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Experimental paper

An investigation of thrust, depth and the impedance cardiogram as measures of cardiopulmonary resuscitation efficacy in a porcine model of cardiac arrest☆

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ABSTRACT

Objective: Optimising the depth and rate of applied chest compressions following out of hospital cardiac arrest is crucial in maintaining end organ perfusion and improving survival. The impedance cardiogram (ICG) measured via defibrillator pads produces a characteristic waveform during chest compressions with the potential to provide feedback on cardiopulmonary resuscitation (CPR) and enhance performance. The objective of this pre-clinical study was to investigate the relationship between mechanical and physiological markers of CPR efficacy in a porcine model and examine the strength of correlation between the ICG amplitude, compression depth and end-tidal CO_2 ($ETCO_2$).

Methods: Two experiments were performed using 24 swine (12 per experiment). For experiment 1, ventricular fibrillation (VF) was induced and mechanical CPR commenced at varying thrusts (0–60 kg) for 2 min intervals. Chest compression depth was recorded using a Philips QCPR device with additional recording of invasive physiological parameters: systolic blood pressure, ETCO2, cardiac output and carotid flow. For experiment 2, VF was induced and mechanical CPR commenced at varying depths (0–5 cm) for 2 min intervals. The ICG was recorded via defibrillator pads attached to the animal's sternum and connected to a Heartsine 500P defibrillator. ICG amplitude, chest compression depth, systolic blood pressure and $ETCO_2$ were recorded during each cycle. In both experiments the within-animal correlation between the measured parameters was assessed using a mixed effect model.

Results: In experiment 1 moderate within-animal correlations were observed between physiological parameters and compression depth ($r = 0.69–0.77$) and thrust ($r = 0.66–0.82$). A moderate correlation was observed between compression depth and thrust ($r = 0.75$). In experiment 2 a strong within-animal correlation and moderate overall correlations were observed between ICG amplitude and compression depth ($r = 0.89$, $r = 0.79$) and $ETCO_2$ ($r = 0.85$, $r = 0.64$).

Conclusion: In this porcine model of induced cardiac arrest moderate within animal correlations were observed between mechanical and physiological markers of chest compression efficacy demonstrating the challenge in utilising a single mechanical metric to quantify chest compression efficacy. ICG amplitude demonstrated strong within animal correlations with compression depth and $ETCO_2$ suggesting its potential utility to provide CPR feedback in the out of hospital setting to improve performance.

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1. Introduction

Current resuscitation guidelines emphasise the importance of high quality cardiopulmonary resuscitation (CPR) and early defibrillation to optimise outcome following cardiac arrest.[1,2] Quality chest compressions (CCs) remain the cornerstone of effective CPR. Optimising the depth of chest compressions is critical in maintaining coronary and cerebral perfusion and correlates with return of spontaneous circulation (ROSC) in a number of studies.[3–5] However chest compressions are performed poorly by the majority of lay responders.[6,7] Several automatic external defibrillators (AEDs) now provide audiovisual feedback on chest compression depth via an accelerometer based device applied over the patient's sternum.[8,9] These devices have yielded improvements in CPR metrics but have yet to confer an outcome benefit.

Trans-thoracic impedance (TTI) is a measure of the biological resistance to current flow across the thoracic cavity. The impedance cardiogram (ICG) can be recorded by passing a high frequency (30 kHz) low amplitude (0.05 mA) current between electrodes applied over the chest and measuring the resultant voltage change. Typically values vary from 50–150 Ω based on patient characteristics. The impedance cardiogram (ICG) is a measure of the change in baseline impedance with time and reflects changes in ventilation, circulating blood volume and inherent signal variation. Chest compressions produce a characteristic ICG waveform with amplitude and frequency characteristics reflecting the depth and rate of compressions respectively.[10–12] The ability to measure ICG in real time via standard defibrillator pads provides the potential to quantify chest compression performance and provide audio-visual feedback to the resuscitator to optimise CPR efficacy. Retrospective analysis of the ICG has confirmed its utility in confirming chest compression rate and fraction but a correlation with compression depth has proved more challenging.[13–15]

This two phase experimental study investigated the relationship between mechanical and physiological markers of chest compression efficacy during CPR. The study also sought to establish the strength of correlation between the ICG and mechanical and invasive physiological measurements during CPR.

2. Materials and methods

This research was conducted in accordance with the United Kingdom Home Office UK Animals (Scientific Procedures) Act of 1986. Experiments were performed within a Good Laboratory Practice (GLP) accredited facility with full veterinary support.

Two experiments were performed using an identical porcine model. The first experiment investigated the relationship between compression depth, thrust and multiple invasive physiological measurements during mechanical CPR. The second experiment investigated the relationship between ICG amplitude measured via standard defibrillator pads, compression depth and $ETCO_2$. A total of 24 commercially sourced female White Landrace swine (9–10 weeks), (weight 30–34 kg) were used for the study (12 animals per experiment).

3. Animal preparation

Each animal was sedated by intramuscular injection (0.3 mg kg^{-1} of midazolam, 0.3 mg kg^{-1} of morphine, 7 mg kg^{-1} of ketamine and 7 μg kg^{-1} of medetomidine). Anaesthesia was induced with propofol intravenously to effect (approximately 60 mg) and mechanical ventilation was used to maintain normocapnia with 1.5–2% isoflurane in oxygen. An $ETCO_2$ sample line (Oridien, USA) was connected to the endotracheal tube to provide continuous monitoring. A pulse oximeter (SpO_2) sensor was connected to the animal's snout. Internal body temperature was monitored rectally and maintained between 36 °C and 38 °C. For experiment 1, 8 French introducer sheaths were placed in the right and left jugular veins and a 7 French sheath in the right common carotid artery by cut down. A pacing wire (Medtronic, USA) was placed in the right ventricle via the left jugular venous sheath. A Swan Ganz catheter (Edwards Lifesciences, USA) was placed in the right jugular vein (via introducer sheath) to enable determination of cardiac output (CO) using thermodilution. A Medex blood pressure transducer (Smith Medical, USA) was attached to the right common carotid sheath and blood pressure measured continuously. An ultrasonic flow probe (Transonic Systems, USA) was attached to the left carotid artery (without introducer sheath) to monitor carotid flow. A CPR/STAT Heart Lung Resuscitator 301 (HLR) mechanical resuscitator (Brunswick Biomedical Technologies, USA) was positioned over the animal's sternum using a customised frame. The location for HLR placement and point of compression was the midline of the sternum. A Philips QCPR puck was attached to the compression pad of the HLR and connected to a Philips MRx defibrillator (Philips, USA). Heartsine defibrillation electrodes (Heartsine Technologies, UK) were applied to the thorax in the sterno-apical (SA) position and connected to the HeartSine Samaritan PAD 500P defibrillator. Experiment 2 was performed in an identical manner, with the omission of Swan Ganz catheter placement and carotid flow measurement.

Ventricular fibrillation (VF) was induced in each model using the S44, Grass Single channel Stimulator (Grass Technologies, USA) at a frequency of 100 Hz and pulse duration of 2ms. Ventilation prior to the induction of VF was adjusted to maintain normocapnia with mechanical ventilation maintained for the duration of the protocol. No adrenaline was administered during either protocol.

4. Experiment 1: Protocol

After 3 min of untreated VF, CPR was initiated with the CPR/STAT Heart Lung Resuscitator 301 (HLR) mechanical resuscitator (Brunswick Biomedical Technologies, USA). THE HLR system was mounted on a customisable frame independently supported over the animal with adjustment prior to each experiment to ensure adequate contact. Compressions were applied at a rate of 110 compressions per minute and a selected thrust between 0–60 kg (0 kg, 20 kg, 30 kg, 40 kg, 50 kg, 60 kg) according to a predefined randomisation schedule. The HLR and Q-CPR puck were adjusted via the frame arrangement after each compression series to ensure continuous sternal contact. After 2 min the compression thrust was altered at 10 kg increments. This process was repeated for 12 min until all compression thrusts had been administered. The depth of compressions in mm was measured using the Philips MRx with Q-CPR (Philips, USA). Average compression depth (mm), systolic blood pressure (SBP) (mmHg), $ETCO_2$ (kPa), carotid flow (ml/min), cardiac output (CO) (L/min) were recorded prior to initiation of VF, at 1 min during untreated VF and after 1 min of each pre-defined compression thrust. Venous blood samples were collected and analysed using the i-Stat (Vet Scan, UK), prior to any interventions, after 3 min of untreated VF and following 12 min of CPR.

5. Experiment 2: Protocol

After 3 min of untreated VF mechanical CPR was initiated and maintained at 110 compressions per minute via the HLR device at incrementally increasing depths of 1 cm, ranging from 0 to 5 cm. CPR was performed for 2 min at each depth until all compression depths had been administered. The depth of compressions in mm was measured using the Philips MRx with Q-CPR (Philips, USA). $ETCO_2$, SBP and ICG amplitude were recorded at 30 s