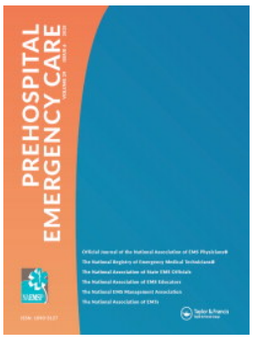

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Nebulized Nitroglycerin Improves Carotid Blood Flow During Cardiopulmonary Resuscitation in a Swine Model of Cardiac Arrest

Christopher Kelly[a], Helen Palatinus[a], Russell Johnson[a], Taryn Hunt-Smith[a], Nicole Ewer[a], Laurence Saint-Pierre[a,b], Guillaume L. Hoareau[a,b,c], M. Austin Johnson[a] and Scott T. Youngquist[a]

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ABSTRACT

Objectives: Pulmonary vasodilators have shown promise in reducing pulmonary vascular resistance during CPR. Most are not currently available outside of an intensive care unit (ICU) setting. Nitroglycerin is widely used by emergency medical services to treat chest pain. If beneficial, the inhalation route of nitroglycerin administration could be implemented by basic life support personnel at the scene of a cardiac arrest. The aim of this investigation was to assess the hemodynamic effects of nebulized nitroglycerin (NIN) in a swine model of ventricular fibrillation (VF)-induced cardiac arrest.

Methods: Seventeen Mixed breed Yorkshire swine (54 + 5 kg) were anesthetized. Ventricular fibrillation was then induced by connecting a 9-volt battery to a pacing catheter in the right ventricle. After 7 min of untreated VF, mechanical chest compressions were initiated. Three minutes later, mechanical ventilation was resumed at pre-arrest settings with 100% FiO2. Two minutes later, 5 mL of normal saline (NIS controls, $n = 9$) or with 10 mg of nitroglycerin (NIN treatment, $n = 8$) were randomly nebulized through a nebulizer in line with the respiratory circuit. After 4 min of nebulization, an intravenous dose of epinephrine (0.015 mg/kg) was administered, followed 2 min later by defibrillation. Standard advanced cardiac life support resuscitation was continued along with NIN or NIS until full delivery, and continued until return of spontaneous circulation or 40 min had elapsed since arrest. Data were analyzed using mixed effects models.

Results: Prearrest arterial and right atrial pressures, chemistries and arterial blood gas values were similar between groups. There were no differences in systolic, diastolic, or coronary perfusion pressures. There was a statistically significant increase in carotid blood flow (CBF) following NIN. Before administering epinephrine, CBF in NIN-treated animals were approximately double those of NIS controls: 92.2 (95%CI 70.69–113.74) versus 41.96 (95% CI 22.28–61.63) mL/min, respectively, a mean difference of 50.26 mL/min (95% CI 24.91–75.61, $p < 0.0001$).

Conclusions: Early nebulized nitroglycerin treatment resulted in superior CBF without decreasing systemic arterial pressures or coronary perfusion pressures in this model of cardiac arrest.

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Introduction

Improving the rates of return of spontaneous circulation (ROSC) and favorable long-term neurological outcomes after cardiac arrest requires optimizing blood flow to vital organs during cardiopulmonary resuscitation (CPR) (1–5). Hypoxic pulmonary vasoconstriction, an increased pulmonary vascular resistance (PVR) in response to systemic hypoxia, is a major limitation to adequate blood flow during CPR (5–7). This increased resistance restricts blood flow through the pulmonary vasculature to the left atrium, reducing left ventricular (LV) filling volume. Consequently, each chest compression during CPR delivers less blood from the LV to vital organs, compromising organ perfusion, ROSC rates, and favorable outcomes (5,8). Interventions that acutely decrease PVR without drastically affecting systemic vascular resistance may improve blood flow during closed chest compressions.

Inhaled nitric oxide and nebulized prostacyclin are pulmonary vasodilators that have shown promise in reducing PVR and increasing transpulmonary blood flow during CPR (5,9). However, their use is currently limited to the intensive care unit (ICU) setting as they require specialized and costly equipment for safe administration (5).

Of the approximately 648,000 cardiac arrests in the United States (U.S.) annually, only 27% (172,280) occur in an ICU, with out-of-hospital locations representing 55% (356,000) (10,11). This limitation creates a significant barrier to broader usage of pulmonary vasodilators and, therefore, investigating alternative pulmonary vasodilation techniques

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suitable for use in cardiac arrest outside of the ICU is warranted.

Nitroglycerin, a widely accessible vasodilator, is used by emergency medical services (EMS) to treat chest pain and possesses known pulmonary vasodilatory properties. Its established safety profile and widespread availability make it an attractive alternative pulmonary vasodilator strategy in cardiac arrest. Intravenous (IV) nitroglycerin has been explored as an adjunct during cardiac arrest, demonstrating a favorable hemodynamic profile and improved organ perfusion (12). Nebulization represents a common method of drug delivery that is within the scope of practice of basic life support (BLS) EMS personnel and largely does not interfere with other resuscitative needs (13). Its effects, however, when nebulized directly to the pulmonary circuit in cardiac arrest remain unexplored in the literature. Notably, nebulized inhaled nitroglycerin (NIN) functions as an effective and selective pulmonary vasodilator, reducing pulmonary arterial pressures (PAP) in animal models of chronic pulmonary hypertension (14). Although this administration route and resulting hemodynamic effects in cardiac arrest have not been investigated, NIN has the potential for selectively lowering PAP and increasing vital blood flow in cardiac arrest.

This investigation aimed to evaluate the hemodynamic effects of NIN in a clinically relevant pig model of cardiac arrest and resuscitation. We hypothesized that treatment with NIN during CPR would improve carotid blood flow without reducing coronary perfusion or arterial pressures.

Methods

Study Design

This was a randomized, investigator-blinded experimental trial comparing the hemodynamic effects of NIN to NIS during cardiopulmonary resuscitation in a pig model of cardiac arrest caused by electrically-induced ventricular fibrillation.

A sample size of 18 animals (9 in each group) was determined by a priori statistical analysis to provide 80% power to detect a 15 mL/min difference in carotid blood flow (CBF) with alpha of 0.05 (G*Power, version 3.1).

This study included adult male and female mixed-breed Yorkshire swine weighing 40–60 kg. The study protocol allowed for pigs to be excluded from the study if baseline temperature was greater than 39 degrees C or white blood cell count was greater than 25,000 cells/mL, although none were ultimately excluded based on these criteria. Animals were randomized to NIS or NIN following a previously generated permuted block scheme generated in an Excel spreadsheet (Microsoft Excel for Mac, version 16.88, Microsoft Corp, Redmond, WA). Investigators were blinded to treatment allocation throughout the experiment. A designated laboratory staff member prepared and provided the undisclosed nebulized solution according to the randomization scheme. This study was approved by the Institutional Animal Care and Use Committee (IACUC) at the University of Utah. The animals were housed and treated in accordance with The Guide for the Use and Care of Laboratory Animals by the National Research Council.

Outcome Measures

The primary outcome measure was the difference in mean CBF at minute 16 of resuscitation attempts, just before administering epinephrine. Secondary measures included the mean difference in coronary perfusion pressures and arterial blood gas parameters: Partial pressure of arterial oxygen (PaO2), partial pressure of arterial carbon dioxide ($PaCO_2$), and arterial lactate concentration, ROSC, and 2-h survival.

Statistical Methods

Data were abstracted directly from laboratory software (LabChart, ADInstruments, Colorado Springs, CO) into an Excel spreadsheet and imported into STATA statistical software (StataBE v 17.0, StataCorp, College Station, TX) for analysis. Coronary perfusion pressure was calculated as the right atrial pressure subtracted from the time-coincident aortic pressure during the release phase of chest compressions just prior to the subsequent compression (15). Parametric and non-parametric summary measures were used as appropriate to the underlying distribution: the two-sample t-test and Mann-Whitney U test, respectively. Time-dependent trajectories of blood gas results and hemodynamic measurements were analyzed using mixed effects linear regression models. The models used a random intercept and time variable with the intervention (NIS vs NIN) entered as a fixed slope effect. The higher-order term of the squared value of time was also used to model curvature in the trajectory of some response variables when this was statistically significant. A discontinuous response trajectory term was included when the intervention was applied to allow for a change in the response trajectory. Rates of ROSC and 2-h survival were compared using a binomial test for independent proportions. For this analysis, we considered a p value of <0.05 to be statistically significant. Of note, CBF measurement was lost in one animal in the nitroglycerin cohort during the latter half of the observation period. Additionally, we were unable to obtain tracings from the transonic probe during an additional animal and, therefore, was excluded from analysis.

Housing and Husbandry

The pigs used in these experiments were allowed to acclimatize in their enclosure 7–10 days prior to the experiment. They were fasted overnight prior to the experiment with free access to water.

Experimental Procedures

Anesthesia was induced with intramuscular injections of tiletamine/zolazepam (4.4 mg/kg), ketamine (2.2 mg/kg), and xylazine (2.2 mg/kg) followed by isoflurane (3–5% by nose cone, as needed). Pigs that met inclusion criteria were

intubated with a cuffed endotracheal tube and ventilated on assist control mode with a tidal volume of 8 mL/kg, fraction of inspired oxygen of 21%, and a positive end-expiratory pressure of 4 cmH2O. The respiratory rate was adjusted to maintain an end-tidal CO2 between 35 and 45 mmHg and the fraction of inspired oxygen was titrated to a target a PaO_2 of 80–100 mmHg. Body temperature was maintained throughout the experiment by use of a heated water blanket set at 38 degrees Celsius. Anesthesia was maintained with isoflurane (1.5–3% by endotracheal tube). Conventional monitoring of the depth of anesthesia was assessed every 15 min. All animals were administered a 10–20 mL/kg PlasmLyte148 bolus IV before the start of the experiment for subclinical dehydration followed by a maintenance rate of 10 mL/kg/hr throughout the study. Additional boluses were administered once a right atrial (RA) catheter had been placed to achieve a normal pre-arrest RA pressure of 3–7 mmHg.

Under ultrasound guidance (Figure 1), a 9 Fr introducer sheath with 7 Fr triple lumen central venous catheter in the right external jugular vein (to accommodate a right atrial pressure catheter), a 9 Fr introducer sheath in the right external jugular vein (for IV fluids and medication administration, temporary use of a pacing lead, and right atrial (RA) pressure monitoring), a 12 Fr introducer sheath in the right femoral artery (aortic pressure (Ao), arterial blood gas sampling), a 7 Fr introducer sheath in the left femoral artery (arterial blood gas sampling), and a 9 Fr introducer sheath (SuperSheath, Boston Scientific) in the left femoral vein (venous blood sampling) were placed. Aortic (Ao), RA, and pulmonary artery (PA) micromanometer pressure catheters (Millar Pressure Catheters, ADInstruments) were then placed under fluoroscopic guidance. The right carotid artery was surgically exposed and an ultrasonic flow probe was placed (Transonic Systems, Inc, Ithaca, New York, USA) to record CBF. Single lead electrocardiogram, Ao, RA and PA pressures were recorded continuously through a data acquisition device (PowerLab, ADInstruments, Colorado Springs, CO) and extracted from data analysis software (LabChart, ADInstruments, Colorado Springs, CO). Waveform capnography was recorded on a defibrillator/monitor (Zoll R Series, Zoll Corp, Chelmsford, MA). Arterial and venous blood gas analyses were performed on a handheld analyzer (iSTAT, Abbott, Abbott Park, IL).

Experimental Protocol

To generate cardiac arrest, a standard bipolar pacing catheter lead was placed in contact with the right ventricular endocardium under fluoroscopic guidance. Ventricular fibrillation was then induced through brief contact of the leads of the pacing catheter with the terminals of a 9-volt battery. Once VF was induced, the pacing catheter was removed and the ventilator turned off. After 7 min of untreated cardiac arrest, mechanical chest compressions were initiated at 100 compressions/minute (Lucas Chest Compression System, Stryker, Portage, MI). Following 3 min of chest compressions only (10 min of cardiac arrest), asynchronous breaths were delivered by the ventilator at pre-arrest settings except for the addition of an FiO2 of 100% and 0 positive end-expiratory pressure. Following 12 min of cardiac arrest, the animals

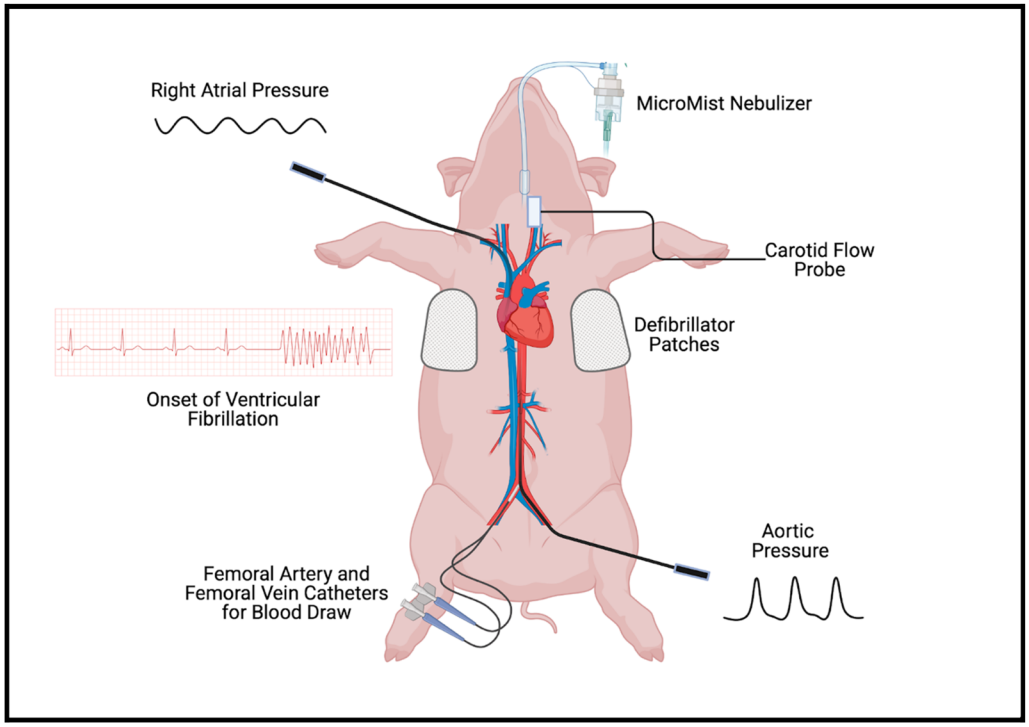

Figure 1. Experimental design diagram.
Diagram denoting location of arterial and venous line placement, carotid flow probe, Micro Mist® Nebulizer device, and defibrillation pads. Lines include: 9 Fr introducer sheath with 7 Fr triple lumen central venous catheter in the right external jugular vein, a 9 Fr introducer sheath in the right external jugular vein, a 12 Fr introducer sheath in the right femoral artery, a 7 Fr introducer sheath in the left femoral artery, and a 9 Fr introducer sheath in the left femoral vein.

were randomized to the control (NIS) or intervention (NIN) group following a previously computer-generated permuted block randomization scheme.

A designated laboratory staff member prepared and provided the undisclosed nebulized solution. The research team was blinded to the contents of the nebulizer. The intervention group received continuous nebulization of 10 mg of nitroglycerin: 2 mL of 5 mg/mL concentration nitroglycerin (American Reagent Inc, Shirley, New York, USA) in 3 mL of normal saline (to a total volume of 5 mL) with *via* a standard nebulizer (Micro Mist® Nebulizer, Medline Industries, Inc, Salt Lake City, UT) in line with the endotracheal tube and delivered in 100% oxygen at 10 L/min. Control animals received a volume equivalent of nebulized normal saline under the same flow parameters. After 16 min of CPR, an intravenous epinephrine bolus (0.015 mg/kg) was administered and repeated, as needed for continued cardiac arrest, every 4 min. Given epinephrine administration is standard in advanced cardiac life support (ACLS), epinephrine was administered prior to any defibrillations to allow us to observe possible interactions on hemodynamic effects when co-administered with NIN. At 18 min of resuscitation, defibrillation at 200 Joules was performed and repeated every 2 min as needed. Defibrillation was intentionally delayed in order to ensure a long enough period to study the effects of the intervention as early defibrillation may lead to immediate ROSC and thus an insufficient window to evaluate any possible effects. If VF persisted beyond 3 shocks, IV amiodarone (Viatris Corp, Pittsburgh, Pennsylvania, USA) boluses (5 mg/kg followed by 2.5 mg/kg 5 min later) were administered. Arterial and venous blood samples were obtained pre-arrest and every 2 min during the first 18 min of cardiac arrest to measure blood gas parameters and lactate concentration. The experimental timeline is represented in Figure 2.

Return of spontaneous circulation was defined as an untreated systolic blood pressure of >50 mmHg for at least 20 min. Resuscitative efforts were terminated if ROSC was not achieved after 40 min of cardiac arrest. If ROSC was achieved, post-resuscitative care with IV fluids and epinephrine infusions were provided until the end of the experiment at 120 min. Additional arterial blood samples were collected at times 40, 60, 120 min if animals achieved ROSC before 40 min.

Results

Data from the 17 animals that completed the experiment were analyzed (8 randomized to NIN treatment and 9 to NNS control). Two animals were excluded from the analysis; one animal died during instrumentation, believed to be due to an air embolism and another animal was used for model development. No differences were observed in pre-arrest variables between two groups, presented in Table 1.

Main Results

Carotid blood flow was significantly higher in treated animals with an average difference of 25.4 mL/min (1.0–49.8 mL/min, $p = 0.041$) in CBF across the observation period. Differences prior to treatment were not statistically significant. Just prior to the administration of epinephrine, the average CBF in NIN animals was more than double that of NIS, 92.2 mL/min (95%CI 70.69–113.74 mL/min) versus 41.96 mL/min (95% CI 22.28–61.63 mL/min), a mean difference of 50.26 mL/min (95% CI 24.91–75.61, $p < 0.0001$) (Figure 3).

Secondary Outcomes

Coronary perfusion pressure was similar between groups throughout the experiment. The response to a bolus dose of epinephrine was comparable between groups (Figure 4).

Mean systolic blood pressure and mean arterial pressure declined in the NIN group after the start of treatment were not statistically significant. Mean diastolic blood pressure slightly increased in NIN treated animals, but this was also not statistically significant (Figure 5).

There were no differences in PaO2 values throughout the experiment. The NIN-treated cohort demonstrated significantly higher PaCO2 values prior to treatment but ventilation seemed to improve with the nebulization of nitroglycerin (Figure 6). Lactate concentration values were similar at all time points between groups.

ROSC and 2-h survival were achieved in 3/9 NIS controls (33.3%) and in 5/8 NIN-treated animals (62.5%), a difference that was not statistically significant.

Sensitivity Analysis

Because control animals had a non-statistically significant lower average CBF prior to treatment, we reran our primary analysis after dropping the control animal that consistently had the lowest CBFs, defined as >1.5 times below the IQR. The results were essentially unchanged, with an average difference in CBF between NIN and NIS animals of 47.4 mL/

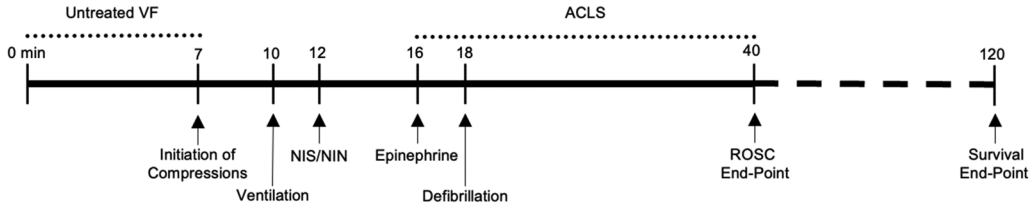

Figure 2. Overview of protocol timeline.
Experimental protocol timeline. VF: ventricular fibrillation; ACLS: advanced cardiac life support; NIS: nebulized normal saline; NIN: nebulized nitroglycerin; ROSC: return of spontaneous circulation.

Table 1. Baseline variables.

	NNS Controls ($N=9$)	NIN Treatment ($N=8$)
Weight (kg)	55 (+4)	53 (+6)
Males (%)	5 (56%)	3 (38%)
Hemodynamics		
Systolic Blood Pressure (mmHg)	104 (+11)	102 (+7)
Diastolic Blood Pressure (mmHg)	75 (+9)	75 (+7)
Right Atrial Pressure (mmHg)	7 (+2)	6 (+5)
Carotid Blood Flow (mL/min)	199 (195–253)	182 (124–294)
Arterial Blood Gas Values		
pH	7.45 (+0.04)	7.44 (+0.04)
pO_2 (mm Hg)	94 (+13)	99 (+11)
pCO_2 (mm Hg)	44 (+4)	42 (+6)
HCO_3 (mEq/L)	31 (+2.5)	29 (+1.5)
Base Excess (mEq/L)	7 (+3.2)	5 (+1.7)
Lactate (mmol/L)	2.2 (+0.9)	2.0 (+0.7)
Serum Chemistries		
Na^+ (mmol/L)	137 (+2)	138 (+2)
K^+ (mmol/L)	3.9 (+0.3)	3.8 (+0.3)
Cl- (mmol/L)	98 (+1.3)	99 (+1.1)
iCa^{++} (mmol/L)	1.3 (+0.1)	1.3 (+0.1)
Total CO_2 (mmol/L)	28 (+2)	30 (+1)
Blood Urea Nitrogen (mg/dL)	6 (+3)	5 (+2)
Creatinine (mg/dL)	1.6 (+0.4)	1.4 (+0.2)
Hemoglobin (g/dL)	8.6 (+1.3)	8.6 (+0.7)
Anion Gap (mmol/L)	*16 (+3)*	*14 (+2)*

Baseline variables prior to the initiation of cardiac arrest comparing pigs randomly assigned to nebulized normal saline (NNS) or to nebulized inhaled nitroglycerin (NIN). Data are presented as mean ± SD or median (IQR).

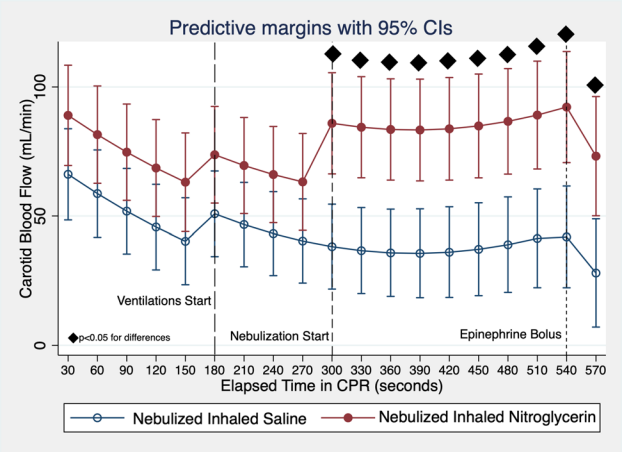

Figure 3. Carotid blood flow.
Carotid blood flow response trajectory in animals treated with nebulized inhaled saline compared to nebulized inhaled nitroglycerin. CI: confidence interval; CPR: cardiopulmonary resuscitation.

min (95% CI 18.3–76.6 mL/min, $p=0.001$) at the end of the observation period.

Discussion

This study evaluated the hemodynamic effects of NIN in a VF model of cardiac arrest in swine. We found a statistically significant difference in mean CBF as well as CBF trajectory compared to NIS without compromising systemic mean arterial pressure or coronary perfusion pressure (CPP). We did not find that the addition of NIN blunted the effects of bolus dose epinephrine.

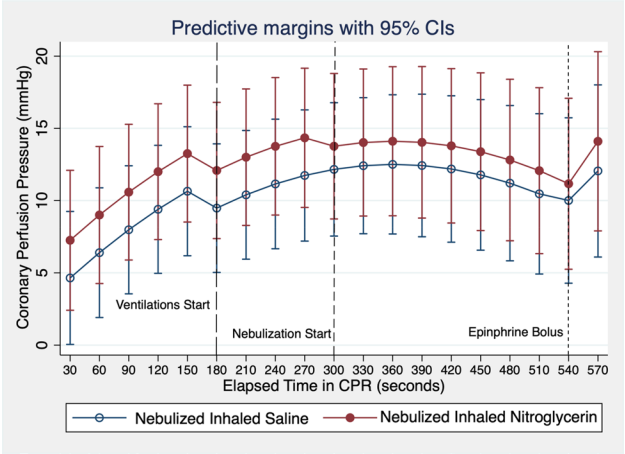

Figure 4. Coronary prefusion pressure.
Coronary perfusion pressure response trajectory in animals treated with nebulized saline compared to animals treated with nebulized nitroglycerin. CI: confidence interval; CPR: cardiopulmonary resuscitation.

The concept behind using pulmonary vasodilators during cardiac arrest revolves around decreasing pulmonary vascular resistance and thus increasing trans-pulmonary blood flow. This study was partially modeled on previous swine cardiac arrest studies evaluating systemic administration of vasodilators, such as sodium nitroprusside, which found similar improvements in CBF (16,17). Additionally, inhaled nitric oxide has been shown to have selective pulmonary vasculature vasodilation with a resultant decrease in right ventricular afterload, allowing for improved oxygenation and ventilation-perfusion distribution (5,18,19). Although demonstrating possible benefits, these studies are limited by multiple factors that we attempted to address in this study. For one, administration of systemic pulmonary vasodilators has the potential deleterious effect of decreased mean arterial blood pressure thus also affecting coronary perfusion (20,21). Yannopoulos et al., attempted to proactively counter this negative effect *via* abdominal binding, although this would represent an additional complexity to prehospital care (16). Additionally, nearly all available pulmonary vasodilators require in-hospital administration, specialized equipment for delivery, or are cost-prohibitive for widespread adoption.

Nitroglycerin, which is metabolized to nitrous oxide (NO), has the benefit of widespread availability in prehospital EMS systems (22–24). Delivery *via* nebulization represents a route of administration that BLS EMS personnel are already capable of providing without requiring specialized equipment or training (13). Nebulized inhaled nitroglycerin represents a possible early intervention in out-of-hospital cardiac arrest care that would not interfere with standard resuscitative practices.

The underlying mechanism of action requires ongoing research. We observed an improved CBF without a significant change in CPP. Further investigation is needed to determine whether the increased CBF represents a benefit from direct pulmonary vasodilation or whether NIN also has an effect on cerebral arterial resistance. Ideally, direct pulmonary vascular resistance measurements could be directly assessed; however, these methods are challenging to implement in cardiac arrest models using closed chest compressions. Ultrasonic flow

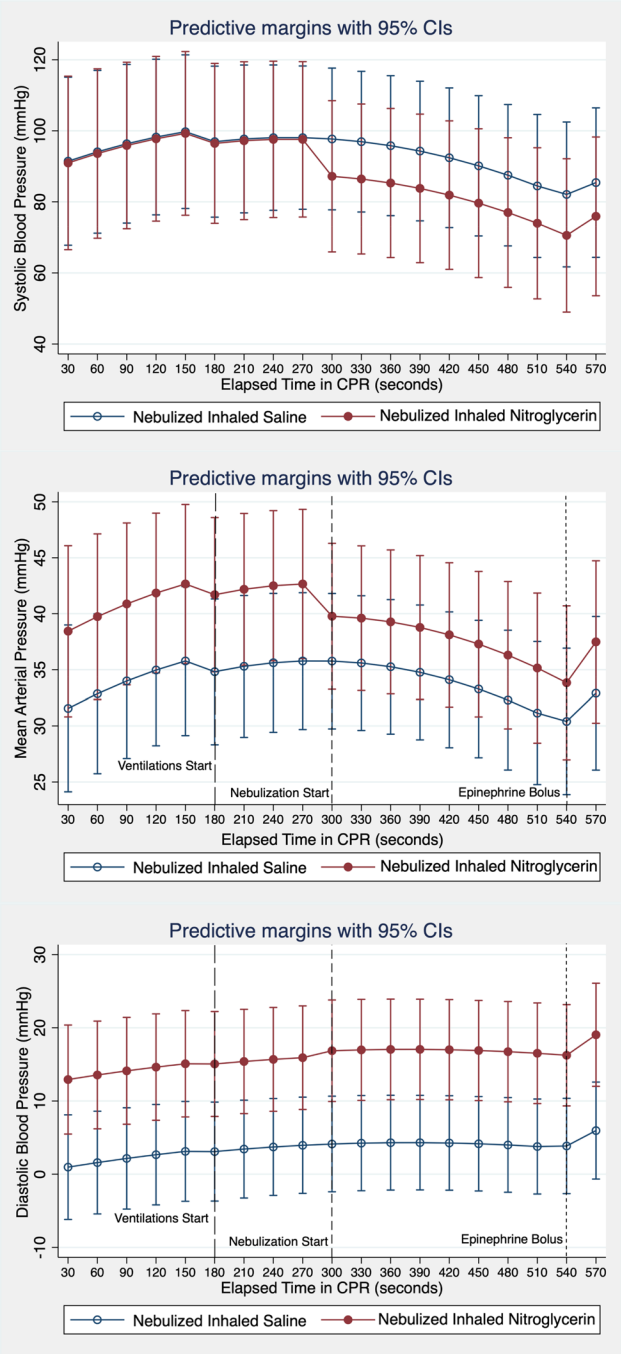

Figure 5. Systemic hemodynamic response.
Hemodynamic response trajectories over time of animals treated with nebulized saline and nebulized nitroglycerin. (a) Systolic blood pressure, (b) Mean arterial pressure, (c) Diastolic blood pressure. CI, confidence interval; CPR: cardiopulmonary resuscitation.

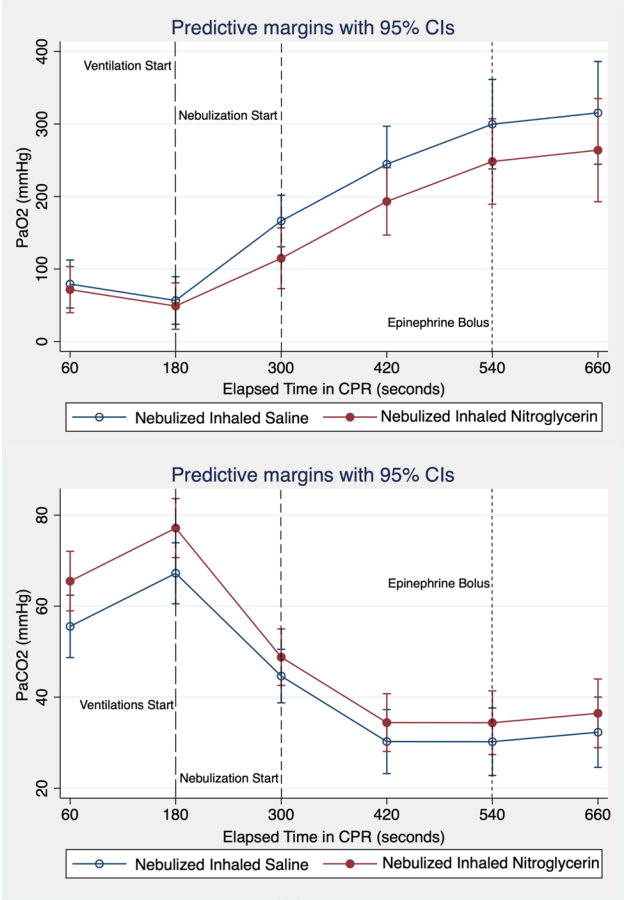

Figure 6. Arterial blood gas parameters.
Partial pressure of oxygen (a) and carbon dioxide (b) over time in animals treated with nebulized saline and nebulized nitroglycerin. CI: confidence interval; CPR: cardiopulmonary resuscitation; $PaCO_2$, partial pressure of CO_2.

probes require thoracotomy, compromising the thoracic pump function of CPR, and intravascular flow probes and pressure catheters impair function of the pulmonary valve. Additional studies of dose optimization as well as evaluating alternative inhalation methods *via* supraglottic airways are warranted. In addition, it is unclear how asynchronous ventilation at guidelines-recommended rates or 30:2 ventilation would interact with the intervention.

Our study was the first to evaluate the usage of nebulized nitroglycerin in a swine cardiac arrest model to demonstrate feasibility and proof of concept to allow for prehospital system adoption. As opposed to alternatively available pulmonary vasodilators, nebulized nitroglycerin offers the possibility of widespread adoption given its ubiquitous availability among prehospital care systems as well as its relatively low cost. Additionally, this study was conducted as a randomized, blinded trial ensuring a standardized resuscitation approach.

Limitations

In our study protocol, NIN was delivered *via* endotracheal tube with the acknowledgment that not all prehospital cardiac arrest cases will have a secured airway in place. Alternative ventilation devices such as bag-valve-mask or a laryngeal mask airway device may have differing delivery results and, as such, represent avenues for potential future investigation. Given delivery was conducted *via* an endotracheal tube, as designed, this study represents a possible advanced life support intervention and its use by emergency medical technicians may be limited by scope of practice until delivery *via* other airway devices are studied. As mentioned above in the sensitivity analysis, removing outliers did not alter the lack of significance and we were still able to demonstrate a significant mean difference as well as trend line in CBF favoring the NIN cohort. Additionally, as mentioned above, direct measurements of pulmonary vascular resistance intra-arrest are difficult and as such indirect measurements were used to

determine effect size. Other invasive measurements such as intracranial pressure to calculate cerebral perfusion pressure was not undertaken in this study but may have provided additional information regarding the effect of NIN and may be useful in future studies.

Conclusions

This study demonstrated that nebulized nitroglycerin significantly improved CBF compared to nebulized normal saline without compromising mean arterial pressure or CPP in a ventricular fibrillation swine cardiac arrest model. Given the widespread availability among prehospital emergency medical systems of nitroglycerin as well as the ease of nebulization, nebulized inhaled nitroglycerin may represent an early beneficial intervention in out-of-hospital cardiac arrest care.

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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.

Data Sharing Statement

Data are available by reaching out to the corresponding author.

Authorship Statement

All listed authors had substantial contributions to the acquisition and/or analysis of the work, were involved in drafting the work, approve of the final version to be published, and agree to be accountable for all aspects of the work.

Disclosure Statement

All authors declare no potential financial conflicts of interest unless otherwise stated below. Christopher Kelly, MD: Principal recipient of ZOLL Foundation Grant. Scott T. Youngquist, MD MS: Advisory Board Member, Emergency Scientific, Salt Lake City, UT

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