Resuscitation 86 (2015) 49–53

Contents lists available at ScienceDirect

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

Experimental paper

A tourniquet assisted cardiopulmonary resuscitation augments myocardial perfusion in a porcine model of cardiac arrest☆,☆☆

Zhengfei Yang[a,d], David Tang[a,e], Xiaobo Wu[a], Xianwen Hu[a], Jiefeng Xu[a], Jie Qian[a], Min Yang[a], Wanchun Tang[a,b,c,d,*]

[a] Weil Institute of Critical Care Medicine, Rancho Mirage, CA, United States
[b] Keck School of Medicine of the University of Southern California, Los Angeles, CA, United States
[c] UC San Diego School of Medicine, San Diego, CA, United States
[d] Sun Yat-sen Memorial Hospital, Sun Yat-sen University, Guangzhou, China
[e] Eisenhower Medical Center, Rancho Mirage, CA, United States

ARTICLE INFO

Article history:
Received 20 June 2014
Received in revised form
27 September 2014
Accepted 6 October 2014

Keywords:
Cardiopulmonary resuscitation
Cardiac arrest
Hemodynamics
Myocardial perfusion
Peripheral vascular resistance

ABSTRACT

Objective: During cardiopulmonary resuscitation (CPR), myocardial blood flow generated by chest compression rarely exceeds 35% of its normal level. Cardiac output generated by chest compression decreases gradually with the prolongation of cardiac arrest and resuscitation. Early studies have demonstrated that myocardial blood flow during CPR is largely dependent on peripheral vascular resistance. In this study, we investigated the effects of chest compression in combination with physical control of peripheral vascular resistance assisted by tourniquets on myocardial blood flow during CPR.

Methods: Ventricular fibrillation was induced and untreated for 7 min in ten male domestic pigs weighing between 33 and 37 kg. The animals were then randomized to receive CPR alone or a tourniquet assisted CPR (T-CPR). In the CPR alone group, chest compression was performed by a miniaturized mechanical chest compressor. In the T-CPR group, coincident with the start of resuscitation, the thin elastic tourniquets were wrapped around the four limbs from the distal end to the proximal part. After 2 min of CPR, epinephrine (20 μg/kg) was administered via the femoral vein. After 5 min of CPR, defibrillation was attempted by a single 150 J shock. If resuscitation was not successful, CPR was resumed for 2 min before the next defibrillation. The protocol was continued until successful resuscitation or for a total of 15 min. Five minutes after resuscitation, the elastic tourniquets were removed. The resuscitated animals were observed for 2 h.

Results: T-CPR generated significantly greater coronary perfusion pressure, end-tidal carbon dioxide and carotid blood flow. There was no difference in both intrathoracic positive and negative pressures between the two groups. All animals were successfully resuscitated with a single shock in both groups. There were no significant changes in hemodynamics observed in the animals treated in the T-CPR group before-and-after the release of tourniquets at post-resuscitation 5 min.

Conclusions: T-CPR improves myocardial and cerebral perfusion during CPR. It may provide a new and convenient method for augmenting myocardial and cerebral blood flow during CPR.

1. Introduction

The main goal of cardiopulmonary resuscitation (CPR) is to provide forward blood flow to the heart and brain until spontaneous circulation is restored.[1] Both experimental and clinical studies have consistently demonstrated that the success of resuscitation during CPR is largely dependent on the efficacy of cardiac output generated by chest compression.[2–5] However, cardiac output and myocardial perfusion generated by conventional CPR rarely exceeds 30% and 50% of normal levels, respectively.[6,7] In addition, cardiac output gradually decreases during prolonged CPR in spite

☆ A Spanish translated version of the summary of this article appears as Appendix in the final online version at http://dx.doi.org/10.1016/j.resuscitation.2014.10.009.
☆☆ Protocol number: P1301.

* Corresponding author at: Weil Institute of Critical Care Medicine, 35-100 Bob Hope Drive, Rancho Mirage, CA 92270, United States.
E-mail addresses: yangzhengfei@vip.163.com (Z. Yang), dtangips@aol.com (D. Tang), mike_wu@weiliccm.org (X. Wu), xianwenhu001@sina.cn (X. Hu), jeff_xu11@gmail.com (J. Xu), qjssmu@gmail.com (J. Qian), 456ym@163.com (M. Yang), wanchun.tang@me.com (W. Tang).

http://dx.doi.org/10.1016/j.resuscitation.2014.10.009
0300-9572/

of continuous precordial compression.[8] Therefore, global myocardial ischemia is persistent during the conventional resuscitation effort.[9,10] Coronary perfusion pressure (CPP), the most reliable predictor of the success of defibrillation and restoration of spontaneous circulation, is highly correlated with coronary blood flow and, therefore, myocardial perfusion.[11,12] Early investigations have demonstrated that the magnitude of myocardial perfusion during CPR is highly dependent on peripheral vascular resistance, which can be enhanced by vasopressor drugs such as epinephrine or the compression and binding of the abdomen.[13–20]

However, our previous study has demonstrated that epinephrine significantly increases the severity of post-resuscitation myocardial dysfunction. This is a result from its β-adrenergic effect during CPR.[21] Two clinical studies further demonstrated that administration of epinephrine during resuscitation compromises the outcomes of CPR.[22,23] In addition, several studies in the early 1980s have shown disappointing results of hemodynamics and survival on the effects of physical control of peripheral vascular resistance implemented by military anti-shock trousers (MAST) or abdominal binder (AB).[24,25] However, in their studies, the detrimental effects including impediment of ventilation, inadequate gas exchange, proportionate increase in diastolic right atrial pressure (RAP) and intrathoracic pressure (ITP) may be due to the increase in intra-abdominal pressure and the limited movement of diaphragm during utilization of MAST or AB.

An ideal method to increase peripheral vascular resistance and therefore improve the perfusion to vital organs during CPR should be simple, safe and noninvasive. An Esmarch tourniquet is a constricting or compressing device, specifically a bandage, used to control venous and arterial circulation to an extremity. It has been used routinely to limit or decrease blood flow in surgery. We developed a method to physically control the peripheral vascular resistance by tightening the elastic tourniquets from distal to proximal around the four limbs.

In the present study, we compared the effects of a tourniquet assisted CPR (T-CPR) on myocardial perfusion during CPR in a porcine model. We hypothesized that T-CPR would improve myocardial perfusion during CPR.

2. 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 (8th edition; Washington, DC, National Academic Press, 2011). The protocol was approved by the Institutional Animal Care and Use Committee of the Weil Institute of Critical Care Medicine.

2.1. Animal preparation

Ten male domestic pigs weighing 34 ± 2 kg were fasted overnight except for free access to water. Anesthesia was initiated by intramuscular injection of ketamine (20 mg/kg), followed by intravascular injection with sodium pentobarbital (30 mg/kg). When the animals awakened or showed signs of restlessness, an additional dose of sodium pentobarbital (8 mg/kg) was injected, or at intervals of approximately 1 h to maintain anesthesia, if necessary. A cuffed endotracheal tube was advanced into the trachea and the 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 F_{iO2} of 0.21. End-tidal carbon dioxide ($ETCO_2$) was monitored with an infrared capnometer (NPB-70, Nellcor Puritan Bennett Inc., Pleasanton, CA).

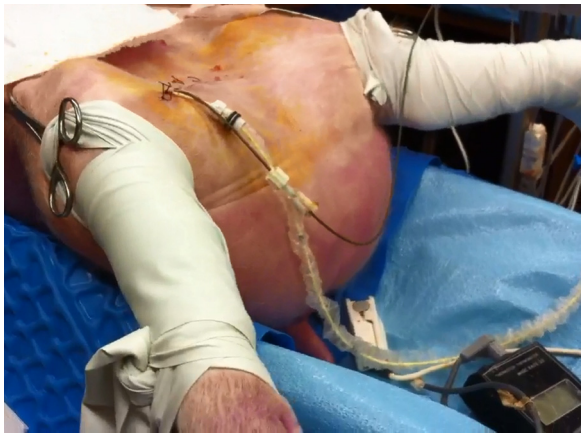

Fig. 1. A sketch of physical control of peripheral vascular resistance by tightening the elastic tourniquets from distal to proximal around the four limbs.

Respiratory frequency was adjusted to maintain $ETCO_2$ between 35 and 40 mm Hg before cardiac arrest. For recording electrocardiogram (ECG), three adhesive electrodes were applied to the shaved skin of the proximal right-and-left, upper-and-lower limbs. For the measurement of aortic pressure and the collection of the blood samples, a 5 F transducer-tipped Millar catheter (Model SPC-450S, Millar Instruments Inc., Houston, TX) was advanced from the right femoral artery into the thoracic aorta. For the measurements of RAP and core blood temperature, a 7 F catheter (Abbott Critical Care, Salt Lake City, UT) was advanced from the right femoral vein and directed into the right atrium. Both catheters were flushed intermittently with saline containing 5 IU bovine heparin per ml. For inducing ventricular fibrillation (VF), a 5 F pacing catheter (EP Technologies Inc., Mountain View, CA) was advanced from the right external jugular vein into the right ventricle. Carotid blood flow (CBF) was continuously measured with the aid of a flow probe (Ultrasonic Blood Flow Meter, T420, Transonic Systems Inc, Ithaca, NY) positioned around the right common carotid artery. The position of all catheters was confirmed by characteristic pressure morphology and with fluoroscopy. For the measurement of ITP, an additional 5 F Millar catheter was advanced from the incisor teeth into the esophagus for a distance of 35 cm. The piston of the compressor was positioned in the midline at the level of the fifth interspace. Body temperature was maintained at 37.5 ± 0.5 °C with the aid of a cooling/warm blanket (Blanket ROL, Cincinnati Sub-Zero Products, Cincinnati, OH) throughout the entire experiment.

2.2. Experimental procedures

Fifteen minutes prior to inducing cardiac arrest, baseline measurements were obtained. The animals were then randomized by the *Sealed Envelope Method* to receive CPR alone or T-CPR. Cardiac arrest, due to VF, was induced by 1 mA alternating current through a 5 F pacing catheter and into the right ventricular cavity. Mechanical ventilation was discontinued after the onset of VF. Prior to initiating the resuscitation procedure, the pacing catheter was withdrawn to avoid heart injury during chest compression. After 7 min of untreated VF, CPR was performed. The mechanical chest compressor (MCC) was programmed to provide 100 compressions-per-minute. The compression depth was adjusted to decrease the anterior–posterior diameter of the chest by 25%. For the T-CPR group, during fibrillation, thin elastic Esmarch tourniquets were simultaneously wrapped around the four limbs from the distal end to the proximal part as tight as possible (Fig. 1). Coincident with the start of precordial compression, all animals were mechanically ventilated with a tidal volume of 15 mL/kg and F_{iO2} of 1.0, with

a rate of 10 breaths-per-minute. After 2 min of CPR, epinephrine (20 μg/kg) was administrated via the femoral vein. After 5 min of CPR, defibrillation was attempted with a single 150 J biphasic electrical shock delivered between the conventional right infraclavicular electrode and the apical electrode with a Zoll E-Series defibrillator (Zoll Medical Corporation, Chelmsford, MA). If an organized rhythm with a mean aortic pressure of >50 mm Hg persisted for an interval of 5 min or more, the animal was regarded as successfully resuscitated, also known as the return of spontaneous circulation (ROSC). With failure to achieve ROSC, chest compression and ventilation were immediately resumed for 2 min prior to the attempt of an additional single shock. The procedure was repeated for a maximum of 5 cycles. Additional doses of epinephrine were given at an interval of 3 min after the first administration. If ROSC was not achieved, resuscitation maneuvers were terminated. When recurrent VF occurred within 30 min after ROSC, another 150-J countershock was attempted. After resuscitation, the animal was monitored for an additional 2 h. Mechanical ventilation was continued with 100% inspired oxygen for the first 30 min, 50% for the second 30 min and 21% thereafter. After a period of 2 h, post-resuscitation measurements were completed. All catheters were removed and wounds were surgically sutured. The animals were then euthanized with an intravenous injection of 150 mg/kg pentobarbital. A necropsy was routinely performed to identify any injuries to the bony thorax or the thoracic or abdominal viscera.

2.3. Measurements

Hemodynamics, $ETCO_2$, CBF and ECG were continuously measured and recorded on a PC-based data acquisition system, supported by CODAS/WINDAQ hardware/software (Computer Acquisition System, Cambridge, MA). The CPP was digitally computed from the differences in time-coincident diastolic aortic and right atrial pressures and displayed in real time. ITP was measured in real time during CPR. Acute ECG changes after CPR and defibrillation shocks were measured by continuous ECG recordings. Arterial blood gases including hemoglobin and lactate concentrations were measured with a Stat Profile pHOx Plus L analyzer (Model PHOX-plusL, Nova Biomedical Corporation, Waltham, MA) at baseline and then hourly after resuscitation.

2.4. Statistical analysis

Continuous variables were presented as mean ± SD when data were normally distributed or as a median (25th, 75th percentiles) when data were not normally distributed. Normal distribution was confirmed with the Kolmogorov–Smirnov test. Variables were compared with the parametric Student's *t*-test or the Mann–Whitney *U* test for nonparametric data. Comparisons between time-based measurements within each group and hemodynamics between the pre- and post-release of the tourniquets in T-CPR group were performed with a paired sample of the Student's *t*-test. For the comparison of categorical variables such as ROSC, the Fisher's exact test was used. A value of $p < 0.05$ was considered significant.

3. Results

Baseline heart rate, mean aortic pressure, RAP, $ETCO_2$, CBF, arterial PaO_2, and lactate did not differ significantly between the two groups (Table 1).

During CPR, consistent intrathoracic positive and negative pressures were observed in the two groups (Fig. 2). However, significantly greater CPP, $ETCO_2$ and CBF were achieved in the T-CPR group when compared with the CPR alone group (Figs. 3 and 4).

Table 1
Baseline characteristics.

	T-CPR	A-CPR	*p*-Value
Body weight, kg	35 ± 2	34 ± 1	0.51
Heart rate, beats/min	123 ± 9	118 ± 10	0.46
Mean aortic pressure, mm Hg	115 ± 9	110 ± 6	0.40
Right atrial pressure	2.8 ± 0.5	3.1 ± 0.6	0.53
End-tidal carbon dioxide, mm Hg	38.2 ± 1.5	37.6 ± 2.0	0.60
Carotid blood flow, ml/min	205 ± 18	199 ± 23	0.67
P_aO_2, mm Hg	100 ± 13	104 ± 14	0.68
Arterial lactate, mmol/L	1.1 ± 0.4	1.3 ± 0.6	0.56

Values are presented as mean ± SD.
CPR, cardiopulmonary resuscitation; T-CPR, tourniquet assisted CPR group; A-CPR, CPR alone group.

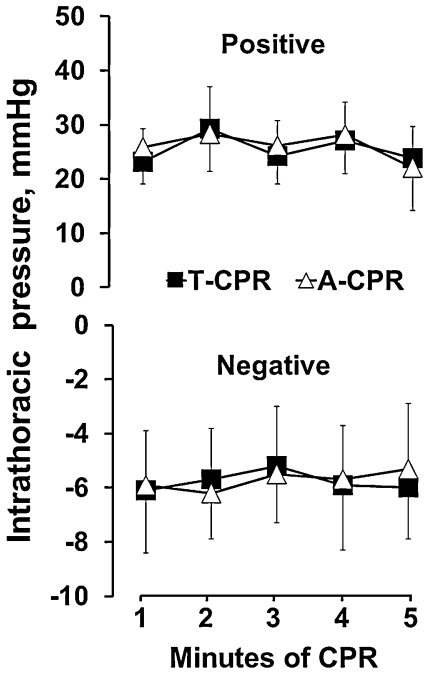

Fig. 2. The positive and negative intrathoracic pressure during cardiopulmonary resuscitation (CPR). T-CPR, tourniquet assisted CPR group; A-CPR, CPR alone group.

All animals were successfully resuscitated in both groups. There were no significant differences in the number of defibrillations, doses of epinephrine and duration of CPR (Table 2).

After 5 min of resuscitation, the tourniquets were released from the limbs of the animals treated with T-CPR group. There was no significant difference in the heart rate, mean aortic pressure and RAP before and after releasing tourniquets (Table 3).

At necropsy, no visceral injuries or rib fractures were observed in either group (Table 2).

4. Discussion

The present study demonstrated that T-CPR improved the hemodynamic efficacy of CPR in a porcine model. With this technique, we were able to observe augmentation on myocardial and cerebral perfusion during CPR. Significantly greater CPP, $ETCO_2$ and CBF were achieved in the T-CPR group when compared with the CPR alone group. There were no significant differences in positive and negative ITP between the two groups.

During cardiac arrest, when arterial and systemic venous pressures reach equilibrium, the circulation ceases. Re-establishment of myocardial blood flow is the most critical hemodynamic determinant for ROSC and minimizing complications. However, myocardial perfusion generated by conventional

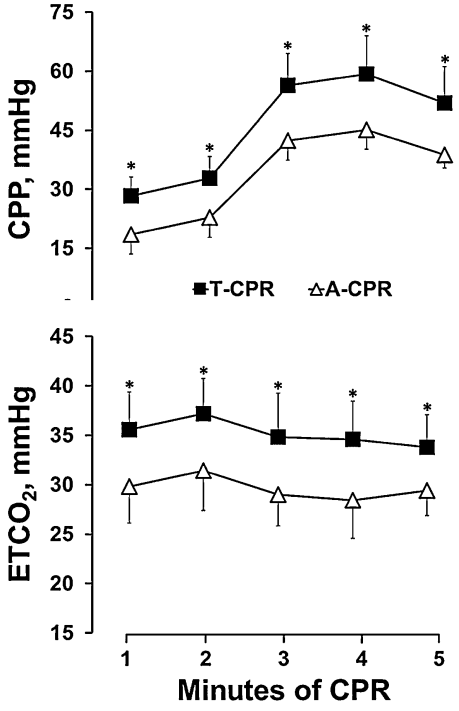

Fig. 3. The coronary perfusion pressure and the end-tidal carbon dioxide during cardiopulmonary resuscitation (CPR). CPP, coronary perfusion pressure; $ETCO_2$, end-tidal carbon dioxide; T-CPR, tourniquet assisted CPR group; A-CPR, CPR alone group. *$p < .05$ vs. the A-CPR group.

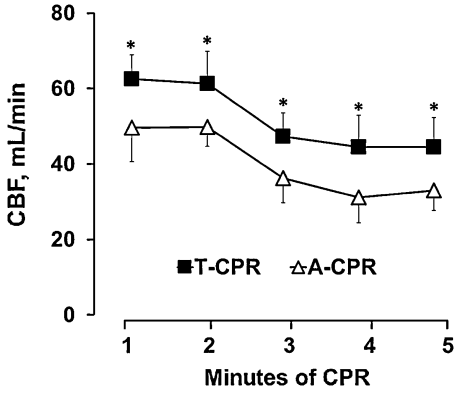

Fig. 4. The carotid blood flow during cardiopulmonary resuscitation (CPR). CBF, carotid blood flow; T-CPR, tourniquet assisted CPR group; A-CPR, CPR alone group. *$p < .05$ vs. the A-CPR group.

CPR fails to meet the need of the metabolic requirements of the fibrillating heart, especially during prolonged cardiac arrest. Augmentation of venous return and prevention of forward blood flow generated by chest compression from shunting to peripheral region could theoretically improve myocardial perfusion. In this study, we utilized the thin elastic tourniquets to restrict the blood flow to the four limbs. As a result, the cardiac output was increased by redistributing pooling blood from the periphery back to the central venous pool. Peripheral vascular resistance was enhanced to control preferential blood flow to vital organs.

A tourniquet has been used routinely to limit or decrease blood flow in surgery for bleeding control during trauma and first aid. It was demonstrated in adult patients that both limbs exsanguination performed by tourniquet banding increased circulating blood volume up to 800 ml and systemic vascular resistance.[26] Antegrade flow occurs in both the thoracic aorta and the pulmonary artery during the chest compression phase of CPR. Retrograde flow

Table 2
Selected hemodynamics and outcomes of cardiopulmonary resuscitation.

	T-CPR	A-CPR	*p*-Value
Hemodynamics at 1 min of CPR			
Systolic AP, mm Hg	75 ± 28	61 ± 21	<.05
Diastolic AP, mm Hg	34 ± 8	20 ± 6	<.05
Systolic RAP, mm Hg	51 ± 16	43 ± 12	<.05
Diastolic RAP, mm Hg	7 ± 3	3 ± 1	<.05
Hemodynamics at 5 min of CPR			
Systolic AP, mm Hg	92 ± 23	77 ± 18	<.05
Diastolic AP, mm Hg	59 ± 9	42 ± 7	<.05
Systolic RAP, mm Hg	63 ± 13	54 ± 10	<.05
Diastolic RAP, mm Hg	9 ± 4	6 ± 2	0.09
Duration of cardiopulmonary resuscitation, min	5 (5–5)	5 (5–5)	1
Numbers of defibrillation	2.4 (2–3)	2.6 (2–3)	0.81
Epinephrine administration, mg	0.70 ± 0.04	0.68 ± 0.02	0.51
Ribs fracture, number	0 (0–0)	0 (0–0)	1

Values are presented as mean ± SD or median plus interquartile range.
AP, aortic pressure; RAP, right atrial pressure; CPR, cardiopulmonary resuscitation; T-CPR, tourniquet assisted CPR group; A-CPR, CPR alone group.

Table 3
Hemodynamics before and after releasing four limbs at post-resuscitation 5 min in animals with physical control of peripheral vascular resistance.

	Before releasing	After releasing	*p*-Value
Heart rate, beats/min	142 ± 11	145 ± 9	0.75
Mean aortic pressure, mm Hg	108 ± 6	105 ± 10	0.65
Right atrium pressure	3.6 ± 0.7	3.3 ± 0.5	0.51

Values are presented as mean ± SD.

occurs in these vessels during the decompression phases. During CPR, augmentation of venous return followed by physical control of peripheral vascular resistance in the decompression phases can increase forward blood flow in the compression phases. Furthermore, physical control of peripheral vascular resistance improves vital organ perfusion by blocking the limited blood flow into periphery. This resulted in better CPP, $ETCO_2$, and CBF during CPR, as observed in our study.

During CPR, negative ITP generated in the decompression phases draws venous blood back to the heart then provides cardiac preload prior to the next chest compression.[27–31] Improved negative ITP during CPR augments myocardial and cerebral perfusion and yields better survival and neurological function.[32] There were no differences in positive and negative ITP between the two groups in this study. We believe the increases in hemodynamic efficacy of chest compressions observed in animals treated with the T-CPR are usually a result from increased peripheral vascular resistance.

In spite of the disappointing results of hemodynamics during MSAT or AB assisted CPR, our study demonstrated optimal CPP and CBF without change in ITP by tightening the elastic tourniquets around the four limbs during CPR. Small changes in diastolic RAP were observed in the T-CPR animals and may be a result from stable ITP. We can determine that the key reasons for stable ITP are limited excursions of the diaphragm and no increase in the abdominal pressure.

No visceral injuries or rib fractures were observed at necropsy in our study which is also coincident with previous studies.[33] After post-resuscitation 5 min, the tourniquets were released one-by-one to maintain stable hemodynamics and to avoid the detrimental effects caused by the release, as previously reported.[34] No significant differences in heart rate, mean artery pressure and right atrium pressure were observed before or after releasing the tourniquets.

In this study, physical control of peripheral vascular resistance was easy to perform by tightening the elastic tourniquets around

the four limbs. However, we acknowledge that the longer and larger limbs of humans would be more difficult to bind quickly and tightly. It is not difficult to design low-cost special tourniquets (long and large enough) which could be convenient to use during in-hospital or out-of-hospital CPR without the interruption of chest compression.

We acknowledge that there were several limitations in this study. First, the healthy porcine model does not always indicate the real condition of patients in a clinical setting. Second, direct myocardial perfusion during CPR, cardiac and neurological functions after resuscitation were not measured. Third, epinephrine was administrated after 2 min CPR in both groups. Epinephrine was used in the present study since it is a standard protocol of human CPR. Furthermore, our results have demonstrated that significant increases in CPP or CBF were observed in the T-CPR group before or after the administration of epinephrine during CPR. In addition, a 7-min down time in a healthy porcine model may not result in severe ischemia injury.

5. Conclusion

T-CPR improves the hemodynamic efficacy of CPR. It may provide a new and convenient method to assist CPR.

Conflict of interest statement

None of the authors have any conflicts of interest to report.

Acknowledgement

Lisa Luna contributed to the editing of this manuscript.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.resuscitation.2014.10.009.

References

1. Kouwenhoven WB, Jude JR, Knickerbocker GG. Closed-chest cardiac massage. JAMA 1960;173:1064–7.
2. Ristagno G, Tang W, Chang YT, et al. The quality of chest compressions during cardiopulmonary resuscitation overrides importance of timing of defibrillation. Chest 2007;132:70–5.
3. Yu T, Weil MH, Tang W, et al. Adverse outcomes of interrupted precordial compression during automated defibrillation. Circulation 2002;106:368–72.
4. Aufderheide TP, Frascone RJ, Wayne MA, et al. Standard cardiopulmonary resuscitation versus active compression-decompression cardiopulmonary resuscitation with augmentation of negative intrathoracic pressure for out-of-hospital cardiac arrest: a randomized trial. Lancet 2011;377:301–11.
5. Bradley SM, Gabriel EE, Aufderheide TP, et al. Survival increases with CPR by Emergency Medical Services before defibrillation of out-of-hospital ventricular fibrillation or ventricular tachycardia: observations from the Resuscitation Outcomes Consortium. Resuscitation 2010;81:155–62.
6. Barsan WG, Levy RC. Experimental design for study of cardiopulmonary resuscitation in dogs. Ann Emerg Med 1981;10:135–7.
7. Gudipati CV, Weil MH, Bisera J, et al. Expired carbon dioxide: a noninvasive monitor of cardiopulmonary resuscitation. Circulation 1988;77:234–9.
8. Klouche K, Weil MH, Sun S, et al. Stroke volumes generated by precordial compression during cardiac resuscitation. Crit Care Med 2002;30:2626–31.
9. Von Planta M, Weil MH, Gazmuri RJ, et al. Myocardial acidosis associated with CO_2 production during cardiac arrest and resuscitation. Circulation 1989;80:684–92.
10. Kette F, Weil MH, von Planta M, et al. Buffer agents do not reserve intramyocardial acidosis during cardiac resuscitation. Circulation 1990;81:1660–6.
11. Ditchey RV, Winkler JV, Rhodes CA. Relative lack of coronary blood flow during closed-chest resuscitation in dogs. Circulation 1982;66:297–302.
12. Deshmukh HG, Weil MH, Gudipati CV, et al. Mechanism of blood flow generated by precordial compression during CPR. I. Studies on closed chest precordial compression. Chest 1989;95:1092–9.
13. Babbs CF, Ralston SH, Geddes LA. Theoretical advantages of abdominal counterpulsation in CPR as demonstrated in a simple electrical model of the circulation. Ann Emerg Med 1984;13:660–71.
14. Kern KB, Ewy GA, Voorhees WD, et al. Myocardial perfusion pressure: a predictor of 24 h survival during prolonged cardiac arrest in dogs. Resuscitation 1988;16:241–50.
15. Pearson JW, Redding JS. Influence of peripheral vascular tone on cardiac resuscitation. Anesth Analg 1965;44:746–52.
16. Lindner KH, Prengel AW, Brinkmann A, et al. Vasopressin administration in refractory cardiac arrest. Ann Intern Med 1996;124:1061–4.
17. Babbs CF. The evolution of abdominal compression in cardiopulmonary resuscitation. Acad Emerg Med 1994;1:469–77.
18. Chandra N, Snyder LD, Weisfeldt ML. Abdominal binding during cardiopulmonary resuscitation in man. JAMA 1981;246:351–3.
19. Person JW, Redding JS. The role of epinephrine in cardiac resuscitation. Anesth Analg 1963;42:599–606.
20. Redding JS. Abdominal compression in cardiopulmonary resuscitation. Anesth Analg 1971;50:668–75.
21. Tang W, Weil MH, Sun S, et al. Epinephrine increases the severity of postresuscitation myocardial dysfunction. Circulation 1995;92:3089–93.
22. Marwick TH, Case C, Siskind V, et al. Adverse effect of early high-dose adrenaline on outcome of ventricular fibrillation. Lancet 1988;2:66–8.
23. Herlitz J, Ekstrom L, Wennerblom B, et al. Adrenaline in out-of-hospital ventricular fibrillation. Does it make any difference? Resuscitation 1995;29:195–201.
24. Bircher N, Safar P, Stewart R. A comparison of standard, MAST-augmented, and open-chest CPR in dogs. A preliminary investigation. Crit Care Med 1980;8:147–52.
25. Niemann JT, Rosborough JP, Criley JM. Continuous external counterpressure during closed-chest resuscitation: a critical appraisal of the military antishock trouser garment and abdominal binder. Circulation 1986;74:102–7.
26. Bradford EM. Haemodynamic changes associated with the application of lower limb tourniquets. Anaesthesia 1969;24:190–7.
27. Lurie KG, Mulligan KA, McKnite S, et al. Optimizing standard cardiopulmonary resuscitation with an inspiratory impedance threshold valve. Chest 1998;113:1084–90.
28. Lurie KG, Voelckel WG, Plaisance P, et al. Use of an inspiratory impedance threshold valve during cardiopulmonary resuscitation: a progress report. Resuscitation 2000;44:219–30.
29. Babbs CF. Interposed abdominal compression CPR: a comprehensive evidence based review. Resuscitation 2003;59:71–82.
30. Ralston SH, Babbs CF, Niebauer MJ. Cardiopulmonary resuscitation with interposed abdominal compression in dogs. Anesth Analg 1982;61:645–51.
31. Babbs CF, Weaver JC, Ralston SH, et al. Cardiac, thoracic, and abdominal pump mechanisms in cardiopulmonary resuscitation: studies in an electrical model of the circulation. Am J Emerg Med 1984;2:299–308.
32. Lurie KG, Zielinski T, McKnite S, et al. Use of an inspiratory impedance threshold valve improves neurologically intact survival in a porcine model of ventricular fibrillation. Circulation 2002;105:124–9.
33. Chen W, Weng Y, Wu X, et al. The effects of a newly developed miniaturized mechanical chest compressor on outcomes of cardiopulmonary resuscitation in a porcine model. Crit Care Med 2012;40:3007–12.
34. Maurer NH, Voegeli Jr PT, Sorkin BS. Non-cardiac circulatory overload secondary to pneumatic thigh tourniquets. J Am Podiatry Assoc 1983;73:589–92.