Augmentation of Cerebral Perfusion by Simultaneous Chest Compression and Lung Inflation with Abdominal Binding After Cardiac Arrest in Dogs
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SUMMARY Recent studies have demonstrated that for the same chest compression force during mechanical cardiopulmonary resuscitation (CPR), the carotid artery-to-jugular vein pressure gradient and carotid blood flow are increased when the phasic rise of intrathoracic pressure is enhanced by abdominal binding and simultaneous ventilation at high airway pressure with each chest compression (SCV). The objective of the present study was to assess whether cerebral blood flow is also enhanced, since it is known that fluctuations in intrathoracic pressure are transmitted to the intracranial space and affect intracranial pressure (ICP). In two series of pentobarbital-anesthetized dogs, one of two CPR techniques was initiated immediately after inducing ventricular fibrillation. Brain blood flow was measured by the radiolabeled microsphere technique immediately before cardiac arrest and at 1 and 3 minutes after commencing CPR. Evidence of adequate mixing of spheres and lack of sedimentation under these low-flow conditions was verified by correlation with brain venous outflow, comparison of the arterial concentration–time profile of spheres and a nonselective marker (thallium-201 in solution), and use of multiple arterial sampling sites. During SCV CPR with abdominal binding, mean carotid artery pressure (60 ± 3 mm Hg) was higher than that during conventional CPR (25 ± 2 mm Hg). Pulsations of ICP occurred that were in phase with chest compression and greater than jugular venous pressure. Mean ICP was higher during SCV (46 ± 2 mm Hg) than conventional CPR (20 ± 2 mm Hg). However, the net brain perfusion pressure gradient (carotid artery pressure × ICP) was greater with SCV (14 ± 3 mm Hg) than with conventional CPR (5 ± 0.4 mm Hg). Cerebral blood flow was significantly greater during SCV CPR (32 ± 7% of prearrest cerebral flow) than during conventional CPR (3 ± 2%). We conclude that SCV CPR combined with abdominal binding substantially improved brain perfusion by enhancing cerebral perfusion pressure in this experimental model.

GENERATION of peripheral blood flow during cardiopulmonary resuscitation (CPR) after cardiac arrest requires the production of an extrathoracic arteriovenous pressure gradient. Recent evidence indicates that this gradient is produced in most animal models and humans by the phasic increase in intrathoracic pressure during chest compression.1-4 While extrathoracic arterial pressure increases almost as much as intrathoracic pressure, the increase in jugular venous pressure is minimized by jugular venous valves and the large peripheral venous capacitance.1,2,5 Attempts to optimize peripheral blood flow have centered on techniques for maximizing the increase in intrathoracic pressure. These newer experimental techniques include lung inflation at high airway pressure (60–90 mm Hg) in synchrony with each chest compression. Chest compression is useful in preventing lung overinflation. Binding the abdomen minimizes movement of the diaphragm and mobilizes blood volume. Abdominal binding appears to be both independently and additively beneficial with synchronous high-pressure lung inflation.6 Improvement in carotid blood flow in animals6 and increased arterial pressure in man7,8 have been reported with these techniques.

Optimization of brain perfusion during CPR is more complex than in other peripheral vascular beds because of the role of intracranial pressure (ICP) in determining brain blood flow. ICP acts as the effective downstream pressure when this pressure is greater than jugular venous pressure.9-11 Increases in intrathoracic pressure12,13 and chest compression, in particular,14 increase ICP. Thus, it is not clear whether strategies that maximize intrathoracic pressure will necessarily improve cerebral perfusion.15 In a preliminary study, Bircher and Safar16 found that in small dogs, the cerebral perfusion pressure is relatively unaltered by increasing intrathoracic pressure, although flow was not measured. The purpose of the present study was to examine the effect of increasing intrathoracic pressure on cerebral hemodynamics and blood flow in dogs by comparing two methods of CPR: conventional CPR with lung inflation after every fifth chest compression, and CPR with intrathoracic pressure enhanced by simultaneous ventilation at high airway pressure with every chest compression (SCV) combined with abdominal binding.

Few studies have examined cerebral blood flow during CPR.17 A major difficulty in obtaining quantitative information relates to the difficulty of accurately measuring cerebral blood flow under conditions of low-flow states. We used the radiolabeled microsphere technique for measuring cerebral blood flow in dogs.
Detailed evidence to support the validity of the use of microspheres under the present experimental conditions is presented.

Methods

Preparation

Eighteen mongrel dogs that weighed 25–33 kg were anesthetized with ketamine (7 mg/kg i.m.) and pentobarbital sodium (15 mg/kg i.v.). Supplemental pentobarbital was administered as needed during surgery. Dogs were ventilated through an endotracheal tube tightly secured in a tracheostomy. From the femoral veins, a catheter was placed into the right atrium and a pacing electrode catheter was inserted into the right ventricle. From the femoral arteries, a catheter was placed into the thoracic aorta and a pigtail catheter was inserted into the left ventricle for injection of microspheres. A catheter inserted into an omocervical artery, with its tip lying in a subclavian artery, served as a sampling site for the collection of blood during the microsphere injection. The left carotid artery was used as a second sampling site. In four dogs a catheter was inserted into the carotid artery through the thyroid artery. In 10, a cannulating electromagnetic flow probe was inserted in the carotid artery. A side arm connector on the distal port of the probe served as the microsphere sampling site in these animals. Jugular venous pressure was measured from a small catheter inserted from a side branch high in the neck. Intracranial pressure was measured from a 16-gauge cannula inserted into the lateral ventricle through a small drill hole in the skull. Dogs were studied in the supine position with the head at approximately the same level as the heart. They were given 300 U of heparin before CPR.

Experimental Protocol

Before cardiac arrest, the first injection of microspheres was made to determine control blood flows. Ventricular fibrillation was then induced by high-frequency stimulation of the pacing electrodes. Mechanical chest compression was instituted within 15 seconds by a programmable pneumatic piston (Thumper) placed over the sternum about 5 cm above the xiphoid, as previously described.6 The CPR technique to be performed on a particular day was chosen before obtaining the unselected dog. In one group of seven dogs, conventional CPR was performed at a chest compression rate of 60/min, with compression lasting 60% of each cycle. Every fifth compression was followed by 1 second of ventilation using a gas mixture of 95% O₂–5% CO₂ by a pressure-limited ventilator to a peak airway pressure of 30 mm Hg. Pressure-limited ventilation was used because it provides a better standardized increase in intrathoracic pressure than a volume delivery system.

In a second group of seven dogs, the lungs were inflated synchronously with every chest compression at a rate of 40/min over a duration of 0.9 second (60%) of each cycle (SCV CPR). Peak airway pressure was set at 80–90 mm Hg. Overinflation of the lungs at this high airway pressure was prevented by the simultaneous chest compression. At the release of chest compression, the airway was opened, allowing airway pressure to fall to atmospheric pressure. Also in these dogs, a wide binder was tightly wrapped around the abdomen after arrest. In both groups of dogs, piston force was adjusted to produce sternal displacement of about 6 cm, which was then held constant. All dogs received 300 ml of saline into the right atrium within the first 45 seconds of CPR. The adequacy of ventilation was checked by sampling arterial blood after 5 minutes of CPR. Arterial Po₂ was greater than 100 mm Hg in both groups of dogs, consistent with previous experience using these techniques.6

The second and third set of microspheres were injected at 1 and 3 minutes after initiation of CPR. Reference blood samples were collected from both sites for the entire 9-minute duration of CPR. After CPR was stopped, the chest was opened to confirm catheter placement and to sample blood from the left ventricle to check for any remaining radioactivity. The entire brain was then removed for further dissection, and tissue samples of other organs were also obtained and weighed.

Measurements

Pressures were recorded from the intrathoracic aorta, carotid artery, jugular vein, right atrium and lateral ventricle using Statham P23Db transducers that were referenced to the level of the right atrium. Mean pressures were obtained by planimetry.

Regional blood flow was measured with 15 ± 3-μm diameter microspheres.18,19 The vial of microspheres was vigorously shaken and then further dispersed by ultrasonic agitation. The spheres were injected as a bolus into the left ventricle, followed by a 10-second flush of 10 ml of saline. Approximately 2.5 × 10⁶ spheres were injected for the control measurement and about 0.8 × 10⁶ spheres were injected for each of the postarrest measurements. The lower energy-emitting isotope, 124I-Sn (New England Nuclear), was used for the prearrest measurement when brain blood flow was high, and higher energy-emitting isotopes, 85Sr and 46Sc (3M Co.), were used after arrest when blood flow was low. This sequence minimizes the error from stripping the overlap counts of the high-energy isotopes into the low-energy windows for the dogs in which the percent of cardiac output going to the brain fell during CPR.

Reference arterial blood samples were obtained from two sites by allowing the blood to continuously drip passively into scintillation vials over timed intervals. The amount of blood in each vial was weighed and converted to milliliters, assuming a specific gravity of 1.05. The drip rate remained relatively steady for the entire duration of CPR, particularly over the first few minutes after sphere injection when their concentration in arterial blood was high. The flow collection rate was approximately 4 ml/min before and 2 ml/min after arrest. These rates were sufficient to yield at least 1000 spheres, typically 5000, in the entire reference sample. For each vial, a value for the counts/ml of
blood per minute was calculated. The values from each vial were added to obtain the total counts/ml/min for the entire reference sample. The average from the two sample sites was then used to calculate peripheral blood flow. This drip collection method of obtaining the reference sample was used rather than the usual syringe pump withdrawal method to obtain the concentration profile of microspheres in arterial blood over time.

Vials of blood and tissue were counted on a multi-channel autogamma scintillation spectrometer (Packard 9042). The energy windows used for $^{113}$Sn, $^{85}$Sr and $^{40}$Sc were 370–430, 480–550, and 800–1300 keV, respectively. The overlap of counts from high-energy isotopes into windows of lower-energy emission was subtracted to obtain a corrected count value for each isotope using the method of differential spectroscopy. Tissue blood flow was then calculated by dividing tissue corrected counts by the total corrected counts/ml/min in the two reference blood samples.

Since blood flows during CPR were low, the standard deviation of flow for many regions was similar in magnitude to the mean flow. Because this implies that the data are not normally distributed, the nonparametric Wilcoxon rank-sum test was used to compare the two groups. The significance level was 0.05.

Validation Studies

In addition to sampling arterial blood from two sites, microsphere mixing and lack of sedimentation under a low-flow state was tested by using a nonsedimentary marker. A solution of 20 $\mu$Ci of thallium-201 ($^{201}$Tl) was mixed in the syringe with one of the microsphere suspensions injected during CPR. The concentration profile of $^{201}$Tl over time at the arterial sampling sites was compared with that of the microspheres. Blood was collected in vials over 15-second intervals for the first 2 minutes after injection, followed by 30-second intervals for the next 2 minutes and then by 60-second intervals for the remainder of CPR. Radioactivity of $^{201}$Tl in the blood was counted over a 40–100-keV window after subtracting the crossover counts from the microspheres.

To compare the activity of $^{201}$Tl and microspheres in the blood, the respective counts were normalized by the total amount of counts injected. Because the dose of injected counts was too hot to be counted directly by the scintillation counter, the injectate was diluted to 5 ml with saline and small samples of the mixture were drawn. From the counts of these small samples and the ratio of their weight to the weight of the amount injected, an estimate of injected counts was obtained. To ensure that the microsphere sample was representative of the entire injectate, the suspension was thoroughly mixed by a magnetic stirrer and samples were taken in quintuplicate. After injection, the inside of the injectate syringe was wiped with gauze, the gauze and syringe were cut into approximately 30 small pieces, each piece was placed in a separate counting vial, and their total radioactivity was subtracted from the estimate of injected counts.

In four additional dogs, cerebral venous outflow was measured simultaneously with the microsphere technique for comparison during CPR. The venous outflow technique of Rapela and Green was used in which blood was collected from a cannula in the confluence of the sinuses and pumped back through a femoral vein. The normal outflow tracts through the lateral sinuses were occluded by wax to prevent retrograde flow from extracranial sources. The collection reservoir was open to atmosphere about 25 cm below the level of the heart.

Results

Validation Evidence

In the four preliminary dogs in which cerebral venous outflow was measured, there was general agreement with the cerebral blood flow determined by microspheres (fig. 1). During CPR, both blood flow measurements fell to a similar extent.

Collection and counting of cerebral venous blood indicated that microsphere shunting across the bed was 2.3 ± 0.5% before arrest and 4.1 ± 2.3% (± SEM) during CPR. These values are comparable to what others have found with 15-μ spheres under other conditions or with higher flows. Blood from the left ventricle was evacuated at the end of CPR. The counts per milliliter of blood were less than 0.02% of the counts injected. Also, subendocardial tissue samples did not show any unusual regional hot spots from spheres settling within the chamber.

To test for possible sedimentation of microspheres along the arterial system during the low-flow conditions of CPR, a solution of $^{201}$Tl was mixed and injected together with one of the microsphere suspensions. Examples of the arterial concentration curves over time from two different sampling sites are given in figure 2 (conventional CPR) and figure 3 (SCV CPR).

![Figure 1](http://circ.ahajournals.org/FIGURES/1.jpg)  
**Figure 1.** Cerebral blood flow measured simultaneously by microspheres and venous outflow technique before arrest and twice during cardiopulmonary resuscitation (CPR) in four dogs ($r = 0.97$).
SCV CPR produced a larger net perfusion pressure that was sustained during the compression (fig. 5).

The pressures were averaged over an integral number of ventilatory cycles and the means from both series of dogs are given in table 2. Mean carotid artery pressure was significantly greater during SCV than during conventional CPR. The mean ICP was also significantly greater during SCV CPR. Mean jugular venous pressure was lower than the respective ICP of each series. Nonetheless, net cerebral perfusion pressure, calculated as mean carotid artery pressure minus mean ICP, was significantly greater in the SCV series (14 ± 3 mm Hg) than in the conventional CPR series (5 ± 0.5 mm Hg). Total brain blood flow was also significantly higher during SCV CPR. Cerebral blood flow fell from 34.3 ± 2.4 ml/min/100 g before to 1.2 ± 0.7 ml/min/100 g at 1 minute and 0.8 ± 0.6 at 3 minutes of conventional CPR. With SCV CPR, cerebral blood flow fell from 40.0 ± 3.6 to 9.4 ± 1.5 ml/min/100 g at 1 minute and 14.1 ± 3.0 ml/min/100 g at 3 minutes. The mean cerebral blood flow was correlated with the mean perfusion pressure generated in each dog (flow = 0.75 pressure − 0.7; r = 0.77) (fig. 6).

Regional cerebral blood flow was significantly greater during SCV CPR than during conventional CPR throughout all brain regions (table 3). Comparison among regions shows that the percent fall in flow

The shape and amplitude of the 201Tl and microsphere curves were closely matched, although the 2–4-minute washout times for microspheres during CPR was an order of magnitude longer than occurs with a normal cardiac output.

Table 1 gives the mean (± SEM) of the area under the concentration curves for 10 dogs. The ratio of the areas for microspheres to 201Tl was 1.00 ± 0.03 from the subclavian artery and 1.00 ± 0.05 from the carotid artery. The absolute value of the error difference of the areas between sampling sites was 1.4 ± 0.3% for microspheres and 1.3 ± 0.2% for 201Tl. Thus, there was no evidence for poor mixing or sedimentation of the microspheres.

**Conventional vs SCV CPR**

Original records during conventional and SCV CPR are shown in figure 4. Large increases in ICP, which were in phase with chest compression, were observed. The compression after lung inflation during conventional CPR (first beat in figure 4, top) produced a larger increase in carotid artery pressure and ICP. With SCV CPR, carotid pressures were greater. Peak ICP was also greater. However, when the difference between carotid artery pressure and ICP was measured instantaneously over the entire compression cycle,
from control was uniform, with the exception of the medulla. The fall in medulla blood flow with SCV CPR (60 ± 8% and 36 ± 19% at 1 minute and 3 minutes, respectively) was significantly less than the drop in total brain blood flow (74 ± 6% and 62 ± 9% at 1 minute and 3 minutes, respectively) (Wilcoxon paired sign-rank test).

Tissue samples taken from extracranial regions of the head and from abdominal organs did not reveal any significant differences between the two methods of CPR (table 4). Left ventricular blood flow in these fibrillating hearts was low in both series. Samples of endocardium/epicardium ratio in five trials showed a large decrease from 1.12 before arrest to 0.56 during both types of CPR. Cardiac output was estimated from the microsphere washout curve in arterial blood for the one injection in which the total number of injected counts was estimated (i.e., the sphere injection with simultaneously injected 201TI). The low cardiac outputs were not significantly different between the two groups (table 4).

### Discussion

Previous work has shown that for the same compression force during mechanical CPR, augmenting the rise in intrathoracic pressure by inflating the lungs simultaneously with every chest compression combined with binding of the abdomen results in a greater arteriovenous pressure gradient and blood flow in extrathoracic carotid vessels than the standard CPR technique.1, 6, 7 The current study extends this work. Although CPR produced large phasic increases in ICP, the increased intrathoracic pressure generated during SCV CPR was transmitted to a greater extent to the carotid artery than to the intracranial space. The resulting augmentation of cerebral perfusion pressure was correlated with a greater cerebral blood flow during SCV CPR than during conventional CPR.

We examined the effect of increased intrathoracic pressure on cerebral perfusion and did not strictly compare simultaneous chest compression and ventilation with conventional CPR by having all other variables equally set. Thus, high airway pressure and abdominal binding were used with the SCV technique to maximize intrathoracic pressure swings for the same sternal displacement. A compression rate of 40/min rather than 60/min was chosen for SCV CPR because it is technically simpler when inflating the lungs simultaneously and it results in less hyperventilation. Previous experience with SCV CPR has demonstrated little effect of compression frequency on carotid blood flow.6 We believe that the effect on brain blood flow in going from a rate of 40/min to 60/min would be small compared with the difference between SCV and conventional groups. Even if cerebral blood flow were to be lower at a rate of 60/min than at 40/min with SCV CPR, we do not see any inherent problem of comparing a new method with its optimum variables to the conventional method with its standard variables.

Myocardial blood flow was approximately 5% of control with SCV CPR and abdominal binding. This was not significantly different from conventional CPR. Comparable percent values were obtained in a previous report using a cannulating flow probe.6 Phasic coronary arterial flow revealed retrograde flow during chest compression and a larger antegrade component in the early release phase. Because the increase in intrathoracic pressure is transmitted equally to all intrathoracic vascular compartments, SCV CPR provided no benefit for coronary flow during the compression phase. During the release phase, aortic pressure falls nearly as rapidly with SCV CPR as with conventional CPR, and a moderate but brief enhancement of antegrade flow was obtained.6 Therefore, increasing intrathoracic pressure during chest compression improves blood flow to the brain, but is of little benefit to the heart without adjunctive therapy for elevating aortic pressure during the release phase.

The high intraabdominal pressure with SCV CPR probably accounts for the lack of improvement in splanchnic perfusion. The enhanced carotid artery-to-jugular venous pressure gradient with SCV CPR predicts a greater perfusion of the extracranial cephalic circulation. However, samples of skin, muscle and tongue failed to show a significant effect on blood flow. These tissues have a low metabolic rate and a low prearrest blood flow. The large coefficient of variation between subjects that is associated with such low flows necessarily makes statistical detection of small blood flow differences between groups difficult to measure. Blood flow to the tongue, expressed as a percent of control, was relatively high in both groups. This suggests that the tongue is effective in its ability to autoregulate at low perfusion pressure.

### Table 1. Comparison of Microspheres and Thallium-201 in Blood from Subclavian and Carotid Artery Sampling Sites

<table>
<thead>
<tr>
<th>Counts/ml/min/counts injected</th>
<th>Subclavian</th>
<th>Carotid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Microspheres</td>
<td>0.00595 ± 0.00052</td>
<td>0.00603 ± 0.00054</td>
</tr>
<tr>
<td>201TI</td>
<td>0.00596 ± 0.00050</td>
<td>0.00604 ± 0.00076</td>
</tr>
<tr>
<td>Ratio of microsphere:Tl</td>
<td>1.00 ± 0.03</td>
<td>1.00 ± 0.05</td>
</tr>
<tr>
<td>% error between sampling sites (100 [carotid - subclavian] / [carotid + subclavian] /2)</td>
<td>1.4 ± 0.3%</td>
<td>1.3 ± 0.2%</td>
</tr>
</tbody>
</table>
Use of the radiolabeled microsphere technique to measure cerebral blood flow during low cardiac output states in the present experimental conditions requires that the assumption of the technique remain valid. The assumption that spheres were well mixed upon leaving the heart appears to be valid because of the good agree-

### TABLE 2. Cerebral Hemodynamics

<table>
<thead>
<tr>
<th></th>
<th>Prearrest control</th>
<th>Conventional CPR</th>
<th>SCV CPR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1 min</td>
<td>3 min</td>
</tr>
<tr>
<td>Mean carotid artery pressure (mm Hg)</td>
<td>133 ± 7</td>
<td>27 ± 2</td>
<td>22 ± 1</td>
</tr>
<tr>
<td>Mean ICP (mm Hg)</td>
<td>12 ± 2</td>
<td>22 ± 2</td>
<td>17 ± 1</td>
</tr>
<tr>
<td>Mean jugular venous pressure (mm Hg)</td>
<td>2 ± 0.7</td>
<td>18 ± 2</td>
<td>14 ± 2</td>
</tr>
<tr>
<td>Mean carotid artery pressure – mean ICP (mm Hg)</td>
<td>122 ± 7</td>
<td>5.0 ± 0.6</td>
<td>4.4 ± 0.5</td>
</tr>
<tr>
<td>Cerebral blood flow (ml/min/100 g)</td>
<td>34.3 ± 2.4</td>
<td>1.2 ± 0.7</td>
<td>0.8 ± 0.6</td>
</tr>
</tbody>
</table>

Values are mean ± SEM. Abbreviations: CPR = cardiopulmonary resuscitation; ICP = intracranial pressure; SCV-CPR = CPR with simultaneous compression-ventilation.
ment of microsphere concentration at two different arterial sampling sites. There was no evidence of microspheres settling within the left ventricle from blood sampled at the end of CPR. The assumption that the spheres remain well mixed while traversing the arterial system also appears valid. Simultaneous injection of a nonsedimentary marker, 201TI in solution, demonstrated that its time course in arterial blood was superimposable with that of the microsphere suspension. This would not be expected if there was significant sedimentation of microspheres along the arterial system. Although sedimentation could have occurred distal to the carotid site, this is not likely. The distribution of microspheres to right and left structures of the brain were similar. One could argue that right-left comparisons would not reflect sedimentation because gravity acts perpendicularly to right-left arterial branching symmetry. However, because rostral and caudal areas of the brain, which are supplied by different branching geometries of the carotid and vertebral arterial systems, also showed similar flow patterns, sedimentation is not a significant problem. The observation that one caudal area, the medulla, exhibited a smaller decrease in flow does not violate this argument because others have found that medullary blood flow is better autoregulated at low perfusion pressures.21,22

Another assumption of the microsphere technique is that all spheres are trapped on the first pass. This assumption is valid because cerebral venous sampling during CPR did not show a substantial increase in shunting. Finally, the low blood flows obtained simultaneously with the cerebral venous outflow technique further support the validity of the microsphere technique. The venous outflow technique was not chosen as the standard method for comparing CPR techniques because of a potential problem: Previous observations suggest that when a large venous pressure gradient is produced between the area being drained at the confluence of sinuses and the remaining cranial drainage, venous-venous anastomoses may open up under certain conditions.21 Thus, the area being drained by the venous outflow technique may not remain constant when different CPR techniques are applied that generate different venous pressures.

The cerebral blood flows in our experiments are much lower than those in a study by Voorhees et al.,7 using microspheres during conventional CPR. Methodologic considerations may account for this difference. In their study,7 the arterial reference withdrawal pump was stopped 2 minutes after the microsphere injection and the dog was then defibrillated. Possibly, at the end of CPR any spheres remaining in the left ventricle, though small in number, would then go to the brain in high concentration upon commencing reperfusion and would then substantially overestimate cerebral blood flow. Our data indicate that the washout of microspheres is usually not complete by 2 minutes. In addition, few hemodynamic measurements were reported in these dogs. Higher cerebral flows may have been the result of marked increases in intrathoracic pressure in these much smaller dogs (6–12 kg) with sternal compression as a result of marked chest deformation, tracheal obstruction during compression, and/or pneumothorax. Also, heart compression, with consequent higher cerebral flow, may be more commonly seen in small dogs.1,16

In a recent extended abstract, Bircher and Safar16 did not report significant improvement in cerebral perfusion pressure with SCV CPR. Differences in experimental design may explain these discrepant findings. First, much lower intrathoracic and carotid artery pressures were generated during SCV CPR than in the present study. Second, the use of small dogs (10–15 kg) in their study may have resulted in direct cardiac compression and, therefore, better results with conventional CPR. In situations in which direct cardiac compression can be achieved, cerebral perfusion may be enhanced because arterial pressure would increase without as large an increment as in ICP.

Cerebral perfusion during SCV CPR without direct vascular compression is apparently limited by the large phasic increases in ICP. Understanding the mechanism whereby intrathoracic pressure is transmitted to the cranium and assessing its relative role in man is important before clinically applying newer CPR techniques.15 Transmission may occur through the arterial system, the venous system, or directly through the intervertebral foramina to the spinal fluid and venous plexus. Studies on dogs with the application of positive end-expiratory pressure suggest that pressure transmission occurs primarily by the venous system under these conditions.24 However, our own preliminary data with SCV CPR indicate that direct spinal transmission of intrathoracic pressure does play a role in generating the phasic rise of ICP in conjunction with the jugular venous system.25 This was based on the observation that ICP more closely tracked large, rapid fluctuations in intrathoracic pressure than the small jugular pressure...
responses. This study also showed that the arterial system was not essential for the pressure transmission to ICP. The nonlinear pressure-volume characteristic of the cranium is an important determinant of the rise in ICP when a patient is placed on positive end-expiratory pressure.\textsuperscript{13} It may also be a key variable in determining the amplitude of the ICP rise during CPR in a particular patient. This is supported by our preliminary observations in dogs in which raising baseline ICP increased the degree of intrathoracic pressure transmission to ICP.\textsuperscript{25}

Although cerebral perfusion was substantially improved with SCV CPR in the present study, the flow was, on the average, only one-third of control. The brains of most species extract 40–60% of the $O_2$ supply under control conditions.\textsuperscript{26} Thus, the $O_2$ extraction reserve is presumably not sufficient for fully maintaining basal $O_2$ uptake at this level of cerebral blood flow.

The question of whether this level of cerebral blood flow is adequate for full cerebral resuscitation is complicated because this matter is also a function of anesthesia, duration of total cerebral ischemia before instituting CPR, duration of CPR, and a variety of reperfusion factors. However, data from the literature on blood flow thresholds for different stages of cerebral ischemia do provide some frame of reference. Significant changes in the characteristics of the EEG and somatosensory-evoked potential begin to occur when cerebral blood flow is reduced to approximately one-third of control,\textsuperscript{27, 28} and these electrical changes correlate with the neuropathology.\textsuperscript{29, 30} The large increase in extracellular potassium activity and fall in extracellular calcium activity during cerebral ischemia appear to require a greater reduction in cerebral blood flow, to less than 10 ml/min/100 g.\textsuperscript{31} Thus, one can conclude that the improvement in cerebral blood flow
provided by SCV CPR over the conventional technique is in the range that would result in a significant amelioration of cerebral function in the present animal model.

The improvement in brain blood flow observed with SCV CPR with abdominal binding over that with conventional CPR is of obvious clinical interest. Application of the present findings to man is limited by the lack of information with regard to the frequency of occurrence of direct cardiac and vascular compression during conventional CPR in man and to its effectiveness in perfusing the brain and heart relative to SCV CPR. In a recent study of five patients by Werner et al., no echocardiographic evidence of direct heart compression was found, which suggests that it does not occur frequently during conventional CPR in man. Other considerations for applying the SCV technique is its limited benefit for myocardial blood flow and the need for patient intubation, specialized equipment and well-trained personnel. Although initial clinical observations of blood pressure changes appear promising with these new approaches, studies of efficacy in terms of patient survival and neurologic sequela and complication rate are essential.

Acknowledgment
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References

Table 4. Peripheral Organ Blood Flow

<table>
<thead>
<tr>
<th></th>
<th>Conventional CPR</th>
<th></th>
<th>SCV CPR</th>
<th></th>
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<tbody>
<tr>
<td></td>
<td>Prearrest 1 min</td>
<td>3 min</td>
<td>Prearrest 1 min 3 min</td>
<td></td>
</tr>
<tr>
<td>Left ventricle</td>
<td>161 ± 23.5</td>
<td>5.3 ± 2.8</td>
<td>2.9 ± 1.7</td>
<td>126 ± 14.7</td>
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<tr>
<td>Regions of head</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skin</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Ventral</td>
<td>4.9 ± 1.0</td>
<td>0.6 ± 0.1</td>
<td>0.7 ± 0.1</td>
<td>6.5 ± 2.3</td>
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<tr>
<td>Dorsal</td>
<td>7.6 ± 2.4</td>
<td>1.2 ± 0.4</td>
<td>2.7 ± 1.7</td>
<td>6.8 ± 3.0</td>
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<tr>
<td>Muscle</td>
<td></td>
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<tr>
<td>Ventral</td>
<td>(myloboideus)</td>
<td>29.8 ± 8.1</td>
<td>3.2 ± 1.1</td>
<td>3.7 ± 1.1</td>
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<tr>
<td>Dorsal</td>
<td>(temporalis)</td>
<td>31.3 ± 7.2</td>
<td>1.9 ± 0.6</td>
<td>2.5 ± 0.4</td>
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<td>Bone (frontal)</td>
<td>7.9 ± 2.3</td>
<td>0.1 ± 0.05</td>
<td>0.1 ± 0.03</td>
<td>12.6 ± 5.5</td>
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<tr>
<td>Tongue</td>
<td>7.2 ± 2.4</td>
<td>3.0 ± 0.6</td>
<td>3.8 ± 1.2</td>
<td>5.4 ± 1.5</td>
</tr>
<tr>
<td>Abdominal organs</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kidney</td>
<td>362 ± 22</td>
<td>3.4 ± 1.1</td>
<td>4.9 ± 1.3</td>
<td>328 ± 21</td>
</tr>
<tr>
<td>Jejunum</td>
<td>40.1 ± 5.9</td>
<td>1.5 ± 0.3</td>
<td>2.0 ± 0.5</td>
<td>36.3 ± 8.0</td>
</tr>
<tr>
<td>Pancreas</td>
<td>28.0 ± 3.4</td>
<td>0.8 ± 0.3</td>
<td>1.4 ± 0.7</td>
<td>29.5 ± 7.3</td>
</tr>
<tr>
<td>Spleen</td>
<td>183 ± 43</td>
<td>0.0 ± 0.0</td>
<td>0.0 ± 0.02</td>
<td>229 ± 63</td>
</tr>
<tr>
<td>Liver</td>
<td>(hepatic artery)</td>
<td>43.6 ± 8.2</td>
<td>0.1 ± 0.06</td>
<td>0.1 ± 0.06</td>
</tr>
<tr>
<td>Cardiac output</td>
<td>147 ± 21</td>
<td></td>
<td></td>
<td>184 ± 19</td>
</tr>
</tbody>
</table>

Values are mean ± SEM.
Cardiac output (ml/min) is based on estimate of number of microspheres injected at either 1 or 3 minutes. Organ flows (ml/min/100 g) are not significantly different between conventional and SCV CPR.
Abbreviations: CPR = cardiopulmonary resuscitation; SCV CPR = CPR with simultaneous compression-ventilation.
Augmentation of cerebral perfusion by simultaneous chest compression and lung inflation with abdominal binding after cardiac arrest in dogs.
R C Koehler, N Chandra, A D Guerci, J Tsitlik, R J Traystman, M C Rogers and M L Weisfeldt

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