Experimental paper

Left ventricle chest compression improves ETCO$_2$, blood pressure, and cerebral blood velocity in a swine model of cardiac arrest and cardiopulmonary resuscitation

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Abstract

Introduction: During cardiopulmonary resuscitation (CPR), high quality chest compressions are critical to organ perfusion, especially the brain. Yet, the optimal location for chest compressions is unclear. It was hypothesized that compared with the standard chest compression (SCC) location, left ventricle chest compressions (LVCCs) would result in greater ETCO$_2$, blood pressure (BP), and cerebral blood velocity (CBV) during CPR in swine.

Methods: Female Landrace swine (N = 32; 35 ± 2 kg) underwent two mins of untreated asphyxiated cardiac arrest (CA). Thereafter, swine were treated with three 2-min cycles of either SCC or LVCC mechanical basic life support CPR (LUCAS 3). ETCO$_2$ (in-line sampling), BP (arterial catheter line), and CBV (transcranial Doppler) were measured during the pre-CA, untreated-CA, and CPR-treated phases.

Results: ETCO$_2$, BP, and CBV were similar between groups at pre- and during untreated-CA (P $>$ 0.188). During CPR, ETCO$_2$ (36 ± 6 versus 24 ± 10 mmHg, P < 0.001), mean arterial BP (MAP; 49 ± 9 versus 37 ± 9 mmHg, P = 0.002), and CBV (11 ± 5 versus 5 ± 2 cm/s, P < 0.001) were significantly greater in the LVCC versus SCC group. Moreover, a greater proportion of animals obtained targets for ETCO$_2$ (ETCO$_2$ $>$ 20 mmHg; 52 % (17/33) versus 100 % (32/32), P < 0.001) and diastolic BP (DBP $>$ 25 mmHg; 82 % (33/40) versus 97 % (48/49), P = 0.020) in the LVCC versus SCC group.

Conclusion: Indicators of cardiac output, BP, and cerebral perfusion during CPR were greatest in the LVCC group, suggesting the quality of chest compressions during BLS CPR may be improved by performing compressions over the left ventricle compared to the centre of the chest.

Keywords: Left Ventricle Chest Compressions, Cerebral Blood Flow, Chest Compressions, Basic Life Support, CPR, Cardiac Arrest

Introduction

Cardiac arrest (CA) occurs over 500,000 times annually in the United States. Reduced cerebral perfusion during CA and cardiopulmonary resuscitation (CPR) is implicated in poor survival rates (<10 %) and post-arrest neurological deficits. Improvements to CPR that increase cerebral blood flow may lead to profound life saving benefits.

Current CPR guidelines define the surface landmark of standard chest compression (SCC) as the centre of the chest on the lower half of the sternum. This SCC location overlies the upper third of the heart, which encompasses the aortic root, ascending aorta, left ventricular outflow tract, or atria in over 80 % of patients. Compressing these structures does not compress the greatest volume of the heart and may increase cardiac outflow resistance. left ventricle chest compression (LVCC) during CPR may offer hemodynamic benefits compared to SCC. LVCC is performed by identifying the location overlying the left ventricle on the anterior chest wall and delivering external chest compressions at that site. Specifically, LVCC targets a larger volume of the heart and avoids compression of outflow vessels, thereby increasing stroke volume.
and potentially decreasing outflow resistance. In swine models of ventricular fibrillation and traumatic CA, LVCC has been reported to improve systemic hemodynamics and return of spontaneous circulation (ROSC).19–22 Whether the benefits of LVCC extend to the cerebral circulation remains unknown. Using a swine model of asphyxiated CA and the purpose of this study was to compare standard indicators of hemodynamic status and cerebral blood flow between SCC and LVCC during basic life support (BLS; chest compressions and ventilations only) CPR. It was hypothesized that compared to SCC, LVCC would result in greater ETCO2 (established indicator of cardiac output23), blood pressure (BP), and cerebral blood velocity (CBV; validated indicator of cerebral blood flow24–27) during BLS CPR.

**Methods**

**Study design**

This prospective interventional trial was approved by the University of Saskatchewan Animal Research Ethics Board (#20200042). Briefly, animals underwent asphyxiated CA and received either SCC or LVCC BLS CPR while hemodynamics (ETCO2, BP and CBV) were monitored.

**Animal preparation**

Following acclimatization and a 12-hour overnight fast, female Landrace swine (N = 32; 35 ± 2 kg; Prairie Swine Center, CAN) were sedated with an intramuscular injection of 20–30 mg/kg of ketamine (Vetoquinol, CAN). Swine were intubated and mechanically ventilated (10 ml/kg tidal volume; Excel 210 SE, 7900 SmartVent, Datex Ohmeda, FIN). Anesthesia depth was maintained with 1–3% isoflurane (Baxter, CAN). Body temperature was maintained at ~ 38°C (rectal thermometer). An intravenous (IV) catheter was inserted into an auricular vein for fluid maintenance (10 ml/kg/hr normal saline) and medication delivery.

Animals were placed supine in a V-shaped holder with the limbs and head secured to prevent displacement during CPR. A gas analyzer (ADI, USA) sampling line was connected to the ventilatory circuit and the ventilation rate was adjusted to maintain an ETCO2 range of 35–40 mmHg. Continuous heart rate and cardiac activity were monitored by electrocardiogram (ECG) using a standard 3-lead limb system (ADI). Beat-by-beat BP was measured from a femoral artery catheter (BD, USA) connected to a pressure transducer (DELTRAN II, Utah Medical Products, USA). To prevent clotting, an initial IV 300 IU/kg bolus of heparin was administered with an additional 75 IU/kg bolus administered every hour.19,21

Following instrumentation, transthoracic echocardiography (GE Vivid I, GE 3Sc-Rs Probe; CCE Medical, CAN) was used to locate and mark the surface site for either SCC (sternal midline at the level of the aortic root; Fig. 1A and 1B) or LVCC (intersection of the parasternal long and short axes of the left ventricle; Fig. 1C and 1D). Although the level of the aortic root is not indicative of all clinical SCC locations, this normalized location was selected as the experimental control as it encapsulates the majority of clinical

Fig. 1 – Transthoracic echocardiography 2-dimensional images of the aortic valve (A & B) to guide surface markings for the standard chest compression (SCC) location. Left ventricular long (C) and short (D) axes used to guide surface markings for the left ventricle chest compression (LVCC) location. The white lines in C and D indicate the corresponding axes.
A transcranial Doppler (TCD, Multigon Industrial Inc., USA) signal was then obtained to assess indices of cerebral blood flow. Briefly, a pencil probe (2 MHz) was positioned superficially over the right transorbital window to obtain middle cerebral artery CBV. Time-aligned indices of systemic hemodynamic status (ETCO₂, heart rate, BP) and CBV were recorded using a digital-analog converter (Powerlab; ADI) interfaced with a data collection software program (LabChart 8, ADI) during baseline, CA and CPR.

**Experimental protocol**

Following a 60-min stabilization period, a mechanical chest compression device (LUCAS 3, Stryker, SWE) was positioned and secured over the previously assigned SCC or LVCC surface landmark. Once fully instrumented, pre-CA baseline data were collected for 2 mins. Asphyxiated CA was induced by cessation of mechanical ventilation and occluding the endotracheal tube. Animals were given a 5–20 mg/kg bolus of propofol to prevent gasping (Baxter). CA was confirmed by loss of cardiac motion (echocardiography) and total absence of a carotid Doppler pulse (long axis view; GE Vivid I, GE 9L Probe; CCE Medical). Following CA confirmation, 2 mins of untreated CA was allotted prior to beginning BLS CPR.

During BLS CPR, mechanical ventilation was resumed and kept constant on 100 % oxygen. Consistent with the American Heart Association (AHA) 2020 CPR guidelines, the chest compression rate was 100/min, compression depth was 5 cm, and full thoracic recoil ensured. BLS CPR was performed for three 2-min rounds separated by 10-sec circulation checks. Hemodynamic data during BLS CPR were grouped and averaged for each 2-min round. If ROSC was achieved during a circulation check, the protocol was terminated. Successful ROSC was defined as the presence of a carotid Doppler pulse and sustained regular electrical and mechanical cardiac activity producing a systolic BP (SBP) ≥ 60 mmHg without
intervention for at least 1-min. If an animal did not achieve ROSC for a full min then the protocol was resumed at the point at which it was paused. Animals that achieved ROSC were kept on mechanical ventilation (1–3 % isoflurane) without additional intervention for up to 10 mins prior to euthanization. Following euthanasia (exsanguination), post-mortem dissection was conducted to assess for injuries.

**Statistics**

Owing to the blatant difference in chest compression locations, experimenters could not be blinded during CPR administration. However, data analysis was completed by a researcher blinded to experimental condition. Data during the entire 2 mins of baseline, untreated CA and each round of BLS CPR were binned independently and averaged for each period. Erroverse waves that exceeded 1 standard deviation from the mean were removed. Statistical analyses were completed using SigmaPlot 14.0 (SysStat, USA). Sample size was estimated using ETCO2 data from previous SCC versus LVCC swine work. This prior report demonstrated an 11 mmHg difference (standard deviation approximating 50 % of mean difference i.e., 8 mmHg). Employing a modified version of the ARRIVE guidelines’ simple randomization procedure, the first animal was randomized (SCC or LVCC; random.org) and thereafter animals were rotated systematically (i.e., SCC, LVCC, repeat) to achieve the necessary sample size. Potential nuisance variables were balanced between groups with strict standardized animal care, housing, feeding interaction and handling procedures, using the same investigators for animal instrumentation and experimentation, and using animals of the same sex and similar mass.

Indices of hemodynamic status were compared using a group time (baseline, CA, BLS CPR round 1, 2, and 3) mixed model ANOVA. A priori between group pairwise comparisons at baseline, CA, and round 1, round 2 and round 3 of BLS CPR were completed using a post hoc Tukey’s test. Within group differences were not explored. Grouped data are presented as mean with individual data points. Cohen’s d effect size analysis (small effect $\geq 0.20$; medium effect $\geq 0.50$; large effect $\geq 0.80$) for end-tidal carbon dioxide (ETCO2), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MAP), pulse pressure (PP), systolic cerebral blood velocity (SCBV), diastolic cerebral blood velocity (DCBV), mean cerebral blood velocity (MCBV) in each round of basic life support (BLS) cardiopulmonary resuscitation (CPR).

### Table 1 – Effect of LVCC on systemic and cerebral hemodynamics.

| Measure       | Round 1 d | Round 2 d | Round 3 d |
|---------------|-----------|-----------|-----------|
| ETCO2 (mmHg)  | $+0.3$    | $+0.4$    | $+0.5$    |
| SBP (mmHg)    | $+0.3$    | $+0.3$    | $+0.3$    |
| DBP (mmHg)    | $+0.2$    | $+0.2$    | $+0.2$    |
| MAP (mmHg)    | $+0.3$    | $+0.3$    | $+0.3$    |
| PP (mmHg)     | $+0.6$    | $+0.5$    | $+0.4$    |
| SCBV (cm/s)   | $+0.6$    | $+0.6$    | $+0.7$    |
| DCBV (cm/s)   | $+0.6$    | $+0.7$    | $+0.8$    |
| MCBV (cm/s)   | $+0.7$    | $+0.9$    | $+0.8$    |

Cohen’s d effect size (d: + indicates a positive effect; small effect $\geq 0.20$; medium effect $\geq 0.50$; large effect $\geq 0.80$) for end-tidal carbon dioxide (ETCO2), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MAP), pulse pressure (PP), systolic cerebral blood velocity (SCBV), diastolic cerebral blood velocity (DCBV), mean cerebral blood velocity (MCBV) in each round of basic life support (BLS) cardiopulmonary resuscitation (CPR).
baseline and during untreated CA (P ≥ 0.506), but greater in the LVCC versus SCC group in all three rounds of CPR (P < 0.001; Fig. 2A). There was no group x time interaction effect for diastolic CBV values (P = 0.309; Fig. 2B). There was a significant group x time interaction effect (P < 0.001) for mean CBV values. Pairwise comparisons reveal groups were similar at baseline and during untreated CA (P ≥ 0.512), but greater in the LVCC versus SCC group in all three rounds of CPR (P ≤ 0.006; Fig. 2C). The effect size analysis reveals LVCC had a medium-large, positive effect on systolic CBV, diastolic CBV and mean CBV in the LVCC versus SCC group in all three rounds of CPR (Table 1).

Return of spontaneous circulation and injuries

Swine that did not achieve ROSC presented with asystole (n = 29) during all three circulation checks. Swine that did achieve ROSC (n = 3) presented with asystole during untreated CA and/or circulation checks prior to regaining independent circulation. The occurrence of ROSC was not dependent on chest compression location (Fig. 2D; Table 2). Similarly, the occurrence of rib fractures, sternal fractures, liver lacerations, spleen lacerations, or hemothoraces were not dependent on chest compression location (Table 2).

End-tidal carbon dioxide

There was a significant group x time interaction effect (P < 0.001) for ETCO2 values. Pairwise comparisons reveal groups were similar at baseline and during untreated CA (P ≥ 0.226), but greater in the LVCC versus SCC group in all three rounds of CPR (P ≤ 0.003; Fig. 3A). The effect size analysis reveals LVCC had a small-medium, positive effect on ETCO2 throughout CPR (Table 1). Moreover, a greater percent of animals achieved the preclinical target for ETCO2 in the LVCC versus SCC group in each round of CPR (P ≤ 0.038; Fig. 3B) as well as across all rounds of CPR (P < 0.001; Fig. 3C).

Fig. 3 – Average end-tidal carbon dioxide (ETCO2; Panel A) values for standard chest compression (SCC; white fill bars) and left ventricle chest compression (LVCC; black fill bars) groups during baseline, cardiac arrest (CA), basic life support (BLS) cardiopulmonary resuscitation (CPR) round 1 (SCC n = 11, LVCC n = 12), CPR round 2 (SCC n = 11, LVCC n = 11), and CPR round 3 (SCC n = 11, LVCC n = 9). Data were analyzed using a mixed model ANOVA. A priori between group comparisons at baseline, CA, and round 1, round 2 and round 3 of BLS CPR were completed using a post hoc Tukey’s test. Significantly greater than SCC ***(P < 0.01), ***(P < 0.001). Bars represent mean. Individual data points reflect individual animals. Some individual datapoints may overlap, thus obscuring visualization. The sample sizes listed herein are accurate. Dashed black line represents preclinical target for ETCO2 (≥20 mmHg). Percent of animals that achieved the preclinical target for ETCO2 in the SCC and LVCC groups during baseline, CA, and round 1, round 2 and round 3 of CPR (B), and across all three CPR rounds cumulatively (C). Panel B and C data were analyzed using Fisher’s exact tests. Significant dependencies between chest compression location and achieving the preclinical target by round *(P < 0.05), **(P < 0.01), *** (P < 0.001).
Fig. 4 - Average systolic blood pressure (SBP; Panel A), diastolic blood pressure (DBP; Panel B), mean arterial blood pressure (MAP; Panel C) and pulse pressure (PP; Panel D) values for standard chest compression (SCC; white fill bars) and left ventricle chest compression (LVCC; black fill bars) groups during baseline, cardiac arrest (CA), basic life support (BLS) cardiopulmonary resuscitation (CPR) round 1 (SCC n = 14, LVCC n = 18), CPR round 2 (SCC n = 13, LVCC n = 16), and CPR round 3 (SCC n = 13, LVCC n = 15). Data were analyzed using a mixed model ANOVA. *A priori* between group comparisons at baseline, CA, and round 1, round 2 and round 3 of BLS CPR were completed using a post hoc Tukey's test. Significantly greater than SCC *(P < 0.05), **(P < 0.01), ****(P < 0.001). Bars represent mean. Individual data points reflect individual animals. Some individual datapoints may overlap, thus obscuring visualization. The sample sizes listed herein are accurate. Dashed black line represent preclinical target for DBP (<25 mmHg). Percent of animals that achieved the preclinical target for DBP in the SCC and LVCC groups during baseline, CA, and round 1, round 2 and round 3 of BLS CPR (Panel E), and across all three CPR rounds cumulatively (Panel F). Panel E and F data were analyzed using Fisher's exact tests. Significant dependency between chest compression location and achieving the preclinical target *(P < 0.05).
Blood pressure

There was a significant group x time interaction effect (P < 0.001) for SBP values. Pairwise comparisons reveal groups were similar at baseline and during untreated CA (P ≥ 0.277), but greater in the LVCC versus SCC group in all three rounds of CPR (P ≤ 0.003; Fig. 4A). There was a main effect of group (P = 0.049) and time (P < 0.001), but the group x time interaction effect for DBP values was not significant (P = 0.078). Given there were two main effects, and in line with the stated a priori comparisons, between group comparisons were made at each time point. Pairwise comparisons reveal DBP was similar at baseline and during untreated CA (P ≥ 0.188), but greater in the LVCC versus SCC group in all three rounds of CPR (P ≤ 0.040; Fig. 4B). There was a significant group x time interaction effect (P = 0.002) for MAP values. Pairwise comparisons reveal groups were similar at baseline and during untreated CA (P ≥ 0.190), but greater in the LVCC versus SCC group in all three rounds of CPR (P ≤ 0.007; Fig. 4C). There was a significant group x time interaction effect (P < 0.001) for pulse pressure values. Pairwise comparisons reveal groups were similar at baseline and during untreated CA (P ≥ 0.766), but greater in the LVCC versus SCC group in all three rounds of CPR (P ≤ 0.002; Fig. 4D). The effect size analysis reveals that LVCC had a small, positive effect on SBP, DBP and MAP, and a medium, positive effect on pulse pressure in all three rounds of CPR (Table 1). Achieving the preclinical target for DBP was not dependent on chest compression location during each BLS CPR round independently (P < 0.001), but greater in the LVCC versus SCC group in all three rounds of CPR (P < 0.003; Fig. 4B). There was a significant group x time interaction effect (P = 0.002) for MAP values. Pairwise comparisons reveal groups were similar at baseline and during untreated CA (P ≥ 0.017; Fig. 4E). However, a greater percentage of animals achieved the preclinical target for DBP in the LVCC versus SCC group across all rounds of CPR (P = 0.020; Fig. 4F).

Discussion

The current study revealed that compared with the SCC location, BLS CPR performed in the LVCC location resulted in significantly higher values for indices of systemic and cerebral perfusion. Further, the total number of rounds in which AHA targets for ETCO2 and DBP were achieved was greater during LVCC versus SCC. Concerning potential risks of LVCC, no group differences in prevalence of CPR-related injury were observed. Overall, the data provide evidence that, compared to SCC, LVCC results in enhanced hemodynamic status and cerebral blood flow in a swine model of BLS CPR.

In the present study, both systolic CBV and mean CBV were greater during LVCC compared to SCC. This was likely the result of increased stroke volume and potential redistribution of blood flow to the brain during compression systole. Both ETCO2 (accepted surrogate for cardiac output in CPR18–19) and pulse pressure (surrogate for stroke volume20) were greater in the LVCC versus SCC condition, suggesting stroke volume increased with LVCC. Potentially, LVCC does not cause the same narrowing of the left ventricular outflow tract as SCC (up to 83 %),14,17 facilitating a larger stroke volume per compression. The present study is in agreement with clinical data where transition from SCC to LVCC following 30 mins of conventional CPR produced improvements in ETCO2.18 Mechanistically, with the descending aorta being located posterior to the heart, indirect aortic compression at the level of the left ventricle may cause an advantageous aortic ballooning effect that directs an increased portion of stroke volume cranially.17 Indeed, previous swine work reported improved ETCO2 and non-significant increases in cerebral oxygenation with LVCC versus SCC (estimated effect size: ~0.3).19 In view of the present results, LVCC potentially promotes superior systemic hemodynamics and cerebral perfusion compared with SCC.

Given the increased cardiac output, it follows that there would be a concomitant increase in BP. Improvements in BP with LVCC versus SCC were in concordance with previous swine work using a model of ventricular fibrillation arrest.19 However, despite increased BP with LVCC, no differences in diastolic CBV were observed. This may be explained by impaired autoregulation in the post-arrest hypoxic brain13 or BP during CPR decompression (compression diastole) being below the autoregulatory threshold of adequate cerebral perfusion,44–46 either resulting in minimal or absent observed diastolic flow. Given increases in CBV yield from the compression phase, these data highlight the importance of high-quality compressions in minimizing cerebral perfusion deficits.

In the present study, the AHA target for ETCO2 (≥20 mmHg) was reached in merely 52 % of SCC versus 100 % of LVCC rounds. The AHA target for DBP (≥25 mmHg) was reached in 82 % of SCC versus 98 % of LVCC rounds throughout BLS CPR. AHA targets were met more routinely with LVCC, raising the possibility that LVCC improves the effectiveness of CPR in real-world settings.

Table 2 – ROSC and Injury data between groups.

| Event                  | SCC % [occurrence/n] | LVCC % [occurrence/n] | P       |
|------------------------|----------------------|-----------------------|---------|
| ROSC                   | 0 % [0/14]           | 17 % [3/18]           | 0.238   |
| Rib Fractures          | 100 % [14/14]        | 100 % [18/18]         | 1.000   |
| Sternal Fractures      | 0 % [0/14]           | 0 % [0/18]            | 1.000   |
| Liver Lacerations      | 0 % [0/14]           | 0 % [0/18]            | 1.000   |
| Spleen Lacerations     | 0 % [0/14]           | 0 % [0/18]            | 1.000   |
| Hemothoraces           | 21 % [3/14]          | 28 % [5/18]           | 1.000   |

Percent, proportion, and Fisher’s exact values for return of spontaneous circulation (ROSC), rib fractures, sternal fractures, liver lacerations, spleen lacerations, and hemothoraces between standard chest compression (SCC) and left ventricle chest compression (LVCC) groups following basic life support (BLS) cardiopulmonary resuscitation (CPR).
In the current study, although more swine achieved ROSC in the LVCC (n = 3) versus SCC group (n = 0), the prevalence of ROSC was not different between groups. However, the current BLS-only study was likely underpowered to assess differences in this secondary outcome. Anderson and colleagues reported ROSC in 69% of LVCC versus 0% of SCC swine, and all during advanced life support CPR. Ergo, LVCC may improve cerebral blood flow and potentiate the prevalence of ROSC when epinephrine, defibrillation, or both are administered. Given the risk for CPR-related injury was similar among groups, the results herein provide a rationale for future investigations to examine the efficacy of LVCC for increasing the prevalence of ROSC in out-of-hospital CA. Indeed, our group published work suggesting that localizing the heart in CA is possible using a facile ultrasound device with artificial intelligence. Optimized LVCC may be achievable in seconds with a novel and simple semi-automated device in the out-of-hospital setting without the current need for expertise and impractical equipment.

Limitations

Data from swine models of resuscitation must be interpreted cautiously and confirmed in the clinical setting. Compression of the heart and underlying structures may differ between species. Underlying structures during CPR were not tracked and may have shifted. This was not a randomized control trial, and after the first animal was randomized, subsequent animals were assigned using a systematic rotation, alteration protocol. This approach was employed to obtain a sufficient number of complete data sets in both groups, using the fewest number of animals. An advantage of TCD is that it can be used in both preclinical and clinical settings. However, TCD quantifies blood velocity and not volumetric flow. Furthermore, ETCO$_2$ is a surrogate for cardiac output. Thus, more invasive preclinical work and human trials are requisite to confirm these findings.

Conclusion

The present study provides novel insight into the benefits of LVCC versus SCC on hemodynamic performance, demonstrating that LVCC improves systemic and cerebral hemodynamic status in a swine model of asphyxiated CA and CPR. Given the observed benefits of LVCC and vitality of cerebral preservation to ROSC and survival, further studies are warranted to determine the translational relevance of these discoveries.

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