Coronary blood flow during cardiopulmonary resuscitation in swine

RONALD F. BELLAMY, M.D., LEONIDES R. DEGUZMAN, M.D., AND DEAN C. PEDERSEN, B.S.

ABSTRACT Recent papers have raised doubt as to the magnitude of coronary blood flow during closed-chest cardiopulmonary resuscitation. We will describe experiments that concern the methods of coronary flow measurement during cardiopulmonary resuscitation. Nine anesthetized swine were instrumented to allow simultaneous measurements of coronary blood flow by both electromagnetic cuff flow probes and by the radiomicrosphere technique. Cardiac arrest was caused by electrical fibrillation and closed-chest massage was performed by a Thumper (Dixie Medical Inc., Houston). The chest was compressed transversely at a rate of 66 strokes/min. Compression occupied one-half of the massage cycle. Three different Thumper piston strokes were studied: 1.5, 2, and 2.5 inches. Mean aortic pressure and total systemic blood flow measured by the radiomicrosphere technique increased as Thumper piston stroke was lengthened (mean ± SD: 1.5 inch stroke, 23 ± 4 mm Hg, 525 ± 195 ml/min; 2 inch stroke, 33 ± 5 mm Hg, 692 ± 202 ml/min; 2.5 inch stroke, 40 ± 6 mm Hg, 817 ± 321 ml/min. Both methods of coronary flow measurement (electromagnetic [EMF] and radiomicrosphere [RMS]) gave similar results in technically successful preparations (data expressed as percent prearrest flow mean ± 1 SD): 1.5 inch stroke, EMF 12 ± 5%, RMS 16 ± 5%; 2 inch stroke, EMF 30 ± 6%, RMS 26 ± 11%; 2.5 inch stroke, EMF 50 ± 12%, RMS 40 ± 20%. The phasic coronary flow signal during closed-chest compression indicated that all perfusion occurred during the relaxation phase of the massage cycle. We concluded that coronary blood flow is demonstrable during closed-chest massage, but that the magnitude is unlikely to be more than a fraction of normal.


SINCE a deficiency of coronary blood flow is probably an etiologic factor in many instances of cardiac arrest, it would be important to perform cardiopulmonary resuscitation in a manner that would optimize myocardial perfusion. The validity of the method used to measure coronary flow is a crucial factor in evaluating studies of experimental cardiopulmonary resuscitation, which are directed toward developing techniques to optimize myocardial perfusion. It is by no means certain that the techniques that have been used with such success to measure coronary blood flow in the beating heart — electromagnetic flow probes and the radiomicrosphere technique — will be equally satisfactory during cardiac massage. We describe experiments in which these techniques can be simultaneously used to ascertain their effectiveness in measuring coronary blood flow during cardiopulmonary resuscitation.

Methods

In conducting our research we adhered to the Guide for Laboratory Animal Facilities and Care as promulgated by the Committee on the Guide for Laboratory Animal Resources, National Academy of Sciences, National Research Council.

Several ancillary experiments were performed before the main study. These experiments answered questions about methods, such as the appropriate direction in which to compress the chest and the optimal duration of collection of the microsphere reference sample. We will first describe the experiments of the main study and then indicate in what manner the ancillary experiments differed.

Blood flow in the distribution of the circumflex branch of the left coronary artery was measured with both an electromagnetic flow probe and radiomicrospheres. A mechanical chest compressor (Thumper) was used in all experiments, and the effect of different piston strokes was determined. We paid special attention to determining radiomicrosphere labeling in the distributions of the anterior descending and circumflex arteries. It was assumed that obstruction of the circumflex artery by a mechanically unstable flow probe would be apparent as a decrease in the radioactivity in the distribution of the circumflex artery compared with the distribution in the anterior descending artery. Finally, the adequacy of microsphere-mixing was assessed by simultaneously withdrawing reference samples from both the abdominal aorta and the ascending aorta at the level of the coronary ostia.

Nine immature domestic swine, either barrows or gilts, with an average weight of 27.2 kg, were instrumented as follows: Animals were premedicated with xylazine HCl 0.5 mg/kg, ketamine 2 mg/kg, and atropine 0.1 mg/kg. They were made to
Human subjects were anesthetized by intubation of the trachea and administration of halothane through a specially designed snout mask until an endotracheal tube could be inserted. This tube was connected to an Ohio Anesthesia Ventilator, and anesthesia was maintained with a mixture of O₂, N₂O, and methoxyflurane. Normal blood gases were maintained before cardiac arrest by adjusting ventilatory parameters.

The femoral arteries were exposed in the groin. A Millar velocity/pressure transducer (model VPC 684T) was passed via one femoral artery into the descending thoracic aorta; this device was used to measure aortic pressure. The velocity signal was recorded but otherwise not used. A 2.5 mm catheter, used for withdrawal of the reference sample, was passed into the abdominal aorta via the other femoral artery. The right carotid artery was exposed in the neck and a 2.5 mm catheter, used for withdrawal of the reference sample, was passed through an arteriotomy into the ascending aorta. A left thoracotomy was performed. The pericardium was opened and the ascending aorta was palpated while the catheter in the carotid artery was positioned as close as possible to the coronary ostia. The circumflex artery was exposed and a Carolina Medical Electronics Model EP4XXRC electromagnetic flow probe of appropriate size was fitted around the artery. Usually a key was inserted to ensure retention. The flow probe and its lead were sutured to the epicardium to prevent motion relative to the artery. A small catheter, which was used for microsphere injection, was inserted into the left atrium. Electrodes for electrocardiographic examination were sutured to the epicardium. The pericardium was closed as much as possible. The descending aorta was palpated to ensure that the femoral microtipped manometer was placed in the thoracic aorta. A Millar PC 350 microtipped manometer was placed in the pleural space. In some animals, a PC 350 microtipped manometer was inserted into the myocardium of the free wall of the left ventricle. The thoracotomy was closed securely after air and fluid had been evacuated from the pleural space.

The animal was positioned so that its left side rested on the base plate of the Thumper. The signal from the flow probe was processed by a Carolina Medical Electronics two-channel electromagnetic flowmeter. During the period of cardiac arrest, zero flow was determined by stopping chest compression for several seconds. A pressure zero from one of the fluid-filled aortic catheters was used to correct the Millar electronic pressure zero. Phasic and mean pressure and flow data were recorded with a Honeywell Visicorder. At least 1 min of phasic and mean data were recorded for each intervention.

The first injection of microspheres was made after baseline coronary and pressure data had been recorded; 5–6 × 10⁹ 15 μm microspheres, labeled with ⁴⁵Sc, ⁴⁷Ru, ⁵¹Cr, and ¹⁵¹Ce (New England Nuclear), were sonicated in a mixing vial and injected over 20 sec. The vial and left atrial line were flushed with 10 ml of saline. Gamma-activity in the mixing vial was determined before and after injection with a Packard Auto-Gamma Scintillation Spectrometer, Model 5986. A Harvard dual infusion/withdrawal pump (Harvard Apparatus Co., Model 600-910-920, Dover, MA) was used to remove reference samples. Withdrawal began 15 sec before injection and continued for 3 min at a rate of 7.9 ml/min.

Cardiac arrest was caused by electrically fibrillating the heart with a brief 10 V/60 cycle discharge delivered through the electrocardiographic wires from a Grass Model S9 Stimulator. After 1 min, transverse chest compression was begun at a rate of 66 strokes/min at a location midway between the scapula and sternum. Compression occupied 50% of the massage cycle. Our Thumper was designed to deliver 55 strokes/min, with a 0.8 sec pause after every 5 strokes for respiration. We decided to delete this mode so that chest compression could occur at a constant rate of 66 strokes/min without interruption. Respiration was provided by the anesthesia machine at a rate of 12 strokes/min.

The gas mixture was N₂O and O₂. No effort was made to synchronize respiration and a specific phase of the massage cycle because inflation/deflation and compression always overlapped. Three successive Thumper piston strokes (1.5, 2, and 2.5 inches) were studied, and an injection of microspheres was made for each.

After the last injection of microspheres had been made, respiration and chest compression were stopped, body cavities were opened, chests and abdomens were carefully inspected for signs of injury, flow probes and contiguous arteries were removed for calibration, and tissue samples were removed for storage in 10% formaldehyde. Four full-thickness samples were removed from the anterior wall adjacent to the anterior descending coronary artery, and four samples were removed from the posterior wall adjacent to the circumflex coronary artery. Tissue samples were divided in two and then cut so that they were all the same size, shape, and weight (1 g). Distilled water was added to blood samples to lyse red cells. Samples were counted in a gamma-spectrometer. The technique of spectral stripping was used to separate the activities of the individual isotopes. Activities were corrected for background and decay that occurred during storing and processing. Tissue flow was calculated as 7.9 times tissue activity divided by activity of the reference sample. Cardiac output was calculated as 7.9 times the difference in vial activity before and after injection divided by activity of the reference sample. The term “cardiac output” will be used to describe total systemic blood flow, regardless of whether the impetus for flow comes from the beating heart or compression of the chest.

The specific activity of the radiomicrospheres used in two experiments was determined. The total number of radiomicrospheres in 16 tissue samples was calculated by dividing the activity for a given radionuclide by the specific activity of each microsphere.

Data for mean pressure and flow were calculated from recordings at least 20 sec long. The difference between aortic and intrathoracic pressures during the relaxation phase was calculated by planimetry of phasic tracings of 4 successive beats. Data are presented as mean ± 1 SD. Student’s test for paired measurements was used to determine if a statistically significant difference existed (H₀: difference between two paired measurements equals 0, alpha equals 0.05).

Three large swine (average weight 59.6 kg) were prepared for a comparison of anterior-posterior (AP) and transverse (T) compression. Instrumentation differed from that used in the main study as follows: (1) Only a femoral reference-sample catheter was used. (2) The pericardium was not opened since no flow probe was used. (3) The microsphere-injection catheter was inserted into the left atrium via a pulmonary vein. Four injections of microspheres were made after cardiac arrest had been induced. The sequence of chest compression was T, AP, T, and AP.

The time required for microspheres to disperse was determined in two swine. Instrumentation differed from that used in the aforementioned comparison only in that a catheter was inserted into the right atrium. Separate femoral reference samples were collected during the first 2 min after injection of radiomicrospheres and during the succeeding 1 min. A reference sample was also aspirated from the right atrium for 3 min after injection. Four injections of microspheres were made in each animal.

**Results**

We will first report the data that pertain to the validity of using radiomicrospheres to measure coronary flow during cardiopulmonary resuscitation. First, application of this technique requires that the number of
microspheres trapped in a given tissue sample exceeds a minimum number, usually taken to be 400. We counted the number of radiomicrospheres in 16 myocardial samples obtained from two preparations in which satisfactory function of the flow probe had been observed. Every sample was calculated to contain at least 400 and usually more than 1000 radiomicrospheres of each of the four isotopes injected. Second, the adequacy of radiomicrosphere-mixing was studied by comparing the activity per gram of reference sample blood withdrawn simultaneously from the ascending aorta close to the coronary ostia and the abdominal aorta. Thirty-six paired determinations were made in nine animals — 27 when the circulation was maintained by chest compression and nine with the heart beating. During chest compression, blood withdrawn from the abdominal aorta had 94 ± 14% of the activity of blood in the ascending aorta (H1, accepted, p > .05). When the heart was beating, blood withdrawn from the abdominal aorta had 96 ± 7% of the activity of the blood in the ascending aorta (H0, accepted, p > .05). Thus, mixing of microspheres before arrival at the target organ appeared to have occurred.

The time required for microspheres to disperse during chest compression was studied in two animals. Reference blood samples withdrawn for 1 min, starting 2 min after microsphere injection had begun, contained 6% of the activity found in blood collected in the initial 2 min. No activity above background was found in blood withdrawn from the right atrium during and for 3 min after microsphere injection.

A comparison was made between AP and T chest compression in three animals. Although peak pressure was about 20% higher with AP compression, mean aortic pressures were similar: AP = 32 ± 5 mm Hg and T = 31 ± 9 mm Hg. Blood flows were also similar: cardiac output, AP = 712 ± 175 ml/min; cardiac output, T = 843 ± 202 ml/min; myocardial AP = 0.32 ± 0.17 ml/g/min; myocardial T = 0.46 ± 0.32 ml/g/min (p > .05 for all by Student’s paired t test). A constant finding in animals that had both AP and T chest compression was near-total disruption of the bony integrity of the thorax (bilateral segmental fractures involving six or more ribs). Since rib fractures were rare in animals having only T compression, it was decided to use T compression in the main study.

Table 1 summarizes the data from nine preparations in which a comparison was made of electromagnetic and radiomicrosphere techniques for coronary flow measurement. As Thumper piston stroke was increased, there was a concomitant increase in cardiac output, aortic pressure, and coronary blood flow. Mean aortic pressure increased primarily because of an increase in pressure during compression. Pressure during the relaxation phase of the massage cycle was increased by only one-half as much. Aortic pressure during compression was clearly a function of intrathoracic pressure, with the relation being: aortic pressure equals 0.58 times intrathoracic pressure plus 14.9, r = .96. In four animals, intramyocardial pressure was also measured, and its phasic appearance closely followed intrathoracic pressure throughout the massage cycle. During relaxation, both intrathoracic and intramyocardial pressures decreased much more rapidly than did aortic pressure. Pressure in the Thumper piston progressively increased with stroke: 1.5 inches equals 40 to 60 psi, 2 inches equals 80 to 90 psi, and 2.5 inches equals more than 120 psi. We estimate the area of contact between the Thumper and the chest wall to be about 2 square inches. Thus, the force applied to the chest was 100 pounds to more than 250 pounds.

Coronary flow, albeit at a level much below normal, was demonstrated by both techniques of flow measurement. Table 1 indicates that both the electromagnetic (EMF) and radiomicrosphere (RMS) techniques are in agreement in that as piston stroke lengthens, coronary flow increases. The proportional increase in flow according to the two techniques is similar (data expressed as percentage of prearrest flow, n = 9): 1.5 inch stroke, EMF 5 ± 7% and RMS 13 ± 6%; 2 inch stroke, EMF 17 ± 15% and RMS 21 ± 11%; 2.5 inch stroke, EMF 27 ± 26% and RMS 29 ± 18%.

The data for coronary flow in table 1 are the average for all nine preparations. It is instructive to consider the individual experiments in more detail. We observed in only four animals a coronary flow signal that consistently had a morphologic characteristic similar to the familiar pattern. In the remaining animals, although occasionally recognizable complexes were recorded, more often than not the signal had the appearance of noise. Examples from one of the four animals showing consistently reproducible phasic flow complexes are shown in figure 1. Capacitance effects such as retrograde flow and a forward flow "spike" occurring with relaxation clearly dominated the signal when stroke was 1.5 inches. With more powerful strokes, phasic complexes appeared that resembled the coronary flow signal seen in beating hearts. Figure 1 shows that the act of chest wall compression inhibits coronary flow more than a normal ventricular systole does. Coronary flow during cardiopulmonary resuscitation occurs entirely during the relaxation phase of the massage cycle.
TABLE 1
Hemodynamic measurements during cardiopulmonary resuscitation

<table>
<thead>
<tr>
<th></th>
<th>Beating heart</th>
<th>1.5</th>
<th>2</th>
<th>2.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac output (ml/min)</td>
<td>2306 ± 311</td>
<td>525 ± 195</td>
<td>692 ± 202</td>
<td>817 ± 321</td>
</tr>
<tr>
<td>Aortic pressure mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>79 ± 10</td>
<td>23 ± 4</td>
<td>33 ± 5</td>
<td>40 ± 6</td>
</tr>
<tr>
<td>Max</td>
<td>96 ± 7</td>
<td>34 ± 8</td>
<td>46 ± 10</td>
<td>55 ± 12</td>
</tr>
<tr>
<td>Min</td>
<td>66 ± 9</td>
<td>17 ± 4</td>
<td>24 ± 4</td>
<td>27 ± 6</td>
</tr>
<tr>
<td>Intrathoracic pressure mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Max</td>
<td>9 ± 4</td>
<td>31 ± 10</td>
<td>57 ± 12</td>
<td>67 ± 11</td>
</tr>
<tr>
<td>Min</td>
<td>2 ± 5</td>
<td>8 ± 6</td>
<td>6 ± 6</td>
<td>10 ± 8</td>
</tr>
<tr>
<td>Circumflex coronary blood flow</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Electromagnetic flow probe (ml/min)</td>
<td>47 ± 12</td>
<td>2 ± 3</td>
<td>8 ± 8</td>
<td>12 ± 12</td>
</tr>
<tr>
<td>Percent of flow in beating heart</td>
<td>-</td>
<td>5 ± 7</td>
<td>17 ± 15</td>
<td>27 ± 26</td>
</tr>
<tr>
<td>Radiomicrosphere (ml/g/min)</td>
<td>0.98 ± 0.20</td>
<td>0.13 ± 0.05</td>
<td>0.20 ± 0.09</td>
<td>0.28 ± 0.15</td>
</tr>
<tr>
<td>Percent of flow in beating heart</td>
<td>-</td>
<td>13 ± 6</td>
<td>21 ± 11</td>
<td>29 ± 18</td>
</tr>
</tbody>
</table>

*Mean ± 1 SD, n = 9.

In the five animals with little or no evidence of perfusion by electromagnetic flow measurement, flow in the distribution of the circumflex measured by radiomicrospheres was absent in three animals (<0.05 ml/g/min, 2.5 inch stroke). In the remaining two animals, radiomicrosphere flow was similar to that found in the four animals with phasic flow signals (0.28 and 0.29 ml/g/min, 2.5 inch stroke). Flow measured at the same time in the five animals with little evidence of flow according to flow probe measurement was 32% of normal in the distribution of the anterior descending coronary artery. It would appear that failure of the flow probe to measure flow in the circumflex artery in these experiments can mean either that no flow occurred (three animals) or that there was flow but it was not sensed (two animals). We suspect, but cannot prove, that in the former situation the flow probe obstructed the artery, while in the latter situation we know from postmortem examination that the flow probe had dislodged. These data indicate that five of nine preparations must be considered failures from the standpoint of the function of the flow probe. The flow data from the four technically successful preparations (table 2) better indicate, therefore, the actual magnitude of coronary blood flow during closed-chest massage. The relative increase in flow according to the two tech-

![Figure 1](http://circ.ahajournals.org/)

**Figure 1.** Phasic pressure and flow waveforms recorded during Thumper chest compression. Redrawn from original record. When the stroke was 1.5 or 2 inches, the phasic coronary waveform is dominated by capacitance effects. Forward flow in the coronary circulation occurs exclusively during the relaxation phase of the massage cycle. Note that the aortic (heavy line) and intrathoracic (narrow line) waveforms have different scales and zeros.
TABLE 2
Coronary hemodynamics in selected experiments

<table>
<thead>
<tr>
<th>Circumflex coronary blood flow</th>
<th>Beating heart</th>
<th>Thumper Piston stroke (inches)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electromagnetic flow probe (ml/min)</td>
<td>49 ± 13</td>
<td>6 ± 3</td>
</tr>
<tr>
<td>Percent of flow in beating heart</td>
<td>—</td>
<td>12 ± 5</td>
</tr>
<tr>
<td>Radiomicrosphere (ml/g/min)</td>
<td>0.97 ± 13</td>
<td>0.16 ± 0.03</td>
</tr>
<tr>
<td>Percent of flow in beating heart</td>
<td>—</td>
<td>16 ± 5</td>
</tr>
<tr>
<td>Mean aorto-intrathoracic pressure gradient relaxation phase (mm Hg)</td>
<td>—</td>
<td>6.5 ± 1.3</td>
</tr>
</tbody>
</table>

* mean ± 1 SD, n = 4.

Techniques in the selected experiments is as follows (data expressed as percentage of prearrest flow, n = 4): 1.5 inch stroke, EMF 12 ± 5% and RMS 16 ± 5%; 2 inch stroke, EMF 30 ± 6% and RMS 26 ± 11%; 2.5 inch stroke, EMF 50 ± 12% and RMS 40 ± 20%.

Table 2 shows the mean pressure gradient that existed between the aorta and the intrathoracic cavity during the relaxation phase of the massage cycle in the four selected experiments in which the electromagnetic flow probes functioned. This gradient (ΔP) was related to flow (Q) measured by electromagnetic flow probes by the expression

\[
Q(\text{ml/min}) = 1.54 \Delta P - 4.15, \ r = .99
\]

and to flow measured by radiomicrospheres by the expression

\[
Q(\text{ml/g/min}) = 0.018 \Delta P + 0.037, \ r = .83
\]

Figure 2 is an example of the phase relation between coronary flow and pressures in the aorta and thorax. At the end of compression, the fall in intrathoracic pressure is more rapid and of greater magnitude than is the fall in aortic pressure.

**Discussion**

Experiments in cardiopulmonary resuscitation are fraught with methodologic problems. A defect common to all is the dissimilar thoracic geometry of humans and experimental animals. Thoracic viscoelastic properties may also differ depending upon age and species. With respect to the latter, immature animals such as the piglets used in our study no doubt have a thorax that can be deformed more than the thorax of adult humans. The study of swine, however, may have an advantage compared with the study of other experimental animals in that the transverse shape of the swine thorax is square and thus resembles more the oval shape of the human thorax than does the triangular thorax of dogs and small primates.

Our purpose is to report a comparison of two different techniques for measuring coronary blood flow during cardiopulmonary resuscitation. We conclude that given optimal conditions, both may be useful.
Respect to the radiomicrosphere technique, the major requirements for its use, namely, the collection of adequate numbers of microspheres in individual tissue samples and complete mixing of microspheres before their arrival at the target organ, appear to have been satisfied. Assessing the results obtained with the electromagnetic flow probes is more difficult because the absence of flow by this technique may mean either that no flow was in fact occurring or that the flow probe was not functioning. By simultaneously measuring flow with both techniques, we believe that we have shown that in those experiments in which flow was not measured by the electromagnetic flow probe, the flow probe was actually inoperative.

Our finding that coronary blood flow may approach one-half of normal during cardiopulmonary resuscitation is in fair agreement with the results of several earlier studies: Voorhees et al.,3 who found flow to be 35% of normal and Maier et al.,4 who found flow to be 65% of normal. Conversely, our results are not in agreement with Chandra et al.,5 and Ditchey et al.6 who found coronary blood flow during experimental cardiopulmonary resuscitation to be 1% to 5% of normal. We can only guess at the reason for this difference, but one intriguing aspect of the work of Ditchey et al.6 is the low aortic pressure generated in their experiments. Their maximum aortic pressure was only slightly greater than the aortic pressure obtained in our study with the minimum piston stroke. In the study of Ditchey et al.,6 the maximum force used to compress the chest (140 pounds) resulted in a peak aortic pressure of 40 mm Hg and coronary flow 4% of normal. In our study, the minimum Thumper stroke of 1.5 inches required a piston pressure of 40 to 60 psi and resulted in a peak aortic pressure of 34 mm Hg and coronary flow about 12% of normal. The area of the piston in their compressor was 3 square inches, so that a force of 140 pounds indicated a piston pressure of 45 to 50 psi. Thus, comparable degree of chest compression in our studies led to similar results. We observed coronary flow about one-half of normal when we used a stroke of 2.5 inches. The required piston pressure was 120 psi and therefore was greatly in excess of the maximum pressure studied by Ditchey et al.6 It would appear that one reason why some investigators have found substantial coronary perfusion, while other investigators have found little flow, is that the former have compressed the chest in a more forceful manner.

We offer the following explanation as to how chest compression can generate coronary blood flow. It must be acknowledged that any explanation will suffer from the present uncertainty as to the true back pressure in the coronary circulation.7 We have taken intrathoracic pressure to be the effective back pressure during cardiopulmonary resuscitation, but it should be understood that if, as demonstrated by Maier et al.,4 vaso-motor tone exists in the arrested heart, use of intrathoracic pressure will overestimate the actual pressure gradient of coronary perfusion. There is no perfusion during the compression phase because aortic and intrathoracic pressures are similarly increased. As shown in figure 2, there is no gradient for forward flow across the coronary bed during compression, but a substantial gradient exists during relaxation. The reason for the substantial gradient during relaxation is that pressure decays more slowly in the aorta than in other intrathoracic viscera such as the right atrium. This slower decay no doubt results from differences in the viscoelastic properties and the rate of discharge from these compartments. Put in another way, during compression some of the energy transferred to the chest goes into distending the aorta; the ensuing elastic recoil of the stretched aortic wall provides the impetus for coronary flow.

Whether coronary flow of the magnitude observed in this study occurs in resuscitation of humans is uncertain. We are concerned that coronary flow during closed-chest massage in humans is more often than not minuscule. We come to this conclusion for two reasons. First, since coronary flow occurs only during the relaxation phase of the massage cycle, prolongation of the compression phase, as is presently recommended, will result in suboptimal perfusion. Second, the recommended chest wall deflection of 1.5 to 2 inches4 causes little coronary flow in swine and dogs and is hardly likely to cause greater flow in humans. There is a need to know whether a massage stroke that is both shorter and more forceful than that presently used could be used to optimize coronary perfusion without (at the same time) impairing perfusion of the brain.

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