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Original Contribution

Combination of chest compressions and interposed abdominal compressions in a swine model of ventricular fibrillation☆

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ABSTRACT

Purpose: The aim of this study was to investigate the effects of the combination of chest compressions and interposed abdominal compressions (IAC-CPR) in a swine model of ventricular fibrillation (VF).
Methods: Twenty healthy female Landrace-Large White pigs were the study subjects. At the end of the eighth minute of VF, animals in the control group (Group A) received chest compressions at a rate of 100/min, while animals in the experimental group received chest compressions and simultaneous interposed abdominal compressions (CC-IAC – Group B), both at a rate of 100/min. The primary end point of the experiment was return of spontaneous circulation (ROSC). Secondary outcomes were 48-h survival rate and 48-h neurologic outcome.
Results: Six animals (60%) from Group A and 9 animals (90%) from Group B achieved ROSC ($P = .121$). There was a statistically significant difference in systolic aortic pressure, mean aortic pressure, right atrial pressures, and end-tidal carbon dioxide ($ETCO_2$) between the two groups during the first cycle of CPR, while during the second cycle, diastolic aortic pressure was significantly higher in Group B. Coronary perfusion pressure (CPP) values in group B were significantly higher compared with those in Group A during the first and second cycle of CPR. Neurologic examination was statistically significantly better in Group B (75.00 ± 10.00 vs. 90.00 ± 10.00, $P = .037$).
Conclusion: ROSC did not differ statistically significant in the IAC-CPR compared to the CPR group only, while CPP was significantly higher in IAC-CPR–treated animals.

1. Introduction

The amount of blood flow to the heart at any given time and the compression-related cardiac output may be the most critical determinants of the efficacy of cardiopulmonary resuscitation (CPR) and survival after cardiac arrest [1].

Augmenting cardiac output during CPR may enhance both the coronary and cerebral perfusion and will increase return of spontaneous circulation (ROSC) rates [1]. However, maintenance of high-quality compressions during out-of-hospital resuscitation is difficult; with manual CPR, many factors come into play, including fatigue, physical ability, focus on several simultaneous tasks, poor-quality CPR, interruptions during movement of patient, and variation in technique and training. On the other hand, various mechanical devices allow high-quality CPR to be performed, but until now, there is no evidence regarding their superiority with regard to survival to discharge or neurological function [2–5]. Moreover, in a recent Cochrane review, the authors reported that there is insufficient evidence from human randomized control trials to conclude that mechanical chest compressions during CPR are associated with benefit or harm, highlighting the need for further research [6,7].

Research so far has shown that although abdominal only CPR may potentially increase the coronary and cerebral perfusion pressure, there is very little evidence to support its application. Considering that the long-term survival following cardiac arrest remains poor and high quality continuous CPR is a critical aspect of resuscitation, mechanical interposed abdominal compression (IAC-CPR), i.e. the addition of manual mid-abdominal compressions to standard external CPR, remains an attractive alternative. The aim of this study was to investigate the effects of the combination of mechanical chest compressions and interposed abdominal compressions (IAC-CPR) in a swine model of ventricular fibrillation (VF). The primary end point of the experiment was ROSC.

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Secondary outcomes were hemodynamics, as well as survival rate and neurologic outcome at 48 h.

2. Methods

Twenty healthy female Landrace-Large White pigs with an average weight of 20 (SD 1 kg), aged 19 (SD 2 weeks) were the study subjects. The animals were transported 1 week before experimentation to the research facility (Experimental Research Center ELPEN, European Ref No. EL 09 BIO 03). All pigs were purchased from the same breeder (Validakis, Koropi, Greece) and were prepared in a standardized fashion in the research facility as previously described [8]. The animals were fasted overnight but had free access to water.

The protocol was approved by the General Directorate of Veterinary Services (License no. 1188/28–02-2014), according to Greek legislation regarding ethical and experimental procedures (Presidential Decree 160/1991, in compliance with the EEC Directive 86/609 and Law 2015/1992 and in conformance with the European Convention for the protection of vertebrate animals used for experimental or other scientific purposes, 123/1986).

2.1. Animal preparation

The protocol has been described in detail elsewhere [8]. Briefly, initial sedation in each animal was achieved with intramuscular injection of 10 mg/kg ketamine hydrochloride (Merial, Lyon, France), 0.5 mg/kg midazolam (Roche, Athens, Greece), and 0.05 mg/kg atropine sulfate (Demo, Athens, Greece). The animals were subsequently transported to the operation research facility, and intravascular access was obtained through the auricular veins. Induction of anesthesia was achieved with an intravenous bolus dose of propofol (Diprivan 1% wt/vol; Astra Zeneca, Luton, United Kingdom) 2 mg/kg and fentanyl (Janssen Pharmaceutica, Beerse, Belgium) 2 μg/kg [9]. The animals were still in spontaneous breathing when endotracheal intubation was performed with a size 6.0 mm cuffed endotracheal tube (Portex, Mallinckrodt Medical, Athlone, Ireland). The endotracheal tube was secured on the lower jaw, and successful intubation was ascertained with auscultation of both lungs while ventilating with a self-inflating bag. The animals were then immobilized in the supine position on the operating table. Propofol 1 mg/kg, *cis*-atracurium (Nimbex 2 mg/mL; GlaxoSmithKline, Athens, Greece) 0.15 mg/kg, and fentanyl 0.01 mg/kg were administered to achieve synchrony with the ventilator, whereas anesthetic depth was maintained using propofol infusion (0.1 mg/kg per minute) and additional doses of cis-atracurium (20 μg/kg per minute) and fentanyl (0.6 μg/kg per minute).

The animals were mechanically ventilated (Alpha Delta lung ventilator, Siare, Bologna, Italy) with 21% oxygen (FIO_2, 21%). All animals were volume-controlled ventilated with a total tidal volume of 10 mL/kg. End-tidal carbon dioxide pressure ($ETCO_2$) was monitored (Tonocap-TC200; Datex Engstrom, Helsinki, Finland), and the respiratory rate was adjusted to maintain an $ETCO_2$ of 35 to 40 mmHg. Arterial blood gases were obtained using heparinized syringe, and they were measured on a blood-gas analyzer (IRMA SL Blood Analysis System, Part 436,301; Diametrics Medical Inc, Roseville, MN, USA). Electrocardiographic monitoring was used using leads I, II, III, aVR, aVL, and aVF, which were connected to a monitor (Mennen Medical; Envoy, Papapostolou, Athens, Greece). The pulse oximeter (Datascope Expert DS-5300 W ECG; Fukuda Denshi, Tokyo, Japan) was placed on the tongue of the animal. For measurement of aortic systolic and diastolic pressure (SAP and DAP, respectively), an arterial catheter was inserted into the aorta via the right common carotid artery (model 6523, USCI CR; Bart Inc, Papapostolou, Athens, Greece). Mean arterial pressure (MAP) was electronically determined. Cardiac output (CO) was measured as the product of time-velocity integral of Doppler-determined transaortic flow, the diameter of the aortic valve, and heart rate, as previously described [10].

The internal jugular vein was surgically prepared, and a Swan-Ganz catheter (Opticath 5.5F, 75 cm; Abbott, Ladakis, Athens, Greece) was inserted into the right atrium for continuous measurement of right atrial systolic (RASP) and diastolic pressure (RADP). Coronary perfusion pressure (CPP) was electronically calculated as the difference between minimal DAP and the simultaneously measured right atrial diastolic pressure. Subsequently, a catheter (18 gauge; Vitroflon, Vitromed Healthcare, India) was placed into the left femoral vein for blood sampling. The animals were then left to be stabilized for 30 min.

2.2. Experimental procedure

Before the experimental procedure, the piglets were randomly assigned to two different groups of 10 subjects each by means of a sealed envelope. After baseline data were collected, VF was induced with a 9-V ordinary cadmium battery via a pacing wire forwarded into the right ventricle through the exposed right jugular vein, as previously described [8]. Ventricular fibrillation was confirmed by ECG and a sudden drop in MAP. Mechanical ventilation and administration of anesthetics were discontinued simultaneously with the onset of VF, and the animals were left untreated for 8 minutes.

At the end of the eighth minute of VF, resuscitation was immediately initiated according to the European Resuscitation Council guidelines on resuscitation with ventilation in 100% oxygen [1]. Animals in the control group (Group A) received chest compressions at a rate of 100/min (LUCAS; Jolife, Lund, Sweden), while animals in the second group received chest compressions and simultaneous interposed abdominal compressions (CC-IAC – Group B) (LUCAS; Jolife, Lund, Sweden), both at a rate of 100/min [11]. Both groups received intravenous adrenaline 0.02 mg/kg at the onset of CPR, followed by 20-mL flush of isotonic sodium chloride solution. Defibrillation was attempted with a 4 J/kg monophasic waveform shock delivered between the right infraclavicular area and the cardiac apex (Primedic Defi-B Defibrillator; Metrax GmbH, Rottweil, Germany).

Successful resuscitation was defined as ROSC with an MAP of at least 60 mmHg for a minimum of 5 min. After ROSC, the animals were monitored closely and mechanically ventilated for 6 hours under general anesthesia at the pre-arrest settings. No other interventions (drugs, cardioversion or defibrillation attempts) were made after ROSC. After 6 hours, anesthesia was discontinued, all catheters were removed as previously described, and manual ventilation was initiated [12]. Atropine 0.2 mg/kg followed by neostigmine 0.05 mg/kg was administered when spontaneous swallowing reflex was detected, whereas extubation was performed after adequate inspiration depth was confirmed. Each animal was then transferred to the animal house for observation for 48 h.

The primary end point of the experiment was ROSC. Secondary outcomes were hemodynamics, as well as survival rate and neurologic outcome at 48 h. A neurological alertness score was performed at 48 hours by a veterinarian blinded as to the allocation of each animal, as previously described [10]. Alertness was scored from 0 (coma) to 100 (fully alert). Finally, the animals that survived were humanely euthanized by an intravenous overdose of pentobarbital and underwent necropsy [9].

2.3. Statistical analysis

Continuous variables were expressed as mean ± SD and categorical variables as percentages. Kolmogorov-Smirnov's test was used to assess the normality of the distribution of continuous variables. Differences between groups were assessed with the χ^2 test or Fisher's exact test for categorical variables, and with Student t test and the Mann-Whitney U test for continuous variables, as appropriate. Multivariable analysis was performed in order to investigate differences between the two groups at different periods of time. Statistical significance was set at the 5% level. Regarding sample size, for an expected 30% of

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ΒΙΒΛΙΟΘΗΚΗ ΕΠΙΣΤΗΜΩΝ ΥΓΕΙΑΣ

ΥΠΟΥΡΓΕΙΟ ΥΓΕΙΑΣ ΚΥΠΡΟΥ – Ι. Υ. & Υ. Δ. Υ.

ΕΙΔΟΠΟΙΗΣΗ: ΠΡΟΕΙΔΟΠΟΙΗΣΗ ΣΧΕΤΙΚΑ ΜΕ ΤΟΥΣ ΠΕΡΙΟΡΙΣΜΟΥΣ ΠΝΕΥΜΑΤΙΚΩΝ ΔΙΚΑΙΩΜΑΤΩΝ

Ο νόμος πνευματικών δικαιωμάτων των Ηνωμένων Πολιτειών (τίτλος 17, Ηνωμένος κώδικας) ελέγχει την παραγωγή των φωτοτυπιών ή άλλων αναπαραγωγών του υλικού. Υπό ορισμένους όρους που διευκρινίζονται στο νόμο, οι βιβλιοθήκες και τα αρχεία εξουσιοδοτούνται για να εφοδιάσουν μια φωτοτυπία ή άλλη αναπαραγωγή. Ένας από αυτούς τους διευκρινισμένους όρους είναι ότι η φωτοτυπία ή η αναπαραγωγή δεν πρόκειται «να χρησιμοποιηθεί για οποιοδήποτε σκοπό εκτός από την ιδιωτική μελέτη, την υποτροφία ή την έρευνα». Εάν ένας χρήστης υποβάλλει ένα αίτημα, ή αργότερα χρησιμοποιήσει, μια φωτοτυπία ή την αναπαραγωγή για λόγους παραπάνω από τη «δίκαιη χρήση», αυτός ο χρήστης μπορεί να είναι υπεύθυνος για την παράβαση πνευματικών δικαιωμάτων. Αυτό το όργανο διατηρεί το δικαίωμα να αρνηθεί να δεχτεί μια διαταγή πνευματικών δικαιωμάτων εάν, στην κρίση της, η εκπλήρωση της διαταγής θα περιελάμβανε την παραβίαση του νόμου πνευματικών δικαιωμάτων.

(η παραπάνω ειδοποίηση αποτελεί μετάφραση από τον πρωτότυπο σχετικό νόμο των Η.Π.Α. σχετικά με τα πνευματικά δικαιώματα που παρατίθεται παρακάτω).

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ΒΙΒΛΙΟΘΗΚΗ ΕΠΙΣΤΗΜΩΝ ΥΓΕΙΑΣ – ΔΙΚΤΥΟ ΒΙΒΛΙΟΘΗΚΩΝ

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subjects regaining ROSC (considered as primary outcome) after standard care and 85% after IAC-CPR, at the $\alpha = 5\%$ significance level and with 80% power, a total sample size of 20 subjects would be required. All statistical analysis was performed using SPSS version 13.0 (SPSS Inc, Chicago IL, USA).

3. Results

In terms of ROSC, 6 animals (60%) from Group A and 9 animals (90%) from Group B achieved ROSC; however, the observed difference was not statistically significant ($P = .121$). More specifically, after the first shock, 3 animals from Group A and 4 animals from Group B restored ROSC. After the second shock, 1 animal from Group A and 3 animals from Group B restored ROSC. After the third shock, 1 animal from Group A and 1 from Group B restored ROSC. After the fourth shock, 1 animal from Group A and 1 from Group B restored ROSC.

No statistically significant differences were observed at baseline and at 8-min untreated VF hemodynamic parameters between the 2 groups (Table 1). In our study, there was a statistically significant difference in SAP, MAP, RASP, RADP, and $ETCO_2$ between the two groups during the first cycle of CPR, while during the second cycle, DAP was significantly higher in Group B (Table 2, Figs. 1-3). Coronary perfusion pressure values in group B were significantly higher compared with those in Group A during the first and second cycle of CPR. In our study, we found no significant difference between arterial blood gases at baseline and 8 minutes of untreated VF, as well as during CPR and post-resuscitation period.

All animals that were successfully resuscitated were monitored for 6 h. The largest differences (which were also statistically significant) in hemodynamic parameters were observed at 2 hours post-ROSC (Table 3). Although there was not a significant difference between the two groups regarding the 48-h survival rate (6 Group A vs. 9 Group B animals, $P = .121$), neurologic examination was significantly better in the animals of Group B (75.00 ± 10.00 vs. 90.00 ± 10.00, $P = .037$).

4. Discussion

The amount of blood flow to the heart at any given time may be the most important determinant of survival and has emerged as a critical determinant of CPR efficacy. In our study, we found a statistically significant difference in SAP, MAP, RASP, RADP, and $ETCO_2$ in favor of CC-IAC during the first cycle of CPR, while during the second cycle, DAP and RADP were also significantly higher in Group B. As a result, CPP values in group B were significantly higher compared with those in Group A during the first two cycles of CPR. Our study sheds light in foggy landscape of IAC-CPR, especially because of the close similarity of Landrace/Large White swine hemodynamics to those of humans [12].

One of the most interesting results is the improved hemodynamics during the first cycle of CPR with the difference in CO being insignificant between the two groups. Although this can be partly explained by the effect of exogenous adrenaline which increased systemic vascular

Table 1
Hemodynamic variables between the 2 groups after at baseline and before the onset of cardiopulmonary resuscitation

Variable	Baseline		After 8 min of untreated VF	
	Group A	Group B	Group A	Group B
HR (beat min^{-1})	109.8 ± 22.51	116.5 ± 9.05	NA	NA
SAP (mm Hg)	101.7 ± 6.57	104.6 ± 13.82	21.10 ± 3.80	19.40 ± 4.10
DAP (mm Hg)	74.6 ± 6.49	77 ± 8.2	18.30 ± 2.65	17.70 ± 1.59
RASP (mm Hg)	10.1 ± 1.55	11.6 ± 1.45	18.10 ± 2.26	17.05 ± 3.62
RADP (mm Hg)	7.9 ± 1.77	7.7 ± 1.32	17.00 ± 1.88	16.75 ± 1.20
MAP (mm Hg)	83.6 ± 8.56	85.59 ± 7.81	19.23 ± 2.34	18.26 ± 2.93
CPP (mm Hg)	66.7 ± 5.78	69.3 ± 4.28	1.30 ± 0.77	0.95 ± 0.39
CO (L/min)	6.3 ± 0.7	6.2 ± 0.5	NA	NA

HR, heart rate; NA, non-applicable.

Table 2
Hemodynamic variables during cardiopulmonary resuscitation

Variable	First cycle of CPR			Second cycle of CPR			Third cycle of CPR			Fourth cycle of CPR		
	Group A	Group B	p-value	Group A	Group B	p-value	Group A	Group B	p-value	Group A	Group B	p-value
HR (beat min^{-1})	104.3 ± 3.47	104.85 ± 5.12	0.402	101 ± 3.28	102.57 ± 5.72	0.312	103 ± 3.1	100 ± 4.27	0.082	104.45 ± 6.76	101.78 ± 11.27	0.079
SAP (mm Hg)	126.6 ± 13.43	132.8 ± 21.1	0.036	131.2 ± 19.35	128.44 ± 34.1	0.039	114.45 ± 21.11	109.21 ± 34.28	0.034	107.02 ± 19.16	106.46 ± 23.15	0.233
DAP (mm Hg)	65.32 ± 7.09	68.69 ± 14.51	0.077	56.7 ± 14.78	72.54 ± 14.04	0.012	55.87 ± 20.31	61.32 ± 11.44	0.026	42.67 ± 12.05	40.54 ± 16.29	0.128
RASP (mm Hg)	63.2 ± 25.53	70.2 ± 46.91	0.031	71.51 ± 30.12	77.4 ± 49.71	0.041	50.24 ± 29.56	65.33 ± 12.65	0.011	45.5 ± 29.65	53.80 ± 37.41	0.042
RADP (mm Hg)	9.21 ± 4.62	7.38 ± 3.85	0.038	14.77 ± 5.36	19.8 ± 5.56	0.040	12.2 ± 3.51	19.3 ± 3.74	0.030	11.87 ± 4.32	11.52 ± 12.57	0.277
MAP (mm Hg)	85.32 ± 21.57	90.46 ± 24.49	0.049	81.67 ± 4.67	91.383 ± 6.71	0.029	75.83 ± 4.78	77.22 ± 5.98	0.101	64.88 ± 6.26	62.06 ± 4.61	0.213
CPP (mm Hg)	56.34 ± 17.72	62.73 ± 13.56	0.042	42.52 ± 4.23	53.82 ± 7.98	0.033	43.67 ± 3.99	42.88 ± 6.23	0.125	31.54 ± 2.78	29.65 ± 6.34	0.194
$ETCO_2$ (mm Hg)	17.35 ± 2.67	22.18 ± 3.37	0.036	16.88 ± 1.99	20.76 ± 2.01	0.048	17.62 ± 2.32	17.89 ± 1.34	0.316	14.99 ± 2.1	15.01 ± 1.78	0.266
CO (L/min)	1.89 ± 0.4	1.96 ± 0.7	0.274	1.93 ± 0.3	1.95 ± 0.6	0.191	1.92 ± 0.9	1.88 ± 0.5	0.114	1.77 ± 0.3	1.75 ± 0.6	0.214

HR, heart rate.

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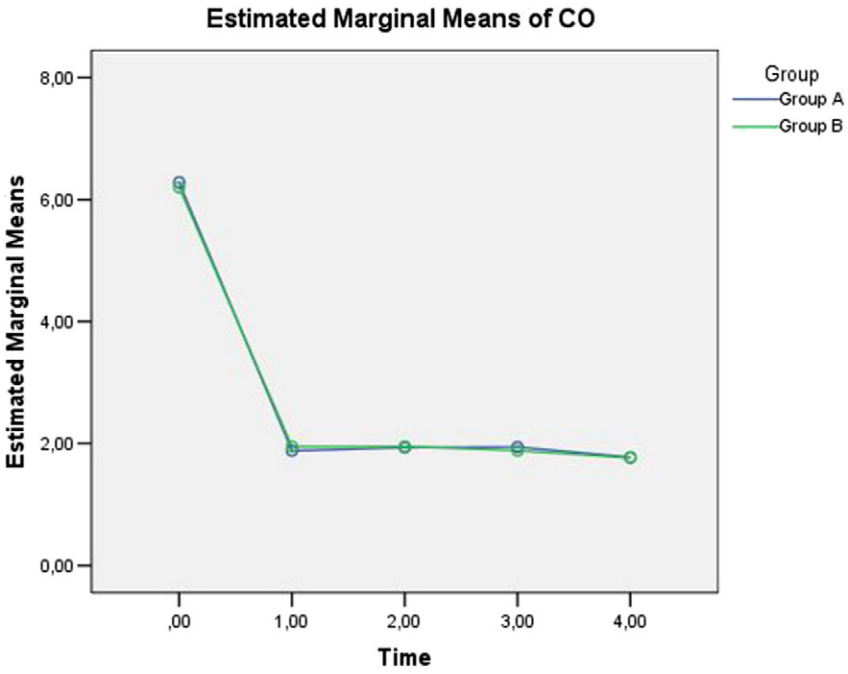

Fig. 1. Cardiac output fluctuation during cardiopulmonary resuscitation. Time .00, baseline; Time 1.00, first cycle; Time 2.00, second cycle; Time 3.00, third cycle; Time 4.00, fourth cycle.

resistance, our results support the concept that the intra-abdominal pressure generated by abdominal compressions can cause blood flow in the absence of any mechanism of chest compression [13,14]. In a very recent randomized clinical trial, IAC-CPR resulted in a significant increase in $ETCO_2$ and CO but did not increase ROSC rates compared to standard CPR (STD-CPR) [15]. An earlier human study reported that CO was significantly increased with IAC-CPR as evidenced by the significantly greater increases in $ETCO_2$ with IAC-CPR compared with STD-CPR [16]. This discrepancy with our data may reflect the larger lower extremity venous blood network capacity of humans compared to piglets, which may result in more pronounced hemodynamic differences. Nevertheless, other studies of IAC-CPR have shown varying results. Berryman and Phillips performed IAC-CPR in a group of six patients after STD-CPR was unsuccessful and reported a 50% increase in MAP [17]. Also, Matter et al. reported that IAC-CPR was at least as effective as STD-CPR [18]. Nonetheless, McDonald demonstrated that IAC-CPR failed to consistently enhance SAP or increase CPP [19], while Howard et al. reported that IAC-CPR significantly increased DAP [20].

Two in-hospital studies of interposed abdominal counterpulsation CPR showed a significant improvement in outcome, while an out-of-hospital study showed that it may actually decrease survival rates [16,18,21]. The authors of the later study reported that a main reason

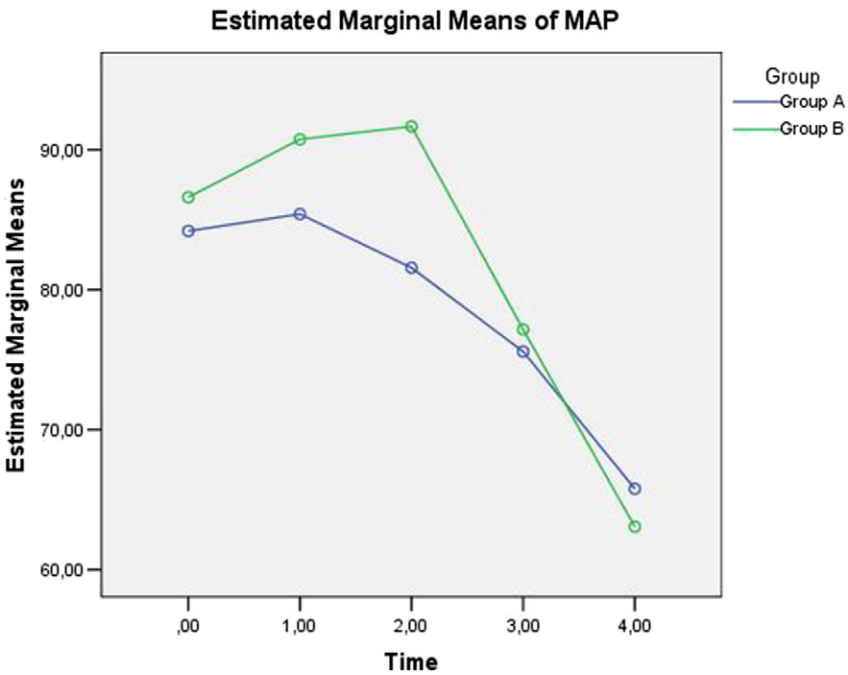

Fig. 2. Mean aortic pressure fluctuation during cardiopulmonary resuscitation. Time .00, baseline; Time 1.00, first cycle; Time 2.00, second cycle; Time 3.00, third cycle; Time 4.00, fourth cycle.

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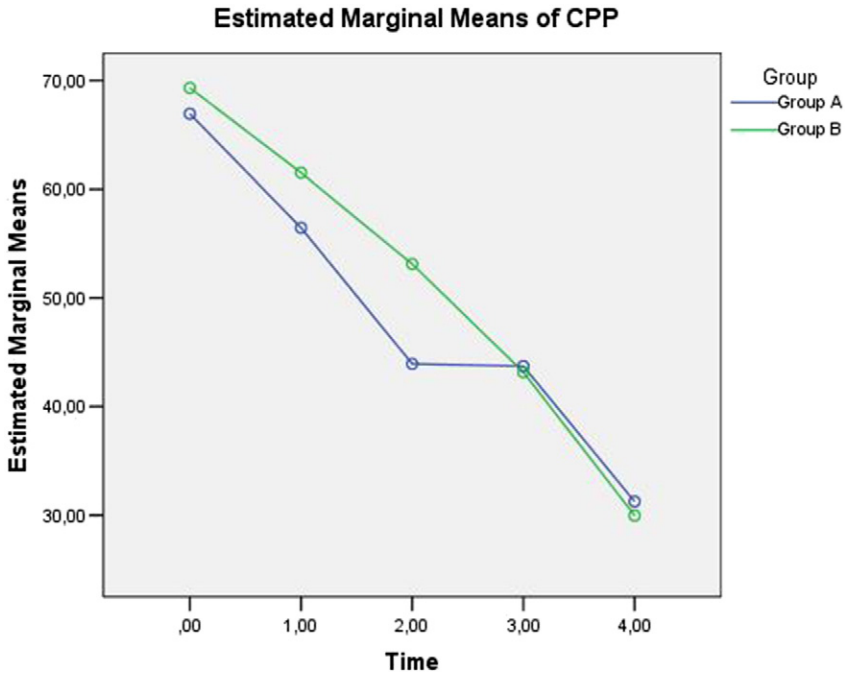

Fig. 3. Coronary perfusion pressure fluctuation during cardiopulmonary resuscitation. Time .00, baseline; Time 1.00, first cycle; Time 2.00, second cycle; Time 3.00, third cycle; Time 4.00, fourth cycle.

for the decreased survival was the effect of abdominal counterpulsation on right atrial pressure and left ventricular pressure during diastole, which decreased CPP. In our study, however, CPP was higher in Group B during the first two cycles. Considering also that RASP was higher in Group B, while RADP was higher in Group A during the same period, it seems that the combined effect of interposed active abdominal decompression and chest compression resulted in backward flow from the right atrium to the vena cava, which decreased RADP in Group B during the first cycle.

Our findings during the first cycle of CPR highlight the role of intrathoracic pressure and thoracic pump on forward blood flow [22]. Similarly, in most animal IAC-CPR studies, blood flow during thoracic compression was most likely generated only by intrathoracic pressure variations [23–28]. This is also supported by the mathematical model of Babbs et al. who predicted that the hemodynamic benefit of IAC-CPR is much greater in a thoracic than in a cardiac pump model [29,30]. Nevertheless, there are no data on the effects of IAC-CPR in a cardiac compression model that confirm this suggestion until now [14]. Of note, although the cardiac and thoracic pump mechanisms have been reported to predominate early and late after human cardiac arrest, respectively [31–34], it is possible that IAC-CPR-related blood flow may be based on thoracic pump in the early stages of CPR, while in the latter stages (i.e. after the second cycle), both theories may have an effect due to the increase in myocardial stiffness and aggravation of cardiac-arrest-induced vasodilatory state. In addition, abdominal compressions result in aortic pressure waves that propagate from the abdominal toward the ascending aorta, promoting retrograde blood flow. This, together with the effect of increased intra-abdominal pressure during abdominal compression may enhance retrograde volume loading of the aorta, increasing DAP and CPP [35,36]. Therefore, we agree with Einagle et al. that the thoracic and abdominal compartments act as a thoracoabdominal unit during IAC-CPR [27]. Based on our findings, however, we propose that this unit involves three major mechanisms of blood flow, the thoracic and cardiac pump, as well as the retrograde aortic volume loading. In our study, however, the retrograde aortic volume loading and the increased afterload may be also responsible for the insignificant difference in CO during the experiment.

On the other hand, IAC-CPR forces venous blood back into the thorax and may cause an increase in RADP during manual chest compressions [37], although the use of devices may further improve hemodynamics in patients who had been treated with mechanical precordial compression regardless of the cardiac arrest rhythm [38–41]. In our study, the increase in RAPD was evident during the second and third cycle of CPR, possibly due to the retrograde volume loading and the effect of myocardial stunning on ventricular filling and compression-related cardiac output [13,20,22,42].

In either case, the alternating compression of the chest and abdomen seem to increase venous return per unit of time, although this increase reach a zenith and then reduces as IAC-CPR evolves [43]. Interestingly, in a canine model with predominant direct cardiac compressions, IAC-CPR–induced augmentation of total forward blood flow and vital organ perfusion depended on the efficacy of thoracic compressions, not on the time after cardiac arrest [14]. This may not the case in a non-predominant model, as in our study, in which blood flow may depend on time. In such case, this issue could be solved if rescuers compress the abdomen during the latter half of the diastolic period, well after the chest wall has recoiled. In this way, the brief pause between the initial chest relaxation phase and the initiation of abdominal compression may be of major importance because during this time the coronary perfusion gradient is increased by the chest recoil, while once the chest recoil is complete, the abdominal compression will enhance forward blood flow and CPP [13,21]. Furthermore, this technique can minimize the effect of upward diaphragmatic placement during abdominal compression which otherwise will increase intrathoracic pressure [43,44], thus decreasing preload during CPR.

In the trial by Movahedi et al, IAC-CPR did not increase ROSC rates compared to STD-CPR [15]. In our study, although the observed differences in ROSC and 48-h survival rate between the two groups were not statistically significant, neurologic examination was significantly better in the animals of Group B. The main reason for this difference is the earlier ROSC rates in Group B and the increased SAP which alleviated the post-resuscitation brain injury [45]. Similarly, in a study of cardiac surgical patients who suffered a sudden cardiac arrest during the first 24 h after surgery, there were more patients in terms of ROSC with IAC-CPR [46]. Also, Tang et al. reported that the greater cerebral flows

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Table 3
Hemodynamic variables between the two groups after return of spontaneous circulation

Variable	ROSC – 0 min			ROSC – 2 h			ROSC – 4 h			ROSC – 6 h		
	Group A	Group B	p-value	Group A	Group B	p-value	Group A	Group B	p-value	Group A	Group B	p-value
HR (beat min^{-1})	165.3 ± 34.5	161.6 ± 40.4	0.048	131.43 ± 17.13	141.32 ± 18.13	0.028	132.45 ± 23.57	123.52 ± 21.59	0.036	135 ± 78	144 ± 37	0.039
SAP (mm Hg)	147.5 ± 31.3	145.8 ± 31.5	0.106	119.78 ± 22.54	123.76 ± 11.43	0.044	109 ± 7.45	95.05 ± 12.22	0.022	92 ± 48	94 ± 66	0.135
DAP (mm Hg)	119.3 ± 23.6	123.9 ± 35.1	0.039	95.5 ± 10.08	99.12 ± 13.61	0.046	61.91 ± 10.34	63.46 ± 12.54	0.082	60 ± 41	55 ± 68	0.045
RASP (mm Hg)	15.2 ± 1.6	14.3 ± 2.4	0.178	14.6 ± 3.89	15.31 ± 3.1	0.155	10.94 ± 1.02	11.31 ± 0.9	0.204	10.67 ± 1.06	10.75 ± 1.1	0.242
RADP (mm Hg)	7.9 ± 0.8	8.1 ± 2.9	0.118	8.3 ± 1.14	9.16 ± 2.78	0.046	7.4 ± 0.63	7.67 ± 1.49	0.088	7.1 ± 1.33	7.3 ± 1.25	0.123
MAP (mm Hg)	128.3 ± 11.5	131.1 ± 23.43	0.104	103.3 ± 13.56	107.31 ± 22.56	0.072	77.12 ± 13.67	73.6 ± 9.38	0.068	70.63 ± 12.79	68.39 ± 11.82	0.133
CPP (mm Hg)	111.5 ± 19.7	116.3 ± 21.44	0.047	87.59 ± 9.51	90.76 ± 10.13	0.114	55 ± 8.89	56.62 ± 7.72	0.178	53.07 ± 5.89	48.71 ± 10.38	0.044
CO (L/min)	8.1 ± 0.6	7.9 ± 0.8	0.131	6.1 ± 0.6	6.3 ± 0.4	0.136	5.3 ± 0.7	5.2 ± 0.3	0.223	5.1 ± 0.9	4.9 ± 0.6	0.171

HR, heart rate.

during IAC-CPR may explain the observed cerebral recovery [47]. In addition, the active abdominal decompression and the downward diaphragmatic movement may decrease the magnitude of transmission of intrathoracic pressure to the intracranial space during chest compression, which together with the retrograde volume loading of the aorta enhance cerebral perfusion pressure [14,48]. The increased carotid blood flow and cerebral oxygen delivery during CPR contributes to the minimization of cerebral ischemia and post–cardiac arrest brain injury. Also, the increased cerebral perfusion pressure during IAC-CPR may ameliorate the severity of total body ischemia/reperfusion injury and, thus, the amount of reactive oxygen species that intensify endothelial injury, contribute to blood-brain barrier disruption, and increase the exchange vessel's permeability and microvascular filtration [45].

We recognize several limitations in this experimental study. Firstly, the sample size was small to recognize accurately significant differences in ROSC. Secondly, our experiment was conducted on apparently healthy pigs with no underlying disease. This is not the case in human cardiac arrest victims who, most of the time, have various comorbidities. Finally, body temperature during 48 h after ROSC could act as confounders on neurologic outcome.

5. Conclusion

In this study, we found a statistically significant difference in hemodynamics between the two groups during the first cycle of CPR in favor of CC-IAC, while CPP in IAC-CPR–treated animals was significantly higher compared to animals treated with STD-CPR.

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