A PRELIMINARY STUDY OF CARDIOPULMONARY RESUSCITATION BY CIRCUMFERENTIAL COMPRESSION OF THE CHEST WITH USE OF A PNEUMATIC VEST

HENRY R. HALPERIN, M.D., JOSHUA E. TSITLIK, PH.D., MARK GELFAND, M.S., MYRON L. WEISFELDT, M.D., KREG G. GRUBEN, PH.D., HOWARD R. LEVIN, M.D., BARRY K. RAYBURN, M.D., NISHA C. CHANDRA, M.D., CAROL JACK SCOTT, M.D., BILLIE JO KREPS, R.N., B.S.N., CYNTHIA O. SIU, PH.D., AND ALAN D. GUERCI, M.D.

Abstract *Background.* More than 300,000 people die each year of cardiac arrest. Studies have shown that raising vascular pressures during cardiopulmonary resuscitation (CPR) can improve survival and that vascular pressures can be raised by increasing intrathoracic pressure.

Methods. To produce periodic increases in intrathoracic pressure, we developed a pneumatically cycled circumferential thoracic vest system and compared the results of the use of this system in CPR (vest CPR) with those of manual CPR. In phase 1 of the study, aortic and right-atrial pressures were measured during both vest CPR (60 inflations per minute) and manual CPR in 15 patients in whom a mean (±SD) of 42±16 minutes of initial manual CPR had been unsuccessful. Vest CPR was also carried out on 14 other patients in whom pressure measurements were not made. In phase 2 of the study, short-term survival was assessed in 34 additional patients randomly assigned to undergo vest CPR (17 patients) or continued manual CPR (17 patients) after initial manual CPR (duration, 11±4 minutes) had been unsuccessful.

Results. In phase 1 of the study, vest CPR increased the peak aortic pressure from 78±26 mm Hg to 138±28 mm Hg ($P<0.001$) and the coronary perfusion pressure from 15±8 mm Hg to 23±11 mm Hg ($P<0.003$). Despite prolonged unsuccessful manual CPR, spontaneous circulation returned with vest CPR in 4 of the 29 patients. In phase 2 of the study, spontaneous circulation returned in 8 of the 17 patients who underwent vest CPR as compared with only 3 of the 17 patients who received continued manual CPR ($P = 0.14$). More patients in the vest-CPR group than in the manual-CPR group were alive 6 hours after attempted resuscitation (6 of 17 vs. 1 of 17) and 24 hours after attempted resuscitation (3 of 17 vs. 1 of 17), but none survived to leave the hospital.

Conclusions. In this preliminary study, vest CPR, despite its late application, successfully increased aortic pressure and coronary perfusion pressure, and there was an insignificant trend toward a greater likelihood of the return of spontaneous circulation with vest CPR than with continued manual CPR. The effect of vest CPR on survival, however, is currently unknown and will require further study. (N Engl J Med 1993;329:762-8.)

THERE are more than 300,000 victims of cardiac arrest each year, and attempts to resuscitate them are usually unsuccessful. Both laboratory studies[1-3] and clinical studies[4] have shown that the restoration of cardiac function after cardiac arrest is related to the vascular pressures generated during resuscitation, especially the coronary perfusion pressure. In addition, over the past decade, a number of studies[3,5-8] have provided evidence that the cyclic increase in intrathoracic pressure produced by chest compression is an important mechanism for the generation of vascular pressure and flow during cardiopulmonary resuscitation (CPR).

On the basis of the assumption that induced increases in intrathoracic pressure can produce blood flow during cardiac arrest, we have been studying circumferential compression of the thorax by means of inflation of a pneumatic vest as a more effective and safer means of resuscitation than manual CPR.[3] The vest is analogous to a large blood-pressure cuff that compresses the thorax as it is inflated. Whereas manual CPR changes the thoracic volume by producing a large displacement at one point (the sternum), use of the vest in CPR (vest CPR) should more efficiently and safely decrease thoracic volume and raise intrathoracic pressure by small, circumferential changes in the dimensions of the thorax (Fig. 1). In addition, severe chest-wall trauma and abdominal and thoracic visceral injury occur frequently during manual CPR,[9-11] and the avoidance of large sternal excursions by the use of vest CPR may reduce trauma. In this study, we evaluated the effect of vest CPR on vascular pressures and the return of spontaneous circulation in patients who had not been successfully resuscitated with manual CPR.

From the Peter Belfer Cardiac Mechanics Laboratory, Cardiology Division, Department of Medicine (H.R.H., J.E.T., M.G., M.L.W., H.R.L., B.K.R., N.C.C., B.J.K., C.O.S., A.D.G.), the Department of Biomedical Engineering (H.R.H., J.E.T., K.G.G.), and the Department of Emergency Medicine (C.J.S.), Johns Hopkins Medical Institutions, Baltimore. Address reprint requests to Dr. Halperin at the Cardiology Division, Johns Hopkins Hospital, 600 N. Wolfe St., Baltimore, MD 21205.

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The vest system used in this study is the subject of a U.S. patent licensed to CardioLogic Systems, Inc. Dr. Halperin, Dr. Tsitlik, Mr. Gelfand, Dr. Weisfeldt, Dr. Gruben, Dr. Levin, Dr. Rayburn, Dr. Chandra, and Dr. Guerci all have an equity interest in CardioLogic Systems, Inc.

METHODS

The Vest System for CPR

The vest system, developed by the investigators, consists of a bladder-containing vest, analogous to a large blood-pressure cuff, and a computerized pneumatic system (Fig. 1). The pneumatic system directs compressed air into and out of the bladder by the proper sequencing of solenoid valves and represents the evolution of a previous laboratory system.[12] As used in this study, during four sequential inflation–deflation cycles, the vest was deflated to approximately 10 mm Hg, and during the fifth compression cycle, the

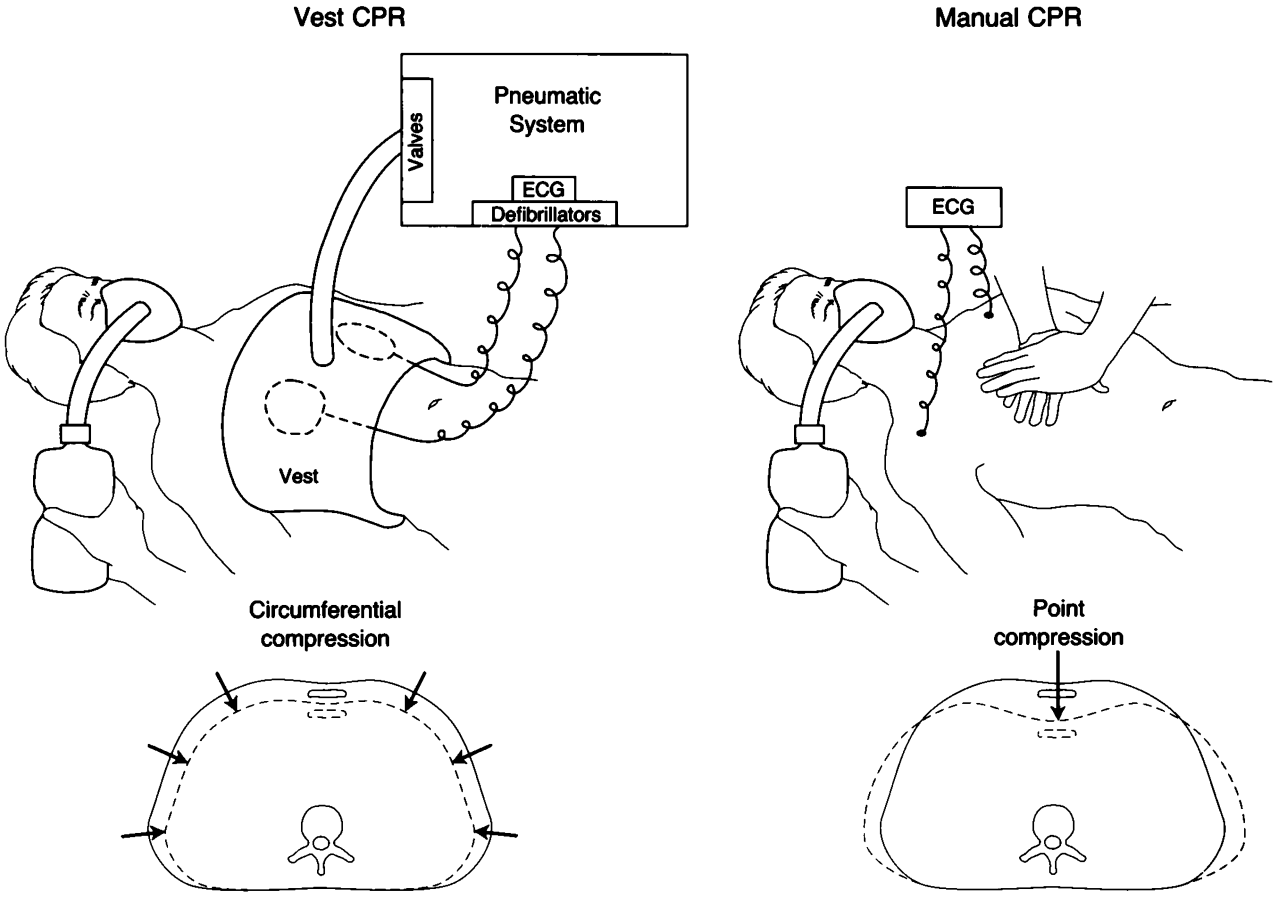

Figure 1. A Comparison of the Thoracic-Vest System for Cardiopulmonary Resuscitation (Vest CPR) with Standard Manual CPR.
The vest contains a bladder that is inflated and deflated by the pneumatic system. Defibrillation can be accomplished during chest compression through the flat defibrillator electrodes (dashed circles) under the vest. The electrocardiogram (ECG) can be recorded through the same electrodes.

The lower panels show schematic representations of transverse sections of the midthorax during vest CPR and manual CPR. The thoracic size during chest relaxation is shown by the solid lines. The arrows indicate force applied to the thorax during chest compression. With vest inflation, there is a relatively uniform decrease in the dimensions of the thorax. With manual CPR, the sternum is displaced during compression (arrow) and the lateral thorax can bulge, thereby increasing thoracic volume and reducing the intrathoracic pressure generated during compression.

vest was completely deflated to atmospheric pressure. This deflation scheme maintained a low positive pressure on the thorax between most cycles of vest inflation, thus keeping the vest tight against the thorax to increase intrathoracic pressure, while completely releasing pressure every fifth cycle to allow full expansion of the thorax for ventilation.

Study Design

Phase 1

In phase 1 of the study, which was approved by our institutional review board, we compared the vascular pressures produced by vest CPR with those produced by manual CPR. The protocol required that at least 20 minutes of manual CPR be performed before vest CPR was used, because spontaneous cardiac function rarely returns after 20 minutes of unsuccessful manual CPR.[13] This approach was designed to allow the assessment of the vascular pressures produced by manual and vest CPR without denying standard resuscitative measures initially or performing CPR on patients who had already sustained irreversible brain injury. Patients were enrolled from the emergency department and the intensive care units. These patients were thought to be comparable, since all had had prolonged cardiac arrest before enrollment. Obtaining informed consent was not possible, but the use of subjects in such circumstances is considered ethically acceptable in research on cardiopulmonary resuscitation.[14]

After verifying the entry criteria, an investigator placed vascular sheaths percutaneously into the femoral artery and vein. The distance from each sheath to the midsternum was noted, and micromanometer-tipped catheters (Model PC-470, Millar) were advanced through the sheaths for those predetermined distances. Preliminary studies showed that with this procedure, the tip of the arterial catheter was placed near the ascending aorta and the tip of the venous catheter was placed near the right atrium. Exact positioning of the catheters was not critical, since pressures varied little as the catheters were advanced to their final positions. Catheter placement was deemed successful if the pressure wave forms from the two catheters were not identical and the color of blood from them was visibly different, indicating that they were in different vascular spaces.

After placement of the catheters, the study of vest CPR and manual CPR began. For vest CPR, the vest was placed around the patient's thorax (Fig. 1) and was cyclically inflated 60 times per minute, with 180 to 250 mm Hg pressure in the vest maintained for

40 to 50 percent of each cycle. For manual CPR, the chest was compressed without the vest in place, with computer-generated sounds used to time chest compression and relaxation. There were 90 compressions per minute, with compression for approximately 50 percent of the cycle. The sternal compression force was measured with a precision force transducer[15] and recorded on an oscillograph (2400 Series, Gould). By looking at the recordings and adjusting the force accordingly, the investigator applied a peak force of 400 N (1 newton = 1 kg · m per second squared, or 0.225 lb-force). That force level was selected because it generally exceeds the maximum that house officers at our institution use, because we wanted to maximize the pressures generated by manual CPR, and because higher levels have been shown to produce substantial trauma in animal models.[3] Sternal displacement was measured by a calibrated mechanical arm.[15] Pressure, force, and displacement signals were digitized and stored by a microcomputer-based data-acquisition system.[16]

Epinephrine was given before and during the study according to our hospital's protocol, with doses of 1 mg, 3 mg, 5 mg, 7 mg, and 10 mg at successive five-minute intervals. Doses of epinephrine above 1 mg can increase vascular pressures during CPR,[17] but their effect on survival remains to be determined. In addition, most patients received at least 1 mmol per liter of sodium bicarbonate per kilogram of body weight before, but not during, the study.

After the prolonged period of unsuccessful manual CPR before the study began, pressures were recorded during manual CPR and vest CPR, as allowed by time constraints. In seven patients, pressures were first recorded during 5 to 15 minutes of manual CPR, then during a similar period of vest CPR. In 18 patients, pressures were first recorded during a period of vest CPR, then during a similar period of manual CPR. In six of the latter patients, pressures were also recorded during a second, subsequent period of vest CPR. No formal mechanism was used to determine whether pressures were first recorded during manual CPR or vest CPR. Finally, in four patients, pressures were recorded only during vest CPR.

Pressure signals adequate for analysis were obtained during manual CPR and vest CPR in 15 patients, designated the phase 1 hemodynamic study group. Pressure signals were not obtained in the remaining patients because 2 were resuscitated with vest CPR before the completion of the study and there were problems with catheter placement in 12.

During manual CPR and vest CPR, ventilation was performed after every fifth compression with a standard manual resuscitator bag delivering 100 percent oxygen through an endotracheal tube. Defibrillation was performed as indicated. Spontaneous circulation was defined as present if the systolic blood pressure was greater than 90 mm Hg and no CPR was being performed.

Phase 2

Phase 2 of this study was undertaken to determine whether the earlier use of vest CPR would increase the likelihood of return of spontaneous circulation or short-term survival. This randomized, controlled study was performed under an investigational-device exemption from the Food and Drug Administration on inpatients, and the protocol was approved by our institutional review board.

In each case of unsuccessful manual CPR after cardiac arrest, an investigator verified that the time since the cardiac arrest was less than 20 minutes, that the patient had undergone endotracheal intubation, and that the patient had received chest compressions, at least 1 mg of epinephrine, and defibrillation, if appropriate. The patient was then randomly assigned (by the opening of a sealed, serially numbered envelope) to receive either vest CPR or continued manual CPR. If the patient was assigned to manual CPR, the investigators left the scene and manual CPR was continued by hospital personnel until spontaneous circulation was restored or until resuscitative measures were stopped by the primary physician. If the patient was assigned to vest CPR, the vest was secured around the patient, and vest pulsations were started as described above; the CPR was continued as with manual CPR. Except for the operation of the vest, all resuscitative measures were specified by the primary physician, who used standard Advanced Cardiac Life Support procedures and high-dose epinephrine. Patient care after resuscitation was also specified only by the primary physician. Ventilation was performed after every fifth compression, and defibrillation was performed as indicated.

Statistical Analysis

Phase 1

The primary objective of this crossover study was to evaluate the efficacy of vest CPR in generating increases in vascular pressure among patients with cardiac arrest who had failed to respond to at least 20 minutes of conventional resuscitative measures. Pressure data are reported for the 15 patients for whom we had adequate aortic and right-atrial pressure recordings during manual CPR and vest CPR. For seven patients manual CPR was studied before vest CPR, and for the remaining eight patients the order was reversed. Coronary perfusion pressure was defined as the mean relaxation-phase pressure gradient between the aorta and the right atrium. Two-tailed, paired t-tests and Wilcoxon's signed-rank tests[18] were used to determine whether vest CPR raised vascular pressures significantly higher than manual CPR. To assess potential order effects, we performed a subgroup analysis using Wilcoxon's signed-rank test to compare treatment effects in the two groups of patients that had received vest CPR and manual CPR in different order. Data are presented as means ±SD.

Phase 2

The objective of this randomized, controlled trial was to determine whether vest CPR increased the frequency of return of spontaneous circulation or short-term survival, as compared with continued manual CPR, in patients with cardiac arrest who had not been successfully resuscitated by up to 20 minutes of standard CPR. The numbers of patients with return of spontaneous circulation produced by manual CPR and vest CPR were compared in an intention-to-treat analysis with use of the two-tailed Fisher's exact test.[18] This analysis was supplemented by an analysis of the overall effects of treatment on survival rates in the 24 hours after resuscitation, with use of the Mantel–Haenszel (log-rank) test in a Kaplan–Meier survival analysis.[18]

Results

Phase 1

Among the 15 patients in the hemodynamic study group, the average age was 51±19 years; there were 7 men, and the average weight was 62±24 kg. As compared with manual CPR, vest CPR markedly increased the peak aortic pressure as well as the relaxation-phase pressure (one of the largest changes in pressure produced by vest CPR is shown for Patient A in Fig. 2). During the compression phase, the aortic and right-atrial pressures were similar, so that the difference between them was small. During the relaxation phase, however, the difference between the aortic and right-atrial pressures (coronary perfusion pressure) generated by vest CPR was markedly higher than that generated by manual CPR. (One of the smallest changes in pressure is shown for Patient B in Fig. 2.)

For the hemodynamic study group as a whole (Fig. 3), vest CPR increased both peak aortic pressure (from 78±26 to 138±28 mm Hg, $P<0.001$) and coronary perfusion pressure (from 15±8 to 23±11 mm Hg, $P<0.003$). Vest CPR also increased the mean aortic

pressure (from 44±13 to 77±17 mm Hg, P<0.001) and the mean relaxation-phase aortic pressure (from 33±12 to 44±13 mm Hg, P<0.001) (Table 1). Similar results were obtained whether the data were analyzed with paired t-tests or with the Wilcoxon signed-rank test. In addition, the effect of vest CPR on the vascular pressures was significant regardless of the order in which the methods of CPR were used (i.e., whether the vest was used first or second). These pressures may not, however, be representative of the 14 patients in whom adequate pressure signals were not obtained.

None of the 15 patients in the hemodynamic study group had return of spontaneous circulation with manual CPR. In addition, none of the other 14 patients had return of spontaneous circulation with manual CPR. These 29 patients had an average age of 51±18 years, 15 were men, and the duration of initial manual CPR was 50±22 minutes; 4 of the 29 (14 percent) had return of spontaneous circulation with vest CPR, but none survived to be discharged from the hospital.

Phase 2

The characteristics of the patients studied in phase 2 are presented in Table 2. Spontaneous circulation returned in 3 of 17 patients (18 percent) randomly assigned to continued manual CPR, as compared with 8 of 17 patients (47 percent) randomly assigned to vest CPR (P = 0.14). Twenty-four hours after resuscitation the difference in survival between the two groups was smaller (1 of 17 given manual CPR vs. 3 of 17 given vest CPR) than at six hours (1 of 17 vs. 6 of 17), suggesting that the benefits of vest CPR in this patient population are often short-lived. During the 24 hours after resuscitation, survival analysis showed improved short-term survival with vest CPR (P = 0.03); there were no differences in medical therapy during this period.

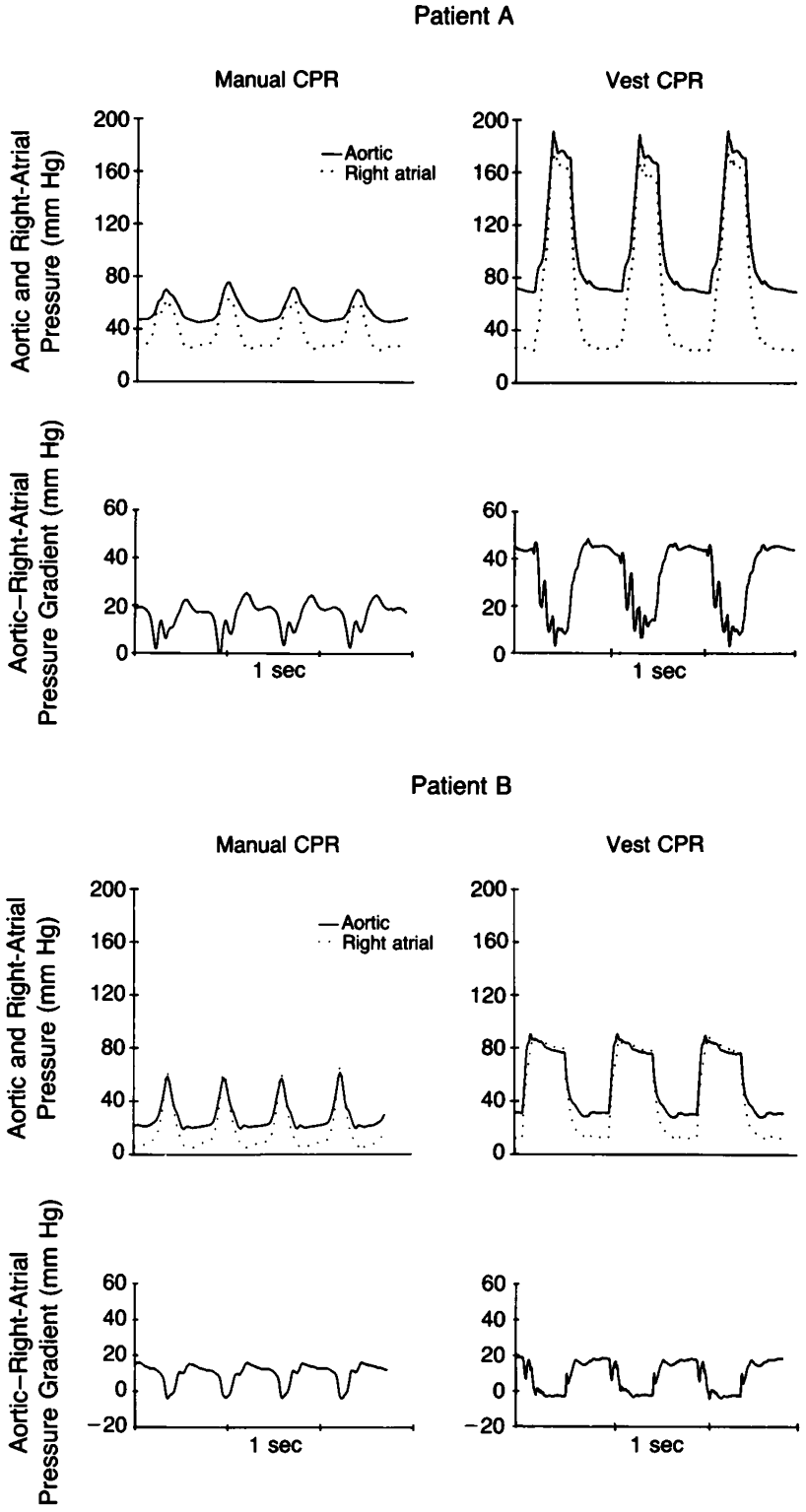

Figure 2. Vascular Pressure during Manual CPR and Vest CPR in Two Patients, as Reproduced from Digital Recordings Made in Phase 1 of the Study.

As compared with the pressures produced by manual CPR, the recording for Patient A shows one of the largest changes in aortic pressure (solid line) and aortic–right-atrial pressure gradient (coronary perfusion pressure; lower line) produced by vest CPR, whereas the recording for Patient B shows one of the smallest changes.

Vest CPR was applied earlier in the course of cardiac arrest in phase 2 than in phase 1 (duration of initial manual CPR, 13±4 vs. 50±22 minutes). This earlier application was associated with an improvement in the return of spontaneous circulation (8 of 17 patients vs. 4 of 29 patients).

Autopsies were performed in four patients who underwent vest CPR and five who underwent manual CPR only. In the patients given vest CPR, congested lungs were present in two and broken ribs in one. In the patients given manual CPR only, congested lungs were noted in five, broken ribs in two, an intrathoracic hematoma in one, and a chest-wall hemorrhage in one. Thus, no additional trauma appeared to have been produced by vest CPR.

Discussion

Studies have shown that an important factor affecting survival after cardiac arrest is the level of vascular pressure, especially the coronary perfusion pressure, generated during resuscitation attempts[1-4] — particularly after the failure of initial defibrillation. Invasive techniques such as open-chest cardiac massage[19] and cardiopulmonary bypass[20] increase vascular pressures as compared with manual CPR, but they have not as yet come into widespread use, partly because they must be performed by physicians. Noninvasive techniques could have more general application, but methods such as chest compression with simultaneous ventilation,[21,22] previously developed techniques for circumferential thoracic-vest inflation,[23-25] and manual chest compression at increased rates[24,26] have generated pressures only marginally different from those produced by manual CPR. Specifically, one previous vest-CPR system used generated coronary perfusion pressures in patients of only 11±13 mm Hg, as compared with 14±15 mm Hg for manual CPR.[24]

Table 1. Measurements in the 15 Patients in Phase 1 of the Study for Whom Data Were Available.*

Variable†	Manual CPR	Vest CPR
Mean aortic pressure (mm Hg)	44±13	77±17‡
Peak aortic pressure (mm Hg)	78±26	138±28‡
Mean aortic relaxation-phase pressure (mm Hg)	33±12	44±13‡
Aortic minus right-atrial mean relaxation-phase pressure (mm Hg)	15±8	23±11§
pH	7.03±0.28	6.92±0.14
PCO_2 (mm Hg)	54±34	47±24
PO_2 (mm Hg)	141±168	193±174
Peak vest pressure (mm Hg)	—	234±25
Peak sternal force (N)¶	395±32	—
Peak sternal displacement (cm)	3.4±0.8	—

*Plus–minus values are means ±SD. Values are for 15 patients, except for arterial-blood gases (n = 5).

†PCO_2 denotes partial pressure of carbon dioxide, and PO_2 partial pressure of oxygen.

‡P<0.001 for the comparison with manual CPR.

§P<0.003 for the comparison with manual CPR.

¶One newton (N) equals 1 kg · min per second squared, or 0.225 lb-force.

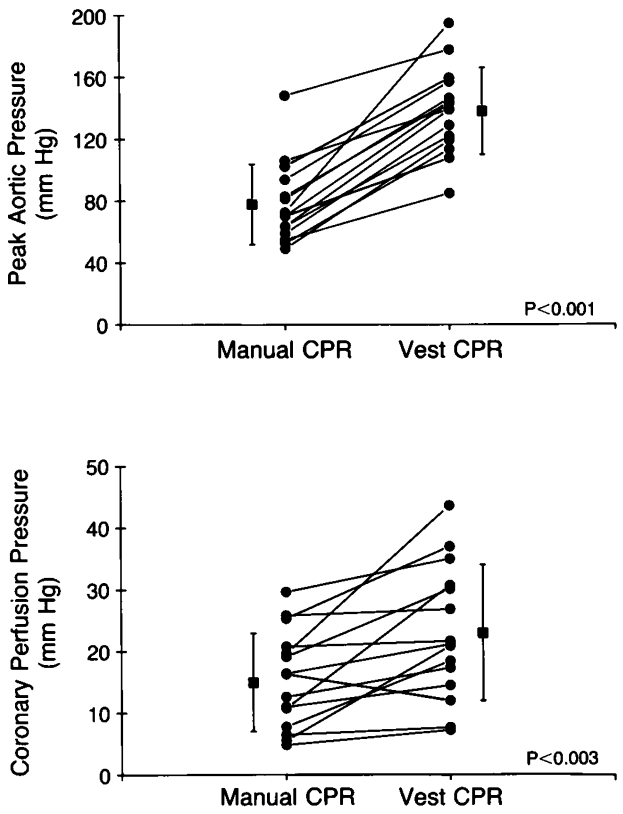

Figure 3. Peak Aortic Pressure and Coronary Perfusion Pressure Measured in 15 Patients during Manual CPR and Vest CPR in Phase 1 of the Study.

The vertical bars and squares are the mean values ±SD for the two types of CPR. There was a significant increase in each vascular pressure with vest CPR as compared with manual CPR.

To optimize the level of vascular pressure during noninvasive methods of resuscitation, it is necessary to understand fully the mechanisms whereby blood flows. It is clear that fluctuations in intrathoracic pressure can generate blood flow[5,6,23,24] and increases in intrathoracic pressure can raise pressure in the intrathoracic vascular structures. When venous valves are competent, arterial blood flows out of the thorax during chest compression. During chest relaxation, intrathoracic pressure falls, with a return of venous blood into the thorax and retrograde flow of arterial blood into the thorax for perfusion of the coronary arteries.

We designed a new vest-CPR system to optimize the effect of intrathoracic pressure on intravascular pressure and flow. Pressure in the thorax rises as the thorax is compressed. Thoracic compression causes medium-sized airways to collapse, trapping air in the lungs,[27] so that further compression of the thorax causes intrathoracic pressure to rise (by Boyle's law) in proportion to the decrease in thoracic volume. With vest CPR, there is circumferential compression of the thorax, which, because of geometric advantages, is more efficient at reducing the volume of the thorax

Table 2. Characteristics of the 34 Patients in Phase 2 of the Study.*

Characteristic	Manual CPR (N = 17)	Vest CPR (N = 17)
Age (yr)	69±18	61±16
Sex (M/F)	10/7	10/7
Weight (kg)	67±18	69±20
Initial rhythm		
VT or VF	8	3
Asystole	3	6
EMD	3	5
Time from arrest to randomization (min)	9±3	13±4
Duration of CPR after randomization (min)		
All patients	15±7	16±13
Patients without return of spontaneous circulation	17±11	18±13
pH	7.22±0.19	7.17±0.19
PCO_2 (mm Hg)	63±25	47±14
PO_2 (mm Hg)	80±67	210±150
Epinephrine dose (mg)	16±9	15±11

*Plus–minus values are means ±SD. The initial rhythm was determined at the start of cardiac arrest; data were not recorded for three patients in each group. VT denotes ventricular tachycardia, VF ventricular fibrillation, EMD electromechanical dissociation, PCO_2 partial pressure of carbon dioxide, and PO_2 partial pressure of oxygen. Arterial-blood gas values (n = 6 for manual CPR and n = 5 for vest CPR) were measured during CPR after randomization; epinephrine doses were also measured after randomization.

than manual CPR. Vest CPR can, therefore, generate large changes in vascular pressures while producing less trauma than manual CPR,[3] because of the reduced deformation of individual portions of the thoracic wall. The improved vest-CPR system is more effective than previous systems because of advances in vest design and vest-inflation techniques. The improved vest covers more of the thorax than previous systems, and the distribution of the compression force over a large area of the thorax allows total compression forces up to 3000 N to be used.

After laboratory studies demonstrating the mechanisms, safety, and efficacy of vest CPR were completed,[3] we designed and built a system for use in patients. We first studied the hemodynamic effects of vest CPR and manual CPR in a situation in which there was essentially no risk to the patient. We chose the end of standard resuscitative measures as an appropriate time to test vest CPR. We found that vest CPR increased vascular pressures as compared with manual CPR. In addition, there was a surprising result — the return of spontaneous circulation in 4 of the 29 patients studied in phase 1. None of these patients survived to be discharged from the hospital, however, probably because irreversible organ damage had been caused by the prolonged period of inadequate perfusion before vest CPR was begun.

The peak aortic pressures reported for manual CPR in this study were at least as high as those reported by other investigators,[2,4] as were the coronary perfusion pressures.[4,24] It is very unlikely, therefore, that inadequate compression force was responsible for the observation that manual CPR produced lower pressures than vest CPR.

It is conceivable that the coronary perfusion pressures produced by vest CPR might have been even higher if the vest had been used earlier after cardiac arrest. Laboratory studies of resuscitation attempts showed that coronary perfusion pressures during manual CPR decreased substantially toward the end of a 20-minute period,[3] probably because decreased peripheral resistance caused increased arterial runoff. This decreased peripheral resistance is probably the result of decreased arterial tone caused by direct damage to the vascular smooth muscle or by changes in smooth-muscle cell receptors.

On the basis of the favorable results of the phase 1 study, we conducted a randomized trial to determine whether earlier use of vest CPR would increase short-term survival. We recognized that long-term survival was unlikely, because of the severity of disease in this high-risk patient population, because of the necessity of waiting for the patient to be intubated, and because we used vest CPR only after the initial round of chest compression, defibrillation, and drugs had been unsuccessful. Despite the time required to enroll patients in the study, there was an improvement in short-term survival among patients treated with vest CPR as compared with those treated with manual CPR only. However, none of the patients survived to be discharged from the hospital.

In summary, our preliminary data demonstrate the capacity of vest CPR to increase aortic pressure and coronary perfusion pressure and show the potential value of this technique in restoring spontaneous and effective cardiac activity. If spontaneous circulation is restored early after cardiac arrest, before irreversible organ damage has occurred, perhaps long-term survival may be possible. The effect of vest CPR on long-term survival will, however, require further study.

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