COMPARISON OF CONVENTIONAL AND SIMULTANEOUS COMPRESSION-VENTILATION CARDIOPULMONARY RESUSCITATION IN PIGLETS

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To determine the mechanism of cardiac output and hemodynamic changes during cardiopulmonary resuscitation (CPR), we performed 60 min of CPR using a mechanical resuscitator ("Thumper", MII, USA) in 20 piglets (13.7 ± 1.2 kg) following cardiac arrest induced by intravenous injection of KCl. Conventional CPR (C-CPR), i.e., 60 external chest compressions (60 psi force, 3 cm deep, and 50% duration) and 12 ventilations (following every 5th compression, with peak airway pressure of 30 cmH2O) per minute, was performed in 10 piglets; and simultaneous compression and ventilation CPR (SCV-CPR), i.e., 60 external chest compressions of the same magnitude, simultaneously with 60 ventilations (with peak airway pressure of 60 cmH2O) per minute, was performed in the other 10 piglets. Cardiac output in C-CPR and SCV-CPR was maintained near 70% or more of baseline throughout the CPR. Systemic vascular resistance dropped to below 50% of baseline. Systolic, mean and diastolic arterial pressures were maintained above 70, 40, and around 20 mmHg, respectively, during the first 30 min of CPR. Central venous pressure rose after arrest and subsequent CPR to above 25 mmHg, and remained high in SCV-CPR, but declined thereafter in C-CPR. Aortic diastolic minus right atrial diastolic pressure was around 15 mmHg early in CPR and dropped to almost zero thereafter. Serial arterial blood gas analyses showed a significant deterioration after 20 min of SCV-CPR. All but one piglet in the SCV-CPR group died after 60 min of CPR. Postmortem examination revealed that pulmonary barotrauma was more extensive and severe in SCV-CPR than in C-CPR. These results suggest that cardiac pumping is the primary mechanism of cardiac output during CPR in piglets. The mechanical resuscitator consistently achieved adequate cardiac output and systolic arterial pressure. Pulmonary barotrauma may have a negative impact on CPR results.

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CARDIAC pump theory, which states that cardiac output (CO) during closed chest cardiopulmonary resuscitation (CPR) is a result of direct compression of the heart between the sternum and the vertebra, has been widely accepted since 1960! Thoracic pump theory, based on Criley's observation; which states that a cough could induce CO in patients with ventricular fibrillation, due to the increased intrathoracic pressure pushing blood out of the heart, has also been proposed and studied in different animal models. The mechanisms of CO during CPR re-

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TABLE 1 CHANGES IN HEMODYNAMIC PARAMETERS DURING C-CPR AND SCV-CPR

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>CO (%)</th>
<th>SVR (mmHg)</th>
<th>SAP (mmHg)</th>
<th>MAP (mmHg)</th>
<th>DAP (mmHg)</th>
<th>CVP (mmHg)</th>
<th>ALD-RAPD (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>5 min</td>
<td>88.2±22.5</td>
<td>84.1±13.3</td>
<td>84.1±12.0</td>
<td>84.1±12.0</td>
<td>84.1±12.0</td>
<td>84.1±12.0</td>
<td>84.1±12.0</td>
</tr>
<tr>
<td>10 min</td>
<td>68.9±8.1</td>
<td>40.0±6.3</td>
<td>24.0±1.7</td>
<td>19.5±5.6</td>
<td>23.5±3.2</td>
<td>24.2±2.6</td>
<td>24.4±2.0</td>
</tr>
<tr>
<td>15 min</td>
<td>72.3±13.3</td>
<td>43.7±15.9</td>
<td>45.8±11.5</td>
<td>45.8±11.5</td>
<td>45.8±11.5</td>
<td>45.8±11.5</td>
<td>45.8±11.5</td>
</tr>
<tr>
<td>20 min</td>
<td>76.2±16.0</td>
<td>40.0±6.3</td>
<td>24.0±1.7</td>
<td>19.5±5.6</td>
<td>23.5±3.2</td>
<td>24.2±2.6</td>
<td>24.4±2.0</td>
</tr>
<tr>
<td>30 min</td>
<td>76.2±16.0</td>
<td>40.0±6.3</td>
<td>24.0±1.7</td>
<td>19.5±5.6</td>
<td>23.5±3.2</td>
<td>24.2±2.6</td>
<td>24.4±2.0</td>
</tr>
<tr>
<td>60 min</td>
<td>74.2±15.8</td>
<td>40.0±6.3</td>
<td>24.0±1.7</td>
<td>19.5±5.6</td>
<td>23.5±3.2</td>
<td>24.2±2.6</td>
<td>24.4±2.0</td>
</tr>
</tbody>
</table>

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CARDIOPULMONARY RESUSCITATION IN PIGLETS

A total of 20 piglets with a mean body weight of 13.7±1.2 kg were chosen. After anesthesia with pentobarbital sodium (20 mg/kg, iv), each piglet was intubated and ventilated with room air by a volume-cycled respirator (Harvard 607), while being maintained in the supine position. Intravascular indwelling catheters were inserted, respectively, into the (1) femoral artery for blood sampling, (2) right atrium via the femoral vein for central venous pressure reading, and (3) ascending aorta via the carotid artery to monitor arterial blood pressure. A CO monitor probe (L3000, Lawrence Medical, USA) was also placed into the esophagus at the level at which an optimal signal of aortic blood flow was detected. Thus, relative CO and systemic vascular resistance were monitored as a percentage of the pre-CPR baseline value. Baseline values of all of the parameters were recorded before the induction of cardiac arrest and subsequent CPR. Seven milliliters of 15% KCl solution was then injected intravenously to induce ventricular fibrillation and cardiac arrest. A mechanical resuscitator ("Thumper", Michigan Instruments Inc., USA) was used to perform 2 different types of CPR.

1. Conventional CPR (C-CPR): Five chest compressions of 60 psi, 3 cm deep and 50% duration, followed by one ventilation with pure oxygen, utilizing a Venturi-equipped ventilating device. The peak airway pressure was set at 30 cmH2O, with a rate of 60 compressions and 12 ventilations per minute.

2. Simultaneous Compression and Ventilation CPR (SCV-CPR): Simultaneous external chest compression of the same magnitude as of in C-CPR, and ventilation of 60 cmH2O peak airway pressure at a rate of 60 per minute.

Each type of CPR was performed for 60

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min in 10 piglets each. The hemodynamic parameters monitored and recorded were relative CO, relative systemic vascular resistance, arterial blood pressure, central venous pressure, and serial arterial blood gas analyses at 5, 10, 20, 30 and 60 min after CPR. After 60 min of C-CPR or SCV-CPR, vasoressor agents, such as epinephrine, electric defibrillation, sodium bicarbonate, and/or xylocaine were administered to revive the piglets. Postmortem examination was done in all of the piglets to check the lungs in terms of weight, gross appearance and injuries.

Statistics
All of the data were expressed as the mean±standard error. Groups were compared using Student's t-test. A p value of less than 0.05 was considered as significant.

RESULTS
I. Hemodynamic Parameters (Table I)
1. Relative Cardiac Output: Around 58% to 88% (mean 72.1±15.0%) of baseline CO was achieved and maintained throughout the 60 min of CPR in both groups, with no significant difference (Fig. 1).
Cardiopulmonary Resuscitation in Piglets

2. Relative Systemic Vascular Resistance: Relative systemic vascular resistances dropped precipitously to 34–51% of baseline values, and further declined to near or below 20% at the final stage in all of the piglets, although this effect was significantly more pronounced in the SCV-CPR group (Fig. 1).

3. Arterial Blood Pressure: Systolic arterial pressure was maintained at 75–98 mmHg, mean arterial pressure at 44–55 mmHg, and diastolic arterial pressure at 20–24 mmHg in both groups during the first 20 min of CPR. In all of the piglets, the pressures started to drop thereafter, and reached very low levels at the end of CPR.

4. Central Venous Pressure: Central venous pressure in all of the piglets rose almost immediately after arrest and remained above 25 mmHg during the first 10 min of CPR. This value continued to increase, to above 30 mmHg, in the SCV-CPR group, but declined at the final stage to below 15 mmHg in the C-CPR group.

5. Aortic Diastolic Minus Right Atrial Diastolic Pressure: This value was only about 15 mmHg early in CPR, and dropped further to around 10 mmHg at 30 min, and to almost zero at 60 min, in both groups.

II. Arterial Blood Gas Analysis (Table II)
1. pH: pH values were maintained above 7.50 before CPR in both groups. After the initiation of CPR, pH started to drop in both groups, and from 20 min on, this decline was more significant in the SCV-CPR group than in the C-CPR group (p<0.05). After 30 min of CPR, pH had declined to below 7.0 in the SCV-CPR group (Fig. 2A).

2. PCO2: Removal of CO2 was adequate with C-CPR, but was inadequate with SCV-CPR. After 20 min of CPR, PCO2 in the SCV-CPR group was above 50 mmHg (vs C-CPR, p<0.05), and continued to rise thereafter (Fig. 2B).

3. PO2: Oxygenation was generally adequate, but was better in the C-CPR group than in the SCV-CPR group during the first 30 min of CPR (p<0.05). PO2 declined to below 50 mmHg in both groups at 60 min of CPR (Fig. 2C).

4. [HCO3-]: Bicarbonate values at the initiation of CPR were 19.4±1.1 mEq/L, ranging from 16.6 to 22.6 mEq/L, in both groups, but decreased soon thereafter. This decrease was more pronounced in the C-CPR group than in the SCV-CPR group (p<0.05) after 10 min of CPR (Table II).

5. Base Excess: Base excess values kept...
TABLE III  RESULTS OF POSTMORTEM EXAMINATIONS OF PIGLETS FOLLOWING C-CPR AND SCV-CPR

<table>
<thead>
<tr>
<th></th>
<th>C-CPR (N=10)</th>
<th>SCV-CPR (N=9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Thoracic cage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>rib fracture</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>2. Lungs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>gross appearance</td>
<td>light</td>
<td>heavier, edematous</td>
</tr>
<tr>
<td>petechial ecchymosis</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>bullae formation (rupture)</td>
<td>0* (0)</td>
<td>4* (1)</td>
</tr>
<tr>
<td>3. Heart</td>
<td></td>
<td></td>
</tr>
<tr>
<td>minor abrasion</td>
<td>10</td>
<td>9</td>
</tr>
</tbody>
</table>

*p<0.05, C-CPR vs SCV-CPR.

worsening, continued to decrease from −10 mEq/L, after 10 min of CPR, to less than −25 mEq/L at 60 min in both groups (Table II).

III. Electrolytes
Serum potassium levels: [K+] values were initially above 10 mEq/L and remained above 7.6 mEq/L throughout CPR in both groups (Table II).

IV. Postmortem Examinations (Table III)
All but one piglet in the SCV-CPR group died after 60 min of CPR. Minor abrasion wounds were found on the surface of the heart in each piglet. Rib fracture was noted in 2 piglets in each group. The lungs in the SCV-CPR group were heavier and edematous, and ecchymosis (6 piglets), bullae formation (n=4), and rupture of lung tissue (n=1) were also observed. The lungs in the C-CPR group only showed ecchymosis in 3 piglets.

DISCUSSION
The thoracic pump theory of CO during CPR claims that intrathoracic pressure fluctuations could push blood out of the heart toward extrathoracic vessels, with the heart acting only as a conduit. If this theory were valid, then the increased intrathoracic pressure created by SCV-CPR should produce more greater CO. However, the present study showed no difference in the CO values induced by SCV-CPR and C-CPR, which suggests that, in this small animal model (<15 kg), the cardiac pump theory, in which blood is squeezed out of the heart by cardiac deformation, is more likely to be the mechanism of CO during CPR. Babbs et al. demonstrated that SCV-CPR produced a significantly greater CO than C-CPR in large dogs weighing 23–37 kg, but not in small dogs weighing 6–9 kg, and concluded that animal models of different sizes responded differently to mechanical systems for artificial circulation, and suggested that the use of small animals would be more appropriate for research in pediatric resuscitation. In contrast, von Planta et al. showed a fairly good resuscitation rate with SCV-CPR in rats. The relative superiority of SCV-CPR to C-CPR remains controversial. Our study suggested that SCV-CPR may not be any more useful than C-CPR in infants and children.

With a mechanical resuscitator, such as that used in this study, CO could reach 70% or more of the pre-arrest value and be maintained without fatigue. Furthermore, chest trauma incurred by the mechanical CPR appears to be less than that due to manual CPR. Although blood pressure does not necessarily imply blood flow, mean arterial pressure is more likely to reflect flow than systolic arterial pressure, and mechanical chest compression is superior to manual chest compression in generating high mean arterial pressure. Mean arterial pressure of less than 50 mmHg has been thought to be incompatible with brain survival. However, during CPR, the brain enjoys a better blood flow, around 90% of normal, than other organs since the ejected flow is directed cephalically. SCV-CPR in large dogs (>20 kg) may produce even more cerebral blood flow than C-CPR. However, although SCV-CPR might be expected to maintain cerebral function, it may not be able to maintain viability of the heart.

Increased carotid blood flow is also not necessarily related to CPR survival. Diastolic arterial pressure and coronary blood flow have been considered to be more important in predicting the success of CPR. As shown in this study, systemic vascular resistance dropped significantly soon after cardiac arrest, as did diastolic arterial pressure. Diastolic arterial pressure
of less than 30 mmHg does not appear to be associated with survival. Therefore, maintenance of adequate diastolic arterial pressure is important for survival. Aortic diastolic minus right atrial diastolic pressure has been considered the coronary perfusion pressure, thus reflecting coronary blood flow. Although not shown in this study and others, coronary perfusion pressure has been reported to be lower with SCV-CPR than with C-CPR. This may be explained, at least in part, by the higher central venous pressure, observed during SCV-CPR. Survival after CPR is possible only if the coronary perfusion pressure is above 20 mmHg.

Using radionuclide microsphere techniques, coronary blood flow during CPR has been shown to be only 10–35% of normal, and slightly more with mechanical resuscitation. The administration of vasopressors, e.g. epinephrine, or other adjunct maneuvers to increase diastolic arterial pressure has been thought to increase coronary perfusion pressure, and therefore coronary blood flow. Since coronary blood flow occurs primarily during the relaxation phase of CPR, the duration of compression should not be too long (<50%). Increasing the applied compressive force (from 60 to 120 psi) may also enhance coronary blood flow (from 12% to 50%).

Barbiturates are believed to induce vasodilation, thereby decreasing diastolic arterial pressure and coronary blood flow. Therefore, Maier et al. suggested that narcotics, e.g. morphine, may be more useful for anesthetizing animals in the evaluation of cardiovascular physiology. Sharff et al. demonstrated that CO, and probably survival as well, decreased with the duration of CPR. In fact, the hemodynamic data observed during the first 20 min of CPR in this study suggest that there may be enhanced survival if additional advanced life supports are administered within that time. The fact that only one piglet in the SCV-CPR group survived after 60 min of CPR was not sufficient to create a significant difference from the C-CPR group. Further study is required to clarify this point.

Chandra et al. reported that oxygenation and removal of CO2 were adequate during the first 10 min of CPR, as shown in the present study. CO2 retention gradually increased after 20 min of SCV-CPR. Although oxygenation was still adequate, it was not proportional to the inspired pure oxygen. Decreased pulmonary compliance after cardiac arrest and gradual development of lung edema after CPR, especially in SCV-CPR, may explain the changes, as may the gross findings of edematous lungs in post-mortem examination of the SCV-CPR group. Progressive acidosis was probably due to prolonged tissue hypoperfusion, as the blood flow during CPR was redistributed among the major organs.

The high peak airway pressure of 60 cmH2O in SCV-CPR, although less than that which has been reported to inevitably cause pulmonary damage, may have had a negative impact on the pulmonary function in this small animal model. Pulmonary barotrauma caused by increased intrathoracic pressure should be considered and avoided in CPR practice and further study. Intravenous KCl solution was useful in inducing cardiac arrest in piglets. Its use in related physiological studies on CPR should be investigated further, in view of the vasodilatory effect of K+ which causes markedly reduced systemic vascular resistance.

In conclusion, in this small animal model (<15 kg), the mechanism of blood flow during CPR favors the cardiac pump theory, and CO can be maintained at 70% or more of pre-arrest values, without fatigue, by using a mechanical resuscitator. If vasopressors or other adjunct maneuvers could be used concomitantly to increase diastolic arterial pressure and enhance coronary blood flow, the chances of survival after CPR might be increased. The peak airway pressure during CPR should not be too high, since subsequent pulmonary barotrauma may have a negative impact on the results of CPR. Further studies on CPR should be conducted in animals of different types and sizes under various conditions, before a better method of CPR can be defined and applied to human beings.

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