Pressure-synchronized Cineangiography During Experimental Cardiopulmonary Resuscitation

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SUMMARY Cardiopulmonary resuscitation (CPR) has been thought to produce blood flow by compression of the heart between the sternum and spine, termed "external cardiac massage," but there has been no direct experimental documentation of this proposed mechanism. Micromanometric pressure recordings were synchronized with cineangiograms during mechanical CPR in 17 dogs with induced ventricular fibrillation. Chest compression produced equivalent pressure increases in the aorta (Ao) and right atrium (RA) (Ao 32 ± 14 mm Hg, RA 30 ± 14 mm Hg; NS), a linear relationship between aortic and intrapleural pressures (r = 0.87, p < 0.001) over a wide range of induced pressures, cineangiographic blood flow through both left-heart chambers, and a pressure gradient (21 ± 14 mm Hg) between all intrathoracic cardiovascular compartments and the jugular veins that resulted from closure of venous valves at the thoracic inlets. Simultaneous chest compression and lung inflation significantly increased all intrathoracic vascular pressures, the aortojugular venous gradient (42 ± 13 mm Hg, p < 0.05 vs chest compression alone), electromagnetically determined carotid arterial blood flow (1.75 ± 0.81 ml/min/kg vs 0.51 ± 0.27 ml/min/kg during chest compression alone, p < 0.005), and angiographic left-heart flow.

We conclude that blood flow during CPR results principally from an increased intrathoracic pressure and that there is selectivity flow to the brachiocephalic vascular bed because of the arteriovenous gradient produced by venous valves at the thoracic inlets. Greater intrathoracic pressure resulting from simultaneous inflation and compression improves left-heart flow. The left heart is therefore a conduit, not a pump, during CPR.

THE ABILITY of closed-chest cardiopulmonary resuscitation (CPR) to produce an arterial pulse and perfuse vital organs in the arrested circulation was demonstrated experimentally and clinically in 1960 and termed "external cardiac massage." As the name implied, the technique was thought to produce selective ventricular compression between the sternum and spine, propelling blood forward through the systemic vasculature. The technique was quickly accepted and its efficacy documented in clinical reports.

However, early hemodynamic observations during experimental CPR by Weale and Rothwell-Jackson showed that chest compression resulted in an equal increase in arterial and venous pressures recorded in the iliac vessels, and the absence of a significant pressure gradient was believed to argue against antegrade flow. The absence of an arteriovenous pressure gradient to account for forward flow was confirmed in human subjects by MacKenzie et al. These investigators found that right atrial pressure increased to a level equal to that of the systemic arterial pressure and suggested that the equivalent pressure increase in the right atrium and arterial bed could result in retrograde flow or reflux into the extrathoracic venous bed. Rudikoff and co-workers showed the absence of a significant net aortic–central venous pressure gradient during chest compression and documented that the pressures in all cardiac chambers and in the thoracic aorta increase proportionately with the increase in intrapleural pressure. These findings suggest that vascular pressures and flow recorded during chest compression are dependent upon the generated intrathoracic pressure rather than ventricular compression. Thus, a number of experimental observations do not support the traditional concept of ventricular compression generating forward blood flow during CPR.

In this study, we used cineangiography synchronized with high-fidelity pressure recordings to examine intravascular pressure and flow events and their genesis during chest compression.

Materials and Methods

The mechanism of blood flow induced by chest compression during circulatory arrest was investigated in 17 mongrel dogs that weighed 18–40 kg. All studies were performed with sodium pentobarbital anesthesia. High-fidelity micromanometer catheters (Millar Mikro-Tip) were positioned via the femoral arteries or veins in three or more of the following sites in each dog: ascending aorta, left ventricle, main pulmonary artery, right atrium, external jugular vein and subdiaphragmatic inferior vena cava (IVC). Intrapleural pressure was measured in six dogs with a micromanometer catheter positioned in the intrapleural space through a 10-gauge polyethylene cannula inserted in the fifth or sixth intercostal space at the level of the midaxillary line. Angiographic catheters (#8F) were placed in two or more cardiac chambers or...
great vessels in each dog. A transjugular catheter was positioned in the left atrium by the transseptal route for angiographic injections in four dogs. A transvenous quadrapolar electrode was placed across the tricuspid valve to induce atrial and ventricular fibrillation. The position of all catheters was verified fluoroscopically. Heart rate and rhythm were monitored continuously with a lead II surface ECG. A standard cuffed endotracheal tube was positioned in all dogs and the cuff was inflated. Data were recorded on an Electronics for Medicine VR 12 recorder and on magnetic tape.

Fibrillation was induced by passing alternating current through the quadrapolar catheter and was confirmed by surface electrocardiographic monitoring. Concomitant fibrillation of the atria did not occur during all episodes of ventricular fibrillation, as determined by the presence of a-waves in the atrial pressure tracing and by atrial contractions seen on cineangiograms.

During circulatory arrest, chest compression was performed with a commercial pneumatic device (Thumper, Michigan Instruments). Piston force (40–120 lbs) and excursion were set to produce 1.5–2 inches of sternal depression. Chest compression was performed at a rate of 30–60 compressions/min, and compression was maintained for 50% of cycle duration. Slower rates were used during cineangiographic studies so that manual injections of contrast could be performed between compressions.

Cineangiograms were obtained at 60 frames/sec and electronically synchronized with pressure recordings. Renografin or Dinosol was injected manually in 10-ml boluses for cineangiography. The oily contrast medium was used to achieve a "tracer bullet" effect, which indicated direction and velocity of blood flow.

Common carotid arterial blood flow was measured in five dogs with a cannulating electromagnetic flow probe and a square-wave flow meter (Narcomatic RT 500, Narco-Biosystems). The area under the flow signal (positive minus negative area) was integrated with an electronic digitizer and net antegrade flow (ml/min/kg) calculated.

Intravascular and intrapleural pressures and carotid flow were recorded during conventional CPR with the lungs deflated and airway open to atmosphere during five chest compressions. After five compressions, the lungs were inflated to an airway pressure of 40–60 cm H₂O. The high airway pressure was sustained during the next five chest compressions (modified CPR) and pressures and flow were recorded. Conventional CPR was compared with modified CPR with respect to pressures and pressures and flow by alternating the two techniques for 2–3 minutes before defibrillation.

Statistical methods included one-way analysis of variance and the S-method for comparison of recorded pressures and the t test for comparison of pressures and flow during conventional and modified CPR. Values are reported as mean ± SD.

**Results**

**Hemodynamic Observations**

Chest compression during circulatory arrest produced pressures of similar magnitude in the intrathoracic cardiovascular compartments and the subdiaphragmatic IVC. The peak aortic pressure was slightly but not significantly higher than those in other intrathoracic vascular compartments during chest compressions (fig. 1). However, the jugular venous pressure (11 ± 4 mm Hg) during chest compression was significantly lower (p < 0.025) than simultaneously recorded aortic (32 ± 14 mm Hg) or right atrial pressure (30 ± 14 mm Hg); the average pressure gradient between the intrathoracic vascular compartments and the extrathoracic jugular veins was 21 ± 14 mm Hg. Pressures recorded in the subdiaphragmatic IVC were not significantly different from aortic or right atrial pressure (fig. 1). The mean common carotid artery flow during chest compression alone was 0.51 ± 0.27 ml/min/kg, or 9% of prearrest flow (mean prearrest flow 5.84 ± 2.74 ml/min/kg).

Chest compression during sustained positive-pressure lung inflation (modified CPR) produced a significant increase in the pressures in all intrathoracic cardiovascular compartments and the IVC (fig. 1). The pressure gradient between the ascending aorta and the jugular veins was greater than that during chest compression alone (42 ± 13 vs 21 ± 14 mm Hg, p < 0.05). This increased arteriovenous gradient was associated with a significant increase in the

**Figure 1.** Mean systolic pressures during conventional cardiopulmonary resuscitation (CPR) (chest compression alone) and modified CPR (chest compression during sustained lung inflation). Modified CPR produced a significant increase in all recorded intravascular pressures. Pressures during conventional or modified CPR were not significantly different in any compartment except the jugular vein (JV). Ao = ascending aorta; RA = right atrium; PA = pulmonary artery; IVC = inferior vena cava; LV = left ventricle. *p < 0.05, **p < 0.025, +p < 0.01, + +p < 0.005.
The magnitude of antegrade carotid blood flow (1.75 ± 0.81 ml/min/kg, \( p < 0.005 \) vs chest compression alone) (fig. 2).

Aortic and intrapleural pressures were measured simultaneously in six dogs. The increase in aortic pressure produced by chest compression approximated the increase in intrapleural pressure. The compression-induced increases in aortic and intrapleural pressures during conventional and modified CPR were positively correlated \( (r = 0.87, p < 0.01) \) (fig. 3).

Cineangiographic Observations

Left Ventriloculograms

Injections in the left lateral projection during ventricular fibrillation opacified the left ventricle and left atrium, indicating mitral regurgitation. If the atrium remained in sinus rhythm, each contraction of the atrium was followed by additional ventriculoatrial reflux. During sternal depression, the heart moved abruptly dorsally and rotated clockwise (from an apical reference). The aortic valve opened 150–200 msec after the ventricle achieved its most dorsal position. The rotation of the ventricle made it difficult to state whether or not the ventricular cavity was compressed, for the projection rotated from left lateral to left anterior oblique; however, in several dogs, forward flow was seen without apparent distortion or compression of the left ventricular cavity.

Left Atrioograms

Chest compression displaced, without significantly compressing, the left atrium. Injections of conventional and oily contrast media in the left atrium and pulmonary veins revealed that blood flowed forward from the pulmonary veins through both left-heart chambers to the aorta during a single chest compression, beginning 150–200 msec after the heart was displaced dorsally. Between compressions, slow antegrade flow was observed in the pulmonary veins.

Sustained lung inflation during compression subjectively increased the quantity and the velocity of blood flow through the left heart. Inflation alone initiated antegrade flow through the left heart before compression, and the ensuing compression markedly accelerated this antegrade flow.

**Aortograms**

Injections of conventional and oily contrast media in the aorta during ventricular fibrillation showed variable degrees of aortic regurgitation between con-
pressions. During the first 100 msec of compression, the aortic diameter decreased and antegrade blood flow occurred before aortic valve opening. After valve opening, the rate of flow increased and the aortic diameter remained constant. Simultaneous lung inflation and chest compression greatly augmented antegrade flow and decreased the severity of aortic regurgitation (fig. 4).

When 3–4 inches of sternal depression was used to achieve cineangiographically visible cardiac compression, the aorta was markedly displaced and compressed, and the severity of aortic regurgitation was greater than that with 1.5 inches of sternal depression. Forward flow was minimal and principally due to aortic compression despite the generation of up to 60 mm Hg of pressure in the aorta. The increased aortic regurgitation during compression appeared to result from compression and torsion of the aortic root. Lung inflation during exaggerated sternal compression resulted in less distortion of the heart, an increase in forward flow, and decreased the amount of aortic regurgitation during chest compression alone.

**Right-heart Angiograms**

Conventional and oily contrast media were injected into the main pulmonary artery and right ventricular outflow tract to assess the timing of pulmonary blood flow with respect to the sternal compression/relaxation cycle. An initial surge of flow into the distal pulmonary vascular bed occurred with the onset of sternal compression, but antegrade pulmonary flow ceased as compression continued. A second and qualitatively larger surge of forward flow occurred shortly after the onset of the relaxation phase, as blood moved through both right-heart chambers to the pulmonary artery. The tricuspid valve was incompetent and reflux from the right atrium into the venae cavae occurred with compression. When sternal compression was combined with sustained lung inflation, there was imperceptible forward flow during compression or

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**Figure 4.** (A and B) Aortography during conventional cardiopulmonary resuscitation (CPR). During relaxation (A), aortic valve incompetence is evident. During chest compression (B), the cardiac silhouette is displaced posteriorly, and antegrade flow is produced without significant left ventricular compression. (C and D) Aortography during modified CPR in the same dog. Immediately after the preceding sequence the lungs were inflated to an airway pressure of 40 cm H$_2$O. No aortic regurgitation is present during relaxation (C). During chest compression and lung inflation (D), greater clearance of contrast medium suggests that aortic outflow is improved over conventional CPR.
relaxation. Lung deflation and simultaneous relaxation of sternal compression markedly augmented forward flow through the right heart and pulmonary arteries as compared with the flow seen on relaxation with sustained deflation or with sustained inflation. Marked differences were apparent in the amount of flow contributed by the venae cavae. During compression, there was more reflux into the IVC than into the superior vena cava (SVC). During relaxation, forward flow was seen from the SVC through the right heart, but little or no net flow came from the IVC except after simultaneous deflation of the lungs and relaxation of compression.

Cervical Venograms

Injections into the axillary vein revealed antegrade venous flow in the brachiocephalic bed between compressions and a brief surge of retrograde flow at the onset of compression. Retrograde flow into the extra-thoracic brachiocephalic veins was abruptly halted by closure of venous valves as compression continued (fig. 5). An abrupt transition from the low-pressure jugular veins to the brachiocephalic vein at the inlet to the thorax was consistently observed during withdrawal of micromanometer catheters; the brachiocephalic pressure was equal to that in the right atrium and aorta during compression (fig. 6).

An anatomic dissection was performed after sacrificing a dog with a micromanometer catheter in situ at the point of pressure transition, and confirmed that a bicuspid venous valve immediately cephalad to the junction of the axillary vein and external jugular vein was responsible for the abrupt pressure transition and the angiographic contrast gradient. This jugular venous valve was seen by cineangiography to be incompetent during low-pressure events. Atrial contraction waves caused reflux through the valves, but the valves closed when intrathoracic pressure was increased by chest compression.

Summary of Angiographic Findings

The atrioventricular and aortic valves were incompetent during ventricular fibrillation. Valvular incompetence was shown by reflux of contrast medium across these valves before the initiation of chest compression as well as during compression in the right heart and between compressions in the left heart. Contrast media injected in the aorta frequently refluxed across both left-heart valves. Blood passed from the pulmonary veins through both left-heart chambers to the aorta during a single compression. The magnitude of left-heart flow was augmented by sustained inflation during compressions, and the degree of aortic regurgitation was reduced.

Sternal compression resulted in retrograde surges of blood in both venae cavae, tricuspid regurgitation, and a brief initial surge of forward blood flow in the pulmonary artery. Most of the right heart and pulmonary artery flow occurred during relaxation of sternal depression, and blood traversed both right-heart chambers and advanced through the pulmonary arteries. Most of the right-heart flow came from the SVC, and no net flow was seen from the IVC except during simultaneous deflation and relaxation. Venous valves at the thoracic inlets closed during compression and inhibited reflux into the SVC.

Discussion

These hemodynamic and cineangiographic observations in dogs do not support the traditional "car-

**Figure 5.** A manual injection of contrast medium into the axillary vein (V) during chest compression shows an abrupt contrast cutoff (arrows) above the junction of the axillary vein and external jugular vein (R. Ext. Jug.) and immediately below the tip of a micromanometer catheter that recorded a pressure of 20 mm Hg less than that in the right atrium and aorta. When the micromanometer catheter was withdrawn across this contrast interface during a subsequent compression, there was an abrupt transition to a pressure equal to that in the thoracic aorta (see text and figure 6). SVC = superior vena cava.
diac pump” mechanism for blood flow during CPR. Systemic perfusion appears instead to be the result of a functioning “thoracic pump”: Chest compression produces a generalized increase in intrathoracic pressure that is differentially transmitted to the brachiocephalic arterial and venous beds by functioning venous valves at the thoracic inlet. Cineangiographically, the heart appears to serve largely as a passive conduit during CPR. During chest compression, blood traverses both left-heart chambers in the absence of significant cardiac compression. During relaxation, antegrade flow through the right-heart chambers occurs principally from the SVC. Antegrade flow does not seem to depend on cardiac compression or competent cardiac valves. Actual compression of the heart, produced in these experiments by increasing the extent of sternal depression, was shown to be hemo-dynamically detrimental. The heart is not truly an anatomic axial structure, and thus its compression is accompanied by some degree of clockwise rotation along its major axis as it “rolls off” the vertebral column.

Pressure recordings during chest compression did not reveal a significant intrathoracic arteriovenous pressure gradient or ventriculoatrial pressure separation, which would be the expected finding if cardiac compression and atrioventricular valve closure occurred during CPR. The fact that atrial pressures increased to the same levels as the ventricular pressure during compression suggests that either the atrioventricular valves were grossly incompetent, that compression was applied to all cardiac chambers, or that there was a generalized increase in all intrathoracic pressures.

Our hemodynamic observations suggest that antegrade carotid flow during chest compression is the result of a pressure gradient between the intrathoracic and peripheral arterial compartment and the brachiocephalic veins. Intrathoracic and aortic pressures are transmitted directly to the brachiocephalic (carotid) arterial bed but not to the venous bed. Our data confirm the observations in dogs and human subjects by Rudikoff and co-workers that jugular venous pressure during chest compression is significantly lower than intrathoracic vascular pressures. This pressure difference is due to functioning venous valves outside the thorax that appear to close competently at high intrathoracic pressures. This arteriovenous gradient is augmented during chest compression with sustained lung inflation due to the greater increase in arterial pressure than in jugular venous pressure. The increase in the arteriovenous gradient is accompanied by a marked increase in carotid blood flow and angiographic left-heart flow. The absence of a significant right atrial—IVC gradient during either conventional or modified CPR suggests that these regions are not separated by a valving mechanism, and free reflux and minimal net forward flow result from compression and relaxation. These findings were confirmed by cineangiography.

Experimental evidence shows that high intrathoracic pressure may produce blood flow from the pulmonary vascular bed. Studies by Howell et al.7 and Permutt et al.8 indicate that blood can be displaced from the pulmonary vascular bed if the lungs are subjected to high transpulmonary pressures. Our observations and those of others6,9 indicate that producing higher intrathoracic pressures during CPR by simultaneous lung inflation and chest compression results in greater antegrade flow than does chest compression alone. This phenomenon could be accounted for by more forceful compression of and greater blood displacement from the pulmonary capillary bed and the generation of a greater peripheral arterial-to-venous pressure gradient mediated by functioning valves.

Another model of blood flow in the arrested circulation that has been studied extensively in our laboratories is “cough CPR,” in which pulsatile arterial blood pressures and antegrade flow have been produced by rhythmic coughing during ventricular fibrillation in man.11 Studies in animals12 indicate that cardiac compression does not occur with cough, that blood from the lung passes through the left heart in a manner identical to that seen in our studies of conventional CPR, and that generated aortic pressure and carotid flow are proportional to developed airway pressure. In addition, cough-induced closure of the venous valves at the thoracic inlet produces a peripheral arterial-to-venous gradient. Thus, substantial evidence exists to support the concept of the lung
as the reservoir of a functioning thoracic pump during the generation of high intrathoracic pressures, with the ventricles serving principally as conduits in the afferent and efferent limbs of the pump.

We do not know whether these findings in a dog model can be translated directly to human CPR. The anatomy of the canine thorax, particularly its anterior-posterior dimensions, is obviously different from that of man. One and one-half to 2 inches of sternal depression did not produce significant cardiac compression in the 18-40-kg dogs used in this study. Babbs\(^1\) reported that chest compression during circulatory arrest in small, flat-chested dogs produced compression of the heart. However, as our studies with more profound sternal depression indicate, proof of cardiac compression during CPR is not of itself proof that the cardiac compression is responsible for blood flow. Of importance is the observation that blood flows without apparent ventricular compression. Thomsen and co-workers\(^3\) recorded left atrial and femoral artery pressures during CPR in eight adult human subjects and found that these pressures increased to the same level. Mackenzie et al.\(^5\) noted no perfusion gradient between the systemic arterial bed and the right atrium during CPR on human subjects. Preliminary studies in man indicate that an intrathoracic-to-jugular venous pressure gradient is present during CPR (fig. 7). Venous valves in the jugular system have long been known to exist in humans, but their physiologic significance has not been established. We contend that these valves function principally during the generation of high intrathoracic and central venous pressures, as might occur during coughing or closed-chest CPR.

Werner and co-workers\(^6\) reported two-dimensional echocardiographic findings during CPR in man that agree with our cineangiographic findings in dogs. Their studies showed incomplete closure of the cardiac valves between compressions and lack of appreciable ventricular compression as well as simultaneous opening of both left-heart valves during sternal depression. Thus, although Werner et al.\(^7\) used different methods, their findings support the "thoracic pump" mechanism of CPR.

More direct observations in experimental animals and man are needed to determine which model, the cardiac pump or the thoracic pump, best explains the mechanism of blood flow during CPR.

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