Improving Active Compression-Decompression Cardiopulmonary Resuscitation With an Inspiratory Impedance Valve

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Background Active compression-decompression (ACD) cardiopulmonary resuscitation (CPR) has recently been demonstrated to provide significantly more blood flow to vital organs during cardiac arrest. To further enhance the effectiveness of this technique, we tested the hypothesis that intermittent impedance to inspiratory gas exchange during the decompression phase of ACD CPR enhances vital organ blood flow.

Methods and Results ACD CPR was performed with a pneumatically driven automated compression-decompression device in a porcine model of ventricular fibrillation (VF). Nine pigs were randomized to receive ACD CPR alone, while 8 pigs received ACD CPR plus intermittent impedance to inspiratory gas exchange with a threshold valve set to 40 cm H_2O. Results comparing 2 minutes of ACD CPR alone versus ACD CPR with the inspiratory impedance threshold valve (ITV) revealed significantly higher mean (±SEM) coronary perfusion pressures (diastolic aortic minus diastolic right atrial pressures) in the ITV (31.0±2.3 mm Hg) group versus with ACD CPR alone (21±3.6 mm Hg) ($P<.05$). Total left ventricular and cerebral blood flows, determined by radiolabeled microspheres, were 0.77±0.095 and 0.47±0.06 mL/min per gram, respectively, with ACD CPR plus the ITV versus 0.45±0.1 and 0.32±0.016 mL/min per gram, respectively, with ACD CPR alone ($P<.05$). Similar improvements in the ITV group were observed after 7 minutes of ACD CPR. After 16 minutes of VF and 13 minutes of ACD CPR, 6 of 8 pigs in the ITV group were successfully resuscitated with less than three successive 150-J shocks, whereas only 2 of 9 pigs with ACD CPR alone were resuscitated with equivalent energy levels ($P<.02$). With up to three additional and successive 200-J shocks, all pigs in the ITV group and 7 of 9 pigs with ACD CPR alone were resuscitated ($P=.18$).

Conclusions Intermittent impedance to inspiratory flow of respiratory gases during ACD CPR significantly improves coronary perfusion pressures and vital organ blood flow and lowers defibrillation energy requirements in a porcine model of VF. (***Circulation*****. 1995;91:1629-1632.)**

Key Words • cardiopulmonary resuscitation • fibrillation

Active compression-decompression (ACD) cardiopulmonary resuscitation (CPR) has recently been described as a method to improve vital organ blood flow and the currently poor resuscitation rates in patients suffering from cardiac arrest.[1] This new method increases blood flow to the heart and brain when compared with other methods of CPR by enhancing the "bellows-like" action of the chest.[1-5] However, the degree to which active decompression augments coronary perfusion pressure is dependent on a number of critical factors including the time between cardiac arrest and initiation of CPR, chest wall compliance, fluid status, myocardial size, ventricular wall compliance, and diaphragmatic tone. In previous studies of humans mechanically ventilated and in ventricular fibrillation (VF), we observed that intrathoracic pressure was significantly lower when the endotracheal tube was temporarily occluded during ACD CPR.[2] Since greater negative intrathoracic pressures during the decompression phase may enhance venous blood return to the thorax, we recently hypothesized that intermittent impedance to the inflow of respiratory gases during the decompression phase should further enhance the effectiveness of ACD CPR.[5] The purpose of the present investigation was to test this hypothesis by insertion of an inspiratory impedance threshold valve (ITV) in the respiratory circuit. This report describes the measurements of central aortic pressures, coronary perfusion pressures, vital organ blood flow with radiolabeled microspheres, arterial blood gases, and the rate of return of spontaneous circulation (ROSC) in a well-established porcine model of VF.

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Methods

The experiments described in this manuscript were approved by the Committee on Animal Experimentation at the University of Minnesota. Healthy female domestic farm pigs (28 to 33 kg) were fasted overnight and anesthetized with pentobarbital (20 mg/kg IV bolus followed by 2.5 mg/kg per hour IV infusion) via an ear vein. The surgical approach has been described previously.[3,4] Once anesthetized, pigs were placed in the dorsal recumbent position and intubated by standard endotracheal intubation technique. They were ventilated during the preparatory phase of the experiment and after ROSC at the end of the experiment with a mechanical respirator (model 607, Harvard Apparatus Co, Inc.). The tidal volume was set at 450 mL and delivered between 11 and 15 breaths per minute with supplemental oxygen at 2 L/min. Normal saline solution was administered intravenously through the preparative and study periods by an infusion pump (Flo-Gard 6201, Baxter Healthcare).

The preparatory phase, which included cannulation of both femoral arteries and the right jugular vein, as well as calibration

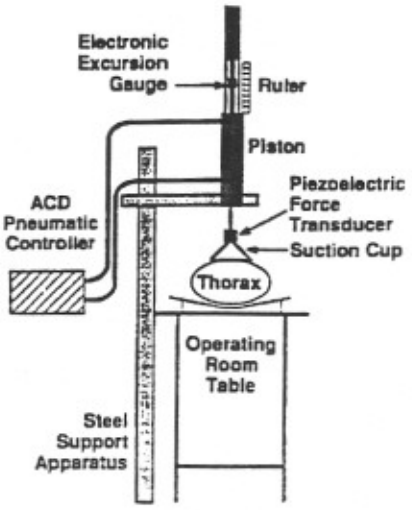

FIG 1. Schematic representation of the active compression-decompression (ACD) cardiopulmonary resuscitation suction apparatus showing position of the force transducer and excursion gauge in relation to the thorax.

of all instruments, took approximately 2 hours. Once venous access was obtained, animals received normal saline solution at approximately 300 to 400 mL/h to maintain diastolic right atrial pressures of 3 to 5 mm Hg. Arterial blood gases were analyzed (Instrumentation Laboratory SpA) every 30 minutes to ensure adequate acid base status and oxygenation. Left ventricular and ascending aortic arch blood pressures were monitored using a single high-fidelity micromanometer catheter (Millar). This aorto–left ventricular catheter had a lumen for injecting radiolabeled microspheres and it was positioned, under fluoroscopic guidance, 15 minutes before initiation of VF. Right atrial pressures were also monitored using a micromanometer catheter (Millar) inserted through a right jugular vein sheath. The micromanometer catheters were calibrated to atmospheric pressure immediately before they were inserted into the pig. A 5F bipolar pacing catheter (Daig, Inc), used to induce VF with alternating current at 7 V and 60 Hz, was inserted through a second right jugular vein sheath and positioned by fluoroscopy in the right ventricular apex. For withdrawal of reference blood samples to measure organ blood flow, a 7F catheter was advanced by femoral arterial access to the aortic arch. Body temperature was monitored continuously via a rectal probe (Yellow Springs Instrument Co). Core temperatures were maintained between 36.5 and 38.5°C with a heating pad. Five minutes before induction of VF, 5000 U IV of sodium heparin was administered.

ACD CPR was performed with a 9.0-cm silicon suction cup positioned fluoroscopically over the right and left ventricles. It was attached to a pneumatically driven automatic piston device for ACD CPR (ACD Controller, Ambu International) as previously described.[3] The automatic piston device was modified such that it was attached to the base of the surgical table with a rigid steel pole (diameter=7 cm) as shown in Fig 1. Compression and decompression forces were measured continuously using a piezoelectric force transducer (model 208A02, PCB Piezotronics) mounted directly in the force path between the plunger cup and piston arm of the automatic compression-decompression device. Compression and decompression excursion was measured continuously by the voltage output of a linear variable differential transformer (model 3000 HR-DC, Lucas Schaevitz DC-LVDT). The transformer core moved orthogonally to the chest wall such that voltage output was linearly proportional to distance traveled above and below the neutral chest wall position. The compression depth was also monitored visually with a ruler attached to the piston housing. Both compression depth and force were monitored on a chart paper recorder (ES 1000, Gould Instrument Systems) and digitized on-line (SUPERSCOPE II v1.295, GW Instruments) with a computerized recording system (Power Macintosh 7100/66 computer) to allow for instantaneous feedback to the operators.

Digitized data were analyzed electronically to provide hemodynamic measurements. The compression-decompression phases were divided in half by using the point at which the suction cup passed the neutral position at the beginning of compression and the beginning of active decompression. Coronary perfusion pressure was calculated during systole and diastole and was defined as the aorto–right atrial pressure difference (time-coincident difference between aortic and right atrial diastolic pressures) as previously defined.[2-4]

The protocol was designed to compare ACD CPR alone with ACD CPR plus an inspiratory ITV. Before induction of VF, baseline hemodynamic and arterial blood gas measurements were obtained. Radiolabeled microspheres were injected into the left ventricle to determine baseline vital organ blood flows, as previously described.[4] VF was induced with a single 5-second application of alternating current. The endotracheal tube (ET Tube Hi-Lo Jet, Mallincrott, Inc) was immediately disconnected from the mechanical ventilator and the cuff pressure was assessed to ensure that it was adequate to seal the trachea. The animals were then randomized to receive either ACD CPR alone or ACD CPR with the ITV. After 3 minutes of VF, during which time no CPR or ventilation was performed, ACD CPR was performed with the automatic ACD device. The compression-decompression rate was 80 per minute with a 50% duty cycle, a depth of 25% of the anterior-posterior diameter of the chest wall, and a velocity of 7.5 in/s. Active decompression was performed to produce a sternal displacement of 10% greater than the resting anteroposterior diameter, for which a suction force of approximately 200 N was applied. Compression and decompression excursion were continuously monitored and adjusted with the control module, as necessary, during the experiment.

Ventilatory support and ACD CPR were performed simultaneously. During ACD CPR, ventilation was provided by manual bag ventilation (Ambu bag) with 10 L/min oxygen. Respirations were delivered as previously described[4] at a rate of one breath every five compressions with a constant tidal volume of approximately 500 mL. In the eight pigs randomized to the ITV group, respiratory gas exchange was limited by the inspiratory valve. In these experiments two 20 cm H_2O threshold valves (Ambu, Inc) were connected in series between the endotracheal tube and the Ambu bag such that during active decompression, but in the absence of a manual ventilation, the valves would open only after 40 cm H_2O of inspiratory pressure. In this manner, more than -40 cm H_2O of intrathoracic pressure was required for inspiration of respiratory gases during four of every five compression-decompression cycles.

Once either ACD CPR alone or ACD CPR plus the ITV was initiated, the same method was performed continuously for 13 minutes during VF. Arterial blood gases were measured immediately before VF and after 2 and 7 minutes of ACD CPR. Radiolabeled microspheres were injected into the left ventricle after 2 minutes and 7 minutes of CPR and after 15 minutes of ROSC. After 16 minutes of VF and 13 minutes of ACD CPR, defibrillation was attempted by transthoracic shock. Defibrillation (43100 A Defibrillator, Hewlett-Packard) was performed initially with a 150-J shock delivered immediately after the automatic ACD CPR machine was turned off. At this time the pig was reconnected to the mechanical ventilator. If the 150-J shock was not successful, two additional 150-J shocks were delivered within the next 15 seconds. Assessment of heart rate and arterial pressure was performed on a continuous basis to monitor for ROSC. If after three successive 150-J shocks spontaneous circulation was not restored, ACD CPR was performed for an additional 10 seconds before delivery of a 200-J shock. A total of three successive 200-J shocks were

normalizing blood flow during CPR to baseline blood flows on a per-animal basis, there was a statistically significant improvement in myocardial and cerebral blood flow in animals treated with the ITV (Fig 2).

ROSC was achieved in all 8 pigs with the ITV and 7 of 9 pigs treated with ACD CPR alone. However, 6 of 8 pigs in the ITV group and only 2 of 9 pigs with ACD CPR alone had an ROSC after one to three 150-J shocks ($P<.03$). In animals that had an ROSC, blood flow to the vital organs was similar after 15 minutes between groups. In the ITV group (n=8), left ventricular and cerebral blood flows were 2.1±0.42 and 0.45±0.06 mL/min per gram, respectively, versus 2.7±0.40 and 0.37±0.03 mL/min per gram without the ITV (n=6). One pig in the ACD CPR alone group died within 10 minutes after ROSC.

Arterial blood gas data are shown in the Table. There were no statistically significant differences in arterial blood pH, Po_2, or Pco_2 after 2 minutes of ACD CPR. By 7 minutes of ACD CPR, the arterial pH and Po_2 levels were significantly lower in the ITV group.

Discussion

Return of venous blood into the thorax is a fundamental and critical element of any effective method of CPR. Closed-chest manual cardiac massage, ie, standard CPR, relies on the natural elasticity of the chest to generate a transient period of negative intrathoracic pressure immediately after maximal compression. ACD CPR augments this process by increasing both the duration and extent of intrathoracic pressure during the decompression phase of CPR.[2-4] The results of the present study demonstrate that insertion of an inspiratory ITV into the respiratory circuit during performance of ACD CPR significantly improves vital organ blood flow and the chances of ROSC with defibrillation when compared with ACD CPR alone. If inflow of respiratory gases is limited with an inspiratory ITV, equilibration of the negative intrathoracic pressure generated by active chest wall expansion occurs to a greater extent secondary to enhanced venous return. Both hemodynamic pressure measurements and myocardial blood flow data support this conclusion. On the basis of the pathophysiology involved, we speculate that the use of a threshold valve should also benefit other types of CPR, including standard CPR and "vest" CPR.

Relatively little is known about the effects of ACD CPR on lung function. We have previously observed that ACD CPR alone significantly enhances minute ventilation compared with standard CPR.[1,2] No evidence of lung damage was found in either group at autopsy. A theoretical disadvantage of a very negative intrathoracic pressure during active decompression is negative pressure pulmonary edema. However, this was not observed grossly and is generally observed only with negative inspiratory pressures greater than -50 cm H_2O.

Insertion of the ITV during ACD CPR eventually leads to lower pH and Po_2 values. When minute ventilation was decreased with the ITV, progressive acidosis occurred to a greater extent when compared with ACD CPR alone. However, these values remained within normal physiological limits throughout the 16 minutes of VF and ACD CPR when supplemental O_2 was used. Although the blood pH was >7.3 in both groups after 16 minutes of VF and pH differences did not alter the chances for successful defibrillation with lower energy requirements in the ITV group, it will be important to assess whether the observed decreases in Po_2 and pH are significant in the clinical setting.

Venous blood flow is increased clinically by the Mueller maneuver, a technique in which inspiration is performed when the trachea is simultaneously occluded by the epiglottis.[6] By using a mechanical valve in an analogous physiological fashion during ACD CPR, we demonstrated in this study that blood flow to the vital organs and the chances for ROSC with defibrillation were significantly increased. Additional mechanisms may also contribute to these improvements. On the basis of these favorable results, the potential benefits of intermittent impedance of inspiratory gas exchange during ACD CPR should be studied in patients in cardiac arrest.

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References

1. Cohen TJ, Tucker KL, Lurie KG, Redberg RF, Dutton JP, Dwyer KA, Schwab TM, Chin MC, Gelb AM, Scheinman MM, Schiller NB, Callaham ML. Active compression-decompression: a new method of cardiopulmonary resuscitation. *JAMA*. 1992;267:2916-2923.
2. Shultz JJ, Coffeen P, Sweeney M, Detloff B, Kohler C, Pineda E, Yakshe P, Adler SW, Chang M, Lurie KG. Evaluation of standard and active compression-decompression CPR in an acute human model of ventricular fibrillation. *Circulation*. 1994;89:684-693.
3. Lindner K, Pfenninger E, Lurie K, Schurmann W, Lindner I. Effects of active compression-decompression resuscitation on myocardial and cerebral blood flow in pigs. *Circulation*. 1993;88:1254-1263.
4. Chang MW, Coffeen P, Lurie KG, Shultz J, Bache RJ, White CW. Active compression-decompression CPR improves vital organ perfusion in a dog model of ventricular fibrillation. *Chest*. 1994;106:1250-1259.
5. Lurie KG. Active compression-decompression cardiopulmonary resuscitation: a progress report. *Resuscitation*. 1994;28:115-122.
6. Sharpey-Schafer EP. Effect of respiratory acts on the circulation. In: Hamilton WF, ed. *Handbook of Physiology*. Washington, DC: American Physiological Society; 1965;3:1883-1884.