Mechanism of Blood Flow Generated by Precordial Compression during CPR*

I. Studies on Closed Chest Precordial Compression

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The mechanism of forward flow produced by precordial compression during CPR was investigated with the aid of echocardiographic and hemodynamic measurements in anesthetized, mechanically ventilated domestic pigs. Both mitral and tricuspid valves were opened during compression diastole and closed during compression systole. Valve motion persisted throughout resuscitation in 17 of 22 animals which were hemodynamically resuscitated. There was a 25 percent reduction in left ventricular area during compression systole. Maximum pressure generated during compression systole in the aorta exceeded that of the right atrium throughout the 12-min interval of precordial compression in successfully resuscitated animals. These observations provide evidence of direct cardiac compression as the mechanism accounting for effective forward blood flow during CPR. The persistence of valve function, chamber compression, and pressure gradients during precordial compression was predictive of successful resuscitation. The absence of these factors prognosticates failure of resuscitation and explains, in part, the inconsistency of prior reports.

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There is consensus that CPR is lifesaving; yet the mechanism by which precordial compression produces and maintains forward flow is in dispute. In their initial report which launched modern day CPR, Kouwenhoven et al. assumed that there was direct cardiac compression during precordial compression which accounted for forward blood flow. Within the past decade, and spurred by the observation of Criley et al. that voluntary coughing during cardiac arrest can sustain blood flow, this "cardiac pump" theory was challenged. Rudikoff and co-workers and Niemann et al. proposed that precordial compression maintains blood flow by the ejection of blood from an intrathoracic reservoir with the implication that the heart serves as a passive conduit. They termed this the "thoracic pump" mechanism of blood flow.

This continuing controversy prompted us to undertake studies in a previously described canine model of cardiac arrest which was adapted to domestic pigs. More specifically, we investigated cardiac valve function, changes in the ventricular chamber dimensions and pressure gradients across intracardiac valves and between the right atrium and aorta during precordial compression. A preliminary report of the echocardiographic observations previously has been published. In addition, echocardiographic recordings were obtained on one human patient at the time of onset of cardiac arrest. This allowed us to document the effects of precordial compression on valve function and chamber deformation in the clinical setting.

METHODS

Experimental

Studies were performed on 27 immature domestic pigs weighing between 25 and 35 kg. The animals had been fasted for 12 h except that they were allowed free access to water. Anesthesia was induced with ketamine, 10 to 15 mg/kg body weight, by intramuscular injection and subsequently by ear vein injection of pentobarbital, 30 mg/kg. Neuromuscular blockade was induced with pancuronium bromide, 0.09 mg/kg. Animals were then intubated and ventilated with a volume-controlled ventilator (MA-1, Puritan-Bennett, Overland Park, KS) at a frequency of 12/min, tidal volume of 12 cc/kg, maximal flow rate of 40 L/min and FIO2 of 0.5. Anesthesia and neuromuscular blockade were subsequently supplemented with additional intravenous doses of pentobarbital and pancuronium.

Through the right femoral vein, a flow-directed thermobilization catheter (7F Pentalumen catheter, Gould Inc., Oxnard, CA) was surgically inserted under oscillographic guidance and flow-directed into the pulmonary artery. A Ducow 7F catheter (521-735, Cordis Corp., Miami, FL) was surgically inserted into the right femoral artery and advanced into the thoracic aorta. The right internal jugular vein was surgically isolated; a 5-F balloon-tipped CPV catheter (4.8-50-56A Cook, Inc., Bloomington, IN) was inserted through a catheter introducer sheath and flow-directed into the pulmonary artery. It was subsequently withdrawn into the right ventricle. An introducer sheath, 25 cm in length, was then inserted over the catheter into the right ventricle and secured. The balloon-tipped catheter then was removed and, after saline flush, the sheath was capped. This sheath provided the entry point for a wire...
electrode for ventricular fibrillation. All catheter positions were confirmed during fluoroscopy. Patency of catheters was maintained with minimal pressure flushes with saline containing 10 IU of heparin per milliliter. In five animals, a DuVoc 7-F catheter (Cordis Corp, Miami, FL) was inserted surgically through the left femoral artery and advanced to the aortic root and subsequently into the left ventricle with both oscilloscopic and fluoroscopic guidance.

Pressures were recorded in the intrathoracic aorta, pulmonary artery, and right atrium with fluid-filled catheters and pressure transducers (Statham P23DB, Gould Inc., Oxnard, CA) or by transducer-tipped catheters (Camino Laboratories, Model 110, San Diego, CA). These were referenced to midchest level at the fourth intercostal space. Immediately before insertion, transducer-tipped catheters were prewarmed, balanced, and calibrated. Right ventricular pressure was measured in 14 of the 22 animals with the transducer-tipped catheters. In five animals, left ventricular and aortic pressures simultaneously were measured with transducer-tipped catheters.

Cardiac output was measured by the continuous infusion thermodilution technique. The validity and reproducibility of this continuous infusion method at the levels of blood flow measured during precordial compression were established by in vitro flow-controlled studies on an isolated porcine heart (r = 0.99) and in vivo by comparison of cardiac output by bolus injection and continuous infusion thermodilution methods. Based on 14 observations in eight animals with a cardiac index ranging from 53 to 91 ml/kg, a correlation of 0.92 (p < 0.001) was obtained.

All dynamic data were continuously recorded on a multichannel recorder (Hewlett-Packard, Waltham, MA) with paper speed of 0.5 mm/sec and intermittent recordings at 25 mm/s. M-mode and two-dimensional echocardiography were performed with an Irex System III. A window was selected outside the mechanical compressor site over the left lateral midventricular area. Standard long axis and short axis views were recorded at mitral valve, aortic valve, and papillary muscle levels. The site of echocardiographic recording was marked on the skin and kept constant throughout the study. M-mode strips were obtained at a paper speed of 25 mm/s. Contrast echocardiograms were obtained by injecting 3 ml of agitated saline solution into the right ventricle in 22 animals and into the left ventricle in five animals. Data were recorded on conventional videotape for subsequent analysis.

After baseline recordings of hemodynamic and echocardiographic parameters were completed, ventricular fibrillation was induced by progressive increases in alternating current from 5 to 20 mA delivered to the right ventricular endocardium. Ventricular fibrillation was allowed to persist for one minute prior to the initiation of precordial compression. The compression pad of a mechanical compressor (Michigan Thumper model 10042, Michigan Instruments, Grand Rapids, MI) was centered over the lower one third of the sternum. Compression was maintained at rates of 60/min, a duty cycle of 50 percent, and depth of compression adjusted to decrease anteroposterior diameter by 25 percent. Animals were continuously ventilated with 100 percent oxygen at a minute volume of 180 ml/kg and a frequency of 12. The compression ventilation ratio was 5:1. Cardiac output was measured at 2, 6 and 10 min after beginning precordial compression and 2 min after restoration of spontaneous circulation in resuscitated animals. After 12 min of precordial compression, one or more 330 J DC countershocks were applied across the precordium (Life Pack 911, Physio-Control Corp, Redmond, WA).

Pressures were computed from ten sequential pressure pulses. Statistical significance was analyzed with multiple analysis of variance for subsets of resuscitated and nonresuscitated animals.

Echocardiographic data were analyzed with frame-by-frame review of recorded videotapes. Compression systole was readily identified by posterior movement of cardiac structures and diastole by anterior movement. Compression systole was also confirmed by auditory recording of the unique sound created by the initial downward travel of the mechanical compressor. Right-sided chambers were identified by injection of saline contrast medium into the

Table 1—Hemodynamic Measurements on 17 Resuscitated and Five Unresuscitated Animals (Mean ± SEM)

<table>
<thead>
<tr>
<th>Time After Ventricular Fibrillation</th>
<th>Aorta</th>
<th>Pulmonary Artery</th>
<th>Right Atrium</th>
<th>Cardiac Index (ml/kg/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Systolic</td>
<td>Diastolic</td>
<td>Mean</td>
<td>Systolic</td>
</tr>
<tr>
<td>Baseline**†‡</td>
<td>151 ± 5</td>
<td>120 ± 4</td>
<td>135 ± 5</td>
<td>31 ± 3</td>
</tr>
<tr>
<td>Resuscitated</td>
<td>122 ± 25</td>
<td>99 ± 10</td>
<td>113 ± 10</td>
<td>23 ± 4</td>
</tr>
<tr>
<td>Nonresuscitated</td>
<td>68 ± 5</td>
<td>26 ± 3</td>
<td>45 ± 3</td>
<td>54 ± 6</td>
</tr>
<tr>
<td>2 min‡</td>
<td>39 ± 4</td>
<td>18 ± 3</td>
<td>29 ± 3</td>
<td>55 ± 5</td>
</tr>
<tr>
<td>Resuscitated</td>
<td>65 ± 4</td>
<td>26 ± 2</td>
<td>46 ± 3</td>
<td>59 ± 5</td>
</tr>
<tr>
<td>Nonresuscitated</td>
<td>47 ± 7</td>
<td>17 ± 2</td>
<td>32 ± 4</td>
<td>48 ± 5</td>
</tr>
<tr>
<td>4 min‡</td>
<td>77 ± 5</td>
<td>28 ± 3</td>
<td>52 ± 3</td>
<td>72 ± 6</td>
</tr>
<tr>
<td>Resuscitated</td>
<td>51 ± 11</td>
<td>15 ± 4</td>
<td>32 ± 5</td>
<td>58 ± 11</td>
</tr>
<tr>
<td>Nonresuscitated</td>
<td>80 ± 5</td>
<td>29 ± 2</td>
<td>54 ± 3</td>
<td>76 ± 6</td>
</tr>
<tr>
<td>10 min‡</td>
<td>45 ± 6</td>
<td>13 ± 2</td>
<td>30 ± 3</td>
<td>56 ± 10</td>
</tr>
<tr>
<td>Resuscitated</td>
<td>77 ± 5</td>
<td>27 ± 2</td>
<td>51 ± 3</td>
<td>68 ± 5</td>
</tr>
<tr>
<td>Nonresuscitated</td>
<td>46 ± 7</td>
<td>12 ± 2</td>
<td>29 ± 3</td>
<td>52 ± 7</td>
</tr>
<tr>
<td>POST-CPR§</td>
<td>126 ± 8</td>
<td>87 ± 10</td>
<td>105 ± 8</td>
<td>36 ± 3</td>
</tr>
</tbody>
</table>

*Baseline measures 5 min prior to ventricular fibrillation.
†Values not significant for columns headed Aorta and Cardiac Index.
‡Values had p < 0.01 for columns headed Aorta and Cardiac Index.
§Values for 2 min after successful cardioversion.
Coronary Perfusion Pressure

**Figure 1.** A comparison of coronary perfusion pressure during precordial compression in resuscitated and nonresuscitated animals. Mean ± SEM are shown. The differences during precordial compression were highly significant (p<0.003).

right atrium. The opening and closing of valves were echocardiographically identified. The direction of flow was established by echocardiographic imaging of the contrast tracer. Right ventricular internal diameter was measured in the long axis at the maximal anteroposterior diameter at end-diastole and end-systole. Left ventricular area was computed by electronic planimetry. Differences between diastolic and systolic dimensions were statistically analyzed with the Student's t test for paired samples.

**Clinical Study**

An echocardiographic study was performed on a 35-year-old man, a known heroin addict, who was admitted because of fever and progressive shortness of breath of two days duration. On clinical examination, the heart rate was 130/min, and the blood pressure was 90/70 mm Hg. Jugular venous distension and pulsus paradoxus of 20 mm Hg were documented. The bedside echocardiogram revealed pericardial effusion with diastolic collapse of the right ventricle indicative of pericardial tamponade. Blood cultures subsequently yielded pneumococci. Because of rapid hemodynamic deterioration, a pericardiocentesis by epigastric approach was performed with echocardiographic guidance. During this procedure, the onset of ventricular fibrillation prompted immediate CPR with manual chest compressions at a rate of 60/min. Normal sinus rhythm with return of spontaneous circulation was reestablished after 3 min of CPR immediately following electrical defibrillation with a single 200 J DC shock. However, pericardiocentesis, fluid resuscitation and vasopressor administration failed to reverse progressive fall in blood pressure and the patient died six h after initial resuscitation from cardiac arrest.

**Results**

**Hemodynamic Measurements:** The hemodynamic observations on 17 resuscitated and five nonresuscitated animals are summarized in Table 1. The baseline aortic and pulmonary artery pressures were lower in the nonresuscitated animals although the differences were not statistically significant. During 12 min of precordial compression, the mean aortic pressure was 51 ± 3 (mean ± SEM) in successfully resuscitated animals and 29±3 mm Hg in animals which failed to resuscitate (p<0.01). Cardiac index was 27.3±2 in resuscitated and 19.4±3.2 ml/kg/min in nonresuscitated animals (p<0.01). Coronary perfusion pressure was computed as the difference between aortic diastolic pressure during precordial compression and time coincident right atrial pressure. In 17 resuscitated animals, coronary perfusion pressure averaged 15 mm Hg. In fatalities it was 4 mm Hg. The differences were significant at the p<0.003 level (Fig 1). Significant systolic pressure gradients between the aorta and the right atrium of 16 mm Hg or greater were observed in animals which were successfully resuscitated. To the contrary, no such gradient was observed in nonresuscitated animals (Fig 2). After 12 min of precordial compression, the systolic pressure gradient between the aorta and right atrium was 20.3±3.0 in 17 resuscitated animals and 1.2±0.5 in five nonresuscitated animals.

**Experimental Studies**

**Intrathoracic Systolic Pressure Gradient during Precordial Compression**

**Figure 2.** The pressure gradient between the aorta and the right atrium during precordial compression, termed the intrathoracic pressure gradient.
Valve Motion: Mitral valve opening and closure were documented in each of the 17 animals in which satisfactory echocardiograms were recorded during the initial 5 min of precordial compression. The same applied to 12 pigs in whom the tricuspid valve was visualized. During compression diastole, the atrioventricular valves opened widely (Fig 3). During compression systole, there was complete closure of the atrioventricular valves. After 5 min, atrioventricular valves remained in the open position in five animals in which DC countershock subsequently failed to restore a viable ventricular rhythm. This contrasted with 12 of the 17 animals in whom valve motion persisted throughout the 12 min of CPR and who were hemodynamically resuscitated and each of these animals maintained spontaneous circulation for 2 h or more. Aortic valve opening also was observed during compression systole and valve closure during compression diastole in 20 animals. However, aortic valve closure was absent after 5 min in the five animals in which resuscitation efforts failed.

Contrast Studies: Studies in 17 animals indicated forward flow into the right ventricular outflow tract during compression systole with mild regurgitation from the right ventricle to right atrium. Five animals demonstrated forward flow into the aortic root during compression systole following injection of contrast into the mid-left ventricle (Fig 4, top). There was, at most, mild systolic mitral regurgitation in a minority of studies. During compression diastole, a negative contrast image was observed in the left ventricular cavity.

This represented diastolic blood flow from left atrium to left ventricle across the mitral valve (Fig 4, bottom).

Valvular Pressure Gradients: A peak systolic pressure gradient of 19.3 ± 6 mm Hg (mean ± SEM) was documented between the right ventricle and right atrium at peak of compression systole in 14 resuscitated animals. In each of the resuscitated animals, this gradient exceeded 11 mm Hg (Fig 5). However, there were no pressure differences between the right atrium and right ventricle during diastole. Time coincident diastolic pressure gradients between the pulmonary...
Figure 5. Right ventricular-right atrial systolic peak pressure gradients (RV-RA) during precordial compression (mean ± SEM). Measurements were obtained on 14 animals, each of which was successfully resuscitated.

The pressure gradient between the left ventricle and the aorta was measured in five successfully resuscitated animals. The pressure in the left ventricle exceeded that of the aorta by 10 ± 1.2 mm Hg during compression systole and the pressure in the aorta exceeded that of the left ventricle by 12 ± 1.2 mm Hg during compression diastole.

Ventricular Systolic and Diastolic Dimensions: Satisfactory long axis echocardiographic recordings of the right ventricle were obtained in 20 (15 resuscitated and 5 nonresuscitated) animals during precordial compression. The anteroposterior diameter was decreased from 1.8 cm ± 0.5 to 1.2 cm ± 0.02 (p < 0.005) during peak precordial compression.

Figure 6. Pulmonary artery diastolic-right atrial (PA-RA) diastolic pressure gradients during precordial compression. The gradient declined after 4 min of CPR in five unresuscitated animals (mean ± SEM).

Figure 7. Echocardiographic (2D) long axis view during CPR. A diastolic image of the left ventricle is shown in the top panel and the effects of systolic compression in the lower panel.

Long and short axis views of the left ventricle were technically satisfactory in 15 resuscitated animals prior to, during, and following reversal of cardiac arrest. The systolic left ventricular area during precordial compression was 6.95 cm² ± 0.52 cm² and diastolic area was 9.14 cm² ± 0.34 cm². Accordingly, precordial compression was accompanied by an approximately 25 percent decrease in ventricular area (p < 0.005). Representative images are shown in Figure 7.

Clinical Data

Apical four-chamber views during manual chest compression systole and compression diastole (release phase) of the patient are shown in Figure 8. Manual
precordial compression at a rate of 60/min was accompanied by both mitral and tricuspid valve closure together with decreases in right and left ventricular chamber dimensions. During compression diastole, both mitral and tricuspid valve opening were observed along with increased right and left ventricular dimensions.

**Discussion**

The predominant importance of the ABCs as determinants of successful cardiopulmonary resuscitation has been securely established. However, there is no consensus on the mechanism by which precordial compression maintains systemic blood flow. Both increases in thoracic pressure ("thoracic pump") and direct cardiac compression ("cardiac pump") theories have been supported. Since these two differing mechanisms imply different interventions for maintaining maximal blood flow, the issue is of great practical moment.

Kowenhaven et al. reported on the combined techniques of artificial respiration, sternal compression and electrical defibrillation for resuscitation. They hypothesized that forward blood flow during resuscitation is maintained by compression of the heart between the sternum and vertebral column. Weale and Bothwell-Jackson found that ilioc vein pressure increases to a level which approximates that of arterial pressure during precordial compression and questioned whether there was forward flow. Criley et al. and Niemann et al. observed that repeated coughing in human patients or experimental animals during cardiac arrest maintained forward blood flow during ventricular fibrillation without external compression. Rudikoff and co-workers measured the aortic-central venous pressure gradient during chest compression. They demonstrated that the pressures in all cardiac chambers and the thoracic aorta increased to the level of the intrapleural pressure. Niemann et al. confirmed these observations and showed by cineangiographic techniques that blood passes from the pulmonary veins through both left heart chambers to the aorta during a single compression. As a result of these observations, they concluded that the blood flow during CPR results principally from an increased intrathoracic pressure. Retrograde flow from the superior vena cava is prevented, in part, by venous valves in the jugular and subclavian veins near their inlet into the thorax. This concept of "thoracic pump" was further supported by echocardiographic studies.

In human subjects during CPR, Werner et al. and Rich et al. observed that the mitral and aortic valves remained open and that there was no significant change in left ventricular cross-sectional area in relation to chest compression.

Yet, the "cardiac pump" theory was supported by Maier et al. who studied chronically instrumented dogs. Babbs et al. demonstrated forward blood flow during precordial compression in dogs when the chest was vented to the atmosphere. The same investigators demonstrated decreased ventricular dimension during chest compression by cineangiography in animals with cardiac arrest.

Following publication of our initial studies in minipigs during CPR, which demonstrated both mitral valve motion and chamber deformation during chest compression, Feneley et al. reported their observations in manual CPR in dogs. They reaffirmed mitral valve closure during "compression systole." They also documented that there was antegrade transmitral blood flow only during "compression diastole." Simultaneous recordings of left ventricular and left atrial pressures yielded peak pressure gradients (mean ± SEM) of 38.5 ± 4 mm Hg.

The present study documents changes which fulfill the criteria of a cardiac pump mechanism. Mitral valve opening was observed during compression diastole and valve closure with compression systole (Fig 3). We documented a substantial right ventriculoatrial pressure gradient in animals which were successfully resuscitated. The same applied to the pulmonic valve. Echocardiographic documentation of valve opening during "compression systole" was obtained only in animals which were successfully resuscitated. Negative contrast images confirming blood flow from the left atrium to the left ventricle documented antegrade blood flow during "compression diastole." Contrast medium from both right and left ventricular chambers moved toward the outflow tracts indicating systolic filling of both pulmonary and systemic beds. The diastolic gradient between the pulmonary artery and the right atrium in our studies supports our observations of systolic forward flow. Finally, our studies documented deformation of both right and left ventricular chambers.

More recently, Raessler et al. noted that systolic pressure gradients between the right atrium and the aorta during cardiac arrest in dogs were greatest during open-chest cardiac massage which represents true cardiac compression. It was intermediate during external mechanical CPR and standard manual CPR, which we regard as cardiac compression. It was lowest after combined abdominal and thoracic vest compression, which is primarily intended to increase intrathoracic pressure and therefore has no direct effect on the heart. Indeed, Weisfeldt and Chandra, proponents of the "thoracic pump" theory, also observed that aortic pressure was much higher than right atrial pressure during chest compression in several of their dogs.

The applicability of these echocardiographic observations to a single human patient provides evidence...
that these studies in immature domestic pigs and the studies by Feneley and his colleagues are of clinical import in medical practice. MacKenzie et al measured brachial artery pressure, right atrial pressure and cardiac output in human patients after cardiac arrest and during external compression and confirmed a gradient of approximately 25 mm Hg between the brachial artery and right atrium during compression systole.

This contrasts with human studies reported by Werner et al and Rich et al who found no valve motion or chamber deformation with echocardiographic imaging. Yet these authors do not specify time relation of their studies to the duration and success of CPR. It is likely that the studies were performed after cardiac viability had been lost. It was under these conditions that chamber deformation and valve function had ceased. This would be expected from our experimental observations in which valve function, decreases in left ventricular dimensions, and pressure gradients were no longer in evidence after 5 min of precordial compression in nonresuscitated animals.

These observations therefore provide evidence, in this animal model, that forward flow is associated with cardiac compression. Our studies also indicate that the aortic right-atrial systolic gradient >20 mm Hg is not only indicative of cardiac pump mechanism during precordial compression but also predicts resuscitation success or failure. As yet unsettled is the possibility that the mechanism may differ both within and between species in part based on chest configuration, body size and maturity of experimental animals. Yet our observations in minipigs were consistent with that of a human case observed by us and selective human cases reported by others.

The practical implications of a primary cardiac pump mechanism relate to the possibility that circumferential chest compression, simultaneous ventilation and chest compression, abdominal binding or other options by which intrathoracic pressure is increased would be likely to improve blood flow to vital organs. As yet, the efficacy of such are not firmly established. The studies herein reported which point to a primary cardiac pump mechanism would further dampen enthusiasm for such interventions.

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