Biphasic Extrathoracic Pressure CPR*
A Human Pilot Study

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Hypothesis: Alternating intrathoracic pressure by means of a chest cuirass can cause perfusion and ventilation equal to or better than standard cardiopulmonary resuscitation (CPR) for humans in cardiac arrest.

Design: Nonrandomized, nonblinded, crossover pilot study.

Setting: Large urban emergency department.

Subjects: Five adult normothermic, nontraumatic, out-of-hospital cardiac arrest patients unresponsive to standard advanced cardiac life support.

Method: Right atrial and aortic catheters were inserted for pressure measurement and blood gas analysis while the patient was receiving standard CPR by a pneumatic compression device (Thumper). The Thumper was then replaced by a chest cuirass (Hayek Oscillator). Pressure and blood gas measurements were then repeated.

Results: The coronary perfusion pressure increased from -1.2 ± 8.6 mm Hg to 6.2 ± 6.9 mm Hg for a mean change of 7.4 ± 3.1 mm Hg (p = 0.006). The compression phase gradient increased 10.0 ± 21.9 mm Hg (p = 0.364). The venous to arterial Pco2 gradient decreased 44.5 ± 32.3 mm Hg (p = 0.070). The oxygen extraction ratio increased 1.6 ± 9.4 percent (p = 0.761). The mean arterial Pao2 and Pco2 changed from 252 to 240 mm Hg (p = 0.836) and from 53 to 66 (p = 0.172) mm Hg, respectively.

Conclusion: The Hayek Oscillator chest cuirass produced a significant improvement in the coronary perfusion pressure. There was a trend for improved systemic perfusion as indicated by an improved compression phase gradient and venous to arterial Pco2 gradient, although this was not supported by the lack of improvement in the oxygen extraction ratio. The cuirass also adequately oxygenates and ventilates unassisted by positive pressure ventilation.

References:

1. Blood flow during cardiopulmonary resuscitation (CPR) has been demonstrated to occur in part by the thoracic pressure pump mechanism. According to this mechanism, systemic blood flow is driven by an intrathoracic to extrathoracic pressure gradient generated during the compression phase of CPR (focal cardiac compression is not required). This gradient is called the compression phase gradient (CPG). Retrograde flow is prevented by functional valving at the venous thoracic inlet. Coronary blood flow is driven by an aortic to right atrium pressure gradient during the decompression (or relaxation) phase of CPR. This gradient is called the coronary perfusion pressure (CPP). Animal and human studies have shown a correlation between CPP and return of spontaneous circulation (ROSC).

2. For this reason, increasing the CPP is a primary goal of CPR.

Blood flow during CPR has been shown to occur in part by the thoracic pressure pump mechanism. According to this mechanism, systemic blood flow is driven by an intrathoracic to extrathoracic pressure gradient generated during the compression phase of CPR (focal cardiac compression is not required). This gradient is called the compression phase gradient (CPG). Retrograde flow is prevented by functional valving at the venous thoracic inlet. Coronary blood flow is driven by an aortic to right atrium pressure gradient during the decompression (or relaxation) phase of CPR. This gradient is called the coronary perfusion pressure (CPP). Animal and human studies have shown a correlation between CPP and return of spontaneous circulation (ROSC).

Methods

This pilot study was approved by the Henry Ford Hospital Institutional Review Board for Human Research. Five patients arriving at the Emergency Department in nontraumatic, normothermic cardiac arrest who failed to respond to advanced cardiac life support (ACLS) and were deemed unsalvageable by the treating clinician were entered into the study. An on-call research team placed an aortic arch catheter (60-cm 5.8F Bunegin-Albin, Cook Critical Care, Bloomington, Ind) via the femoral artery and a right atrial catheter (22-cm 7.5F Triple Lumen, Baxter-Edwards, Irvine, Calif) via the subclavian vein. These catheters were attached to precalibrated pressure transducers.
Figure 1. Schematic drawing of the cuirass during inspiration and expiration.

Figure 2. Patient wearing the cuirass. [Printed with permission of Dr. Michael Bennett, Western Pennsylvania Hospital, Pittsburgh.]

Table 1--Subject Characteristics*

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age, yrs</th>
<th>Time Interval, min</th>
<th>Intraocular Pressures, cm H_2O</th>
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<td></td>
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*Insp = inspiratory (relaxation phase) pressure; exp = expiratory (compression phase) pressure.

Pressures were measured 3 min after receiving the epinephrine and were averaged over five consecutive waveforms. The patients remained apneustic throughout the study period.

Statistical Methods

Two-tailed paired student's t tests were used to compare the Thumper with the chest cuirass for CPP. CPP, venous to arterial PaO_2 gradients, oxygen extraction ratio (OER), and blood gas values. A p value less than or equal to 0.05 was defined as statistically significant. The values presented are means ± standard deviation.

Results

The five male subjects had a mean age of 60 ± 12 years. None had a witnessed arrest, and none had bystander CPR. One patient (subject 3) had prehospital ACLS. The average prehospital time interval (the time from when emergency medical services received the emergency call to when they arrived at the hospital) was 25 ± 5 min. The average inhospital time interval prior to entry into the study was 26 ± 31 min (Table 1).

The intraocular inspiratory and expiratory pressures generated by the Hayek Oscillator were –21 ± 12 cm H_2O and 36 ± 12 cm H_2O, respectively (Table 1).

The Hayek Oscillator chest cuirass increased CPP.

Table 2--Compression and Relaxation Phase Pressures*

<table>
<thead>
<tr>
<th>Subject</th>
<th>Aor(R)</th>
<th>RA(R)</th>
<th>CPP</th>
<th>Aor(C)</th>
<th>RA(C)</th>
<th>CPP</th>
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*All values given in mm Hg. Aor(R) = aortic compression pressure; Aor(C) = aortic relaxation pressure; RA(R) = right atrial compression pressure; RA(C) = right atrial relaxation pressure; CPP = coronary perfusion pressure; CPG = compression phase gradient.
from $-1.2 \pm 8.6$ mm Hg to $6.2 \pm 6.9$ mm Hg for a mean change of $7.4 \pm 3.1$ mm Hg ($p = 0.006$). The increase in CPG by $10.0 \pm 21.9$ mm Hg ($p = 0.364$) is primarily due to one patient (subject 3). Table 2 lists the aortic and right atrial pressures during compression and relaxation phases. The right atrial relaxation phase pressures decreased in three subjects and increased in two subjects. In two cases, the aortic compression phase pressure was higher with the cuirass than with the Thumper. The direction or magnitude of the pressure changes did not seem to correlate to the baseline pressures nor to body habitus. Additionally, there did not appear to be a relationship between the intracuirass pressures and either the change in CPG or CPP.

Table 3 lists the blood gas values obtained on four of the patients. (The blood gas values for subject 3 were clotted.) The mean arterial Po$_2$ and Pco$_2$ changed from 252 to 240 mm Hg ($p = 0.836$) and from 53 to 66 ($p = 0.172$) mm Hg, respectively, and are probably of no clinical significance. However, the venous to arterial Pco$_2$ gradient decreased when the subjects were switched from the Thumper to the cuirass. The mean Pco$_2$ gradient decrease was $44.5 \pm 32.3$ mm Hg ($p = 0.070$). The change in this gradient was due to both a decrease in the venous Pco$_2$ and an increase in the arterial Pco$_2$; however, the arterial change is modest ($13.0 \pm 14.5$ mm Hg) compared with the venous change ($-31.5 \pm 32.0$ mm Hg). None of the subjects received bicarbonate. There was no apparent relationship between the change in Pco$_2$ gradient and the intracuirass pressures.

The OER increased from $75.8 \pm 14.1$ percent to $77.3 \pm 16.2$ percent for a mean change of $1.6 \pm 9.4$ percent ($p = 0.761$). The OER increased for all subjects except for subject 2. In this subject, the OER dropped dramatically from 81 to 69 percent. There was no apparent relationship between the change in OER and the intracuirass pressures.

**Discussion**

Standard CPR is currently performed by focal compression of the sternum which yields a myocardial blood flow only 5 percent of normal and a systemic blood flow 15 percent of normal. These low blood flows are one reason for the poor outcomes from cardiac arrest. Additionally, standard CPR is also associated with severe thoracoabdominal trauma in nearly half of all patients. The effect of this trauma on long-term survival, while not previously studied, is almost certainly significant.

Multiple studies have shown a correlation between CPP and ROSC. Experimental forms of CPR have been developed to try to improve the CPP and thus ROSC and long-term outcome. One such method rhythmically squeezes the thorax by use of a circumferential pneumatic vest. This method (known as vest-CPR) was developed because of the observation that coughing can produce sufficient blood flow to keep a person conscious while in ventricular fibrillation. Vest-CPR attempts to mimic cough CPR by rhythmically increasing intrathoracic pressure. Animal and human studies have been encouraging. When the vest is placed only on the thorax and ventilations are interposed with every fifth compression, the CPP has been shown to increase from 6 mm Hg to 29 mm Hg causing myocardial blood flow to increase from 5 percent to 40 percent of normal. This animal study also demonstrated improved cerebral blood flow, increased ROSC, and decreased thoracoabdominal trauma. In a human study, Swenson et al showed that vest-CPR was equivalent to standard CPR. When vest-CPR was combined with abdominal binding and simultaneous ventilations, the results of animal studies were conflicting with some showing improvement while others demonstrating no change or even deterioration of hemodynamics and outcome. One possible reason for the discordant results was demonstrated by Hausknecht et al in a canine heart/lung preparation. In this study, they showed that if pulmonary pressures are high compared with static blood pressure and intrathoracic pressure, then alveolar vascular collapse occurs causing blood to be trapped in the right side of the heart and thus decreasing cardiac output and
CPP.

Unlike coughing, vest-CPR does not generate negative intrathoracic pressure. In a computer simulation, Lin et al. showed that by alternating negative and positive thoracic pressure (the biphasic intrathoracic pressure model of our study), hemodynamics were shown to be superior to other forms of standard and experimental CPR. Lin et al. proposed that the negative intrathoracic pressure phase facilitates blood return to the thorax by lowering central venous pressure, thus improving systemic perfusion. The CPP is improved because the venous side pressures are decreased to a greater degree on the venous side than on the arterial side because of the difference in vascular tone. According to their model, an intrathoracic pressure swing from −100 to +100 cm H2O would be needed for optimum circulation. To make use of negative intrathoracic pressure in a dog study, Cohen et al. modified standard chest compressions such that during the upstroke the chest was actively pulled up instead of passively recoiling. This was called active compression decompression CPR. They found that the CPP almost tripled and the cardiac output almost doubled. As a side effect, this method also adequately ventilated the dogs, thus obviating the need for positive pressure ventilation and its associated risks. In a pilot human trial using this method, they found that the velocity time interval was significantly improved; however, neither CPP nor cardiac output was measured. Unfortunately, this method compresses the sternum 3.75 to 5 cm as in standard CPR and is thus just as likely to induce trauma.

For three of the subjects in our study, the increase in CPP was driven by a drop in relaxation phase pressure for the right atrium more so than for the aorta as expected by the theory of Lin et al. and as also found by Cohen et al. in their active compression decompression dog studies. In two subjects, however, the right atrial relaxation phase pressure increased. In these subjects, the rise in the right atrial pressures was not as great as in the aortic pressures; thus the CPP improved. Interestingly, these two subjects also had higher compression phase pressures for the cuirass compared with the Thumper.

The changes in the blood gas values give additional insight into the perfusion dynamics of the cuirass. During circulatory failure, it has been shown that the venous to arterial Pco2 gradient increases because of increased blood transit time across systemic and pulmonary capillary beds primarily leading to an increase in venous Pco2, and to a lesser degree to a decrease in arterial Pco2. Improvement in this gradient would then reflect a decrease in capillary transit time and thus improved systemic circulation. Our data are consistent with these studies, showing that the cuirass may have improved systemic circulation.

Contrary to the changes in CPP and Pco2, the OER increased, indicating a decreased systemic perfusion caused by the cuirass. While the change in OER was not statistically significant, it clearly did not even trend in the expected direction with the exception of subject 2. The reason for the apparent conflict between OER and the venous to arterial Pco2 gradient is not clear. Other than for a random sampling error, the OER may have increased because of a redistribution of blood flow to previously dormant tissue beds or to tissue beds with high oxygen extraction capabilities (i.e., heart and brain). This is supported by the increase in CPP from a negative to a positive value suggesting that the myocardium was not being perfused until the cuirass was used.

The arterial Po2, shows no clinically significant changes between the two CPR methods indicating that the cuirass adequately oxygenates. The increase in arterial Pco2 can be explained either by a decrease in minute ventilation or increased pulmonary perfusion. Despite the increase in Pco2, the ventilation appeared to be clinically adequate.

The inspiratory and expiratory pressures generated by the Hayek Oscillator fell short of the target values the device was set at. This was probably due to an insufficiently powerful engine to quickly move the large amount of air in the size 10 cuirass. The advertised pressure limitations of −80 to +80 cm H2O probably pertains to a smaller-sized cuirass.

**Limitations**

The intracuirass pressures generated by the Hayek Oscillator did not meet the optimum levels proposed by Lin et al. We did not measure or standardize chest wall displacement, intrathoracic pressure, and minute ventilation. The results were biased against the cuirass because this was a nonrandomized crossover study with the Hayek Oscillator always being studied after standard CPR. The long duration between patient collapse and initiation of the cuirass may have caused the cuirass to underperform because of increasing chest wall stiffness that occurs with increasing downtime. Additionally, thoracoabdominal trauma was not assessed.

**Conclusion**

Biphasic extrathoracic pressure CPR significantly increases the coronary perfusion pressure when compared with standard CPR. Additionally, there is a trend for improved systemic perfusion as indicated by the improved venous to arterial Pco2.
gradient, although this is not reflected by significant decreases in the OER. Oxygenation and ventilation appear to be adequately maintained, thus obviating the need for positive pressure ventilation with its associated risks.

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