrest who was unconscious and, therefore, likely to develop a recurrent episode of cardiac arrest was considered for the study. The patient's family gave informed consent for placement of micromanometer catheters and for measurement of hemodynamics during experimental methods of CPR after death. Patients with recent chest or abdominal trauma or surgery, with bleeding diathesis, or with ascites were excluded. After informed consent, 8F introducer sheaths (USCI, Billerica, Massachusetts) were inserted in the right femoral artery and vein. After calibration against mercury manometers, 8F Millar manometer-tipped pigtail catheters (Millar Micro-Tip Catheter Pressure Transducer Model SPC 485 A, Millar Instruments, Houston, Texas) were advanced with fluoroscopic guidance into the right atrium and thoracic aorta. Indwelling catheters were continuously irrigated with heparinized saline, and patients were systemically anticoagulated. Eight pressure amplifiers (Gould Electronics, Cleveland, Ohio) were used to record pressures on both a strip-chart recorder (MFE Corporation, Salem, New Hampshire) and calibrated magnetic tape (Hewlett-Packard, Waltham, Massachusetts).

After cardiac arrest occurred and the patient was declared dead, the following protocol was performed. In eight of the nine patients, cardiac arrest occurred several days after the index resuscitation and in the setting of irreversible brain injury. The patients' families had requested no resuscitative efforts be applied but consented to these investigational studies of CPR hemodynamics after death. An American Heart Association-certified rescuer applied conventional CPR at a compression rate of 60 compressions/min.24 The timing of compression and relaxation was aided by use of an audio tape of 1-second tones. Enough force was applied to produce 1.5–2 in. of sternal displacement to comply with AHA guidelines. A transducer was interposed between the investigator's hand and the patient's chest to measure applied force and the proportional period of compression (duty cycle). One ventilation was given through an endotracheal tube after every five compressions, and conventional CPR was thus continued for 2 minutes.

After conventional CPR, alternative CPR methods were then used. The methods were sequenced in random order to prevent any bias that might favor a method applied early during the protocol, assuming there might be a progressive decrease in vascular tone over time and thus progressively lower intravascular pressures. High-impulse CPR was performed by manual compression of the chest at a rate of 120 beats/min. Again, an audio tape recording and the force transducer were used to ensure appropriate compression rate and to verify applied force and duration of the compression-relaxation periods. For simplicity, these are referred to as systolic and diastolic periods. Again, one ventilation was delivered every 5 seconds.

Pneumatic vest CPR was performed with a custom-designed vest (Physio-Control Corporation, Redmond, Washington). The pneumatic vest was coupled to a special controller and ventilator (Physio-Control) that permitted adjustments in delivered tidal volume and accurate control of both vent and abdominal inflation pressures. Vest, abdominal binder, and airway pressures were continuously recorded along with intravascular pressures. The effect of increasing the pressure in the vest was determined by recording the resulting hemodynamics during inflations to pressure of, first, 200 mm Hg pressure; then, 250 mm Hg; and, last, 300 mm Hg. A rate of 72 inflations/min was used, and the inflation time equaled the deflation time (duty cycle, 50%). A pause in compressions every 5 seconds for ventilation resulted in 60 chest compressions/min. Compressed air was used to fill the vest. Inflation and deflation times each approximated 200 msec. Ventilation for all CPR methods was done with 100% oxygen. Pressures were recorded for 2 minutes after each step in the protocol.

The effect of simultaneous ventilation and chest compression was measured by inflating the vest to 250 mm Hg at a rate of 72 beats/min and delivering simultaneous ventilation with a tidal volume of 15 ml/kg. The airway exhaust valve on the controller closed 40 msec before inflation of the vest. Airway inflation and exhalation thus occurred simultaneously with vest inflation and exhaust. Airway pressure was not allowed to exceed 100 mm Hg by means of a relief valve. Abdominal binding was accomplished by inflating the chamber of a 6-in. wide binder to 100 mm Hg simultaneous with each vest inflation. The superior edge of the binder fastened to the inferior edge of the vest with a zipper; the inflation chambers were approximately 3–4 cm apart.

Conventional CPR was done for 2 minutes between each alternative method of CPR as well as at the conclusion of the protocol to provide reference values throughout the study. The entire protocol required about 20 minutes to complete.

In four patients, intrathoracic airway pressure was also measured during simultaneous ventilation.
CPR with special endotracheal tubes with multiple lumens (Hi-Lo Jet Tracheal Tube, Mallinckrodt, Argyl, New York). During these latter studies, we examined the effect of reducing the rate of CPR to 30 cycles/min and of increasing the time of exhalation by opening the airway exhaust during the last half of the period of chest compression to better understand airway pressure during simultaneous ventilation CPR.

Arterial blood samples were obtained for measurement of blood gases before and after each CPR method. No sodium bicarbonate was administered. All patients received a 1-mg bolus of epinephrine into the right atrium 1 minute before beginning the protocol and then a continuous central infusion of epinephrine (6 mcg/kg/min) throughout the CPR protocol. Previous studies in 20–30-kg dogs have shown that this dose increases intra-arterial pressure and prevents arterial collapse during alternative methods of CPR.  

Within the next 24 hours, autopsy examinations were done with particular attention directed toward any evidence of traumatic visceral injuries that might have resulted from the vest and abdominal binder, such as evidence of pneumothorax or traumatic injury to the liver, kidney, or spleen.

Continuous variables were compared with the Student's *t* test, paired when appropriate, and one-way analysis of variance. Results during each of the three alternative methods (high-impulse, vest with simultaneous ventilation, and vest with 5:1 ventilation) were compared with pressures during conventional CPR with the paired *t* test. Because multiple comparisons were done, the stated *p* values may overestimate the significance of differences—a most conservative estimate would be to multiply the stated *p* value by 3 (Bonferroni adjustment). The true *p* value lies between the stated and adjusted values. Multivariate analysis was done with linear and logistic regression techniques. The difference in instantaneous aortic and right atrial pressures in mid-diastole was measured for 10 consecutive beats and averaged to determine coronary perfusion gradient. The duty cycle was calculated from the midpoints of the rise and fall of applied sternal force and expressed as a ratio of the compression and relaxation periods.

**Results**

**Patients**

The CPR protocol was accomplished in nine patients; seven men and two women whose average...
Hemodynamics Recorded During Four Methods of Cardiopulmonary Resuscitation in Humans

<table>
<thead>
<tr>
<th>Patient</th>
<th>Duration of arrest (min)</th>
<th>Force (lb)</th>
<th>Duty cycle (proportion)</th>
<th>Aortic pressure (mm Hg)</th>
<th>Right atrial pressure (mm Hg)</th>
<th>Coronary perfusion gradient (mm Hg)</th>
<th>Force (lb)</th>
<th>Duty cycle (proportion)</th>
<th>Aortic pressure (mm Hg)</th>
<th>Right atrial pressure (mm Hg)</th>
<th>Coronary perfusion gradient (mm Hg)</th>
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<td>63/6</td>
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<td>80/6</td>
<td>17</td>
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<td>66</td>
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<td>39/-1</td>
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<td>0.31</td>
<td>95/15</td>
<td>95/7</td>
<td>8</td>
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</tbody>
</table>

Mean ± SD: 57 ± 24, 73 ± 10, 0.39 ± 0.05, 61 ± 29, 57 ± 28, 9 ± 11, 74 ± 16, 0.33 ± 0.05, 80 ± 39, 80 ± 30, 14 ± 15

Mean ± SD: 13 ± 10, 5 ± 2, 19 ± 13, 5 ± 3

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Hemodynamics During CPR (Figure 1)

**Conventional CPR.** Aortic peak pressure averaged 61 ± 29 (mean ± SD) mm Hg during conventional CPR. The average aortic diastolic pressure in these patients was 13 ± 10 mm Hg, and the coronary perfusion gradient averaged 9 ± 11 mm Hg. The amount of force applied to the chest wall during conventional CPR was 73 ± 10 lb, and the compression period was 39 ± 5% of the compression-relaxation cycle (Table I). Aortic peak pressures and coronary perfusion gradients varied considerably among patients, ranging from 39 to 126 mm Hg (systolic) and from -1 to 36 mm Hg (gradient), respectively. This variability between patients could not be explained by differences in applied force, duty cycle, period from collapse until measurements, body weight, surface area, chest circumference, or chest diameter. In seven of the nine patients, we could determine that peak aortic pressure was related to applied force when the force of chest compression was varied (significance range, p<0.001 to p = 0.006) and explained 23–78% of the variance in pressure observed in any individual patient. In six of the seven patients, the correlation was positive; in one, it was negative, and in the other two, there was no significant relation.

Both the aortic pressure and coronary perfusion gradients measured during conventional CPR at the beginning, middle, or end of the 20-minute protocol were essentially unchanged. One patient (Patient 5) also had pressures measured earlier during attempted resuscitation by the hospital staff. Aortic pressure and the coronary perfusion gradients recorded during resuscitative efforts immediately after the arrest were 118/48 and 38 mm Hg, respectively, and were 127/37 and 36 mm Hg during the protocol 85 minutes later.

The only parameter that appeared to relate to both achieved peak aortic pressure and the coronary perfusion gradient for the entire group was the aortic pressure measured just before CPR was initiated (p<0.005 and p<0.01, respectively; r = 0.84 for both instances). This "resting" pressure that ranged from 3 to 19 mm Hg did not correlate with body size or weight, duration of arrest before the resting measurement, or pharmacological treatments before initiation of the protocol.

**High-impulse CPR.** High-impulse CPR (120 compressions/min) produced a significantly higher aortic pressure of 80 ± 39 mm Hg (p<0.01) and a higher diastolic pressure of 19 ± 13 mm Hg (p<0.005) compared with pressures achieved during conventional CPR methods (Figure 2). The amount of force used to compress the chest was essentially the same as that used during conventional CPR (74 ± 16 vs. 73 ± 10 lb), although the duty cycle was shorter (33 ± 5%, p = 0.06; see Table I). As with conventional CPR, peak aortic pressure varied widely among patients, ranging from 46 to 161 mm Hg. The mean coronary perfusion gradient achieved was 14 ± 15 mm Hg (range, -4 to 48 mm Hg) and was significantly higher than that recorded during con-
veloped chest compression methods \( p<0.05 \) (Figure 3).

**Pneumatic vest CPR.** Inflation of the vest to 200 mm Hg (five compressions followed by one ventilation) produced aortic pressures and coronary perfusion gradients similar to those achieved with conventional CPR. The aortic peak and diastolic pressures achieved with pneumatic vest CPR were 71 ± 33 and 16 ± 13 mm Hg, respectively, and the coronary perfusion gradient was 11 ± 13 mm Hg (Figures 2 and 3).

Increasing vest inflation pressure stepwise from 200 to 300 mm Hg produced significantly higher peak aortic pressures; however, no significant rise in the aortic diastolic pressure (the major determinant of coronary perfusion) occurred (Figures 4 and 5). Peak pressure increased from 60 ± 29 to 81 ± 39 \( p = 0.006 \). Aortic peak pressure increased on average 1.2 mm Hg for each millimeter of mercury increase in vest inflation pressure (SEE of the slope, 0.2; \( p<0.001; r = 0.91 \)).

On the other hand, simultaneous ventilation along with the vest compression caused an even greater increase in aortic systolic pressure (increasing from an average of 71 ± 33 to 93 ± 27 mm Hg, \( p = 0.008 \); Figure 6). However, simultaneous ventilation also caused an abrupt drop in the coronary perfusion gradients in eight of nine patients (Figure 7). The decline in the coronary perfusion gradient was related to a significant rise in right atrial pressure (from 7 ± 3 during conventional CPR to 13 ± 14 mm Hg; \( p<0.002 \)) caused by high end-expiratory airway pressures attributable to incomplete exhalation between ventilations (air trapping). End-expiratory airway pressures during simultaneous ventilation averaged 19 ± 10 mm Hg and ranged from 5 to 38 mm Hg. This finding was not present during any other CPR method. The high intrathoracic airway pressures, measured in the trachea itself, were identical to pressures recorded in the airway of the vest controller during the 40-msec end-diastolic period and just before vest and airway inflation.

To determine if this adverse effect of simultaneous ventilation could be obviated, we attempted to eliminate the high end-expiratory pressures three ways: 1) by decreasing the delivered tidal volume stepwise from 15 to 10 and then to 5 ml/kg (decreasing the volume to be exhausted); 2) by decreasing the compression and ventilation rate from 72 to 30 cycles/min (to increase the exhaust time); and 3) by allowing the airway to exhaust during the second half of the vest-inflation period (active expulsion of air by direct chest compression). Decreasing the tidal volume diminished but did not eliminate end-expiratory pressure (Table 2). In two patients, coronary perfusion gradients improved with this maneuver; in two other patients, no changes were observed. Decreasing the compression rate from 72 to 30/min also lowered but did not eliminate the high end-expiratory airway pressures. Exhausting the airway early was also ineffective in overcoming this problem. The adverse influence of increased airway pressure on the resulting intravascular pressures is dramatically illustrated in Figure 8, which shows an atrial pressure of 50 mm Hg during rapid ventilation.

Intermittent, abdominal binding during vest CPR did not significantly alter aortic peak, diastolic, or right atrial pressures and had no effect on the coronary perfusion gradient (Figures 9 and 10).

**Effect of epinephrine on aortic pressure.** During the 20-minute CPR protocol, an average of 3.59 ± 0.72 mg epinephrine was administered by continuous infusion. After the hemodynamics during the various CPR methods had been recorded, an additional 1.0-mg bolus was administered through the thoracic aortic catheter to eight of the nine patients. After this additional bolus of epinephrine, there was significant increase in both aortic peak and diastolic pressures [rising from 66 ± 20 to 79 ± 21 mm Hg \( p = 0.01 \) and from 12 ± 12 to 18 ± 12 mm Hg \( p<0.01 \), respectively] (Figure 11).
Pressure gradients between vascular structures during CPR. In one patient (the only one in which the maneuver was attempted), withdrawal of the micromanometer-tipped catheter from the right atrium to the internal jugular vein during CPR revealed a systolic gradient of 80 mm Hg. Withdrawal of the catheter from the right atrium into the inferior vena cava while under fluoroscopic guidance showed a significant systolic gradient in all nine patients (Figure 12). The average gradient recorded at the atrial-inferior caval junction was 49 ± 30 mm Hg (range, 16–118 mm Hg). There was no gradient measured between the thoracic and abdominal aorta in two patients in whom this was also measured. The catheter was advanced from the aorta into the left ventricle during CPR in four patients. Aortic end-diastolic pressure exceeded left ventricular end-diastolic pressure by 5–20 mm Hg. No systolic gradient was present.

Arterial blood gas alterations during CPR. Serial arterial blood gas measurements were made in eight patients. Before CPR, all patients were profoundly acidotic and hypercapnic (mean pH, 6.82 ± 0.18; mean PCO₂, 102 ± 54 mm Hg). After 2 minutes of conventional CPR, the pH increased slightly to 6.93 ± 0.23 (p = 0.1) because of a drop in PCO₂ (67 ± 63 mm Hg; p = 0.009) compared with control values. As would be expected, there was also an improvement in oxygenation (PO₂ rose from 58 ± 72 to 191 ± 196 mm Hg). At the end of the series of CPR techniques, arterial pH was slightly lower (6.70 ± 0.17) than the initial value and is consistent with circulation of venous blood. Arterial PO₂ at the end of the procedure was 343 ± 215 mm Hg (p = 0.015 compared with initial values).

CPR-induced trauma. Postmortem examinations showed evidence of either sternal or rib fractures in all patients. In all but one patient, these occurred during the clinical resuscitation attempt and were evident before the CPR protocol. One patient had a small area of hemorrhage into the abdominal wall (probably from the binder). One patient had evidence of a left pneumothorax (possibly caused by simultaneous ventilation), and six patients had pneumonia. No patient had either hepatic contusions or lacerations. One had a very small retroperitoneal hemorrhage associated with placement of the femoral arterial sheath. Patient 3 also had the incidental finding of extensive pleural asbestosis.

Discussion
CPR is an artificial means of providing blood flow during cardiac arrest to maintain cerebral and myocardial viability until normal circulation can be restored by electrical defibrillation or pharmacological treatment. The majority of experimental and clinical evidence suggests that threshold levels of flow exist, below which myocardial and cerebral viability cannot be maintained for more than a few minutes. Studies in animals show that conventional manual CPR provides very low levels of myocardial and cerebral blood flow, on the order of 5–10% of baseline values.²⁰ It has also been demonstrated, at least in the experimental setting, that the instanta-

Figure 2. Plot of aortic systolic (peak compression) and diastolic (relaxation) pressure (mean and standard deviations shown) during conventional and experimental methods of CPR. High-impulse CPR caused a modest increase in both aortic peak and diastolic pressures over that recorded during the conventional manual method. Peak aortic pressure was also higher during simultaneous chest compression and ventilation methods.

Figure 3. Plot of coronary perfusion gradients (+SD) during each CPR method. Range is given within each bar. With essentially the same applied force to the chest, the coronary perfusion gradient was significantly higher with the high-impulse method than with the conventional method. Average gradient achieved with high-impulse CPR was 14 ± 15 mm Hg. Unlike the high-impulse method, simultaneous ventilation and vest CPR did not augment the perfusion gradient over levels achieved with the conventional manual method.
neous diastolic difference between aortic and right atrial pressures, or the so-called "coronary perfusion gradient," is directly related to myocardial blood flow during cardiac arrest and is highly predictive of outcome. Gradients of 15 mm Hg or more routinely result in successful resuscitation, whereas lower levels are almost never successful.\textsuperscript{14,15} Likewise, aortic systolic pressure is an important determinant of cerebral blood flow.

In the present study, we measured aortic and right atrial pressures and the instantaneous coronary perfusion gradient during conventional and several alternative methods of CPR to better characterize hemodynamic events during CPR in humans. The thoracic peak aortic and diastolic pressures during conventional CPR were similar to those reported with less-rigorous methods, fluid-filled systems, and peripheral cannulas.\textsuperscript{1-12} Aortic systolic pressure varied from 39 to 126 mm Hg among patients, despite minimal differences in applied chest compression force. Unfortunately, this study does not provide any obvious reasons that might account for this wide variability among

\textbf{Figure 4.} Plot of peak aortic pressure in each patient during vest compression of the chest. The vest is inflated first to 200 and then to 250 and 300 mm Hg. Ventilation is nonsimultaneous. Aortic pressure rose significantly as vest inflation pressure was increased.

\textbf{Figure 6.} Plot of peak aortic pressure during vest compression with and without simultaneous ventilation. A significant rise in peak aortic pressure occurred during simultaneous ventilation. The vest was inflated to 250 mm Hg, and 15 ml/kg oxygen was delivered during ventilation.

\textbf{Figure 5.} Plot of aortic diastolic pressures achieved during stepwise increases in vest inflation pressure (see legend for Figure 4). There was no significant rise in aortic diastolic pressure, as seen here, or in the coronary perfusion gradient (not shown) with higher vest pressures. This along with findings in Figure 4 suggests that cerebral but not myocardial perfusion would be improved by increasing vest pressure.

\textbf{Figure 7.} Plot of coronary perfusion gradient with and without simultaneous ventilation. Gradient dropped in eight of nine patients when ventilation was delivered simultaneously with chest compression. This drop was associated with a rise in right atrial pressure caused by positive end-expiratory pressure.
TABLE 2. Effect of Compression Rate, Tidal Volume, and Airway Exhaust Time on End-Expiratory Pressure During Pneumatic Vest Cardiopulmonary Resuscitation With Simultaneous Ventilation

<table>
<thead>
<tr>
<th>Compression rate (cycles/min)</th>
<th>Vest pressure (mm Hg)</th>
<th>Tidal volume (mm Hg)</th>
<th>Airway duty cycle (%)</th>
<th>End-expiratory pressure (mm Hg)</th>
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<tr>
<td>30</td>
<td>200</td>
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</table>

patients. Although the pressures measured during CPR were done several minutes (>20 minutes) after cardiac arrest, we found no statistical relation between aortic pressure and the duration of cardiac arrest preceding the measurement, and the variability was not related to compression force. Indeed, we observed similar aortic pressures over a 90-minute period in one patient who had a very protracted resuscitation attempt. The only parameter that appeared to correlate with the achieved peak aortic pressures for the group of patients studied was the baseline arterial pressure measured just before CPR was initiated. The reason for this variability in baseline pressure was not apparent. It is unclear whether this is related to the volume of blood in the arteries, to arterial compliance, or to both and whether this might explain why some patients develop adequate blood pressure during CPR to sustain life and are resuscitated even after several minutes of cardiac arrest, whereas other similar patients who are treated relatively rapidly succumb even after a short duration of cardiac arrest.

For individual patients, the applied force during CPR was the largest determinant of resulting aortic pressure. Body weight and surface area and chest circumference and diameter did not correlate with the resulting aortic pressure. Pressure was also not related to the duration of cardiac arrest and CPR (i.e., there was no difference in the aortic pressure achieved at the beginning and at the end of the 20-minute protocol).

Aortic and coronary perfusion pressures were significantly but modestly improved during high-impulse CPR. This relatively small augmentation in pressure may not be of major clinical importance, but this is yet to be determined. It would be difficult to manually maintain a consistent rapid compression rate for more than a few minutes because of the amount of work needed to apply this method. Thus, these findings are somewhat discouraging in that none of the previously described alternative CPR techniques that have been used to advantage during experimental CPR caused a major increase in the coronary perfusion gradient and thus, presumably would not increase resuscitation rates over that achieved with present CPR methods.

The pneumatic vest device produced similar aortic pressure to that achieved with conventional CPR. Increasing vest inflation pressure from 200 to 300 mm Hg caused peak aortic but not diastolic pressures to rise. Placement of the vest was an important factor. We found that the highest pressures were obtained when the vest was positioned on the lower thorax. Thus, vest design seems very important, and alterna-

![Figure 8](http://circ.ahajournals.org/)

**Figure 8.** Recordings of the effect of rapid ventilation on right atrial pressure. Ventilation is delivered at 72 cycles/min with 15 ml/kg. Both end-expiratory and right atrial pressures quickly rise, the latter reaching 50 mm Hg. This rise in right atrial pressure adversely affects the coronary perfusion gradient.

![Figure 9](http://circ.ahajournals.org/)

**Figure 9.** Plot of peak aortic pressure before and after phasic abdominal binding simultaneous with vest inflation. Vest is inflated to 200 mm Hg, and the binder inflated to 100 mm Hg. There is no significant improvement in peak aortic pressure.
tive designs to the one used here might yield superior results. Intermittent inflation of an abdominal binder simultaneous with vest inflation was associated with neither hemodynamic benefit nor compromise. Unlike the results in dogs with continuous binding, phasic compression of an abdominal binder in humans seems to have little effect on net venous return. Simultaneous ventilation and vest compression resulted in higher peak aortic pressures. This improvement, however, was accompanied by elevated end-expiratory airway pressures that persisted throughout the relaxation period and, in eight patients, caused the coronary perfusion gradient to drop. We attempted to eliminate air-trapping and its adverse effect on the coronary perfusion gradient by decreasing minute ventilation, increasing the exhaust time, and initiating airway exhaust during chest compression to promote mechanical expulsion of air. None of these maneuvers were effective. Based on this experience, it seems prudent not to use simultaneous ventilation during CPR because of its associated adverse effect on the coronary perfusion gradient.

It is possible that higher doses of epinephrine than what are commonly used might have further increased the aortic and coronary perfusion pressures. Despite the initial epinephrine bolus and continuous infusion administered during this protocol, there was a further significant increase in both peak aortic and coronary perfusion pressures when additional epinephrine was given at the end of the 20-minute protocol. It is also noteworthy that pressure did not deteriorate throughout the 20-minute protocol while epinephrine was being continuously infused. It thus appears that aortic pressure is greatly influenced by vascular resistance and that arterial tone can be further augmented by higher-than-usual doses of epinephrine. Further studies are necessary to determine the amount of epinephrine to maximize aortic pressure in man.

There was also a systolic gradient recorded between the right atrium and inferior vena cava during CPR in every patient. In each, the catheter was withdrawn from the right atrium to the vena cava under fluoroscopic guidance and was noted to be floating freely within each chamber with no evidence of entrapment. Although rudimentary valvular structures exist at the right atrial–inferior vena cava junction, no gradient has previously been reported at this site. In fact, several studies in animals have clearly demonstrated free reflux of blood from the right atrium to the inferior vena cava. The gradient in humans may be attributable to mechanical distortion of the inferior vena cava by the diaphragm during chest compression. The significance of this finding is unclear, but this, along with the observations made at the superior thoracic outlet, provides further evidence that the thoracic pump mechanism is responsible for at least part of the blood flow generated during CPR. The gradient may also affect the magnitude and timing of pulmonary blood flow and could, therefore, have substantial influence on cardiac output.

These findings should be considered in the context that they were made after a long duration of cardiac arrest after the patient had died and that acidosis was present; the resulting pressures may have been influenced by these and other factors. However, in one patient who was monitored for over 1.5 hours, no significant change in pressure
was observed. No attempt was made to correct the metabolic acidosis because it is impossible to accurately titrate this condition and also because of concern about possible direct hemodynamic effects of sodium bicarbonate administration.

Based on these observations, none of the experimental CPR methods evaluated in the present study seem likely to represent a substantial improvement over conventional CPR methods. There is no apparent hemodynamic benefit of abdominal binding during CPR; simultaneous ventilation appears to be detrimental. We could find no evidence to support the use of these latter two techniques during attempted resuscitation in man.

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