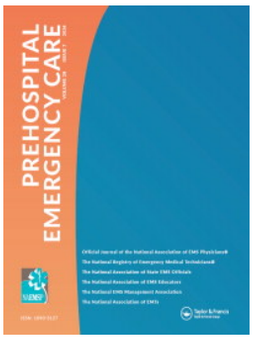

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Proportional Versus Fixed Chest Compression Depth for Guideline-Compliant Resuscitation of Infant Asphyxial Cardiac Arrest

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ABSTRACT

Objectives: Current guidelines for parameters of the delivery of chest compressions (CC) for infants and children are largely consensus based. Of the two recommended depth targets – 1.5 inches and 1/3 anterior-posterior chest diameter (APD) – it is unclear whether these have equal potential for injury. In previous experiments, our group showed in an animal model of pediatric asphyxial out-of-hospital cardiac arrest (OHCA; modeling ~ 7 year-old children) that 1/3 APD resulted in significantly deeper CC and a higher likelihood of life-threatening injury. We sought to examine and compare injury characteristics of CC delivered at 1.5 inches or 1/3 APD in an infant model of asphyxial OHCA.

Methods: Swine were sedated, anesthetized, paralyzed, intubated through direct laryngoscopy, and then mechanically ventilated (10 ml/kg, FiO2:21%). APD was measured and confirmed by two investigators *via* a sliding T-square at the xiphoid. After instrumentation for vital signs monitoring, and while still anesthetized, the endotracheal tube was manually occluded to induce asphyxia, and occlusion was maintained for 9 min. Animals were then randomized to receive CC with a depth of 1.5 inches (Group 1) or 1/3 APD (Group 2), both with a rate of 100 per minute. Advanced life support drugs were administered at 13 min, and defibrillation at 14 min. Resuscitation continued until return of spontaneous circulation (ROSC) or 20 min of failed resuscitation. Survivors were sacrificed with KCl after 20 min of observation. Veterinary staff conducted necropsy to assay lung injury, rib fracture, hemothorax, airway bleeding, great vessel dissection, and heart/liver/spleen contusion. Injury characteristics were summarized and compared *via* Chi-Squared test or Mann-Whitney U-test using an alpha = 0.05.

Results: A total of 36 animals were included for analysis (Group 1: 18; Group 2: 18). Mean (SD) APD overall was 5.58 (0.23) inches, yielding a mean 1/3 APD depth of 1.86 inches. APD did not differ between groups. ROSC rates did not differ between groups. No injury characteristics differed significantly between groups.

Conclusions: In an swine model of infant asphyxial OHCA and resuscitation considering 1/3 APD or 1.5 inches, neither CC depth strategy was associated with increased injury.

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Introduction

Guidelines for the treatment of out-of-hospital cardiac (OHCA) arrest among infant and pediatric patients suffer from a dearth of high quality supporting evidence. These patients are distinct from adults, owing to different anatomical and physiologic features that coincide with the broad phases of human development and which bear implications for hemodynamics, outcomes, and injury. Observational clinical studies have provided some evidence for formulating guidance but also are limited by low incidence of cardiac arrest in the young (1, 2). Among infants or neonates, the evidence is especially sparse. While laboratory studies are at least one step detached from clinical randomized controlled trials, they offer a tightly controlled evaluation of basic resuscitation processes. Process parameters thought to influence outcomes include chest compression rate, depth and pausing durations, although in pediatric OHCA large scale qualitative evidence is limited (3). The current American Heart Association (AHA) and European Resuscitation Council (ERC) guidelines recommend a target rate of at least 100 per minute, minimization of pausing time, and a target depth of 1/3 the anterior-posterior sternal thoracic diameter (APD) or 1.5 inches (4, 5). Depth targets have a moderate class of recommendation coupled with a high potential for significant anatomic injury in this population, leading to interest in establishing an optimum.

In a previous study, our group compared proportional chest compression depth to fixed depth in a juvenile swine model of pediatric asphyxial cardiac arrest with an anatomic homology to a 7-year old child (6). We found that a proportional depth approach likely overestimated the appropriate target depth, resulting in significant likelihood of life-threatening injury and no apparent hemodynamic gain. In the present study we extended this investigation to a porcine

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model more closely homologous to a human 12-month old infant. We hypothesized that we would observe the same associations in younger, smaller animals, i.e., that the proportional depth would produce more serious injuries without a hemodynamic advantage.

Methods

This study was reviewed and approved by the University of Pittsburgh Institutional Animal Care and Use Committee (PRO#18052785) and was conducted in accordance with the *Guide for Care and Use of Laboratory Animals*. Our laboratory is accredited by the Association for the Assessment and Accreditation of Laboratory Animal Care International (AAALAC).

Anesthesia and Surgical Instrumentation

We used 4-week old, approximately 11 kg swine to model cardiac arrest and resuscitation in infants. Each piglet was sedated with intramuscular ketamine (10 mg/kg) and xylazine (4 mg/kg). The anterior-posterior diameter was then measured with a sliding T-square to the nearest $1/8^{th}$ of an inch. We intubated each animal *via* direct laryngoscopy with a 3-0 cuffed endotracheal tube, confirming placement by continuous capnography monitored *via* an inline sensor. Mechanical ventilation was initiated with a tidal volume of 10 mL/kg and a rate sufficient to maintain eucapnia. We then delivered a bolus (50ug/kg) of fentanyl for induction of anesthesia, which was then maintained *via* syringe pump at a rate of 30ug/kg/hr for the duration of instrumentation and the experimental insult. Vecuronium was then administered (5 mg) by bolus and readministered as needed to maintain neuromuscular blockade. Next, we performed a femoral cutdown to advance invasive pressure sensors (Model 350S, Millar, Inc) into the aortic arch and proximal to the right atrium. ECG leads were placed in a Lead I and Lead II configuration. An perivascular flow probe (TransSonic, Inc) was installed on the right-side common carotid artery *via* cutdown. An arterial blood chemistry panel (CG8+, I-Stat, Abbott, Inc) was run at the end of instrumentation, and we observed the stable instrumented animal for at least 10 min until beginning the experimental procedure.

Experimental Procedure

The experimental timeline is shown in Figure 1. We administered a paralytic bolus and waited approximately 3 min for it to circulate to ensure a lack of spontaneous breathing. We then disconnected the ventilator circuit and manually occluded the endotracheal tube for 9 min. The animal was left untreated for this period. After 8 min and 45 s of this period had elapsed, if the animal was not already in ventricular fibrillation, we induced VF with a 3-s transthoracic shock. At the 9-min mark we initiated continuous chest compressions, reconnected the ventilator circuit, and resumed ventilation at baseline settings. After 4 min of chest compressions we administered epinephrine (0.1 mg/kg, i.e., high dose) and sodium bicarbonate (1mEq/kg). After another minute we attempted defibrillation, if indicated by the ECG rhythm. Given a failed shock, we continued chest compressions and attempted additional shocks every 2 min as needed, with epinephrine administered every 3 min (0.015 mg/kg, i.e., standard dose for a 70 kg human). Resuscitation was terminated if return of spontaneous circulation (ROSC) was not achieved 20 min after the start of chest compressions. Animals with ROSC were survived for 1 h, and while still anesthetized were euthanized with a rapid bolus injection of 40 mEq of potassium chloride.

Chest Compression Depth Groups

We randomized each animal to either 1.5 inch depth or 1/3 APD chest compression depth delivered by a custom servo-driven mechanical chest compression device. Chest compressions were delivered with uniform rate (100/minute), duty cycle (50%), and depth for the duration of each experiment.

Postmortem Examination

Immediately after the experiment, the animal was transferred to the necropsy suite for independent assessment by a research veterinarian and veterinary technician who were unaware of which group the animals were treated with. Animals were assessed for presence and number of rib fractures, as well as presence of hemothorax, liver/spleen contusion, myocardial contusion, and aortic or vena cava transection. The veterinary team also assessed the lungs using a scoring system we have described elsewhere (6). In short, each lobe was assigned a visually assessed score of 0 (no injury), 1 (less than 50% injured), and 2 (at least 50% injured). Thus, this score can range from 0 to 10.

Analysis

Baseline case characteristics were summarized and reported as means (SD) or frequency (%) as appropriate. Differential hemodynamic response to asphyxia was examined and characterized by grouping animals into 3 categories based on their mean arterial pressure (MAP) response after occlusion of the airway. In a previous series we noted that animals follow a bimodal trajectory in response to asphyxia, initially crashing, followed by a vigorous compensatory response, followed in turn by a permanent crash. In this series, hypo-compensators were defined as those that did not return to baseline during the 9-min asphyxia period; normo-compensators returned to at least 50% of baseline but did not exceed it; and hyper-compensators exceeded their baseline.

We summarized continuous physiologic measures, including coronary perfusion pressure (CPP), MAP, end tidal cardon dioxide ($EtCO_2$), and carotid blood flow (CBF), for each 30-second window of CPR in the period up to the first shock. CPP was calculated as the average of the difference between the aortic and right atrial pressure during the end-diastolic phase of the chest compression cycle for each compression. We used Generalized Estimating Equations to

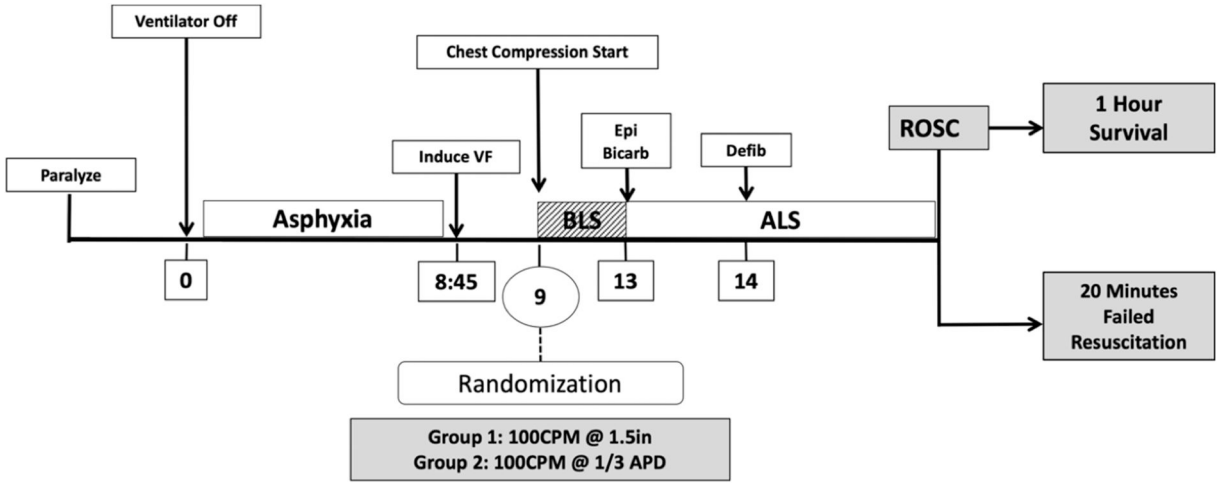

Figure 1. Experimental timeline.
The complete experimental timeline, including experimental groups and associated treatments is shown on a timeline starting at administration of the arrest induction cocktail. Abbreviations: ALS – Advanced Life Support; BLS – Basic Life Support; CPM – Compressions Per Minute; MAP – Mean Arterial Pressure; PEA – Pulseless Electrical Activity; ROSC – Return of Spontaneous Circulation; VF – Ventricular Fibrillation

compare each measure by chest compression group across time.

Injury characteristics were summarized as median (IQR) or frequency (%). Lung injury scores from both inspectors were compared, and if the reviewer's disagreed on score component, the veterinarian's score was overriding. Generally, proportions were compared with chi-squared test, means were compared with Student's t-test, and medians were compared with the Wilcoxon Rank Sum test. All statistical analyses were performed in Stata 15 (Stata Corp. College Station, TX) with a threshold alpha level of 0.05.

Results

A total of 36 animals were enrolled in the study. Table 1 summarizes baseline characteristics by group. The groups did not differ on any baseline characteristics, including weight, APD diameter, blood chemistry and physiologic values. Chest compression depth differed on average by 0.38 inches between the groups, or approximately 7% of chest typical APD among all animals collectively. In Figure 2 the distribution of asphyxial compensation phenotypes prior to resuscitation can be seen in aggregate. Half of all animals were hypo-compensators. Compensator category did not differ between chest compression groups ($p = 0.103$).

Key resuscitation outcomes, as well as injury assessed at necropsy, are reported in Table 2. ROSC rate among all animals was 77.7%, and neither ROSC rates nor number of rescue shocks differed between groups. Median (IQR) total CPR time was 6 (5–8) minutes, and did not differ between groups ($p = 0.88$), but no animals achieved ROSC after longer than 9 min of CPR and the majority of ROSC events occurred at 5 min (57.1%). While all animals demonstrated some level of myocardial contusion, there were no statistically significant differences in any of the injury metrics between the chest compression groups. Hemothorax was only present in the 1/3 APD group (2/16, 11%).

Physiologic time-series are summarized in Figure 3. All measures except CBF, which was unusually rich in mechanical artifact, increased significantly over time and reflected late administration of drugs between the 13 and 14 min mark of the experimental timeline (minutes 4 and 5 of CPR). No measures differed between groups. End tidal carbon dioxide also reflected early post-asphyxia hypercapnia at the start of CPR.

Table 1. Baseline characteristics.

	Group		
	1.5 inch	1/3 APD	p-value
N	18 (50.0%)	18 (50.0%)	
Sex			
Female	9 (50.0%)	8 (44.4%)	0.738
Male	9 (50.0%)	10 (55.6%)	
Weight, kg	10.45 (0.74)	10.78 (0.96)	0.251
APD, in	5.54 (0.25)	5.63 (0.20)	0.210
Compression Depth, in	1.50 (0.00)	1.88 (0.07)	<0.001
HR, BPM	89.07 (17.11)	92.21 (17.01)	0.584
MAP, mmHg	76.14 (9.28)	79.39 (14.70)	0.433
EtCO2, mmHg	41.45 (2.12)	41.47 (1.43)	0.975
CBF, mL/Min	53.52 (19.21)	54.95 (22.99)	0.841
pH	7.30 (0.71)	7.43 (0.09)	0.437
pCO2, mmHg	41.51 (3.16)	44.74 (13.42)	0.340
pO2, mmHg	78.18 (9.78)	74.22 (15.82)	0.384
BEecf, mmol/L	7.35 (2.47)	5.11 (3.22)	0.028
HCO3, mmol/L	30.72 (1.88)	29.41 (2.59)	0.097
TCO2, mmol/L	31.88 (1.93)	33.44 (17.91)	0.723
sO2, %	96.24 (1.39)	90.76 (19.83)	0.265
Na, mmol/L	142.12 (2.57)	142.78 (1.59)	0.365
K, mmol/L	3.29 (0.27)	3.43 (0.49)	0.313
iCa, mmol/L	1.46 (0.12)	1.49 (0.14)	0.563
Glucose, mmol/L	82.35 (20.41)	84.72 (25.49)	0.764
Hct, %	23.71 (3.98)	24.61 (4.33)	0.525
Hb, g/dL	12.21 (17.01)	8.36 (1.48)	0.346

Animal characteristics at baseline are shown stratified by group with associated group comparison p-value parameters. All characteristics unless noted were determined to be normally distributed. Continuous variables represented as mean (SD) and were compared *via* t-test. Dichotomous variables represented as frequency (percentage) and compared *via* Chi Squared test.
Abbreviations: APD – Anterior-Posterior Diameter; CBF – Carotid Blood Flow; CPP – Coronary Perfusion Pressure; EtCO2 – End Tidal Carbon Dioxide; Hb – Hemoglobin; Hct – Hematocrit; HR – Heart Rate; MAP – Mean Arterial Pressure. Total Sample = 36.

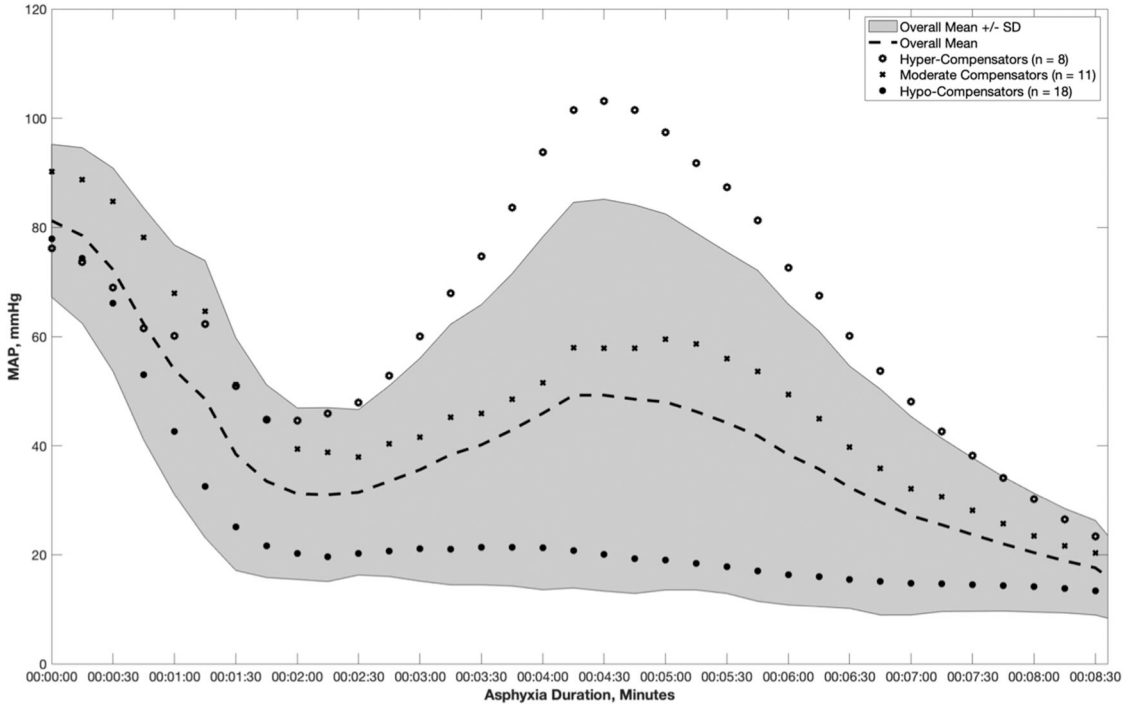

Figure 2. Hemodynamic crash period.
Mean arterial pressure is shown with trajectory groupings based on compensatory response. Abbreviations: MAP – Mean Arterial Pressure.

Table 2. Injury and outcomes.

	Group		
	1.5 inch	1/3 APD	p-value
n	18 (50.0%)	18 (50.0%)	
ROSC			
No	5 (27.8%)	3 (16.7%)	0.423
Yes	13 (72.2%)	15 (83.3%)	
Rescue Shock Count	1 1 - 3	1 1 - 2	0.176
Any Rib Fracture			
No	3 (16.7%)	1 (5.6%)	0.289
Yes	15 (83.3%)	17 (94.4%)	
Fib Fracture Count	3 0 - 4	4 0 - 5	0.532
LIS	5 4 - 5	5 3 - 6	0.651
Hemothorax			
No	18 (100.0%)	16 (88.9%)	0.146
Yes	0 (0.0%)	2 (11.1%)	
Aortic Transection			
No	18 (100.0%)	18 (100.0%)	.
Vena Caval Transection			
No	18 (100.0%)	18 (100.0%)	.
Myocardial Contusion			
Yes	18 (100.0%)	18 (100.0%)	.
Liver / Spleen Contusion			
No	16 (88.9%)	15 (83.3%)	0.630
Yes	2 (11.1%)	3 (16.7%)	

Injuries sustained (including aggregate lung injury score) during resuscitation are summarized, along with resuscitation outcome variables, and stratified by group with associated group comparison p-value parameters.
Abbreviations: LIS – Lung Injury Score; ROSC – Return of Spontaneous Circulation. Total Sample = 36.

Discussion

The present study is part of a preclinical series exploring the impact of chest compression parameter targets throughout the pediatric phase of life. In a previous study, our group demonstrated a strong potential for injury when 1/3 APD was used as a chest compression target, compared to 1.5 inches, in a swine model of pediatric asphyxial cardiac arrest in 25 kg pigs modeling a 7-year old child (6). The present study considered the same depth target comparison in a similar model of infant asphyxial cardiac arrest, utilizing pigs of roughly half their size. In contrast, this time we did not see statistically significant separation between chest compression depth target groups. Clues as to why the two studies differed begin with the observation that the APD of the infant model animals were such that the two groups in the present study had very similar absolute depth targets, differing by less than half an inch. This compares to the previous pediatric model in which depth targets differed by more than 1 inch on average. In our observations, APD in swine does not scale linearly with weight in the infant-to-pediatric age range; in our pediatric model, the APD-to-weight ratio was 0.29, compared to 0.53 in the infant model. It is then not unreasonable to imagine that APD-scaled compression depth would have differential impact on the anatomy between these life stages. Several studies utilizing pediatric chest tomography have estimated ranges of safe compressible distance in infants and older children and generally acknowledge the evolving compressible depth of the pediatric thorax with age (7–11). The same studies vary on inferring to the contribution of fixed or proportional chest compression depth targets to cause injury, possibly reflecting the variability in human chest composition around the world.

The hemodynamic results of both studies suggest that depth target group did not influence hemodynamics, although likely for different reasons. In the present series, the degree of absolute difference in depth between groups was likely physiologically and kinematically negligible, at least on average. Conversely, in our previous series the level of injury sustained by the 1/3APD group may have simply

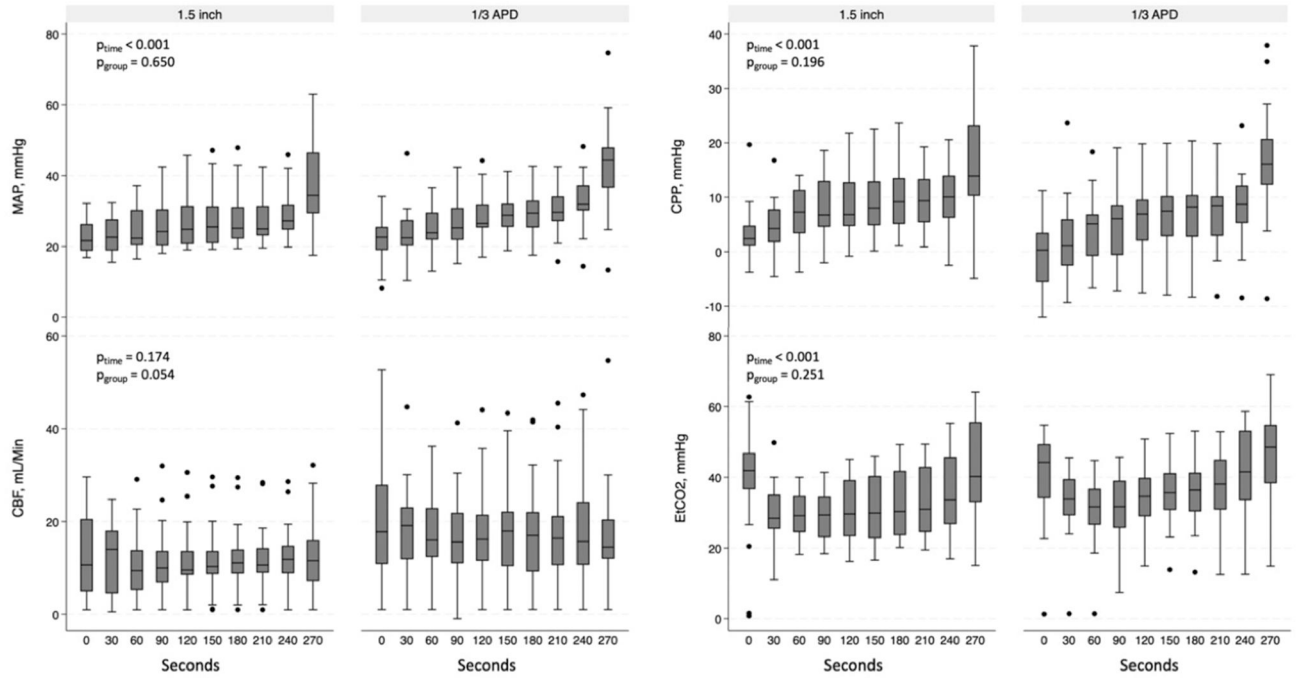

Figure 3. Hemodynamics during CPR until first pulse check.
Box plots are shown during CPR until the first defibrillation / pulse check by treatment group for CPP, CBF, $EtCO_2$ and MAP. Abbreviations: APD – Anterior-Posterior Diameter; CBF – Carotid Blood Flow; CPP – Coronary Perfusion Pressure; EtCO2 – End Tidal Carbon Dioxide; MAP – Mean Arterial Pressure.

attenuated the mechanisms of forward blood flow during CPR, while the 1.5 inch group may not have been deep enough to fully maximize both venous return and cardiac output, when viewed through the most common theories of CPR-generated blood flow (12). Both groups in the present study responded to drug administration, as indicated by the changes in hemodynamic and capnography measures toward the end of the analytic period, which likely also contributed to relatively high rates of ROSC in both groups.

On both injury and hemodynamics, the clinical literature offers only a limited basis for comparison with our findings in infants, respectively owing to low incidence of infant OHCA and the limitations of invasive instrumentation of this population. A number of small studies have found that rib fracture is comparatively much lower among human infants than what we observed in our studies overall (13–16). While useful as a general surrogate for injury, these findings lack information about true chest compression depth delivered, which is required to make an accurate comparison. This is relevant owing to the observed variability in chest compression quality in both adult and pediatric patient populations (3, 17). And, while controlling depth delivered may be one mechanism of avoiding injury, it is noteworthy that CPR quality feedback devices predominantly designed for adults may also not function optimally for infants without adaptation (18).

Regarding the pre-resuscitation phase of the model, animals in the present series displayed three noteworthy hemodynamic trajectories during the crash phase leading to asphyxial arrest, an observation that did not present clearly in the pediatric model, which showed a singular response trajectory. This may be reflective of phase of life, or could be related to difference is pre-arrest protocol; animals in our previous pediatric series received a large additional bolus of fentanyl prior to asphyxia. Extrapolating to differential compensatory responses during asphyxia is difficult and may warrant further investigation.

There are a number of important next steps ahead toward further understanding our findings to date. CPR is a multi-dimensional process parameterized in terms of not only depth, but also rate, duty cycle, up- and down-stroke accelerations, pauses, and the permissible variability and interdependence of all of those. A more advanced understanding of how our findings change when accounting for variation across this complex parameter space may help more generally identify important constraints to optimizing safety and efficacy of CPR in this phase of life. Moreover, the relationship between the selection of CPR parameters and evolving physiology remains a very active field of development. Future work should investigate whether and how to adapt parameters within individual cases.

Our study has a number of limitations that hold bearing on interpretation of the results. Foremost, this was a basic science laboratory study using swine to model infants. A number of significant dimensions of the laboratory model affect the generalizability of our findings, including the different anatomical characteristics of the human infant and swine chests, the highly controlled conditions of apnea and ventricular fibrillation induction, and the timing, method and dosage of resuscitation drug delivery (i.e., high dose epinephrine). Our experiment also utilized a robotic chest compression device that is designed to provide precise rate and depth, but does specifically not replicate the nuance of human performed chest compressions, which infants are most likely to receive (as there are currently no commercially available infant chest compression devices). Moreover, our study only considered a relatively narrow range of the

entire resuscitation timeline, i.e., the first 5 min leading up to defibrillation. Despite this, we observed nearly uniform CPR duration exposure between groups. We would expect differences in injury rates if the groups differed in the amount of time necessary to achieve ROSC, however neither the former nor the latter came to pass. Lastly, our study was only blinded during the necropsy phase, not during the resuscitation phase. Even so, we believe the most important opportunity for introduction of unintentional bias (position and function of the chest compressor) was mitigated by the workflow we used with the robotic compressor.

Conclusions

In a swine model of infant asphyxial out-of-hospital cardiac arrest, animals receiving chest compressions delivered at 1/3 APD or 1.5 inches did not differ in extent of anatomical injury, hemodynamic response or ROSC. A relatively high ROSC rate among both groups suggests that either depth target is reasonable in this phase of life.

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Authorship Statement

JJM acquired grant funding to sponsor the research. DDS and JJM designed the experiment, oversaw its execution, and were responsible for overseeing all data analysis. DDS, JJM, ACK, and CG contributed to the execution of each experiment. JAG provided data quality control/review, analyzed data, developed figures and tables, and provided administrative support. DDS, JJM, ACK, CG, and JAG contributed to writing of the manuscript. All authors read, edited, and approved the final manuscript.

Declaration of Generative AI in Scientific Writing

The authors did not use a generative artificial intelligence (AI) tool or service to assist with preparation or editing of this work. The author(s) take full responsibility for the content of this publication.

Disclosure Statement

The authors have no conflicts to disclose related to this work.

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Data Sharing Statement

The final analyzed dataset from this study is available upon request from the corresponding author.

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