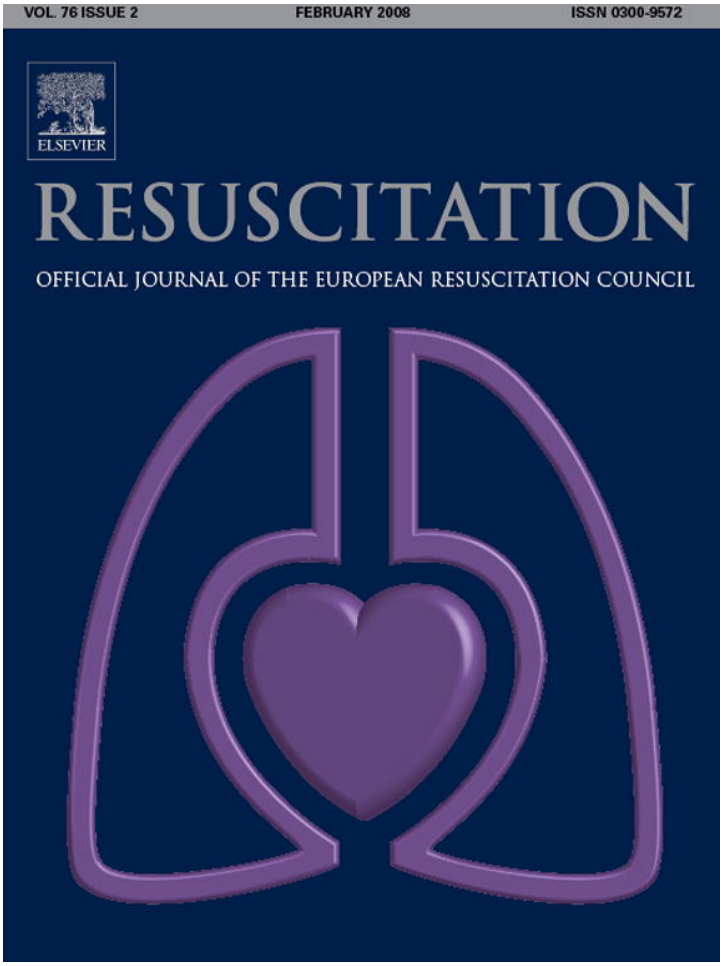

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CLINICAL PAPER

Miniaturized mechanical chest compressor: A new option for cardiopulmonary resuscitation☆

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Post-resuscitation survival

Summary
Aim of study: After cardiac arrest, uninterrupted chest compressions with restoration of myocardial blood flow facilitates restoration of spontaneous circulation. We recognized that this may best be accomplished with a mechanical device and especially so during transport. We therefore sought to develop a lightweight, portable chest compressor which may be carried on the belt or attached to the oxygen tank typically carried on the back of the first response rescuer. A miniaturized pneumatic chest compressor (MCC) weighing less than 2 kg was developed and compared with a currently marketed ''Michigan Thumper®'', which weighed 19 kg. We hypothesized that the 2 kg, low profile, portable device will be as effective as the standard pneumatic Thumper® for restoring circulation during CPR.
Material and methods: Ventricular fibrillation was electrically induced in 10 domestic male pigs weighing 39 ± 2 kg, and untreated for 5 min. Animals were then randomized to receive chest compressions with either the MCC or the Thumper®. After 5 min of mechanical chest compression, defibrillation was attempted with a 150 J biphasic shock. Coronary perfusion pressure (CPP) and end tidal PCO_2 ($EtPCO_2$) were measured by conventional techniques together with right carotid artery blood flow (CBF).
Results: Four of five animals compressed with the Thumper® and each animal compressed with the MCC were successfully resuscitated. No significant differences in CPP, $EtPCO_2$, CBF and post-resuscitation myocardial function were observed between groups. Resuscitated animals survived for more than 72 h without neurological impairment.
Conclusion: The low profile, 2 kg miniaturized chest compressor is as effective as the conventional Thumper® in an experimental model of CPR.

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Introduction

Cardiovascular disease continues to be the leading cause of death and more than 400,000 Americans and 700,000 Europeans are victim of cardiac arrests each year.[1] Despite major efforts to improve outcomes from cardiac arrest, fewer than 5% of victims are hospital survivors.[2–5] Both in heavily populated larger cities and in sparsely populated rural communities, delayed response by emergency medical services compromises outcomes such that survival is even more disappointing, namely as low as 1%.[6,7]

There is now evidence that the highest priority of intervention is to re-establish systemic blood flow promptly by external chest compression and thereby achieve and maintain threshold levels of coronary and cerebral perfusion. Accordingly, effective, consistent and uninterrupted chest compression is now designated as the primary intervention for management of cardiac arrest. Both survival and neurological recovery are contingent upon initiating chest compression within less than 5 min.[8–10] Accordingly, bystander initiated chest compressions by minimally trained, non-professional rescuers subsequently supported by well organized professional emergency medical providers have significantly increased survival from out-of-hospital cardiac arrest.[11–13]

In addition to the benefits of prompt intervention, it is also the quality of chest compressions delivered in both in- and out-of-hospital settings, which has proven to be a determinant of outcomes. Even well-trained professional providers cannot maintain effective chest compression for intervals that exceed 2 min.[14–17] This limitation is in addition to the documented inconsistency of depth and rate of compressions.[18–20] The challenges are even greater during evacuation and transport of victims. Therefore, the option of using mechanical devices is attractive. Mechanical chest compression potentially overcomes operator fatigue, slow rates of compression, and inadequate depth of compression. A mechanical compressor would also allow for the delivery of an electrical shock without interruption of manual compression for the protection of the rescuer.

The present study in a porcine model was therefore undertaken to compare the effectiveness of a newly developed miniaturized pneumatic chest compressor (MCC) with that of a conventional and commercially available compression device (Figure 1). The MCC was so designed that it may be carried on the belt or attached to the oxygen gas tank carried routinely by the professional rescuer. It is pneumatically powered with oxygen or compressed air. In Table 1, the principal features of the two devices are compared. The biomedical engineering details of design, construction, and pneumatic operation of the MCC will be addressed in detail in a separate medical engineering publication.

We tested the hypothesis that such a lightweight device would be as effective as a current standard, the Thumper® (Model 1004, Michigan Instruments, Grand Rapids, MI), for restoring circulation during CPR after cardiac arrest.

Materials and methods

All animals received humane care in compliance with the ''Principles of Laboratory Animal Care'' formulated by the National Society for Medical Research and the *Guide for the Care and Use of Laboratory Animals* prepared by the Institute of Laboratory Animal Resources and published by the National Institutes of Health (NIH publication 86-32, revised 1985). The protocol was approved by the Institutional Animal Care and Use Committee of the Weil Institute of Critical Care Medicine. The animal laboratories of the Weil Institute are fully accredited by American Association for Accreditation of Laboratory Animal Care (AAALAC) International.

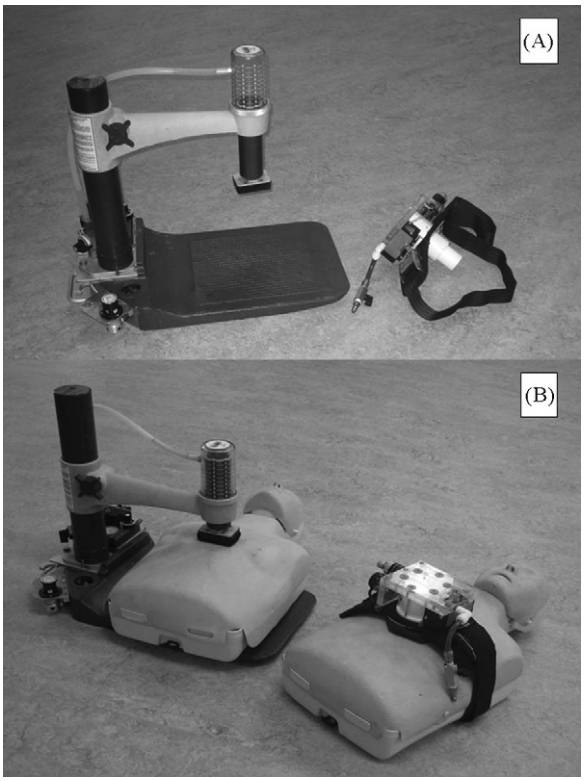

Figure 1 (A) The Michigan Thumper® shown on the left and the MCC on the right. (B) The Michigan Thumper® applied to a manikin on the left and the MCC applied to a manikin on the right.

Table 1 A comparison of the features of the two chest compression devices

	MCC	Thumper®
Weight (kg)	2	19
Length (cm)	35	61
Width (cm)	15	30
Height (cm)	9	139.5
Force at pneumatic pressure of 50 psi (kg)	48	55
Gas consumption (L/min)	46	45
Compression rate (compressions/min)	90 ± 5	90 ± 5
Maximal piston descent (cm)	10	10

Animal preparation

Ten male domestic pigs weighing 39 ± 2 kg were fasted overnight except for free access to water. Anesthesia was initiated by an intramuscular injection of ketamine (20 mg/kg) and completed by ear vein injection of sodium pentobarbital (30 mg/kg). Additional doses of sodium pentobarbital (8 mg/kg) were injected at intervals of approximately 1 h to maintain anesthesia. A cuffed tracheal tube was advanced into the trachea. Animals were mechanically ventilated with a volume-controlled ventilator (Model MA-1, Puritan-Bennett, Carlsbad, CA) with a tidal volume of 15 mL/kg, peak flow of 40 L/min, and FiO_2 of 0.21. End-tidal PCO_2 ($EtPCO_2$) was monitored with an infrared capnometer (Model NPB-75, Nellcor Puritan Bennett Inc., Pleasanton, CA). Respiratory frequency was adjusted to maintain $EtPCO_2$ between 35 and 40 mm Hg. For measurement of left ventricular function, a transesophageal echocardiographic transducer was advanced from the incisor teeth into the esophagus for a distance of approximately 35 cm. For measurement of mean aortic pressure (MAP), a fluid-filled catheter was advanced from the right femoral artery into the thoracic aorta. For the measurements of right atrial pressure (RAP), mean pulmonary artery pressure (MPAP), and thermodilution cardiac output (CO_{TD}), a 7-Fr pentalumen, thermodilution-tipped catheter was advanced from the right femoral vein and flow directed into the pulmonary artery. Carotid blood flow (CBF) was continuously measured with the aid of a flowprobe (Ultrasonic Blood Flow Meter, T101, Transonic Systems Inc., Ithaca, NY) positioned around the right common carotid artery. For inducing VF, a 5-Fr pacing catheter (EP Technologies, Inc., Mountain View, CA) was advanced from the right subclavian vein into the right ventricle. The position of catheters was confirmed by characteristic pressure morphology and/or fluoroscopy. The pacing catheter was removed after onset of VF. The piston of the compressor was positioned in the midline at the level of the fifth interspace and this locus was defined prior to randomization. Repositioning of the devices was not required because they were stable once positioned. Precordial compression was started with either the Thumper® or the MCC. The chest compressors were programmed to provide equal compression—relaxation intervals, i.e. a 50% duty cycle. Coincident with the start of precordial compression, the animals were ventilated asynchronously with a tidal volume of 15 mL/kg and FiO_2 of 1.0 and with a rate of 10 breaths per min.

Experimental procedures

Before inducing cardiac arrest, the animals were randomized by the sealed envelope method to receive chest compression by either of the two piston-driven devices. VF was induced with 1—2 mA alternating current delivered to the endocardium of the right ventricle. Mechanical ventilation was discontinued after onset of VF. After 5 min of untreated VF, mechanical chest compression with one of the two devices was begun. Animals were ventilated simultaneously with 100% oxygen for the ensuing 5 min. Electrical defibrillation was then attempted with a single biphasic 150 J electrical shock, delivered between the conventional right infraclavicular electrode and the apical electrode with a Heartstart XL defibrillator (Philips Medical Systems, Andover, MA). If spontaneous circulation was not restored, mechanical chest compressions with the same compressor and ventilation were resumed and continued for 2 min before a subsequent defibrillation attempt. The same resuscitation procedure was continued until successful resuscitation or for a maximum of 15 min. No vasopressor drugs were used. The animals were regarded as successfully resuscitated if an organized cardiac rhythm with MAP of more than 60 mmHg persisted for an interval of 5 min or more. Anesthesia was continued and animals were monitored for an additional 4 h. With the aid of an image intensifier and fluoroscopy, we visualized the bony thorax and counted the number of fractured ribs. Catheters were then removed, wounds were surgically repaired, and animals were extubated before they were returned to their cages. The animals were then observed for an additional 68 h. At the end of the 72-h post-resuscitation observation interval, animals were reanesthetized with ketamine and pentobarbital. Echo-Doppler measurements of myocardial functions were then repeated. Animals were then sacrificed painlessly with an intravenous injection of 150 mg/kg pentobarbital. Autopsy was performed routinely for documentation of potential injuries to the thoracic and abdominal viscera occurring during CPR, including fractured ribs, or due to obfuscating disease.

Measurements

Hemodynamic data, $EtPCO_2$, and ECG were measured continuously and recorded on a PC-based data acquisition system supported by CODAS hardware/software as described previously.[21] Coronary perfusion pressure (CPP) was digitally computed from the differences in time-coincident diastolic aortic and right atrial pressures and displayed in real time. Arterial and mixed venous blood gases, hemoglobin and oxyhemoglobin were measured on 200 μL aliquots of blood with a stat profile analyzer (ULTRA C, Nova Biomedical Corporation, Waltham, MA) adapted for porcine blood. These measurements were obtained at 15 min before inducing cardiac arrest and at 4 min after starting chest compression. Cardiac output was measured by conventional thermodilution techniques after injection of 5 mL of saline maintained at 2 °C. Echocardiographic measurements were obtained with the aid a S7-t3 mini-multi, transesophageal echocardiographic transducer (Model HD11XE, Philips Medical Systems, Eindhoven, Netherlands). Long axis two-chamber views were obtained. Left ventricular end-systolic and end-diastolic volumes were calculated by the method of discs, as previously described.[21] From these, stroke volumes (SV), ejection fractions (EF) and fractional area change (FAC) were computed. Measurements were obtained at baseline and at hourly intervals thereafter for a total of 4 h. These measurements were repeated at 72 h following resuscitation to quantify myocardial function. A neurological alertness score (NAS), developed by our group[22] was used for evaluating neurological recovery at 24, 48, and 72 h.

Statistical analyses

The independent variables were the two mechanical chest compressors, the Thumper® and the MCC. The dependent variables were initial resuscitation; CPP, $EtPCO_2$ and CBF during chest compression; numbers of shocks delivered before restoration of spontaneous circulation (ROSC) and incidence of recurrent VF. Additional independent variables included duration of CPR; numbers of fractured ribs; post-resuscitation myocardial function including left ventricular SV, EF and FAC; post-resuscitation neurological recovery; and post-resuscitation duration of survival. For measurement between groups, ANOVA with Scheffe's method for multiple comparisons was used. When the dependent variable was categorical such as 24, 48, and 72 h survival, Fisher's exact test was used. Values are reported as mean ± standard deviation. A p value of <0.05 was regarded as significant.

Results

There were no differences in the baseline values blood gas measurements, heart rate (HR), MAP, $EtPCO_2$, RAP, MPAP and CO_{TD}, between the two groups (Table 2). In each animal treated with the MCC device, ROSC was achieved with only a single shock (Table 3). Four of the five animals treated with the Thumper® achieved ROSC.

During chest compression, we observed no significant differences in CPP, $EtPCO_2$, CBF (Figure 2) or in the arterial blood concentrations of lactate (Figure 3) between the two groups. Both CPP and CBF were numerically greater with the MCC, but the differences were only of borderline statistical significance. We observed a numerical but not statistically significant lower incidence of fractured ribs with the MCC (Figure 3).

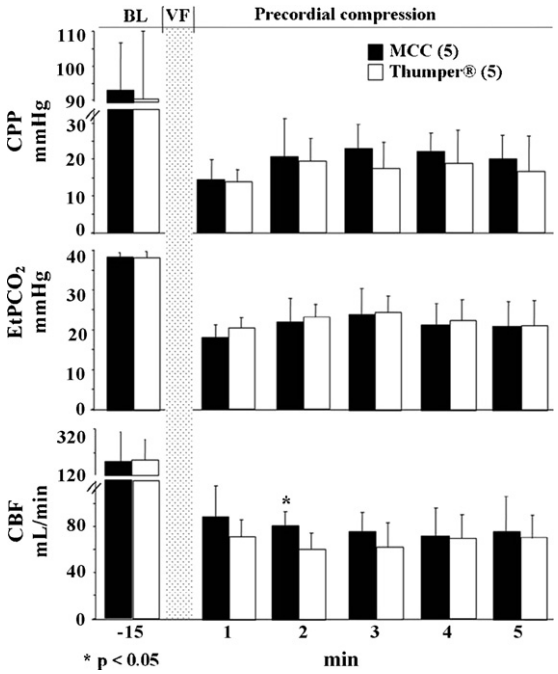

Figure 2 A comparison of coronary perfusion pressures (CPP), end-tidal PCO_2 ($EtPCO_2$) and carotid blood flows (CBF) at baseline (BL) and during precordial compression. Mean ± S.D.; *$p < 0.05$.

Each resuscitated animal survived for 72 h with full neurological recovery. No differences in baseline or post-resuscitation thermodilution cardiac output, MAP or

Table 2 Baseline measurements (mean ± S.D.)

	MCC (5)	Thumper® (5)	p (MCC vs. Thumper®)
Animal weight (kg)	39.8 ± 1.8	37.6 ± 2.3	0.13
PaO_2/FiO_2	410 ± 72	414 ± 89	0.93
LAC (mmol/L)	1.2 ± 0.7	0.8 ± 0.3	0.28
HR (beats/min)	140 ± 44	123 ± 16	0.44
MAP (mmHg)	107 ± 7	108 ± 13	0.91
RAP (mmHg)	3 ± 1	4 ± 2	0.41
MPAP (mmHg)	21 ± 2	20 ± 3	0.65
CO_{TD} (L/min)	8 ± 1.2	7 ± 0.8	0.15

PaO_2 = arterial partial pressure of oxygen; FiO_2 = inspiratory fraction of oxygen; LAC = arterial blood lactate; HR = heart rate; CO_{TD} = thermodilution cardiac output.

Table 3 Resuscitation outcomes (mean ± S.D.)

	MCC	Thumper®	p (MCC vs. Thumper®)
Resuscitated	5/5	4/5	0.3
Number of shocks prior to ROSC	1	1.25 ± 0.5	0.29
Incidence of recurrent of VF	2 ± 2.8	0.5 ± 1	0.35
Duration of CPR prior to ROSC	306 ± 11	334 ± 76	0.39

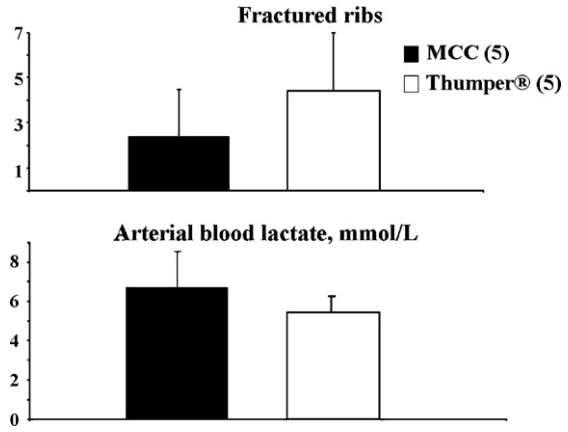

Figure 3 A comparison of the number of fractured ribs produced by chest compressions with the two devices and arterial blood lactate concentration measured at 4 min after start of precordial compression. Mean ± S.D.

echocardiographically measured myocardial function were observed between the two groups (Table 4).

Discussion

The present study documented that the new miniaturized chest compressor was as effective as the Thumper® with respect to increases in CPP and $EtPCO_2$ during chest compressions. The MCC generated marginally greater CBF. There were no statistically significant differences in outcomes with respect to the success of initial resuscitation, post-resuscitation myocardial function, post-resuscitation neurological recovery, and 72 h survival. Accordingly, the goal of our effort was achieved, namely equivalency of the effectiveness and outcomes when the two compression devices were compared.

Current guidelines strongly recommend chest compression to restore blood flow to vital organs as the primary intervention after ''sudden death'', especially when the duration of untreated cardiac arrest is greater than 5 min.[8] Following prolonged untreated cardiac arrest progressive energy imbalances develop since chest compression produces less than 50% of pre-arrest stroke volumes.[23–26] However, threshold levels of myocardial and cerebral blood flows are restored such as to minimize ischemic myocardial and cerebral ischemic injury.[27] CPP is a key predictor of the likelihood of successful ROSC and both the Thumper® and the MCC consistently increased CPP to levels that exceeded the threshold of 15 mmHg for successful ROSC.[28–30] $EtPCO_2$, which has also emerged as an indirect measure of pulmonary blood flow and therefore cardiac output produced by chest compression, continuously exceeded the threshold level of approximately 15 mmHg, which is predictive of successful resuscitation.[31–34]

Several new devices have recently been introduced to facilitate mechanical chest compression. Both the LUCAS (Jolife, Medtronic, Sweden) and the AutoPulse (Revivant Corporation, CA, USA) have demonstrated equivalency and potentially even greater effectiveness than manual chest compression.[35–38] In each instance, however, the weight of the devices ranges from 6.5 to 12.2 kg and width from 44 to 46 cm. These physical dimensions compromise portability. The MCC weighs <2 kg and is only 15 cm in width. The MCC complies with the inventors' intent, namely that it may be carried without significant burden by the first response professional rescuer (Figure 4) such as to facilitate prompt start of mechanical chest compression on arrival. The MCC is anchored in position on the patient's thorax regardless of the victim's body position, a major advantage during evacuation through stairways and around corners.

In addition, to its modest dimensions and weight, the MCC therefore fulfils the need of a mechanical device for both early implementation and uninterrupted, effective chest compressions. The advantages of size and weight also allow for chest compression during ambulance transport and transport through hospital hallways and elevators. Perhaps most important, effective and uninterrupted precordial compres-

Table 4 Post-resuscitation hemodynamic measurements (mean ± S.D.)

Hours	MCC	Thumper®	*p* (MCC vs. Thumper®)
Stroke volume (mL)			
BL	29 ± 11	30 ± 12	0.91
PR 1	19 ± 6	23 ± 4	0.31
PR 2	22 ± 4	26 ± 5	0.29
PR 3	23 ± 4	25 ± 6	0.53
PR 4	27 ± 7	25 ± 6	0.68
PR 72	31 ± 14	27 ± 2	0.77
Ejection fraction (%)			
BL	64 ± 6	61 ± 3	0.27
PR 1	54 ± 7	55 ± 6	0.78
PR 2	54 ± 4	57 ± 7	0.34
PR 3	54 ± 3	58 ± 2	0.11
PR 4	56 ± 4	61 ± 8	0.24
PR 72	63 ± 2	62 ± 3	0.77
Fractional area change (%)			
BL	51 ± 4	47 ± 5	0.59
PR 1	36 ± 6	37 ± 10	0.76
PR 2	38 ± 2	39 ± 6	0.77
PR 3	40 ± 3	42 ± 3	0.39
PR 4	42 ± 3	43 ± 7	0.81
PR 72	47 ± 8	48 ± 4	0.64
CO_{TD} (L/min)			
BL	8 ± 1.2	7 ± 0.8	0.15
PR 1	7.2 ± 1	6 ± 1	0.12
PR 2	7.5 ± 1.4	5.8 ± 1.5	0.13
PR 3	7.2 ± 1.5	5.4 ± 0.9	0.07
PR 4	6.9 ± 1.3	6.2 ± 1.4	0.41
MAP (mmHg)			
BL	107 ± 7	108 ± 13	0.91
PR 1	104 ± 11	94 ± 4	0.14
PR 2	107 ± 11	101 ± 11	0.47
PR 3	107 ± 8	103 ± 6	0.47
PR 4	111 ± 3	108 ± 4	0.29

Baseline (BL) measurements on five animals per group. Post-resuscitation (PR) measurements on five animals with the MCC and four animals with the Thumper®; CO_{TD} = thermodilution cardiac output.

Figure 4 The MCC worn on the belt of the professional rescuer.

sion provided by mechanical compressors is not limited by operator fatigue.

We recognize limitations in the interpretation of our findings. The studies were conducted in healthy animals and therefore in the absence of underlying diseases or injuries that are causative of cardiac arrest. The investigators were not blinded to the intervention and, accordingly, the possibility of observer bias in the physical application of the mechanical chest compressors to the chest of the animal is not excluded although the location of the piston was the same for both groups. The close relationship among measurements of CPP, $EtPCO_2$, and CBF during chest compression provides additional evidence. Finally, the present study demonstrates equivalency with an approved device but without comparison to conventional manual chest compressions. However, the evidence that with manual compression, rescuer fatigue, interruption of chest compression for the delivery of defibrillating shock and the difficulty of manual compression during transport, would provide persuasive evidence to the contrary.

Conclusions

The miniaturized mechanical chest compressor represents a new option for CPR. It is as effective as the conventional Thumper® with respect to increases in CPP, $EtCO_2$ and carotid blood flow during chest compressions. The MCC has the advantage of lightweight and compact dimensions so that it may be carried routinely on the belt of first response rescuers and allow for uninterrupted CPR during transport.

Conflict of interest

No conflicts of interest are reported. No financial benefits to the researchers are reported. All benefits of all inventions of the Weil Institute of Critical Care Medicine are totally reserved for research support and none accrue to the personal benefit of any of the inventors or researchers.

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