Evaluation of Standard and Active Compression-Decompression CPR in an Acute Human Model of Ventricular Fibrillation

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Background The mechanisms that underlie cardiopulmonary resuscitation (CPR) in humans remain controversial and difficult to study. This report describes a new human model to evaluate CPR during the first 1 to 2 minutes after the onset of ventricular fibrillation (VF). With this model, standard CPR was compared with active compression-decompression (ACD) CPR, a method that uses a handheld suction device to actively compress and actively decompress the chest.

Methods and Results During routine inductions of VF as part of a transvenous lead cardioverter/defibrillator implantation procedure, CPR was performed in 21 patients if the first defibrillation shock failed and until a successful rescue shock was delivered. Compressions during CPR were performed according to American Heart Association guidelines. For ACD CPR, decompression was performed with up to 30 lbs. Radial arterial and right atrial pressures were measured in all patients. Esophageal pressures, intrathoracic pressures, or minute ventilation was measured in the last 13 patients. Application of both CPR techniques increased arterial and right atrial pressures. The mean coronary perfusion pressure was increased throughout the entire CPR cycle with ACD CPR (compression, 21.5±9.0 mm Hg; decompression, 21.9±8.7 mm Hg) compared with standard CPR (compression, 17.9±8.2 mm Hg; decompression, 18.5±6.9 mm Hg; P<.02 and P<.02, respectively). Ventilation per compression-decompression cycle was 97.3±65.6 mL with standard CPR and 168.4±68.6 mL with ACD CPR (n=7, P<.001). Negative inspiratory pressure was −0.8±4.8 mm Hg with standard CPR and −11.4±6.3 mm Hg with ACD CPR (n=6, P<.04).

Conclusions Patients undergoing multiple inductions of VF during cardioverter/defibrillator implantation with transvenous leads provide a well-controlled and reproducible model to study the mechanisms of CPR. Using this model, ACD CPR significantly increased arterial blood pressure, coronary perfusion pressure, minute ventilation, and negative inspiratory pressure compared with standard CPR. (Circulation. 1994;89:684-693.)

Key Words • cardiopulmonary resuscitation • perfusion • pressure

Despite its widespread use, the mechanisms that underlie cardiopulmonary resuscitation (CPR) in humans remain controversial, difficult to study, and poorly understood. Although a number of innovative modifications have been developed to try to improve the dismal CPR survival statistics,1-14 relatively few changes have been made in the basic method of CPR since it was first described more than 30 years ago.15 One significant impediment to improvement in current methods of CPR has been the lack of an acute human model to study this complex process. To date, much of our knowledge has been derived from animal studies or studies in humans after a prolonged cardiac arrest. Although both kinds of studies are important, it has been difficult to extrapolate many of the results from animal investigations to human application because of differences in size, chest geometry, and sensitivity to different drug therapies. Furthermore, despite the importance of information gained from studies in patients in cardiac arrest who have undergone prolonged periods of resuscitation,2-4,5,16-20 invasive monitoring needed for studying mechanisms of CPR acutely is difficult to implement rapidly. As a result, it has been impossible to study cardiopulmonary function in a reproducible manner in patients during the critical seconds to minutes after the onset of cardiac arrest, the time when CPR may be most effective in preserving vital organ function.

In this report, we describe a new, well-controlled, and reproducible model of ventricular fibrillation (VF) in humans to evaluate the acute hemodynamic effects of CPR. This model uses patients undergoing placement of implantable cardioverter/defibrillators with transvenous lead systems in the operating room. The advantage of the transvenous leads is that no thoracotomy is required for implantation of the defibrillator. This is critical to this model, because the chest remains intact. Since VF must be induced a number of times during the standard implantation testing procedure to optimize defibrillation thresholds, chest compressions can be performed in the interval between the unsuccessful defibrillation and the subsequent rescue defibrillation. With this model, we compared standard CPR with a newly described method of CPR, active compression-decompression CPR (ACD CPR),16,21 that uses a handheld suction device to assist in chest compression as well as active chest decompression. This acute model provides an opportunity to assess the hemodynamic and ventilatory parameters of different methods of CPR during repet-
Fig 1. Study protocol flow diagram indicating when either standard (STD) or active compression-decompression (ACD) cardiopulmonary resuscitation (CPR) was performed. Methods were alternated on subsequent repetitions of this protocol.

itive inductions of “controlled” VF without risk to the patient.

Methods

This study was approved by the University Committee on the Use of Human Subjects in Research of the University of Minnesota. Before the procedure, informed consent was obtained from each patient.

We studied 21 consecutive patients undergoing placement of an implantable cardioverter/defibrillator (Medtronic PCD model 7217B, Medtronic Inc, Minneapolis, Minn) with transvenous lead systems between February and December 1992 in the operating rooms of the University of Minnesota Hospital. Hemodynamic monitoring was routinely performed by the anesthesiologist using pulmonary artery (Baxter-Edward Swan Ganz Thermomodule Pace Port Catheter, Baxter Health Care Corp, Irvine, Calif) and radial artery fluid-filled catheters. During the implantation procedure, two and sometimes three transvenous leads were positioned in either the right ventricular apex, the superior vena cava, and/or the coronary sinus by a standard subclavian vein approach. An elliptical subcutaneous patch electrode was also routinely positioned on the left anterior chest by making a 4- to 8-cm incision under the left breast lateral to the midclavicular line. Either method of CPR could be performed without affecting the area immediately adjacent to the incision. Once the electrodes were in place, ventricular fibrillation was induced repeatedly to obtain optimal defibrillation thresholds. One or more potential lead configurations and pulse delivery sequences were tested to fulfill the manufacturer and Food and Drug Administration implant criteria of three successful defibrillations at \( \leq 18 \) J.

The protocol used in this study is illustrated in Fig 1. VF was induced by low-voltage alternating current applied to the endocardium for 3 to 6 seconds through the right ventricular lead. The defibrillator was programmed to detect VF, charge, and deliver a shock to the patient through the transvenous lead system. If defibrillation was successful, no CPR was performed. If the initial defibrillation attempt failed and the patient remained in VF, chest compressions were started with either standard CPR or ACD CPR. The initial method of CPR was determined randomly before the first VF induction. Standard CPR or ACD CPR was performed for 12 to 15 seconds during the time needed for the defibrillator to charge to the maximum output of the implantable device, 34 J, and to deliver a rescue shock. Since approximately 20 seconds is required to induce, detect, and discharge, CPR was initiated 20 to 25 seconds after induction of VF when the first shock was ineffective. In the event that the “rescue shock” failed to terminate VF, the same CPR intervention was performed for an additional 10 to 90 seconds until defibrillation was successful. During each subsequent VF induction, the alternate CPR method was used. In this way, each patient served as his or her own control. There were usually several opportunities to perform CPR in a given patient while different lead configurations and pulse delivery sequences were being evaluated. There was at least 5 minutes between successive VF inductions. VF was not induced for the sole purpose of performing CPR.

ACD CPR was performed with a sterile Ambu CardioPump™ (Ambu Inc, Denmark). This device (Fig 2) has three essential parts: (1) a neoprene suction cup, (2) a plastic circular handle with an undercut hand grip, and (3) a force gauge. The gauge is calibrated to ensure a depth of compression equivalent to standard CPR (1.5 to 2 inches), regardless of the compliance of the patient’s chest. The gauge also measures force of decompression, up to \( \approx 30 \) lb, to aid the operator in this function. During use, the device is positioned over the midsternum at the level of the nipples. Compressions with both standard CPR and ACD CPR were performed with a 50% duty cycle to a depth of 1.5 to 2 inches and at a rate of 80 compressions per minute, according to CPR guidelines of the American Heart Association. Compression rate was kept constant with the use of a metronome. Active decompression was performed at the same rate, immediately at the end of the compression phase, by actively lifting up on the handle with up to \( \approx 30 \) lb per cycle but without dislodgment of the device from the chest. CPR was performed with strict adherence to sterile operating room technique.

Radial arterial and right atrial pressures were measured in all patients during baseline native rhythm immediately before VF induction, during VF immediately before commencement of CPR, and during CPR. Intrathoracic pressures during CPR were assessed with esophageal manometry in five patients, by recording distal intrathracale pressures in five patients, and with both techniques in one patient. Minute ventilation was measured during CPR in seven patients by routine spirometric techniques. Inspiratory and expiratory pressures were mea-
sured while CPR was performed by measuring distal endotracheal tube (Mallinckrodt Broncho-Cath endobronchial tube with CPAP system, Mallinckrodt Medical, Inc, St Louis, Mo) pressures over a 10- to 15-second period during VF.

Data Analysis

A single CPR cycle was 750 milliseconds in duration and was defined as beginning with chest compression and terminating at the end of either passive or active decompression. The onset of a given CPR cycle was determined on the pressure tracings as the first rapid upstroke in the right atrial pressure associated with the initiation of compression. Because of the peripheral position of the arterial line, the arterial line recordings were phase shifted 50 to 70 milliseconds to correct for the time delay in the onset of compression in the aorta versus the radial artery. The number of milliseconds needed to correct for this delay in the abrupt rise of the radial artery pressure curve with CPR was determined by measuring the delay between the rise in right atrial pressure and the rise in the radial artery pressure in the first compression beat after the initiation of standard CPR. This was confirmed by overlapping the right atrial and radial artery pressure curves by use of a computer (see below) to ensure that the rapid rise in both pressure curves was coincident. The degree of offset determined with the standard CPR tracings was then applied to ACD CPR as well.

Pressure waveforms were analyzed with the assistance of a Macintosh IX computer (Apple Computer, Inc, Cupertino, Calif). In the first 10 patients, pressure waveforms were recorded with a Mingograph 7 recording system (Siemens-Elema AB, Sweden). Pressure tracings during CPR were then digitized with a Kurta Pad IS/ADB input system (Kurma Corp, Phoenix, Ariz). In the last 11 patients, all recordings were made directly onto a standard VHS recorder (Panasonic model PV-4760, Panasonic Corp, Japan) with a Vettermatic recording adapter, model 4000 (AR Vettermatic, Rebersburg, Pa) and Sony digital audio processor, model PCM-801ESD (Sony Corp, Japan) recording system, which was then interfaced directly in the computer using a MacADIOS analog-digital conversion system. Pressure waveforms obtained during CPR were analyzed with Microsoft Excel software (Microsoft Corp, Redmond, Wash) and Superscope program (GW Instruments, Somerville, Mass) to determine the mean pressure for any particular measurement during different portions of the CPR cycle. Five CPR cycles were analyzed for each intervention, beginning with the compression that began 20 to 25 seconds after induction of VF. Mean arterial, right atrial, and coronary perfusion pressures (defined as the arterial minus the simultaneous right atrial pressure) were determined three different ways: for an entire CPR cycle (750 milliseconds), for each half of the CPR cycle (375 milliseconds), and for each third of the CPR cycle (250 milliseconds). Esophageal manometry was performed with an Arndorfer fluid-filled multilumen catheter (Arndorfer Inc, Greendale, Wis) by standard techniques. A Narco Biosystems MMS 200 recorder (International Biomedical Corp, Houston, Tex) was used to measure midesophageal (endocardiac) pressures in the first three patients, and a Mingograph 7 recording system (see above) was used for the remaining three patients.

Ventilation per CPR cycle, in liters, was determined by measuring the amount of expired gas during the total time CPR was performed divided by the number of CPR cycles. All patients were preoxygenated with 100% FiO2 before induction of VF. Patients were apneically oxygenated (100% FiO2) during the first 10 to 15 seconds of CPR when ventilation or expiratory and inspiratory pressures were being measured. To calculate minute ventilation, the volume of ventilation per CPR cycle was multiplied by 80, the number of CPR cycles per minute. Positive expiratory and negative inspiratory pressures during CPR were determined by averaging the peak and trough values, respectively, over five consecutive CPR cycles.

Statistical analysis was performed by Student’s paired t test. Statistical significance was defined as a value of P≤.05. All data are expressed as mean±SD.

Results

Twenty-one consecutive patients (5 women and 16 men) undergoing implantable cardioverter/defibrillator placement with transvenous leads were included in this study. The mean age was 62.1±13.3 years. Sixteen patients had a history of hemodynamically unstable VT, and 5 had a history of a VF arrest. Sixteen patients had coronary artery disease, 4 had idiopathic cardiomyopathy, and 1 had congenital heart disease. The mean ejection fraction for all patients was 34.9±14.4%. One patient underwent coronary artery bypass surgery 3 weeks before our study. After performance of both types of CPR after a total of four failed initial defibrillations, his sternum had significantly less rigidity. Although we included data from this patient, for safety reasons we subsequently excluded any patient from the study who had undergone a thoracotomy within the previous 6 months.

Hemodynamic measurements during sinus (or native) rhythm and during baseline VF immediately before the failed first defibrillation are shown in Table 1. Arterial and right atrial pressures as well as heart rates were similar for each individual patient in both the standard and ACD CPR groups during normal sinus rhythm and during VF. Waveforms obtained from a patient during the induction protocol demonstrating arterial and right atrial pressures in sinus rhythm, VF without CPR, and VF with standard CPR are shown in Fig 3. If we waited at least 5 minutes after each successful defibrillation, there was <10% deviation between the blood pressure and heart rate in any given patient before each subsequent induction of VF and during VF but before CPR. During VF, but before CPR, there was a mean positive coronary artery perfusion gradient of 19.1±6.7 mm Hg and 20.1±5.8 mm Hg in the standard and ACD CPR groups, respectively (P=NS; range, 8 to 37 mm Hg). The coronary perfusion gradient was positive in all patients. With the onset of VF, arterial blood pressure fell rapidly, and there was a small rise in right atrial pressure (Table 1). When either standard or ACD CPR was performed during VF, the peak and mean radial arterial pressures and right atrial pressures were markedly higher compared with hemodynamic measurements in the absence of CPR (Table 2).

Hemodynamic parameters were assessed during the entire CPR cycle as well as when the CPR cycle was divided in half and into thirds. As shown in Table 2, mean arterial pressures during the entire CPR cycle were significantly higher with ACD CPR compared with standard CPR. The right atrial pressures tended to be lower during the last third of the CPR cycle in the ACD group (13.8±6.8 mm Hg with ACD CPR versus 16.1±8.2 mm Hg with standard CPR, P=.097), probably as a result of the greater negative intrathoracic pressure generated with active chest wall decompression. Representative tracings of the radial arterial and right atrial pressure curves during standard CPR and ACD CPR from the same patient, obtained 5 minutes apart, illustrate these findings further (Fig 4). With standard CPR, there was a rapid rise in the arterial and venous pressures with chest compression. With ACD CPR, the
rise in the arterial pressure began earlier than with standard CPR. This was observed after the first active decompression in nearly every patient. That is, after the first complete ACD CPR cycle, the arterial pressure increased earlier, during late diastole, in the subsequent ACD CPR cycles compared with the corresponding standard CPR cycles.

These differences between the two methods of CPR became more apparent during apneic respiration in a different patient when intratracheal pressures were obtained. In Fig 5, the negative intratracheal pressures obtained with temporary occlusion of the endotracheal tube demonstrate the more negative intrathoracic pressure obtained with ACD CPR compared with standard CPR. Although peak positive intratracheal pressures were similar, implying equal force of compression with both techniques, the arterial and right atrial systolic pressures were higher and the right atrial diastolic pressures were lower when gases were prevented from entering the lungs during the decompression phase of ACD CPR. That is, when equalization of thoracic pressures during active decompression was forced to occur solely by movement of venous blood into the thorax (since the endotracheal tube was temporarily occluded, preventing movement of gases from contributing to this rapid equilibration process), the differences between standard and ACD CPR hemodynamics were even more pronounced.

The coronary perfusion pressures obtained during VF with both methods of CPR are shown in Tables 2 and 3. By measuring maximum and minimum pressures during the compression (systolic) and decompression (diastolic) phases in the traditional manner,28 we observed that coronary perfusion during the compression phase was 10.3±8.8 mm Hg with standard CPR versus 18.2±8.1 mm Hg with ACD CPR (P<.001) and during the decompression phase was 20.1±5.6 mm Hg with standard CPR versus 21.8±9.4 mm Hg with ACD CPR (P=.22). By measuring mean coronary perfusion gradients, we determined that the mean perfusion pressures over the whole CPR cycle were 17% higher in the ACD CPR group (22.6±9.3 mm Hg) than in the standard CPR group (19.3±8.4 mm Hg; P=.02).

Measurements of mean coronary perfusion pressures during the compression and decompression phases and after the CPR cycle were divided into thirds are shown in Tables 2 and 3. With this method of analysis, the largest differences between mean coronary perfusion pressures with the two CPR techniques were observed in the first and last thirds of the CPR cycle, in which coronary perfusion pressures were 28.5% (P=.03) and 18.7%
TABLE 2. Comparison of Arterial, Right Atrial, and Coronary Perfusion Pressures During ACD and STD CPR

<table>
<thead>
<tr>
<th></th>
<th>Peak Pressures</th>
<th>Mean Pressures (Compression/Decompression Phases)</th>
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<tr>
<td></td>
<td>ACD</td>
<td>STD</td>
</tr>
<tr>
<td>Arterial pressure, mm Hg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Compression</td>
<td>60.1±13.7</td>
<td>54.3±11.3</td>
</tr>
<tr>
<td>Decompression</td>
<td>30.7±7.0</td>
<td>30.4±4.4</td>
</tr>
<tr>
<td>Right atrial pressure, mm Hg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Compression</td>
<td>42.1±13.1</td>
<td>44.0±14.0</td>
</tr>
<tr>
<td>Decompression</td>
<td>7.8±7.9</td>
<td>10.3±5.9</td>
</tr>
<tr>
<td>Coronary perfusion pressure, mm Hg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Compression</td>
<td>18.2±4.7</td>
<td>10.3±8.8</td>
</tr>
<tr>
<td>Decompression</td>
<td>21.8±9.4</td>
<td>20.1±5.6</td>
</tr>
</tbody>
</table>

ACD indicates active compression-decompression; STD, standard; and CPR, cardiopulmonary resuscitation. Data were analyzed by directly measuring the maximum and minimum pressures during each intervention (peak pressure analysis) and by measuring the mean pressures during the compression and decompression phases of the CPR cycle (mean pressure analysis) (n=21 patients).

(P=.02) higher, respectively, with ACD CPR versus standard CPR. The higher coronary perfusion pressures observed during the first third of the CPR cycle were secondary to higher arterial pressures in the ACD group, whereas the higher perfusion pressures during the final third of the cycle are probably secondary to lower right atrial pressures.

In conjunction with the hemodynamic parameters, a number of respiratory parameters were also measured. Ventilation per CPR cycle, measured with a spirometer during VF when the patient was apneically oxygenated (FiO2=100%), was nearly twofold greater with ACD CPR than with standard CPR (Table 4). When expressed as minute ventilation, 7.8±5.3 L/min was measured during standard CPR versus 13.5±5.5 L/min measured with ACD CPR (P<.001). After a mean of 80 mL is subtracted for respiratory dead space (average dead space in an intubated patient is 80 to 100 mL29), ventilation is 5 times greater with ACD CPR than with standard CPR (7.2 L/min versus 1.4 L/min, respectively).

In addition to minute ventilation, we also measured maximum positive expiratory and negative inspiratory
pressures (Table 4). There were no significant differences between the two methods with respect to the positive expiratory pressures generated during compressions (17.0±16.8 mm Hg with ACD versus 18.0±15.3 mm Hg with standard CPR, *P*=.51). In contrast, the negative inspiratory pressures generated were more than 14-fold greater with ACD CPR than with standard CPR (−11.4±6.3 versus −0.8±4.8 mm Hg, respectively, *P*<.04). Negative inspiratory pressures and minute ventilation were the two parameters in which the greatest differences between the two methods of CPR were observed.

In an effort to maintain a constant compression force with both CPR techniques, we used the compression gauge on the CardioPump™ and visual inspection during standard CPR to achieve a compression depth of 1.5 to 2.0 inches. We also measured esophageal pressures in five patients, distal intratracheal pressures in five additional patients, and both parameters in one patient during the compression phase using these indirect measurements of intrathoracic pressure when standard CPR was compared with ACD CPR (Table 4). There was an increase in intrathoracic pressure of 7.9±2.4 mm Hg with standard CPR using esophageal manometry versus 8.1±3.6 mm Hg with ACD CPR. Results with the intratracheal pressures were similar. It is important to note that in some patients, simply placing our hands or the suction device in position to perform CPR increased the intrathoracic pressure as much as 7 mm Hg. This was particularly striking for the patient who had undergone a recent thoracotomy. In general, patients with thinner chests demonstrated this increase in intrathoracic pressure when the CPR performers’ hands were laid on the chest. In view of this observation and of the potential implications related to changes in right atrial pressures, all measurements of intrathoracic pressure using esophageal manometry were calculated using a baseline value obtained after the CPR performers’ hands or the ACD device was resting on the chest. There

**Fig 5.** Tracings showing simultaneous arterial, right atrial, coronary perfusion, and intratracheal pressures obtained 5 minutes apart during apneic respiration (FIO₂=100%) (see text for details). STD indicates standard; ACD, active compression-decompression; and CPR, cardiopulmonary resuscitation.

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**Table 3. Comparison of Coronary Perfusion Pressures in STD and ACD CPR**

<table>
<thead>
<tr>
<th></th>
<th>Whole Cycle</th>
<th>First 1/3</th>
<th>Second 1/3</th>
<th>Third 1/3</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACD CPR, mm Hg</td>
<td>22.6±9.3</td>
<td>21.3±9.6</td>
<td>23.6±9.2</td>
<td>22.9±10.0</td>
</tr>
<tr>
<td>STD CPR, mm Hg</td>
<td>19.3±8.4</td>
<td>16.9±10.3</td>
<td>21.5±8.2</td>
<td>19.3±8.8</td>
</tr>
<tr>
<td><em>P</em></td>
<td>.02</td>
<td>.03</td>
<td>.07</td>
<td>.02</td>
</tr>
</tbody>
</table>

STD indicates standard; ACD, active compression-decompression; and CPR, cardiopulmonary resuscitation. Whole cycle refers to the mean coronary perfusion pressure over an entire CPR cycle. The mean coronary perfusion pressures were also calculated for each portion of the CPR cycle after the cycle was divided into equal thirds.
were no significant adverse effects from either method of CPR during the study. All patients survived without any significant complications. There were no rib fractures in either group as determined by postoperative chest radiographs, and no internal organ injury occurred. The one patient who had undergone a coronary bypass 3 weeks earlier experienced a mild degree of sternal discomfort, which resolved over the course of several days. The suction device worked well in all but one case. It is not clear why suction was poor in this 65-year-old woman, since she did not have unusually large breasts or a chest deformity.

**Discussion**

The purpose of this study was twofold: first, to develop a well-controlled model of VF in humans to evaluate the acute ventilatory and hemodynamic effects of cardiopulmonary resuscitation and second, to use this model to compare standard CPR with ACD CPR. The patients studied were similar to those who suffer from out-of-hospital cardiac arrest. Many had a history of aborted sudden death, and all had malignant ventricular arrhythmias. The results from this study demonstrate that anesthetized patients receiving transvenous leads during placement of implantable cardioverter/defibrillators provide a reproducible model to assess cardiovascular and pulmonary function during the first 1 to 2 minutes of VF. In addition, the results demonstrate that ACD CPR improves coronary perfusion pressures and minute ventilation compared with standard CPR in patients with VF.

**The Model**

Measurement of cardiopulmonary function immediately after the onset of VF is routinely studied in animal models to assess the effects of CPR but has been more difficult to accomplish in humans. Acute studies describing the importance of “cough CPR” in patients in VF during cardiac catheterization has led to widespread support of the “thoracic pump” mechanism of CPR.30,31 Miller et al31 described one patient in VF who was able to maintain consciousness up to 90 seconds by repeatedly coughing. Although intrathoracic pressures were not measured, it was presumed that rapid increases and decreases in intrathoracic pressure resulted in increased systemic blood pressure. With this notable exception, most studies in humans have been performed after a more prolonged arrest, at a time well past the critical window for long-term meaningful survival.24,5,16-20 Nonetheless, these studies have provided the most important information to date related to the complex mechanisms underlying CPR in humans. The present study describes a new model of acute VF. In addition to providing an opportunity to study CPR in a controlled setting in the same patient population as those susceptible to sudden cardiac death, this model affords the opportunity to safely compare different types of CPR in the same patient during repetitive inductions of VF, a standard part of the cardioverter/defibrillator implantation procedure. Furthermore, the potential usefulness of this model goes beyond the measurement of cardiopulmonary function, as in the present study. A number of other important pathophysiological processes can be readily studied during VF by using a variety of additional intraoperative techniques, including transesophageal manometry, transcranial and transthoracic Doppler ultrasonography, and electroencephalography.

The results of this study demonstrate that CPR improves systemic arterial blood pressures during VF in patients undergoing cardioverter/defibrillator implantation. During VF, arterial blood pressure increased significantly with either method of CPR compared with no CPR at all. Use of either method of CPR may be particularly beneficial in patients with significant reversible ischemia who undergo multiple inductions of VF during the implantation procedure.32

One potential concern with this acute model is the short duration of VF. However, within 20 seconds after the onset of VF, the arterial pressures reached a new and stable level. The effect of CPR was dramatic at this point, and differences were consistently observed when the different CPR techniques were compared, despite the short time frame. Two additional observations further support the relevance of the acute model: (1) with up to 2 minutes of CPR in several patients who were difficult to defibrillate, the hemodynamic response to CPR remained constant; and (2) the hemodynamic effects observed with acute VF and CPR were similar to those previously reported in patients with prolonged VF.18,28

The difference between arterial and right atrial pressures was used in our studies as an estimate of coronary perfusion pressure.20,24-26 During VF and before CPR, there was always a positive coronary perfusion gradient, which ranged from a low of 8 to a high of 37 mm Hg. By measuring the maximum and minimum pressure values during the compression and decompression phases of standard CPR, we found that coronary perfusion was significantly greater during the decompression phase (20.1±5.6 mm Hg) than during the compression phase (10.3±8.8 mm Hg, P<.001) (Table 2). We extended this analysis by calculating the mean arterial, right atrial, and coronary artery perfusion pressures over the CPR cycle as a whole and at the aforementioned one-third intervals (Tables 2 and 3). This more comprehensive analysis provided further insight into the mechanism of forward blood flow throughout the CPR cycle. We observed the largest coronary perfusion gradient during the middle third of the standard CPR cycle, with positive but smaller gradients during the first and last thirds of the CPR cycle. It is noteworthy that despite marked increases in arterial pressure with standard CPR compared with no CPR, there was only a minimal

**TABLE 4. Ventilation and Esophageal Parameters**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>ACD CPR</th>
<th>STD CPR</th>
<th>P</th>
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<tbody>
<tr>
<td>Minute ventilation, L/min (n=7)</td>
<td>13.5±5.5</td>
<td>7.8±5.3</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Negative inspiratory pressure, mm Hg (n=6)</td>
<td>-11.4±6.3</td>
<td>-0.8±4.8</td>
<td>&lt;.04</td>
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<tr>
<td>Positive expiratory pressure, mm Hg (n=6)</td>
<td>17.0±16.8</td>
<td>18.0±15.3</td>
<td>.51</td>
</tr>
<tr>
<td>Esophageal manometry, mm Hg (n=6)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Compression</td>
<td>8.1±3.6</td>
<td>7.9±2.4</td>
<td>.86</td>
</tr>
<tr>
<td>Decompression</td>
<td>-5.9±2.7</td>
<td>-3.6±3.5</td>
<td>&lt;.02</td>
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</table>

ACD indicates active compression-decompression; CPR, cardiopulmonary resuscitation; and STD, standard.
increase in coronary perfusion pressure during the middle third of the CPR cycle. These results are similar to those of Paradis et al., who demonstrated the greatest positive coronary perfusion gradient immediately after chest compression. In this study, we measured coronary perfusion pressure, calculated as the difference between the arterial and right atrial pressures, during VF. Ideally, we would have measured aortic rather than radial artery pressures. However, since patients could not be safely anticoagulated to prevent thrombus formation on central aortic catheters, we relied on radial artery measurements. Although this was a potential limitation, since radial artery pressures may be 5 to 10 mm Hg lower than central aortic pressures, a recent study has shown that these pressures are similar, even when large doses of catecholamines are administered concurrently. Use of either arterial or aortic pressures in this simplified formula provides an indirect yet commonly used assessment of coronary perfusion pressure.

**Comparison of Standard and Active Compression-Decompression CPR**

Using this acute model, we compared the effectiveness of standard CPR and ACD CPR. The results demonstrated that systemic arterial pressures and coronary artery perfusion pressures were consistently higher with ACD CPR than with standard CPR. The increase in coronary perfusion pressure with ACD CPR was observed after the first CPR cycle, most likely secondary to the earlier rise in arterial blood pressure that occurred consistently with ACD CPR. The increase in coronary perfusion was observed throughout the CPR cycle. The highest coronary perfusion gradients with ACD CPR were observed during the middle third of the CPR cycle, at the time of maximum active decompression, and during the same portion of the CPR cycle when standard CPR generates the greatest coronary perfusion pressure. However, compared with standard CPR, the greatest hemodynamic benefit from active decompression was observed in both the first and the last thirds of the CPR cycle. We believe that improvements in coronary perfusion with ACD CPR observed during the last third of the decompression phase are secondary to greater compliance of the venous bed, which results in a larger arterial:venous gradient, whereas improvements observed during the compression phase of the CPR cycle result from increased cardiac output secondary to active transport of venous blood into the chest with active decompression.

Support for this hypothesis can be found in the analysis of measurements of inspiratory and expiratory pressures, minute ventilation, and intrathoracal and intraesophageal pressures measured during standard CPR and ACD CPR. ACD CPR was associated with a marked increase in movement of gases into and out of the chest compared with standard CPR (Table 4). With the chest transformed into an active bellows, active decompression of the chest resulted in a more than fivefold net increase in movement of gases into the chest, assuming that the dead space in an intubated patient averages about 80 to 100 mL. This was accomplished by a significant decrease in intrathoracic pressures. With passive relaxation of the chest during standard CPR, the intrathoracic pressures measured at the tip of the endotracheal tube were $-0.8 \pm 4.8$ mm Hg during the relaxation phase, whereas with active decompression, the pressure decreased significantly further, to $-11.4 \pm 6.3$ mm Hg. Similar trends were observed by measuring intraeosophageal pressures. These changes were reflected in changes in right atrial pressures that were consistently lower during the final third of the CPR cycle with ACD CPR than with standard CPR. Although we did not measure blood flow during the different portions of the CPR cycle, the greater negative intrathoracic pressures generated by active decompression of the chest, together with the increase in minute ventilation associated with active decompression of the chest, both support the hypothesis that venous return is augmented with ACD CPR. Thus, the overall potential improvement in ACD CPR compared with standard CPR stems from a significant improvement in ventilation as well as an increase in cardiac output and coronary perfusion.

In this study, compression depth was not quantitatively standardized. Appropriate depth of compression was confirmed by visual inspection of the chest. Three different individuals performed CPR throughout the course of the study to try to avoid operator bias. In addition, the pressure gauge on the CardioPump™ was designed specifically to guide the operator in delivering constant compression and decompression. Equally important, when we measured intrathoracic pressures indirectly using either intrathoracal, intraesophageal, or right atrial systolic pressures, the increases in intrathoracic pressures during the compression phase were similar with both CPR techniques. Although none of these parameters are direct measures of intrathoracic pressure, they reflect changes in thoracic pressures.

Whether the changes in coronary artery pressure, intrathoracic pressure, and minute ventilation observed during our study fit best with the "thoracic pump" or the direct cardiac compression model of CPR remains uncertain. We speculate that both mechanisms are operative and that chest wall compliance with ACD CPR is the principal factor underlying which mechanism is more important in a given patient. Previous reports have observed two different types of hemodynamic response during standard CPR: the majority of patients have a positive coronary artery perfusion pressure only during the decompression phase, whereas a minority of patients have a positive gradient during both the compression and the decompression phases. We observed that some patients had an increase in intrathoracic pressure as soon as we placed our hands on the chest in preparation for standard CPR. This increase in intrathoracic pressure ranged from 0 to 7 mm Hg. Such differences in chest wall compliance may predict the predominant mechanism of forward blood flow in any given patient.

After the report of multiple successful resuscitations of a patient with a common household plumber's helper, several studies using ACD CPR have been performed that support our conclusions that increased coronary perfusion with ACD CPR is secondary to higher arterial pressures with compression and lower right atrial pressures with decompression. Studies in humans after prolonged resuscitation attempts have demonstrated improvements in arterial blood pressure, minute ventilation, and end-tidal CO2 when results from ACD CPR were...
compared with standard and mechanical thumper CPR. Studies in animals have further supported the importance of the decrease in negative intrathoracic pressures by demonstrating that with active chest wall decompression in porcine and canine models of VF, there were higher arterial pressures during the compression phase, lower right atrial pressures during the decompression phase, and an overall increase in cardiac output as determined by radiolabeled microspheres. The indirect measurements of intrathoracic pressure in the present study, using intratracheal and intraesophageal pressures, represent the first attempts to quantify the changes in intrathoracic pressure generated with ACD CPR in any model of VF. In addition, this is the first report of coronary perfusion pressures in humans using ACD CPR. Taken together, these studies support the conclusion that active chest compression and decompression during VF help to “prime the pump” before each subsequent chest compression and thus improve overall vital organ perfusion. These results suggest that future efforts aimed at improving coronary perfusion with ACD CPR should be directed toward maximizing coronary artery vasodilation and arterial pressure while minimizing central venous pressure, perhaps with a combination of a vasopressor and nitroglycerin.

The results from this study provide insight into the mechanisms of ACD CPR in humans and add further support to the hypothesis that there is a significant improvement in cardiopulmonary function with ACD CPR over standard CPR. However, the most important test of the utility of this technique will be whether or not its application can improve the rate of return of spontaneous circulation in victims of cardiopulmonary arrest. Investigations are currently under way to study this question.

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