Simultaneous Aortic, Jugular Bulb, and Right Atrial Pressures During Cardiopulmonary Resuscitation in Humans

Insights Into Mechanisms

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Pressure gradients across and between the head and chest were studied during mechanical cardiopulmonary resuscitation (CPR) in 22 humans. Patients in medical cardiac arrest, managed by ACLS guidelines, underwent placement of aortic arch (Ao), jugular venous bulb (JVB), and right atrial (RA) catheters. Simultaneous pressures were measured, and intercatheter gradients were calculated. The JVB to RA pressure difference is the gradient between the cervical and central venous circulations. It was negative when averaged throughout the CPR cycle and was more negative during compression than relaxation, $-19 \pm 12$ and $-2 \pm 6$ mm Hg, respectively. This indicates that the intrathoracic pressure rise was not transmitted to the jugular venous system, supporting the concept of a competent jugular valve mechanism during CPR. It is consistent with the thoracic pump model of cerebral perfusion. JVB to RA was positive only during early relaxation, allowing blood return from the head. The Ao to JVB gradient, although not equal to cerebral perfusion pressure, is the maximum potential pressure gradient for blood flow across the cerebral vasculature. It was positive throughout CPR, $25 \pm 17$ mm Hg during compression, and $9 \pm 10$ mm Hg during relaxation. The Ao to RA gradient during the relaxation phase is CPR coronary perfusion pressure. In most patients, it was minimally positive in both phases of the CPR cycle: $7 \pm 14$ in compression and $7 \pm 9$ mm Hg during relaxation. This appears to be inadequate in providing sufficient blood flow to meet the metabolic needs of the myocardium. Four patients had larger gradients during compression suggestive of cardiac compression. We conclude that in most patients there are functional venous valves at the thoracic inlet and the potential for a thoracic pump mechanism for cerebral perfusion. A subset of patients may have direct cardiac compression. Thoracic and cardiac pump models of CPR are not mutually exclusive. (Circulation 1989;80:361–368)
were measured during CPR in humans as a means to evaluate pressure gradients across and between the brain and the heart during mechanical CPR. The gradient between the JVB and RA was of particular interest because it allows the evaluation of the venous valving mechanism at the thoracic inlet. These valves have been investigated in animals during CPR,4 in humans during spontaneous circulation,8 and anecdotally during CPR in humans.2,4 The present study evaluated these valves prospectively in a consecutive series of patients. If a thoracic pump mechanism were present, venous valves would prevent transmission of RA pressure to the JVB during compression,7 and a negative JVB to RA (JVB-RA) gradient would occur.

The jugular venous bulb is an enlargement of the proximal internal jugular vein. It is located above the jugular foramen at the intersection of the sigmoid sinus and internal jugular vein. A radiograph of a properly placed JVB catheter is seen in Figure 1. Pressures measured at the JVB may be indicative of those on the venous side of the cerebral circulation, and blood sampled at this location is almost exclusively intracranial in origin.12 Although the Ao to JVB pressure gradient (Ao-JVB) is neither cerebral perfusion pressure nor directly predictive of cerebral blood flow, it does represent the maximum pressure available for brain perfusion.

The Ao to RA gradient (Ao-RA) is equivalent to coronary perfusion pressure during CPR and is predictive of myocardial blood flow and return of spontaneous circulation.9,13–16

**Methods**

Adult emergency department patients in normothermic, nonhemorrhagic medical cardiac arrest were eligible for entry. Down time was defined as time from collapse until initiation of basic life support. Patients having return of spontaneous circulation before or during placement of the catheters were excluded. The protocol was approved by the Institutional Review Board of Henry Ford Hospital. Clinical management was by physicians staffing the department at the time of cardiac arrest and was in accordance with ACLS guidelines.17 All catheter placement was by guide-wire technique. A double lumen 7.5F catheter (Cook, Bloomington, Indiana) was placed in the RA through the subclavian vein. The proximal port was used for drug administration, and the distal port was used for pressure monitoring. Members of an on-call research team placed a 60-cm 5.8F catheter (Cook) into the aortic arch through the femoral artery using a percutaneous or cut-down technique. A 4F 15-cm catheter (Cook) was simultaneously inserted percutaneously into the internal jugular vein and advanced retrograde into the JVB.18 This was done above the thoracic inlet so as not to interfere with any valve mechanism that may be present. The fluid-filled catheters were connected to pressure transducers (Abbott Systems, North Chicago, Illinois). The resulting signals were amplified (Hewlett-Packard 78205D, Palo Alto, California), and simultaneous pressure tracings were recorded (Hewlett-Packard Systems, North Chicago, Illinois).
7758 multichannel). Blood samples for gas analysis were obtained simultaneously from all three locations. The heparinized fluid flush and amplifier/recorder system were set up and calibrated before patient arrival.

Patients received intravenous epinephrine (1 mg) every 5 minutes and sodium bicarbonate according to arterial blood gas results. CPR was performed in all but one patient with a pneumatic compression device (Thumper, Michigan Instruments, Grand Rapids, Michigan) with settings of 60 compressions/min, 50% duty cycle, a ventilation after each fifth compression and 80–100 lb chest pressure as needed to achieve a piston excursion of 2 in.

Paper tracings were converted to digitized data with an optical/magnetic microcomputer scanning system (Jandel Scientific, Corte Madera, California). All pressures were measured just after catheter placements. Intercatheter gradients were calculated by microcomputer subtraction of the digitized tracings. Individual patient pressures are the average of five consecutive compressions/relaxation cycles, which is one ventilation to ventilation cycle.

Compression phase was defined as beginning with the inflection point at the onset of downstroke and as ending with the inflection point at the beginning of the upstroke (Figure 2). The rest of the cycle was defined as the relaxation phase. Sample means and standard deviations were determined in milligrams of mercury for compression and relaxation phases and the time points of early relaxation (0.06 second after the onset of relaxation), compression maximum, and end relaxation. The latter two are reported to allow comparison with earlier studies. JVB catheters were withdrawn 2 cm during CPR to assure that transmitted pressures were not dampened by the catheter tip being pressed against the vessel wall.

Catheter positions were confirmed by postmortem radiographs of the neck (Figure 1) and chest, and appropriateness of blood gas measurements (i.e., \( \text{PO}_2 < 60 \text{ mm Hg} \) for venous catheters). Patients with improperly positioned catheters were excluded. Internal jugular venography was performed in 15 patients to confirm that no obstruction, such as hematoma, caused dampening of the waveforms with resultant loss of pressure.

Pressures and gradients were prospectively defined to be different if statistical testing, either parametric (t test) or nonparametric (Wilcoxon's rank-sum test), indicated that the difference was significant at the \( p=0.05 \) level of confidence.

**Results**

Three patients had return of spontaneous circulation before catheter placement was attempted. Thirty patients were entered into the protocol. Of these patients, data are reported for those \( n=22 \) in whom the protocol was successfully completed and catheter placements confirmed. Patients were excluded from data analysis for the following reasons: improperly placed JVB or Ao catheters (four and one patients, respectively), and uninterpretable pressure tracings (three patients). Two patients subsequently had return of spontaneous circulation; this sample was too small to allow comparison with the nonsurvivors, but the Ao-RA pressures in these two patients were above the mean for the group as a whole.

Mean age of patients was 66±15 years. Accurate down times were available in 11 patients and averaged 12±10 minutes. In all patients but two, basic life support was initiated only upon arrival of Detroit Emergency Medical System personnel, whose response and transport times were between 5 and 15 minutes. Catheter placement time averaged 13±7 minutes. Thus, in many patients, the time from arrest to pressure measurement (down time plus transport time plus catheter placement time) was more than 30 minutes. There were three patients who had return of spontaneous circulation during catheter placement and had nearest with catheters in place after a period of relative hemodynamic stability. Immediate measurements were obtained, and this was considered zero down time. The initial rhythm was ventricular fibrillation in 12 patients (55%), asystole in five (23%), electromechanical dissociation in two (9%), and not reported in three. The average down time of patients admitted while
in ventricular fibrillation was not statistically different than those in asystole.

The data did not satisfy the assumption of a normal distribution, so the nonparametric Wilcoxon’s rank-sum test was used.

Compression phase pressures (Table 1) were greater than relaxation phase pressures at all three locations \( (p<0.0001) \). JVB pressure was less than the RA and Ao pressures during compression \( (p<0.0001) \) and less than Ao during relaxation \( (p<0.0001) \). Ao pressures were greater than RA pressures during both compression \( (p=0.05) \) and relaxation \( (p=0.01) \). Pressures at peak compression were considerably higher than for the entire compression phase at each location. Aortic end-relaxation pressures were not different than averages for the entire relaxation phase (80% power to detect a difference of at least 1.7 mm Hg).

Pressure gradient data are shown in Table 2. The JVB-RA gradient (Figure 3) was negative when averaged throughout each phase and was more negative during compression than relaxation \( (p=0.0001) \). This gradient became positive only during the early part of the relaxation phase. At 0.06 second after the onset of relaxation, the gradient was 8±8 mm Hg. Pulling back the JVB catheter resulted in no change in pressures or waveforms.

The Ao-JVB gradient (Figure 2) was positive throughout the CPR cycle and greater during compression than relaxation \( (p=0.0001) \).

For the patients as a whole, the Ao-RA gradients were similar during either phase of CPR (Table 2, Figure 4). However, four patients (18%) had Ao-RA gradients greater than 15 mm Hg during compression, the maximum being 50 mm Hg (Figure 5). In the remaining patients, Ao-RA was slightly greater during relaxation but not to a statistically significant degree.

There was no correlation between the Ao-RA and JVB-RA gradients \( (r=0.15) \). There were individual patients with all combinations of these gradients, ranging from both gradients being large to both being absent. The most common pattern \( (n=12) \) was a JVB-RA gradient of at least \(-15 \) mm Hg and a small Ao-RA gradient that was slightly greater in relaxation.

**Discussion**

Methodology

A mechanical compression device was used because of its consistency. CPR performed by humans has more variability moment to moment and between providers. Although the mechanism of mechanical CPR appears to be different than that for manual CPR in certain animal models, this has not been well studied in humans.10 The terms “compression” and “relaxation” have been used instead of “systole” and “diastole” because they accurately reflect the events occurring with each part of the CPR cycle and do not imply that the heart is acting as a pump. It is important to realize, however, that because the compression phase includes the rise of the compression waveform (downstroke of the piston) and because the relaxation phase includes its descent (upstroke of piston), their average values are lower and higher, respectively, than previously reported.14,19

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**Table 1. Blood Pressures for All Patients in Series**

<table>
<thead>
<tr>
<th></th>
<th>Compression (mm Hg)</th>
<th>Relaxation (mm Hg)</th>
<th>Peak compression (mm Hg)</th>
<th>End relaxation (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aorta</td>
<td>48±16</td>
<td>23±10</td>
<td>61±22</td>
<td>22±10</td>
</tr>
<tr>
<td>Jugular venous bulb</td>
<td>23±10</td>
<td>15±8</td>
<td>29±12</td>
<td>14±8</td>
</tr>
<tr>
<td>Right atrium</td>
<td>41±14</td>
<td>16±6</td>
<td>55±17</td>
<td>16±7</td>
</tr>
</tbody>
</table>

Data are mean±SD.

Ao-RA was greater than jugular vein bulb in compression \( (p<0.0001) \) and relaxation \( (p=0.001) \). Jugular vein bulb was less than right atrium during compression \( (p<0.0001) \). Ao-RA was greater than right atrium in compression \( (p=0.05) \) and relaxation \( (p=0.01) \).

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**Table 2. Gradients for All Patients Included in Series**

<table>
<thead>
<tr>
<th></th>
<th>Compression (mm Hg)</th>
<th>Relaxation (mm Hg)</th>
<th>Early relaxation (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>JVB-RA</td>
<td>-19±12*</td>
<td>-2±6</td>
<td>8±8</td>
</tr>
<tr>
<td>Ao-JVB</td>
<td>25±17</td>
<td>9±10</td>
<td>—</td>
</tr>
<tr>
<td>Ao-RA</td>
<td>7±14</td>
<td>7±9</td>
<td>—</td>
</tr>
</tbody>
</table>

Data are mean±SD.

JVB-RA, jugular vein bulb to right atrial gradient; Ao-JVB, aortic to jugular vein bulb gradient; Ao-RA, aortic to right atrial gradient.

*Compression/relaxation difference significant \( (p=0.0001) \).

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**Figure 3. Tracings of jugular vein bulb (JVB), right atrial (RA) pressures, and JVB minus RA gradient (thick line) from a typical patient.**

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The prolonged average down time needs to be considered when applying these data to other groups. The results may also have been biased by the exclusion of patients who had return of spontaneous circulation before catheter placement. These individuals may have had higher gradients.

**Jugular Venous Bulb to Right Atrial Gradient**

JVB-RA is the pressure gradient between the cervical and central venous circulations. The negative gradient during compression shows failure of the intrathoracic pressure rise, as measured in the RA, to be transmitted up the internal jugular vein.

Our study substantiates most animal studies, as well as the limited human data, and shows that most patients maintain a functioning venous valve at the thoracic inlet even after prolonged CPR. This venous valve is consistent with the thoracic pump model. The valve itself may be anatomic or simply functional in nature, resulting from the collapse of the great veins during compression in the manner of a Starling resistor. The results confirm that the valve is located between the RA and internal jugular vein at the thoracic outlet or base of the neck. Twice, when the JVB catheters presumably went into the chest and were drawn back into the neck, the wave forms and pressures changed from those typical of intrathoracic catheters (RA in Figure 3) to a JVB pattern (JVB in Figure 3) as the catheter tip entered the neck. This has been previously observed. Demonstration that there is not a similar drop in pressure between the Ao and carotid arteries, as has been done in dog models, would further substantiate a positive gradient across the brain and would lend further support to the thoracic pump being the pump for cerebral perfusion in humans.

The JVB-RA gradient also indicates the potential for blood return from the head. The small negative value during relaxation does not indicate absence of forward flow during the complete cycle but results from averaging of pressures for the entire relaxation phase. The gradient is positive during early relaxation and would allow blood flow back to the chest at this time. Because blood flow during CPR is quite reduced, only a small part of the CPR cycle may be needed for return flow through these large vessels. Also, cerebral venous drainage may occur during the entire cycle using the compliant internal jugular vein as a reservoir. The typical pattern of increasing JVB pressure throughout the compression phase, whereas RA pressure tended to plateau (Figure 3), suggests an increase in internal jugular volume. This could be retrograde leakage from the chest or continued cerebral venous drainage.

**Aortic to Jugular Venous Bulb Gradient**

The Ao-JVB pressure gradient represents the potential for perfusion across the cerebral vasculature. It was positive throughout the CPR cycle and was greater during compression. However, this does not prove that blood flow occurs or even that perfusion pressure is greater during the compression phase. During normal circulation, cerebral perfusion pressure is equal to mean arterial pressure minus the intracranial pressure. Guerci et al and others showed that intracranial pressure rises with each (CPR) compression because of pressure transmission through both cerebrospinal fluid and vessels other than the internal jugular vein. Their work, in an animal model without the use of epinephrine, indicates that mean arterial minus intracranial pressure (MAP-ICP) may be cerebral perfusion pressure during CPR. However, it is clear that pressures in the internal jugular vein must be below

![Figure 4](image-url)  
*Figure 4. Tracings of aortic (Ao), right atrial (RA) pressures, and Ao minus RA gradient (thick line) from a typical patient with a compression gradient less than 15 mm Hg.*

![Figure 5](image-url)  
*Figure 5. Tracings of aortic (Ao), right atrial (RA) pressures, and Ao minus RA gradient (thick line) from one of the three patients with compression gradients greater than 15 mm Hg.*
those in the Ao for net forward flow to occur across the cerebral vasculature. The positive gradient we found throughout the CPR cycle indicates the potential for blood flow. In one patient, this gradient was zero or negative during both phases of CPR, and it can be assumed that there was no cerebral blood flow in this individual.

**Aortic to Right Atrial Gradient**

The Ao-RA gradient during relaxation phase is coronary perfusion pressure during CPR.9,25,26 and is positively correlated with coronary blood flow and return of spontaneous circulation.15,16,26,27 Because the magnitude of the pressures and the shape of the Ao and RA waveforms were similar in most of our patients, it is reasonable to conclude that there was no selective ventricular compression or that there was tricuspid incompetence allowing right ventricular pressure to be transmitted to the RA in these patients. This equalization of pressures on the left and right sides of the heart is consistent with the thoracic pump model of CPR.28 It may also reflect time-dependent deterioration of perfusion pressure consistent with the minimal effectiveness of CPR after prolonged arrest.29

When positive Ao-RA gradients and coronary blood flow have been shown in thoracic pump models, they are usually greater during relaxation9 and have been attributed to differing elastic properties of the arterial and venous systems within the chest. Regardless of the actual mechanism of coronary blood flow, it is important to note that the coronary perfusion pressures in most patients in this study would not have been large enough to supply blood flow adequate to meet the metabolic needs of the arrested myocardium22 and were probably insufficient to overcome wall tension in those that were fibrillating.20 Recent work in our department has shown that an Ao-RA gradient of at least 15 mm Hg is needed before return of spontaneous circulation occurs.16

Four patients (18%) in this series had large positive Ao-RA gradients during the compression phase, which were suggestive of cardiac compression. In a previous study in our department, three of 22 patients (14%) had compression phase Ao-RA gradients of more than 25 mm Hg.14

If the data are examined without the patients who had large Ao-RA during compression, that is, those that may have had a component of cardiac pump, then the gradient will tend to be greater during the relaxation but not to a statistically significant degree.

**Mechanism of Cardiopulmonary Resuscitation**

Although it is now widely believed that during standard external CPR the chest and noncardiac vasculature are responsible for blood flow,2,4,30,31 this thoracic pump model has not been universally accepted.10,11,32,33 One study using broad-chested dogs, whose chest configuration may more closely resemble humans, showed direct cardiac compression and suggested that an intact thorax was not necessary for cerebral blood flow.14 A recent echocardiographic investigation in dogs showed that the heart and its valves act as a pump moving blood from the venous to the arterial side of the circulation.11 However, similar studies in humans have failed to demonstrate appropriate valve motion,3 but this may have been because patients were examined late in resuscitation when the cardiac mechanism had failed. In the over 40 patients from both series within our department, a subset of patients had Ao-RA gradients during compression that were large enough to suggest cardiac compression. Rudikoff and associates2 using a different methodology concluded that approximately 30% of patients had some component of cardiac pump. Because our studies have involved measurement of pressure gradients, the mechanism by which these gradients occur can only be inferred. The role of specific structures, such as cardiac valves, cannot be commented on.

Demonstration of possible cardiac compression does not, however, prove that a cardiac pump is responsible for extrathoracic cerebral perfusion. Our data indicate that in most humans undergoing mechanical CPR most of the cranial arteriovenous pressure gradient, Ao-JVB, is secondary to the pressure drop between the chest and internal jugular vein. Animal studies indicating that cerebral blood flow is correlated with MAP-ICP also show that techniques, such as simultaneous ventilation compression, and abdominal binding, that raise intrathoracic pressure also increase cerebral blood flow.23 Thus, both human and animal data support the conclusion that cerebral perfusion primarily results from a thoracic mechanism. In certain patients, a mix of cardiac and thoracic pump mechanisms may occur, and the contribution of each mechanism may vary even in a single patient at different times after arrest. At one end of this spectrum would be the purely thoracic pump model of cough-induced CPR.5 At the other end, patients with large Ao-RA gradients during compression but with minimal JVB-RA gradients may be an example of a pure cardiac pump.14 If the JVB-RA gradient is considered to indicate function of the thoracic pump and if the Ao-RA gradient during compression indicates the function of a cardiac pump, then the independence of the two mechanisms is supported by data from individuals in our series. These data showed that correlations did not occur between any of the possible combinations of gradients. Some patients had large JVB-RA and Ao-RA compression gradients, indicating effective thoracic and cardiac pumps, whereas others had significant pressure differences across only one or the other. The cardiac and thoracic pump models need not be mutually exclusive. The cardiac pump may move blood from the venous to the arterial side of the circulation, whereas the thoracic pump may be the basis for most of the cerebral perfusion gradient. This
interpretation is consistent with the limited human data available at this time.

In chronically instrumented dogs, Maier and associates found the mechanism of mechanical CPR to be different from that of manual high-rate CPR and found the aortic and coronary blood flows to be higher with the latter technique. In their model, manual CPR appeared to have a cardiac pump mechanism. Whether or not this difference exists in humans at standard compression rate is not clear. Weisfeldt and Halperin have been concerned that adhesions may result in greater-than-normal cardiac compression in this dog model. Working in a dog model with an intact chest, they found manual CPR to have a thoracic pump mechanism and found no difference in the hemodynamics when a mechanical vest was used. They have also found that blood flow increases not with the rate of CPR but rather as the duration of compression increases. They point out that as the rate of manual compression increases, the fraction of the CPR cycle taken up by compression increases. At high rates, the percentage approaches 50%, which is the amount found optimal in their model and the fraction delivered by the Thumper. Whether or not manual and mechanical CPR have differing mechanisms remains controversial, and this should be kept in mind when interpreting our data.

Pressures measured with intravascular catheters alone cannot determine which structure transmits pressure to the intravascular space. In the few patients with positive Ao-RA compression gradients, direct ventricular compression and appropriate cardiac valve motion may be present, but this cannot be proved with our techniques, and other structures could be involved. One possibility is that the pump may not be thoracic so much as mediastinal in nature. The mediastinum has enough structural integrity to efficiently transfer force from the chest wall to the great vessels. Combined with the valves at the thoracic inlet, it may be a thoracic pump. With appropriate cardiac valve motion, it may also be the basis of a cardiac pump.

Because we have measured only pressures, direct conclusions about blood flow must also be limited. As stated above, the Ao-JVB gradient is the maximum potential for cerebral perfusion, not the actual cerebral perfusion pressure. The relation between Ao-JVB during CPR and cerebral blood flow, much less neurologic outcome, is unknown. That this gradient is positive is reassuring. A zero or negative gradient across the cerebral vascular system throughout the CPR cycle would indicate absence of perfusion. Our demonstration that in most patients CPR produces a positive gradient across the head, as reflected by Ao-JVB, is consistent with previous animal data showing cerebral blood flow. Anecdotal clinical experience indicates that CPR is capable of maintaining cerebral viability for limited periods. However, most of the patients studied in our department had Ao-RA gradients less than that necessary for return of spontaneous circulation. This has important clinical implications. In patients similar to ours, the thoracic pump and its venous valve mechanism may continue to function after prolonged down times. This may maintain cerebral viability, but the environment within the myocardium is not likely to improve. Should standard measures not meet with success during the initial period of resuscitation, eventual return of spontaneous circulation would be unlikely, and alternative measures to raise Ao-RA, such as open-chest CPR or cardiopulmonary bypass, should be considered. We conclude that during standard mechanical CPR in patients with relatively long down times most have functional venous valves at the thoracic inlet and that a thoracic pump mechanism is the basis for potential cerebral perfusion. A subset of patients had gradients indicative of cardiac compression. Thoracic and cardiac pump models, however, are not mutually exclusive and may operate alone or in combination in any given patient.

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