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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

Table 1
Biochemical indices.

Variable	Pre-arrest mean (SD)	3 min untreated VF mean (SD)	12 min CPR mean (SD)
pH	7.44 (0.04)	7.7 (0.12)	7.34 (0.11)
PO_2 (kPa)	30.9 (4.9)	27.3 (6.1)	11.9 (7.1)
PCO_2 (kPa)	6.4 (0.6)	3.9 (1.0)	6.3 (2.3)
HCO_3 (mmol L^{-1})	24.0 (3.4)	31.4 (4.5)	24.1 (3.7)
Base excess (mmol L^{-1})	9.1 (2.4)	10.6 (5.6)	−2.0 (3.4)
Glucose (mmol L^{-1})	7.7 (2.4)	8.9 (5.3)	20.3 (9.8)
Na (mmol L^{-1})	138 (2.3)	138 (2.3)	132 (2.1)
K (mmol L^{-1})	3.6 (0.3)	3.7 (0.3)	6.2 (1.1)
Haemoglobin (g dL^{-1})	9.8 (1.8)	9.6 (2.4)	9.8 (1.5)

intervals. ICG amplitude (recorded in analogue-to-digital units (ADU) and converted to Ohms) was recorded using a HeartSine samaritan PAD 500P (Heartsine Technologies, Northern Ireland). This device detects transthoracic impedance by passing a high frequency, low amplitude sinusoidal current (30 kHz; 0.05 mA) between the defibrillation pads and measuring the resulting voltage at a sampling rate of 170.667 Hz. The ICG signal is derived with a bandwidth of 1.5–20 Hz. The root mean square (RMS) of the ICG amplitude was averaged over each 30 s window.

6. Data processing

Physiological data were captured manually in real time with simultaneous recording using customised data acquisition software. Compression depth was analysed retrospectively using proprietary Philips 'Event Review' software (Philips, USA). The impedance cardiogram data were retrospectively analysed using HeartSine Viewer Version 1.1 software (HeartSine Technologies, Northern Ireland). Samples were exported at 30 s time intervals and then converted from automated digital units (ADUs) into Ohms (19.91 ADU is equivalent to 1 Ω).

7. Statistical analysis

Statistical analysis was carried out independently of the investigators using SAS version 9.3. For both experiments mixed effect models were used in order to derive the within-animal correlation estimates for each pair of continuous variables, with animal as the random effect. A statistical analysis was performed for each pair of continuous variables. All pairs of non-missing observations were entered in the statistical model. In order to avoid non-normally distributed residuals with untransformed data, the values of each variable were replaced by their corresponding ranks prior to performing the statistical analyses.

8. Results

8.1. Experiment 1

Baseline pre-arrest physiological data were within normal limits and comparable between models: (SpO_2 mean (SD) 98% (2.4%), SBP mean (SD) 83 mmHg (10 mmHg), $ETCO_2$ mean (SD) 5.8 kPa (0.70 kPa), CO mean (SD) 3.8 l min^{-1} (0.80 l min^{-1}), carotid flow mean (SD) 196 ml min^{-1} (28 ml min^{-1}), temperature 37.9 °C (0.40 °C). Biochemical indices at baseline, 3 min and following completion of CPR are summarised in Table 1. Twelve measurements of CO and 1 measurement of carotid flow were not recorded during the protocol as a consequence of an error result during acquisition.

Moderate within animal correlations were observed between invasive physiological markers and chest compression depth ($r = 0.69–0.77$) and thrust (0.66–0.82) (Table 2). Scatter plots of compression depth and physiological parameters are presented in Fig. 1a–c There was a moderate within-animal correlation between applied thrust and achieved compression depth ($r = 0.75$) with scatter plots suggesting non-linearity and plateauing of depths at higher thrusts (Fig. 1d). Chest wall compliance was further analysed

Table 2
Experiment 1: summary within animal correlations for compression depth, thrust and physiological parameters.

Endpoint	Thrust	Depth	$ETCO_2$	SBP	Cardiac output	Carotid Flow
Thrust						
Samples (*N*)						
Correlation coeff						
95% CI						
Depth						
Samples (*N*)	72					
Correlation coeff	0.75					
95% CI	(0.65, 0.87)					
ETCO2						
Samples (*N*)	72	72				
Correlation coeff	0.66	0.72				
95% CI	(0.54, 0.79)	(0.59, 0.84)				
SBP						
Samples (*N*)	72	72	72			
Correlation coeff	0.82	0.77	0.80			
95% CI	(0.75, 0.91)	(0.68, 0.86)	(0.68, 0.90)			
Cardiac output						
Samples (*N*)	60	60	60	60		
Correlation coeff	0.73	0.76	0.73	0.82		
95% CI	(0.65, 0.85)	(0.62 0.85)	(0.50, 0.86)	(0.70, 0.91)		
Carotid flow						
Samples (*N*)	71	71	71	71	59	
Correlation coeff	0.72	0.69	0.74	0.75	0.75	
905% CI	(0.63, 0.83)	(0.54, 0.80)	(0.59-0.84)	(0.62, 0.87)	(0.56, 0.86)	

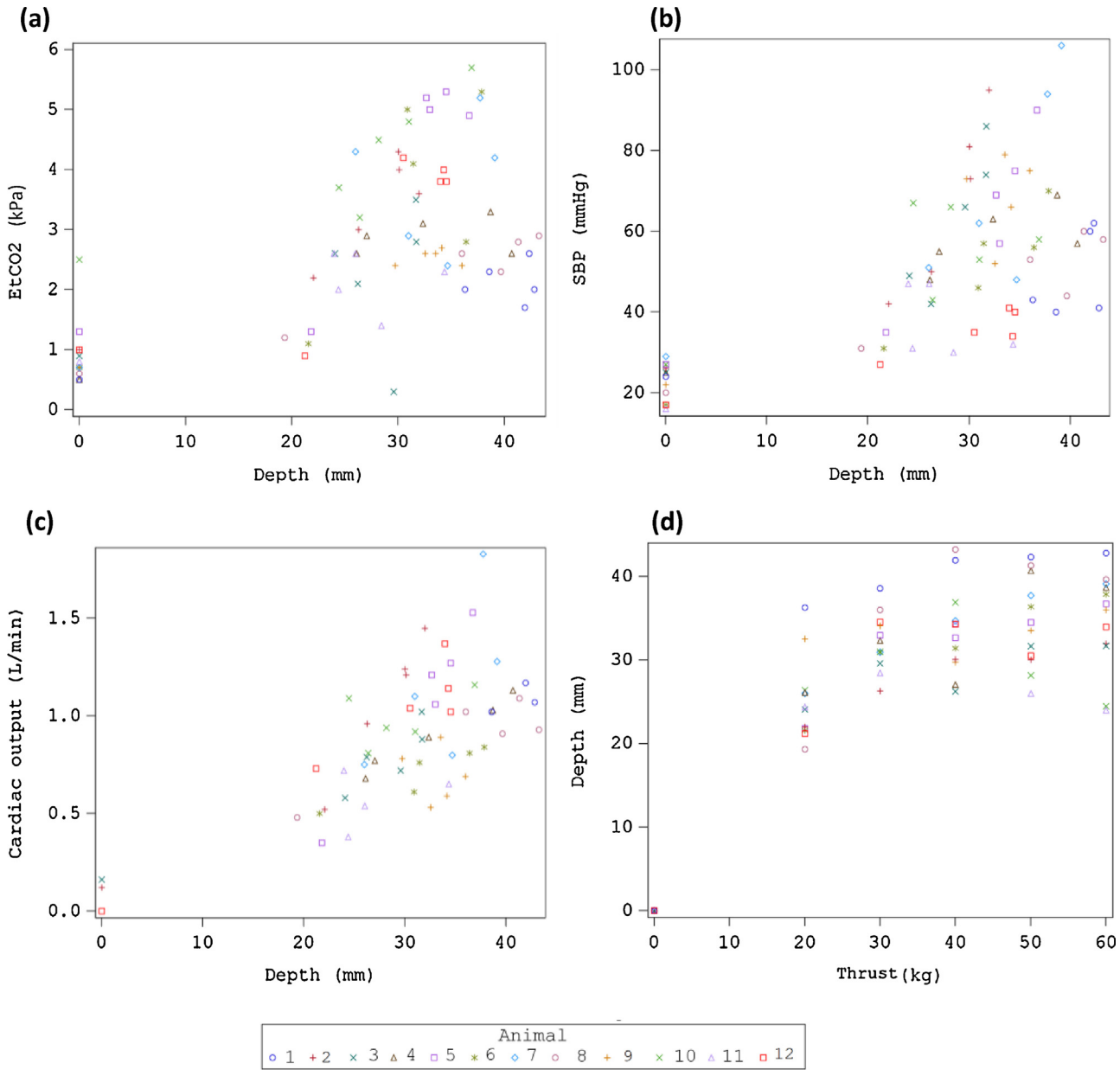


Fig. 1. Scatter plots of compression depth and physiological parameters (a) $ETCO_2$, (b) SBP (c) CO and (d) applied thrust.

by calculating the force necessary to achieve 30 mm of compression for each model using linear regression modelling. A mean thrust of 40.4 kg (±8.6 kg) was required to achieve 30 mm compression depth with a broad range in required thrust (28–55 kg).

9. Experiment 2

A strong within-animal correlation was observed between ICG amplitude and compression depth ($r = 0.89$) (Table 3). A strong within-animal correlation was also observed between ICG amplitude and $ETCO_2$ ($r = 0.85$) and a moderate correlation between ICG amplitude and SBP ($r = 0.69$). Scatter plots of ICG vs depth and $ETCO_2$ are shown in Fig. 2 A representation of corresponding depth and ICG recordings is presented in Fig. 3 All correlations of ICG amplitude with depth showed large point estimates with confidence intervals above 0.8 indicating a strong association.

Table 3
Experiment 2: Summary within animal correlations for ICG, compression depth, ETCO2 and Systolic blood pressure.

Endpoint	ICG amplitude	Depth	$ETCO_2$	SBP
ICG amplitude				
Samples (*N*)				
Correlation coeff				
95% CI				
Depth				
Samples (*N*)	280			
Correlation coeff	0.89			
95% CI	(0.87, 0.91)			
$ETCO_2$				
Samples (*N*)	283	281		
Correlation coeff	0.85	0.81		
95% CI	(0.82, 0.87)	(0.77, 0.85)		
SBP				
Samples (*N*)	283	281	285	
Correlation coeff	0.69	0.63	0.86	
95% CI	(0.62, 0.75)	(0.56, 0.69)	(0.83, 0.89)	

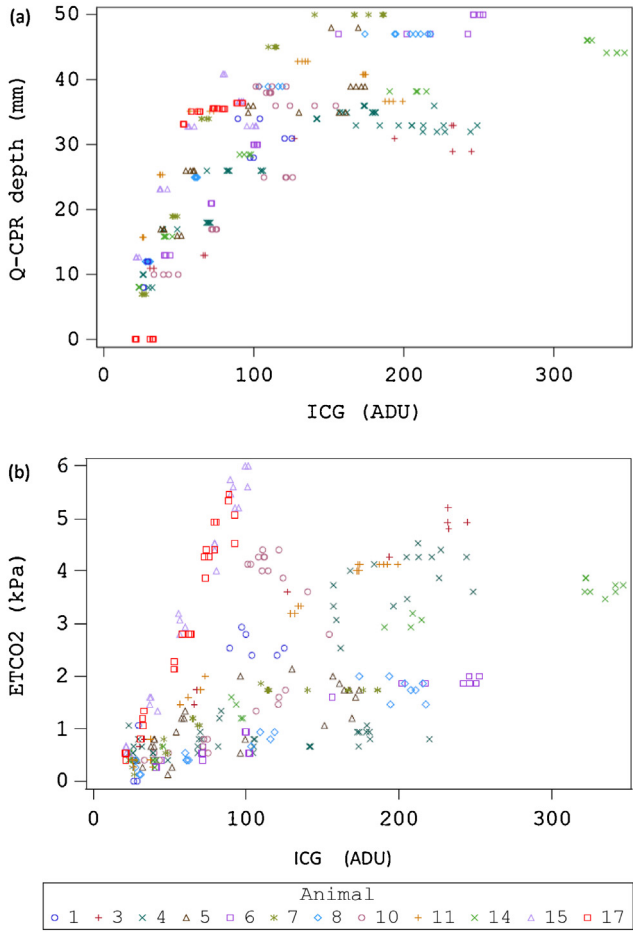


Fig. 2. Scatter plot of ICG amplitude versus (a) Q-CPR depth (b) $EtCO_2$.

10. Discussion

There is now overwhelming evidence highlighting the importance of optimising CC depth, rate and fraction to achieve successful resuscitation.[16–18] Despite widespread dissemination of resuscitation guidelines and training, CPR performance by lay responders remains suboptimal.[3] The development of accelerometer based CPR feedback devices has improved CPR metrics but not outcome and a non-invasive physiological indicator of resuscitation performance remains elusive.

Previous studies have demonstrated the utility of the ICG to quantify chest compression rate in the retrospective setting however the ability to quantify compression depth has proved more contentious.[10–15] The objective of this study was to investigate the relationship between mechanical and physiological markers of chest compression efficacy and explore the relationship between ICG and the most clinically applicable resuscitation parameters, compression depth and $ETCO_2$.

Experiment 1 confirmed moderate within animal correlations between compression depth and invasive physiological parameters during CPR in a porcine model ($r = 0.69–0.77$). However when looking at the overall correlation estimates (incorporating inter-animal variability) there was a significant reduction in correlation between compression depth and $ETCO_2$ ($r = 0.51$) and SBP ($r = 0.62$). These results demonstrate the significant inter-animal variability in achieved physiological markers during CPR and the challenge in applying a single mechanical metric to accurately quantify chest compression efficacy in the context of complex and evolving resuscitation physiology. A moderate correlation was also demonstrated between applied compression force and achieved depth with plateauing of depth at higher thrusts (40–60 kg) (Fig. 1d). These findings reflect the dynamic changes in chest wall compliance and thoracic morphology as chest compressions are administered and maximal sternal displacement is achieved. A prior analysis of 91 out of hospital cardiac arrest victims support these findings, reporting a strong, non-linear relationship between compression depth and thrust and significant variation in thrust necessary to achieve 38 mm compression depth.[19]

Experiment 2 sought to more closely examine the relationship between ICG amplitude and compression depth, $ETCO_2$ and SBP. A depth range of 0–5 cm was chosen to reflect achievable sternal displacement within this size and age of model whilst avoiding mechanically mediated thoracic injury. The results demonstrated a strong within-animal correlation between chest compression depth and ICG amplitude ($r = 0.89$). A strong within-animal correlation was also observed between ICG amplitude and end-tidal CO_2 ($r = 0.85$) which compared favourably to the correlation between compression depth and $ETCO_2$ ($r = 0.81$). Mean TTI was 49.3 Ohm and baseline TTI did not significantly alter the amplitude of the ICG signal or derived correlations. Zhang et al. compared TTI amplitude to both CC depth and coronary perfusion pressure (CPP) in a porcine study.[12] The study compared suboptimal (35 mm) and optimal (50 mm) CCs from 14 pigs during 2 min cycles of CPR. The authors reported similarly high correlation coefficients between TTI amplitude and depth (0.89) and CPP (0.83) and concluded that ICG amplitude could potentially be used as a predictor of CC depth.

Alonso et al. subsequently performed a retrospective analysis of ICG and compression depth in 60 cardiac arrest patients and reported marked inter-patient variability in the linear relationship between TTI and compression depth.[20] The authors concluded that TTI was an unreliable marker of compression depth during clinical CPR. We applied a mixed effect models in order to derive the within-animal correlation estimates for each pair of continuous variables. When overall correlation estimates were calculated for the cohort of animals in experiment 2 there was a decrease in the correlation between ICG amplitude and compression depth ($r = 0.79$) and $ETCO_2$ ($r = 0.64$). The results confirm significant inter-animal variability in the ICG waveform as observed for mechanical measures during CPR but relatively preserved correlation estimates.

This study demonstrates the challenge in utilising any mechanical parameter as an accurate predictor of chest compression efficacy when correlated with invasive physiological measurements. Despite age and weight matched models significant inter-animal variation between achieved mechanical and physiological metrics was observed. ICG amplitude demonstrated strong within animal correlations with compression depth. The correlation between ICG and end-tidal CO_2 also compared favourably with compression depth demonstrating a potential utility in optimising chest compression efficacy. Further research is needed to overcome the observed inter-animal variability in ICG correlations to optimise performance and realise this potential clinical benefit.

11. Limitations

The reported experiments utilised age and weight matched animals in a controlled experimental setting with accurate, mechanical application of chest compressions at a pre-specified rate. Manual adjustment of the HLR position was required between compression series to ensure consistent sternal contact. The incremental, small, repositioning needed to maintain contact with the sternum impacted the absolute anterior-posterior depth and thus may have impacted the reported outcomes. ICG performance was



Fig. 3. Example of within-animal recording of (a) compression depth and (b)–(f) corresponding ICG amplitude.

not assessed in the context of subject and resuscitation variation that is routinely encountered in the clinical setting. The maximal achieved compression depth within this experimental model was 5 cm and therefore below the most relevant clinical range of 5–6 cm. We performed a within animal analysis and acknowledge that inter-animal variability adversely affects the derived correlations for ICG and mechanical and physiological parameters. Experiment 1 collected averaged data at 1 min following each intervention. Experiment 2 collected data at 30 s intervals at incremental depths in contrast to experiment 1 in which thrust was randomised and data collected at 2 min intervals. Continuous ventilation during CPR produce changes in the ICG waveform though this is of low amplitude and should not distort the analysed amplitude during chest compressions.

12. Conclusions

Chest compression depth and force demonstrated moderate correlations with invasive physiological markers of chest compression efficacy during CPR in a porcine model. The ICG amplitude measured via defibrillator pads achieved strong within-animal correlations with compression depth and $ETCO_2$ suggesting a potential

utility to accurately quantify and communicate chest compression performance to optimise outcome.

Conflict of interest statement

Dr Andrew Howe received a research grant from HeartSine Technologies Ltd in 2010 as part of a 2 year research doctorate through Queen's University Belfast. This research grant provided funding for the submitted experiments. Dr Peter O'Hare and Dr Rebecca Di Maio are employees of HeartSine Technologies Ltd. Dr David McEneaney and Prof Jennifer Adgey are members on the HeartSine Clinical Advisory Board. Bruno Delafont received consultancy payment from HeartSine Technologies Ltd for statistical services. Paul Crawford has received payments for veterinary consultancy from Heartsine Technologies Ltd.

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