Determinants of blood flow to vital organs during cardiopulmonary resuscitation in dogs

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ABSTRACT Whether blood flow during cardiopulmonary resuscitation (CPR) results from intrathoracic pressure fluctuations or direct cardiac compression remains controversial. From modeling considerations, blood flow due to intrathoracic pressure fluctuations should be insensitive to compression rate over a wide range, but dependent on the applied force and compression duration. If direct compression of the heart plays a major role, however, flow should be dependent on compression rate and force, but above a threshold, insensitive to compression duration. These differences in hemodynamics produced by changes in rate and duration form a basis for determining whether blood flow during CPR results from intrathoracic pressure fluctuations or from direct cardiac compression. Manual CPR was studied in eight anesthetized, 21 to 32 kg dogs after induction of ventricular fibrillation. There was no surgical manipulation of the chest. Myocardial and cerebral blood flows were determined with radioactive microspheres. At nearly constant peak sternal force (378 to 426 newtons), flow was significantly increased when the duration of compression was increased from 14 ± 1% to 46 ± 3% of the cycle at a rate of 60/min. Flow was unchanged, however, after an increase in rate from 60 to 150/min at constant compression duration. The hemodynamics of manual CPR were next compared with those produced by vest inflation with simultaneous ventilation (vest CPR) in eight other dogs. Vest CPR changed intrathoracic pressure without direct cardiac compression, since sternal displacement was less than 0.8 cm. At a rate of 150/min, with similar duration and right atrial peak pressure, manual and vest CPR produced similar flow and perfusion pressures. Finally, the hemodynamics of manual CPR were compared with the hemodynamics of direct cardiac compression after thoracotomy. Cardiac deformation was measured and held nearly constant during changes in rate and duration. As opposed to changes accompanying manual CPR, there was no change in perfusion pressures when duration was increased from 15% to 45% of the cycle at a constant rate of 60/min. There was, however, a significant increase in perfusion pressures when rate was increased from 60 to 150/min at a constant duration of 45%. Thus, vital organ perfusion pressures and flow during manual external chest compression are dependent on the duration of compression, but not on rates of 60 or 150/min. These data are similar to those observed for vest CPR, where intrathoracic pressure is manipulated without sternal displacement, but opposite of those observed for direct cardiac compression. We conclude that intrathoracic pressure fluctuations generate blood flow during manual CPR.


THERE IS continuing controversy concerning the mechanisms of blood flow during cardiopulmonary resuscitation. In 1960, Kouwenhoven et al.1 proposed, but never proved, that blood moved due to direct compression of the heart between the sternum and vertebral column. If this mechanism is correct, stroke volume should be determined by the amount of cardiac deformation, and prolongation of compression beyond the time necessary to squeeze the heart will have no effect on stroke volume because ejection ceases as soon as sternal displacement is maximal. At constant sternal displacement, increases in the rate of compression will augment flow because a fixed stroke volume is pumped into the systemic arteries more frequently per unit time.

Early evidence pointed to the fact that heart compression alone could not account for the equivalence of...
thoracic arterial and venous pressures during chest compression.\textsuperscript{2, 3} There had been speculation about an alternative theory,\textsuperscript{4} but it was not until 1980 that Rudikoff et al.\textsuperscript{5} provided compelling evidence that blood moves during chest compression because of phasic changes in intrathoracic pressure, not cardiac compression. According to this model, chest compression produces a relatively uniform rise in the pressure in all intrathoracic vascular structures. This pressure is transmitted from intrathoracic to extrathoracic arteries. Competent venous valves\textsuperscript{6, 7} and the large extrathoracic venous compliance, however, prevent the full transmission of pressure to the extrathoracic veins. The difference in pressure between extrathoracic arteries and veins causes blood to move from the thorax into the extrathoracic arterial system and into the cerebral bed. During release of chest compression, intrathoracic pressure falls below venous pressure, causing return of blood to the thorax.\textsuperscript{8} The heart serves as a passive conduit for blood flow in this model. Two-dimensional echocardiography, which has shown both the aortic and mitral valves to be open during chest compression in man,\textsuperscript{9} and angiography\textsuperscript{7} support the idea that the heart is a passive conduit. Since rises in intrathoracic pressure decrease the size of the vessels leaving the thorax, flow limitation will occur, causing a relatively constant flow per unit time. Flow should thereby be dependent on the duration of compression per cycle, but insensitive to the rate of compression.

Recently, Maier et al.\textsuperscript{10} in studies in animals instrumented over a long term, found that peak pleural pressure was significantly lower than peak vascular pressures during manual CPR, thus reviving the notion that cardiac compression, not intrathoracic pressure, was generating vascular pressures and flow. However, if the same data are examined comparing the rise in pleural pressure with the rise in vascular pressure (as has been done previously\textsuperscript{11}) rather than with the rise in peak pressure, the differences are far less striking. In addition, pleural pressure was measured with a sensor in a solid sheath. Maier et al. used similar techniques to measure pleural pressure during mechanical ventilation with positive end-expiratory\textsuperscript{12} and pericardial pressure. Their pleural and pericardial pressures were significantly lower than those measured by other investigators using different techniques.\textsuperscript{13–15}

Since methods for measurement of pleural pressure are controversial,\textsuperscript{16} we sought other evidence supporting one of the two mechanisms. Response to the rate of compression and the duration of compression during CPR should provide a test for the two theories: if the magnitude of flow is determined by the rate of compression but not the duration of compression, then direct cardiac compression should be the cause of flow; if the rate of compression does not affect flow but duration does, then intrathoracic pressure is likely the cause. These assumptions about the hemodynamic effects of changes in rate, compression duration, and rate were predicted by mathematical modeling,\textsuperscript{17} and are summarized in Table 1. Other models have predicted similar effects.\textsuperscript{18, 19}

Maier et al.\textsuperscript{10} noted an increase in cardiac output, mean aortic pressure, and mean aortic diastolic pressure as rate was increased; they did not, however, take into account the covariance of compression duration with rate. Thus, their observations may be due to changes in peak force or compression duration, but not rate per se.

The hemodynamic effects of changes in rate, compression duration, and applied external force were studied during manual CPR in dogs. The hemodynamics of manual CPR were then compared with those of vest CPR at similar changes in right atrial pressure to determine if intrathoracic pressure changes could account for flow. Lastly, the hemodynamics of manual CPR were compared with those of direct cardiac compression during open-chest cardiac massage to determine if cardiac compression could account for flow.

## Methods

### Preparation.

Sixteen mongrel dogs weighing 21 to 32 kg were anesthetized with ketamine (7 mg/kg im) and pentobarbital sodium (15 mg/kg iv). Supplemental pentobarbital was administered as needed. An endotracheal tube was secured in a tracheostomy, and the dogs were ventilated with room air by a volume-cycled respirator (Harvard 607). From a femoral cutdown, micromanometer-tipped catheters (Millar PC-470) zeroed and calibrated at 37°C were placed into the right atrium and ascending aorta, a pigtail catheter for microsphere injection was placed.

## Table 1

<table>
<thead>
<tr>
<th>Predicted effects of rate, duration, and force on perfusion pressure or flow</th>
<th>Intrathoracic pressure</th>
<th>Cardiac compression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Duration</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Force</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

A model of the cardiovascular system was used to predict changes in perfusion pressure and flow for changes in rate (60 or 150/min), compression duration (15% to 45% of the cycle), and applied force. The hemodynamics of manual CPR were then studied for changes in rate, duration, and force. The hemodynamics of vest CPR (intrathoracic pressure) and open-chest cardiac massage (cardiac compression) were studied to verify the predictions of the model. Rate insensitivity with duration dependence for manual CPR would indicate that cardiac compression was unimportant.
into the left ventricle, and a pacing catheter was placed into the right ventricle. A fluid-filled catheter was placed into the left common carotid artery through the thyroid artery. From bilateral axillary cutdowns, catheters were placed into each brachiocephalic artery for collection of blood during microsphere injection. Catheter positions were confirmed at autopsy. In nine of the dogs a 16-gauge cannula was placed into the lateral cerebral ventricle through a Burr hole for measurement of intracranial pressure. The dogs were studied in the supine position, and each was given 300 units/kg of heparin. The dogs were also given normal saline intravenously to bring their mean right atrial pressure to 3 mm Hg. This fluid replaced losses resulting from surgery and catheter flushing, and restored the dogs to euohia.

**General protocol.** The first injection of microspheres (for methods see below) was made before cardiac arrest for measurement of prearrest regional flow. Ventricular fibrillation was then induced by 60 Hz alternating current applied to the pacing catheter. Epinephrine was administered as a 1 mg bolus into the left ventricle, then intravenously at a constant rate of 8 µg/kg/min by a syringe pump (Harvard 600) to reverse arterial collapse, which occurs at the thoracic outlet during CPR and can limit flow.20-22 CPR was started within 30 sec after fibrillation. Manual CPR was performed by one of the investigators or a technician, using computer-generated sounds to time chest compression and release. CPR was also performed with the use of a vest (Physio-Control Corp.) around the thorax. The vest contained a bladder that was inflated cyclically by a programmable pneumatic generator.23 During manual CPR, ventilation was performed after every fifth compression, with a mixture of 95% O₂-5% CO₂, by a Venturi-equipped ventilating device (Michigan Instruments) to a peak airway pressure of 30 to 40 mm Hg. The Venturi added room air to the O₂-CO₂ to produce a final ventilating mixture of approximately 55% oxygen. During vest CPR, ventilation was performed synchronously with vest inflation, also at a peak airway pressure of 30 to 40 mm Hg, using the same Venturi-equipped ventilator.

In four additional dogs, prepared in similar fashion, the anterior chest wall was removed. Open-chest cardiac compression was performed with a pneumatic chest compressor (Michigan Instruments) fitted with a spring to return the piston to its initial position after each direct cardiac compression. The piston was positioned so that it just made contact with the heart before each compression.

**Specific protocols.** This study consisted of three sets of experiments. The first set examined the effects of compression rate, compression duration, and force on the hemodynamics of manual CPR in eight dogs. The second set compared the hemodynamics of manual CPR with those of vest CPR in eight additional dogs. The third set compared the hemodynamics of manual CPR with those of direct cardiac compression in four dogs.

**Compression rate and duration at constant force with manual CPR.** This protocol was used to determine whether blood flow was dependent on rate or compression duration. Manual CPR was performed with peak sternal force of ~400 newtons (N) (1 N = 0.102 kg = 0.225 pound) at a rate of 150/min with long duration (~45%), at a rate of 60/min with long duration (~45%), and at a rate of 60/min with short duration (~15%). Force measured is described below. These CPR techniques were performed in randomized order for each dog. Randomization was used to eliminate order-dependent bias. Microsphere blood flows were obtained for each CPR technique. Cerebral and myocardial blood flow were compared at similar durations (~45%) and rates of 60/min and 150/min, and at the same rate (60/min) with short (~15%) and long (~45%) durations.

At the end of each study in which intracranial pressure was measured (n = 6), a limited thoracotomy was performed and the resuscitator’s hand was placed inside the thorax. Open-chest cardiac massage was performed at rates of 60 and 150/min to test whether direct cardiac compression would produce an increase in vascular pressures concomitant with increased rate.

**Manual vs vest CPR.** Two protocols compared the hemodynamics of manual CPR with those of vest CPR. If blood flows during manual CPR because of intrathoracic pressure fluctuations, then the hemodynamics of manual CPR should be similar to those of vest CPR.

The first protocol compared manual CPR at 150/min with that of vest CPR at 150/min. Manual CPR at 60/min was used as a reference. The compression duration at 150/min for manual and vest CPR was ~45%, while the duration at 60/min was ~18%, since each technique used ~180 msec cycle for compression. Manual CPR was performed first in this set of studies at a rate of 60/min with a force of ~400 N. Manual CPR at 150/min with the same 400 N force or vest CPR at 150/min with peak right atrial pressure similar to that during manual CPR were then performed in random sequence. Finally, a second period of manual CPR at 60/min was performed to test the stability of the preparation. Microsphere blood flows during CPR were obtained for each of the four techniques in each dog.

The second protocol compared vascular pressures at rates of 60 and 150/min as sternal force was increased, with vascular pressures obtained during vest CPR at rates of 60 and 150/min as vest pressure was increased. Sternal force was increased in 100 N increments from 100 to 400 N, and then decreased back to 100 N. Each incremental force was applied until vascular pressures stabilized (15 to 30 cycles). Pressure measurements were then taken. Vest CPR was performed at the same duration and rates. Vest pressure was increased in increments to obtain right atrial pressures similar to those obtained during manual CPR. Pressure measurements were recorded as soon as vascular pressures stabilized at each inflation pressure. Flow was not measured.

**Direct cardiac compression.** This protocol was used to determine whether perfusion pressures for direct cardiac compression were dependent on rate or duration.

After an initial period of manual CPR, the anterior chest wall was removed and the pneumatic chest compressor was positioned such that its piston just contacted the heart. For each dog, the heart was then compressed at a rate of 60/min with durations of 15% and 45%, and at a rate of 150/min with a duration of 45%, all with identical piston displacement (~4.6 cm). Each technique was performed until vascular pressures stabilized. Measurements of perfusion pressures were then recorded. These techniques were performed in randomized order for each dog. Cerebral and myocardial perfusion pressures were compared at the same rate (60/min), with durations of 15% and 45%, and at the same duration (45%) at rates of 60 and 150/min.

**Measurements.** For manual CPR, sternal force was measured by a precision force transducer assembly held under the operator’s palms and used to displace the sternum. The hemodynamic effects of changing sternal force have been noted.24 The force-measuring system was calibrated with known weights, and was found to be accurate within 10 N over the range of 0 to 500 N. The frequency response of the force-measuring system was essentially flat to 100 Hz. Sternal displacement was measured by a linear potentiometer assembly fitted with a flat handgrip that was also positioned under the operator’s palms. Figure 1 is a diagram of the measuring system. The force transducer (PCB Piezotronics 208A03) was mounted on a hard rubber pad (2 × 3 inch) that approximates the size and consistency of the human palm. This pad gives the contact surface with the chest. A steel disk 6 cm in diameter, mounted on the top of the force transducer, allowed the operator to hold both the handgrip and the force transducer assembly. The linear potentiometer was attached to the piston of a pneumatic chest compressor (Michi-
Regional blood flows were determined with $15 \pm 3 \mu m$ diameter radioactive microspheres (New England Nuclear) according to techniques previously described. Reference samples were obtained from the two arterial sites with a dual-syringe pump (Harvard 600). Withdrawal was started 15 sec before injection and continued for 6 minutes after injection, allowing full washout of spheres. Withdrawal rate was 3.8 ml/min for prearrest samples and 1.9 ml/min for postarrest samples.

Blood and tissue samples contained at least 2000 spheres and were counted on an automated, multichannel gamma pulse-height analyzer (Packard 9042). For $^{141}$Ce, $^{113}$Sn, $^{103}$Ru, $^{99}$Nb, and $^{48}$Sc, the energy windows were 132 to 172, 352 to 438, 446 to 550, 692 to 820, and 850 to 1200 keV, respectively. The counts measured in any one window were the sum of the counts from that window’s isotope plus overlap from the other isotopes present in the sample. A series of equations were solved that subtract out the overlap counts and determine the correct number of counts for each isotope. Tissue blood flows were then calculated as follows: tissue-corrected counts were multiplied by the reference withdrawal rate and divided by the average counts of the two arterial reference samples. The results were then divided by the weight of the sample and multiplied by 100 to obtain flows in milliliters per minute per 100 grams tissue.

The adequacy of mixing with left ventricular injection of microspheres during CPR has been validated with the use of nonembryonic markers, and blood flows determined with $15 \mu m$ spheres have been validated during CPR by a venous outflow technique for cerebral flow and with electromagnetic flow probes for myocardial flows.

**Accuracy and consistency of rates and durations during manual CPR.** Manual CPR was performed by compressing and releasing the chest in time with computer-generated sounds. The timing of the sounds were accurate to within 0.05%. Preliminary studies showed that, with practice, the time interval from compression to compression (cycle) could be maintained to within 2% at rates of 60 and 150/min. Compression duration could be maintained to within 3% to 5% of the cycle at durations of both 15% and 45% of the cycle. These values exclude the occasional 2 to 3 sec pause in CPR necessitated by personnel changes due to operator fatigue.

**Statistical analysis.** Correlations between myocardial or cerebral flow and their respective perfusion pressures were tested with linear regression. Relationships between myocardial or cerebral perfusion pressures and force or right atrial peak pressure were tested by analysis of variance, with covariance analysis used to test for differences in the relationships among manual and vest CPR at 60/min and 150/min. Differences in pressures and flows generated by manual CPR at 60 and 150/min with short and long duration, as well as differences in pressures and flows generated by manual CPR at 60 and 150/min and vest CPR at 150/min were tested by repeated-measures analysis of variance, with orthogonal contrasts. Differences in pressures measured during cardiac compression were also tested by repeated-measures analysis of variance, with orthogonal contrasts. Differences in paired data were tested with the paired $t$ test. The significance level was .05.

**Results**

Compression rate and duration at constant force during manual CPR. This protocol was used to determine whether blood flow was dependent on rate or compression duration during manual CPR. Myocardial and cerebral flows were measured during randomized-order manual CPR, with 400 N sternal force, at a rate of
60/min with short (15%) duration, at a rate of 60/min with long (45%) duration, and at a rate of 150/min with long duration. This protocol allowed comparisons at the same duration (45%) of rates of 60 and 150/min and comparisons at the same rate (60/min) of short (15%) and long (45%) durations of CPR. Oxygenation was good for all techniques (Po2 > 100 mm Hg). Pco2 values were in the 20 to 35 mm Hg range, and there were no differences among the techniques.

Pressures, sternal force, and displacement for this protocol are shown in Table 2. At near-constant sternal force, cerebral perfusion pressure (mean carotid minus mean intracranial pressure) was higher for manual CPR of long duration at 60 and 150/min than at 60/min with short duration. As predicted from these pressures, cerebral blood flow was higher for the long-duration techniques than for the short-duration technique (Table 3). For all parts of the brain, there was no difference in flow during manual CPR of long duration at 60/min or at 150/min. However, both of these techniques produced greater blood flow to the brain than manual CPR of short duration at 60/min. Myocardial perfusion pressure (mean diastolic aortic minus mean diastolic right atrial pressure) was also higher with longer compression duration (Table 2). If analyzed as a pair, manual CPR at 60/min with long duration produced higher myocardial flow than manual CPR at 60/min with short duration (p < .02), but this difference did not achieve statistical significance (p < .09) when the variation of manual CPR at 150/min was added. Thus, cerebral and myocardial blood flow was unchanged for the rates studied, while cerebral flow was strongly dependent on compression duration.

**Manual vs vest CPR — regional flow.** Manual and vest CPR were compared directly. There is no direct cardiac compression with the vest because of the small sternal displacement (Table 4), so vest CPR produces "pure" intrathoracic pressure fluctuations. During the inflation phase of vest CPR, right atrial and intrathoracic venous pressures should approximate intrathoracic pressure, because the large venous compliance would make venous transmural pressure close to zero. If manual CPR generates flow also because of changes in intrathoracic pressure, right atrial pressure would be a good estimate of intrathoracic pressure during manual CPR. Therefore, vest pressure was set to produce a peak right atrial pressure similar to that achieved by manual CPR. If blood moves because of intrathoracic pressure fluctuations, then manual and vest CPR performed at a similar rate, duration, and peak right atrial pressure should produce similar vascular pressures and flow.

A continuous record during vest and manual CPR at 150/min is shown in Figure 2. For this record, the myocardial perfusion pressures (31 vs 29 mm Hg) and the cerebral perfusion pressures (30 vs 27 mm Hg) are similar. The myocardial perfusion pressure during vest CPR was not degraded by the early diastolic downward deflection on the aortic pressure tracing because it was balanced by a similar deflection on the right atrial

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**TABLE 2**

<table>
<thead>
<tr>
<th>Effect of rate and duration at constant force during manual CPR</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CPR</strong></td>
</tr>
<tr>
<td>---------</td>
</tr>
<tr>
<td>Mean aortic pressure (mm Hg)</td>
</tr>
<tr>
<td>Aortic systolic pressure (mm Hg)</td>
</tr>
<tr>
<td>Aortic diastolic pressure (mm Hg)</td>
</tr>
<tr>
<td>Mean right atrial pressure (mm Hg)</td>
</tr>
<tr>
<td>Right atrial systolic pressure (mm Hg)</td>
</tr>
<tr>
<td>Right atrial diastolic pressure (mm Hg)</td>
</tr>
<tr>
<td>Myocardial perfusion pressure (mm Hg)</td>
</tr>
<tr>
<td>Mean carotid pressure (mm Hg)</td>
</tr>
<tr>
<td>Mean intracranial pressure (mm Hg)</td>
</tr>
<tr>
<td>Cerebral perfusion pressure (mm Hg)</td>
</tr>
<tr>
<td>Sternal force (N)</td>
</tr>
<tr>
<td>Sternal displacement (cm)</td>
</tr>
<tr>
<td>Compression duration (% of cycle)</td>
</tr>
</tbody>
</table>

Values are mean ± SEM; n = 8 (n = 6 for intracranial pressure).
Myocardial perfusion pressure was computed as mean diastolic aortic minus mean diastolic right atrial pressure and cerebral perfusion pressure as mean carotid minus mean intracranial pressure. M60S = manual CPR at 60/min with short duration; M60L = manual CPR at 60/min with long duration; M150L = manual CPR at 150/min with long duration.

*p < .01 vs M60S.
tracing. This downward deflection was likely due to the more rapid recoil of the chest wall with vest CPR, producing transient negative intrathoracic pressure, as shown by the early diastolic negative right atrial pressure. This figure also contrasts the sternal displacements produced by vest and manual CPR. Sternal displacement during vest CPR was variably toward or away from the vertebral column. Absolute values for all experiments are shown in table 4.

There was no difference in myocardial or cerebral blood flows during vest or manual CPR at 150/min (table 5). Both CPR techniques produced more cerebral flow than did manual CPR at 60/min with short duration. The absolute time of compression (176 ± 19 msec) for each cycle was similar for the three techniques, so that at more rapid rates, more of the cycle was occupied by compression, and more cerebral flow was produced. At the same rate (150/min), similar right atrial peak pressures, and similar compression duration, manual and vest CPR produced similar blood flow.

As expected, flow in the left ventricular free wall correlated with the myocardial perfusion pressure (r = .88, p < .001), and occipital lobe flow correlated with the cerebral perfusion pressure (r = .89, p < .001) for manual CPR of long duration at 60 and 150/min and vest CPR of long duration at 150/min (figure 3). A similar relationship has been shown for CPR performed with mechanical compressions.28 These perfusion pressures are reasonable estimates and most likely determine myocardial and cerebral flow.

**Comparison of manual and vest CPR**
= .90, p < .001) for manual CPR at 60/min with long duration (figure 4). Similar relationships exist for manual CPR at 150/min, and for CPR performed with mechanical compressions.24

Perfusion pressures produced by manual CPR with increasing sternal force were compared with perfusion pressures produced by vest CPR with increasing inflation pressure, at rates of 60/min and 150/min, and of long duration. Under all conditions, perfusion pressure was related to peak right atrial pressure. The absolute value of perfusion pressure at each level of right atrial pressure was different among the dogs; therefore, to compare the relationship of perfusion pressure to right atrial pressure across dogs, the perfusion pressures were normalized to peak right atrial pressure. In each dog, the perfusion pressures produced by increasing right atrial peak pressures during manual and vest CPR at 60 and 150/min with long duration were divided by the perfusion pressure produced by a right atrial peak pressure of 80 mm Hg during manual CPR at 60/min for that dog. The values were then multiplied by 100 for conversion to percentages. Figure 5 shows myocardial and cerebral perfusion pressures plotted against right atrial peak pressure for the four CPR techniques.

![FIGURE 2. Continuous record of vest and manual CPR at 150/min. For each technique the chart speed was increased, then decreased. Vest pressure was adjusted to produce peak right atrial pressure similar to that achieved with manual CPR performed before recording these tracings. Myocardial and cerebral perfusion pressures were similar. Right atrial pressure became negative in early diastole during vest CPR. The vest was deflated rapidly with negative pressure, causing rapid recoil of the thorax.](image)

### TABLE 5
Regional blood flow during manual and vest protocol

<table>
<thead>
<tr>
<th>Prearrest control</th>
<th>CPR</th>
<th>M60S</th>
<th>M150L</th>
<th>V150L</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV free wall</td>
<td></td>
<td>14.6 ± 4.4</td>
<td>29.7 ± 11.2</td>
<td>37.1 ± 11.5</td>
</tr>
<tr>
<td>LV septum</td>
<td></td>
<td>12.9 ± 5.0</td>
<td>30.2 ± 15.0</td>
<td>27.8 ± 10.8</td>
</tr>
<tr>
<td>Frontal lobe</td>
<td></td>
<td>3.6 ± 1.2</td>
<td>10.5 ± 3.8</td>
<td>12.0 ± 4.2</td>
</tr>
<tr>
<td>Parietal lobe</td>
<td></td>
<td>4.4 ± 1.7</td>
<td>13.3 ± 5.3</td>
<td>13.7 ± 5.0</td>
</tr>
<tr>
<td>Occipital lobe</td>
<td></td>
<td>5.4 ± 1.9</td>
<td>16.1 ± 6.8</td>
<td>17.4 ± 6.3</td>
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<tr>
<td>Brainstem</td>
<td></td>
<td>8.7 ± 3.4</td>
<td>19.7 ± 9.2</td>
<td>30.2 ± 8.8</td>
</tr>
</tbody>
</table>

Values are ml/min/100 g (mean ± SEM).

LV = left ventricular; M60S = manual CPR at 60/min with short duration; M150L = manual CPR at 150/min with long duration; V150L = vest CPR at 150/min with long duration.

*p < .01 vs M60S; **p < .025 vs M60S.
produce higher perfusion pressures than vest CPR at corresponding right atrial peak pressures, indicating that intrathoracic pressure fluctuations account for all of the perfusion pressure generated.

Direct cardiac compression. Direct cardiac compression with the mechanical compressor was used to determine the influence of compression rate and duration on myocardial and cerebral perfusion pressures. In contrast to manual CPR, myocardial and cerebral perfusion pressures were not dependent on duration. At a rate of 60/min, perfusion pressures did not change when compression duration was changed from 15% to 45% (table 6). Again, as opposed to manual CPR, there was a significant increase in the perfusion pressures when compression duration was held fixed at 45% and rate was changed from 60 to 150/min (table 6). The measured anterior-posterior cardiac deformation was 4.6 ± 0.1 cm for all dogs and did not change with change in rate or duration.

Discussion

Determining the mechanisms of blood flow during CPR is important for further understanding of its phys-
io logic effects, and is necessary for optimization of vital organ flow. Since all investigators seem to agree that blood flow during manual CPR is due either to intrathoracic pressure fluctuations or direct cardiac compression, we sought to identify the consequences of each mechanism that might be measured and compared, while avoiding the controversy of pleural pressure measurements.

When we examined the effects of direct cardiac compression after thoracotomy, we found that perfusion pressures were insensitive to the duration of compression, but highly dependent on rate. For direct compression, stroke volume should be proportional to the amount of mechanical deformation of the heart, not the duration of compression, because ejection would cease as soon as the heart reached maximum compression. If the rate of compression were increased at the same amount of cardiac compression, then more stroke volumes per unit time would be pumped into the arterial system, and perfusion pressures and flow would increase accordingly, provided filling were adequate.

On the other hand, we found with vest CPR — a “pure” intrathoracic pressure manipulation — that movement of blood was insensitive to the rate of compression when the duration of compression was fixed. When the entire thoracic contents are pressurized, blood moves from the compliance in the thorax through a resistance to the compliance in the periphery. The flow of blood is determined by the amount of time the thorax is compressed — if the rate is changed, but the percent of the cycle for compression is fixed, then the amount of compression per unit time is maintained constant and blood flow should remain relatively constant.

Thus, the differences in perfusion pressures and flow when rate and duration of compression are changed provide an additional test of the two models for the mechanisms for blood flow during manual CPR. Our findings that the hemodynamics of manual CPR are similar to those produced by vest CPR, but opposite to those produced by direct cardiac compression, support the hypothesis that blood moves during manual CPR because of changes in intrathoracic pressure.

We found no differences across rate for myocardial

![Graph](image)

**FIGURE 5. A.** Correlation between normalized myocardial perfusion pressure and peak right atrial pressure for manual CPR at 60/min (M60L), manual CPR at 150/min (M150), vest CPR at 60/min (V60), and vest CPR at 150/min (V150). The perfusion pressures for each CPR technique for each dog are expressed as a percent of the perfusion pressure obtained at a peak right atrial pressure of 80 mm Hg for M60L for that dog. Data for vest CPR are described by the relationship \( y = 2.59x - 68.3 \) (\( r = .86, p < .001 \)), while data for manual CPR are described by the relationship \( y = 2.14x - 64.0 \) (\( r = .82, p < .001 \)). There is a difference between the two lines (\( p < .05 \)). B. Correlation between normalized cerebral perfusion pressure and peak right atrial pressure for manual CPR at 60/min (M60L), manual CPR at 150/min (M150), vest CPR at 60/min (V60), and vest CPR at 150/min (V150). All data are described by the same relationship \( y = 2.01x - 52.6 \) (\( r = .88, p < .001 \)). Normalization was performed as above.

### TABLE 6

<table>
<thead>
<tr>
<th></th>
<th>Mechanical compression (n = 4)</th>
<th>Manual compression (n = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>60S</td>
<td>60L</td>
</tr>
<tr>
<td>Myocardial perfusion pressure (mm Hg)</td>
<td>28 ± 1.5</td>
<td>27 ± 0.9</td>
</tr>
<tr>
<td>Cerebral perfusion pressure (mm Hg)</td>
<td>24 ± 2.0</td>
<td>25 ± 2.6</td>
</tr>
<tr>
<td>Cardiac compression (cm)</td>
<td>4.6 ± 0.1</td>
<td>4.6 ± 0.1</td>
</tr>
</tbody>
</table>

Values are mean ± SEM.

60S = 60/min with short duration (15%); 60L = 60/min with long duration (45%); 150L = 150/min with long duration. Manual compression was performed by the rate-duration protocol.

\(^a\)p < .01 vs 60S and 60L; \(^b\)p < .01 vs 60/min; \(^c\)p < .02 vs 60/min.
or cerebral blood flow when CPR was performed at the same duration (table 3), but there was a significant increase in cerebral flow at the same rate when the duration was increased (table 3). There were also no differences in flow during manual or vest CPR performed at 150/min at similar right atrial peak pressures and durations (table 5).

There was good correlation between the perfusion pressures normalized to force and the sternal force (figure 4). The question arises: Is this correlation between perfusion pressure and force due to compression of the heart with greater force or simply to generation of greater intrathoracic pressure? In this study the relationship between the perfusion pressures and the right atrial peak pressure was similar for all CPR techniques that had the same compression duration, independent of rate (figure 5). Right atrial pressure is an estimate of intrathoracic pressure during vest CPR. Since manual CPR generates the same or slightly lower perfusion pressures than vest CPR at the same right atrial peak pressure, the critical determinant of flow at the same duration and rate is intrathoracic pressure. Even if there is cardiac compression during chest displacement, there is no evidence that its contributions to flow are any more than those achieved by fluctuations in intrathoracic pressure. An electrical model of the circulation has predicted that cardiac compression should produce approximately twice the perfusion produced by changes in intrathoracic pressure at the same right atrial peak pressure.18

Epinephrine was used to augment myocardial and cerebral blood flow. Studies done without epinephrine showed very low flows.22, 25 With low flows, it would not be possible to distinguish between the two mechanisms, since all CPR techniques would result in low flows and any differences would likely be obscured by the variability of the flow measurements. The flows were increased significantly by epinephrine, but the drug did not change the mechanisms of blood flow, since the same dogs that showed no change in flow or perfusion pressure with manual CPR showed a marked increase in perfusion pressures with open-chest cardiac massage (cardiac compression). Epinephrine may have contributed to the ability to distinguish between the two mechanisms.

It has been proposed by Maier et al.10 that changes in dimension of the heart observed during manual CPR demonstrate that cardiac compression is the mechanism for movement of blood. These dimensional changes, however, could be occurring without a change in ventricular volume. If there is a decrease in ventricular volume, the flow could be backward or forward, or the ventricle could be just one of many structures from which blood moves as a result of increases in intrathoracic pressure. Studies in isolated heart-lung preparations have demonstrated that pleural pressure changes alone can move volume from the left ventricle.29

This study shows that hemodynamics of CPR are insensitive to rate in 21 to 32 kg dogs without surgical manipulation of the chest, but are dependent on the duration and magnitude of applied force. Increased flow with higher rates has been shown in other studies.10, 30 The first possible explanation for this increased flow at higher rates is that these studies were performed in dogs instrumented over a long term in which fibrosis could restrict cardiac mobility, allowing cardiac compression and its concomitant rate effects to occur. A second explanation is that compression force may have increased at the more rapid rates, accounting for the increase in flow observed, since compression force was not measured. The most likely explanation is that the longer compression duration at rapid rates accounts for the increased blood flow in these studies. Short rise-time manual CPR was deliberately used and probably resulted in a relatively constant time of compression for each beat at each rate studied. A constant time of compression per cycle produces a marked increase in the percent of the cycle during which compression occurs at rapid rates. Another study31 showed rate dependence of cardiac output at rates less than 50/min, but showed no significant rate dependence for rates between 50 and 120/min.

To determine the effects of varying rate or duration on perfusion pressures and flow during manual CPR, it is necessary that sternal force or displacement remain constant. If either sternal force or displacement varies when rate or duration is changed, then a change in flow may represent a change in force or displacement, rather than a change in rate or duration. In our study, peak sternal force was controlled, and when duration of compression was increased (at a rate of 60/min), there was minimal change in displacement (4.6 ± 0.3 vs 4.7 ± 0.4, p = NS; table 2). In addition, there was no significant change in displacement when rate was changed from 60 to 150/min (4.7 ± 0.4 vs 4.3 ± 0.4 cm, p = NS; table 2).

The 400 N force used in the study of flow was chosen because it was the highest force that could be maintained steadily. This force produced a sternal displacement of 4.5 cm, an average peak aortic pressure of 88 mm Hg, and marked chest distortion.

For clinical application, rapid-rate manual CPR has the disadvantage of being extremely tiring to perform
for prolonged periods, and it appears to be no more efficacious than properly performed manual CPR at 60/min and of prolonged duration. Studies in man have already shown that increases in compression duration from 30% to 60% of the cycle produce significant increases in arterial pressure and the area under the Doppler carotid flow-velocity signal. Rates from 40 to 80/min at an optimal duration of 60% did not change mean arterial pressure or change this carotid flow index.32 This duration dependence but rate insensitivity in man is similar to the canine findings presented. Long-duration compressions are also difficult to perform and require a distinct effort at a rate of 60/min.

It should be emphasized that this study was performed in dogs, and that differences in chest geometry or other anatomic factors may limit its usefulness with regard to understanding the mechanisms of blood flow in man. However, the similarity of the results with those in man cannot be overlooked.

The sternal displacement-force relationship is one factor in assessing the applicability of results with this particular dog preparation to man. There was a good correlation (r = .82, p < .001) between all the sternal displacement and force measurements obtained with the different protocols. The compliance in this dog preparation (0.0117 cm/N) is very close to the compliance value of 0.0109 cm/N in man.33

Thus, this study provides confirmation that the major mechanism for blood flow during manual CPR in large dogs is manipulation of intrathoracic pressure. Vital organ flow is unchanged when rate is increased from 60 to 150/min, but is markedly enhanced when the duration of each compression is increased from 15% to 45%. This duration dependence but rate insensitivity is diametrically opposed to the influence of changes in compression duration and rate on the hemodynamics of direct cardiac compression. Vest CPR, where there is a rise in intrathoracic pressure without significant sternal displacement, produces perfusion pressures and flow similar to those produced by conventional external chest compression.

We thank Physio-Control Corporation for supplying the vests used in these studies, and PCB Piezotronics for supplying the force transducer. We thank Julius Mayo and Tony Dipaula for technical assistance. We also thank Jean Cadden, Karen Paetow, and Susan Edmunds for help in preparing the manuscript.

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Circulation. 1986;73:539-550
doi: 10.1161/01.CIR.73.3.539

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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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:
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