Long-term survival with open-chest cardiac massage after ineffective closed-chest compression in a canine preparation


ABSTRACT The ultimate goal of cardiopulmonary resuscitation (CPR) is long-term, neurologically intact survival. This study examined whether open-chest cardiac massage could improve 7 day survival and neurologic function when instituted after the failure of standard closed-chest compression CPR. Twenty-nine mongrel dogs were anesthetized and then instrumented with catheters to monitor right atrial and ascending aortic pressures. Ventricular fibrillation was induced and after 3 min standard CPR was begun. Standard CPR was performed with a Thumper programmed for 2 inch chest compressions at 60/min with a 50% duty cycle. External defibrillation was attempted twice after 15 min of ventricular fibrillation. Unsuccessfully defibrillated animals were randomly assigned to either an additional 2 min of continued closed-chest compressions, or 2 min of open-chest cardiac massage. All animals underwent a period of advanced cardiac life support and were followed until they were resuscitated or died. Follow-up care, including scoring of neurologic deficit, was performed for 7 days. In dogs receiving open-chest cardiac massage there was significantly more immediate resuscitation success (14/14 vs 5/14; p < .005), 24 hr survival (12/14 vs 4/14; p < .005), and 7 day survival (11/14 vs 4/14; p < .02) than in those receiving continued closed-chest compression. Open-chest cardiac massage significantly improved long-term outcome when instituted after 15 min of ineffective closed-chest compression. Circulation 75, No. 2, 498–503, 1987.

OPEN-CHEST cardiac massage is the treatment of choice for cardiac arrest associated with penetrating thoracic trauma, or cardiac arrest during an intrathoracic surgical procedure.1-4 However, the role of thoracotomy and open-chest cardiac massage after failure of initial closed-chest cardiopulmonary resuscitation (CPR) during nontraumatic cardiopulmonary arrest is controversial. Recent efforts to improve the treatment of cardiac arrest has rekindled interest in the use of the “emergency” thoracotomy.

Open-chest cardiac massage was compared with closed-chest compression during the 1960s and found to produce significantly more cardiac output.7-9 Recent comparisons have found that open-chest cardiac massage produces not only higher cardiac outputs, but also higher aortic systolic pressures, aortic diastolic pressures, mean aortic pressures, cerebral perfusion pressures, and carotid blood flow.10-12 Nevertheless, open-chest massage is recommended with hesitancy by the American Heart Association, and then only under very specific and unusual circumstances.13

A recent study in humans failed to show any survival benefit when open-chest massage was initiated 30 min after beginning closed-chest compression.14 However, animal studies have shown that open-chest cardiac massage is capable of improving success of short-term resuscitation when it is begun after 15 min, rather than 30 min, of closed-chest compression.15, 16 Survival and neurologic function at 24 hr after cardiac arrest have also been shown to be improved with open-chest cardiac massage in one other experimental study.17
It is not known whether open-chest cardiac massage can significantly improve long-term survival and neurologic function. Accordingly, this study was undertaken to determine whether open-chest cardiac massage could significantly improve 7 day survival and neurologic function when instituted after a reasonable attempt at standard closed-chest resuscitation had failed.

Methods

Animal preparation. All studies were performed under the guidelines of the American Physiologic Society and approval of the Animal Research Board at Purdue University was obtained. Twenty-nine large mongrel dogs (24.2 ± 0.7 kg) underwent a 2 week conditioning program to ensure good health and nutrition. All animals tested negative for heart worms by two separate methods. General anesthesia was induced with 2 mg/kg iv morphine sulfate and maintained with 1% to 1.5% halothane in oxygen. Under full anesthesia and by an aseptic technique in a sterile operating room, skin electrocardiographic (ECG) leads were placed, and a cutdown over the right femoral vessels was performed. Fluid-filled catheters were placed in the right femoral vein and right femoral artery. The catheters were connected to pressure transducers (Gould/Microswitch) and subsequently to a direct-inking physiograph recorder (Narco-Biosystems, Houston). The system was calibrated with a mercury manometer and to atmospheric zero. Under fluoroscopic and manometric guidance, these catheters were advanced to the right atrium and left ventricle, respectively. On completion of instrumentation and catheter positioning, halothane was discontinued, decreasing the depth of anesthesia until corneal reflexes returned and the arterial blood pressure was approximately 110/70 mm Hg. Baseline pressures were recorded from the right atrium and the ascending aorta (after pullback of the left ventricular catheter across the aortic valve). The aortic catheter, containing a 1 mm intraluminal stainless steel wire, was then readvanced across the aortic valve into the left ventricle.

Experimental protocol. Ventricular fibrillation was produced before full emergence from anesthesia by delivering a 60 Hz current through the stainless steel wire in the left ventricular catheter to the left ventricular endocardium. After documentation of ventricular fibrillation by both ECG and arterial pressure tracings, the left ventricular catheter was pulled back into the ascending aorta for the recording of aortic pressure during resuscitation. No treatment was instituted for the first 3 min of ventricular fibrillation to simulate the usual clinical “down time” occurring with cardiac arrest. After 3 min, all animals received external chest compressions and ventilation performed as recommended by the American Heart Association for standard basic life support with the use of a programmable mechanical resuscitator (Thumper; Michigan Instruments, Inc.). A compression rate of 60/min, a duty cycle of 50%, a compression-ventilation ratio of 5:1, and a compression depth of 2 inches were used in all animals. Aortic and right atrial pressures were measured continuously throughout the resuscitation period. Calibrations with the zero reference set to air at the midchest level were done repeatedly throughout the experiment. However, no pressure data were observed by the resuscitation team during the experiment. At 13 min of ventricular fibrillation, 1 mg of epinephrine was administered intravenously into the right atrium. At 15 min of ventricular fibrillation, external defibrillation was attempted with two consecutive 80 J shocks. Animals were divided into three distinct groups after the initial attempts at external defibrillation.

Animals successfully defibrillated with a resultant perfusing cardiac rhythm formed the first group. Animals unsuccessfully defibrillated, those that remained in ventricular fibrillation, or those in which defibrillation resulted in a nonperfusing rhythm were divided alternately into two groups, A and B. Group A animals received an additional 2 min of external closed-chest compression and then entered into an advanced cardiac life support (ACLS) protocol in which a sequential protocol was followed (table 1).

Group B animals underwent a left lateral thoracotomy immediately after failure of external defibrillation. The left lateral thoracotomy was performed after placing the animal in right lateral recumbancy. With use of a scalpel, a skin and subcutaneous tissue incision was made, followed by an intercostal incision, and finally, a pleural incision. Manual spreading of the ribs was performed on exposure of the intrathoracic cavity. Open-chest cardiac massage was performed with the right hand with the fingers over the left anterior-lateral portion of the heart while the thumb was medial and over the right heart. Open-chest cardiac massage was performed for 2 min. Figure 1 shows the hemodynamic tracing of one animal that received open-chest massage. ACLS was then provided following a sequential protocol similar to that adapted for internal defibrillation (table 1). Full resuscitation efforts were continued until response or completion of the ACLS protocol.

Successful resuscitation was defined as a mean arterial pressure of 50% or greater of the prearrest baseline. If resuscitated, the animal was entered into a 2 hr period of intensive “cardiac care unit” follow-up, which included: ventilation support, blood pressure support with dopamine when needed, and halothane plus oxygen for anesthesia. Thoracotomies were closed during this cardiac care unit period. Closure was accomplished by first

<table>
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<td>ACLS protocols adapted for dogs (20 to 25 kg)</td>
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<td>VF (external)</td>
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<tr>
<td>(1) Defib, 100 J</td>
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<td>(2) Defib, 100 J</td>
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<td>(3) CPR + oxygen</td>
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<td>(4) Epi, 1 mg iv</td>
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<td>(5) Bicarb, 1 meq/kg</td>
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<td>(6) Defib, 160 J</td>
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<td>(7) Defib, 160 J</td>
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<td>(8) CPR + oxygen</td>
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<td>(9) Defib, 160 J</td>
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<td>(10) Epi, 1 mg iv</td>
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<td>(11) CPR + oxygen</td>
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<td>(12) Defib, 240 J</td>
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<td>(13) Defib, 240 J</td>
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EMD

Asystole

VF = ventricular fibrillation; Bicarb = bicarbonate; Defib = defibrillation; EMD = electromechanical dissociation; Epi = epinephrine.
placing a chest tube through a stab wound one or two rib inter-
spaces below the incision, followed by rib approximation and
placement of interrupted sutures around the ribs with No. 1
chromic gut sutures. A three-layer closure was then performed
including the intercostal muscles, subcutaneous fascia, and skin
with the use of 4-0 chromic suture internally and 2-0 polypropy-
lene sutures for skin closure. Weaning from the ventilator and
extubation were performed in all animals during the cardiac care
unit period. Arterial and right atrial lines were removed after
arterial pressure had stabilized.

After this intensive care, the animals were transferred to a
postoperative ward for observation. Morphine sulfate was used
as needed for analgesia and sedation. All animals were main-
tained for 7 days or until death. Each animal received 240 mg of
trimethoprim-sulfamethoxazole antibiotic daily and 325 mg as-
pirin as needed for fever. All animals were offered water and
food within 8 hr of the procedure. Animals unable to drink
sufficiently were given fluids by subcutaneous infusion of Ring-
er's lactate solution. Each animal underwent daily evaluations,
including wound and thorax examinations and temperature rec-
CORDINGS. Extensive neurologic evaluations using a modified
neurologic deficit scoring system from the University of Pitts-
burgh Resuscitation Research Center17 were performed at 24 hr,
48 hr, and 4 and 7 days. All surviving animals were given an
intravenous euthanizing solution at the end of the 7 day follow-
up.

Data analysis. All hemodynamic data are listed as the mean
values ± SEM. Coronary perfusion pressure was calculated by
subtracting the right atrial mid-diastolic pressure from the si-
multaneous aortic mid-diastolic pressure.15, 16, 18 Fisher's exact
testing was performed for comparison of initial success of resus-
citation, 24 hr survival, and 7 day survival of animals receiving
closed-chest compression and those receiving open-chest cardia-
c massage.19, 20 Student's t testing (two-tailed) for unpaired
samples was used in comparing weights, hemodynamic vari-
bles, and neurologic deficit scores from the two groups of an-
imals. Morbidity for each group was compared with use of
Fisher's exact test.

Results

Resuscitation. Twenty of 29 animals were resuscitated-
cluding five of 14 animals in group A, 14 of 14 in
group B, and one animal that was eliminated from
further study because of successful external defibrilla-
tion after 15 min of cardiac arrest treated with closed-
chest CPR. Open-chest cardiac massage significantly
improved success of resuscitation when compared
with closed-chest compression (p < .005). Ease of
resuscitation was calculated with the use of criteria
from Ralston et al.21 and showed no difference be-
tween closed-chest compression and open-chest cardia-
c massage. Two of the five group A animals were
easy to resuscitate, requiring only electrical defibrilla-
tion; the remaining three were difficult to resuscitate,
requiring numerous defibrillation attempts and drug
therapy. Four of the 14 animals that received open-
chest cardiac compression were easy to resuscitate,
while 10 of 14 required more than one defibrillation
attempt. Figure 2 depicts the results of resuscitation.

Twenty-four hour survival. There was a marked dif-
fERENCE in 24 hr survival in the two groups. In group B
12 of 14 animals survived 24 hr; in group A four of 14
animals survived (p < .005; figure 2). One of the five
group A animals that was resuscitated died approxi-
mately 8 hr after resuscitation. Two of the 14 success-
fully resuscitated animals in the open-chest cardiac
compression group also died before 24 hr, one at 6 hr
and one at 12 hr.
Seven day survival. Open-chest cardiac compression significantly improved 7 day survival when compared with closed-chest compression. Eleven of 14 animals receiving open-chest cardiac compression survived for 7 days while four of 14 receiving closed-chest compression survived this length of time (p < .02). Figure 2 shows the difference in 7 day survival in the two groups.

Scoring of neurologic deficit. Neurologic deficit scores were calculated for each surviving animal at 24 hr, 48 hr, 4 days, and 7 days. The mean neurologic deficits of the four surviving animals from group A were not significantly different from those of the 12 survivors (24 hr) or 11 survivors (48 hr, 4 days, or 7 days) in group B (figure 3).

Hemodynamics. Baseline hemodynamics, including aortic systolic, aortic diastolic, right atrial systolic, right atrial diastolic, and calculated coronary perfusion pressures, were similar in both groups (table 2). There were no differences with respect to any hemodynamic variable between groups A and B during the first 15 min of ventricular fibrillation, when both groups received closed-chest compression. After the initiation of open-chest cardiac massage in group B, there was a significant difference noted in aortic systolic pressure, aortic diastolic pressure, and coronary perfusion pressure. The animals receiving open-chest cardiac massage had significantly higher values for these variables when compared with those receiving closed-chest compression (p < .001; figure 4).

Morbidity. Low-grade fever was common during the follow-up period. However, there was no significant difference in incidence of fever in animals that underwent thoracotomy and that in animals that received closed-chest compression. Of the 11 animals that underwent thoracotomy and were alive at 7 days, one had...
mild fluid accumulation in the subcutaneous tissue around the incision site. An additional animal had evidence of inner layer dehiscence with marked movement of the external layers with respiration, but was without respiratory compromise. No animal had objective evidence of active wound infection at 7 days.

Discussion

This study demonstrates that after 15 min of cardiac arrest, including closed-chest compression CPR for 12 min, open-chest cardiac massage significantly improves short- and long-term survival. The success with respect to long-term outcome seen with open-chest cardiac massage reflects in part the marked improvement in hemodynamics produced with direct cardiac massage. Open-chest cardiac massage was shown two decades ago to be superior to closed-chest compression for the production of cardiac output and mean aortic pressure. Weiser et al.,7 using normal animals and those that had suffered myocardial infarction, showed that open-chest cardiac massage produced two to three times the amount of cardiac output and mean aortic pressure produced by closed-chest compression CPR.7 Del Guercio et al.,8 comparing hemodynamics in man, found that the cardiac index with open-chest cardiac massage was twice that with closed-chest compression. Cohn and Del Guercio9 also found a significant increase in cardiac index with open-chest cardiac massage in a second expanded human series. More recently, Bircher et al.10 used a canine preparation to show that even after 2 hr of external chest compression, open-chest cardiac massage significantly improved systolic and diastolic arterial pressures and perfusion pressures, while doubling common carotid arterial blood flow. Bartlett et al.12 have also shown in a canine preparation that arterial systolic, diastolic, and mean pressures are substantially increased with open-chest cardiac massage and that cardiac index is two- to three-fold higher with this method than with closed-chest compression.

We observed a similar impressive increase in arterial systolic pressure and, more importantly, in arterial diastolic pressure, with open-chest cardiac massage. This increase in aortic diastolic pressure without a substantial simultaneous rise in right atrial diastolic pressure produced an increase in coronary perfusion pressure that was both statistically significant and most likely an important factor in the improved outcome with open-chest cardiac massage.

Numerous studies have shown that coronary perfusion pressure correlates well with measured coronary blood flow and improved resuscitation from cardiac arrest.15, 16, 18, 21-23 The improved initial resuscitation rate with open-chest cardiac massage seen in this study corresponds with the remarkable increase in coronary perfusion pressure seen with CPR by this method.

Sanders et al.15 found that 3 min of open-chest cardiac massage after 15 min of inadequate closed-chest compression resuscitated significantly more animals than did continued closed-chest compression. In a second study, Sanders et al.16 showed that if closed-chest compression was continued for 25 min before open-chest compression was initiated, there was no resuscitation benefit from open-chest cardiac massage. Geehr et al.14 have recently reported similar findings in a human study in which open-chest cardiac massage was instituted after arrival at the emergency department, usually about 30 min or more from the time of initial cardiac arrest. No difference in rates of immediate resuscitation or survival was found. Hence, open-chest cardiac massage, although effective in improving hemodynamic responses, must be instituted within a relatively short time to improve success of resuscitation. Open-chest cardiac massage is unlikely to improve survival if used as a “last-ditch effort.”

In this study and one other,17 24 hr survival rate was significantly higher with open-chest cardiac massage than with closed-chest compression resuscitation. Bircher and Safar17 found that animals given 30 min of open-chest cardiac massage had a significantly higher 24 hr survival rate than did those receiving either standard external chest compression or simultaneous compression and ventilation external CPR.

Bircher and Safar17 also reported a significant improvement in neurologic function at 24 hr with open-chest resuscitation. In calculating the mean neurologic
deficit scores they included all animals, with animals that died receiving a score of 100%. In our study, in which the mean neurologic deficit score was calculated with data from survivors only, no benefit with respect to neurologic outcome was noted with open-chest resuscitation. However, the small number of survivors and the possibility of a large beta (type 2) error limits any definitive conclusions concerning long-term neurologic outcome with open-chest cardiac massage.

Open-chest cardiac massage did not produce an increase in apparent morbidity when compared with closed-chest compression. There was no evidence of thoracic infections in any of the group B animals, nor was there a significant difference in the incidence of fever during the follow-up period in animals given open-chest cardiac massage and those receiving closed-chest compression. Altemeier and Todd reported a similar low incidence of infection after thoracotomy and open-chest cardiac massage. Only two of their 43 patients had evidence of infection of the thoracotomy wound. They also noted that fever was common, but it did not correlate with active wound infection.

The emergent minilateral thoracotomy used in this animal study was easily learned and performed capably by all principal investigators. The entire thoracotomy procedure routinely took less than 20 to 30 sec to perform. These results indicate that in this preparation the emergency thoracotomy necessary to perform open-chest cardiac massage need not be difficult and may be performed quickly.

Experimental work has shown the benefits of open-chest cardiac massage for resuscitation from cardiac arrest. However, all but one of the previous studies have used healthy animal preparations without heart disease such as myocardial infarction or obstructive coronary lesions. It remains unknown if the dramatic improvements seen in these experimental preparations without heart disease will be evident in the clinical arena, in which victims of cardiac arrest commonly have poor left ventricular function and obstructive coronary disease. Experimental preparations with poor left ventricular function, previous myocardial infarctions, and/or coronary obstructive lesions are needed to confirm the benefits of open-chest cardiac massage. Hence, caution must be exercised in extrapolating these data from normal healthy animals to humans.

This study showed that open-chest cardiac massage, performed when external defibrillation failed after 15 min of ventricular fibrillation and ineffective closed-chest CPR, can improve not only immediate resuscitation success and 24 hr survival, but also 7 day survival.

Further confirmation of these benefits is needed in experimental preparations with organic heart disease.

References

Long-term survival with open-chest cardiac massage after ineffective closed-chest compression in a canine preparation.
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