External Cardiac Massage Using a Hand-powered Chest Compressor on Dogs with Ventricular Fibrillation

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We devised a hand-powered portable chest compressor for external cardiac massage. The purpose of this study was to assess the efficacy and safety of this device in comparison to manual chest compression in dogs with ventricular fibrillation. Five out of 7 dogs that received manual chest compression during cardiopulmonary resuscitation (CPR) were successfully resuscitated. Seven out of 8 dogs that received mechanical chest compression with this device during CPR were successfully resuscitated. There were no differences between the two methods in maximum arterial pressure at 1 and 10 min after the initiation of CPR. There was also no difference between the two methods in pulmonary arterial pressure or arterial and mixed venous blood gases during CPR. Minimum arterial pressure during CPR was higher in dogs receiving mechanical chest compression than those receiving manual chest compression. This study reveals that the hand-powered chest compressor is equally efficient for external cardiac massage as manual cardiac massage. Moreover, this device can be useful in a situation where manual compression has to be interrupted, such as during litter transport of the patient, and so on. (Key words: cardiac arrest, cardiopulmonary resuscitation, cardiac massage, transportation)

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Intermittent positive pressure ventilation (IPPV) and external manual chest compressions are standard resuscitative procedures for a patient with cardiac arrest[1]. However, effective cardiac massage may not be possible for patients with cardiac arrest when being transported through narrow passageways or stairs on a litter. This problem prompted us to devise a hand-powered portable chest compressor for external cardiac massage during transport (fig. 1)[2]. The purpose of this study was to assess the efficacy and safety of this device in comparison to the standard method of manual chest compression.

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Methods

Animal preparation

Fifteen healthy mongrel dogs were used in this study (9.8 to 13.4 kg). Each animal was anesthesized with 10 $mg \cdot kg^{-1}$ of intramuscular ketamine and 3 $mg \cdot kg^{-1}$ of intravenous diazepam, followed by a constant intravenous infusion of 40 $mg \cdot hr^{-1}$ ketamine and 1.6 $mg \cdot hr^{-1}$ pancuronium bromide. The

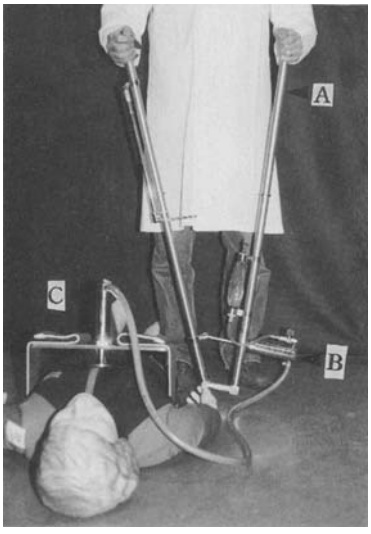

Fig. 1. Overview of the Hand-powered chest compressor with hydrolic cylinder piston. The piston of the sternal compressor moves synchronously with another cylinder piston incorporated between two manual levers with a pivot at the top.

A: the levers, B: cylinder piston, C: piston of sternal compressor and compression pad.

animals were restrained in the supine position on a V-shaped board. The abdomen was stretched with a wide band to limit caudal movement of the diaphragm and to prevent dissipation of the intrathoracic pressure during CPR. An endotracheal tube with an inflatable cuff (Blue Line Tracheal Tube 9.0 mm ID, Portex) was inserted and the animals were ventilated by a preset-volume type ventilator (KMA-1300, Acoma Medical Co.) with room air and a tidal volume of 200 ml at a rate sufficient to maintain Pa_{CO_2} at 35 to 45 mmHg.

A femoral vein cannula for intravenous drug administration, and a femoral artery cannula for arterial blood pressure monitoring and obtaining arterial blood samples were inserted. A Swan-Ganz thermodilution catheter (Edwards Laboratories Inc. 93A-131F-7F) was inserted via the right jugular vein for monitoring pulmonary arterial pressure and obtaining pulmonary arterial blood samples. The femoral arterial line and pulmonary arterial line were both connected to a pressure transducer (P23ID, Gould Stathan) and were continuously recorded on a polygraph (RM-6200, Nihon Kohden). The left jugular vein was surgically isolated to insert a wire electrode to induce ventricular fibrillation (VF). Heparin (1 $mg \cdot kg^{-1}$ iv) was administered to prevent clot formation in the catheters and to reduce intravascular coagulation during circulatory arrest. Lead II of the ECG was monitored continuously.

Airway pressure was monitored through a catheter (2.2 mm ID) inserted 10 cm into the endotracheal tube through a side port of the tube connector and was then connected to a pressure transducer (TP-200T, Nihon Kohden) for continuous recording on a polygraph. Body core temperature was monitored with a rectal thermometer (MGA-III, Nihon Kohden) and maintained within the normal range. Arterial blood gases and pH were measured with a Corning 170 pH/blood gas analyzer.

Experimental protocol

The animals were randomly assigned to two groups. The control group received manual chest compressions and IPPV. The other received mechanical chest compression using the hand-powered portable chest compressor and IPPV.

After femoral and pulmonary arterial blood samples were drawn for baseline values, VF was induced by a 15 volt alternating current delivered into the right ventricular endocardium. VF was confirmed by ECG and a sudden decrease in arterial blood pressure to less than 25 mmHg.

The dogs received no therapy for 1 min. An intravenous injection of epinephrine 0.5 mg was then given. Simultaneously, chest compression by either method at a rate of $92 \cdot min^{-1}$ and IPPV with an FI_{O_2} of 1.0 were initiated. A tidal volume of 200 ml and a respiratory rate of $18 \cdot min^{-1}$ were used for the IPPV. The chest compression rate was maintained using a metronome and the sternum was depressed between 3 and 5 cm. A constant infusion of epinephrine, 0.001 $mg \cdot kg^{-1} \cdot min^{-1}$, was started and increased, if necessary, to maintain a maximum arterial pres-

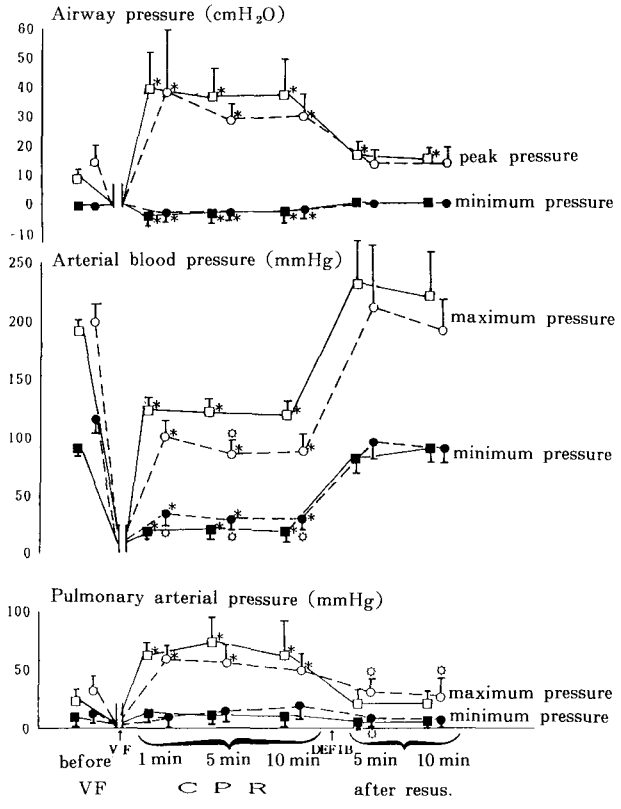

Fig. 2. Changes in airway pressure, femoral arterial blood pressure, and pulmonary arterial pressure before VF, during CPR, and after resuscitation.

The mean ± SE are shown. Significantly different from the value of the control group, ☼ $P < 0.05$; Significantly different from the value before VF, * $P < 0.05$.

□ or ■: control group, ○ or ●: mechanical group.

sure greater than 100 mmHg during CPR.

After 10 min of CPR, a nonsynchronized 100-Joules defibrillation shock was administered to the anterior chest using a direct-current defibrillator (MDV-2, Nihon Kohden). If a viable cardiac rhythm was not produced, a second and third defibrillation shock of 200 and 300-Joules was administered. Before each shock, manual or mechanical chest compression was performed for at least 30 seconds. A bolus 0.5 mg intravenous epinephrine was administered before each defibrillation shock. Resuscitative therapy was discontinued if the third defibrillation shock failed to restore a viable cardiac rhythm. The resuscitated animals were observed for 10 min while maintaining IPPV. No changes in the respirator settings were made during the whole procedure.

Femoral and pulmonary arterial blood was taken for blood gas analysis and hemodynamic parameters were determined before VF, at 1, 5, and 10 min during CPR and at 5, and 10 min after resuscitation. An autopsy was performed on all animals. The resuscitated animals were sacrificed for autopsy with intravenous potassium chloride (10 mEq). The paired Student t-test was used for comparison with values before VF (base-line values), and previous values. The unpaired t-test was used for comparison between the two groups. A value of $P < 0.05$ was considered significant.

Results

Circulatory restoration was obtained in five of seven dogs who received manual chest compression along with IPPV (control group) and in seven of eight dogs who received mechanical chest compression along with IPPV (mechanical group).

Figure 2 shows the changes in the peak and minimum airway pressures, maximum and minimum femoral arterial pressures, and maximum and minimum pulmonary arterial pressures before VF, during CPR, and af-

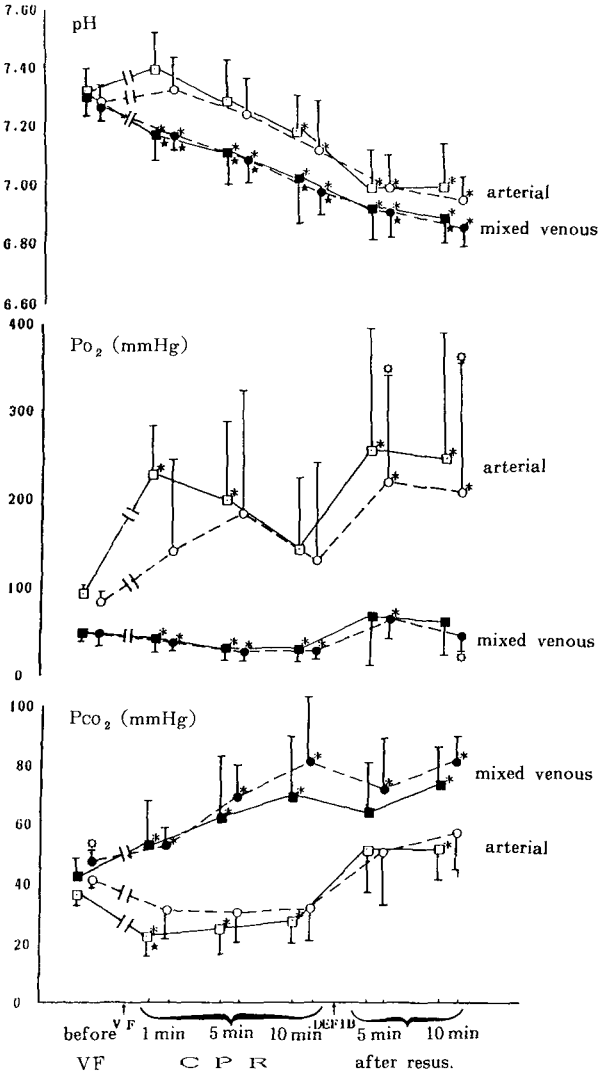

Fig. 3. The changes in arterial (□ or ○) and mixed venous (■ or ●) pH, P_{O_2} and P_{CO_2} before VF, during CPR and after resuscitation.

The mean ± SD are shown. Significantly different from the value of the control group, ☼$P < 0.05$; Significantly different from the value before VF, $*P < 0.05$; Significantly different from the previous value, ★$P < 0.05$.

□ or ■: control group, ○ or ●: mechanical group.

ter resuscitation for both groups. The peak airway pressure and maximum pulmonary artery pressure significantly increased in both groups after the initiation of CPR. The maximum arterial pressures in both groups were maintained around 100 mmHg during CPR. There was no significant difference between the two groups in maximum femoral arterial pressure at 1 and 10 min after the initiation of CPR. There was no significant difference between the two groups in terms of peak and minimum airway pressure, or maximum and minimum pulmonary artery pressure during the course of CPR. The minimum femoral arterial pressure in the mechanical group was significantly higher than those of the control group during the course of CPR. The amount of epinephrine used during CPR was 1.43 ± 0.45 mg in the mechanical group and 1.55 ± 0.54 mg in the control group. There was no significant difference between the two groups in the amount of epinephrine.

Figure 3 shows the changes in arterial and mixed venous pH, P_{O_2}, and P_{CO_2} before VF, during CPR, and after resuscitation for both groups. The arterial and mixed venous pH values decreased gradually in both groups during CPR and after resuscitation. The arterial P_{O_2} values in both groups were

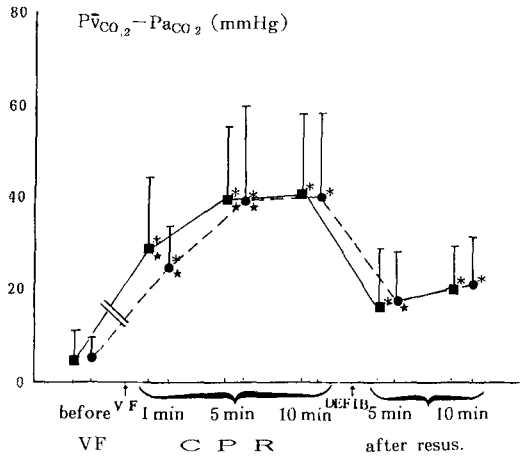

Fig. 4. The change in Venous-arterial P_{CO_2} gradient ($P\bar{v}_{CO_2}$-Pa_{CO_2}) before VF, during CPR and after resuscitation.

The mean ± SD are shown. Significantly different from before VF, $*P < 0.05$; Significantly different from the previous value, $\bigstar P < 0.05$.

■: control group, ●: mechanical group.

maintained greater than 100 mmHg during the course of CPR. The mixed venous P_{O_2} values decreased significantly in both groups after the initiation of CPR. The arterial P_{CO_2} values in the control group and in the mechanical group significantly decreased after the initiation of CPR but there was no significant difference between the two groups. After resuscitation, the arterial P_{CO_2} values in both groups tended to increase. The mixed venous P_{CO_2} values in both groups increased significantly after the initiation of CPR and were maintained at high levels even after resuscitation. However, there were no significant differences between the two groups in arterial and mixed venous pH, P_{O_2} and P_{CO_2}. Figure 4 shows the venous-arterial P_{CO_2} gradient for both groups. The venous-arterial P_{CO_2} gradient in both groups increased significantly after the initiation of CPR, but this decreased after circulatory restoration. However, there was no significant difference between the two groups in the venous-arterial P_{CO_2} gradient before, during and after CPR.

Autopsy revealed no major visceral injuries. However, one had mild spotty hemorrhage in the apex of the heart, and the other had the same condition in the posterior wall of the heart in the mechanical group.

Discussion

Various emergency cardiac care systems have been recently organized to reach out-of-hospital cardiac arrest victims promptly and to provide definitive management. The outcome of CPR depends on the length of time prior to the initiation of CPR. Eisenberg et al.[3,4] showed that survival rate was higher in the patients who received CPR within 4 min and definitive care within 8 min after cardiac arrest. The objective of CPR is to maintain oxygenation of the brain and other vital organs until satisfactory cardiopulmonary function is restored. In order to have successful resuscitation without brain damage, CPR should not be interrupted. However, discontinuation of CPR is unavoidable during transportation up or down a flight of stairs, through narrow passageways and certain out-of-hospital situations. No devices are currently available to continue effective CPR in these situations. The external cardiac massage chest compression and lung ventilation device, Thumper (Michigan Instruments, Inc., MI), could be used for this purpose[5]. But, this device requires compressed air or oxygen as a power source and is too heavy to carry with a victim during an emergency situation. A simple and light weight device is desirable for this purpose. Our hand-powered portable chest compressor requires no compressed air or oxygen as a power source and weighs only about 5.0 kg making it practicable to carry during the emergency situations[2].

We were able to show in this study that this mechanical chest compressor maintained adequate maximum arterial pressure during CPR in animals. The minimum arterial pressure with the mechanical chest compressor was higher than that with manual chest compressions. The difference of the arterial pressure and the minimum right arterial pressure represents the coronary perfusion pressure. Sanders et al.[6] suggested that maintainance of an adequate minimum arterial pressure

during CPR appeared to be important for successful resucitation of the experimental dogs. They suggested that it was necessary to maintain a diastolic blood pressure above 30 mmHg for successful restoration of spontaneous circulation. Paradis et al.[7] measured the maximal coronary perfusion pressure during CPR in humans with cardiac arrest. They found that the coronary perfusion pressure was 25.6 ± 7.7 mmHg in the successfully resuscitated group and 8.4 ± 10.0 mmHg in the unsuccessful group. We did not measure the right arterial diastolic pressure in this experiment. However, the higher minimum arterial pressure during mechanical chest compression implies that the coronary perfusion pressure may be higher in the mechanical group than in the manual group. The reason for the higher minimum arterial pressure in the mechanical group is not clear. Michael et al.[8] suggested that epinephrine increases myocardial perfusion by increasing aortic diastolic pressure. However, there was no significant difference in the amount of epinephrine administered in both groups.

In the present study, the mixed venous P_{CO_2} values in both groups increased significantly after the initiation of CPR and were maintained at high levels even after circulatory restoration. On the other hand, the venous-arterial P_{CO_2} gradient ($P\bar{v}_{CO_2}$-Pa_{CO_2}) in both groups increased after the initiation of CPR and decreased after circulatory restoration (fig. 4). These results are consistent with those of Weil et al.[9] and Grundler et al.[10].

Weil et al.[9] demonstrated that there was a striking increase in mixed venous P_{CO_2} in spite of the decline of arterial P_{CO_2} during CPR in humans with cardiac arrest, resulting in a marked increase in $P\bar{v}_{CO_2}$-Pa_{CO_2}. They suggested that this increase in mixed venous P_{CO_2} reflect critical decreases in the excretion of carbon dioxide due to the marked reduction of pulmonary blood flow during CPR. Grundler et al.[10] found that there was a marked widening in $P\bar{v}_{CO_2}$-Pa_{CO_2} during CPR in dogs with cardiac arrest and this widened $P\bar{v}_{CO_2}$-Pa_{CO_2} was restored after resuscitation. An increase in mixed venous P_{CO_2} may occur by the reduction of pulmonary blood flow or hypoventilation. But the widening in $P\bar{v}_{CO_2}$-Pa_{CO_2} can be produced by the reduction of pulmonary blood flow, but not by hypoventilation[11]. We look at this phenomenum as follows: Decreasing P_{CO_2} and increasing P_{O_2} in arterial blood during the course of CPR in both groups was enough to presume that the alveolar ventilation and pulmonary shunting blood are maintained near normal even if the total pulmonary blood flow decreased considerably. The increased P_{CO_2} in mixed venous blood probably is resulted from the bicarbonate-buffer system in buffering the aciditic substances originated from underperfused tissues. There was no significant difference in $P\bar{v}_{CO_2}$-Pa_{CO_2} during CPR between the two groups in the present study. This finding may suggest that pulmonary blood flow during CPR between the two groups was not different.

External chest compression is sometimes associated with serious complications like hemothorax, hemopericardium, and laceration of the liver, spleen, stomach, colon and inferior vena cava. We did not observe any of these serious complications attributable to the mechanical compressor in this study. However, we observed slight spotty hemorrhage of the heart in 2 dogs in the mechanical group. Babbs et al.[12] suggested that the heart of small dogs can be more directly compressed than that of large dogs during external cardiac compression. Small dogs (9.8–13.4 kg) were used for this study and a round, flat, and hard "compression pad", designed for the broad human chest, was used. As the chest is keel-shaped in dogs, the heart of the dogs might therefore have been compressed more strongly by the "pad" than during manual compression. This might be the explanation for the spotty hemorrhage seen in the two dogs in the mechanical group.

In conclusion, this study on dogs with VF demonstrated that a hand-powered portable chest compressor for external cardiac massage can maintain circulation during CPR

comparable to that of the standard manual method. There were no serious injuries due to the device. These results suggest that this device can be useful in a situation where manual compression has to be interrupted, such as during litter transport of the patient, and so on.

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References

1. Standard for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). JAMA 255:2905–2932, 1986
2. Morioka T, Sugiyama G, Kon Y, Sakai H, Takeshita J, Kishi H: A portable manual sternal compressor for cardiac massage of a patient under transportation. –1. Evaluation after trials and errors–. Masui (Jpn J Anesthesiol) 38:784–790, 1989
3. Eisenberg SM, Bergner L, Hallstrom A: Cardiac Resuscitation in the Community. JAMA 241:1905–1907, 1979
4. Cummins RO, Eisenberg MS: Prehospital cardiopulmonary resuscitation. JAMA 253:2408–2412, 1985
5. Tsitlik JE, Weisfeldt ML, Chandra N, Effron MB, Halperin HR, Levin HR: Elastic properties of the human chest during cardiopulmonary resuscitation. Crit Care Med 11:685–691, 1983
6. Sanders AB, Ewy GA, Taft TV: Prognostic and therapeutic importance of the aortic diastolic pressure in resuscitation from cardiac arrest. Crit Care Med 12:871–873, 1984
7. Paradis NA, Martin GB, Rivers EP, Goetting MG, Appleton MF, Nowak RM: Coronary perfusion pressure and the return of spontaneous circulation in human cardiopulmonary resuscitation. JAMA 263:1106–1113, 1990
8. Michael JR, Guerci AD, Koehler RC, Shi AY, Tsitlik J, Chandra N, Niedermeyer E, Rogers MC, Traystman RJ, Weisfeldt ML: Mechanism by which epinephrine augments cerebral and myocardial perfusion during cardiopulmonary resuscitation in dogs. Circulation 69:822–835, 1984
9. Weil MH, Rackow EC, Trevino R, Grundler W, Falk JL, Griffel MI: Difference in acid-base state between venous and arterial blood during cardiopulmonary resuscitation. N Engl J Med 315:153–156, 1986
10. Grundler W, Weil MH, Rackow EC: Arteriovenous carbon dioxide and pH gradients during cardiac arrest. Circulation 74:1071–1074, 1986
11. Adrogue HJ, Rashad MN, Gorin AB, Yacoub J, Madias NE: Assessing Acid-base status in circulatory failure. Engl J Med 320:1312–1316, 1989
12. Babbs CF, Tacker WA, Paris RL, Murphy RJ, Davis RW: CPR with simultaneous compression and ventilation at high airway pressure in 4 animal models. Crit Care Med 10:501–504, 1982