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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 (TonocapTC200; 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