Relative Lack of Coronary Blood Flow During Closed-chest Resuscitation in Dogs

ROY V. DITCHLEY, M.D., JAMES V. WINKLER, M.D., AND C. ALLEN RHODES, M.D.

SUMMARY  Recent studies have shown that blood flow during closed-chest cardiopulmonary resuscitation (CPR) results primarily from generalized changes in intrathoracic pressure rather than direct compression of the heart. Since ascending aortic and right atrial pressures rise and fall synchronously and to comparable levels during CPR, we hypothesized that the absence of a pressure difference across the coronary vascular bed during CPR precludes coronary blood flow. To test this hypothesis, we compared high-fidelity ascending aortic and right atrial pressures and carotid and coronary blood flow (electromagnetic flowmeters) during closed-chest CPR in 12 fibrillating dogs. Chest compression force was increased from 40 to 140 pounds in 20-pound increments using a pneumatic chest compression device. Although ascending aortic and right atrial pressures were always similar, high-compression-force CPR produced small mean pressure differences across the coronary vascular bed (5.6 ± 0.8 mm Hg [± SEM] at 140 pounds). These pressure differences were accompanied by low levels of coronary blood flow. However, coronary flow was less than 1% of control (prearrest) values whenever chest compression force was less than 100 pounds, and coronary flow exceeded coronary flow under all conditions (carotid and coronary flows at 140 pounds = 26.2 ± 6.4% and 4.3 ± 2.0% of prearrest values, respectively, p < 0.01). We conclude that generalized changes in intrathoracic vascular pressures during closed-chest CPR promote carotid but not coronary blood flow. High-compression-force CPR produces small pressure differences across the coronary vascular bed, allowing low levels of coronary flow. However, high-compression-force CPR is over six times more effective in maintaining carotid flow than coronary flow.

RECENT STUDIES have focused attention on the importance of generalized changes in intrathoracic pressure as a mechanism of blood flow during closed-chest cardiopulmonary resuscitation (CPR). This concept evolved from the observation that intrathoracic arterial and venous pressures are similar during chest compression, a finding inconsistent with selective ventricular compression and effective atrioventricular valve closure. Increased intrathoracic pressure during CPR is transmitted unequally to extrathoracic arteries and veins due to the presence of venous valves at the thoracic inlet and unequal arterial and venous capacitance and resistance to collapse. As a result, chest compression produces an extrathoracic arteriovenous pressure difference that allows forward blood flow.

Although cyclic variations in intrathoracic pressure can produce carotid blood flow, the observation that intrathoracic vascular pressures are nearly equal during CPR suggests that closed-chest resuscitation may be ineffective in delivering blood to the heart itself. Specifically, equality between ascending aortic and right atrial pressures throughout each chest compression cycle would preclude the development of a significant pressure difference across the coronary circulation at any time during CPR. Theoretically, the lack of a pressure difference, or driving force, would prevent coronary blood flow.

Based on these considerations, we hypothesized that the absence of a pressure difference across the coronary circulation precludes effective coronary blood flow during closed-chest resuscitation. However, preliminary studies demonstrated that both the difference between ascending aortic and right atrial pressures and the level of coronary blood flow during CPR were dependent upon the force with which the chest was compressed. To better characterize these effects, we studied intrathoracic arteriovenous pressure differences and carotid and coronary blood flow during CPR performed with a wide range of external compression forces.

Methods

Studies were performed in 12 closed-chest mongrel dogs that weighed 32.0–52.7 kg. A preliminary left lateral thoracotomy was performed to allow placement of a Zepeda circumferential electromagnetic flow probe around the proximal portion of the circumflex coronary artery. Probe size (2.5 or 3.0 mm i.d.) was varied to avoid constriction of the coronary vessel. A bipolar electrode was sewn to the surface of the left ventricular free wall. The electrical leads from both the epicardial electrode and the coronary flow probe were exteriorized through small incisions in the lateral chest wall. The chest was then closed in layers, and the dogs were allowed to recover for 3–5 days.

On the day of study, the dogs were anesthetized with pentobarbital (30 mg/kg i.v.), intubated, and mechanically ventilated. High-fidelity, micromanometer-tip catheters (Millar), each with a separate lumen for recording fluid-filled pressures, were inserted into a femoral artery and vein and positioned in the ascending aorta and right atrium, respectively. Catheter position was confirmed both fluoroscopically and by appropriate pressure recordings in each case. The lumen of each Millar catheter was connected to a Gould P23Db pressure transducer with zero reference set at the mid-chest level. The solid-state pressure transducers were calibrated before insertion, and high-fidelity and fluid-
filled pressures were matched prior to each set of recordings to correct for baseline drift. The coronary flow probe was connected to a Zepeda model SWF-4 square-wave electromagnetic flowmeter. A second flow probe was placed around the carotid artery several centimeters above the thoracic outlet and connected to a Carolina Medical Electronics model 501D square wave electromagnetic flowmeter. Each flow probe was calibrated in vitro. Reference for zero flow was determined by recording flow signals after intravascular pressures had equilibrated after induction of ventricular fibrillation. All pressures and flow signals were recorded on a Beckman model R611 eight-channel recorder. The instantaneous difference between ascending aortic and right atrial pressures was recorded on a separate channel using an electronic subtraction circuit.

Normal saline (500–1000 ml) was administered intravenously before data collection. This was done to increase the levels of arterial pressure and blood flow achieved during CPR. In addition, a single i.v. dose of heparin (5000 U) was given to retard clot formation. Control pressures and flow signals were recorded with respiration temporarily suspended at end-expiration. Ventricular fibrillation was then induced by applying a low voltage alternating current to the epicardial electrode. After recording carotid and coronary flow signals under conditions of zero flow, anteroposterior chest compression was initiated using a modified pneumatic chest compression device (Michigan Instruments Life Aid model X1004 cardiopulmonary resuscitator). Compression rate was 60 times per minute, with a compression duration equal to 50% of each cycle. The relaxation phase was prolonged by 0.5 second every fifth compression to allow lung inflation to a peak inspiratory pressure of 20 cm H₂O by a synchronized, pressure-limited ventilator. Pressure and flow signals during CPR were first recorded with chest compression force set at 40 pounds. Compression force was then increased to a maximum of 140 pounds in 20-pound increments, with pressure and flow measurements repeated at each level. All signals were checked for baseline drift after each set of recordings. Studies were completed within an average of 35 minutes of the onset of ventricular fibrillation.

Pressure and flows during control and all CPR studies were averaged over five cycles. Pressure recordings in two dogs were technically inadequate and were excluded from analysis. Results are reported as mean ± SEM. Correlation coefficients for the relationship between the mean ascending aortic–right atrial pressure difference and coronary blood flow during CPR were derived using standard linear regression methods. Statistical significance was determined by the t test for paired samples, or analysis of variance with multiple comparison testing (Student-Newman-Keuls), when applicable.

Results

Pressure and flow recordings from a representative study are shown in figure 1. Ascending aortic and right atrial pressures were similar during CPR under all con-
### Table 1. Pressure and Flow Measurements

<table>
<thead>
<tr>
<th></th>
<th>Peak aortic pressure (mm Hg)</th>
<th>Peak right atrial pressure (mm Hg)</th>
<th>Mean Ao-RA pressure difference (mm Hg)</th>
<th>Carotid blood flow (ml/min)</th>
<th>Coronary blood flow (ml/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>136.9±5.7</td>
<td>1.3±0.5</td>
<td>128.5±5.1</td>
<td>78.7±22.7</td>
<td>30.9±5.0</td>
</tr>
<tr>
<td>40 pounds</td>
<td>14.3±1.7</td>
<td>13.9±1.5</td>
<td>1.4±0.8</td>
<td>3.1±1.3</td>
<td>0.1±0.1</td>
</tr>
<tr>
<td>60 pounds</td>
<td>17.8±3.1</td>
<td>16.6±2.1</td>
<td>1.8±1.0</td>
<td>4.2±1.1</td>
<td>0.3±0.2</td>
</tr>
<tr>
<td>80 pounds</td>
<td>22.5±4.1</td>
<td>20.2±2.8</td>
<td>2.1±0.7</td>
<td>6.5±1.4</td>
<td>0.1±0.0</td>
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<tr>
<td>100 pounds</td>
<td>28.2±4.7</td>
<td>24.8±3.6*</td>
<td>4.1±1.0§</td>
<td>10.9±2.2</td>
<td>0.4±0.3</td>
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<tr>
<td>120 pounds</td>
<td>34.8±5.0</td>
<td>30.6±4.5†</td>
<td>4.1±1.0§</td>
<td>25.8±6.5</td>
<td>1.3±0.5§</td>
</tr>
<tr>
<td>140 pounds</td>
<td>40.9±5.4</td>
<td>37.4±5.6‡</td>
<td>5.6±0.8§</td>
<td>26.2±6.4</td>
<td>1.2±0.5**</td>
</tr>
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</table>

Values are mean ± SEM. Control (prearrest) data are shown for comparison, but were not included in the statistical analyses.

Abbreviations: CPR = closed-chest cardiopulmonary resuscitation; mean Ao-RA pressure difference = mean difference between ascending aortic and right atrial pressures throughout an average chest compression-relaxation cycle; % = percent of control, calculated individually for each dog.

* p < 0.05, † p < 0.01, § p < 0.001 (right atrial vs aortic pressure; percent coronary vs percent carotid blood flow).

<table>
<thead>
<tr>
<th></th>
<th>Carotid blood flow (%)</th>
<th>Coronary blood flow (%)</th>
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<tbody>
<tr>
<td>Control</td>
<td>100</td>
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### Conditions (Table 1). However, small differences between peak pressures were present when CPR was performed with an applied chest compression force of 100 pounds or more. The mean pressure difference over a five-beat CPR cycle also increased with more forceful chest compression, reaching a maximum of 5.6 ± 0.8 mm Hg when compression force was 140 pounds.

Cardiopulmonary resuscitation during CPR at each level of chest compression force are summarized in Table 1. Coronary blood flow was less than 1% of control (prearrest) values whenever chest compression force was less than 100 pounds and was proportionately much less than carotid flow under all conditions (Fig. 2). For example, when CPR was performed with a compression force of 140 pounds, relative carotid and coronary blood flows were 26.2 ± 6.4 and 4.3 ± 2.0% of control values, respectively (p < 0.01). Coronary blood flow under these conditions was significantly greater than that during CPR at all levels of compression force less than 120 pounds.

Coronary blood flow during CPR was a linear function of the mean pressure difference generated across the coronary circulation (r = 0.97, p < 0.01, based on average values of both coronary flow and mean ascending aortic–right atrial pressure differences at each level of chest compression force) (Fig. 3). In general, both coronary blood flow and the difference between ascending aortic and right atrial pressures were highest during the relaxation phase of each chest compression cycle (Fig. 4). The pattern of coronary flow during actual chest compression was more variable, ranging from low levels of forward flow to low levels of retrograde flow.

The ascending aortic–right atrial pressure difference generated by chest compression was dependent upon the state of lung inflation. The first compression-relaxation cycle after lung inflation produced higher mean pressure differences than did any subsequent cycle (Fig. 5A). Pressure differences during the remaining four cycles were all similar. A trend toward increased coronary flow in the cycles after lung inflation was also apparent, although these differences were not statistically significant (Fig. 5B).

### Discussion

The results of this study demonstrate that closed-chest CPR produces relatively little coronary blood flow in large dogs. Coronary flow was virtually absent whenever the chest was compressed with a force of less than 100 pounds and was proportionately much less than carotid flow under all conditions. More forceful chest compression produced only small amounts of coronary blood flow and was over six times more effective in maintaining carotid flow than coronary flow.
Pressure have little effect on coronary arteriovenous pressure differences, since the entire coronary circulation lies within the chest. Thus, the potential driving force for coronary blood flow during closed-chest CPR is inherently limited. Although autoregulatory mechanisms normally maintain coronary flow at or near levels sufficient to meet myocardial oxygen demands, compensation is incomplete when perfusion pressure falls below a critical level (determined by myocardial oxygen requirements, and averaging approximately 60 mm Hg in a beating heart). As a result, coronary blood flow is a function of perfusion pressure in the low range, and cannot occur without an arteriovenous pressure difference.

Based on these considerations and the assumption that generalized pressure changes are solely responsible for forward blood flow during CPR, we expected coronary flow to be absent during closed-chest resuscitation. Although our findings strongly support these basic concepts, we did observe small ascending aortic–right atrial pressure differences and low levels of coronary blood flow when CPR was performed with high-force chest compression. The cause of this pressure difference (and therefore coronary blood flow) is uncertain. High compression force CPR, by causing greater sternal deflection, may produce some direct heart compression in addition to a generalized increase in intrathoracic pressure. However, selective aortic or ventricular compression seems unlikely in view of the anatomic relationship between the sternum, great vessels and cardiac chambers. Furthermore, the mean ascending aortic–right atrial pressure difference during CPR was highest in the cycle immediately after lung inflation (fig. 5A), a circumstance in which the separation between the sternum and vertebral column is increased. This observation is indirect evidence against cardiac compression as a major factor. An alternative possibility is that high-force chest compression produces regional differences in intrathoracic pressure that are transmitted to the underlying cardiac chambers and great vessels. Whether significant pressure differences can develop in the limited extravascular space

**Figure 3.** Relationship between coronary blood flow and the mean ascending aortic–right atrial pressure difference generated during cardiopulmonary resuscitation. Data points represent average values during cardiopulmonary resuscitation performed with six levels of chest compression force in 10 dogs.

Although we did not attempt to measure myocardial oxygen requirements directly, coronary flow was always well below levels appropriate for myocardial metabolic demands during ventricular fibrillation.

This distribution of blood flow during CPR reflects the relative pressure differences generated across the carotid and coronary circulations. Recent studies have demonstrated that external chest compression produces a generalized increase in intrathoracic pressure, with little or no direct compression of the heart. Differential transmission of this increased pressure to extrathoracic arteries and veins creates a substantial pressure difference across the carotid circulation, and therefore an effective driving force for carotid blood flow. However, generalized changes in intrathoracic pressure...
that separates the aorta and right atrium is unknown. However, intravascular pressure differences were often highest after release of applied compression force. An increase in pressure difference due to more forceful chest compression, but occurring largely during the relaxation phase of each cycle, is difficult to explain on this basis. Another possibility is that intrathoracic arteriovenous pressure differences are simply a function of the level of systemic blood flow generated during CPR. Cyclic blood flow through arterial and venous systems that have intrinsic differences in resistance and capacitance might well result in slightly unequal pressures, regardless of the mechanism by which flow is generated. If so, higher levels of flow would create predictably greater pressure differences. The fact that high-compression force CPR increased carotid blood flow, as well as the difference between ascending aortic and right atrial pressures, is consistent with this concept, although a cause-and-effect relationship has not been demonstrated.

Almost 20 years ago, MacKenzie et al. \(^5\) first suggested that closed-chest resuscitation may be less effective in supplying blood to the heart than to other vital organs. This suggestion was based on both an observed reduction in the normal arterial–central venous pressure difference during CPR in human subjects, and presumed compression of the entire heart, including the coronary arteries and veins, during external massage. However, these observations and the possibility of disproportionately low coronary blood flow during CPR have since received little attention. Rudikoff et al. \(^1\) questioned the adequacy of coronary perfusion during closed-chest resuscitation on the basis of low aortic pressures during the relaxation phase of each chest compression cycle, but did not measure coronary blood flow directly. To our knowledge, quantitative assessment of coronary flow during closed-chest CPR was first reported by Voorhees et al. \(^10\) who studied regional blood flow distribution during CPR in dogs using radioactive microspheres and concluded that the heart is perfused in proportion to cardiac output. The reason for this difference in results is uncertain. Since their studies were performed in relatively small dogs (6–12 kg), chest compression could have produced coronary arteriovenous pressure differences larger than those present in our studies, owing to direct compression of the heart or to other mechanisms. However, the limited pressure data reported suggest that this was not the case. \(^10\) It is, therefore, difficult to explain such high levels of coronary flow (average 35% of control values) on a physiologic basis. Alternatively, the microsphere technique could have yielded spurious results under the low-flow conditions of CPR. Although dogs with grossly unequal blood flow to paired organs were excluded from analysis, \(^10\) this does not eliminate the possibility of nonuniform streaming of microspheres to different body regions. Such an effect, due to incomplete mixing of microspheres with blood, could produce substantial error in estimates of regional blood flow distribution, despite comparable uptake by paired organs in the same region.

Although the applicability of our findings to human subjects is uncertain, available evidence suggests that generalized changes in intrathoracic pressure are an important mechanism of blood flow during CPR in man. \(^1\) \(^4\) \(^5\) \(^11\) Therefore, human CPR probably maintains carotid blood flow more effectively than coronary flow. Furthermore, these same concepts apply to other resuscitation techniques in which forward flow is dependent upon cyclic changes in intrathoracic pressure, such as repetitive coughing. \(^12\) \(^13\) Although it is reasonable to presume that this relative lack of coronary blood flow has an adverse effect on the likelihood of successful resuscitation, the fact remains that standard CPR is effective in many patients. Whether improved coronary flow would enhance either survival or postresuscitation ventricular function is unknown.

We conclude that generalized changes in intrathoracic vascular pressures during CPR promote carotid but not coronary blood flow. Since this appears to be the major effect of external chest compression, closed-chest resuscitation is ultimately ineffective in maintaining blood flow to the heart. Further studies will be required to determine the cause of the small aortic–right atrial pressure difference present during CPR, and whether this pressure difference (and therefore coronary flow) can be augmented by modifications in resuscitation technique.

**Acknowledgment**

We express our appreciation to Stephen Bell, Jane Connell, and Holly Collier for their expert technical assistance and to Barbara Brock- enridge and Ruth Hueiker for their secretarial services.
Addendum

While this manuscript was being considered for publication, Chandra et al.\textsuperscript{14} reported that coronary blood flow averaged less than 2% of prearrest values during standard CPR in six dogs. Their findings are consistent with the data presented in this study, and provide additional evidence that standard CPR is ineffective in producing blood flow to the heart.

References

Relative lack of coronary blood flow during closed-chest resuscitation in dogs.
R V Ditchey, J V Winkler and C A Rhodes

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