Active Compression-Decompression CPR Improves Vital Organ Perfusion in a Dog Model of Ventricular Fibrillation*

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Objectives: This study was designed to assess whether a new method of cardiopulmonary resuscitation (CPR), termed active compression-decompression CPR, or ACD-CPR, improves organ perfusion when compared with standard (S) CPR in a dog model of ventricular fibrillation.

Background: ACD-CPR has recently been shown to improve hemodynamic and respiratory parameters during cardiac arrest when compared with standard CPR. However, to our knowledge, the effects of ACD-CPR on tissue perfusion have not been investigated.

Methods: Ventricular fibrillation was induced in eight anesthetized, intubated animals. ACD-CPR and standard CPR were each performed twice in alternating order. All interventions were preceded by 1 min of ventricular fibrillation, in which no CPR was performed, and consisted of 6 min of CPR with either technique during which tissue perfusion was measured. Compressions were performed at 80/min with a 50 percent duty cycle and 175 to 200 N downward force applied to the chest wall for both techniques. Epinephrine was administered at the beginning of each 6-min CPR interval. Hemodynamic monitoring of aortic and right atrial pressure was performed continuously and myocardial, cerebral, and renal blood flows were measured using the radiolabeled microsphere technique at baseline and during all interventions.

Results: Baseline organ perfusion and hemodynamics were similar for all dogs. Baseline left ventricular, brain, and renal blood flows were 62.0 ± 5.5, 14.1 ± 2.1, and 478.3 ± 55.5 ml/min/100 g, respectively (mean ± SEM). Compared with standard CPR, ACD-CPR resulted in higher global left ventricular (22.5 ± 6.2 vs 14.1 ± 4.0 ml/min/100 g, p<0.01), cerebral (12.0 ± 2.4 vs 8.5 ± 2.3 ml/min/100 g, p<0.01), and renal cortical (27.8 ± 5.0 vs 17.5 ± 5.0 ml/min/100 g, p<0.05) blood flows. Regional flows to the epicardium, endocardium, and midmyocardium as well as to the frontal, parietal, and occipital lobes of the brain were all significantly improved by ACD-CPR. Aortic systolic (61.7 ± 4.1 vs 49.5 ± 3.1 mm Hg, p<0.01), aortic mean (31.6 ± 2.8 vs 27.2 ± 2.2 mm Hg, p=0.001), and myocardial perfusion pressure (12.9 ± 3.4 vs 10.4 ± 3.4 mm Hg, ACD-CPR vs standard CPR, p<0.01) were all higher during ACD-CPR than during standard CPR.

Conclusions: We conclude that ACD-CPR improves tissue perfusion and systemic hemodynamics compared with standard CPR.

| ACD-CPR=active compression-decompression cardiopulmonary resuscitation; S=standard |
|---------------------------------|---------------------------------|
| Key words: cardiopulmonary resuscitation; cerebral blood flow; hemodynamics; left ventricular blood flow; renal blood flow |

The standard method of cardiopulmonary resuscitation (S-CPR) has undergone few changes since its introduction by Kouwenhoven et al. more than three decades ago. Despite its nearly universal acceptance, the number of patients who benefit from S-CPR is small (<20 percent), probably secondary to inadequate blood flow during resuscitative efforts.2-4 While newer methods of CPR designed to enhance tissue perfusion have been investigated,5-16 at present there is insufficient evidence to warrant changes in current clinical S-CPR techniques. Recently, Lurie et al15 described a patient who was successfully resuscitated using an ordinary bathroom plunger during CPR. This anecdotal report has stimulated the development of a new method of CPR using a handheld suction device which, when applied to the chest surface, allows active decompression as well as compression of the chest wall. Recent studies using active compression-decompression (ACD) CPR in humans during cardiac arrest demonstrated improvements in hemodynamic and respiratory parameters during ACD-CPR compared with S-CPR.16,17 To date, and to our knowledge, the effects of ACD-CPR on tissue...
perfusion have not been investigated. This study was designed to compare regional organ blood flows achieved with ACD-CPR and S-CPR, and to gain new insights into the basic mechanisms by which active decompression of the chest wall might improve CPR hemodynamics.

METHODS

Preparation

This study was approved by the University of Minnesota Committee on Animal Research and was performed in accordance with the position of the American Heart Association on Research Animal Use. Eight beagles weighing 10 to 14 kg were anesthetized with pentobarbital sodium (30 mg/kg intravenously [IV]). Supplemental pentobarbital sodium was administered as needed during surgery. Dogs were intubated with a 6-F endotracheal tube and ventilated with 10 L of supplemental oxygen at a minute ventilation required to maintain arterial pH between 7.3 and 7.4. Arterial blood gas monitoring was performed every 30 min to ensure adequacy of ventilatory parameters. The chest was shaved and animals were placed in the supine position until immediately prior to induction of ventricular fibrillation; 5,000 U of heparin was given IV prior to initiation of the study. After surgical exposure of the femoral arteries and veins, a 5-F pigtail catheter (Cordis Corp, Miami) was placed into the right atrium and a 5-F bipolar catheter (Daig Corp, Minnetonka, Minn) was advanced under fluoroscopic guidance into the right ventricle. One pigtail catheter was also positioned in the apex of the left ventricle and a second was positioned in the descending thoracic aorta, immediately distal to the left subclavian artery. Two 5-F sampling catheters (Cordis Corp, Miami) were placed in the proximal iliac arteries, which served as the sites for collection of blood during microsphere injection. Esophageal pressure was measured using a multilumen water perfusion catheter (Arndorfer, Medical Specialties Inc, Greendale, Wis) as an index of intrathoracic pressure. The catheter was continuously flushed with 5 percent dextrose at 15 mL/h. Hemodynamic measurements using fluid-filled catheters were recorded on a multichannel recorder (Astro-Med, Astro-Med Inc, West Warwick, RI). Pressures from the thoracic aorta, right atrium, and esophagus were recorded using transducers (Spectramed, Spectramed Inc, Oxnard, Calif) referenced to the level of the right atrium. For analysis of hemodynamics during CPR with each technique, data were acquired at the end of each minute of each intervention. Maximum and minimum aortic and right atrial pressures obtained at end-expiration during compression (systole) and decompression (diastole) were averaged for six compressions over two respiratory cycles. Mean aortic and right atrial pressures were obtained electronically; 5,000 U of heparin was given IV prior to initiation of the study.

CPR Techniques

Both S-CPR and ACD-CPR were performed using a hand-held modified household plunger (internal diameter, 8 cm) shown schematically in Figure 1. At the base of the suction cup, a fluid-filled manometry system was constructed to allow monitoring of the force delivered to the chest wall during compressions. For each experiment, a new suction device was used to ensure adequate suction. Standard CPR was defined as compression and release with no suction adherence of the plunger to the chest, thus allowing passive relaxation of the chest wall to resting position. During S-CPR only, suction was prevented by placing two 10X10-cm gauze pads between the chest wall and the suction cup. ACD-CPR was defined as compression and active withdrawal of the plunger with adequate suction to actively decompress the chest wall to an anteroposterior diameter approximately 10 percent beyond normal resting position. CPR was performed with the dogs in the left lateral oblique (45°) position. Compressions were delivered at the midventricular level, determined fluoroscopically, slightly lateral to the sternum where adequate suction could be achieved.

Using a metronome, both techniques were performed at a rate of 80/min with a 50 percent duty cycle. To ensure that the force of compressions was equal during both methods of CPR, two monitoring systems with continuous feedback to the person applying compressions were used. Direct measurements of force applied to the chest wall were made with the manometer constructed at the base of the plunger. In addition, esophageal manometry was used as an indirect assessment of intrathoracic pressure to record the degree of compression and decompression. The delivered force and resultant esophageal pressures were recorded on the recorder (Coulbourn) and also displayed for viewing by the operator throughout the experiment on an electronic oscilloscope. Using these techniques, 175 to 200 N force was applied to the chest wall with each compression resulting in approximately 45 mm Hg intrathoracic pressure (end-expiratory), and 3 to 5 cm downward displacement of the chest wall.

Experimental Protocol

This protocol was designed to allow the comparison of tissue flows and hemodynamics during multiple periods of ACD-CPR and S-CPR in the same animal. It was modeled in part from previous studies of CPR in animals by the Hopkins group18 in which a short period of no CPR and relatively high-dose epinephrine were used. In each of seven dogs, both ACD-CPR and S-CPR were performed twice, 6 min per intervention, in alternating order. In one dog, ACD-CPR and S-CPR were each performed once. Each round of CPR was preceded by 1 min during which no CPR was performed. Using this experimental design, hemodynamic and organ blood flow measurements during each method of CPR were made twice in the same animal. Thus, comparison of tissue flows achieved with the two CPR techniques could be made with each animal serving as its own control.

Before cardiac arrest, baseline hemodynamic measurements were obtained and radiolabeled microspheres were injected to determine control blood flows by methods previously described.19,20 During baseline measurements, saline solution was infused at 25 ml/h and adjusted as needed to maintain mean right atrial pressure at 3 to 8 mm Hg. Ventilatory support was conti-
used throughout all experiments using manual bag ventilation with 10 L oxygen supplementation. Hand-held ventilation was utilized after preliminary experiments showed that it was easier to interpose manual ventilatory efforts at the end of the decompression phase with bag ventilation than with mechanical ventilation. Respiration were delivered at a rate of 16/min (one breath every five chest compressions) at a constant tidal volume required to maintain the same minute ventilation delivered during surgical preparation. Ventricular fibrillation was induced by a single 5-s application of alternating current applied to a 5-F bipolar electrode lead in direct contact with the endocardium of the right ventricle. After 1 min of fibrillation, during which no CPR or ventilation was performed, either ACD-CPR or S-CPR was initiated. The decision to use S-CPR or ACD-CPR was made randomly at the beginning of each experiment. In four dogs, S-CPR was performed first, and in four dogs, ACD-CPR was performed first. Epinephrine was administered using a protocol similar to that of Michael et al.\(^3\) with a bolus (1 mg) directly into the left ventricle at the onset of CPR, followed by a continuous infusion (8 \(\mu\)g/kg/min) into the right atrium throughout the remaining 27 min of the experiment. The first CPR method (either S-CPR or ACD-CPR) was continued for 6 min during which blood flows and hemodynamics were measured. After 5.5 min of the first intervention, a 5-ml aliquot of blood was obtained from the left ventricle for blood gas analysis and to assess residual counts in the ventricle at the end of the intervention. After 6 min, CPR was stopped. After 1 min of no CPR and no ventilation, the alternate CPR technique was performed for 6 min. At the onset of this second round of CPR, a repeat bolus of epinephrine (1 mg) was administered into the left ventricle. Hemodynamic and organ perfusion data were obtained as in the first intervention. In seven experiments, this cycle of 1 min of ventricular fibrillation with no CPR and 6 min of CPR was repeated two additional times alternating the two techniques for each intervention, for a total of four CPR interventions per dog. In one experiment, only two interventions were performed.

**Tissue Flow Measurements**

Regional blood flow was measured using 15-\(\mu\)m-diameter microspheres with techniques similar to those previously reported and validated during CPR in dogs by Koehler et al.\(^6\) Our laboratory has extensive experience in the use of radioactive microsphere technique. The vials of microspheres were shaken and then dispersed by ultrasonic agitation. Studies have shown that tissue flows can be accurately measured within 1 min of instituting CPR and that peripheral blood flow and tissue perfusion remain relatively steady throughout the entire duration of CPR up to 50 min.\(^7\) For each intervention, approximately \(2 \times 10^6\) spheres were injected as a bolus into the left ventricle 30 s after the onset of chest compressions. Each injection was followed by a 10-ml flush of saline solution. Reference arterial blood samples were obtained in 30-s aliquots from both iliac arteries in the first five dogs at a continuous rate of 5 ml/min per site using a mechanical peristaltic pump (Harvard, Harvard Apparatus, South Natick, Mass). For the subsequent three dogs, only one peripheral withdrawal site was used. In order to maintain adequate volume status during the experiment, 1 U of packed red blood cells was infused peripherally at the same rate as withdrawal from the iliac arteries. Animals were killed at the end of each experiment using bolus injections of potassium chloride. Organs were fixed in 10 percent formalin for sectioning and microsphere analysis.

Myocardium from the left ventricle was taken at the midventricular level and sectioned into epicardial, endocardial, and midmyocardial regions. A total of 12 samples were available for each region. Brain tissue was sectioned into frontal, parietal, and occipital regions, with each region yielding four samples. From each kidney, the outer 1 to 2 mm of tissue was taken for counting, yielding 12 renal cortical samples per experiment. Vials of blood and tissue were counted on a multichannel autogamma scintillation spectrometer. The energy windows used for \(^{141}\)Ce, \(^{51}\)Cr, \(^{52}\)Co, \(^{55}\)Fe, \(^{90}\)Tc, and \(^{188}\)Re were 128 to 168, 304 to 348, 484 to 548, 718 to 804, and 834 to 1160 keV, respectively. Overlap of counts was subtracted to obtain corrected count values for each isotope using the method of differential spectroscopy.\(^8\) Counts from each sample of myocardium, brain, and both kidneys were examined for excessive variability within an individual animal. Because sample variability was judged not large, counts from all tissue samples were used to calculate the mean and variance of the data. Tissue blood flow was then calculated by dividing tissue-corrected counts by the total corrected counts per milliliter per minute in the reference blood samples. The two determinations of tissue blood flow for each CPR intervention were then averaged to provide a single value for S-CPR and ACD-CPR for each animal.

**Statistical Analysis**

All values are expressed as mean values \(\pm\) SEM. Statistical analysis of regional blood flows and clearance of microspheres from the peripheral circulation was performed using the nonparametric Wilcoxon signed rank test because the SD in the values was similar to the magnitude of the mean, suggesting the data were not normally distributed. For analysis of hemodynamic and arterial blood gas data, the paired Student’s \(t\) test was used.

**RESULTS**

**Global Organ Blood Flow**

Global organ blood flows using radiolabeled microspheres are summarized in Table 1 and Figure 2. Mean baseline global left ventricular blood flow was 62.0 \(\pm\) 5.5 ml/min/100 g. Left ventricular flow during either method of CPR was significantly lower than during the baseline period (\(p<0.01\)). However, during ACD-CPR, left ventricular flow (22.5 \(\pm\) 6.2 ml/min/100 g) was significantly greater than during S-CPR (14.1 \(\pm\) 2.1 ml/min/100 g, \(p<0.01\), ACD-CPR vs S-CPR).

Average baseline total brain flow was 14.2 \(\pm\) 2.1 ml/min/100 g. During S-CPR, the flow was 8.5 \(\pm\) 2.3 ml/min/100 g (significantly lower than baseline, \(p<0.05\)), but this value increased to 12.0 \(\pm\) 2.4 ml/
min/100 g during ACD-CPR (p<0.01, ACD-CPR vs S-CPR flows). The difference between ACD-CPR and baseline brain blood flow was not statistically significant (p=0.24). Renal cortical flow fell dramatically during CPR with average flows of 476.3±55.5 ml/min/100 g during baseline, 17.5±5.0 ml/min/100 g during S-CPR, and 27.5±5.0 ml/min/100 g during ACD-CPR (p<0.05, ACD-CPR vs S-CPR flows). Thus, when compared with S-CPR, ACD-CPR resulted in a significant increase in total left ventricular perfusion, cerebral perfusion, and renal cortical flow. Furthermore, ACD-CPR resulted in cerebral blood flows that were statistically equivalent to baseline values.

Global mean left ventricular and cerebral blood flows were significantly greater during ACD-CPR than during S-CPR in each of the eight individual experiments. As shown in Figure 2, organ flows were improved with ACD-CPR throughout a broad range of measured flows. These differences were statistically significant in each experiment (p≤0.02 ACD-CPR vs S-CPR). Thus, whether tissue flows during resuscitation in an individual experiment were low or high, ACD-CPR resulted in greater perfusion than S-CPR. In the kidney, flow was improved with ACD-CPR in five of the seven animals (p<0.02). In one experiment, renal cortical flows were no different between the two techniques, and in another, flow during S-CPR was higher than during ACD-CPR. In one experiment, renal blood flow was below the limit of detection with both ACD-CPR and S-CPR.

Improvement in organ perfusion seen with ACD-CPR is further supported by the time course of recovery of microsphere activity from the circulation. Figure 3 reveals that the time to peak recovery of counts in the peripheral blood was the same for both methods of CPR, occurring 1.5 min after injection into the left ventricle. However, microspheres appeared more rapidly in the peripheral blood during ACD-CPR than during S-CPR. At 1.0 min after injection, 25.4±6.8 percent of all counts recovered were seen with ACD-CPR vs 14.6±5.3 percent dur-

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**Figure 2.** Mean organ flows during active compression decompression CPR (ACD-CPR) and standard CPR (S-CPR). The measured flows from individual studies are interconnected. Mean flows (with SEMs) are shown to the left and right of the data from individual dogs. P values refer to mean flows during ACD-CPR vs mean flows during S-CPR.

**Figure 3.** Time course of recovery of radiolabeled microspheres from the peripheral circulation during baseline, ACD-CPR, and S-CPR. Percent counts per minute (cpm) was calculated as the corrected counts recovered during each 30-s interval divided by the total number of corrected counts recovered during the 6-min intervention. The time scale was not adjusted for the approximate 15-s delay arising from catheter dead space.
ing S-CPR (p=0.02). After peak recovery, microspheres were also cleared from the peripheral circulation significantly faster with ACD-CPR than with S-CPR. With ACD-CPR, 13.1±2.4 percent and 6.5±1.9 percent of total counts recovered were collected at 2.0 and 2.5 min, respectively, compared with 17.6±2.1 percent and 9.2±1.6 percent at the same time points during S-CPR (p<0.05, both time points). Taken together, these clearance data are consistent with a significantly higher peripheral blood flow state during ACD-CPR than during S-CPR and are consistent with the organ blood flow data discussed above.

**Regional Tissue Perfusion**

In addition to total organ blood flow, regional tissue perfusion was also significantly improved with ACD-CPR compared with S-CPR. Regional organ flow in the heart and brain are shown in Table 1. Epicardial, midmyocardial, and endocardial left ventricular flows were all significantly improved with ACD-CPR compared with S-CPR. During ACD-

<table>
<thead>
<tr>
<th>Pressure</th>
<th>Baseline, mm Hg</th>
<th>S-CPR, mm Hg</th>
<th>ACD-CPR, mm Hg</th>
<th>p Value (ACD- vs S-CPR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic systolic</td>
<td>128.3±5.5</td>
<td>49.5±3.1</td>
<td>61.7±4.1</td>
<td>0.001</td>
</tr>
<tr>
<td>Aortic diastolic</td>
<td>50.4±5.2</td>
<td>20.5±2.4</td>
<td>23.5±3.3</td>
<td>0.063</td>
</tr>
<tr>
<td>Mean aortic systolic</td>
<td>108.5±4.9</td>
<td>27.2±2.2</td>
<td>31.6±2.8</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Right atrial systolic</td>
<td>5.7±1.2</td>
<td>53.9±3.3</td>
<td>65.4±4.1</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Right atrial diastolic</td>
<td>3.4±0.9</td>
<td>8.5±1.4</td>
<td>8.6±1.9</td>
<td>0.65</td>
</tr>
<tr>
<td>Mean right atrial</td>
<td>4.7±1.1</td>
<td>23.5±1.6</td>
<td>27.5±1.6</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Myocardial perfusion</td>
<td>87.0±5.0</td>
<td>11.3±2.7</td>
<td>13.7±2.8</td>
<td>&lt;0.02</td>
</tr>
</tbody>
</table>

*All values are mean±SEM; n=8. Myocardial perfusion pressure was computed as aortic diastolic minus right atrial diastolic pressure.

CPR, regional flows to the epicardium and mid-myocardium were >40 percent of baseline levels while flow to the endocardium reached nearly 30 percent of baseline. In contrast, during S-CPR, epicardial and midmyocardial flows were <30 percent of baseline.

**FIGURE 4.** Representative hemodynamic, force, and esophageal recordings obtained during standard CPR (S-CPR, left) and active compression decompression CPR (ACD-CPR, right) in the same dog. Both were obtained after 3 min of CPR. Arrows depict the beginning of compression.
and endocardial flows were only 16 percent baseline flow. The ratio of endocardial to epicardial flows decreased from 1.76 at baseline to 0.92 (p<0.02) during ACD-CPR and to 0.79 (p<0.01) during S-CPR. While the endocardial/epicardial ratio was greater during ACD-CPR compared with S-CPR, this difference was not statistically significant (p=0.20). In the brain, ACD-CPR resulted in flows to the frontal, parietal, and occipital lobes to nearly 85 percent of baseline levels, while flows during S-CPR reached only 60 percent of baseline.

**Hemodynamics and Blood Gas Values**

The hemodynamic data from these experiments are summarized in Table 2. Compared with S-CPR, ACD-CPR resulted in significantly higher aortic systolic and aortic mean pressures. Systolic right atrial and mean right atrial pressures were also significantly higher during ACD-CPR than during S-CPR. In contrast, there were no significant differences between diastolic aortic and diastolic right atrial pressures during CPR with the two techniques. Myocardial perfusion pressure, computed for each experiment as the diastolic aortic pressure minus the diastolic right atrial pressure, was significantly greater during ACD-CPR than during S-CPR. Left ventricular microsphere flow correlated with myocardial perfusion pressure for both ACD-CPR (r=0.845, p<0.01) and S-CPR (r=0.811, p<0.02).

Representative hemodynamic data acquired during ACD- and S-CPR are shown in Figure 4. Simultaneously recorded force of compression and intrathoracic pressure tracings are also shown. This example suggests that with constant force of compression and similar peak intraesophageal pressure, aortic systolic pressures are higher with ACD-CPR than with S-CPR. Furthermore, during active decompression, somewhat greater negative pressures within the esophagus are achieved, suggesting increased negative intrathoracic pressures were generated with ACD-CPR. Also worth noting is the difference in the right atrial pressure tracings during diastole with each technique. With active decompression, right atrial pressure appears to reach its nadir earlier and remain lower than with S-CPR for a greater proportion of diastole, despite similar duty cycles for the two techniques.

A total of 28 blood gas samples taken during the last minute of baseline measurements and of each CPR intervention were analyzed from six of the eight experiments. Table 3 reveals there were no significant differences in the arterial blood gas data during ACD-CPR compared with S-CPR and that animals were adequately oxygenated throughout the experiment. When compared with baseline, animals were significantly more acidic during S-CPR but not during ACD-CPR. During CPR with either technique, animals were significantly more hypocapnic than during baseline.

**Validation Data**

In order to validate the use of radiolabeled microspheres in this acute model of ventricular fibrillation and to assess differences in ACD-CPR vs S-CPR, peripheral clearance of microspheres was estimated by dividing the corrected counts obtained in each of the 30-s aliquots by the total number of counts recovered during an intervention. In Figure 3, the three curves reveal that microspheres had sufficiently cleared from the peripheral circulation by 3 min following their injection into the left ventricle during baseline, S-CPR, and ACD-CPR. The raw counts recovered from the left ventricle 5.5 min after initiation of each intervention were also less than 3 percent of the total number of counts recovered during the intervention. These data suggest that adequate ejection of microspheres from the ventricle had occurred during both CPR techniques. These findings are consistent with previous work that validated the microsphere technique for measuring blood flow during CPR.4

The data in this study represent means of values obtained during separate CPR interventions within individual experiments. It is possible that differences in blood flows seen with ACD-CPR vs S-CPR were affected by the particular time frame in a given experiment during which the intervention was performed. To assess whether time was an important factor in our results and to assess the stability of our model during the course of an experiment, mean flows achieved with both ACD-CPR and S-CPR early in each of the experiments (ie, the first two interventions) were compared with mean flows obtained with the same technique late in the experiment (the last two interventions) (Table 4). Also, the absolute differences and percentage of change in flows achieved with S-CPR and ACD-CPR early in each experiment were compared with the differences obtained late in the protocol. With both techniques, mean flows to the heart and the brain tended to fall as the experiment progressed to the later stages, while

### Table 3—Arterial Blood Gas Data*

<table>
<thead>
<tr>
<th>pH</th>
<th>P_{CO_2}</th>
<th>P_{O_2}</th>
<th>O_2 Sat.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mm Hg</td>
<td>mm Hg</td>
<td>%</td>
</tr>
<tr>
<td>Baseline</td>
<td>7.35 ± 0.02</td>
<td>32.0 ± 2.9</td>
<td>420.3 ± 65.5</td>
</tr>
<tr>
<td>S-CPR</td>
<td>7.25 ± 0.04</td>
<td>24.9 ± 4.2</td>
<td>369.8 ± 72.6</td>
</tr>
<tr>
<td>ACD-CPR</td>
<td>7.28 ± 0.06</td>
<td>24.6 ± 4.5</td>
<td>351.7 ± 73.3</td>
</tr>
</tbody>
</table>

*All values are mean ± SEM; n=6.

\( p < 0.05 \), compared with baseline.

\( p < 0.01 \), compared with baseline.
mean flows to the renal cortex tended to increase. However, these changes over time were not statistically significant, suggesting that the model remained relatively stable throughout the experiments. Furthermore, the absolute differences in mean ACD-CPR and S-CPR flows remained constant throughout the experiment, suggesting that compared with S-CPR, ACD-CPR improved flows by a constant magnitude that was independent of time. Therefore, mean ACD-CPR tissue flows and hemodynamic parameters for the early and late periods were compared with mean S-CPR flows and hemodynamics.

**DISCUSSION**

This study demonstrates that compared with S-CPR, ACD-CPR improves blood flow to three vital organs and significantly improves systemic blood pressure in a dog model of ventricular fibrillation. Global and regional blood flows to the heart, brain, and kidney, as well as systolic blood pressure and calculated myocardial perfusion pressure were all improved with ACD-CPR. Despite wide variations in basal organ flow, improvements in flows to the heart and brain were seen with ACD-CPR in each of eight individual experiments that comprise this study. The measured myocardial flow during S-CPR was comparable to that observed in previous reports. However, myocardial flows of 20 to 25 ml/min/100 g tissue, higher than those observed with S-CPR in this study, may be necessary to meet the heart’s metabolic needs during ventricular fibrillation. Previous authors suggest that flows during prolonged CPR of at least 10 to 20 percent of baseline are necessary for successful defibrillation and return of spontaneous circulation. If these criteria are accurate, both the higher absolute flows and percentage of baseline flows achieved with ACD-CPR in our study suggests that ACD-CPR may provide a significant advantage over S-CPR in overcoming these critical threshold values.

The brain is extremely sensitive to anoxic insult with irreversible neuronal damage beginning within 4 to 6 min after complete ischemic insult. Metabolic and clinical studies suggest that neurons can survive blood flow rates as low as one third of normal values. Above this level of perfusion, cells appear to maintain normal ATP content and intracellular homeostasis, and patients demonstrate normal electroencephalograms and somatosensory-evoked potentials. In this study during ACD-CPR, cerebral blood flows averaged nearly 80 percent of baseline flows. These levels of perfusion during ACD-CPR were statistically equivalent to baseline brain flows. In contrast, S-CPR resulted in 60 percent of baseline flows. Taken together, the significant increase in cerebral flows achieved with ACD-CPR over S-CPR might be important with regard to the potential recovery of neurologic function following a period of CPR.

The minimal metabolic requirements of the kidney are less well documented than for the heart and brain. Blood flow is preferentially shunted from the kidney during ventricular fibrillation. Renal blood flow during CPR in the range of 1 percent of baseline values has been a consistent finding in previous studies. The magnitude of flows seen in this study is comparable to those found previously. It is unlikely that the significant differences in ACD-CPR vs S-CPR renal blood flow during ventricular fibrillation would provide a greater chance of preserving renal function during and after arrest.

The exact mechanisms responsible for improvement in organ flow and hemodynamics with ACD-CPR remain unclear. Indeed, the mechanisms by which circulation is sustained during S-CPR continues to be the subject of debate. Kouwenhoven et al proposed that blood flow results from direct cardiac compression, a theory recently supported by several investigators. However, significant laboratory data also support the thoracic pump theory, which suggests that blood flow during CPR is a result of a generalized increase in intrathoracic vascular pressures that is transmitted to the extrathoracic arteries during compression of the chest wall. Competent venous valves and the high systemic venous compliance prevents full retrograde pressure transmission to the extrathoracic veins, thus creating the arteriovenous pressure gradient necessary for forward blood flow. Other work has suggested that both mechanisms play important roles and are not mutually exclusive.

The mechanisms responsible for blood flow within the thorax, specifically in the myocardium, have been investigated separately. Using an electromagnetic flow probe, Ditchey et al demonstrated that epicardial coronary blood flow and the corresponding

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**Table 4—Organ Flow vs Time**

<table>
<thead>
<tr>
<th></th>
<th>Early</th>
<th>Late</th>
<th>%Δ</th>
<th>p Value (Early vs Late)</th>
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<tbody>
<tr>
<td>Left ventricle</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACD-CPR</td>
<td>24.2±7.9</td>
<td>16.7±5.2</td>
<td>−31.0</td>
<td>NS</td>
</tr>
<tr>
<td>S-CPR</td>
<td>16.7±4.6</td>
<td>10.2±4.8</td>
<td>−38.9</td>
<td>NS</td>
</tr>
<tr>
<td>Difference</td>
<td>7.5±6.4</td>
<td>6.6±4.6</td>
<td></td>
<td>NS</td>
</tr>
<tr>
<td>Brain</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACD-CPR</td>
<td>12.8±2.3</td>
<td>10.9±3.3</td>
<td>−14.8</td>
<td>NS</td>
</tr>
<tr>
<td>S-CPR</td>
<td>9.2±2.4</td>
<td>8.1±3.0</td>
<td>−12.0</td>
<td>NS</td>
</tr>
<tr>
<td>Difference</td>
<td>3.6±1.6</td>
<td>2.9±1.9</td>
<td></td>
<td>NS</td>
</tr>
<tr>
<td>Renal cortex</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACD-CPR</td>
<td>25.9±6.3</td>
<td>31.6±8.4</td>
<td>22.0</td>
<td>NS</td>
</tr>
<tr>
<td>S-CPR</td>
<td>16.8±5.5</td>
<td>20.4±4.9</td>
<td>21.4</td>
<td>NS</td>
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<tr>
<td>Difference</td>
<td>9.1±7.7</td>
<td>11.2±5.5</td>
<td></td>
<td>NS</td>
</tr>
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</table>

*Early and late values are expressed in milliliter per minute per 100 g of tissue. All values are mean±SEM; n=8. NS=not significant.
aortic-to-right atrial pressure gradient occur predominantly during the relaxation phase of the chest compression cycle of CPR. Halperin et al.\textsuperscript{31} were able to demonstrate that myocardial flows depended on diastolic myocardial perfusion pressures and that both varied significantly with alterations in intrathoracic pressures.

In this study, we have demonstrated that during ACD-CPR there is an improvement in myocardial perfusion pressure leading to greater myocardial flows during ACD-CPR (Tables 1 and 2). The tracings shown in Figure 4 suggest that the right atrial pressure during decompression in ACD-CPR remains lower for a greater duration than during S-CPR. This difference may be important and may account for the improvement in calculated myocardial perfusion pressure and measured flows. Furthermore, esophageal manometry, an indirect measure of intrathoracic pressure, reveals a marked decrease in pressure during active decompression (greater negative intrathoracic pressure) with ACD-CPR (Fig 4). To our knowledge, demonstration of intrathoracic pressure alterations with ACD-CPR has not been reported previously. In preliminary studies, we have evaluated the effect of ACD-CPR on phasic coronary flow velocity. These studies reveal that peak late diastolic flow velocity during decompression is markedly augmented during ACD-CPR vs S-CPR.\textsuperscript{34}

Based on these results, we speculate that ACD-CPR improves organ flow and systemic hemodynamics predominantly by increasing negative intrathoracic pressure during active decompression. The improvement in the aortic-to-right atrial pressure gradient and blood flow to intrathoracic structures such as the heart is likely to be a direct result of this alteration in intrathoracic pressure. Furthermore, during active decompression, the compliant thoracic venous bed is subjected to greater negative pressure both in duration and in magnitude. The resultant increase in the arterial to venous gradient may lead to greater filling of the thoracic venous bed as well as the right side of the heart, accounting for greater flows and pressures during compression. Further studies investigating the underlying mechanisms of organ perfusion during ACD-CPR are the focus of our current laboratory efforts.

\textit{Limitations}

A large variance in absolute organ flow was noted during both methods of CPR. This is likely due to variations in the efficacy of either method of CPR secondary to differences in the canine chest configuration from animal to animal as they affect the performance of both chest compression and decompression. Substantial variations in organ blood flow are commonly reported in animal studies of CPR. In our study, the use of paired observations in the same animal, each performed twice, however, permitted a comparison of the two methods to be made regardless of the absolute organ flow in any individual animal. A similar “down-time” was used before each replication. In order to maintain a viable experimental preparation for both replications, the duration of ventricular fibrillation in these studies was kept relatively short.

The measured blood flows to the brain during baseline in this study are lower than previously reported, despite similar hemodynamics.\textsuperscript{4,18,22} The hypocarbia seen during the basal state, which was more pronounced during both methods of CPR, may be, in part, responsible. However, since baseline renal and myocardial blood flow are comparable to previously reported values, it is unlikely that these findings are simply a result of generalized methodologic considerations regarding microsphere blood flow measurements. Furthermore, the measured vital organ flows during S-CPR in this study are comparable to those seen in previous reports using similar S-CPR techniques.\textsuperscript{4,18} The dose of epinephrine used in these studies was selected following the work of Michael et al.\textsuperscript{18} This dosage would represent high-dose epinephrine if used in humans. Our results in this regard are different from those of Lindner et al\textsuperscript{35} in swine in which ACD-CPR was not shown to have beneficial effects on organ blood flow when utilized with high-dose epinephrine. There may be important species differences relative to epinephrine between the swine and canine models.

Critical to accepting the results of this investigation is the assumption that the systolic phases of compression were similar with the two CPR techniques. In this study, a hand-held compressive device was utilized. Preliminary studies convinced us that this method of chest compression more closely modeled the clinical application of CPR than the mechanical “thumper.” We attempted to assure equal systolic compression with both techniques by monitoring the direct force applied to the chest wall as well as the resultant intrathoracic pressure, measured indirectly with esophageal manometry.\textsuperscript{31,36} Using constant monitoring, these parameters were kept as equal as possible. Both S-CPR and ACD-CPR were subjected to identical methods of data analysis. Given these considerations, we believe it is unlikely that the improvement seen in organ flows and hemodynamics with ACD-CPR in this study is due to differences in systemic compressive force.

Finally, application of results of a CPR study in an animal model to the clinical setting is limited, primarily due to differences in the chest configuration between animals and humans. Chest dimensions
are known to be important determinants of the effects of CPR on hemodynamics and organ flows.\textsuperscript{5,33,37} The canine chest configuration is not ideal for studies of a technique requiring consistent chest decompression to be achieved using active suction. In these studies, the plunger would occasionally lose suction and require reapplication. Nevertheless, the technique is not difficult to apply in animals and preliminary data show it to be even easier in humans.\textsuperscript{37} Further refinements into techniques to achieve the most effective rate and depth of chest decompression may yield even better hemodynamic and myocardial perfusion results. Certainly, any alterations in currently accepted CPR techniques must be supported by proven benefits in the laboratory setting.

CONCLUSIONS

ACD-CPR is a novel method of CPR. Based on this study, we conclude that ACD-CPR significantly improves vital organ perfusion and systemic blood pressure during CPR in a dog model of ventricular fibrillation. The mechanism by which ACD-CPR improves blood flow and CPR hemodynamics remains speculative, but it is likely due to the development of greater negative intrathoracic pressure during active decompression resulting in the active transport of greater blood volumes into the thoracic cavity and right side of the heart. Clinically relevant issues such as whether ACD-CPR leads to improved recovery of organ function or facilitates the return of spontaneous circulation following a period of CPR are currently ongoing in prehospital studies. We believe that further studies designed to evaluate these outcomes using this novel method of CPR, both in animal models and in the clinical setting, are warranted.

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REFERENCES


36 Standards and guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). JAMA 1986; 255:2905-89