The physiology of external cardiac massage: high-impulse cardiopulmonary resuscitation

George W. Maier, M.D., George S. Tyson, Jr., M.D., Craig O. Olsen, M.D., Kemp H. Kernsnein, M.D., James W. Davis, M.S.E.E., Eric H. Conn, M.D., David C. Sabiston, Jr., M.D., and J. Scott Rankin, M.D.

ABSTRACT In intact chronically instrumented dogs, left ventricular dynamics were studied during cardiopulmonary resuscitation (CPR). Electromagnetic flow probes measured cardiac output and coronary blood flow, ultrasonic transducers measured cardiac dimensions, and micromanometers measured left ventricular, right ventricular, aortic, and intrathoracic pressures. The dogs were anesthetized with morphine, intubated, and fibrillated by rapid ventricular pacing. Data were obtained during manual external massage with dogs in the lateral and supine positions. Force of compression was varied from a peak intrathoracic pressure of 10 to 30 mm Hg, and compression rate was varied from 60 to 150/min. Increasing force of compression increased stroke volume up to a peak intrathoracic pressure of approximately 20 mm Hg, beyond which stroke volume remained constant or declined. Stroke volume appeared to result primarily from direct transmission of manual compression force to the heart rather than from positive intrathoracic pressure because peak cardiac or vascular pressures or the change in these pressures were consistently two to four times greater than the corresponding intrathoracic pressures during manual compression. With increasing compression rate, stroke volume remained relatively constant, and total cardiac output increased significantly: 425 ± 92 ml/min at 60/min, 643 ± 130 ml/min at 100/min, and 975 ± 219 ml/min at 150/min (p < .05). Left ventricular dimensions decreased minimally at higher manual compression rates. In four patients undergoing CPR, systolic and diastolic arterial blood pressure increased with faster compression rates, correlating well with data obtained in the dog. Dynamic coronary blood flow in canine experiments decreased to zero or negative values during compression. Antegrade coronary flow occurred primarily during noncompression periods and seemed to be related to diastolic aortic perfusion pressure; coronary flow at a compression rate of 150/min averaged 75% of control. Therefore stroke volume and coronary blood flow in this canine preparation were maximized with manual chest compression performed with moderate force and brief duration. Increasing rate of compression increased total cardiac output while coronary blood flow was well maintained. Direct cardiac compression appeared to be the major determinant of stroke volume during manual external cardiac massage.


CARDIOPULMONARY RESUSCITATION (CPR) has been practiced in various forms since the beginning of recorded history. Galen in 175 AD described the use of bellows to inflate the lungs of a dead animal.1,2 In 1775, standards were set for resuscitation by the Royal Humane Society, which stated that the most efficacious method was “to blow with force into the lung, by applying the mouth to that part of the patient, closing his nostrils with one hand, and gently expelling the air again by pressing the chest with the other.”3 Sternal compression was suggested in 1786 by John Sherwin, a surgeon of Enfield: “The surgeon should go on inflating the lungs and alternately compressing the sternum.”4

Relevant experimentation in CPR began in 1874 when Schiff performed the first open-chest cardiac massage in the dog. Boehm in 1878 administered closed-chest cardiac massage to cats by compressing the sides of the thoracic cage together.4 Dr. Franz Koenig, professor of surgery at Gottingen, Germany, is credited as the father of external cardiac compression and reported in 1885 six successful resuscitative efforts in man. Maass in 1892 reported a modification...
of the Koenig technique and proposed that the "rate of compression should be very rapid — 120 or more per minute." However, it was not until 1960 that Kouwenhoven et al. combined the techniques of artificial respiration, sternal compression, and electrical defibrillation to resuscitate dogs and eventually human beings. At this point, modern CPR was born.

Initially, it was assumed that blood flow during CPR occurred by direct compression of the heart between the sternum and spine. Recently, however, Niemann and associates and Rudikoff and co-workers proposed that blood flow occurs not by direct cardiac compression but rather by a thoracic pump mechanism. According to this theory, increased intrathoracic pressure or the change in intrathoracic pressure during external compression forces blood from the thoracic vessels into the systemic circulation, with the heart acting as a conduit and not as a pump. This hypothesis was supported by Criley et al., who observed that the repetitive coughing of patients having cardiac arrest in the catheterization laboratory maintained blood flow without external compression. Further work by this group emphasized the passive role of the heart and the closure of venous valves at the thoracic inlets. Werner et al., using two-dimensional echocardiography, observed that the mitral and aortic valves were open and that cardiac chamber size did not change during external chest compression. On the basis of these data, a thoracic pump mechanism of blood flow seemed highly plausible. However, few investigators have directly measured cardiac dimensions, flows, and pressures simultaneously during external massage, and little is known about cardiac dynamics during chest compression. Therefore this study was undertaken to investigate the effects of varying manual compression rate, force, and duration on the physiology of external cardiac compression in the intact heart.

Methods

Experimental preparation. Twenty-four adult mongrel dogs (25 to 35 kg) were surgically instrumented for long-term studies. The dogs were anesthetized with sodium thiamylal (25 mg/kg) and ventilated with a volume respirator (Model 550; Ohio Medical Products, Madison, WI). As described in previous communications, a left thoracotomy was performed under sterile conditions through the fifth intercostal space. A pericardiotomy was made from the apex of the heart to the great vessels. Pairs of pulse transit ultrasonic dimension transducers (resonant frequency 3 to 5 MHz) were implanted on the left ventricle to measure minor axis and septal-free wall diameters (figure 1). In eight dogs, major axis diameter and right ventricular free wall to left ventricular free wall dimension were measured as well. Through a purse-string suture in the base of the left atrial appendage, a heparin-filled silicone rubber catheter (od 4.9 mm, id 2.6 mm; Dow Corning, Midland, MI) was placed directly into the left atrium for subsequent passage of a micromanometer across the mitral valve into the left ventricle. A similar catheter was positioned in the cavity of the right ventricle, and another catheter with multiple side holes was sutured to the epicardial surface of the base of the heart for subsequent measurement of pleural pressure with a micromanometer. An electromagnetic aortic flow probe (series TTQ; Howell Instruments, Camarillo, CA) was placed around the ascending aorta of all dogs. In 13 dogs an electromagnetic coronary flow probe (Series HST; Howell Instruments) was placed around the left circumflex coronary artery as well. An inflatable balloon occluder was secured distal to the coronary flow probe. Bipolar epicardial pacing electrodes were sutured to the right ventricle, and the pericardium was left open in all animals. The transducer leads, catheters, and occluder tubes were implanted in a subcutaneous pouch dorsal to the thoracotomy incision until the time of study. Postoperatively, the dogs received a single dose of intramuscular fentanyl hydroxide (100 mg) and daily intramuscular injections of procaine penicillin G (200,000 U) and dipyridamole (250 mg).

Three additional dogs were instrumented specifically for multiple pressure measurements with implantation of introducers in the right and left atria, right and left ventricles, pulmonary artery, and intrapleural space. At the time of study, a balloon catheter was connected to a Statham P23dB transducer and positioned in the retrocardiac esophagus for correlation of eso-
phageal balloon pressure to intrathoracic pressure measured with the micromanometer.

**Instrumentation and data acquisition.** Each dog was studied 10 to 14 days after instrumentation. One hour before study, 5 to 7 mg of morphine sulfate was administered intramuscularly. The subcutaneous pouch was opened with animals under 1% lidocaine local anesthesia, and the transducer leads were exteriorized. The dimension transducers were coupled directly to a third-generation sonomicrometer designed and constructed in our laboratory. With this system the time delay from transmission of a burst of ultrasound from one piezoelectric transducer to the reception of the sonic wave by an identical transducer was converted into an analog output. A single-pole filter was used to produce a 6 dB per octave attenuation above 100 Hz; therefore, the practical frequency response was 0 to 50 Hz. The minimum resolution of the dimension system was approximately 0.08 mm, and the maximum electronic drift was 0.05 mm/hr.

Pressure measurements were obtained with high-fidelity micromanometers (Millar PC-350; Houston) coupled to pressure amplifiers (Hewlett-Packard 8805-C, Waltham, MA). Each micromanometer has a useful frequency response in excess of 10 kHz, and the pressure waveforms were filtered with an upper cut-off of 50 Hz, determined by a two-pole Butterworth filter. The manometers were kept in a water bath at 38°C, were constantly excited electrically, and were simultaneously calibrated and balanced immediately before each experiment. Prewarmed in this manner, the transducers had negligible temperature sensitivity and drift (less than 0.5 mm Hg/hr). At the time of study, the manometers were passed through the implanted silicone rubber catheters to obtain left ventricular, right ventricular, and intrathoracic pressures. Ascending or descending thoracic aortic pressure also was measured in all dogs by a manometer passed through either the carotid or femoral artery. In four dogs additional Millar manometers measured pressure in the descending thoracic and abdominal aorta. In three dogs micromanometers were introduced through implanted tubes into both atria, both ventricles, the pulmonary artery, and the pleural space, while a Statham P23dB transducer measured esophageal balloon pressure.

The electromagnetic flow probes were connected to Statham M4001 gated sine-wave flowmeters and were calibrated before and after implantation by a gravity-fed saline system. Calibrations were constant to within ±6% before and after each study, and the slope of the calibration curve was linear to within ±2%. At the time of study, zero aortic flow was assumed at end-diastole or just before compression during CPR. Zero coronary blood flow was obtained either with a brief coronary occlusion in the control state or during a prolonged noncompression period during fibrillation. Coronary physiology was assumed to be normal if peak reactive hyperemic blood flow after a 20 sec coronary occlusion was in excess of 250% of control values. In addition, data were accepted for analysis only if no baseline drift of the flow measurements occurred during the study and if no motion artifacts were noted on the coronary flow curve with compressions during complete coronary occlusion. If these criteria were present, the probes were firmly seated on the vessels, and reproducible results were obtained. The coronary flow data presented in this article represent five studies that met these criteria out of a total of 13 dogs undergoing implantation of coronary flow probes. The validation of these preparations and more detailed descriptions of the data acquisition techniques have been published elsewhere.16-19

Data were recorded with an FM tape system (Model A; Vetter, Rebersburg, PA) while dogs were conscious and under steady-state control conditions. General anesthesia then was induced by intravenous injection of morphine sulfate (2 mg/kg), and succinyl choline (1 to 2 mg/kg) was administered intrave-

nously. Each dog was intubated with a cuffed endotracheal tube and ventilated with a Harvard respirator at a tidal volume of 650 ml and a respiratory rate of 12/min. The dogs were volume loaded as necessary by intravenous infusion of Normosol R (pH 7.4) to a left ventricular end-diastolic transmural pressure of 13 to 15 mm Hg. Ten thousand units of sodium heparin was administered intravenously, and the ventricular pacing wires were connected to a programmable pacemaker. Ventricular fibrillation was induced in all studies by rapid ventricular pacing.

After a cardiac arrest period of 30 sec, manual external cardiac massage was begun with the dog lying in the supine or lateral position. Evaluation of the flow probes was performed as previously described; if baseline drift or motion artifacts occurred as a result of mobility of the probes on the vessel, the dog was not used for study. In general, drift was infrequent; all experiments were performed 1 to 2 weeks after implantation because of firm healing of the probes to the vessel wall. Compression force was first varied while compression rate was held constant at 100/min. Compression force was assessed by measuring peak intrathoracic pressure and was increased in an arbitrary range of low to middle to high, corresponding approximately to 10, 20, and 30 mm Hg peak intrathoracic pressure, respectively. Dynamic intrathoracic pressure was displayed on a storage oscilloscope to regulate compression force. Compression rate then was varied from 60 to 100 to 150/min, while compression force was held constant at approximately 20 mm Hg peak intrathoracic pressure. Manual compression rate was regulated by synchronizing compressions to a crystal-controlled digital beeper apparatus. At least 45 to 60 sec of steady-state data were recorded at each intervention. In each experiment the sequence of interventions was varied randomly and did not seem to affect the results. All quantitative studies were completed within 5 min of cardiac arrest. Seven of the dogs underwent external compression in the supine position with a pneumatic mechanical device (Thumper, Michigan Instruments). For this portion of the study, the dogs were held firmly in a cradle and a specially designed yoke attachment on the Thumper compressed the sternum. A complete autopsy was performed at the conclusion of each experiment.

**Data analysis.** Analog data were converted to digital form by an analog-to-digital converter (Model 1012; ADAC, Woburn, MA) either directly on-line at the time of study or subsequently from FM tape. The sampling rate for each sweep of eight channels was 200 Hz. Total conversion time per channel (hardware and software) was approximately 30 μsec. Therefore the delay from the first to the eighth channel was 240 μsec, or approximately 5% of the sampling interval. For 50 Hz signals if the experiment was performed 1 and 8 (worst case), the phase delay was 4.5 degrees. Computer analysis of all data was accomplished with a microprocessor (Model PDP 11/23; DEC, Maynard, MA) and interactive programs developed in our laboratory. Short-term data storage was accomplished on hard disc (DEC, RL01); digital magnetic tape was used for long-term data storage (Model 100X; Cipher Data Products).

In each study, two 10 sec periods of control data recorded just before arrest were digitized. During CPR, 10 sec segments of data were digitized at each intervention of the protocol during steady-state conditions. Variations in the end-diastolic diameters were assumed to represent changes in ventricular volume and filling during the noncompression period as discussed elsewhere.20

Stroke volume was calculated digitally from the aortic blood flow measurement. Zero flow was assumed before compression, and the curve was integrated from the zero value before compression to the point where the flow again reached that value. Stroke volume was averaged over the 10 sec period, and for each intervention cardiac output was obtained by multiply-

Downloaded from http://circ.ahajournals.org/ by guest on November 12, 2017
ing the average stroke volume by the average compression rate. Average coronary blood flow was calculated by integrating the coronary flow measurements over each 10 sec steady-state period. Peak pressure data were computed by averaging maximum pressures during compression over a 10 sec period for each intervention. Mean diastolic aortic pressure was calculated by averaging all diastolic values for aortic pressure over the entire 10 sec for each intervention. Stroke volume, cardiac output, mean coronary blood flow, peak pressure data, mean and diastolic aortic pressure, and end-diastolic diameter were compared by Student’s t test for paired data with data measured during each preceding intervention.

Hemodynamic data from the eight studies of compression rate performed with morphine anesthesia were compared with similar data obtained in six preliminary studies that used intravenous sodium thiamylal (25 mg/kg) for anesthesia. Differences were evaluated by Student’s t test for unpaired data.

Patient protocol. After approval by the Duke University Human Investigation Committee, hemodynamic studies were performed in four patients undergoing CPR. Three patients sustained cardiac arrest caused by left ventricular failure after myocardial infarction; a fourth had intractable ventricular tachycardia and fibrillation from cardiomyopathy. As part of the resuscitative effort, a femoral arterial catheter was inserted to sample arterial blood gases and to measure blood pressure with a Statham P23Db transducer. During the resuscitation, manual sternal compression force was maintained at moderate levels, and compression rate was increased from 60 to 100 to 150/min. After 1 min at each rate, the resuscitation was continued at whichever rate achieved the highest blood pressure. Measurements of femoral arterial pressure were recorded continuously, and every effort was made to optimize hemodynamics during the resuscitation. Thus the interventions were considered to be potentially therapeutic.

Results

Typical control data are compared in figure 2 with those obtained during manual compression at a rate of 60/min. During control conditions, concentric ejection-phase shortening of all cardiac dimensions was observed, characteristic of normal stroke volume. The relationships between left ventricular pressure, right ventricular pressure, ascending aortic pressure, and aortic flow also were normal. During manual CPR, with the dog lying in the lateral position, left ventricular dimensions changed with compression but not in a concentric fashion. During compression, left ventricular minor axis diameter decreased, but major axis diameter and right ventricular to left ventricular free wall diameter initially increased as the cardiac chambers were flattened. End-diastolic dimensions during CPR and ventricular fibrillation were equivalent or slightly smaller than control in most dogs and changed very little with any intervention. Aortic blood flow during compression was similar in configuration to control but had a sharper upstroke and decline; stroke volume and peak aortic flow during CPR were significantly reduced. Right and left ventricular pressures during CPR were essentially equivalent, reaching peak values of approximately 100 mm Hg. Peak intrapleural pressure was 12 mm Hg, while ascending aortic pressure reached approximately 70 mm Hg. Average values are presented in table 1.

Hemodynamic measurements during manual compression in the lateral and supine positions are illustrated in figure 3. Aortic flow and left ventricular pressure were qualitatively similar in both positions, but pressure and flow were greater in the lateral position. In this particular study, compression force and cardiac filling were somewhat higher in the lateral position than in the supine position, accounting for the augmentation of left ventricular pressure and aortic flow. In addition, generation of pressure and flow by direct chamber compression seemed to be better in the lateral position in most studies. The observed changes in cardiac dimensions seemed to be dependent on the direction of the compression force. In the lateral position, the minor axis diameter decreased and the septal free wall diameter expanded during compression, while in the supine position the opposite change in shape was noted.

Multiple aortic pressure data are illustrated in figure 4. During compression, left ventricular and intrapleural pressures increased, attaining peak values at approximately the same time. Aortic flow increased later in the compression, and peak aortic flow occurred after peak left ventricular pressure. Aortic pressure measured in the descending thoracic and abdominal aorta showed a bimodal pressure rise. The first pressure increment occurred during peak compression, and the second was observed after peak aortic flow. The first pressure increment was fairly synchronous along the aorta and seemed to be temporally related to external compression. The second increment was observed progressively later as measurements were obtained distally along the aorta. It is hypothesized that the second aortic pressure rise represented propagation of ventricular stroke volume along the aorta after compression.

Data obtained during mechanical compression in the supine position with the mechanical compression device are illustrated in figure 5. During mechanical compression, peak intracardiac and aortic pressures were approximately equivalent at 45 mm Hg. However, the pressure at the epicardial surface was considerably lower than that in the vascular structures, attaining a peak of only 15 mm Hg. Left ventricular minor axis diameter increased with sternal compression in the supine position, and in all dogs a flattening of cardiac shape was observed with mechanical compression.
Esophageal balloon pressure did not correlate well with intrapleural pressure during compression, primarily because of impact artifacts on the balloon, which made balloon pressure appear considerably higher than intrathoracic pressure. Furthermore, multiple oscillations occurred in esophageal balloon pressure during transmission of compression force through the chest. This finding is characteristic of a fluid-filled system with low-frequency response, and the physical nature of a balloon catheter makes it extremely sensitive to motion artifacts.

Typical measurements of minor axis diameter, aortic blood flow, coronary blood flow, aortic pressure, and left ventricular pressure are depicted in figure 6 during mechanical and manual compression with dogs in the supine position. Compression rate was 60/min for the mechanical data and 100/min for the manual data. Mechanical compression was performed at a force of 80 psi with a depth of 2 inches. Both methods produced similar dimension changes, but the duration of mechanical compression was longer with the mechanical device. Aortic flow was quite different with the two methods. Peak aortic flow was higher with manual compression, whereas mechanical compression produced an initial peak and then a plateau in the flow curve. A greater negative aortic flow in early diastole was noted with the manual method, which correlated well with the greater peak diastolic coronary blood flow. As shown in figure 6, coronary blood flow was slightly negative early in compression with both techniques. An initial retrograde coronary flow was not surprising, since left ventricular pressure was higher than aortic pressure during the early phase of compression. Peak and mean coronary flow were consis-
## TABLE 1
Effects of manual compression rate on hemodynamics in dogs with cardiac arrest

<table>
<thead>
<tr>
<th>Compression rate</th>
<th>PPLP (mm Hg)</th>
<th>MDPLP (mm Hg)</th>
<th>PLVP (mm Hg)</th>
<th>MDLVP (mm Hg)</th>
<th>PAP (mm Hg)</th>
<th>EDAP (mm Hg)</th>
<th>MAP (mm Hg)</th>
<th>MDAP (mm Hg)</th>
<th>EDD (mm)</th>
<th>Stroke volume (ml/min)</th>
<th>Cardiac output (% control)</th>
<th>CBF (% control)</th>
</tr>
</thead>
<tbody>
<tr>
<td>60/min</td>
<td>20 ± 2</td>
<td>−2 ± 1</td>
<td>108 ± 9</td>
<td>19 ± 2</td>
<td>84 ± 8</td>
<td>35 ± 6</td>
<td>44 ± 5</td>
<td>40 ± 5</td>
<td>63 ± 2</td>
<td>7.1 ± 1.5</td>
<td>425 ± 92/16</td>
<td>64 ± 15</td>
</tr>
<tr>
<td>100/min</td>
<td>21 ± 1</td>
<td>−2 ± 1</td>
<td>110 ± 8</td>
<td>20 ± 2</td>
<td>88 ± 7</td>
<td>42 ± 6b</td>
<td>52 ± 5b</td>
<td>47 ± 5b</td>
<td>60 ± 2</td>
<td>6.4 ± 1.3</td>
<td>643 ± 130/24b</td>
<td>76 ± 12</td>
</tr>
<tr>
<td>150/min</td>
<td>20 ± 3</td>
<td>−1 ± 1</td>
<td>122 ± 8</td>
<td>20 ± 3</td>
<td>91 ± 7</td>
<td>44 ± 4c</td>
<td>59 ± 4c</td>
<td>52 ± 4c</td>
<td>60 ± 2</td>
<td>6.5 ± 1.4</td>
<td>975 ± 220/36c</td>
<td>75 ± 6</td>
</tr>
</tbody>
</table>

PPLP = peak pleural pressure; MDPLP = mean diastolic pleural pressure; PLVP = peak left ventricular pressure; MDLVP = mean diastolic left ventricular pressure; PAP = peak thoracic aortic pressure; EDAP = end-diastolic aortic pressure; MAP = mean aortic pressure; MDAP = mean diastolic aortic pressure; EDD = end-diastolic diameter; CBF = coronary blood flow.

Data represent mean ± SEM of observations in eight dogs, except for coronary flow data (CBF), which represent mean ± SEM of observations in five dogs. Each observation was averaged from 10 sec of steady-state digital data.

| Significant difference (p < .05) between 60 and 100/min by two-tailed Student’s t test for paired data. |
| Significant difference (p < .05) between 100 and 150/min by two-tailed Student’s t test for paired data. |

The effect of varying compression force is shown in figure 7. Manual compression force was progressively increased from a peak intrapleural pressure of 10 to 20 mm Hg with a constant rate of 100/min. Cardiac dimensions were qualitatively similar over the range of compression forces, and peak left ventricular, right ventricular, and aortic pressures increased with each increment in compression force. Aortic blood flow and stroke volume increased in every dog up to a peak intrapleural pressure of approximately 20 mm Hg. Beyond this level, stroke volume was not significantly

---

**FIGURE 3.** Dimension, flow, and pressure data during manual chest compression in the supine and lateral positions. See text for details.

Vol. 70, No. 1, July 1984
augmented and in fact decreased in some dogs (table 2).

Average data comparing peak left ventricular, intrapleural, and thoracic aortic pressures at each compression force are illustrated in figure 8. During manual compression, peak left ventricular and aortic pressures were consistently three to four times greater than peak intrapleural pressure at each level of compression force, and the change in vascular pressures was two to four times greater than the change in pleural pressure (table 1). These findings, in conjunction with the measurements of dimension, flow, and multiple aortic pressure, strongly suggest significant direct cardiac compression during manual CPR.

Typical digital data showing the effects of increasing rate of manual compression on hemodynamics are depicted in figure 9. As compression rate was increased from 60 to 100 to 150/min, force of compression was held constant at a peak intrapleural pressure of 20 mm Hg. Peak left ventricular, right ventricular, and aortic pressures remained relatively constant with increasing rate and seemed to be determined more by the force of compression. Moreover, stroke volume was relatively constant with each increment in rate, so that total output was augmented significantly. Cardiac dimensions at each compression rate were similar, and ventricular filling did not seem to be impaired.

Average hemodynamic data obtained in eight stud-

---

**TABLE 2**

<table>
<thead>
<tr>
<th>Compression force</th>
<th>Rate (compressions/ min)</th>
<th>PPLP (mm Hg)</th>
<th>PLVP (mm Hg)</th>
<th>PAP (mm Hg)</th>
<th>Stroke volume (ml)</th>
<th>Cardiac output (ml/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>133 ± 5</td>
<td>-2 ± 1</td>
<td>131 ± 5</td>
<td>119 ± 6</td>
<td>20.7 ± 1.8</td>
<td>2760 ± 269</td>
</tr>
<tr>
<td>Low</td>
<td>95 ± 5</td>
<td>12 ± 1</td>
<td>71 ± 8</td>
<td>68 ± 9</td>
<td>4.6 ± 0.6</td>
<td>438 ± 67</td>
</tr>
<tr>
<td>Middle</td>
<td>97 ± 6</td>
<td>20 ± 2b</td>
<td>92 ± 7b</td>
<td>79 ± 14b</td>
<td>6.2 ± 0.7b</td>
<td>599 ± 70b</td>
</tr>
<tr>
<td>High</td>
<td>97 ± 6</td>
<td>32 ± 2c</td>
<td>122 ± 10c</td>
<td>88 ± 14c</td>
<td>5.7 ± 0.6</td>
<td>557 ± 70</td>
</tr>
</tbody>
</table>

PPLP = peak pleural pressure; PLVP = peak left ventricular pressure; PAP = peak thoracic aortic pressure.

*Data represent mean ± SEM of observations in 10 dogs. Each observation was averaged from 10 sec of steady-state digital data. Data obtained just before cardiac arrest (control) are presented for comparison.

*Significant difference (p < .05) between low and middle force by two-tailed Student’s t test for paired data.

*Significant difference (p < .05) between middle and high force by two-tailed Student’s t test for paired data.
MECHANICAL COMPRESSION

FIGURE 5. Left ventricular diameter and multiple thoracic vascular pressures obtained with micromanometers during mechanical chest compression with animals in the supine position.

ies of compression rate are presented in figure 10 and table 1. At each rate, the force of compression (as indicated by peak intrapleural pressure) remained constant, stroke volume was unchanged, and left ventricular diameter decreased insignificantly. Mean and diastolic aortic pressure increased with each increment in compression rate, and cardiac output at 150/min was 2.3 times that observed at 60/min. In addition, coronary blood flow was well maintained at all rates, ranging from 64% to 75% of control (table 1).

Hemodynamic data obtained in the six barbiturate-anesthetized dogs differed significantly from those obtained in the morphine-anesthetized animals. Cardiac output at a compression rate of 100/min was 525 ± 43 ml/min, peak left ventricular pressure was 75 ± 7 mm Hg, peak aortic pressure was 72 ± 10 mm Hg, and mean diastolic aortic pressure was 30 ± 8 mm Hg. All values were significantly depressed as compared with those of the morphine experiments (table 1) (p < .05).

At autopsy, all transducers and flow probes were confirmed to be in the proper positions and no evidence of pulmonary or cardiac injury was noted. Upon opening of the abdomen, no hepatic, splenic, or other abdominal injury was found.

Representative femoral arterial pressure recorded with increasing manual compression rate in a patient undergoing CPR is illustrated in figure 11. An increase in systolic, diastolic, and mean femoral arterial pressure was noted as rate was increased from 60 to 100 to 150/min. Because the frequency response of the fluid-filled catheter/manometer system was less than optimal in the emergent clinical setting, it was difficult to make firm quantitative conclusions about the pressure waveforms. However, mean and diastolic arterial blood pressure improved with increasing compression rate in all patients, correlating well with the experimental data shown in figure 9.

**Discussion**

CPR is a well-established therapeutic technique allowing support of the circulation after cardiac arrest and preservation of organ viability until defibrillation can be performed. External cardiac massage can be applied for this purpose in virtually any situation, whether the arrest occurs in the hospital setting or in the community. In recent years, CPR has been formalized and taught to large segments of the population with the hope of preventing some portion of the 1000 prehospital sudden deaths occurring daily in the United States. Up to 40% of out-of-hospital arrest victims with documented ventricular fibrillation can be resuscitated by appropriately trained personnel,21-23 and it is estimated that 100,000 lives could be saved annually by providing effective CPR at the time of arrest. Furthermore, in-hospital CPR has become standardized, following specific guidelines established by the Subcommittee on Emergency Cardiac Care of the American Heart Association.24

One continuing problem with external cardiac massage is the relatively low cardiac output and coronary blood flow generated by current methods of compression. Absolute values of cardiac output observed in the dog vary considerably, and it is not known whether this variability is caused by differences in methodology or in compression techniques. Barsan and Levy,25 using thermodilution Swan-Ganz catheters, measured a cardiac output of 17% of control during manual external compression in dogs at 60/min. Weiser et al.26 used similar compression techniques and achieved 22% of the control cardiac output as measured by indocyanine green dye curves. Fitzgerald et al.27 used a
saline-conductivity method and observed a cardiac output of 42 ml/min/kg during mechanical compression, although control values were not given. Voorhees et al.,28 using mechanical compression, observed a cardiac output of 27% of control as determined by radioactive microspheres. It is not clear from the literature that measurements of cardiac output obtained with different methods in different laboratories or on dogs of different sizes can be validly compared. Moreover, carotid blood flow, as measured in several studies, may not be a reliable index of total cardiac output. Although some validation was performed in most studies of cardiac output, each method has theoretical problems during external chest compression and comparison with a direct measurement such as provided by an electromagnetic flow probe, would have been desirable. From each of these studies, however, it is clear that cardiac output is severely depressed during CPR.

Before technical modifications to improve cardiac output can be devised, a better basic understanding of external cardiac massage is required. Accordingly, several investigators over the past 5 years have attempted to elucidate the mechanisms of blood flow during CPR. As a result of this work, the most widely accepted theory at present is the thoracic pump mechanism. According to this hypothesis, the principal driving force for cardiac output during CPR is intrathoracic pressure or the change in intrathoracic pressure. During compression, the veins at the thoracic inlets are closed and a pressure gradient is generated from the thoracic vascular structures to the periphery. Experimental observations from a number of laboratories support this theory.9-12 In most studies, thoracic vascular and intracardiac pressures seemed to be similar to esophageal balloon or intrathoracic pressure measurements during external compression. Increasing intrathoracic pressure by lung inflation was associated with simultaneous augmentation of intracardiac pressures and arterial blood flow. In classic studies of patients in ventricular fibrillation,13 increasing intrathoracic pressure by repetitive coughing generated a significant cardiac output and arterial blood pressure. Finally, angiographic as well as echocardiographic studies suggested that all cardiac valves were open during external compression and that the heart acted as a passive conduit and not as a pump during CPR.14-15

Several potential problems with these studies need to be addressed. First, virtually all experimental data in recent years have been acquired in barbiturate-anesthetized animals. It has long been recognized that barbiturates produce significant peripheral vasodilatation and autonomic derangements so that, whenever possible,
conscious preparations are preferred in studies of cardiac physiology. Anesthesia obviously is required in animal experiments of cardiac arrest. When our group first approached this problem, it was our preconception that choice of anesthetic was not important and that chest compression was a mechanical phenomenon in an inanimate system; it was soon discovered that this was not the case. In point of fact, arterial and venous tone as well as possible reflex factors seemed to influence pressure generation and cardiac output during chest compression, a principle that has been demonstrated previously. When barbiturate anesthetics were used, arterial blood pressures and flows were uniformly lower than those obtained with morphine. Certainly, most would agree that narcotic anesthetics alter hemodynamics minimally, so that narcotics should be used whenever possible in cardiac physiology studies requiring anesthesia. Cardiac filling pres-
Pressures are uniformly lower with barbiturate anesthesia, possibly because of peripheral venous dilatation. This factor, along with the lower peripheral arterial resistance, seemed to account for the less satisfactory hemodynamics obtained during CPR with barbiturates. With lower diastolic arterial blood pressure, coronary blood flow would be considerably less than reported in table 1. These same phenomena also tended to occur with time after arrest, and cardiac output progressively deteriorated as vascular tone was lost. In the final experiments, morphine anesthesia was used and studies were completed within 5 min after arrest, providing better reproducibility and a more valid assessment of compression techniques. Without question, the results of previous animal experiments have been affected by barbiturate anesthesia, and future investigations should attempt to minimize such problems.

Another factor influencing recent studies is the use of mechanical compression devices. It seems logical to use such devices so that compression characteristics can be standardized. However, the physiology of mechanical compression is different from manual techniques (figure 6), and extrapolation of mechanical principals to manual CPR may not be entirely proper. Recent data from our laboratory indicate that intracardiac pressure generation is linearly related to compression momentum defined as the product of compression mass and velocity. This observation may be represented by impulse theory, where force transferred to the heart during chest compression is a direct function of the initial momentum of impact. By impulse theory, the force transferred between colliding bodies is proportional to the magnitude of the force integrated from time of initial impact to maximum impact. Since force equals mass times acceleration, the force transferred to the chest from initial impact to peak compression is proportional to the mass of the resuscitator's upper body times the deceleration as compression proceeds from initial impact to peak compression. Because the magnitude of the deceleration is determined...
by the velocity at initial impact as it approaches zero at peak compression, total force transferred to the chest equals mass times initial velocity, which is momentum of impact. Thus more force can be imparted to the chest during CPR by increasing either mass or velocity of compression. With manual external massage, the mass of compression, which is the resuscitator’s upper body, is considerably greater than that with mechanical techniques, and significant differences probably exist in compression velocity and momentum. Thus physiologic principles derived from studies with low-momentum mechanical compression may not be totally applicable to manual CPR, in which impulse characteristics are usually greater.

Most studies of CPR have measured intrathoracic pressure either with an esophageal balloon, a free catheter, or an unprotected micromanometer in the chest cavity or mediastinum. It is well understood that impact artifacts of manometers are proportional to kinetic energy components or to the product of mass and velocity squared. During normal respiration, the rate of change of intrathoracic pressure is small and the propagation velocity of pressure waves is minimal. During chest compression, however, the velocity of force vectors transmitted through the chest is significant, predisposing to impact artifacts. For these reasons, free intrathoracic catheters or esophageal balloons are not acceptable for investigation of CPR, and validation studies performed in our laboratory showed that peak intrathoracic pressure measured with balloons or free catheters was markedly exaggerated as compared with the technique described in this paper. The Millar manometer within the lumen of the silicone rubber pleural tube freely communicated with the potential space of the pleural cavity via the multiple side holes, while the wall of the tube protected the manometer from impact artifacts. With this method, only the scalar quantity of intrapleural pressure was measured, and velocity-dependent forces and motion artifacts were minimized.

Even under normal conditions, intrapleural pressure is difficult to measure. Because gravitational pressure gradients exist throughout the chest, esophageal or peripheral pleural pressure measurements may yield baseline pressure shifts. For studies of cardiac physiology, pleural pressure must be measured on the surface of the heart and at approximately the same vertical level as the intracavitary manometer. The accuracy of the present technique is illustrated by previous observations that subtracting pleural pressure from intracavitary left ventricular pressure virtually eliminated respiratory variation in the diastolic pressure-dimension relationship. In recent studies of positive pressure
FIGURE 11. Analog femoral arterial pressure data from a patient undergoing manual external chest compression early after cardiac arrest. The upper panel represents a rate of 60/min, the center panel a rate of 100/min, and the lower panel a rate of 150/min. See text for details.

ventilation, measuring pleural pressure on the surface of the heart and subtracting this pressure from diastolic intracavitary left ventricular pressure yielded changes in transmural cardiac filling pressure that were appropriate to alterations in end-diastolic ventricular volume, no matter what combination of internal and external pressures was examined. Thus, based on extensive published and unpublished validation, the technique for measuring pleural pressure reported in this article is probably the most accurate yet devised.

Carotid blood flow cannot be used to assess cardiac output, and most investigators who have measured carotid blood flow during CPR have stated this clearly. During chest compression, pressure gradients exist along the aorta, so that arterial pressures and flows may tend to be higher in the brachiocephalic vessels when techniques are used that produce a prolonged elevation of intrathoracic pressure. While carotid blood flow and pressure are increased with prolonged compression with or without simultaneous ventilation, flow to other organs may be impaired. Most important, it is now clear that myocardial blood flow is reduced to nearly zero by maneuvers that generate pressure in the left ventricle. This finding is probably another example of a vascular waterfall phenomenon and is similar qualitatively to that occurring during normal myocardial contraction. The reduction in coronary blood flow during external cardiac massage is even greater than during normal contraction, possibly because intramyocardial pressure integrated across the left ventricular wall is higher with chest compression. Therefore, it appears from our studies that prolonged compression techniques tend to limit diastolic perfusion time and coronary blood flow, and consistent with the results of previous studies, total coronary flow is depressed.

One might question the possible influence of thoracotomy and mediastinal scarring on the observations presented in this article. In all experiments reported, the studies were delayed 1 to 2 weeks after implantation to provide adequate seating of the flow probes.
separate validation experiments, however, four dogs underwent CPR studies 1 to 2 days after implantation. Pressure, dimension, and flow data were indistinguishable from those presented in this study, although the flow probes tended to unseat after a few minutes of compression, as evidenced by zero drift. In other studies, four dogs with intact pericardia were submitted to the CPR protocol, and again, results did not appear to be different from those in this article, indicating that an intact pericardium does not significantly influence the physiology of CPR. In more recent studies, Millar manometers protected by silicone rubber tubes measured regional pleural pressures in four different areas of the pleural space, as compression techniques were varied. Methods that optimized the thoracic pump mechanism, such as simultaneous compression and ventilation, were observed to have evenly distributed pleural pressures of similar magnitude to intracardiac pressures. However, with manual compression in the supine position, pleural pressures were generally low (as reported in this study) except in the retrosternal space. In a very localized area between the sternum and the cardiac surface, regional pleural pressure was found to be similar to intracardiac pressures. It is hypothesized that this regional high-pressure zone represents an area of direct transfer of force from the sternum to the heart, again suggesting a major component of direct cardiac compression. The low peak pleural pressure in other regions of the chest emphasizes differences in the physiology of various compression techniques.

Data from the present study suggest that several modifications should be considered in current concepts of systemic blood flow during CPR. Certainly, a thoracic pump mechanism exists, and observations from our laboratory with an inflatable pneumatic vest device indicate that intrathoracic pressure with simultaneous circumferential vest inflation and ventilation is equivalent to intracardiac pressure. Under the circumstances of pneumatic vest or cough CPR, the thoracic pump mechanism probably accounts for the majority of cardiac output. Experiments performed in our preparations indicate that prolonged mechanical compression with simultaneous ventilation and abdominal binding significantly improves cardiac output, reproducing the results of others and emphasizing the importance of a thoracic pump mechanism under these conditions. However, it seems that the relative contributions of the thoracic pump and direct compression varies with the physiologic situation. With high-momentum manual chest massage, direct cardiac compression appears to be considerable based on the findings of low pleural pressure relative to ventricular pressure and a flattening of ventricular shape at right angles to the force vector. The multiple aortic pressure data show possible propagation of a cardiac stroke volume along the aorta, which is more prominent with high-impact manual techniques. It is hypothesized that momentum of compression determines the relative contribution of direct cardiac compression to total cardiac output. During high-impact manual CPR, as studied in this article, direct cardiac compressive effects appear to predominate, whereas thoracic pump effects are more significant with low-momentum prolonged compression techniques.

When direct compression was optimized by using high-impact CPR, peak ventricular pressures increased directly with the force of compression. However, stroke volume increased only to moderate levels of compression and plateaued. This cannot be explained at present but may relate to obstruction of the ventricular outflow tract during more forceful compression. The observation of increasing peak pressure gradient from the left ventricle to the aorta (figure 8) would support this hypothesis. It was interesting to find that as long as compression force (as judged by peak intrathoracic pressure) was held constant, stroke volume remained constant despite marked variations in compression rate. Thus cardiac output could be augmented significantly by increasing compression rate when high-momentum compressions were used (table 1). These findings are somewhat contrary to those of previous publications, in which experiments were performed with low-momentum compression devices and prolonged compression times. However, a significant improvement in total cardiac output with increasing manual compression rate was observed consistently throughout the present study under all physiologic conditions.

Although cardiac output with any method of compression tended to decrease with time after arrest, total output still increased with rate of manual compression at any point in time and was always better with manual compression at 150/min than with any other technique. The increase in mean and diastolic aortic pressures with increasing cardiac output at higher rates was seen routinely early after arrest when peripheral vascular resistance was relatively normal. Arterial pressure increased less consistently with rate late after arrest, although cardiac output was still augmented. It seemed that directional changes in arterial blood pressure did not always correlate with changes in cardiac output, especially under conditions of profound vaso-dilatation. In our experience, arterial blood pressure
increases with higher manual compression rates in most patients, although again, increasing peripheral arterial pressure may or may not reflect improved cardiac output. Although the femoral arterial pressure data shown in figure 11 correlated well with observations in the dog, detailed investigation of this problem in human beings awaits better clinical methods of measuring cardiac output during cardiac arrest.

Because duration of compression could be minimized with the high-momentum manual technique, maximal diastolic intervals were available for coronary perfusion. This principle, together with the generally higher diastolic aortic pressures, resulted in coronary blood flow values of approximately 65% to 75% of control, which seemed to be independent of compression rate. As rate was increased, diastolic perfusion pressure and peak coronary flow velocity increased, but the diastolic time interval decreased, which may have contributed to the relatively constant total coronary flow. In addition, a phenomenon similar to reactive hyperemia was observed in several studies, suggesting that coronary vascular tone may have regulated coronary flow to some extent, according to the requirements of the fibrillating heart. In either case, coronary blood flow values of 65% to 75% of control are better than previously observed during CPR and represent one significant advantage of high-frequency, high-impulse manual compression. With high-impulse CPR an initial negative coronary flow velocity was observed commonly when left ventricular pressure exceeded aortic pressure. This negative flow, was small in all studies and, when taken into account by the computer integration, reduced total coronary flow minimally.

In summary, extensive investigation in intact animals instrumented for long-term studies suggests that significant improvements can be made in the techniques of manual external chest compression. To maximize cardiac stroke volume and coronary blood flow, manual massage should be performed with brief compressions of moderate force. Increasing the rate of compression significantly improves cardiac output, while coronary blood flow is well maintained. The favorable physiologic effects observed with high-impulse external cardiac massage in the dog seem to justify judicious human application, including more detailed hemodynamic studies in patients during cardiac arrest. In addition, previous experimental data and clinical experience suggest that open-chest cardiac massage when feasible should be considered whenever a brief application of closed-chest massage is unsuccessful. In the final analysis, methods of chest compression, including the choice between open-chest vs closed-chest techniques, may have to be individualized to each clinical situation by means of empirical measurements such as arterial blood pressure to optimize hemodynamic parameters.

We gratefully acknowledge Mr. James Bradsher and his staff for technical assistance and Mrs. Vicki Poe for aid in preparing the manuscript. We also thank Mr. Robert Margulies of the Duke Audiovisual Department for the illustrations.

References

23. Eisenberg MS, Gergner L, Hallstrom A: Cardiac resuscitation in the community: importance of rapid provision and implication for program planning. JAMA 241: 1905, 1979
24. McIntyre KM, Parker MR: Standards and guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). JAMA 244: 453, 1980
The physiology of external cardiac massage: high-impulse cardiopulmonary resuscitation.
G W Maier, G S Tyson, Jr, C O Olsen, K H Kernstein, J W Davis, E H Conn, D C Sabiston, Jr and J S Rankin

_Circulation_. 1984;70:86-101
doi: 10.1161/01.CIR.70.1.86

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1984 American Heart Association, Inc. All rights reserved.
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:
http://circ.ahajournals.org/content/70/1/86

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org/subscriptions/