Published in final edited form as:

Prehosp Emerg Care. 2011 ; 15(3): 405–409. doi:10.3109/10903127.2011.569847.

Does Early Postresuscitation Stress Hyperglycemia Affect 72 Hour Neurologic Outcome? Preliminary Observations In The Swine Model

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Abstract

Background—Hyperglycemia is common in the early period following resuscitation from cardiac arrest and has been shown to be a predictor of neurologic outcome in retrospective studies. The purpose of this study was to evaluate neurologic outcome and early post-arrest hyperglycemia in a swine cardiac arrest model.

Methods—Electrically-induced ventricular fibrillation cardiac arrest was induced in 22 anesthetized and instrumented swine. After 7 min, CPR and advanced cardiac life support was initiated. Twenty-one animals were resuscitated and serum glucose was measured at intervals for 60 min post-arrest. Animals were observed for 72 hours and neurologic score determined at 24 hr intervals.

Results—Ten animals demonstrated a peak plasma glucose ≥ 226 mg/dl during the initial 60 min post-arrest. Neurologic scores at 72 hrs in these animals (mean score = 0, mean overall performance category = 1) was the same as that in animals with a serum glucose < 226 mg/dl. End-tidal CO₂ measured during CPR, time to restoration of spontaneous circulation, and epinephrine dose were not significantly different between animals with a peak glucose ≥ 226 mg/dl and those with lesser values. The sample size afforded a power of 90% to detect a 50 point difference from the lowest score (0 points) of the porcine neurologic outcome scale.

Conclusion—In this standard porcine model of witnessed out-of-hospital cardiac arrest, early stress post-resuscitation hyperglycemia did not appear to affect neurologic outcome. During the prehospital phase of treatment and transport, treatment of hyperglycemia by emergency medical service providers may not be warranted.

Keywords

cardiopulmonary resuscitation; post-resuscitation period; glucose; outcome

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Declaration of interest:

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

INTRODUCTION

Although 30–40% of patients with sudden out-of-hospital cardiac death and cardiac arrest achieve restoration of spontaneous circulation following resuscitative efforts, only about 8% survive to hospital discharge.(1) In this population, the most common cause of death is brain injury.(2) The pathobiology of brain injury and death following resuscitation is complex yet amenable to interventions which may minimize the extent of neuronal death.(2–4)

Laboratory investigations completed three decades ago demonstrated that hyperglycemia preceding the induction of focal or global cerebral ischemia in the rodent and feline model worsened neurologic outcome.(5–7) In these experiments, pre-ischemic plasma glucose concentrations in glucose-infused animals ranged between 500–1000 mg/dl. Hyperglycemia was associated with an increase in cerebral lactate and derangements of the recovery of brain energy metabolism. Hyperglycemia after the ischemic event has since been shown in the clinical population to worsen the extent of neurologic injury in focal cerebral ischemia (stroke).(8,9)

Hyperglycemia is common after resuscitation from cardiac arrest and it has been suggested that post-arrest hyperglycemia may decrease the likelihood of survival following recovery of spontaneous circulation.(10,11) Although an association between blood glucose and mortality and neurologic outcome has been established in patients with acute stroke (9), such an association has not been consistently or conclusively demonstrated for post-resuscitation hyperglycemia and neurologic outcome. Although post-resuscitation hyperglycemia is generally believed to be the result of a stress response, resuscitation interventions can potentially exacerbate the hyperglycemic response, ie, glucose administration for treatment of intra-arrest hypoglycemia or intra- or post-arrest hyperkalemia.

The effect of post-arrest hyperglycemia on neurologic outcome has not been studied in large animal laboratory models. The conventional porcine cardiac arrest model lends itself to such an assessment because many of the variables impacting resuscitation and survival, e.g., down time, time to CPR, CPR quality, post-resuscitation hemodynamics, can be standardized and controlled or their effect minimized. The purpose of this study was to assess the effect of plasma glucose, measured during the first hour post-arrest, a time which includes prehospital and emergency department care as well as critical care interfacility transport, on 72 hr neurologic outcome in swine.

METHODS

This study followed the NIH guidelines for the use of laboratory animals in biomedical research and was approved the Biological Resource Center and Animal Care and Utilization Committee of our institution.

This study represents a post hoc analysis of data collected during a comparative trial of standard CPR versus CPR using a new CPR device.(12) In brief, after an overnight fast but with ad libitum access to water, 22 male and female mixed-breed swine (34–43 kg) were sedated and general anesthesia was induced with isoflurane via nose-cone and, following endotracheal intubation, maintained with inhaled isoflurane and 50% oxygen and 50% nitrous oxide. Minute ventilation was adjusted to maintain an end-tidal pCO₂ of 35–45 mm Hg. Standard lead II of the surface electrocardiogram was monitored during instrumentation and throughout the study protocol.

An internal jugular vein and femoral artery were surgically exposed and micromanometer-tipped catheters with side holes (Millar Instruments, Houston, TX) were inserted and

positioned in the right atrium (RA) and aortic arch (Ao) for pressure monitoring and blood withdrawal. The tip of a bipolar pacing catheter was positioned in contact with the right ventricular endocardium for induction of VF. Catheter tip positions were confirmed fluoroscopically.

Following instrumentation, heart rate, systolic and diastolic aortic pressure and mean right atrial (RA) pressure, were recorded and arterial blood was analyzed (I-Stat EG7+, I-Stat Corp, Princeton, NJ). Ventricular fibrillation (VF) was then induced with a brief 60 Hz AC current pulse delivered to the right ventricular endocardium via the pacing catheter. After 7 min of untreated VF, chest compressions were initiated either manually or with a manually operated sternal compression/thoracic constraint device.(12) Randomization to CPR type was by permuted block design and each type of CPR was performed by a single rescuer. Chest compressions were performed at a rate of 100 compressions/min with force sufficient to depress the sternum 1.5 to 2.0 inches with either method. Chest compression depth was confirmed videographically. One minute after starting chest compressions, end-tidal CO₂ was measured and a transthoracic biphasic countershock (Physio-Control LifePak 12, Physio-Control Corporation, Redmond, WA) at 200 J was given. If VF persisted, a second shock at 300 J was administered. If necessary, the third and subsequent countershocks were delivered at 360 J. (13) Chest compressions were performed between shocks and positive pressure ventilations (F_iO₂=1.00) were performed at a rate of 8–10 ventilations/min. If VF persisted after the initial three shocks, epinephrine, 0.5 mg (approximately 0.01 mg/kg), was administered and CPR continued for one to three minutes before additional shocks at 360 J were given. If asystole or pulseless electrical activity (PEA) followed shocks, CPR and additional epinephrine were administered until spontaneous arterial pressures of 60 mm Hg appeared or for 15 min. At the end of 15 min of CPR, animals remaining in VF, PEA, or asystole were considered resuscitation failures and resuscitative efforts terminated.

In those animals achieving return of spontaneous circulation (ROSC), defined as an arterial systolic pressure >60 mm Hg for >10 min (14), hemodynamic and blood gas measurements and glucose determinations were made at 15, 30, 60 min following ROSC. When mean arterial pressure had returned to prearrest values, incisions were surgically repaired and animals were weaned from ventilator support. When breathing spontaneously and responding to stimuli, animals were returned to their cages and underwent neurologic scoring at 24, 48, and 72 hours following ROSC.(15) In brief, the neurologic deficit score assigns values for deficits in swine neurologic function. A score of 0 is normal, and a score of 400 indicates brain death. The cerebral performance category represents a global assessment of neurologic function with category 1 being normal and category 5 being brain death. Neurologic scoring was performed by the institutional veterinarian who was blinded to the experimental protocol and had no information on glucose levels.

For the purposes of the study, animals with a peak serum glucose value ≥ 226 mg/dl during the first hour following resuscitation were considered “hyperglycemic”.(16) With at least 8 animals in the “hyperglycemic” group and 8 in the “non-hyperglycemic” group, we calculated a power of 95% to detect a 50 point mean difference in neurologic deficit score, assuming a standard deviation of 25. A 50 point difference in the swine scoring system would allow recognition of changes in consciousness, respiratory pattern, motor or sensory function, or behavior that would be easily detected by an experienced research veterinarian.

Control, CPR, and post-resuscitation hemodynamic data were recorded and stored on a laptop computer using PowerLab Chart v. 5.2 (ADInstruments, Colorado Springs, CO). Data are presented as the mean $\pm$ SD. For all comparisons, $p < 0.05$ was considered statistically significant. Two-way repeated measures ANOVA was used to compare glucose levels over time for study groups with p values adjusted for multiple comparisons according to the

method of Dunnett-Hsu. Student t-test was used to compare resuscitation variables. Binomial confidence intervals are reported for proportions.

RESULTS

Twenty one animals were successfully resuscitated. Ten of these animals demonstrated a peak serum glucose of ≥ 226 mg/dl. There were no statistically significant differences in resuscitation variables between animals with a peak serum glucose ≥ 226 mg/dl and those with a lower value (table 1). Four animals in the low glucose group and 3 animals in the high glucose group received no epinephrine. All animals were breathing spontaneously, extubated, and returned to the vivarium within 3 hours of ROSC.

Serum glucose concentrations for the groups are shown in figure 1. Glucose levels measured after resuscitation were significantly greater than pre-arrest values at all time points within study groups. Plasma glucose was significantly greater in the high glucose group at all time points. The peak glucose of ≥ 226 mg/dl was observed at 15 min in 81% of these animals and peak glucose was > 240 mg/dl in all animals in the high glucose group.

Twenty of the 21 resuscitated animals survived 72 hours and all had a neurologic deficit score of 0 and cerebral performance category of 1. Ten of 20 (50 %) of these animals had an elevated glucose, as defined previously. Thus, the specificity of an early, elevated post-ROSC glucose for predicting poor neurologic outcome in this model was only 50% (95% CI 28–72%). One animal was euthanized 18 hours after resuscitation due to coma and intractable generalized seizure activity. This animal had a peak glucose of 213 mg/dl at 15 min post-resuscitation. While sensitivity cannot be reliably calculated from this single animal, the lack of an elevated glucose above the threshold in this neurologically deficient animal suggests it is less than 100%.

DISCUSSION

To our knowledge, this is the first study to assess the effect of early post-resuscitation hyperglycemia on long-term (72 hr) neurologic outcome in the conventional swine model of cardiac arrest and resuscitation. We observed that plasma glucose level during the first hour after resuscitation had no effect on 72 hour neurologic outcome determined using a standardized neurologic scoring instrument for swine. In this standard porcine model of witnessed out-of-hospital cardiac arrest, differences between glucose groups were not observed for resuscitation variables previously reported to be predictors of neurologic outcome, namely, cardiac arrest rhythm, time from arrest to CPR, quality of CPR (systemic perfusion reflected by end-tidal CO_2), ischemia time (time to ROSC), and epinephrine dose.

The plasma glucose value (≥ 226 mg/dl) used to categorize our sample into the “high” glucose group is the maximum median value observed in diabetic patients after resuscitation from in-hospital cardiac arrest.(15) Our selected cutoff value also approximates the median glucose value in patients observed within the first hour following resuscitation from out-of-hospital arrest and the median value of patients resuscitated from out-of-hospital cardiac arrest who did not survive 6 months as reported by others.(16,17)

Nearly all prior laboratory studies designed to assess the effect of hyperglycemia on neurologic outcome after a cerebral ischemic insult have included glucose infusion or injection before the ischemic event.(5–7) In some instances, plasma glucose has exceeded 1000 mg/dl prior to vessel occlusions. A recent report using a porcine arrest model in which prearrest hyperglycemia (153–180 mg/dl) was induced experimentally found an increase in post-resuscitation cerebral tissue oxygenation in the hyperglycemic group when compared to a normoglycemic group.(18) However, the investigators were unable to demonstrate

differences in cerebral perfusion or markers of oxidative stress and inflammation. In that study, an early increase in plasma glucose in the normoglycemic control groups following resuscitation was also noted and of a similar magnitude and duration to that observed in our study with prearrest normoglycemia.

Hyperglycemia is not unexpected in resuscitated victims of cardiac arrest and is one manifestation of the stress response. Mediators include the counter-regulatory hormones cortisol, catecholamines (endogenous and administered during resuscitation efforts), and glucagon.(19,20) The glycemic response is more pronounced in the setting of insulin deficiency. The magnitude and duration of hyperglycemia are associated with the duration of cardiac arrest and the severity of the post-resuscitation syndrome, characterized by hemodynamic instability and an innate immune response similar to that observed in sepsis. (21)

Diabetics resuscitated from cardiac arrest have been shown to have a higher in-hospital mortality and worse neurologic outcome.(22,23) This is likely due to the pathophysiological changes associated with the disease.(24) These changes have been enumerated by Matchar and colleagues and include atherosclerosis, impaired autoregulation of cerebral blood flow, increased blood viscosity, increased endothelial adhesion of red blood cells and decreased erythrocyte deformability, all of which are likely to limit perfusion of vital organs after restoration of circulation.

Prior studies of the role of hyperglycemia in post-arrest outcome have extensively utilized univariate and multivariate logistic regression analysis to identify hyperglycemia as a significant independent variable utilizing registries of varying size.(15–17) Using these statistical methods, conclusions are dependent upon the variables selected for univariate analysis and are susceptible to confounding by uncontrolled factors. Using similar methods, other reports have not demonstrated an association between glucose and outcome.(25) Such studies share common limitations acknowledged by the authors including inaccuracies related to ischemia and resuscitation durations (“down time”), uncertainty regarding pre-event history and presence of diabetes, absence of pre-arrest blood glucose values, and the quality of post-resuscitation care including insulin use in the registry population. Additionally, clinical trials have failed to demonstrate that glucose control favorably improves ultimate survival and that early glucose control may not be necessary.(26,27)

Limitations

This study has several limitations. Healthy swine served as study subjects. The majority of the clinical population who suffer an out-of-hospital cardiac arrest have one or more co-morbid diseases that may impact the magnitude and effect of post-arrest hyperglycemia. Longer ischemia duration, prolonged resuscitation, the use of a spontaneous, ischemically induced VF model, the administration of larger doses of epinephrine, and the post-arrest infusion of vasopressors would likely induce a more profound and sustained stress response. Of note, however, prior studies in swine with cardiac arrest periods >12 min have been followed by normal neurologic function and outcome in the vast majority of swine.(14) The neurologic scoring system used in this study assesses only primitive function and subtle neurologic dysfunction may have been missed. The CPC score commonly used to assess neurologic outcome in patients has also been shown to miss substantial cognitive deficits, particularly in infants and children (28–32). Lastly, our swine model most closely replicates the clinical scenario of a witnessed cardiac arrest and a “response time” typical of established EMS systems.(1).

Conclusion

Our preliminary study suggests that early stress hyperglycemia does not have a predictive role in post-arrest neurologic function in the swine model. The swine model demonstrates a glucose stress response similar to that observed in patients and the model affords control of other important outcome variables absent from retrospective clinical reports. During the prehospital phase of treatment and transport, treatment of hyperglycemia by first responder emergency medical service providers or during interfacility transport in the early post-resuscitation period may not be warranted.

Acknowledgments

Funded, in part, by grants from the National Institutes of Health, NHLBI R01 HL076671 and NHLBI R42 HL071378.

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Figure 1. Serum Glucose versus Time for Study Groups

Serum glucose measured pre-arrest and at intervals following resuscitation are shown. Glucose levels in both groups were significantly greater than pre-arrest values at all time points following return of circulation. Blood glucose was greater in the “high” glucose” group when compared to values in the “low glucose” group (* $p < 0.001$ vs “low” glucose group).

Table 1

Resuscitation Variables for Low and High Glucose Groups

	Systolic Aortic Pressure mm Hg	Diastolic CPP mm Hg	End-tidal CO ₂ mm Hg [*]	Shock #	Epi Dose mg
Low	79 ± 14	22 ± 9	17 ± 7	1 ± 1	0.49 ± 0.46
High	76 ± 12	22 ± 4	20 ± 6	2 ± 1	0.38 ± 0.32

* End-tidal CO₂ measured after one minute of chest compressions.