Regional Blood Flow During Cardiopulmonary Resuscitation in Dogs Using Simultaneous and Nonsimultaneous Compression and Ventilation

John M. Luce, M.D., Brian K. Ross, Ph.D., Ronald J. O'Quin, M.D., Bruce H. Culver, M.T., Murali Sivarajan, M.D., David W. Amory, M.D., Ph.D., Robert A. Niskanen, M.S., Clif A. Alferness, B.S., Wayne L. Kirk, L. Bruce Pierson, and John Butler, M.D.

SUMMARY We studied regional blood flow (Qr) using radiolabeled microspheres and measured hemodynamic variables in 20 anesthetized dogs in normal sinus rhythm and during ventricular fibrillation treated with cardiopulmonary resuscitation (CPR). Nonsimultaneous compression and ventilation CPR (NSCV-CPR) was performed in seven dogs with a pneumatic piston that gave 50 chest compressions/min with an open artery with 10 ventilations at an airway pressure of 33 mm Hg interposed between each fifth and sixth compression. Simultaneous compression and ventilation (SCV-CPR) was performed in seven dogs with the piston and in six other dogs with a circumferential pneumatic vent. Both devices gave 30 compressions/min simultaneously with 30 ventilations that elevated airway pressure to 80 mm Hg. The abdomen was bound during SCV-CPR. Regional blood flow (mean ± SD) to the cerebral hemispheres, cardiac ventricles, and kidneys, expressed as ml/min/100 g tissue, was 3.1 ± 4.0, 3.4 ± 3.3 and 1.5 ± 1.5, respectively, during NSCV-CPR; 11.5 ± 5.9, 4.9 ± 4.7 and 2.7 ± 2.7 during SCV-CPR (vest); and 16.2 ± 7.2, 11.0 ± 4.0 and 20.1 ± 20.2 during SCV-CPR (piston) (all p < 0.05 compared with NSCV-CPR). These results indicate that Qr to all organs studied is reduced below normal sinus rhythm levels during CPR for ventricular fibrillation. Qr to the brain is proportionately greater than Qr to the heart and kidneys, and Qr to the brain is greater with both forms of SCV-CPR than with NSCV-CPR.

Because cardiopulmonary arrest is one of the greatest stresses the body can suffer, the success of cardiopulmonary resuscitation (CPR) depends upon maintaining vital organ perfusion at a level consistent with full recovery of function after resuscitation. Conventional or nonsimultaneous ventilation and compression CPR (NSCV-CPR) involves the administration of closed-chest compressions with an open airway with ventilations at a low airway pressure interposed between every fifth and sixth compression. In contrast to this approach, chest compressions may be administered simultaneously with ventilations at a high airway pressure, a combination called new or simultaneous compression ventilation CPR (SCV-CPR). Although Chandra et al demonstrated that arterial pressures and blood flow in the carotid artery are greater during SCV-CPR than during NSCV-CPR, comparisons of regional blood flow (Qr) between the two methods have not been reported. We therefore used the radiolabeled-microsphere technique to determine Qr in dogs in normal sinus rhythm (NSR) and during ventricular fibrillation (VF) treated with NSCV-CPR and two forms of SCV-CPR.

Methods

Twenty adult mongrel dogs, mean weight 23.7 kg, unselected for chest configuration, were studied. Anesthesia was induced with thiopental sodium (1 g) and maintained with ketamine (mean dose 0.6 ± 0.3 g; mean time of administration after thiopental 89 ± 44 minutes). The dogs were intubated with an endotracheal tube, the proximal end of which extended 2–4 cm outside the mouth. The cuff was inflated to achieve an airtight seal; the exact amount of air inserted was not measured. The dogs were ventilated with a mixture of O2 and CO2 to give an arterial CO2 tension (Paco2) of 30–45 mm Hg during control and CPR runs. Airway pressure was measured continuously.

A left thoracotomy was performed, and catheters were placed in the left atrium for measurement of left atrial pressure (PLa) and microsphere injection and in the left ventricle for collection of blood samples to detect residual microspheres after control and CPR runs. The chest was evacuated of air and closed to achieve an airtight seal. The dogs were supine. Catheters were inserted in the thoracic aorta via the left femoral artery for collection of arterial blood gases and microsphere reference samples, in the left carotid artery for measurement of left carotid artery pressure (PLeCA), in the left external jugular vein via the left maxillary vein for measurement of left external jugular
venous pressure ($P_{LEV}$) and in the right atrium for measurement of right atrial pressure ($P_{RA}$). Aortic pressure ($P_{AO}$) could not be measured continuously during the study because the aortic catheters were being used for microsphere collection. Right atrial pressure, therefore, was used to reflect intrathoracic pressure changes during NSR and CPR. A transvenous right ventricular pacing catheter was used to induce VF; its location was confirmed by an intracardial electrocardiographic tracing displayed on a monitor. The dogs were heparinized (300 U/kg) and cannulating electromagnetic flowmeters were placed in the left and right carotid arteries to measure left and right carotid artery blood flows ($Q_{LCA}$, $Q_{RCA}$). Left atrial pressure at end-expiration was maintained at 2–5 mm Hg before NSR and CPR by injecting normal saline. All pressure transducers were calibrated with reference to zero at the level of the right atrium.

The study procedure in each dog was as follows. During the control period, microspheres with a mean diameter of 15 ± 3 μm, labeled with $^{85}$Sr or $^{46}$Sc and suspended in 20% dextran, were injected into the left atrium over 45 seconds. A reference blood sample was withdrawn from the aortic catheter using a Harvard pump at a rate of 3.1 ml/min for 5 minutes starting 10 seconds before the microsphere injection. Vascular pressures and carotid artery flows were measured at 2, 4, and 6 minutes. At 7 minutes, arterial blood gases were drawn and a 15-ml blood sample was taken from the left ventricle to determine whether residual microspheres were present. Fifteen minutes later, VF was induced by 60-Hz electric stimulation of the right ventricle. One type of CPR only was initiated immediately in each dog.

NSCV-CPR was performed in seven dogs with a pneumatic piston (Michigan Instruments, Inc.) that gave 50 chest compressions/min with an open airway with 10 ventilations at an airway pressure of 33 mm Hg interposed between every fifth and sixth compression. The compression:relaxation time ratio was 60:40 during NSCV-CPR, and the piston caused a 6.5-cm sternal depression. The combination of 50 compressions and 10 ventilations/min with a compression duration of 60% of the cycle was chosen (rather than the combination of 60 compressions and 12 ventilations/min with a compression duration of 50% as recommended by the American Heart Association1) after a suggestion by Taylor et al.3 that lower compression rates and longer compression durations may be useful in augmenting carotid blood flow during conventional CPR. Chandra et al.3 used a compression rate of 60/min and a duration of 60% with a 6-cm sternal depression during NSCV-CPR.

SCV-CPR was performed in seven dogs with the piston and in six other dogs with a prototype model of a rapidly inflatable circumferential vest (Physio-Control Corp.). During SCV-CPR, both devices gave 30 chest compressions/min simultaneously with 30 ventilations resulting in elevation of airway pressure to 80 mm Hg. Lung inflation was achieved with the piston by a Ven-

turi device in which room air was entrained with an O₂-CO₂ mixture to reach the preset airway pressure of 80 mm Hg. With the vest, this gas mixture was used to rapidly fill a cylinder; once a pressure of 80 mm Hg was reached, the cylinder was emptied into the airway. Tidal volumes were not measured with the devices, but were assumed to exceed 15 ml/kg body weight. The compression:relaxation time ratio was 60:40 with both devices. The piston caused a 6.5-cm sternal depression. The pressure in the vest was 80 mm Hg. An abdominal binder was continuously inflated to 100 mm Hg during SCV-CPR, but was not used during NSCV-CPR. Chandra et al.2 used a compression rate of 40/min and a duration of 60% with a 6-cm sternal depression, but without an abdominal binder during new or SCV-CPR.

Radiolabeled microspheres were injected 2 minutes after CPR was initiated, and the reference sample was collected over the subsequent 5 minutes, as during the control period. Vascular pressures and carotid flows again were measured at 2, 4 and 6 minutes. At 7 minutes, arterial blood gases were drawn, the left ventricular residual sample was collected, and CPR was discontinued. A necropsy was performed immediately to ascertain the presence of major trauma to the intrathoracic and intraabdominal organs and to examine the jugular veins; a microscopic examination was not conducted. The tongue, temporal muscle, left and right cerebral hemispheres, cerebellum, midbrain and brain stem, left and right cardiac ventricles, and left and right kidneys were removed. The organs were weighed and placed in vials for gamma counting. The radioactivity of these samples and of the reference and left ventricular residual samples was measured using a sodium-iodide scintillation counter. Regional blood flow (ml/min/100 g tissue) was calculated from these data by the method described by Heymann et al.4

The unpaired $t$ test was used to compare variables between dogs receiving different types of CPR. Differences were considered statistically significant at $p \leq 0.05$.

Results

No significant differences in body weight or type or amount of anesthesia used were observed among the dogs receiving NSCV-CPR, SCV-CPR (piston), or SCV-CPR (vest). Left atrial pressure was similar before control and CPR runs in all three groups. Arterial pH and $P_{CO_2}$ also were similar in all three groups; $P_{O_2}$ differed because $CO_2$ was added to maintain $P_{O_2}$ at 30–45 mm Hg during CPR and because the piston device entrained room air (table 1). Despite these similarities, $P_{RA}$, a reflection of intrathoracic pressure, was significantly higher in compression and relaxation during both types of SCV-CPR than during NSCV-CPR. $P_{LCA}$ was also significantly higher in compression and relaxation during SCV-CPR of both types than during NSCV-CPR. The increase in $P_{LCA}$ also was greater than the increase in $P_{LEV}$, so a greater pressure gradient for antegrade blood flow across the cerebral circulation was created between the left carotid artery and the left
external jugular vein in the dogs receiving SCV-CPR. The increase in $P_{RA}$ was greater than the increase in $P_{LEV}$ in these dogs, and resulted in a greater pressure gradient between the right atrium and the left external jugular vein in the dogs receiving SCV-CPR. This gradient presumably resulted from closure of thoracic inlet valves and could protect the brachiocephalic venous bed from the high intrathoracic pressure and retrograde blood flow. Valves in the left external jugular vein were found by dissection in two representative dogs from each group.

$Q_{LCA}$ was significantly higher during SCV-CPR of both types than during NSCV-CPR (fig. 1). However, $Q_{RCA}$ did not differ. Subsequent cineangiograms and necropsy studies in dogs treated with SCV-CPR have shown that the right and, rarely, the left carotid arteries can be narrowed by tracheal expansion and dissection related to the high airway pressure achieved during SCV-CPR (Ross BK, Rosborough J, Niemann JT, Ung S, Luce JM, Butler J: unpublished observations). Since these studies may not have duplicated our experimental corrections, we can only assume — but have not demonstrated — that right carotid artery narrowing explains the limited $Q_{RCA}$ in our dogs.

Only 1–2% of the total microspheres injected remained in the left ventricle after control and CPR studies, and no differences in residual counts were observed during different types of CPR. These findings suggested a good washout of microspheres during all situations. Flow to paired organs also was equal, suggesting good microsphere mixing and distribution. No differences in $Q_B$ were observed during NSR in the three groups of dogs, and because statistical comparisons were made only between different types of CPR, $Q_B$ control data were pooled (table 2, figs. 2–6). Regional flow to the cerebral hemispheres, cerebellum, and midbrain and brain stem was greater during both forms of SCV-CPR than during NSCV-CPR. Flow to the left and right ventricles and kidneys was greater during SCV-CPR (piston) than during NSCV-CPR. Flow to the midbrain and brain stem, the left ventricle, and the left and right kidneys was greater during SCV-CPR (piston) than during SCV-CPR (vest). Regional flow to the tongue and temporal muscle was the same during all types of CPR. No gross evidence of trauma to intrathoracic or intraabdominal organs was observed at necropsy after CPR.

**Discussion**

Our investigation shows that $Q_B$ is markedly reduced during the types of CPR we studied. However, $Q_B$ to the brain is proportionately greater than $Q_B$ to the myocardium and the kidneys. Finally, $Q_B$ to the brain is greater with both forms of SCV-CPR than with NSCV-CPR. These findings suggest that redistribution of $Q_B$ occurs during CPR and that the brain receives more blood than the intrathoracic and intraabdominal

---

**TABLE 1. Arterial Blood Gas and Vascular Pressure Data During Cardiopulmonary Resuscitation**

<table>
<thead>
<tr>
<th></th>
<th>Control (n = 20)</th>
<th>NSCV-CPR (vest) (n = 7)</th>
<th>SCV-CPR (vest) (n = 6)</th>
<th>SCV-CPR (piston) (n = 7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood gases</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$PH_a$</td>
<td>7.36 ± 0.03</td>
<td>7.35 ± 0.07</td>
<td>7.25 ± 0.06</td>
<td>7.29 ± 0.11</td>
</tr>
<tr>
<td>$Paco_2$ (mm Hg)</td>
<td>34 ± 2</td>
<td>33 ± 5</td>
<td>36 ± 3</td>
<td>39 ± 5</td>
</tr>
<tr>
<td>$Pao_2$ (mm Hg)</td>
<td>530 ± 94</td>
<td>323 ± 74</td>
<td>518 ± 121</td>
<td>253 ± 120</td>
</tr>
<tr>
<td>Vascular pressures</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$P_{RA}$ (mm Hg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>systole/compress</td>
<td>6 ± 2</td>
<td>24 ± 3</td>
<td>60 ± 3*</td>
<td>71 ± 2*</td>
</tr>
<tr>
<td>diastole/relax</td>
<td>1 ± 1</td>
<td>12 ± 3</td>
<td>12 ± 3</td>
<td>10 ± 2</td>
</tr>
<tr>
<td>$P_{LCA}$ (mm Hg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>systole/compress</td>
<td>155 ± 8</td>
<td>25 ± 2</td>
<td>64 ± 2*</td>
<td>71 ± 3*†</td>
</tr>
<tr>
<td>diastole/relax</td>
<td>114 ± 6</td>
<td>16 ± 2</td>
<td>20 ± 2*</td>
<td>24 ± 2*†</td>
</tr>
<tr>
<td>$P_{LEV}$ (mm Hg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>systole/compress</td>
<td>3 ± 2</td>
<td>15 ± 1</td>
<td>41 ± 3*</td>
<td>46 ± 4*</td>
</tr>
<tr>
<td>diastole/relax</td>
<td>3 ± 1</td>
<td>8 ± 5</td>
<td>19 ± 2*</td>
<td>25 ± 4*†</td>
</tr>
<tr>
<td>$P_{RA}$-$P_{LEV}$ (mm Hg) systole/compress</td>
<td>152 ± 3</td>
<td>10 ± 3</td>
<td>22 ± 5*</td>
<td>46 ± 2*†</td>
</tr>
</tbody>
</table>

* $p < 0.05$ vs NSCV-CPR.
† $p < 0.05$ vs SCV-CPR (vest).

Abbreviations: CPR = cardiopulmonary resuscitation; NSCV-CPR = nonsimultaneous compression and ventilation CPR; SCV-CPR = simultaneous compression and ventilation CPR; $PH_a$ = arterial pH; $Paco_2$ = arterial $CO_2$ tension; $Pao_2$ = arterial $O_2$ tension; $P_{RA}$ = right atrial pressure; $P_{LCA}$ = left carotid artery pressure; $P_{LEV}$ = left external jugular vein pressure.
organisms. Although we did not measure survival or organ function, this redistribution should have major physiologic consequences. Reports of patients undergoing carotid endarterectomy\textsuperscript{5} and animal studies involving middle cerebral artery occlusion\textsuperscript{6-8} suggest that neurologic function can be maintained during cerebral ischemia if $Q_r$ to the brain remains greater than 16-18 ml/min/100 g tissue. By this standard, neurologic recovery could be expected in a majority of the dogs receiving SCV-CPR in our study and in none receiving NSCV-CPR. However, a $Q_r$ of 20-25 ml/min/100 g tissue is considered necessary to meet the heart’s metabolic needs during VF.\textsuperscript{9} This level was achieved in only one of the dogs in our study, a dog treated with SCV-CPR (piston).

Our results regarding left carotid artery pressures and flows during CPR are in accord with those reported by Chandra et al.\textsuperscript{2} However, our $Q_r$ data differ from a recent study involving microspheres by Vorhees et al.\textsuperscript{10} The difference between their study and ours may be attributable to differences in sedation and anesthesia, chest compliance of experimental animals, arterial blood gas tensions, site of microsphere injection, collection time for reference samples, sequence of CPR runs, and possible limitations of the microsphere technique in measuring blood flow during low flow states. Dawson et al.\textsuperscript{11} demonstrated that ketamine decreases cerebral vascular resistance and increases cerebral $Q_r$ by 80\% immediately after its administration, but that cerebral $Q_r$ returns to control levels 30 minutes later. More than 30 minutes elapsed between ketamine administration and data collection in our dogs. Further-

![Figure 1. Left and right carotid blood flow ($Q$) (mean and individual values) during cardiopulmonary resuscitation (CPR) in dogs using nonsimultaneous (NSCV-CPR) and simultaneous compression and ventilation (SCV-CPR). Control $Q$ (mean ± sd) is also shown. The difference between left and right carotid $Q$ during SCV-CPR may be due to tracheal compression from the high airway pressures used.]

<table>
<thead>
<tr>
<th>Table 2. Regional Blood Flow During Cardiopulmonary Resuscitation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control</strong> (n = 20)</td>
</tr>
<tr>
<td>----------------------</td>
</tr>
<tr>
<td>Left cerebral hemisphere</td>
</tr>
<tr>
<td>Right cerebral hemisphere</td>
</tr>
<tr>
<td>Cerebellum</td>
</tr>
<tr>
<td>Midbrain and brainstem</td>
</tr>
<tr>
<td>Left ventricle</td>
</tr>
<tr>
<td>Right ventricle</td>
</tr>
<tr>
<td>Left kidney</td>
</tr>
<tr>
<td>Right kidney</td>
</tr>
<tr>
<td>Tongue</td>
</tr>
<tr>
<td>Temporal muscle</td>
</tr>
</tbody>
</table>

Values are expressed as ml/min/100 g tissue (mean ± sd).
* $p < 0.05$ vs NSCV-CPR.
† $p < 0.05$ vs SCV-CPR (vest).
Abbreviations: CPR = cardiopulmonary resuscitation; NSCV-CPR = nonsimultaneous compression and ventilation CPR; SCV-CPR = simultaneous compression and ventilation CPR.
more, cerebral $Q_R$ decreases and then stabilizes if ketamine is preceded by thiopental, which we used for induction of anesthesia. It is not known whether cerebral $Q_R$ is affected if ketamine is given an hour or more after thiopental, as in our dogs, or whether these anesthetics affect cerebral $Q_R$ when CPR is performed during VF. The effects of arterial pH, $CO_2$ and $O_2$ during VF with or without CPR are also unclear, although we suspect that in this situation the cerebral vessels are maximally vasodilated. We controlled these variables in our study to provide uniformity in arterial blood gas tensions during different types of CPR.

Koehler et al.\textsuperscript{12} compared the use of microspheres to use of the thallium technique and to the cerebral venous outflow method in measuring cerebral $Q_R$ under CPR conditions. They consider the microsphere technique adequate in measuring cerebral $Q_R$ if the microspheres are injected into the left atrium and if the aortic reference sample is withdrawn over a long period. We injected the microspheres into the left atrium, collected the reference sample over 5 minutes, studied paired organs wherever possible, performed only one CPR run per dog, and confirmed the lack of a significant number of residual microspheres in the left ventricle before necropsy. Our data concerning cerebral $Q_R$ are quite similar to those of Koehler et al.,\textsuperscript{12} who used the microsphere technique. Although this technique may have some limitations regarding circulatory beds other than the brain in low-flow states, we believe that the redistribution of $Q_R$ we observed cannot be explained by our experimental procedures per se.

The traditional or cardiac pump model of CPR holds that the heart is squeezed between the sternum and spine during closed chest compressions, as postulated by Jude et al.\textsuperscript{13} This is thought to produce an artificial systole that increases right and left ventricular pressures above those in the pulmonary artery and aorta.
and forces blood through the great vessels. Release of the pressure and recoil of the thoracic cage during artificial diastole allows the heart to fill with blood and creates a negative intrathoracic pressure that enhances venous return. Although positive pressure is exerted on the venous as well as the arterial system with each compression, blood is assumed to flow in an antegrade fashion rather than retrograde up the jugular vein and other vessels because of the one-way arrangement of the heart valves.

In contrast to this model of the heart as a pump, Rudikoff et al.\textsuperscript{14} suggested that the entire thorax also can pump blood. In this model, closed-chest compression is seen as causing a generalized increase in intrathoracic pressure that is transmitted equally to the heart, great vessels and extrathoracic arteries. This renders the heart valves incompetent and prevents the development of the intrathoracic pressure gradient implicit in the model proposed by Jude et al.\textsuperscript{15} However, an extrathoracic gradient between the carotid artery and jugular vein is created because the carotid artery remains patent while the jugular vein collapses because of the high intrathoracic pressure at the thoracic outlet or the presence of venous valves. The actions of these valves have been documented in dogs by Niemann et al.\textsuperscript{16} and in humans by Fisher and colleagues.\textsuperscript{16} This valve mechanism also prevents retrograde flow into the cerebral circulation.

In keeping with the thoracic pump model, the superiority of cerebral $Q_R$ during SCV-CPR in our study may be attributable to the higher intrathoracic pressure and the greater pressure difference between $P_{LCA}$ and $P_{JUG}$ achieved with this modality. That this gradient was greater during SCV-CPR (piston) than during SCV-CPR (vest) may explain the slightly higher $Q_R$ to the midbrain and brain stem with the piston. We predict that $Q_R$ to the brain would have been equal if intrathoracic pressure were the same during SCV-CPR.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure4}
\caption{Regional blood flow ($Q_R$) (mean and individual values) to the tongue and temporal muscle during cardiopulmonary (CPR) in dogs using nonsimultaneous (NSCV-CPR) and simultaneous compression and ventilation (SCV-CPR). Control $Q_R$ (mean ± sd) is also shown.}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure5}
\caption{Regional blood flow ($Q_R$) (mean and individual values) to the left and right ventricular myocardium during cardiopulmonary resuscitation (CPR) in dogs using nonsimultaneous (NSCV-CPR) and simultaneous compression ventilation (SCV-CPR). Control $Q_R$ (mean ± sd) is also shown.}
\end{figure}
with the vest and piston, as can be achieved with a newer model of the vest presently under study.

Despite the adequate cerebral $Q_R$ achieved during SCV-CPR with both devices, $Q_R$ to the heart and kidneys overall was severely compromised during all of the types of CPR we studied. The low $Q_R$ to the heart is probably due to the lack of a significant pressure difference across the coronary circulation ($P_{Ao} - P_{RA}$) when intrathoracic pressure is increased during CPR. This situation might have been aggravated in our study by the fact that only 40% of each artificial cardiac cycle was devoted to the relaxation phase. Unfortunately, we did not measure $P_{Ao} - P_{RA}$ or vary the compression-relaxation in our study, although we are doing so in a current investigation. Similarly, renal $Q_R$ may have been limited because a pressure gradient was not developed across the renal circulation in our present study. Niemann et al. demonstrated that retrograde venous flow is present, but that anterograde arterial flow is negligible below the diaphragm in dogs receiving CPR.

The application of our findings to humans is uncertain. In our dogs, SCV-CPR might be expected to maintain cerebral function but not the viability of the heart or kidneys. At the same time, NSCV-CPR would not be expected to contribute significantly to any organ homeostasis. Nevertheless, bystander-initiated NSCV-CPR for VF was associated with 43% survival rate in the series from Seattle. This suggests that the published $Q_R$ requirements during VF are less than reported (which we doubt), that the microsphere technique in our hands underestimated $Q_R$ during NSCV-CPR (and perhaps SCV-CPR as well), or that CPR is more effective when applied to humans than to dogs. We performed mechanical NSCV-CPR and SCV-CPR in our dogs in a manner similar to that of other investigators and achieved similar results in the variables we studied in common. Byrne et al. administered NSCV-CPR to dogs in the lateral position and reported $Q_R$ values in the same range as we did, so the style of giving NSCV-CPR does not appear to be an important factor. Clearly, further investigation is needed to increase our understanding of how $Q_R$ is redistributed during CPR in humans as well as primates and other experimental animals. We must also improve our ability to deliver blood to the heart as well as to the head before we substitute SCV-CPR for NSCV-CPR.

**References**

1. American Heart Association: Standards and guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). JAMA 244: 453, 1980
13. Jude JR, Kouwenhoven WB, Ing Dr, Knickerbocker GG: Cardiac...
arrest: report of application of external cardiac massage on 118 patients. JAMA 178: 1063, 1961
Regional blood flow during cardiopulmonary resuscitation in dogs using simultaneous and nonsimultaneous compression and ventilation.
J M Luce, B K Ross, R J O'Quin, B H Culver, M Sivarajan, D W Amory, R A Niskanen, C A Alfernness, W L Kirk, L B Pierson and J Butler

Circulation. 1983;67:258-265
doi: 10.1161/01.CIR.67.2.258

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1983 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/67/2/258

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/