Use of impedance threshold device in conjunction with our novel adhesive glove device for ACD-CPR does not result in additional chest decompression


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Abstract

Objective: To evaluate the hemodynamic effects of using an adhesive glove device (AGD) to perform active compression–decompression CPR (ACD-CPR) in conjunction with an impedance threshold device (ITD) in a pediatric cardiac arrest model.

Design: Controlled, randomized animal study.

Methods: In this study, 18 piglets were anesthetized, ventilated, and continuously monitored. After 3 min of untreated ventricular fibrillation, animals were randomized (6/group) to receive either standard CPR (S-CPR), active compression–decompression CPR via adhesive glove device (AGD-CPR) or ACD-CPR along with an ITD (AGD-CPR + ITD) for 2 min at 100–120 compressions/min. AGD is delivered using a fingerless leather glove with a Velcro patch on the palm area and the counter Velcro patch adhered to the pig’s chest. Data (mean ± SD) were analyzed using one-way ANOVA with pairwise multiple comparisons to assess differences between groups. p-Value < 0.05 was considered significant.

Results: Both AGD-CPR and AGD-CPR + ITD groups produced lower intrathoracic pressure (IttP, mmHg) during decompression phase (−13.4 ± 6.7, p = 0.01 and −11.9 ± 6.5, p = 0.01, respectively) in comparison to S-CPR (−0.3 ± 4.2). Carotid blood flow (CBF, % of baseline ml/min) was higher in AGD-CPR and AGD-CPR + ITD (respectively, 64.3 ± 47.3%, p = 0.03 and 67.5 ± 33.1%, p = 0.04) as compared with S-CPR (29.1 ± 12.5%). Coronary perfusion pressure (CPP, mmHg) was higher in ACD-CPR and AGD-CPR + ITD (respectively, 19 ± 4.6, p = 0.04 and 25.6 ± 12.1, p = 0.02) when compared to S-CPR (9.6 ± 9.1). There was statistically significant difference between AGD-CPR and AGD-CPR + ITD groups with reference to intrathoracic pressure, carotid blood flow and coronary perfusion pressure.

Conclusion: Active compression decompression delivered by this simple and inexpensive adhesive glove device resulted in improved cerebral blood flow and coronary perfusion pressure. There was no statistically significant added effect of ITD use along with AGD-CPR on the decompression of the chest.

1. Introduction

Pediatric cardiopulmonary arrest has a poor prognosis, with survival rates ranging from 2% to 17% [1–4]. There are confounding factors that contribute to the low cardiopulmonary resuscitation (CPR) success rate, including difficulty in acquiring vascular access and suboptimal CPR equipment suitable for small patients [2].

Recent CPR guidelines call for continuous uninterrupted chest compression at a fast rate (>100 compressions/min), allowing for
full chest recoil [5–7]. Standard CPR (S-CPR) is intrinsically inefficient as, during the decompression phase of CPR, limited chest recoil is achieved. Incomplete chest recoil during S-CPR produces a limited decrease in intrathoracic pressure (IttP) following compression, leaving a residual positive IttP and, thus, restricting venous return [6]. Different CPR techniques and devices have been designed to decrease IttP and improve CPR efficiency.

A leading candidate method to enhance CPR quality is active compression–decompression CPR (ACD-CPR). ACD-CPR requires active lifting of the anterior chest wall by the rescuer during the decompression phase of CPR, maximizing chest recoil and lowering IttP [8,9]. ACD-CPR improves cardiac output, IttP, vital organ perfusion pressures [8,10] and resuscitation rates [11–13]. ACD-CPR is performed with a suction device fixed to the chest wall suitable for adult CPR. Unfortunately, to date, there is no device available to perform ACD-CPR in the pediatric population.

An impedance threshold device (ITD) may also lower IttP during the chest compression phase of CPR [14]. The ITD prevents inflow of air into the lungs during the decompression phase until a certain “cracking pressure” is reached, creating a more negative IttP and better cardiac output [14–17]. In adult patients, the combination of ACD-CPR in tandem with an ITD exerts a synergistic effect on IttP, improving hemodynamics and survival rates [18–22]. Interestingly, a recent multi-center trial of 8700 patients randomized to ITD or no ITD questions the benefit of using an ITD in adult CPR patients [22,23].

However, few studies have evaluated the combination of ACD-CPR + ITD in the pediatric population [24]. Adult-size ACD devices are bulky and heavy, and do not mold to small chest sizes (i.e. the pediatric thorax) [25–27]. Our group developed a novel adhesive glove device (AGD) to enable ACD-CPR to be efficiently performed in infants and children. The glove device fits a wide range of rescuer hand sizes, can easily be made to conform to different chest sizes, and is also much lighter than the commercially available adult ACD devices [28]. ACD-CPR delivered by AGD was able to improve vital organ perfusion pressures in a pediatric CPR model [28,29].

This study was designed to evaluate the effects of AGD-CPR with and without ITD on cardiac-circulatory variables in a pediatric swine model. We hypothesized that AGD-CPR generates a more negative IttP than S-CPR and that the use of an ITD during AGD-CPR (AGD-CPR + ITD) will further augment this effect, enhancing cardiac and cerebral perfusion.

2. Methods

This controlled, randomized study was approved by the University of Florida Health Science Center Institutional Animal Care and Use Committee (IACUC) and the animals were managed in accordance with American Physiological Society guidelines. Animal care and use was performed by qualified individuals supervised by veterinarians and all facilities meet the standards of the American Association for Accreditation of Laboratory Animal Care.

2.1. Animal preparation and measurement

Eighteen healthy, two-month-old (weight ~15 kg) farm piglets (University of Florida Swine Unit) of either sex were used in this study. Animals were sedated with a single bolus intramuscular injection of ketamine (15 mg/kg). Once the appropriate level of sedation was achieved, anesthesia was induced with 3–5% isoflurane in 100% oxygen delivered by a nose cone, followed by oral endotracheal intubation. Mechanical ventilation was achieved with a rate- and volume-regulated ventilator (Surgivet Vaprostic Anesthesia Machine, Smiths Medical, Dublin, OH) and anesthesia was maintained with titrated isoflurane (1.6 ± 0.2%). Ventilation rate and tidal volume were adjusted to maintain an end-tidal CO₂ of 37 ± 4.1 mmHg. Lactated Ringer’s solution (10 mL/kg/h) was administered continuously throughout the preparation and resuscitation phase using an intravenous infusion pump. EKG leads were placed on the limbs for the continuous monitoring of heart rate and rhythm.

After intubation, the right carotid artery, left internal jugular (IJ) vein and femoral artery were used exposing standard cut-down techniques. A vascular introducer sheath (5F and 15 cm) was placed in the left IJ. Piglets were instrumented with micro-manometer pressure tip catheters (Millar Instruments, Houston, TX) introduced into the pleural cavity and right atrium, for measurement of intrathoracic pressure (IttP) and right atrial blood pressure (RAP), respectively. A 6F introducer catheter was placed in the right femoral artery for invasive aortic pressure (AP) monitoring (by a fluid-filled catheter transducer) and to obtain blood samples for I-STAT analysis of blood samples. Carotid blood flow was measured using a 3 mm transonic flow probe (Animal Blood Flow Meter T206, Transonic Systems Inc, Ithaca, NY) placed around the carotid artery. Correct catheter placement was verified by fluoroscopy.

Once instrumentation was complete, heparin (50 mg/kg) was given to all animals as a single intravenous bolus. Coronary perfusion pressure during diastole (relaxation) was defined as the maximal difference between aortic and right atrial pressures.

2.2. Adhesive glove device and impedance threshold device description

The adhesive glove device consisted of a leather glove modified to expose the fingers and thumb (to allow interlocking of the rescuer’s fingers during CPR) with an adjustable strap on the dorsal aspect of the glove for proper fit (Fig. 1) [28,29]. A Velcro patch was sewn to the Palmer aspect of the glove. The counter Velcro patch was adhered to the animals’ chest wall using an adhesive pad. An impedance threshold device (ITD) was attached between the endotracheal tube and rebreathing hose of the anesthesia machine. The ITD device was turned on just before CPR was to be administered.

3. Experimental protocol

Baseline hemodynamic values and carotid blood flow were recorded over 2 min with the animal anesthetized with isoflurane maintained at 1.6 ± 0.2%. Subsequent to baseline data collection, ventricular fibrillation (VF) was induced with a 100 Hz alternating current applied via a non-coated guide wire advanced into the right ventricle (via IJ catheter), and ventilation and oxygenation were discontinued. Once VF was achieved (confirmed by the characteristic EKG waveform and the precipitous fall in AP) and, after 3 min of untreated VF, animals were randomized (6 per group) to receive either standard CPR (S-CPR), active compression–decompression CPR using AGD (AGD-CPR) or AGD-CPR in tandem with the ITD (AGD-CPR + ITD). The order of treatment was determined by random number generated. After the initial untreated VF, chest compressions were initiated according to the group (delivered by PALS certified health care providers blinded to data recording) and mechanical ventilation was re-instituted at eight breaths per minute. The AGD-CPR + ITD group received chest compressions and breaths by hand bagging as the ITD device indicated. Hemodynamic parameters (BP, RAP, AP, IttP) and carotid blood flow were continuously monitored intra-CPR.

Chest compressions were given by a single rescuer at 100–120 compressions per minute for two full minutes. Following the first two minutes, direct 70 J biphasic shock defibrillation (Cardio Pak, Physio-Control, Redmond, WA) was used as an attempt to restore spontaneous circulation (ROSC). If the animal was still in VF after
defibrillation, CPR was continued for a second cycle of two minutes. During this cycle, an epinephrine bolus of 0.01 mg/kg (1:10,000) was given. At the end of the two minutes of CPR, there was a short pause to assess cardiac rhythm and, if VF was still present, a shock of 150 J was given CPR was continued per AHA (PALS) guidelines for 20 min or until ROSC was achieved. ROSC is defined as an unassisted pulse with an aortic systolic pressure of >60 mmHg, sustained for one minute. Animals achieving ROSC remained anesthetized during the ensuing 30 min post-resuscitation simulated intensive care period and were placed on an epinephrine drip. At the conclusion of the experimental protocol, all animals were humanely euthanized.

4. Measurements and data acquisition

The primary endpoint was evaluating chest decompression efficacy as evident by negative ITD and improved CPP and CBF. Secondary end points were time of ROSC, changes in other hemodynamic variables, number of defibrillatory shocks and epinephrine administration. Pressure tracing, ECG rhythm and carotid blood flow were continuously monitored with a data acquisition and recording system (ADInstruments PowerLab® Systems, Castle Hill, NSW, Australia), which sent real time continuous data to computer software (Chart Pro V7.3 by ADInstruments PowerLab® Systems, Castle Hill, NSW, Australia). The sampling rate for data collection was set at 100 data points per second. Coronary perfusion pressure and ITD were determined by an average of every compression over the first 2 min of CPR. By averaging the entire CPR cycle value variation due to ventilation and rescuer fatigue could be accounted. Venous and arterial blood gases, as well as plasma concentrations of potassium, sodium, lactic acid, hematocrit and ionized calcium (iSTAT Blood Gas Analyzer, Windsor, NJ) were performed at baseline and 30 min post-ROSC for all animals.

4.1. Statistical consideration

Data are reported as mean ± SD. Normality and equality of variances of the data were assessed using the Kolmogorov–Smirnov and Leven tests, respectively. For each group, a one-way ANOVA for repeated measures was used (baseline vs CPR). A p-value < 0.05 was considered statistically significant. All data analyses were conducted using PC-SigmaStat V3.0 (Aspire Software Int., Ashburn, VA). For the major analyses, the primary inference was based on the difference between S-CPR vs AGD-CPR vs AGD-CPR-ITD. The study had over 80% power to detect a difference of 0.37 standard deviations in the raw measures at p = 0.05, two-sided.

5. Results

Eighteen piglets (6/group) were randomized into either AGD-CPR, AGD-CPR + ITD or S-CPR. All three groups were comparable at baseline, as shown in Table 1. Hemodynamic data during the first two minutes of resuscitation are shown in Table 2. There was no difference in peak compression (systolic) BP and RAP between groups (Table 2 and Fig. 2). Fifteen animals survived to 30 min post-ROSC, 5 on S-CPR group, 5 on AGD-CPR group and 5 on AGD-CPR + ITD group (Table 2).

Intrathoracic pressure (mmHg) during the decompression (diastolic) phase of CPR was statistically significantly lower on both AGD-CPR and AGD-CPR + ITD in comparison to S-CPR. There was no statistically significant difference in intra-thoracic pressure (mmHg) during the decompression (diastolic) phase of CPR when AGD-CPR group was compared to AGD-CPR + ITD. CBF (% of baseline mL/min) was significantly higher with both AGD-CPR and AGD-CPR + ITD when compared to S-CPR, although there was no difference in CPP when AGD-CPR was compared to AGD-CPR + ITD. CPP (mmHg) was significantly higher for both AGD-CPR and AGD-CPR + ITD in comparison to S-CPR, though, there was no difference in CPP when AGD-CPR was compared to AGD-CPR + ITD (Fig. 2). Mean systolic RA pressure was significantly lower for AGD-CPR or AGD-CPR + ITD when compared to S-CPR, yet there was no difference in mean systolic RA pressure between AGD-CPR and AGD-CPR + ITD. There was no difference in rate and time of ROSC, doses of epinephrine administration, number of shocks, end-tidal
Table 1
Baseline data.

<table>
<thead>
<tr>
<th></th>
<th>S-CPR</th>
<th>ACD-CPR</th>
<th>ACD-CPR + ITD</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>Gender (F/M)</td>
<td>4/2</td>
<td>2/4</td>
<td>3/3</td>
<td>0.7</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>112 ± 14</td>
<td>103 ± 23</td>
<td>100 ± 17</td>
<td>0.2</td>
</tr>
<tr>
<td>Mean systolic BP (mmHg)</td>
<td>95 ± 2</td>
<td>102 ± 11</td>
<td>95 ± 7</td>
<td>0.7</td>
</tr>
<tr>
<td>Right atrial pressure (mmHg)</td>
<td>12 ± 3.6</td>
<td>9 ± 3</td>
<td>10 ± 3</td>
<td>0.6</td>
</tr>
<tr>
<td>Coronary perfusion pressure (mmHg)</td>
<td>37 ± 6.1</td>
<td>41 ± 9.1</td>
<td>36 ± 7</td>
<td>0.7</td>
</tr>
<tr>
<td>Carotid blood flow (ml/min)</td>
<td>208 ± 49</td>
<td>160 ± 96</td>
<td>177 ± 68</td>
<td>0.1</td>
</tr>
<tr>
<td>End tidal CO2 (mmHg)</td>
<td>35.0 ± 5.1</td>
<td>37.0 ± 2.1</td>
<td>36.0 ± 2.9</td>
<td>0.7</td>
</tr>
<tr>
<td>Arterial PO2 (mmHg) (100% FiO2)</td>
<td>523 ± 168</td>
<td>510 ± 109</td>
<td>505 ± 145</td>
<td>0.6</td>
</tr>
<tr>
<td>Arterial saturation (%)</td>
<td>99 ± 0.0</td>
<td>99 ± 0.4</td>
<td>99 ± 0.3</td>
<td>0.9</td>
</tr>
<tr>
<td>Sodium (mmol/L)</td>
<td>137 ± 13</td>
<td>136 ± 3.1</td>
<td>135 ± 4.1</td>
<td>0.8</td>
</tr>
<tr>
<td>Potassium (mmol/L)</td>
<td>3.89 ± 2.1</td>
<td>4.1 ± 0.8</td>
<td>4.2 ± 16</td>
<td>0.6</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>21.1 ± 2.5</td>
<td>24.2 ± 6.9</td>
<td>23 ± 6.7</td>
<td>0.7</td>
</tr>
<tr>
<td>Lactic acid (mmol/L)</td>
<td>1.5 ± 0.8</td>
<td>1.5 ± 0.4</td>
<td>1.4 ± 0.2</td>
<td>0.8</td>
</tr>
<tr>
<td>Ionized calcium (mmol/L)</td>
<td>1.3 ± 0.1</td>
<td>1.21 ± 0.1</td>
<td>1.41 ± 0.2</td>
<td>0.7</td>
</tr>
</tbody>
</table>

BP indicates blood pressure; bpm beats per minute.

Table 2
Intra-resuscitation parameters (n = 18).

<table>
<thead>
<tr>
<th></th>
<th>S-CPR vs AGD-CPR</th>
<th>S-CPR vs AGD-CPR + ITD</th>
<th>AGD-CPR vs AGD-CPR + ITD</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean aortic “systolic” pressures (mmHg)</td>
<td>90 ± 25</td>
<td>92 ± 25</td>
<td>0.8</td>
<td>90 ± 25</td>
</tr>
<tr>
<td>Mean aortic “diastolic” pressures (mmHg)</td>
<td>17 ± 12</td>
<td>19 ± 19</td>
<td>0.1</td>
<td>17 ± 12</td>
</tr>
<tr>
<td>Mean RA “systolic” pressures (mmHg)</td>
<td>229 ± 79</td>
<td>191.3 ± 42.2</td>
<td>0.8</td>
<td>229 ± 79</td>
</tr>
<tr>
<td>Mean RA “diastolic” pressures (mmHg)</td>
<td>9.5 ± 4.5</td>
<td>−1.7 ± 6.3</td>
<td>0.03</td>
<td>9.5 ± 4.5</td>
</tr>
<tr>
<td>Intrathoracic pressure (mmHg)</td>
<td>−0.3 ± 4.2</td>
<td>−13.4 ± 6.7</td>
<td>0.01</td>
<td>−0.3 ± 4.2</td>
</tr>
<tr>
<td>CPP (mmHg)</td>
<td>9.6 ± 9.1</td>
<td>19.7 ± 4.6</td>
<td>0.04</td>
<td>9.6 ± 9.1</td>
</tr>
<tr>
<td>Actual compressions given/min</td>
<td>119 ± 5</td>
<td>100 ± 16</td>
<td>0.04</td>
<td>119 ± 5</td>
</tr>
<tr>
<td>Mean carotid blood flow (ml/min)</td>
<td>65 ± 29</td>
<td>129 ± 108</td>
<td>0.04</td>
<td>65 ± 29</td>
</tr>
<tr>
<td>Mean carotid blood flow (% baseline)</td>
<td>29.1 ± 12.5</td>
<td>67.5 ± 33.1</td>
<td>0.04</td>
<td>29.1 ± 12.5</td>
</tr>
<tr>
<td>End tidal CO2 (mmHg)</td>
<td>25 ± 9</td>
<td>27 ± 7</td>
<td>0.7</td>
<td>25 ± 9</td>
</tr>
<tr>
<td>Epinephrine administration</td>
<td>1.8 ± 3.7</td>
<td>1.6 ± 1.8</td>
<td>0.7</td>
<td>1.8 ± 3.7</td>
</tr>
<tr>
<td>Shocks delivery</td>
<td>1.75 ± 3.5</td>
<td>1.5 ± 1.0</td>
<td>0.7</td>
<td>1.75 ± 3.5</td>
</tr>
<tr>
<td>Lactic acid (mmol/L)</td>
<td>2.8 ± 3.4</td>
<td>2.5 ± 2.8</td>
<td>0.8</td>
<td>2.8 ± 3.4</td>
</tr>
<tr>
<td>Number of ROSC 30min</td>
<td>5/6</td>
<td>5/6</td>
<td>1</td>
<td>5/6</td>
</tr>
<tr>
<td>Time to ROSC (min)</td>
<td>9.4 ± 9.8</td>
<td>5.5 ± 2.8</td>
<td>0.1</td>
<td>9.4 ± 9.8</td>
</tr>
</tbody>
</table>

CPP indicates coronary perfusion pressure; RA, right atrial.

* Statistically significant difference p value when compared to S-CPR.

CO2, sodium, potassium, hematocrit, ionized calcium, lactic acid or venous and arterial gasses between groups.

6. Discussion

The current study demonstrated that the addition of ITD to our glove device did not improve the decompression of the chest any further than active compression–decompression alone, which significantly lowers intrathoracic pressure and, subsequently, increases vital organ blood flow.

ACD-CPR has been shown to improve cardiac output, intrathoracic pressure, vital organ perfusion pressures [8,10] and resuscitation rates [11–13] in adults. Adult-sized ACD-CPR devices are bulky and heavy, lead to early rescue fatigue and do not mold to pediatric chest sizes [25–27]. To date, there is no device available to perform ACD-CPR in the pediatric population. Our group developed

![Fig. 2](image-url) Magnified and superimposed intravascular pressure (aortic pressure; black line and right atrial pressure; thick gray line) curves for: (A) standard cardiopulmonary resuscitation (S-CPR), (B) adhesive glove device CPR (AGD-CPR) and (C) adhesive glove device in tandem with impedance threshold device CPR (AGD-CPR+ITD). The curve shows three consecutive compressions for each method.
a simple and low cost adhesive glove device (AGD) to perform ACD-CPR in pediatric size patients. The glove device can easily be made to conform to different chest sizes and is much lighter and easier to use than the commercially available adult ACD-CPR devices [28].

The use of an impedance threshold device (ITD) during CPR ACD-CPR may further lower intrathoracic pressure and thus improve venous return and cardiac output. As some arrest victims have gasping breaths during CPR, passive airflow into the lungs can neutralize the negative intrathoracic pressure from ACD-CPR, resulting in lower venous return and lower cardiac output [14–17]. In adults, the combination of ACD-CPR with an ITD exerts a synergistic effect on intrathoracic pressure, improving hemodynamics and survival rate [18–21]. However, few studies have evaluated the combination of ACD-CPR + ITD in the pediatric population [24]. Our previous animal studies showed active compression–decompression CPR delivered by our adhesive glove device improved the effectiveness of chest compressions, thus resulting in improved carotid blood flow and higher coronary perfusion pressure as compared with standard CPR [28,29]. The current work builds upon our previous animal studies and aims at combining AGD-CPR with ITD to further improve intrathoracic pressure, cardiac output and hemodynamics in a pediatric swine model.

AGD-CPR with or without ITD generated more than double the carotid blood flow, and decreased mean diastolic right atrial pressure, as compared to S-CPR. This reduction in right atrial pressure is translated into improved venous return and improved heart and brain perfusion, which is confirmed by increased carotid blood flow. Carotid blood flow can be a good estimation of cerebral perfusion. Improved perfusion is likely due to an increase in venous return. As this study was not designed to evaluate neurologic damage and long term outcome, further studies are needed to assess the effect of AGD-CPR on neurological status.

Both AGD-CPR alone or with ITD improved myocardial perfusion pressure as compared to S-CPR. There were fewer actual chest compressions per minute in the AGD-CPR group with or without ITD when compared to the S-CPR group (respectively 19 and 15% fewer compressions), as shown in Table 2, probably because it takes longer to actively decompress the chest. The fewer number of compressions was counterbalanced by improved ejected volume with each compression, resulting in an overall net increase in cardiac output; as evident by improved CPP and carotid blood flow.

Interestingly, there was no significant difference between AGD-CPR and AGD-CPR + ITD. The use of AGD-CPR in this model might already be maximizing the decompression of the chest and venous return, leaving little room for improvement by adding an ITD. Another possible explanation for the lack of a synergistic effect between ITD and AGD-CPR may be size of our target patient. Even though the chest wall of juvenile piglets (and children) is more compliant than that of adults, they lack an intrinsic tendency to rebound when released from compression during CPR [30]. This may have limited the amount of air movement during chest decompression and thus reducing the effectiveness of the ITD. The natural lack of chest rebound also underscores the importance of active lifting during chest decompression, as provided by the ACD technique. Because intrathoracic venous blood return is highly dependent on the fall of ItpP during the decompression phase of CPR, the use of ACD with or without ITD may therefore be even more important in pediatric patients than adults [24].

There was no difference between short term survival rate between treatment groups. Low ROSC rate (~80%) could be due to relatively high electrical defibrillation dose (5J/kg) used in this study. High defibrillation voltage could have caused myocardium damage and reduced rate of ROSC.

It is important to discuss that the absolute value of coronary perfusion pressure and carotid blood flow detected in the current study was different from our previously reported data [29]. We believe that the variation in values is due to several factors. We used different Transonic probes to measure CBF, T-206 during the first reported study and TS–420 during the current study. Also, RAP was measured using a fluid filled catheter transducer on the first study, while we used a Millar micro-manometer pressure tip catheter (SPR–524) during the current study. Body weight was different between the first and second studies. Animals on the first study had average body weights of 12 kg, while the current study had average body weights of 15 kg. Finally, CPR providers were different between both studies. As the quality of CPR is greatly influenced by the rescuer ability and stamina, having different CPR providers can greatly affect hemodynamic values obtained during CPR. Though all the rescuers used during both studies were among experimental staff. However, although the total CPP and CBF values measured were different between studies, the percentage change from baseline was very similar between studies. In summary, we believe that the differences in absolute values could be due to changes in equipment, use of different rescuers and differences in body weight. Both studies show higher CBF and CPP for the AGD-CPR group as compared to the S-CPR group.

Our study has several limitations. A Hawthorne effect cannot be excluded since the nature of this study precluded the investigators from being completely blinded to the procedures. However, at the peak of compressions, systolic BP (AP), systolic ItpP and systolic RAP during the compression phase were not statistically significantly different in all three groups is an indication that the chest compression quality and effort were the same for all groups.

The limited number of animals did not allow us to answer all pertinent questions. First, this study did not evaluate the effect of CPR with the ITD alone. The current study was focused on evaluating if ITD would synergistically improve ACD and not to evaluate if ITD by itself would improve S-CPR. The use of an ITD alone has been associated with improved CPR efficacy in both animal models and humans [14,16]. Interestingly, a recent multi-center trial of 8700 patients randomized to ITD or no ITD questions the benefit of using an ITD in adult CPR patients [22,23]. Second limitation: electrical ventricular fibrillation was the method used to induce cardiac arrest, while asphyxiation and respiratory arrest are the most likely cause of cardiac arrest in human infants. And finally, data from an animal model (using healthy piglets) may not be fully transferable into a clinical setting. Further animal studies are necessary to allow a more comprehensive estimation of the efficiency of this technique before it can be recommended for clinical use.

7. Conclusions

This study demonstrated that active compression–decompression CPR can be achieved with the use of our simple and inexpensive adhesive glove device. However, there was no significant added effect of ITD use on chest decompression when used along with an adhesive glove for ACD-CPR. Further animal survival studies are required to evaluate if ACD-CPR using the AGD results in improved neurologically intact survival.

Conflict of interest statement

The authors have no conflict of interest to declare.

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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.2013.05.019.

References