Comparison of CPR Outcome Predictors Between Rhythmic Abdominal Compression and Continuous Chest Compression CPR Techniques

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Abstract and Introduction

Abstract

Objective: Bystander cardiopulmonary resuscitation (CPR) provides treatment for out-of-hospital cardiac arrest since perfusion of vital organs is critical to resuscitation. Alternatives to standard CPR are evaluated for effectiveness based upon outcome predictive metrics and survival studies. This study focuses on evaluating the performance of rhythmic-only abdominal compression CPR (OAC-CPR) relative to chest compression (CC-CPR) using a complementary suite of mechanistically based CPR outcome predictors. Combined, these predictors provide insight on the transduction of compression-induced pressures into flow perfusing vital organs.

Methods: Intrasubject comparisons between the CPR techniques were made during multiple 2-min intervals of induced fibrillation in 17 porcine subjects. Arterial pO_2, cardiac output, carotid blood flow, coronary perfusion pressure (CPP), minute alveolar ventilation (MAV), end-tidal CO_2, and time from defibrillation to the return of spontaneous circulation (ROSC) were recorded. Organ damage was assessed by necropsy.

Results: Compared with CC-CPR, OAC-CPR had higher pressure and ventilation metrics with increased relative CPP (+16 mm Hg), MAV (+75/ml/min/kg) and a lower reduction in arterial pO_2(−22% baseline), but suffered from lower carotid flows (−9.3 ml/min). No significant difference was found comparing cardiac outputs. Furthermore, resuscitation was qualitatively more difficult after OAC-CPR, with a longer time to ROSC (+70 s). No abdominal damage was observed over short periods of OAC-CPR.

Conclusions: Although OAC-CPR appeared superior to CC-CPR by pressure and ventilation metrics, lower carotid flow and longer delay until ROSC raise concerns about overall performance. These paradoxical observations suggest that the evaluation of efficacious alternative CPR techniques may require more direct measurements of vital organ perfusion.

Introduction

Cardiopulmonary resuscitation (CPR) by bystanders is essential to improving the resuscitation and neurological outcome after out-of-hospital cardiac arrest.[1,2] However, many bystanders are hesitant to perform CPR,[3] primarily due to an aversion to performing mouth-to-mouth resuscitation (MMR)[4,5] and anxiety over performing it incorrectly, and thereby, causing more harm.[6] In response to these concerns, the American Heart Association (AHA) has endorsed chest compression only CPR (CC-CPR) for untrained bystanders.[7] While the approach does not address concerns of inadequate ventilation over extended periods of CC-CPR,[8] numerous studies support CC-CPR for bystanders, through both experimental and clinical evidence.[9–11]

Rhythmic-only abdominal compression CPR (OAC-CPR) is a potential alternative CPR technique for use by untrained bystanders, proposed to avoid chest injury, promote ventilation and reduce effort for the CPR provider. In OAC-CPR, the abdomen of the victim is rhythmically compressed, without chest compressions. OAC-CPR may be physically less demanding for the bystander over extended periods as it requires less force and lower compression rates to generate similar coronary perfusion pressure (CPP)[12] to that of CC-CPR in porcine subjects. Without manual ventilation, OAC-CPR also generates greater minute alveolar ventilation (MAV)[13] than CC-CPR. By contrast, a recent study found that CC-CPR supplemented with intubation and artificial ventilation improved survival and neurological outcome over OAC-CPR when performed for 2 min following 8 min of unassisted fibrillation.[14] Collectively, these results assess OAC-CPR by several predictive metrics without ventilation and by outcomes with ventilation, with slightly contradictory results. However, they do not clearly establish the performance of OAC-CPR, especially for bystanders; nor do they provide detail on differences from CC-CPR in terms of blood oxygenation and flow to vital tissues. To address the gap, we directly compare performance without ventilation of OAC-CPR with CC-CPR using an expanded set of CPR outcome predictors.

To better investigate the functional differences between OAC-CPR and CC-CPR, we look to measurements predictive of cardiopulmonary performance and vital organ support, including: mean CPP, MAV, arterial pO_2, cardiac output, carotid blood flow, $EtCO_2$, delay until return of spontaneous circulation (ROSC), as well as injuries caused by CPR. CPP, MAV, and blood oxygenation have been previously used as predictors of resuscitation or survival.[15–19] Cardiac output indicates overall performance, but does not address distributions of flow to important tissues. Measuring carotid flow indicates the potential for flow to the brain. While cerebral blood flow is more direct to predict neurological outcome and its relationship to carotid flow is complex,[20,21] it is difficult to accurately measure during the low flow conditions of CPR.[22] For respiratory performance, end-tidal carbon dioxide ($EtCO_2$) is an indirect indicator for hypoventilation or hyperventilation of the subject. The time from defibrillation to ROSC can be used more qualitatively to infer overall performance and heart viability. Finally, it is worth noting the type and severity of injuries resulting from CPR, although such complications are rarely life-threatening.[23] Combining these metrics and using CC-CPR as a reference, we evaluate the strengths and weaknesses of OAC-CPR, as suggested for bystander use.

Methods

All studies were approved by the Purdue Animal Care and Use Committee. Pigs of either gender weighing 31±3 kg were used. To minimise the number of subjects used, while addressing procedural concerns regarding some measurements, two CPR protocols were employed as described below, with eight pigs in the first protocol, and nine pigs in the second. The procedures and instrumentation for anaesthesia, ventricular fibrillation induction, as well as the measurement of ECG, blood pressures, air flow, expired CO_2 content and blood chemistry are measured, as in Pargett *et al*.[13] All continuous data signals were recorded using a 16-channel data acquisition (DAQ) board (model SC-2345, National Instruments, Austin, Texas, USA.) Statistical analysis was performed using Minitab 16 Statistical Software (State College, Pennsylvania, USA).

Instrumentation

Instrumentation was applied as in figure 1. Cardiac output was assessed by the thermodilution method using a cardiac output computer (model 9520A, American

Edwards Laboratories, Irvine, California, USA). Right carotid blood flow was measured continuously using an ultrasonic perivascular flowprobe (model 4PSB, Transonic Systems, Ithaca, New York, USA) with its flowmeter (Module TS420, Transonic Systems). A lubricant (Surgilube, Fougera, Melville, New York, USA) was used as an acoustic couplant for the flowprobe and was thermally equilibrated for 1 h before measurement. CC-CPR and OAC-CPR compressions were administered using a Life Aid CPR Cardiac Compressor 'Thumper' (Michigan Instruments, Grand Rapids, Michigan, USA). Force to the abdomen during OAC-CPR was distributed over approximately 25 square inches, using a 'home plate' applicator, as described in previous studies.[12]
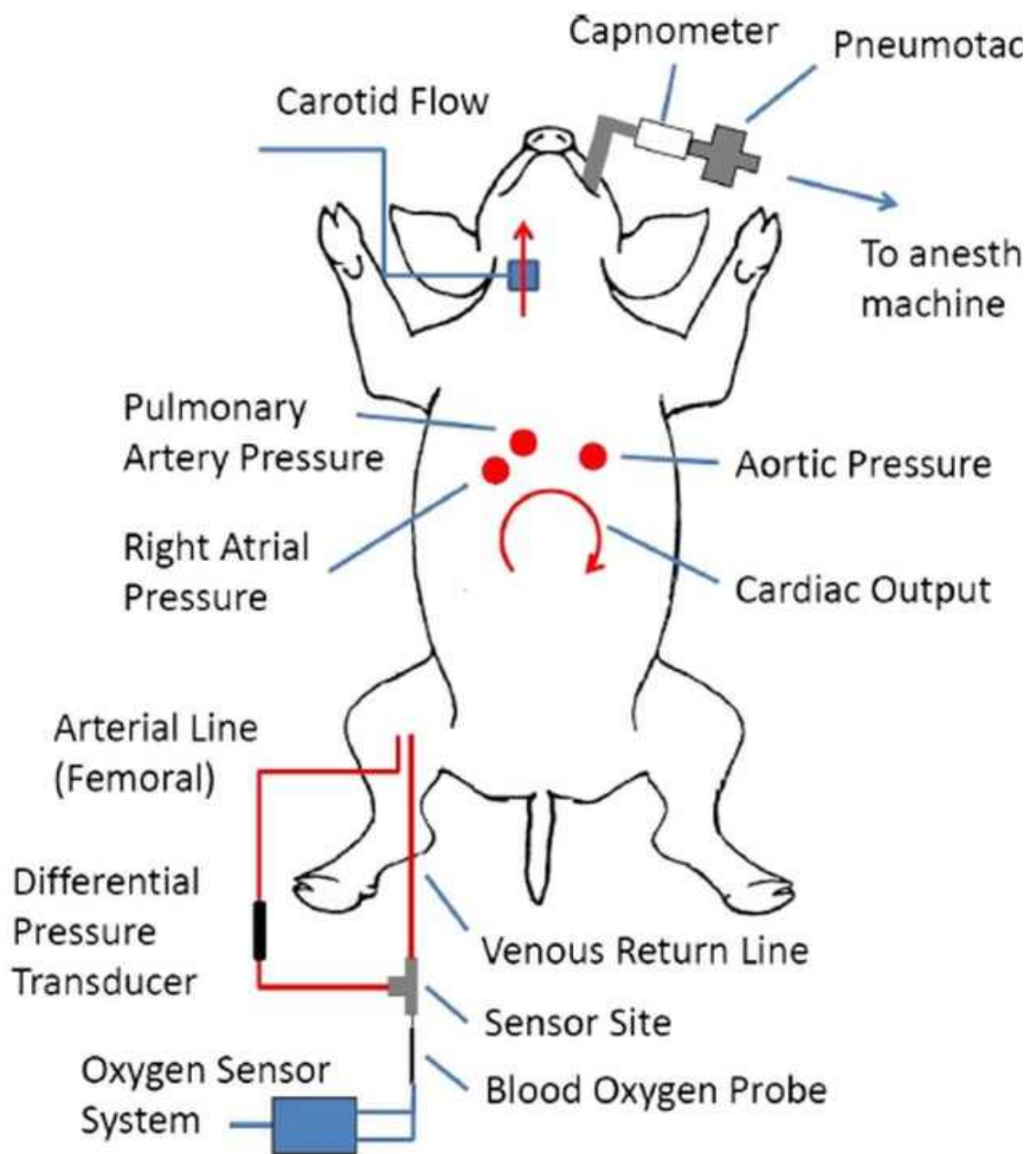
Figure 1.

Instrumentation of porcine subject for measuring predictive metrics.

Blood gas dynamics were measured using a photoluminescence-quenching O_2 sensor with thermistor (FOXY-AL300-TM and MFPF-100, Ocean Optics, Dunedin, Florida, USA), placed into an external arteriovenous shunt connecting the right femoral artery and the right femoral vein. The shunt contained ~12 ml of blood at any given time. A differential pressure transducer (PX26 series, Omega Engineering, Stamford, Connecticut, USA) was used to monitor shunt pressure for indications of clotting or backflow during measurement.

Experimental Protocol

For each subject, a preliminary compression test determined the force needed to compress the chest to the 2005 AHA recommended depth of 1.5 inches (3.8 cm).[24] This force was used for all CC-CPR runs. Severe chest remodelling was observed if the AHA 2010 compression recommendation of 2 inches (5 cm) was performed on the porcine subjects.

Ventricular fibrillation was electrically induced by a catheter electrode placed in the right ventricle, and CPR was started immediately following visual confirmation of fibrillation on the ECG. Following the AHA recommended parameters for a bystander, CC-CPR was performed at 100 compressions per minute. OAC-CPR was performed with 100 lbs (445 N) of force at a rate of 80 compressions per minute, as recommended previously.[13] Cardiac output was measured ~1 min into CPR to allow for a stabilisation of blood flows. Defibrillation shocks were administered approximately 2 min after the start of fibrillation using a biphasic defibrillator (Lifepak 20, Medtronic, Minneapolis, Minnesota, USA) increasing from 50 J as necessary. Several chest compressions were performed immediately after defibrillation,[7] and the presence of a stable electrical rhythm and returning circulation (as indicated by steadily increasing pulse and mean arterial pressures) were evaluated for several seconds. As needed, additional CPR and defibrillations were performed, and epinephrine was administered to treat postdefibrillation pulseless electricity activity. Each set of induced fibrillation and CPR was considered to be an experimental 'run'. The length of these experiment 'runs' was kept short at 2 min, well within the first 4-min 'electrical' phase of CPR,[25] to minimise the risk of unsuccessful resuscitation, and to reliably collect sufficient data per subject for at least one intrasubject comparison between OAC-CPR and CC-CPR.

Once ROSC was achieved, at least 10 min were allowed for the subject to recover to stable cardiorespiratory dynamics before beginning the next run. After each run, the presence of permanent chest deformation or broken ribs was assessed visually and by palpation. Blood chemistry was monitored using an iSTAT (Cat No 5101, Heska, Wankesha, Wisconsin, USA), and a 5% dextrose drip was administered as needed. Heparin was administered as necessary to prevent clotting in catheters.

Following euthanasia, a laparotomy and a median sternotomy were performed to further assess damage to the abdominal and thoracic organs. Any visual abnormalities or damage to the internal organs and tissues were recorded.

CPR Run Protocol 1

Protocol 1 focused on collecting all data, while considering potential interfering effects on

the blood oxygen measurement, giving three distinct run procedures within the protocol. These procedures were (A) all metrics were monitored except cardiac output, (B) cardiac output was measured as well and (C) the anaesthesia unit was replaced with a 5 l rebreathing bag filled with 500 ml of room air immediately after an exhalation and before beginning CPR. Cardiac output measurements were omitted from procedure A to evaluate for interactions with the time series oxygenation measurement under CPR conditions. To observe potential effects of differences in ventilation on circulatory metrics the availability of fresh air was limited in procedure C. As indicated in figure 2, the CPR type and the run procedure were randomised.
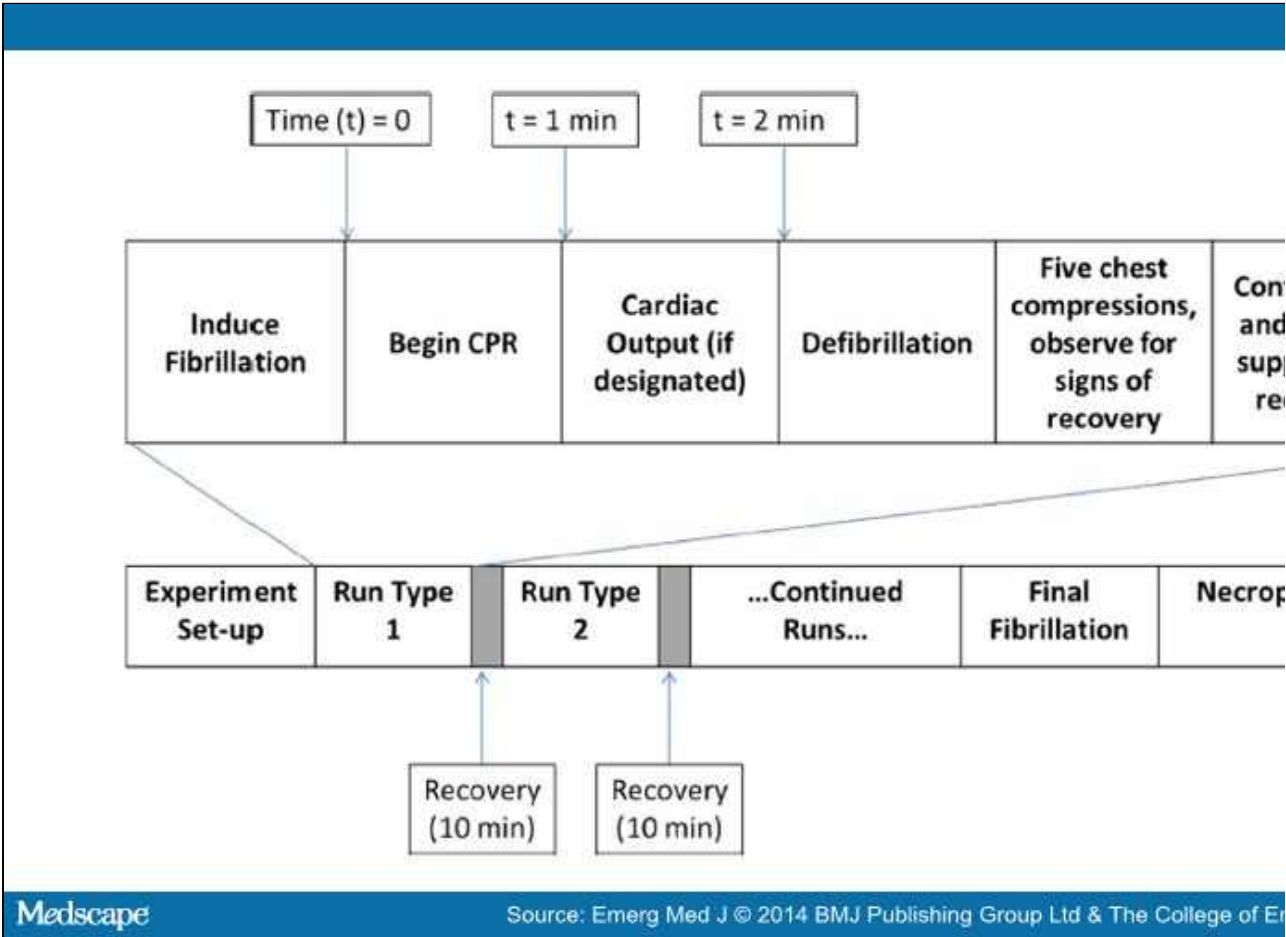

Figure 2.

Experiment design for protocol 1 with run types varying in cardiopulmonary resuscitation (CPR) technique (only abdominal compression or chest compression-CPR), measurement of the cardiac output with the other predictive metrics, and the use of a rebreathing bag.

CPR Run Protocol 2

Protocol 2 focused on refining blood oxygen measurements by preventing spontaneous respiration, as shown in figure 3. Given the short duration of these experimental runs, it was deemed necessary to separate the effects of spontaneous and compression-induced ventilations, as even small gasps may increase pO_2 levels.[26] To suppress respiration, the subjects were hyperventilated using a positive-pressure ventilator (Model 2000, Hallowell EMC, Pittsfield, Massachusetts, USA) for ~5 min immediately before each run

to an $EtCO_2 < 30$ mm Hg. In this protocol, cardiac output was measured with every run and the rebreathing bag was not used. The CPR technique used during the first run was determined at random, and the other technique used in the second run. The third was determined at random, and so on for repeated pairings of CPR techniques.
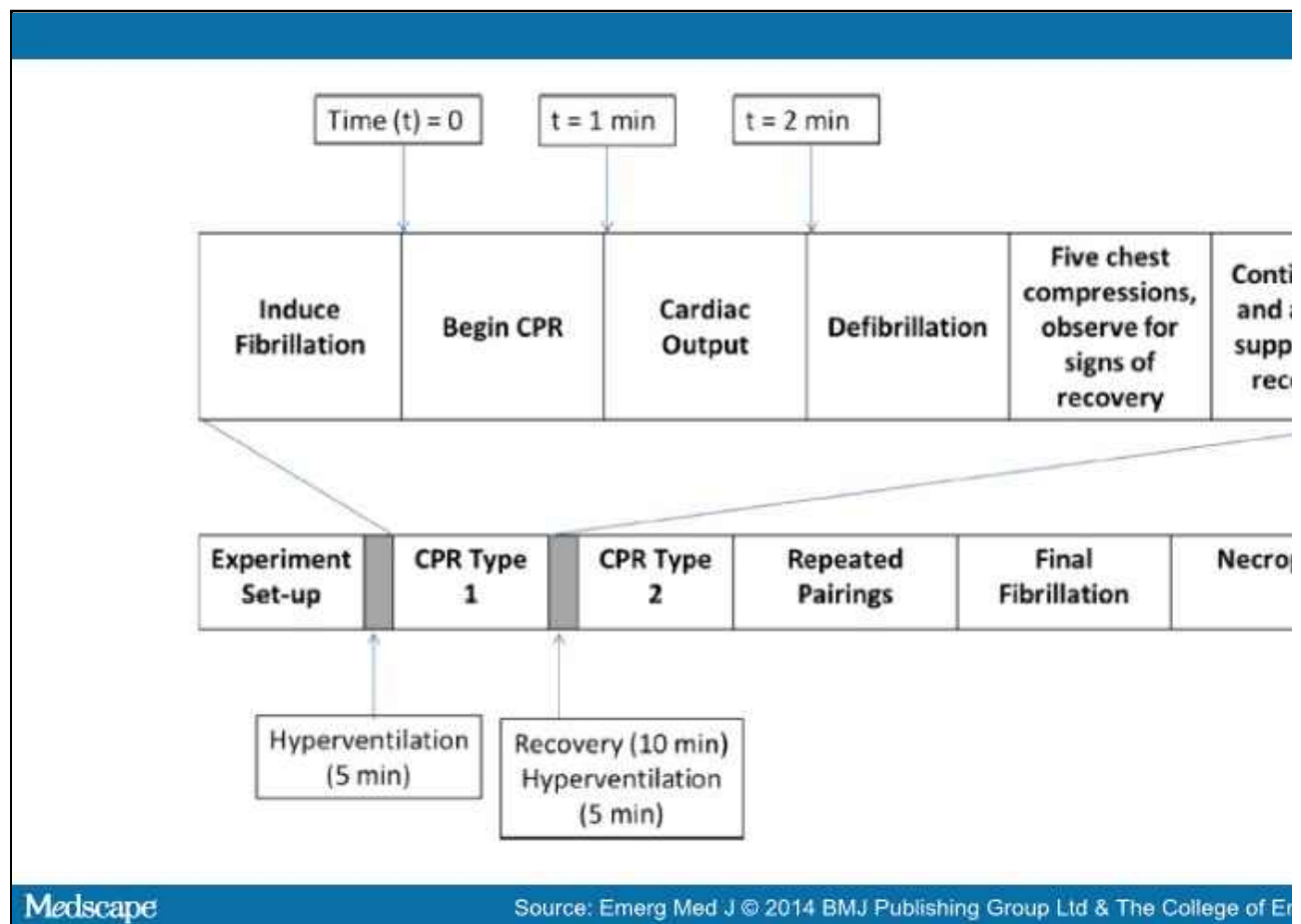

Figure 3.

Experiment design for protocol 2 with hyperventilation period preceding application of only abdominal compression or chest compression-cardiopulmonary resuscitation.

Data Analysis

Baseline values were taken during stable dynamics immediately before the induction of fibrillation, but were not included in analysis. Aortic pressure, right atrial pressure, lung air flow, $EtCO_2$, and carotid blood flow measurements were recorded as continuous waveforms. Mean CPP, MAV and right carotid flow rate during CPR were calculated by averaging over the 120 ± 10 s of application. Stable $EtCO_2$ measurements were taken less than 5 s before defibrillation. $EtCO_2$ was excluded if an extremely low MAV (<-200 ml/min/kg) was observed (artificially low EtCO2s may result from very low ventilation rates). Dead space was calculated as previously described, and negative MAVs set to zero.[13] Arterial pO2 was sampled at 2-s intervals, and noise was filtered using a five-point running average. Arterial pO_2 measurements were only analysed for protocol 2, as spontaneous breathing was confounding, and cardiac output saline injections were not found to interfere. The drop in pO2 was defined as the difference between the average pO2 for the first and last 6 s of each run. As the drop in arterial pO2 may be dependent

on the pO_2 at which it began, all drops were normalised by dividing by the baseline pO_2. The time from a successful defibrillation until ROSC was recorded, with ROSC defined as a haemodynamic state requiring no additional assistance. Measurements were excluded from analysis if they were only obtained for one CPR technique in a subject, or if data-compromising complications occurred. Two to six runs were performed on each subject.

Statistical significance was evaluated by fitting a general linear model (GLM) with fixed effects of CPR type, ventilation type (rebreathing/anaesthesia circuit), protocol type, and the number of previous resuscitations (run ordering), while the individual subjects, nested within each protocol, were random effects. No difference was found between run procedures A and B in protocol 1 (inclusion of cardiac output), so this was not included as a factor. First-order interactions were included, and the significance of each main and interaction effect determined. Treatment effects were then determined using a model including the main effects and any previously determined significant interactions. p Values less than 0.05 were considered significant.

Results

Typical carotid blood flow and arterial pO_2 dynamics for OAC-CPR and CC-CPR are shown in figure 4. The flow waveform for OAC-CPR does not exhibit the rapid spikes that are evident with CC-CPR. A decrease in pO_2 is evident over the course of the fibrillation episode for both types of CPR. Larger oscillations in the arterial pO_2 at the start of each run are due to greater sensitivity to environmental conditions at higher O_2 levels.
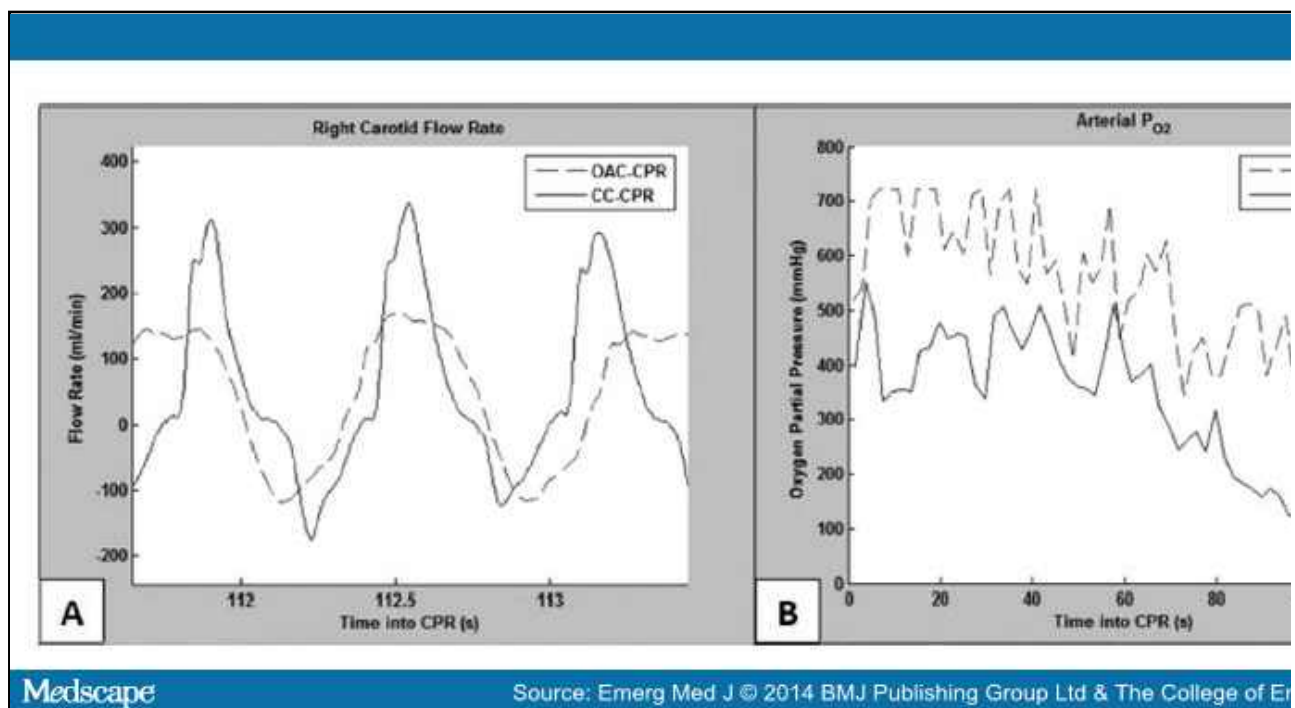

Figure 4.

Examples of (a) the flow waveform for the right carotid artery flow rate and (b) the change in arterial oxygen partial pressure as read by the FOXY sensor.

Using the GLM, differences in treatment effects were determined as follows. The intersubject variation significantly affected all metrics except carotid flow; run sequence was significant for $EtCO_2$ and cardiac output. Rebreathing significantly increased $EtCO_2$ by 36.6 mm Hg, and non-significantly increased time to ROSC by 39 s (SE=25.3 s). The interaction effect of run sequence and rebreathing was significant for the metric of $EtCO_2$ and the time until ROSC, while the interaction effect of protocol and run sequence was significant for the metric of MAV. The first interaction effect likely points to a change in the lung architecture over time, affecting the effect of the rebreathing gas, most likely due to pulmonary oedema/broken ribs. The second interaction effect likely points to the fact that spontaneous respirations were repressed in the second study.

The major findings of the GLM for the CPP, carotid blood flow, MAV and cardiac output are indicated graphically in figure 5. CPP and MAV were greater for OAC-CPR than CC-CPR, although no difference was found for cardiac output. Carotid flow rate, however, was higher for CC-CPR with an average at 50 ml/min compared with 40.7 ml/min for OAC-CPR.
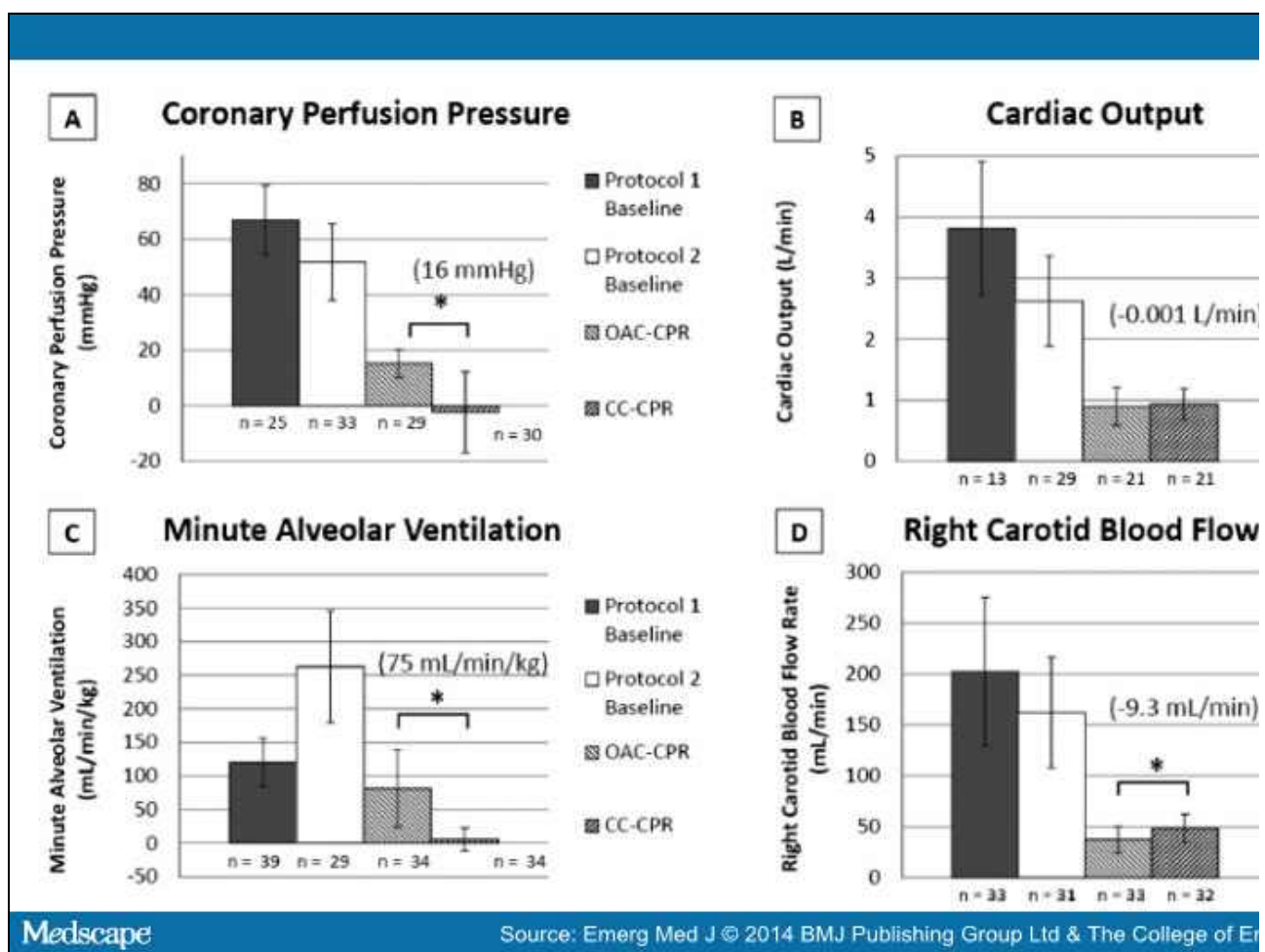

Figure 5.

Comparisons of datasets used in the general linear model for mechanical metrics. Error bars denote SD, and the sample size in each set is given near the bottom of the bars. Asterisks denote a significant difference between cardiopulmonary resuscitation (CPR) treatment effects (p<0.05), with the difference between the only abdominal compression

(OAC)-CPR and the chest compression (CC)-CPR treatment effects (OAC effect minus CC effect) given in parentheses.

The major findings of the GLM for gas exchange and resuscitation metrics are shown in figure 6. End-tidal CO_2 was significantly lower during OAC-CPR than CC-CPR and was comparable with the level during hyperventilation for protocol 2. Arterial pO_2 decreased by nearly 35% during CC-CPR but only 12% for OAC-CPR. However, it was qualitatively more difficult to resuscitate subjects after OAC-CPR, as quantified by a greater delay in ROSC (+70 s).
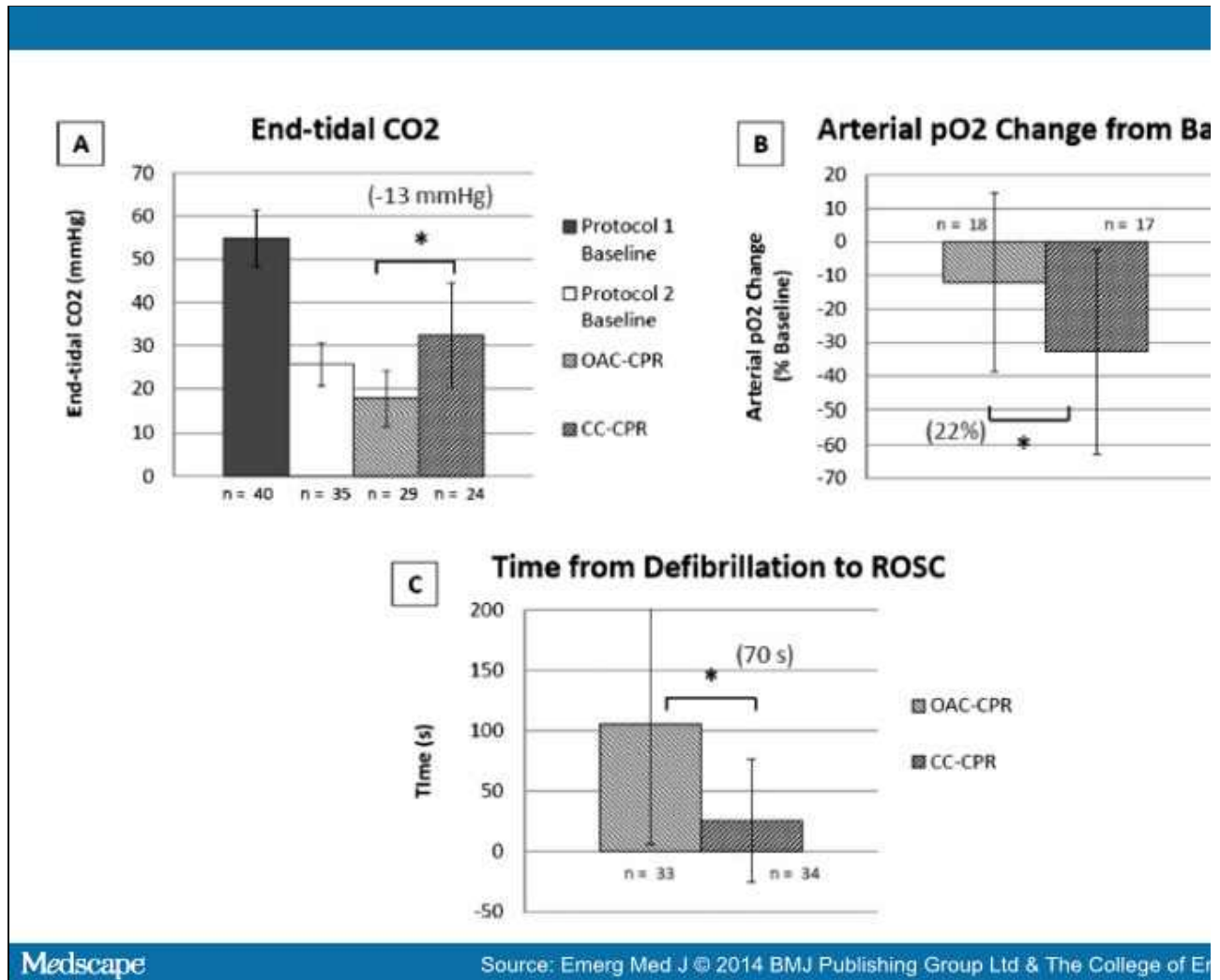

Figure 6.

Comparisons of datasets used in the general linear model for metrics of gas exchange and ease of resuscitation. Error bars denote SD, and the sample size in each set is given near the bottom of the bars. Asterisks denote a significant difference between cardiopulmonary resuscitation (CPR) treatment effects ($p<0.05$), with the difference between the only abdominal compression (OAC)-CPR and the chest compression (CC)-CPR treatment effects (OAC effect minus CC effect) given in parentheses.

For some subjects, unexpected negative CPP values were observed during CC-CPR. We hypothesized that such values were due to permanent physical changes in the chest

structure. To investigate, the aortic and right atrial pressures in the first CC-CPR run for each subject were analysed, and divided by whether or not a negative CPP was observed during that run. The divided sets were compared by t test, as shown in . The mean pressures for the first OAC-CPR run were also calculated. While the mean aortic pressure did not differ between the groups, the mean right atrial pressure was significantly higher for subjects in which a negative CPP was observed. Additionally, 4 out of 5 of the subjects with a negative initial CPP experienced broken ribs, compared with 5 of the 11 subjects that had a positive initial CPP.

Table 1. Comparison of mean aortic and mean right atrial pressures of subjects under different forms of CPR and CPP values

	Mean aortic pressure (mm Hg)	Mean right atrial pressure (mm Hg)	Number of subjects
OAC-CPR	36.2	22.6	16
CC-CPR, positive CPP	23.1	16.6*	11
CC-CPR, negative CPP	21.2	44.6*	5

Asterisks (*) denote that the two values were significantly different (p<0.05).

CC, chest compression; CPP, coronary perfusion pressure; CPR, cardiopulmonary resuscitation; OAC, only abdominal compression.

A necropsy was performed on each of the 17 subjects after the completion of all runs. Of these 17, only one showed signs of abdominal damage. It should be noted that the abdominal damage (liver contusions and a ruptured bladder) was observed after 20 min of OAC-CPR and 40 min of CC-CPR were applied attempting to resuscitate the subject; 12 out of 17 of the subjects had significant remodelling or broken ribs, and 16 of the 17 had pulmonary oedema. Some remodelling of the chest was normally observed following the first run of CC-CPR in the subjects with such damage.

Discussion

The differences in treatment effects for CPP and MAV with regard to OAC-CPR and CC-CPR are similar to those determined in previous studies.[12,13] Though negative CPPs are an unusual observation, these appear to result from increased right atrial pressure alone, and were normally observed along with broken ribs, suggesting that they are caused by more direct compression of the right side of the heart. A significant difference between cardiac outputs in OAC-CPR and CC-CPR was not observed (figure 5). This discrepancy between perfusion pressure and flow in alternative CPR techniques has been observed previously.[27]

As the MAV for OAC-CPR is greater than for CC-CPR, with a similar cardiac output, higher stable arterial pO_2 would be expected.[28] While in this study, we did not continue CPR long enough to observe a stable oxygenation state, we found that the drop in arterial pO_2 was less for OAC-CPR than CC-CPR, suggesting that OAC-CPR would provide greater stable blood oxygenation. The drop in O_2 as measured in the external shunt is dependent on the blood flow to the shunt, but the observation of similar cardiac output suggests that this effect is small.

As seen in figure 6, the lower $EtCO_2$ found in OAC-CPR indicates that the subjects were hyperventilated relative to CC-CPR, and potentially hypocapnic. The ventilations produced by the two CPR techniques were very different, so $EtCO_2$ could not be used as an indicator of cardiac output. Rather, it is important to consider hyperventilation-induced hypocapnia as a potential interaction, which can cause vasoconstriction of the coronary[29] and the cerebral arteries,[30,31] reducing vital organ perfusion. In this study, it is unclear how vascular resistance may have been affected by hyperventilation, due to the transience of the $EtCO_2$ and blood flow rates. However, the hypercapnia in rebreathing runs did not show a significant effect on the haemodynamics measured over these short intervals.

Carotid blood flow was found to be greater for CC-CPR than OAC-CPR, as shown in figure 5. This is a significant finding, though it is important to note the complex relation between carotid flow and cerebral flow during CPR. Cerebral blood flow is influenced by the intrathoracic pressure, which may be transmitted to the intracranial space by non-valved vertebral blood vessels and the cerebrospinal fluid,[21] and increased intracranial pressures may shunt blood flow through the extracranial vessels.[20] Since CC-CPR and OAC-CPR apply force differently, it is likely that cerebral flow is affected by differences in the intrathoracic pressure variations. Speculatively, CC-CPR could be expected to produce higher intrathoracic pressures, diminishing cerebral flow compared with OAC-CPR, but the complexity of pressure transduction to the intracranial space, differences in perfusion pressure, and dynamic interactions prevent a clear interpretation based on the current data. Along with potential effects of hypocapnia, a large number of factors may impact cerebral blood flow, and future studies should measure cerebral blood flow by more direct methods, such as microsphere injection.[32,33]

There was a greater delay in ROSC for OAC-CPR (figure 6), despite higher CPP and similar cardiac output (figure 5). The mechanisms underlying this observation are unclear, but related factors may involve the loading of the heart chambers. Geometric conditions in the heart may affect defibrillation, however, we observed only a small, non-significant, decrease in first shock success with OAC-CPR.[34] An engorged heart increases the preload in heart tissue upon resuscitation, and unloading the heart could require more chest compressions prior to defibrillation than were given in this study.[32] Also related to heart loading, a greater ventricular-thoracic pressure difference could impede myocardial blood flow[35] and increase O_2 consumption during fibrillation.[36] The current data does not directly address these hypotheses, and careful investigation of the mechanics of heart tissue under OAC- and CC-CPR conditions is necessary to investigate this effect.

The limited abdominal damage observed was consistent with a prior study with 20 subjects given 2 min of OAC-CPR[14] and studies on interposed abdominal compression CPR (IAC-CPR).[37–39] Abdominal compressions, at least in short durations, appear unlikely to cause internal damage.

Limitations

This study was designed for an intrasubject comparison in a laboratory setting, to elucidate differences in the mechanisms underlying CC- and OAC-CPR. The results at this stage are not intended to be generalised to bystander-initiated CPR in humans, but to explore the transduction of compression-induced pressures to vital organ perfusion. Herein, we used the compression rates that performed best in previous studies; however, there may be a more optimal combination of parameters (rate and duty cycle) that were not explored within this study. For consistency with previous studies and to approximate

bystander CPR application, a duty cycle of 50% was used for both CC-CPR and OAC-CPR, though bystander-based CPR may result in a lower duty cycle.[40] While a theoretical model of OAC-CPR suggests that a higher duty cycle may be more optimal,[41] a preliminary study observed equivalent CPP in both 50% and 70% duty cycles (unpublished data from four porcine subjects). The predictive outcome metrics used indicate internal conditions difficult to observe, but do not directly indicate survival or neurological outcome.

Conclusions

In this study, we noted significant tradeoffs between predictive outcome pressure and ventilation metrics with perfusion metrics for OAC-CPR and CC-CPR. Compared with CC-CPR, OAC-CPR produces similar cardiac output, greater blood oxygenation and greater CPP and MAV. Despite these benefits, OAC-CPR was characterised by lower carotid flow rates and a greater difficulty of resuscitation. These contrasting observations suggest that traditional predictive metrics, such as CPP and MAV which have been validated for CC-CPR, may not be directly applicable in the evaluation of techniques that generate pressures via alternative sites of compression or manipulations. Future studies will compare the regional blood flows to the cerebrum and myocardium generated by these two CPR techniques.

References

1. Thompson RG, Hallstrom AP, Cobb LA. Bystander-initiated cardiopulmonary resuscitation in the management of ventricular fibrillation. *Ann Intern Med* 1979;90:737.

2. Herlitz J, Ekstrom L, Wennerblom B, *et al*. Effect of bystander initiated cardiopulmonary-resuscitation on ventricular-fibrillation and survival after witnessed cardiac-arrest outside hospital. *Br Heart J* 1994;72:408–12.

3. Waalewijn RA, Tijssen JGP, Koster RW. Bystander initiated actions in out-of-hospital cardiopulmonary resuscitation: Results from the amsterdam resuscitation study (arrest). *Resuscitation* 2001;50:273–9.

4. Boucek CD, Phrampus P, Lutz J, *et al*. Willingness to perform mouth-to-mouth ventilation by health care providers: A survey. *Resuscitation* 2009;80:849–53.

5. Locke CJ, Berg RA, Sanders AB, *et al*. Bystander cardiopulmonary-resuscitation—concerns about mouth-to-mouth contact. *Arch Intern Med* 1995;155:938–43.

6. Hamasu S, Morimoto T, Kuramoto N, *et al*. Effects of BLS training on factors associated with attitude toward CPR in college students. *Resuscitation* 2009;80:359–64.

7. Travers AH, Rea TD, Bobrow BJ, *et al*. Part 4: CPR overview: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation* 2010;122:S676–84.

8. Yannopoulos D, Matsuura T, McKnite S, *et al*. No assisted ventilation cardiopulmonary resuscitation and 24-hour neurological outcomes in a porcine model of cardiac arrest. *Crit Care Med* 2010;38:254–60.

9. Bobrow BJ, Spaite DW, Berg RA, *et al*. Chest compression–only CPR by lay rescuers and survival from out-of-hospital cardiac arrest. *JAMA-J Am Med Assoc* 2010;304:1447–54.

10. Iwami T, Kawamura T, Hiraide A, *et al*. Effectiveness of bystander-initiated cardiac-only resuscitation for patients with out-of-hospital cardiac arrest. *Circulation* 2007;116:2900–7.

11. Nagao K, Kikushima K, Sakamoto T, *et al*. Cardiopulmonary resuscitation by bystanders with chest compression only (SOS-KANTO): An observational study. *Lancet* 2007;369:920–6.

12. Geddes LA, Rundell A, Lottes A, *et al*. A new cardiopulmonary resuscitation method using only rhythmic abdominal compression—a preliminary report. *Am J EmergMed* 2007;25:786–90.

13. Pargett M, Geddes LA, Otlewski MP, *et al*. Rhythmic abdominal compression CPR ventilates without supplemental breaths and provides effective blood circulation. *Resuscitation* 2008;79:460–7.

14. Xanthos T, Bassiakou E, Dontas I, *et al*. Abdominal compressions do not achieve similar survival rates compared with chest compressions: An experimental study. *AmJ Emerg Med* 2011;29:665–9.

15. Idris A, Becker L, Fuerst R, *et al*. Effect of ventilation on resuscitation in an animal model of cardiac arrest. *Circulation* 1994;90:3063–9.

16. Kern KB, Ewy GA, Voorhees WD, *et al*. Myocardial perfusion-pressure—a predictor of 24-hour survival during prolonged cardiac-arrest in dogs. *Resuscitation* 1988;16:241–50.

17. Paradis NA, Martin GB, Rivers EP, *et al*. Coronary perfusion-pressure and the return of spontaneous circulation in human cardiopulmonary resuscitation. *JAMA-J AmMed Assoc* 1990;263:1106–13.

18. Niemann JT, Criley JM, Rosborough JP, *et al*. Predictive indexes of successful cardiac resuscitation after prolonged arrest and experimental cardiopulmonary resuscitation. *Ann Emerg Med* 1985;14:521–8.

19. Yeh ST, Cawley RJ, Aune SE, *et al*. Oxygen requirement during cardiopulmonary resuscitation (CPR) to effect return of spontaneous circulation. *Resuscitation* 2009;80:951–5.

20. Koehler R, Chandra N, Guerci A, *et al*. Augmentation of cerebral perfusion by simultaneous chest compression and lung inflation with abdominal binding after cardiac arrest in dogs. *Circulation* 1983;67:266–75.

21. Guerci A, Shi A, Levin H, *et al*. Transmission of intrathoracic pressure to the intracranial space during cardiopulmonary resuscitation in dogs. *Circ Res* 1985;56:20–30.

22. Rogers MC, Weisfeldt ML, Traystan RJ. Cerebral blood-flow during cardiopulmonary resuscitation. *Anesth Analg* 1981;60:73–5.

23. Krischer JP, Fine EG, Davis JH, *et al*. Complications of cardiac resuscitation. *Chest*

1987;92:287–91.

24. Part 3: Overview of CPR. *Circulation* 2005;112:IV-12–18.

25. Weisfeldt ML, Becker LB. Resuscitation after cardiac arrest: A 3-phase time-sensitive model. *JAMA-J Am Med Assoc* 2002;288:3035–8.

26. Noc M, Weil M, Sun S, *et al*. Spontaneous gasping during cardiopulmonary resuscitation without mechanical ventilation. *Am J Respir Crit Care Med* 1994;150:861–4.

27. Burnett AM, Segal N, Salzman JG, *et al*. Potential negative effects of epinephrine on carotid blood flow and ETCO2 during active compression-decompression CPR utilizing an impedance threshold device. *Resuscitation* 2012;83:1021–4.

28. Boron WF, Boulpaep EL. Medical physiology: a cellular and molecular approach. 2005;1224:700–11.

29. Case R, Greenberg H. The response of canine coronary vascular resistance to local alterations in coronary arterial PCO2. *Circ Res* 1976;39:558–66.

30. Poulin MJ, Liang PJ, Robbins PA. Fast and slow components of cerebral blood flow response to step decreases in end-tidal CO2in humans. *J Appl Physiol* 1998;85:388–97.

31. Dineen NE, Brodie FG, Robinson TG, *et al*. Continuous estimates of dynamic cerebral autoregulation during transient hypocapnia and hypercapnia. *J Appl Physiol* 2010;108:604–13.

32. Adams JA, Mangino MJ, Bassuk J, *et al*. Novel CPR with periodic Gz acceleration. *Resuscitation* 2001;51:55–62.

33. Hoekstra OS, Vanlambalgen AA, Groeneveld ABJ, *et al*. Abdominal compressions increase vital organ perfusion during CPR in dogs—relation with efficacy of thoracic compressions. *Annals of Emergency Medicine* 1995;25:375–85.

34. Ott P, Reiter MJ. Effect of ventricular dilatation on defibrillation threshold in the isolated perfused rabbit heart. *J Cardiovasc Electrophysiol* 1997;8:1013–19.

35. Klocke FJ, Ellis AK. Control of coronary blood-flow. *Annu Rev Med* 1980;31:489–508.

36. Monroe RG, French G. Ventricular pressure-volume relationships and oxygen consumption in fibrillation and arrest. *Circ Res* 1960;8:260–6.

37. Kern KB, Carter AB, Showen RL, *et al*. CPR-induced trauma: Comparison of three manual methods in an experimental model. *Ann Emerg Med* 1986;15:674–9.

38. Howard M, Carrubba C, Foss F, *et al*. Interposed abdominal compression-CPR: Its effects on parameters of coronary perfusion in human subjects. *Ann Emerg Med* 1987;16:253–9.

39. Waldman PJ, Walters BL, Grunau CFV. Pancreatic injury associated with interposed abdominal compressions in pediatric cardiopulmonary resuscitation. *Am J EmergMed* 1984;2:510–12.

40. Maier GW, Tyson GS, Olsen CO, *et al*. The physiology of external cardiac massage: High-impulse cardiopulmonary resuscitation. *Circulation* 1984;70:86–101.

41. Jung E, Lenhart S, Protopopescu V, *et al*. Optimal control applied to a thoraco-abdominal CPR model. *Math Med Biol* 2008;25:157–70.

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Raw data will be made available upon request to the corresponding author.

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