Vest inflation without simultaneous ventilation during cardiac arrest in dogs: improved survival from prolonged cardiopulmonary resuscitation

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ABSTRACT Myocardial and cerebral blood flow can be generated during cardiac arrest by techniques that manipulate intrathoracic pressure. Augmentation of intrathoracic pressure by high-pressure ventilation simultaneous with compression of the chest in dogs has been shown to produce higher flows to the heart and brain, but has limited usefulness because of the requirement for endotracheal intubation and complex devices. A system was developed that can produce high intrathoracic pressure without simultaneous ventilation by use of a pneumatically cycled vest placed around the thorax (vest cardiopulmonary resuscitation [CPR]). The system was first tested in a short-term study of the maximum achievable flows during arrest. Peak vest pressures up to 380 mm Hg were used on eight 21 to 30 kg dogs after induction of ventricular fibrillation and administration of epinephrine. Microsphere-determined myocardial blood flow was 108 ± 17 ml/min/100 g (100 ± 16% of prearrest flow) and cerebral flow was 51 ± 12 ml/min/100 g (165 ± 39% of prearrest). Severe lung or liver trauma was noted in three of eight dogs. If peak vest pressure was limited to 280 mm Hg, however, severe trauma was no longer observed. A study of the hemodynamics during and survival from prolonged resuscitation was then performed on three groups of seven dogs. Vest CPR was compared with manual CPR with either conventional (300 newtons) or high (430 newtons) sternal force. After induction of ventricular fibrillation, each technique was performed for 26 min. Defibrillation was then performed. After 20 min of resuscitation, vest CPR produced a myocardial flow of 54 ± 13 ml/min/100 g (40 ± 9% of prearrest flow) and a cerebral flow of 37 ± 4 ml/min/100 g (99 ± 11% of prearrest). With conventional sternal force, manual CPR produced lower myocardial and cerebral flows than did the vest method (p < .04), and resulted in fewer next-day survivors (7/7 for vest vs 1/7 for manual, p < .003). With high sternal force, flows were similar to those obtained with the vest, but more dogs had severe rib or liver trauma (0/7 for vest vs 4/7 for manual, p < .04), and there were still fewer survivors than with the vest method (3/7, p < .04 vs vest). Thus, at very high pressures, vest CPR can generate essentially normal myocardial and cerebral flow, but can also produce severe trauma. At lower pressures, vest CPR can improve survival after cardiac arrest, while producing less trauma than manual CPR performed with sufficient compression to generate comparable flows. Vest CPR warrants study in man as a potential means for augmenting flow during cardiac arrest without the need for endotracheal intubation and simultaneous ventilation.

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The means by which conventional cardiopulmonary resuscitation (CPR) with sternal displacement maintains blood flow to the heart and brain during cardiac arrest — whether by induced fluctuations in intrathoracic pressure only, or also by direct cardiac compression — remains a subject of controversy.1–6 Recent studies have shown conclusively, however, that fluctuations in intrathoracic pressure alone are sufficient to maintain blood flow.7, 8 With a rise in intrathoracic pressure, there is a similar rise in the pressure in all intrathoracic vascular structures. This pressure is transmitted from intrathoracic to extrathoracic arteries. Competent venous valves9, 10 and extrathoracic venous compliance prevent the full transmission of intrathoracic venous pressure to extrathoracic veins. The resulting difference in pressure between the extrathoracic arteries and veins.
causes blood to move out of the thorax and into the extrathoracic arterial system.

Since intrathoracic pressure is known to produce blood flow, uniform compression of the thorax deserves investigation as a more effective and safer means of CPR than current manual compression techniques. The volume of the thorax would be more efficiently reduced, and intrathoracic pressure thereby increased, by a uniform, small decrease in its dimensions than by distortion at one point from a relatively large sternal displacement. Furthermore, severe chest wall trauma and abdominal and thoracic visceral injury frequently occur during conventional CPR, and avoidance of trauma, as well as maintenance of perfusion, is obviously a goal of resuscitative efforts.

In previous studies, high-pressure ventilation has been used with simultaneous sternal compression or with vest inflation to raise intrathoracic pressure to levels above those obtained conventionally. Endotracheal intubation, however, and the complex devices needed to deliver simultaneous high-pressure ventilation safely, make these techniques cumbersome and have limited their usefulness.

We developed a system that can generate high intrathoracic pressure without the need for simultaneous ventilation. The system uses a vest that contains a bladder that is rapidly inflated and deflated (vest CPR). Prior studies with vest CPR showed that blood flow to vital organs was related to peak vest pressure, the duration of vest inflation per cycle, and arterial tone. Recent preliminary studies showed that severe lung or liver trauma could be produced when the peak vest pressure was greater than 300 mm Hg, that there was no severe trauma if peak vest pressure was limited to 280 mm Hg, that the design of the vest influenced the amount of trauma produced, and that a cycle rate of 60/min with inflation for 45% of the cycle produced optimal flow. When there was a mean diastolic aortic-to-right atrial pressure difference of 25 to 35 mm Hg, myocardial flow was approximately 50% of that before arrest at vest pressures of 200 to 280 mm Hg. At these vest pressures, cerebral flow was near prearrest levels. Our intention in this study was to compare this technique with manual external CPR.

Prior studies of manual CPR showed that vital organ blood flow was related to peak sternal force, the duration of compression per cycle, and as with vest CPR, arterial tone. Recent preliminary studies also showed that severe trauma appeared to be associated with peak sternal forces that were greater than 450 newtons (N; 1 N = 0.102 kg-force = 0.225 pound), or that produced peak aortic pressures greater than 120 mm Hg. A compression rate of 60/min with compression for 45% of the cycle produced optimal flow. CPR performed with lateral chest compression produced less flow and more trauma than CPR performed with anterior-posterior sternal displacement. The compliances of the canine and human chests were shown to be similar, and peak sternal force correlated with peak displacement. Studies in man showed that a peak sternal force of ~300 N is generally used during manual CPR.

Based on these data from studies of manual and vest CPR, we performed two experiments. We first determined the maximum achievable myocardial and cerebral flows from the vest system. We then tested the efficacy and safety of the vest system during prolonged resuscitation in dogs. Twenty-four hour survival, vital organ perfusion, neurologic status, and trauma were compared in dogs treated with vest CPR or manual CPR with conventional compression force, or with manual CPR with high compression force. Epinephrine was administered because it has been established that arterial collapse can limit flow during prolonged CPR, and that arterial collapse is reversed by epinephrine.

**Methods**

**Preparation.** Twenty-nine dogs weighing 21 to 30 kg were anesthetized with pentobarbital sodium (15 mg/kg iv). Supplemental pentobarbital was administered as needed. An endotracheal tube was inserted, and the dogs were ventilated with room air by a volume-cycled respirator (Harvard 607). From a femoral catheter, micromanometer-tipped catheters (Millar PC-470) zeroed and calibrated at 37°C were placed into the right atrium and ascending aorta, a pigtail catheter for microsphere injection was placed into the left ventricle, and a pacing catheter was placed into the right ventricle. From bilateral axillary catheters, catheters were placed into each brachiocephalic artery for collection of blood during microsphere injection. In each of six dogs used in the study of maximum blood flow during vest CPR, a fluid-filled catheter was placed into the right common carotid artery via a side branch, and a 16-gauge cannula was placed into the lateral cerebral ventricle through a burr hole for measurement of intracranial pressure. All dogs were studied while in the supine position, and were given 0.5 to 1 liter of normal saline intravenously to achieve mean right atrial pressures of 3 to 5 mm Hg. This fluid replaced losses during surgery and restored the dogs to a euvolemic state.

The first injection of microspheres was made before cardiac arrest for measurement of prearrest regional blood flow by methods that have been previously described. Ventricular fibrillation was then induced by a 60 Hz alternating current applied to the pacing catheter. CPR was started within 30 sec after fibrillation commenced.

**Protocols**

**Maximum blood flow during vest CPR.** Vest CPR was performed on a group of eight dogs with the use of inflatable vest (PhysioControl) placed around the thorax. The vest was inflated and deflated cyclically by a programmable pneumatic generator. The rate of inflation-deflation cycles was 60/min, with inflation for 45% of the cycle. The rise time of vest pressure was 100 to 150 msec. Peak vest pressure was set to a maximum of...
380 mm Hg. Ventilation was performed after every fifth compression with 100% O₂ by a Venturi-equipped ventilating device (Michigan Instruments) to a peak airway pressure of 30 to 40 mm Hg. The final ventilating mixture was approximately 60% oxygen. Just after induction of ventricular fibrillation, epinephrine was administered as a 1 mg bolus into the left ventricle, followed by a constant intravenous infusion at 4 μg/kg/min by a syringe pump (Harvard 600). Microspheres were injected at least 6 min after resuscitation began.

Vest vs manual CPR during prolonged cardiac arrest. Vest CPR was performed on a group of seven randomly selected dogs with use of the same vest system as above. Peak vest pressure, however, was set to a maximum of 280 mm Hg, or to a level that produced a mean diastolic aortic–to–right atrial pressure difference of 25 to 35 mm Hg.

Manual CPR was performed on two additional groups of randomly selected dogs. In one group of seven dogs, peak sternal force was limited to 300 N (conventional-force manual CPR). In another group of seven dogs, sternal force was increased to 430 N (high-force manual CPR), or to a level that produced the same 25 to 35 mm Hg mean diastolic aortic–to–right atrial pressure difference as vest CPR. Manual CPR was performed by the investigators using computer-generated sounds to time chest compression and release. The rate of compression was 60/min, with compression for approximately 45% of the cycle.

Because a previous study had shown a large difference in survival between dogs treated with conventional CPR and those treated with vest CPR with simultaneous ventilation, and because the latter system produced perfusion pressures that were equal or inferior to those produced with our new vest system, we expected large differences in survival of dogs undergoing manual and vest CPR. Therefore, relatively small groups of dogs were used.

During manual and vest CPR, ventilation was performed after every fifth compression with the same ventilator described above. Ten minutes after fibrillation, epinephrine was administered as a 1 mg bolus into the left ventricle, followed by a constant intravenous infusion at 4 μg/kg/min. Microspheres were injected 2, 11, and 20 min after fibrillation. Two milliequivalents per kilogram of sodium bicarbonate were given intravenously after 24 min of resuscitation, and defibrillation was performed 2 min later. Defibrillation was performed in a 200, 200, and 400 J sequence. Subsequent defibrillation pulses were 400 J, but no more than five pulses were used if there was no return of spontaneous circulation.

After defibrillation, acute medical care, including mechanical ventilation, fluids, antiarrhythmic agents, and pressor agents, was given as needed for up to 3 hr. Morphine was given if there was any evidence of pain. Arterial pH was corrected with additional sodium bicarbonate. Survivors were killed after 24 hr.

Neurologic status was assessed by standard criteria. Survivors were noted to have a level of consciousness that was one of the following: normal, clouded, stuporous, or comatose. It was also noted whether survivors were ambulatory or not.

All dogs were subjected to full autopsies at the time of death. The autopsy protocol included thorough gross and histologic examination of the thorax and abdomen. The brain was examined only in survivors. Standard methods were used.

A three-grade system was used to describe the trauma noted in each of the major organs involved. Severe trauma was defined as gross and/or microscopic abnormalities that were expected to be responsible for either life-threatening rapid clinical deterioration or death. Intermediate trauma was defined as any trauma present that was not severe, and consisted of gross and/or microscopic abnormalities that were not expected to be directly responsible for rapid clinical deterioration or death. The third grade of trauma was defined as “none” (no abnormality was present).

Severe trauma to the major organs consisted of the following: ribs, flail chest from bilateral rib fractures or costochondral separations; liver, liver laceration with hemoperitoneum; lungs, gross laceration of a lung, or laceration of a large pulmonary artery with dissection into a bronchus; heart, gross laceration of the heart or another major mediastinal structure and marked subendocardial, epicardial, or intramyocardial hemorrhage; brain, laceration of an intracranial vessel, or significant subarachnoid or subdural hemorrhage.

Measurements. Sternal force was measured by a precision force transducer system (PCB Piezotronics) that has been previously described.

Pressure and force signals were recorded on an eight-channel Gould-Brush recorder, and were digitized and stored by a microcomputer-based data acquisition system.

Regional blood flows were determined with 15 ± 3 μm diameter radioactive microspheres (New England Nuclear). The isotopes used were 141Ce, 113Sn, 103Ru, and 46Sc. Reference samples were obtained from the two arterial sites with a dual syringe pump (Harvard 600). Withdrawal was started 15 sec before injection, and continued for 6 min after injection to allow for full washout of spheres. Withdrawal was at 3.8 ml/min for prearrest samples, and at 1.9 ml/min for postarrest samples. The use of blood flow measurements obtained with radioactive microspheres for studies of CPR has been previously validated.

Statistical analysis. Differences in pressures and flows generated by manual and vest CPR were tested by analysis of variation with orthogonal contrasts. Differences in survival and trauma were tested with contingency table analysis. The contributions of flow and trauma to survival were tested with stepwise multiple regression. The correlation between myocardial flow and myocardial perfusion pressure was tested with regression analysis. Differences in paired data were tested with the two-tailed Student t test. A probability less than .05 was considered indicative of a statistically significant difference.

Results

Maximum blood flow during vest CPR. Figure 1 is a record obtained during vest CPR showing the high arterial and myocardial perfusion pressure (aortic minus right atrial diastolic pressure) that can be obtained without simultaneous ventilation. In addition, the carotid pressure was much higher than the intracranial pressure, giving a large pressure difference for cerebral perfusion. Figure 2 illustrates myocardial and cerebral flow data for individual dogs. Mean pressure and flow data are listed in table 1. Essentially normal myocardial and cerebral flows were achieved. Three of the eight dogs had severe lung or liver trauma.

Vest vs manual CPR during prolonged cardiac arrest. All dogs treated with vest CPR survived. Three dogs treated with high-force manual CPR survived, but only one treated with conventional-force manual CPR survived (table 2). Both when considered separately and combined, use of the two forms of manual CPR resulted in fewer survivors than did use of vest CPR (table 2).
The dogs treated with vest CPR required 2.6 ± 0.7 defibrillation pulses, while the dogs treated with high-force and conventional-force manual CPR required 4.0 ± 1.1 and 4.4 ± 0.3 pulses, respectively (p = NS).

Records obtained during vest and high-force manual CPR are illustrated in figure 3. These tracings were recorded after the administration of epinephrine had started, and show the similarities in peak aortic and atrial systolic pressures during CPR by these two methods.

There were no differences among the three groups with respect to prearrest pressures or flows (table 3;

TABLE 1
Pressure and flow during the maximization of flow protocol

<table>
<thead>
<tr>
<th></th>
<th>Prearrest control</th>
<th>Vest CPR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean aortic pressure (mm Hg)</td>
<td>133 ± 4</td>
<td>108 ± 4</td>
</tr>
<tr>
<td>Aortic systolic pressure (mm Hg)</td>
<td>147 ± 4</td>
<td>155 ± 7</td>
</tr>
<tr>
<td>Mean right atrial pressure (mm Hg)</td>
<td>5 ± 1</td>
<td>66 ± 4</td>
</tr>
<tr>
<td>Right atrial systolic pressure (mm Hg)</td>
<td>9 ± 2</td>
<td>135 ± 7</td>
</tr>
<tr>
<td>Aortic minus right atrial mean diastolic pressure (mm Hg)</td>
<td>127 ± 4</td>
<td>54 ± 3</td>
</tr>
<tr>
<td>Carotid minus intracranial mean pressure (mm Hg)</td>
<td>106 ± 10</td>
<td>67 ± 10</td>
</tr>
<tr>
<td>Myocardial flow (ml/min/100 g)</td>
<td>108 ± 14</td>
<td>108 ± 17</td>
</tr>
<tr>
<td>Cerebral flow (ml/min/100 g)</td>
<td>31 ± 3</td>
<td>51 ± 12</td>
</tr>
<tr>
<td>Vest pressure (mm Hg)</td>
<td>—</td>
<td>323 ± 24</td>
</tr>
</tbody>
</table>

Values are mean ± SEM. n = 8 (n = 6 for intracranial pressure).

FIGURE 2. Regional flows from the protocol for maximizing flow during vest CPR. Control flows are prearrest measurements. The flows from individual dogs are connected. Mean flows (with SEMs) are shown to the left and right of the data from individual dogs.

TABLE 2
Summary of findings on survival and trauma from manual and vest CPR

<table>
<thead>
<tr>
<th></th>
<th>Survival</th>
<th>Severe trauma^a</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Vest</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>CF manual</td>
<td>1</td>
<td>6^b</td>
</tr>
<tr>
<td>HF manual</td>
<td>3</td>
<td>4^p</td>
</tr>
<tr>
<td>CF + HF manual</td>
<td>4</td>
<td>10^b</td>
</tr>
</tbody>
</table>

Values are number of dogs.
CF = conventional-force, HF = high-force, CF + HF = combined conventional-force and high-force.
^aSevere trauma consisted of liver laceration with hemoperitoneum or flail chest.
^b p < .003 vs vest; ^p = .055 vs vest; ^p < .04 vs vest.
These pressures and flows are similar to those reported previously. There were also no differences in pressures (table 3) or flows (figure 4) in the vest and high-force manual CPR groups, except at the 20 min measurement, at which time vest CPR produced more myocardial flow than did high-force manual CPR. There was a marked difference in peak and mean vascular pressures in dogs undergoing conventional-force manual CPR and those undergoing both vest CPR and high-force manual CPR (table 3). The increased difference between aortic mean diastolic pressure and right atrial mean diastolic pressure (myocardial perfusion pressure) with vest CPR and high-force manual CPR (table 3), correlates with the higher myocardial flows produced by these two techniques compared with flows obtained with conventional-force manual CPR (figure 4). Conventional-force manual CPR also produced less cerebral flow than did vest CPR at all three times that flow was measured, and less cerebral flow than high-force manual CPR at 2 min and at 20 min (figure 4).

Vest CPR produced 40% of prearrest myocardial flow and 99% of prearrest cerebral flow at 20 min, while high-force manual CPR produced 18% and 63%, respectively. Conventional-force manual CPR produced only 5% of prearrest myocardial flow and 15% of cerebral flow at 20 min (figure 4).

Epinephrine caused an increase in myocardial flow in dogs undergoing vest CPR and high-force manual CPR. Flows increased from 21.6 and 27.6 ml/min/100 g, respectively, at 2 min of resuscitation to 68.2 and 67.4 ml/min/100 g (p < .02), at 11 min (figure 4). Epinephrine was started at 10 min.

The myocardial flow produced by high-force manual CPR decreased from 67.4 ml/min/100 g at 11 min of resuscitation to 26.2 ml/min/100 g at 20 min of resuscitation (p < .02), but there was no comparable fall in the myocardial flow produced by vest CPR (figure 4).

In the vest CPR group, five dogs were neurologically normal and ambulatory, one dog was near normal but had difficulty walking, and one was comatose. In the high-force manual CPR group, one dog was neurologically normal and ambulatory, one dog was near normal but had difficulty walking, and one was stuporous. The lone survivor in the conventional-force manual CPR group was neurologically normal and ambulatory.

There was no severe trauma in the dogs treated with vest CPR. Severe trauma was present, however, in two
TABLE 3
Hemodynamics during manual and vest CPR

<table>
<thead>
<tr>
<th></th>
<th>Prearrest control</th>
<th>2 min</th>
<th>11 min</th>
<th>20 min</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean aortic pressure (mm Hg)</strong></td>
<td>V 142±7</td>
<td>55±5</td>
<td>76±5</td>
<td>67±3</td>
</tr>
<tr>
<td></td>
<td>CM 135±6</td>
<td>22±3^</td>
<td>27±5^</td>
<td>28±5^</td>
</tr>
<tr>
<td></td>
<td>HM 135±7</td>
<td>45±4</td>
<td>65±5</td>
<td>47±3</td>
</tr>
<tr>
<td><strong>Aortic systolic pressure (mm Hg)</strong></td>
<td>V 164±9</td>
<td>81±6</td>
<td>110±9</td>
<td>90±6</td>
</tr>
<tr>
<td></td>
<td>CM 156±6</td>
<td>32±4^</td>
<td>45±8^</td>
<td>47±10^</td>
</tr>
<tr>
<td></td>
<td>HM 159±8</td>
<td>65±5</td>
<td>97±6</td>
<td>74±6</td>
</tr>
<tr>
<td><strong>Aortic diastolic pressure (mm Hg)</strong></td>
<td>V 125±6</td>
<td>36±3</td>
<td>49±4</td>
<td>42±2</td>
</tr>
<tr>
<td></td>
<td>CM 118±5</td>
<td>15±3^</td>
<td>16±3^</td>
<td>16±2^</td>
</tr>
<tr>
<td></td>
<td>HM 115±6</td>
<td>27±3</td>
<td>46±4</td>
<td>30±3</td>
</tr>
<tr>
<td><strong>Mean right atrial pressure (mm Hg)</strong></td>
<td>V 5±1</td>
<td>42±4</td>
<td>53±4</td>
<td>46±4</td>
</tr>
<tr>
<td></td>
<td>CM 4±1</td>
<td>21±3^</td>
<td>22±4^</td>
<td>23±3^</td>
</tr>
<tr>
<td></td>
<td>HM 3±1</td>
<td>35±5</td>
<td>40±4</td>
<td>35±4</td>
</tr>
<tr>
<td><strong>Right atrial systolic pressure (mm Hg)</strong></td>
<td>V 9±2</td>
<td>74±6</td>
<td>96±10</td>
<td>84±6</td>
</tr>
<tr>
<td></td>
<td>CM 9±2</td>
<td>33±4^</td>
<td>43±8^</td>
<td>44±9^</td>
</tr>
<tr>
<td></td>
<td>HM 6±1</td>
<td>60±7</td>
<td>87±9</td>
<td>72±8</td>
</tr>
<tr>
<td><strong>Right atrial diastolic pressure (mm Hg)</strong></td>
<td>V 2±1</td>
<td>16±2</td>
<td>18±2</td>
<td>13±2</td>
</tr>
<tr>
<td></td>
<td>CM 1±1</td>
<td>14±2</td>
<td>11±2</td>
<td>9±2</td>
</tr>
<tr>
<td></td>
<td>HM 1±1</td>
<td>7±1</td>
<td>10±2</td>
<td>7±1</td>
</tr>
<tr>
<td><strong>Aortic minus right atrial mean diastolic pressure (mm Hg)</strong></td>
<td>V 138±7</td>
<td>22±4</td>
<td>31±4</td>
<td>29±3</td>
</tr>
<tr>
<td></td>
<td>CM 129±5</td>
<td>2±1^</td>
<td>5±2^</td>
<td>6±3^</td>
</tr>
<tr>
<td></td>
<td>HM 131±7</td>
<td>17±4</td>
<td>34±4</td>
<td>18±3</td>
</tr>
<tr>
<td><strong>Vest pressure (mm Hg)</strong></td>
<td>V —</td>
<td>227±9</td>
<td>219±13</td>
<td>234±14</td>
</tr>
<tr>
<td></td>
<td>CM —</td>
<td>305±6</td>
<td>295±18</td>
<td>305±16</td>
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<tr>
<td><strong>Sternal force (N)</strong></td>
<td>HM —</td>
<td>430±5</td>
<td>345±31</td>
<td>387±20</td>
</tr>
</tbody>
</table>

Values are mean ± SEM. n = 7 for each group. Epinephrine was started at 10 min.
V = vest CPR; CM = conventional-force manual CPR; HM = high-force manual CPR.
^p < .002 vs V, HM.

dogs treated with conventional-force manual CPR and four dogs treated with high-force manual CPR (table 2). After high-force manual CPR, three dogs had flail chests (severe trauma), and two had multiple unilateral rib fractures (intermediate trauma). Four dogs had liver lacerations with hemoperitoneum (severe trauma). All seven dogs had focal pleural petechiae (intermediate trauma) and one dog had diffuse myocardial hemorrhage associated with a blood-tinged pericardial effusion (intermediate trauma).

After conventional-force manual CPR, one dog had severe rib trauma and one dog had severe liver trauma. Two dogs had focal pleural petechiae.

After vest CPR, two dogs had a small subcapsular liver hematoma without hemoperitoneum (intermediate trauma). Three dogs had a fresh thrombus occluding a distal pulmonary arterial branch, but without parenchymal hemorrhage or infarction (intermediate trauma). Two other dogs had focal pleural petechiae.

Data on myocardial flow, cerebral flow, and trauma are shown for survivors and nonsurvivors in figure 5. Stepwise multiple regression performed on these variables indicated that the most potent predictor of survival for all dogs was cerebral flow (p < .001). Among the dogs with higher flows (vest CPR and high-force manual CPR), however, the degree of trauma probably determined which dogs would survive (p < .01).

**Discussion**

At very high vest pressures, vest CPR can produce essentially normal myocardial and cerebral flow for short periods of time, but severe trauma can also occur. At lower vest pressures, however, high levels of flow can be produced without severe trauma. Vest inflation of 200 to 280 mm Hg produced sufficient myocardial and cerebral flow for survival while producing no severe trauma (table 2). Manual CPR, however, could not produce sufficient flow for survival without causing severe trauma, as other studies have shown. Manual CPR with high sternal force produced both high levels of flow and severe trauma (table 2; figure 4). With lower force, trauma was minimized, but vital organ perfusion was inadequate for survival (table 2).
al contusions with intramural hemorrhage or necrosis, tense hemopericardium, or laceration of any major vessel. By these criteria, two of seven animals treated with conventional-force manual CPR, four of seven animals treated with high-force manual CPR, and none of seven animals treated with vest CPR had severe trauma. Rib fractures were not seen in dogs treated with vest CPR, and although small subcapsular liver hematomas were present in two of seven animals, hemoperitoneum was not present. The animals treated with high-force manual CPR, on the other hand, sustained liver lacerations with hemoperitoneum and flail chest that would be considered the cause of death in three of seven animals.

Measured sternal displacement with vest CPR in the dog is less than 0.8 cm, while sternal displacement with manual CPR generating comparable pressure is greater than 4 cm. Since the degree of trauma is probably related to the amount of thoracic distortion, the smaller thoracic displacement during vest CPR would be expected to produce less trauma than manual CPR at comparable vascular pressures.

Previously described complications of external chest compression include rib fractures, hemothorax, hemopericardium, and lacerations of the liver, spleen, stomach, colon, and inferior vena cava. The classification of organ trauma used in this study was modified from that used by Adelson for quantitating myocardial damage from internal cardiac massage. He reported in his description of the pathology of CPR in humans that survivors never had severe myocardial or vascular injuries. In contrast, 40% of nonsurvivors had moderate or severe forms of trauma. Based on this human experience, the following criteria for severe trauma were defined: (1) multiple rib fractures or flail chest, (2) liver laceration with hemoperitoneum, (3) pulmonary hemorrhage or infarction, or (4) myocardial...
Earlier studies comparing conventional and vest CPR in dogs have shown both improved survival\(^1\) and improved flow to the heart and brain\(^1\) with vest CPR. Other studies\(^2\) have shown no difference in carotid flow produced by vest CPR and that produced by mechanical compression of the thorax with simultaneous ventilation. In all of those studies, vest inflation was simultaneous with ventilation and abdominal compression. Mechanical chest compression simultaneous with binding of the abdomen and ventilation has also been used in other studies both with\(^1,14,19\) and without\(^28\) improvement over conventional CPR. Epinephrine has been shown to augment flows when simultaneous ventilation is used.\(^7,29\) Endotracheal intubation and a device to deliver simultaneous pulses of air at different pressures to the airway, to a bladder around the abdomen, and to a vest around the thorax or a mechanical chest compressor were required in those studies. Alternatively, abdominal pressurization could be accomplished by placing a binder around the abdomen without inflation, but in all instances these systems were complicated. A simpler device would seem to have wider clinical applicability. The device used in this study requires neither simultaneous ventilation nor manipulation of the abdomen.

The human and canine chests are different in configuration. The canine chest is longer and narrower. It is unclear what effect these differences will have on the ability of vest inflation to generate high vascular pressures in man.

In humans with relatively small chests and large hearts, direct cardiac compression may occur during manual CPR. It is not known whether flow generated by direct compression in these individuals would be greater than that produced by manipulation of intrathoracic pressure.

In the case of cardiac arrest in which there has been a long delay in starting resuscitation, there is some evidence that open-chest cardiac massage may be superior to conventional CPR.\(^30\) It is not known how vest CPR would perform under similar circumstances, but it could be applied by paramedics, while open-chest cardiac massage must be performed by a physician.

In summary, vest CPR performed without simultaneous ventilation can produce myocardial and cerebral flow adequate to sustain life without causing severe trauma in dogs. This new system is simpler to apply than previous systems, and has the potential for improving blood flow and survival in man.

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