Sequence of mitral valve motion and transmitral blood flow during manual cardiopulmonary resuscitation in dogs

MICHAEL P. FENELEY, M.D., F.R.A.C.P., GEORGE W. MAIER, M.D., J. WILLIAM GAYNOR, M.D., STANLEY A. GALL, B.S., JOSEPH A. KISSLO, M.D., JAMES W. DAVIS, M.S.E.E., and J. SCOTT RANKIN, M.D.

ABSTRACT  According to the thoracic pump model of cardiopulmonary resuscitation (CPR), the heart serves as a passive conduit for blood flow from the pulmonary to the systemic vasculature, necessitating an open mitral valve and anterograde transmitral blood flow during chest compression. To assess the applicability of this model to manual CPR techniques, two-dimensional echocardiograms were recorded from the right chest wall and/or the esophagus in nine dogs (18 to 26 kg) during manual CPR. The aortic valve opened with chest compression and closed with release, while the pulmonary and tricuspid valve leaflets closed with compression and opened during release. The mitral valve remained open during ventilation alone and during abdominal compressions. With the onset of brief, high-velocity (high-impulse) chest compressions, the mitral valve closed rapidly and the left ventricle was deformed, whether compressions were applied to the sternum or the left mid-chest wall. The mitral valve reopened with release of each compression. Left atrial echocardiographic contrast injections confirmed the absence of anterograde transmitral blood flow during high-impulse compression and its presence during release. Failure of mitral leaflet approximation during chest compression was observed only when a very low-velocity, prolonged (low-impulse) compression technique was used, or when regions that did not directly overlie the heart were compressed. Consistent with these observations, simultaneous recordings of the left ventricular and left atrial pressures during high-impulse sternal compressions in five dogs (19 to 25 kg) demonstrated peak and mean left ventricular systolic pressures of 38.5 ± 4.0 and 13.5 ± 2.9 mm Hg, respectively, and the pressure gradients declined with less impulsive compressions. The observations made during all but low-impulse chest compressions are inconsistent with the thoracic pump model, and support direct cardiac compression as the primary mechanism of forward blood flow with more impulsive manual chest compression techniques. Circulation 76, No. 2, 363–375, 1987.

THE MECHANISMS that sustain the circulation during cardiopulmonary resuscitation (CPR) continue to be debated more than 25 years after Kouwenhoven et al. proposed that blood flow results from direct cardiac compression. An alternative theory, the thoracic pump theory, attributes blood flow during CPR to a generalized increase in intrathoracic vascular pressures that is transmitted to the extrathoracic arteries. Compe-
diac compression mechanism with manual CPR, left ventricular pressure was found to exceed intrapleural pressure during compression, and cardiac output increased with increasing compression rate because the stroke volume remained relatively constant.\textsuperscript{12} In contrast, other investigators recently presented evidence that blood flow is determined by the duration rather than the rate of manual compression,\textsuperscript{6} a finding consistent with the thoracic pump model.

In the thoracic pump model, the heart is viewed as “a passive conduit for blood flow” during chest compression.\textsuperscript{5, 15, 17} It follows that two essential features of the thoracic pump model are an open mitral valve and anterograde transmitral blood flow during chest compression. Thus, analysis of the sequence of mitral valve motion and transmitral blood flow during CPR would provide a critical test of this model. The echocardiographic observation in two studies of human subjects undergoing manual CPR that the mitral valve was open during all or part of the compression phase is frequently cited in support of the thoracic pump model, although the observations made concerning both the mitral and aortic valves in these two studies were discordant in several respects.\textsuperscript{18, 19} In one of these studies, none of the four patients survived attempted resuscitation.\textsuperscript{19} In the other study, the survival benefit of the CPR administered could not be assessed because the study was conducted only after a decision had been made to terminate life support in the five patients in the study group.\textsuperscript{18} In both studies, echocardiograms were recorded from the parasternal position during sternal compressions, which were performed in a “conventional manner.”\textsuperscript{18, 19} Mitral valve dynamics have also been examined during mechanical compressions with a pneumatic-piston device in two different animal preparations with divergent results.\textsuperscript{4, 20} Importantly, however, closure of the mitral valve during compression was associated with successful resuscitation in one of these studies.\textsuperscript{20}

We postulated that the divergent findings concerning the mechanism of blood flow during manual CPR in previous studies might reflect differences in manual compression techniques. The purpose of this investigation was to attempt a resolution of this controversy by the echocardiographic observation of mitral valve dynamics with several different manual CPR techniques in dogs. The shape of the canine chest afforded echocardiographic “windows” that were further removed from the sternum, and thus more stable during sternal compression, than is the case in human subjects. In addition, two-dimensional echocardiograms were recorded from the esophagus, a position technically preferable to the mobile chest wall during CPR. It was also possible to assess transmural blood flow during manual CPR in dogs with a contrast echocardiographic technique that would not be appropriate in human studies. Finally, the left ventricular and left atrial pressures were measured simultaneously during manual CPR in a separate group of dogs to correlate the observations of mitral valve dynamics with the hemodynamic effects of different compression techniques.

Methods

Echocardiographic study. Nine mongrel dogs weighing 18 to 26 kg were studied by echocardiography during manual CPR. Five of these dogs had not undergone any previous surgical manipulation. In each of the four remaining dogs, left atrial and pleural catheters and pulse-transit left ventricular dimension transducers had been implanted surgically 1 to 2 weeks before the CPR study via a left lateral thoracotomy, as described fully elsewhere.\textsuperscript{12, 21} Both instrumented and noninstrumented dogs were studied because of an earlier suggestion that direct cardiac compression during manual CPR in instrumented dogs might be attributable to the restraints imposed on cardiac mobility by postoperative adhesions.\textsuperscript{5, 15} With the exception of the left atrial catheter, the implanted hardware was not used in this study.

General anesthesia was induced with thiopental sodium (15 mg/kg iv) and succinyl choline (1 to 2 mg/kg iv), and was maintained with morphine sulfate (2 mg/kg iv). After endotracheal intubation, the dogs were ventilated with room air (Bennett MA1 respirator or Air-Viva ambu-bag), and the anterior and right lateral regions of the thorax were shaved. Surface electrocardiographic leads were attached to both axillae and the xiphisternal region. A micromanometer-tipped catheter (Millar MPC-500) was passed into the ascending aorta via a femoral artery cut-down. Sodium heparin (5000 units) was administered intravenously. In six dogs, ventricular fibrillation was induced by a 60 Hz alternating current applied for 30 sec to two paddles placed across the anterior thorax. In the remaining three dogs, cardiac arrest was induced by bolus infusion of potassium chloride (30 meq). Manual CPR was then commenced.

In random order, two-dimensional echocardiograms were recorded in the absence of ventilation, with and without chest compression, during ventilation alone, during ventilation and chest compression, and during abdominal compression. Chest compression was performed by manually depressing either the sternum with the dog supine, or the left lateral chest wall with the dog in the right lateral position, in either order. The chest compression technique was varied, ranging from brief, high-velocity compressions\textsuperscript{12} to prolonged, low-velocity compressions.\textsuperscript{10, 22} Given the compression force, the compression velocity determines impact momentum and the time taken to reach peak compression determines impact deceleration.\textsuperscript{14} These factors determine the impulsiveness of the compression stroke. Thus, the compression techniques used in this study ranged from the high-impulse (brief, high-velocity) to the low-impulse end of the compression spectrum. The rate of chest compression was also varied, and was synchronized with a variable-rate beeper apparatus.\textsuperscript{12}

The echocardiograms were recorded with a Hewlett-Packard (model 77020A) ultrasonograph. Recordings were made from the right lateral chest wall with a 5 MHz short-focus phased-array transducer. The left ventricle was imaged in both the long- and short-axis planes. Although the primary objective was to obtain optimal images of the mitral valve, an attempt was made
to image all the cardiac valves during CPR. Aortic pressure was monitored continuously on a storage oscilloscope, and was displayed simultaneously with the echocardiographic images on the ultrasonograph screen during videotape (½ inch) recordings.

In four of the dogs, transesophageal two-dimensional echocardiograms were also recorded with a 5 MHz phased-array esophageal probe (Hewlett-Packard prototype) during sternal compression. This device permitted the recording of long-axis images of the heart from a relatively stable position during CPR. Due to anatomic constraints, it was not possible (as it is in human subjects) to image the heart in the short-axis plane or to obtain images of the aortic and pulmonary valves from the esophageal position.

Transmitral blood flow during CPR was assessed by contrast echocardiography. In the five noninstrumented dogs, agitated saline (3 to 8 ml) was injected into the left atrium during CPR via a transthoracic needle (20 gauge, length 16.5 cm) passed percutaneously from the left chest wall. Contrast injections also were made into the implanted left atrial catheters of two of the instrumented dogs. The contrast echocardiograms were recorded from both the esophageal and right chest wall transducer positions. Multiple contrast injections were made in each dog to ensure optimal delivery of the contrast bolus in relation to the compression cycle.

Three independent signals were used to establish the temporal relationship of the echocardiographic observations to the compression-relaxation sequence: (1) the displacement of the intrathoracic structures with the impact of each compression and their recoil with release; (2) the output of the synchronization beeper, which was recorded on the audio track of the videotape recordings, and (3) the simultaneously recorded aortic pressure signal. The videotape recordings were reviewed in real time, slow motion, and frame by frame.

**Left ventriculoatrial pressure gradient study.** In a separate group of five dogs that weighed 19 to 25 kg, the left atrial and left ventricular pressures were recorded simultaneously during manual CPR. Three of these dogs had undergone surgical instrumentation 1 to 2 weeks before the pressure gradient study, as described for the echocardiographic protocol. To control for the putative effects of surgical manipulation of the heart and postoperative adhesions, surgical instrumentation of the remaining two dogs was confined to the implantation of left atrial catheters through a limited left thoracotomy and a small pericardial incision directly over the left atrial appendage; data were acquired immediately after closure of the thoracotomy wound.

The dogs were anesthetized, intubated, and ventilated with room air, as described for the echocardiographic study. Two micromanometer-tipped catheters (Millar MPC-500, or PC-350) were passed into the left ventricle via the left atrial catheters. The micromanometers were prewarmed, balanced, and calibrated simultaneously against a water column immediately before insertion. After ensuring that the left ventricular pressure signals from the two micromanometers were superimposable, one micromanometer was withdrawn into the left atrium. In three dogs, ventricular fibrillation was induced by rapid right ventricular pacing with a transvenous pacemaker. In the remaining two dogs, cardiac arrest was induced by bolus infusion of potassium chloride (30 meq). Manual compression of the sternum was then commenced. The rate and technique of sternal compression were varied in the same manner as described for the echocardiographic study. Two of the dogs also underwent mechanical sternal compressions (duration 530 msec, rate 60/min) with a pneumatic-piston device (Thumper, Michigan Instruments), as previously described.

The left ventricular and left atrial pressures were displayed continuously on a storage oscilloscope throughout each study, and were digitized at 200 Hz (Model 1012, ADAC). The peak and mean pressure gradients between the left ventricle and the left atrium during each compression were computed with a microprocessor by digital subtraction at 200 Hz (Model PDP 11/23, DEC). The results are presented as the mean ± SEM.

**Results**

**Echocardiographic study.** The echocardiographic findings during CPR were very consistent in all of the dogs studied, regardless of whether there had or had not been previous surgical manipulation of the chest. The findings were not dependent on the mode of cardiac arrest.

**Mitral valve motion.** The mitral valve was the most easily visualized of the four cardiac valves during CPR. High-quality images of both mitral leaflets were obtained in all of the dogs studied. Although both long- and short-axis views of the mitral valve were obtained from the right chest wall position, translational cardiac movements frequently produced alterations of the short-axis scanning plane during chest compressions, particularly when applied to the sternum. The long-axis scanning plane was more consistent.

In the absence of both ventilation and compression, the mitral valve was always open. In the fibrillating hearts, rapid small oscillatory movements of the mitral leaflets were consistently observed. With ventilation alone, or with abdominal compression, the mitral valve remained open and the leaflets separated slightly further.

With the onset of high-impulse manual compressions of either the sternum or the midsegment of the left lateral chest wall, the mitral valve closed rapidly, and reopened with release of the compressions (figure 1). These observations were made both in the presence and absence of ventilation. Mitral valve closure coincided with the rise of the simultaneously recorded aortic pressure. When the technique and rate of compression were varied independently in an attempt to characterize the determinants of mitral valvular dynamics during CPR, mitral valve closure was observed with compressions that generated peak aortic pressures ranging from 20 to 200 mm Hg at rates ranging from 60 to 120/min. The extent and rapidity of maximal leaflet separation with release increased with increasing compression rate. Even brief chest thumps with a single hand resulted in rapid "dipping" movements of the mitral leaflets toward each other (figure 2), although insufficient force could be generated in this way to completely coapt the leaflets.

Failure of mitral leaflet approximation during the
compression phase was observed under only two conditions of manual chest compression: (1) during very low-velocity, prolonged compressions, which necessitated compression rates of 40 to 60/min or less — the low-impact end of the compression spectrum (figure 3, top), or (2) when compressions, however brief or forceful, were applied to regions of the chest wall that did not directly overlie the heart (figure 3, bottom).

Transmitral blood flow. Left atrial contrast injections demonstrated that there was no anterograde transmural blood flow during the compression phase when high-impact compressions were applied to the sternum or the left mid chest wall, consistent with the demonstrated coaptation of the mitral valve leaflets. On the contrary, the left atrial contrast bolus moved retrogradely (toward or into the pulmonary veins) during the compression phase, followed by rapid anterograde transit into the left ventricle during the release phase (figure 4). Moreover, once the contrast had entered the left ventricle, the dominant trajectory of the contrast during the subsequent compression strokes was toward the closed mitral valve and the left ventricular outflow tract. During ventilation alone, slight anterograde motion of the contrast across the open mitral and aortic valves was observed, followed by approximately equal retrograde motion with the cessation of the ventilation cycle.

Left ventricular deformation. Deformation of the left ventricle was consistently observed when compressions were applied to the sternum or the left mid chest wall. Particularly notable was the fact that the site of left ventricular deformation corresponded with the direction of the compression force (figure 1). With compressions of the left mid chest wall, the lateral wall of the left ventricle was displaced inward, while the long-axis dimension increased slightly. With sternal compressions, the apicoseptal segment of the left ventricle was deformed inward, while the “anteroposterior” minor-axis dimension (the dimension in the plane joining the anterior and posterior descending coronary arteries) increased. These observations are consistent with the surface anatomy of the canine heart, the apex of which lies closer to the sternum than does the apex of the human heart, and are similar to observations made previously in dogs with implanted ultrasonic dimension transducers.12

It is also notable that cardiac compression was not associated with entrapment of the heart between oppo-
times observed during the release phase with compression rates of 60/min or less. Adequate images of the pulmonary valve leaflets throughout all phases of the chest compression-release cycle were obtained in only two dogs, and only when these dogs were placed in the right lateral position (i.e., only during left lateral chest compressions). In both cases, the pulmonary valve closed during lateral chest compression and reopened with release.

**Left ventriculoatrial pressure gradient study.** Representative illustrations of the left ventricular and left atrial pressures, and the difference between these pressures, during sinus rhythm and under a variety of CPR conditions are shown in figure 6. The original digitized pressure waveforms were traced in ink to permit pho-

---

**FIGURE 2.** Long-axis views of the left ventricle and mitral valve leaflets (arrowheads) before (top) and during (bottom) a brief thump of the sternum with a single hand. The chest thump results in incomplete apposition of the mitral leaflets. Abbreviations as in figure 1.

---

**FIGURE 3.** Long-axis views of the mitral valve (arrowheads) during a very low-velocity, prolonged sternal compression (top), and during a high-impulse compression of the inferior segment of the left lateral chest wall, immediately above the costal margin (bottom). In both cases, the mitral valve remains open during the compression. Abbreviations as in figure 1.
**FIGURE 4.** Release-compression-release sequence of a contrast echocardiogram recorded during high-impulse chest compression. *Left,* Left atrial contrast injection during the release phase. The leading edge of the contrast bolus has just entered the left ventricle (LV). A portion of the open anterior mitral leaflet is visible (white arrowhead). *Middle,* Compression phase. The mitral valve is closed (white arrowheads), completely engulfing the contrast within the left atrium. *Right,* Release phase. Contrast fills the left ventricle. The black arrow in each frame indicates the trajectory of the contrast during each phase as observed in real time.

Toreduction to the size shown. The first panel of this figure shows the superimposed left ventricular pressures measured by the two micromanometers during sinus rhythm, followed by withdrawal of one micromanometer into the left atrium. Although the left atrial pressure increased during manual compression, high-impulse compressions, performed at rates ranging from 60 to 140/min, always produced a higher pressure in the left ventricle than in the left atrium (figure 6, *second panel*) regardless of the mode of cardiac arrest.

**FIGURE 5.** Paired views of the tricuspid (*left*), aortic (*middle*), and pulmonary (*right*) valves (arrowheads) during release and chest compression (see text). All views were recorded from the right chest wall: the tricuspid valve is shown in a modified four-chamber view; the aortic valve is shown in a slightly oblique, left ventricular, long-axis view; the pulmonary valve is shown in the short-axis plane of the proximal aorta at a level just superior to the aortic valve. Ao = aorta; PA = pulmonary artery; RA = right atrium; other abbreviations as in figure 1.
FIGURE 6. In each panel of this figure, the upper section (LV and LA) shows the left ventricular pressure (solid lines) and the left atrial pressure (dots) displayed on the same scale, and the lower section (LV-LA) shows the difference between these pressures, which was determined by digital subtraction at 5 msec intervals. The original digitized pressure waveforms were traced in ink to permit photoreduction to the size shown. First panel; Simultaneous left ventricular pressure recordings from two micromanometers and withdrawal of one micromanometer into the left atrium during sinus rhythm demonstrating the left ventriculoatrial pressure gradient before cardiac arrest. Second panel; High-impulse compressions at 60/min and 140/min. Segments of the left ventricular and left atrial digital pressure data points from the early phase of two of these high-impulse compressions are shown on a larger scale in figure 7. Third panel; Very low-impulse compressions at 30/min. Note beat-to-beat variation in the left ventriculoatrial pressure gradient. Fourth panel; Mechanical compressions with a pneumatic-piston device at 60/min.

FIGURE 7. Segments of the left ventricular (LV) and left atrial (LA) digital pressure data points at 5 msec intervals from the early phase of two of the high-impulse compressions that were illustrated on a smaller scale in figure 6. Both panels demonstrate the early onset of the left ventriculoatrial pressure gradient during compression, and illustrate the maximal observed variation in the pattern of the gradient during early compression.

The average peak pressure gradient from the left ventricle to the left atrium during high-impulse compressions was 38.5 ± 4.0 mm Hg, and the average of the mean left ventriculoatrial pressure gradients was 13.5 ± 2.9 mm Hg. Postoperative adhesions did not appear to favor the generation of a higher ventriculoatrial pressure gradient; the highest mean gradients were observed in one each of the studies performed immediately and 1 to 2 weeks after surgery. With high-impulse compressions, the average peak left ventricular pressure was 129 ± 10 mm Hg, and the average time from the onset of left ventricular pressure elevation to its return to the precompression level (the compression pressure duration) was 255 ± 17 msec.

Figure 7 shows the left ventricular and left atrial digital pressure data points at 5 msec intervals during the early phase of two of the high-impulse compressions from figure 6 on a larger scale, and demonstrates the early onset of the left ventriculoatrial pressure gradient during compression, which is difficult to appreciate on the smaller scale of the traced waveforms. The
two gradient patterns shown illustrate the maximal variation in the observed high-impulse compression gradients. The early gradient pattern shown in the left panel is typical of that observed in most studies. The pattern shown in the right panel was observed at 140/min in one of the dogs. The average left ventricular pressure at the onset of the pressure gradient between the left ventricle and the left atrium (pressure "crossover") during high-impulse compressions was 21.2 ± 3.4 mm Hg.

When the compression duration was prolonged and the compression rate was reduced, the pressure gradient from the left ventricle to the left atrium during compression declined. At a compression rate of 30 to 40/min with a mean compression pressure duration of 751 ± 86 msec and a peak left ventricular pressure of 75 ± 7 mm Hg, the average peak and mean left ventriculoatrial pressure gradients during compression were 13.0 ± 4.0 and 5.1 ± 2.0 mm Hg, respectively. With this very low-impulse compression technique, however, considerable beat-to-beat variability was observed (figure 6, third panel), and the left ventriculoatrial pressure gradient intermittently approached zero during some compressions. Only during pneumatic-piston compressions, which generated peak left ventricular pressures of only 30 to 40 mm Hg, was the mean left ventriculoatrial pressure gradient consistently negligible (figure 6, fourth panel).

Discussion

An accurate understanding of the mechanisms by which forward blood flow is generated during manual CPR is of great importance in optimizing clinical CPR techniques, and thus survival. If cardiac compression were the primary mechanism, then increasing the compression rate would significantly augment cardiac output. According to the thoracic pump model, however, the cardiac output should be determined by the amount of time the thorax is compressed, rather than the compression rate. These divergent theories have quite different implications concerning the optimal technique for manual CPR in human subjects.

There is no doubt that a thoracic pump mechanism exists. In particular, the thoracic pump model appears to account accurately for blood flow during cough CPR, and probably also during vest CPR. It cannot be assumed, however, that the mechanism of blood flow is the same under all conditions of thoracic compression. The mechanical impact on the thorax with manual compression is different from that achieved by coughing, or by inflating a vest that encompasses the entire thorax. Although anterograde transmirtal blood flow has been demonstrated by angiography during the latter phase of pneumatic-piston chest compressions in dogs, mitral valve closure was documented recently by echocardiography during pneumatic chest compressions in minipigs. In addition, previous evidence indicates that the hemodynamic effects of pneumatic chest compressions differ significantly from those of manual compressions, as this study confirmed (figure 6), and that the effects of manual compressions vary with compression technique. The issue addressed in this study was not whether a thoracic pump mechanism exists, but whether the thoracic pump model provides an accurate account of the mechanism by which blood flow is generated under all conditions of manual chest compression.

In the demonstrated absence in this study of an open mitral valve and anterograde transmirtal blood flow during high-impulse manual chest compressions, the stroke volume expelled into the systemic vasculature during the compression phase can have been derived only from the left ventricle, which was deformed by chest compression. Thus, our findings concerning mitral valve motion during high-impulse CPR are the antithesis of those predicted by the thoracic pump model, wherein the heart is viewed as "a passive conduit" for blood flow from the lungs to the systemic circulation through the open mitral valve during chest compression. Moreover, so long as chest compressions were applied to regions that overlie the heart, the mitral valve remained open only during very prolonged, low-velocity manual compressions performed at a slow rate. The same echocardiographic observations were made regardless of whether there had or had not been any previous surgical manipulation of the chest, and regardless of the mode of cardiac arrest.

The left ventriculoatrial pressure gradient data were consistent with these echocardiographic observations. A considerable pressure gradient from the left ventricle to the left atrium was observed during high-impulse compressions, and indeed with all but some very low-impulse compressions (figure 6). Since mitral valve closure is a threshold phenomenon that normally is coincident with the crossover point between the left ventricular pressure and the left atrial pressure at the onset of spontaneous ventricular contraction (figure 6, first panel), it is highly probable that closure of the normal mitral valve would accompany the generation of any pressure gradient from the left ventricle to the left atrium. In this study, pressure crossover occurred at a left ventricular pressure of 21.2 ± 3.4 mm Hg during high-impulse compressions, which is lower.
than aortic diastolic pressures reported previously during manual CPR in dogs. The observation suggests that the mitral valve closed before left ventricular pressure was sufficient to open the aortic valve, consistent with the echocardiographic observation that the mitral valve was closed when the aortic valve was open. During the echocardiographic study, it was possible to modulate the compression technique until, at the low-impulse end of the compression spectrum, the threshold for mitral valve closure was no longer exceeded. The observations made during pneumatic compressions in dogs support previous evidence that the hemodynamic characteristics of such mechanical compressions are sufficiently different from those of manual compressions to raise doubts concerning the applicability of mechanical compression data to recommendations for manual CPR technique.

The present hemodynamic data are also consistent with the previous demonstration of nonequivalent elevations of left ventricular, right ventricular, and pleural pressures during high-impulse CPR, in that both studies indicate that the increase in intrathoracic pressures during CPR is not necessarily uniform with all compression techniques. In that previous study, the increase in left ventricular pressure during manual compression was four to five times greater than the increase in pleural pressure, suggesting direct cardiac compression, and right ventricular pressure also greatly exceed pleural pressure. Similarly, since the increase in left atrial pressure during compression in the present study was of a similar order of magnitude to the increase in left ventricular pressure (figure 6), it cannot be attributed solely to a global increase in intrathoracic pressure. Moreover, the echocardiographic observation that left atrial contrast was displaced toward or into the pulmonary veins during compression indicates that left atrial pressure exceeded intrapulmonary pressure at that time. Cardiac compression would be expected to influence the pressures in all cardiac chambers, but inequality of the chamber pressures is also to be expected since (1) all chambers are not equally exposed to the compressive force, a factor determined by compression site, and (2) the impedance opposing venting of the pressure increase is not the same for all chambers. The left atrium, for example, can vent to the pulmonary veins, but the left ventricular pressure must exceed aortic diastolic pressure before the aortic valve opens.

It should be noted that although equivalent left atrial and left ventricular pressures have been documented during cough CPR, as would be expected, we are not aware of any previous studies in which the left atrial and left ventricular pressures have been measured simultaneously during manual CPR. Even if the importance of compression technique in determining the results obtained in previous studies were ignored, the assumption that the pressures in all cardiac chambers are equally elevated during manual compressions has been based on the observation of approximately equal right atrial or venous pressures and aortic or peripheral arterial pressures. In fact, in one of these studies, the peak aortic pressure exceeded the peak right atrial pressure by 27 mm Hg in both patients in whom these pressures were measured during manual CPR. In one previous study, the left atrial and femoral arterial pressures were measured simultaneously in eight patients during manual CPR, but all eight patients had mitral valve disease with mitral regurgitation and/or stenosis. No conclusions of general validity concerning the left ventriculoatrial pressure gradient during CPR can be drawn from such a study. Given the presence of mitral valve disease in all of the patients studied, it is indeed remarkable that the mean femoral arterial pressure during compression exceeded the mean left atrial pressure by 10 to 13 mm Hg in three of the patients.

The observations made in this study concerning the movements of the tricuspid and pulmonary valves during chest compression are consistent with those reported in human studies and suggest that blood flows from the systemic veins into the lungs through the right heart during the release phase of the CPR cycle. It is notable that the observation of tricuspid valve closure, in human subjects and in dogs, is also inconsistent with the assumed equality of all intracardiac pressures during manual compression. The pulmonary valve was observed in only two of the animals in this study, however, and only during left lateral chest compressions. It was not determined, therefore, whether the pulmonary valve was closed during sternal compressions. Caution is warranted in extrapolating our findings during lateral chest wall compressions to the effects of sternal compressions on pulmonary valve motion, since the relationship of the right ventricle to the compression vector is clearly different in the latter situation. Although Deshmukh et al. did not observe pulmonary valve motion in their minipig study, they did note flow of echocardiographic contrast toward the right ventricular outflow tract during mechanical sternal compressions that were associated with mitral valve closure.

Werner et al. observed that the mitral valve remained widely open throughout manual compression of the sternum in five human subjects, a finding at

Vol. 76, No. 2, August 1987
variance with our observations in dogs during all but low-impulse compressions. If this discrepancy were due to differences between human and canine anatomy or size, then most of the experimental evidence concerning CPR, derived mainly from canine experimental preparations, would be irrelevant to the understanding of CPR mechanisms in human subjects. Like Werner et al., Rich et al. observed that the aortic and mitral valves were open simultaneously during early manual compression in four human subjects, and concluded similarly that flow was attributable to the thoracic pump mechanism. Unlike Werner et al., however, Rich et al. observed that both the aortic and mitral valve leaflets returned to a “less open” position during midcompression. Although this observation seems compatible with diminishing flow in midcompression, the mitral valve leaflets coapted in one case, and in another case they assumed a more approximated position than that observed during relaxation. Since, like Werner et al., we observed that in the absence of CPR (no-flow state) the mitral leaflets of the arrested heart were always widely separated, it is difficult to attribute the midcompression position of the mitral leaflets in two cases studied by Rich et al. solely to a reduction in flow. Moreover, contrary to the finding of both the present study and the study by Werner et al., Rich et al. reported that both the mitral and aortic valves opened widely again during relaxation and concluded that blood flowed through the left heart not only during compression but also during relaxation. Thus, when the findings of the present study are compared with the findings of these two previous human studies, none of the studies are fully concordant. Since the discordant findings of the two human studies cannot be attributed to species differences and must be attributed to technical differences, it is possible that technical differences are also more important than species differences in explaining the discrepancies between these two studies and the present work.

The relatively narrower anteroposterior diameter of the human thorax might appear, if anything, to favor greater cardiac compression in human subjects than in dogs during sternal compression. Indeed, one of the original arguments advanced in support of the thoracic pump model was that “external CPR is effective in dogs despite large antero-posterior thoracic dimensions which obviate cardiac compression.” It is notable, therefore, that in neither of the previous human studies was any prominent deformation of the left ventricle observed during chest compression. In contrast, left ventricular deformation was a very prominent feature of high-impulse chest compressions in dogs in the present study. Furthermore, the left ventricular dimensions along the path of the force vector were shortened, while those perpendicular to the force vector were elongated, as previously demonstrated in dogs with implanted ultrasonic dimension transducers. These observations are consistent with a direct compressive effect.

While deformation of the left ventricle demonstrates that it is subjected to forces that cannot be explained by a uniform increase in the pressures in all intrathoracic structures during compression, deformation is not necessarily synonymous with volume reduction. Ventricular deformation, per se, invalidates the geometric assumptions inherent in two-dimensional volume algorithms. The inference that the stroke volume resulted from a reduction in left ventricular volume was based on the simultaneous observation that the mitral valve was closed when the aortic valve was open. Similarly, Deshmukh et al. recently demonstrated prominent left ventricular deformation and mitral valve closure during pneumatic-piston sternal compressions in minipigs weighing 20 to 30 kg. Presumably, it is possible to generate a left ventriculoatrial pressure gradient in minipigs, if not in dogs (figure 6), with that device.

The theoretical point has been made by others that any reduction in left ventricular volume during manual CPR could be due to mitral regurgitation. If mitral regurgitation occurred during manual compression, it would be an additional factor contributing to the elevation of left atrial pressure (figure 6). The absence of a “negative contrast” effect in the left atrium during our contrast studies of the manual compression phase (figure 4, middle), however, is evidence against significant mitral regurgitation. Similarly, Deshmukh et al. noted only minimal mitral regurgitation during the compression phase when saline was injected into the left ventricle. It should be emphasized, however, that anterograde transmitral blood flow during compression is an essential requirement of the thoracic pump model. It is not the cardiac compression model that would be refuted by the presence of retrograde transmitral flow during manual compression, therefore, but rather the thoracic pump model. If blood were flowing from the left ventricle into the left atrium during compression, then the simultaneous flow of blood through the open aortic valve could not possibly be attributed to the thoracic pump concept of anterograde transmitral blood flow.

The echocardiographic observations of Werner et al. were made during manual CPR in five patients after a decision had already been made to terminate life
support. None of the four patients studied by Rich et al.\textsuperscript{19} during manual CPR were successfully resuscitated. Deshmukh et al. suggested that the observations made in these studies might be attributable to delays between the onset of cardiac arrest and the echocardiographic recordings in nonsurviving patients.\textsuperscript{20} They noted that the closing motion of the mitral valve during pneumatic sternal compressions diminished 5 min after cardiac arrest in nonsurviving minipigs, but that mitral valve closure continued to occur during compression until successful defibrillation after 12 min in three surviving animals. Because the aim of our study was to observe the effects of several different CPR techniques in the same animals, survival was not an end point. Nevertheless, mitral valve closure was observed with high-impulse compressions throughout studies that were continued for a minimum period of 30 min.

Previous evidence from this laboratory has indicated that intracardiac pressure generation is linearly related to compression momentum,\textsuperscript{16} and that, as may be predicted by impulse theory, high-impulse compressions produce more optimal hemodynamic effects than do more prolonged mechanical compressions.\textsuperscript{12, 13} The rationale for the high-impulse CPR technique is that the extent of cardiac compression is determined not only by the force but also by the velocity of chest compression and thus compression momentum. The effect of brief chest thumps illustrated this principle (figure 2), despite the limited force that could be generated in this way.

Moreover, the fact that the heart can be compressed in dogs by lateral chest compression and by sternal compression, despite the relatively large canine anteroposterior thoracic dimension, is not compatible with the view that cardiac compression results from entrapment of the heart between the sternum and the vertebral column, as was postulated originally.\textsuperscript{1} Certainly, there was no echocardiographic evidence for cardiac entrapment in this study, since maximal inward deformation occurred ipsilateral to the compression site, without comparable deformation of the contralateral cardiac surface. This pattern of cardiac deformation strongly suggests that cardiac compression occurs because of the heart’s inertia to displacement from its resting position within the thorax. Consistent with the physics of collision with an inert but deformable body, a brief high-momentum impact would be expected to favor cardiac compression rather than dissipation of the compression momentum by cardiac translocation. This observation is important because several theoretical arguments against the cardiac compression mechanism of CPR have been based on the erroneous notion of compression by entrapment.\textsuperscript{6, 8, 15}

Brief, high-velocity (high-impulse) compressions are more easily performed at faster compression rates. In the context of impulse theory, a brief compression is one of short absolute duration, while the percentage duration (duty cycle) is also a function of compression rate. For example, a duty cycle of 50% would not be considered short, but the duty cycle for high-impulse compressions of only 250 msec duration performed at 120/min is 50%. Of more practical importance is the fact that the stroke volume remains relatively constant with increasing high-impulse compression rates, so that the cardiac output increases when the compression rate is increased.\textsuperscript{12} For this reason, the term “high-impulse CPR” was coined originally to describe a high-rate manual CPR technique.\textsuperscript{12}

In the reports by Werner, Rich, and their associates,\textsuperscript{18, 19} the authors stated that CPR was performed in a “conventional manner.” The emphasis of the recommended CPR technique at that time was on prolonged compression duration at a slow rate (50% to 60% duty cycle at 40 to 60 compressions/min\textsuperscript{10, 22}), the antithesis of the high-impulse CPR technique.\textsuperscript{12-14} Indeed, Werner emphasized that increased mitral leaflet separation was observed during “more prolonged chest compression.”\textsuperscript{18} Since similar observations were made in the present study when a low-velocity, prolonged chest compression technique was used, the discrepancies between the two previous human studies and the present study may also be attributable to differences in manual compression technique.

The American Heart Association recently increased the recommended manual compression rate for clinical CPR from 40 to 60/min to 80 to 100/min.\textsuperscript{11} This decision was recommended by advocates of both the cardiac compression model and the thoracic pump model of manual CPR.\textsuperscript{27} Advocates of the cardiac compression model supported the decision because of experimental evidence that the stroke volume remains relatively constant when compression rate is increased, resulting in increased cardiac output with faster compression rates.\textsuperscript{12, 27} Advocates of the thoracic pump model supported the decision because they believed a faster compression rate would lead to a greater percentage of compression time,\textsuperscript{27} which they hold to be the primary determinant of flow.

Leading proponents of the thoracic pump model have argued that the improved cardiac output and other hemodynamic variables noted with increased manual compression rates in dogs by Maier et al\textsuperscript{12} may have
been attributable to a covariant increase in percentage compression duration with increasing rates.2 This line of argument would be relevant, however, only if blood flow with the high-impulse manual compression technique used by Maier et al. were mediated by the thoracic pump mechanism. The present echocardiographic findings demonstrate that this is not the case. Moreover, in a recently completed collaborative study,28 12 of 13 dogs were successfully defibrillated after 30 min of high-impulse manual CPR at 120 compressions/min, while only two of 13 dogs were successfully defibrillated after 30 min of manual CPR at 60 compressions/min, despite the fact that the percentages of compression time in the two groups were the same. Twenty-four hour survival was also greater with high-rate CPR (8/13) than with low-rate CPR (2/13), despite the fact that neither cardioactive drugs nor advanced life-support techniques were used. The survival data for high-impulse CPR were also superior to those achieved with manual CPR at 60/min by other investigators who used epinephrine during CPR and advanced cardiac life-support techniques, including antiarrhythmic therapy.29 These findings, which would provide experimental support for the decision to increase the recommended compression rate for clinical CPR regardless of the mechanism of the survival benefit, also exclude covariance of compression duration with increased rate as the necessary mechanism of that benefit.

In summary, this study demonstrates that high-impulse manual chest compressions produce left ventricular deformation, mitral valve closure, and no anterograde transmitial blood flow during the period when the aortic valve is open. These findings are inconsistent with the thoracic pump model of CPR, and support direct cardiac compression as the primary mechanism of blood flow with the high-impulse manual CPR technique. Moreover, both the echocardiographic and the left ventriculotrial pressure gradient data support the conclusion that, in dogs, the mitral valve closes during all but very low-impulse compressions performed at a slow rate. These findings provide direct evidence of the mechanistic basis for the hemodynamic and survival benefits of faster manual compression rates in a canine preparation of CPR. Reservations must always be held concerning the relevance of findings in animal preparations to human subjects. Similar studies of the mechanisms and effectiveness of different manual compression techniques in human subjects may be warranted, therefore, despite the difficult ethical considerations, to optimize clinical CPR techniques and thus survival.

References

9. Eisenberg MS, Gergner L, Hallstrom A: Cardiac resuscitation in the community: importance of rapid provision and implication for program planning. JAMA 241: 1905, 1979
10. Standards and guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). JAMA 244: 198, 1980
11. Standards and guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). JAMA 255: 2901, 1986
Sequence of mitral valve motion and transmirtal blood flow during manual cardiopulmonary resuscitation in dogs.
M P Feneley, G W Maier, J W Gaynor, S A Gall, J A Kisslo, J W Davis and J S Rankin

Circulation. 1987;76:363-375
doi: 10.1161/01.CIR.76.2.363

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1987 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/76/2/363