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Hyperoxia and Hypoxia in Children Resuscitated From Cardiac Arrest

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Abstract

Background—Ischemia depletes antioxidant reserves and impairs mitochondrial electron transport. Oxygen within blood reperfusing ischemic tissue can form free radicals, worsen oxidative stress and exacerbate tissue injury (reperfusion injury). One strategy for limiting reperfusion injury is to limit delivery of “luxuriant” oxygen during or after reperfusion. Resuscitation guidelines for children with cardiac arrest recommend early weaning of supplemental oxygen as tolerated. There are currently no studies demonstrating the frequency and outcomes of hyperoxia and hypoxia after pediatric CA.

Objective—To determine the frequency and outcomes of hyperoxia and hypoxia in patients following resuscitation from pediatric cardiac arrest admitted to a tertiary care center.

Design and Methods—This is a retrospective observational cohort study. Charts of children resuscitated from cardiac arrest and admitted to our hospital from 2004-2008 were reviewed. Partial pressures of oxygen (PaO_2) obtained within the first 24 hours following return of spontaneous circulation and mortality at 6 months was recorded. Children who did not survive the initial 48 hours, patients having undergone extracorporeal oxygenation (ECMO) or had congenital heart disease, and those in whom arterial blood gases (ABG) were not obtained were excluded.

Results—Seventy-four patients met inclusion criteria. Of these, 38 (51%) had at least one ABG with a $Pa0_2$ > 300 mm Hg and 10 (14%) had a $Pa0_2$ < 60 mmHg in the first 24 hours. Neither hyperoxia nor hypoxia on initial ABG (p=0.912 and p=0.384) nor any ABG within the first 24 hours after CA (p=0.325 and p=0.553) was associated with 6 month mortality.

Conclusions—Hyperoxia occurs commonly within the first 24 h of management in children resuscitated from cardiac arrest.

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Keywords

Heart Arrest; Resuscitation; Hyperoxia; Pediatrics

Introduction

Oxygen is necessary for life-sustaining aerobic respiration as well as oxidative phosphorylation but oxygen can also form free radicals (molecules with unpaired, highly reactive electrons). Free radical production, when it exceeds the body's antioxidant defenses, can lead to damage of important cellular structures including cell membranes, intracellular proteins and DNA[1-3]. Within healthy cells, the highly volatile nature of oxygen chemistry is carefully balanced with antioxidant defenses and oxidative phosphorylation, with its transfer of free electrons, is controlled and sequestered within the double membrane of the mitochondria [2]. With ischemia, mitochondrial integrity is impaired and the sudden influx of oxygen during reperfusion causes an increase in reactive oxygen species (ROS) that can overwhelm the antioxidant defenses of the cell and thus worsen injury (reperfusion injury)[4]. The challenge in managing ischemic injury is to provide enough oxygen to facilitate cellular recovery without providing excessive oxygen that can contribute to reperfusion injury.

Kilgannon et al found an association between hyperoxia (PaO_2 > 300 mmHg) and to a lesser degree hypoxia (PaO_2 < 60 mmHg) on first arterial blood gas (ABG) and mortality after adult cardiac arrest (CA) [5]. One strategy for limiting oxidative-mediated reperfusion injury following hypoxic-ischemia is to limit delivery of “luxuriant” oxygen during reperfusion. Trials comparing room air to supplemental oxygen for resuscitation of experimental CA and in depressed newborns show better when resuscitated with room air [6-10]. Trials comparing room air to supplemental oxygen in adults with myocardial ischemia or stroke also show better outcomes in those treated with room air [11,12]

Unlike in adult CA, the primary etiologies of pediatric CA are respiratory failure and non-cardiogenic shock, states that can create profound cellular hypoxia[13,14]. Fetuses normally thrive in PaO_2 20-30 mmHg compared with 85-100 mmHg in healthy children, and it is not known whether results seen in newborns will translate into children. The American Heart Association guidelines for resuscitation of children with CA recommend resuscitating with FiO_2 = 1.0 during CA followed by early weaning of supplemental oxygen [15,16]. The opportunity for this intervention (weaning) depends upon the frequency of hyperoxia in the clinical setting. We sought to determine the frequency and outcomes of hyperoxia and hypoxia in pediatric patients following resuscitation from CA and analyze for associations with mortality. We hypothesized that hyperoxia and hypoxia occur commonly in the management of patients resuscitated from CA and may represent an important and unrecognized opportunity to improve patient care and outcomes.

Methods

Approval for the study was obtained through our Institutional Review Board IRB). Patients were identified through a CA database maintained in our pediatric intensive care unit (PICU). The maintenance of this database was also IRB approved. The database includes all children admitted to the PICU having survived initial resuscitation from CA. Children with congenital heart disease were excluded from this database. Patient charts from January 2004 thru August 2008 were reviewed. Patients were excluded from review if the patient survived for < 48 hours, underwent extracorporeal membrane oxygenation (ECMO), and or did not have arterial blood gas (ABG) values recorded during the first 48 hours after admission. Charts were abstracted by the PI and data was collected using standardized data collection

sheets. Nursing notes and respiratory flow sheets were reviewed for all patients. Data collected included demographics, historical data, partial pressures of inspired oxygen (FiO_2), pulse oximetry values, PaO_2/FiO_2 and ABG values.

Patient enrollment (time zero for time to first ABG) began at the time of return of spontaneous circulation (ROSC) for in-hospital CA or the time of arrival to our institution for out-of-hospital CA. In our PICU, ABG values and respiratory data are collected at hourly intervals until deemed unnecessary by the PICU attending. We defined hyperoxia as an ABG with a PaO_2 > 200 or 300 mmHg. If more than one ABG was obtained per hour, the ABG with the highest partial pressure of arterial oxygen (PaO_2) was entered into the database. Hypoxia was defined as PaO_2 < 60 mmHg.

Data analysis

Clinical variables were analyzed for associations with mortality at 6 months using Fisher's exact test. All other statistical tests for binary or categorical variables were also conducted using Fisher's exact test, whereas differences in continuous variable were assessed using the Wilcoxon rank-sum test. Spearman's rank was used to correlate age and etiology of CA with mortality and PaO_2 variables. P-values < .05 were considered statistically significant. A logistic regression was performed for PaO_2 cutoffs < 60 mmHg, > 200 mmHg, and > 300 mmHg and mortality. Data were summarized via either medians (range), for continuous variables, or frequencies (%), for binary or categorical data. All analyses were conducted using Stata software, version 12 (College Station, Texas).

Results

The PICU arrest database from Jan 2004-August 2008 identified 124 patients who suffered a CA. Fifty patients were excluded: 29 expired in less than 48 hours, 9 underwent ECMO, and 12 did not have any ABGs during the 24 hours following admission. Seventy-four patients met inclusion criteria and 53 (72%) survived to 6 months. The median age was 1.8 (0-18) years and 59% were males (*Table 1*). As would be expected with a pediatric population, the majority of CAs were due to respiratory failure and shock, occurred out-of-hospital, and had asystole as the first rhythm.

Highest and lowest PaO_2 were not associated with age or etiology of CA. Median initial PaO_2 was 160 mmHg (range 28-622) drawn at median hour 1 (0-19) (*Figure 1*). The reason for lateness of time to first ABG for some patients was not known. FiO_2 at the time of initial ABG was 0.97 (0.35-1.00), and P/F ratio was 276 (28-718). Highest median PaO_2 in 24 hours was 315 mmHg (69-622) and occurred at hour 4 (0-23). Median FiO_2 at the time of the highest PaO_2 was 0.80 (0.35-1.00) with a corresponding P/F ratio of 418 (77-718). Lowest median PaO_2 was 78 mmHg (28-399) and occurred at hour 12 (0-23). Median FiO2 at the time of the lowest PaO_2 was 0.49 (0.26-1.00) in the first 24 hours with corresponding P/F ratio 176 (28-547). Neither hyperoxia nor hypoxia on initial ABG (p=0.912 and p=0.384) or within the first 24 hours after CA (p=0.325 and p=0.553) was associated with 6 month mortality.

Among ABGs obtained, Figure 2 shows the percentage of patients with hyperoxic and hypoxic results over the first 24 hours. Fifty-two (70%) patients had at least one PaO_2 > 200 mmHg and spent a median of 2 (range 0-20) hours > 200 mmHg. Thirty-eight (51%) patients had at least one PaO_2 > 300 mmHg in the first 24 hours following CA and spent 1 (0-14) hour > 300 mmHg in the first 24 hours. Ten (15%) patient had at least one PaO_2 < 60 mmHg and spent 0 (0-12) hours < 60 mmHg in the first 24 hours. The AUC for PaO_2 > 200 mmHg was 0.459, for PaO2 > 300 mmHg was 0.493 and for PaO2< 60 mmHg was 0.461 to predict mortality at 6 months. See Table 2 for associated logistic regression results.

We examined data prior to and following publication of the AHA Guidelines to determine if clinical practice changed in response to recommendations for weaning oxygen. The frequency of hyperoxia (> 300 mmHg) from 2004-2008 was 65%, 29%, 40%, 60%, 60%, for each year, respectively.

Discussion

Secondary brain insult from hypoxia, hyperthermia, and hypotension have been associated with poorer outcome after pediatric acute brain injury [17,18]. This is the first study to characterize the occurrence and outcome of hyperoxia and hypoxia following resuscitation from pediatric CA. We found hyperoxia to be common in children during the first 24 hours following ROSC from CA with over half of children having a blood gas with a PaO_2 > 300 mmHg while only ten children experienced hypoxia. It is plausible that the frequency of hypoxia and hyperoxia were underestimated as we relied on the performance of ABGs by the treating team. Unlike studies in adult CA, neither hyperoxia nor hypoxia in the first 24 hours following CA was associated with mortality.

The AHA Guidelines for resuscitation published in 2005 and 2010 recommend weaning oxygen to saturations > 94% as tolerated following CA[15,16]. We found that hyperoxia was equally common before and after publication of the 2005 AHA Guidelines. This suggests that avoidance of hyperoxia remains an underappreciated goal of post-resuscitation care.

As early as 2002, Lefkowitz questioned the wisdom of using 100% oxygen during resuscitation in light of emerging evidence for the role of oxygen in potentiating reperfusion injury [19]. Lefkowitz concluded that the use of 100% oxygen was not based on evidence of benefit, but rather on historical notions of its good. The potential detrimental effects of hyperoxia have since been demonstrated in various experimental and clinical populations[20-27]. Studies in newborns show that resuscitation with room air leads to faster transition to normal breathing and crying, less consumption of antioxidants by reactive oxygen species, and that resuscitation with oxygen increases the risk of cerebral palsy [6-8,28,29]. A recent prospective, randomized trial by Vento et al showed that premature infants resuscitated with 30% oxygen compared with 100% oxygen had a shorter requirement for supplemental oxygen, fewer days on the ventilator, and were less likely to develop bronchopulmonary dysplasia [8]. A small, prospective, randomized pilot trial in adults comparing the use of 30% with 100% oxygen in the first hour following resuscitation from CA showed a significant increase in neuron specific enolase (a biochemical marker of neuronal injury) in normothermic patients randomized to 100% oxygen [12].

Kilgannon et al examined a large, multicenter cohort of patients admitted to adult ICUs following resuscitation from CA and found an association between hyperoxia (18% of the cohort, defined as a PaO_2 >300) on the first ABG and increased mortality (odds ratio for death of 1.8 compared to normoxic patients [19% of the cohort]) [5]. The largest group of patients was those with hypoxia, who had an odds ratio for death of 1.3. Similar to traumatic brain injury (TBI), hypotension as a secondary insult had and odds ratio for death of 2.1[17]. Importantly, the “threshold” for hyperoxia resulting in harm has not been defined. In a subsequent study, Kilgannon found that using the highest PaO_2 in the first 24 hours following CA, for every 100 mmHg increase in PaO_2, there was a 24% increase in mortality risk[30]. Patients with hypoxia were excluded from this analysis.

Considering the available evidence that hypoxic-ischemic brain injury can be worsened by exposure to supplemental oxygen, our study demonstrates a potential opportunity for improvement in clinical care. However, there are other important considerations in favor of supplemental oxygen. Both hyperoxia and hypoxia can be harmful and many patients

resuscitated from CA have pulmonary injury requiring supplemental oxygen [5,12]. Indeed, many of our patients required supplemental oxygen to maintain normal PaO_2 values. Some studies in acute brain injury do not support the hypothesis that hyperoxia is detrimental. Bellomo et al found no association of hyperoxia with mortality in a multicenter study after cardiac arrest in adults[31]. Hypoxia, but not hyperoxia, was associated with worse outcome in general adult ICU patients who required mechanical ventilation[32]. Puccio et al found that 2 hours of normobaric hyperoxia (FiO_2 = 1.0) in adult patients with severe TBI did not promote oxidative stress or deplete antioxidant reserves in cerebrospinal fluid (CSF)[33]. Interestingly, using CSF samples from a RCT in children with severe TBI, Bayir et al found that hypothermia treatment preserved antioxidant reserves vs. subjects in the normothermic arm[34]. There is also animal research showing that hyperbaric oxygen delivered several hours following resuscitation is beneficial [35]. The authors speculated that early vulnerability to hyperoxia is secondary to exacerbating reactive oxygen species generation but later in recovery the concentration of ROS has diminished, antioxidant reserves have recovered, and supplemental oxygen has more benefit than risk. Thus, the early episodes of hyperoxia in our patient cohort are potentially injurious whereas it is unclear if the later episodes of hyperoxia are also injurious. Lastly, blood flow is heterogeneous following resuscitation and it is possible that regional tissue hypoxia occurs even when arterial PaO_2 is normal or increased [36].

Limitations. Study subjects were taken from an existing database of children with CA from 2000-2008. Pediatric Cerebral Performance Category scores were not available for patients in this database. A limitation of our study is that the sample size and heterogeneity of patients precludes testing for an association between hyperoxia and neurologic outcome. Similarly, the sample size and heterogeneity of patients limits the interpretation of our mortality data. Larger studies with adjustment for severity of illness are necessary to determine the effect of hyperoxia and hypoxia upon mortality and neurologic outcome. Another limitation is that blood gases were not obtained at all one-hour intervals and thus we are likely underestimating the occurrence of hyperoxia. There were also patients with more than one blood gas obtained in a one-hour interval; each of these “repeat” blood gases was obtained in response to hypoxia, none in response to hyperoxia. This suggests a clinical emphasis on avoiding hypoxia with less concern for hyperoxia. In this regard, it is noteworthy that Kilgannon showed hyperoxia to have a stronger association with worse outcome than hypoxia in adults recovering from CA[5].

Conclusion

This is the first pediatric study to show the frequency and outcomes of hyperoxia and hypoxia following resuscitation from CA. Both hyperoxia and hypoxia are commonly encountered after cardiac arrest and represent opportunities to improve patient care.

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Abbreviations

CA	cardiac arrest

ROSC	return of spontaneous circulation
ED	emergency department
PICU	pediatric intensive care unit
ABG	arterial blood gas
PaO_2	partial pressure of arterial oxygen
ECMO	extracorporeal membrane oxygenation
ROS	reactive oxygen species

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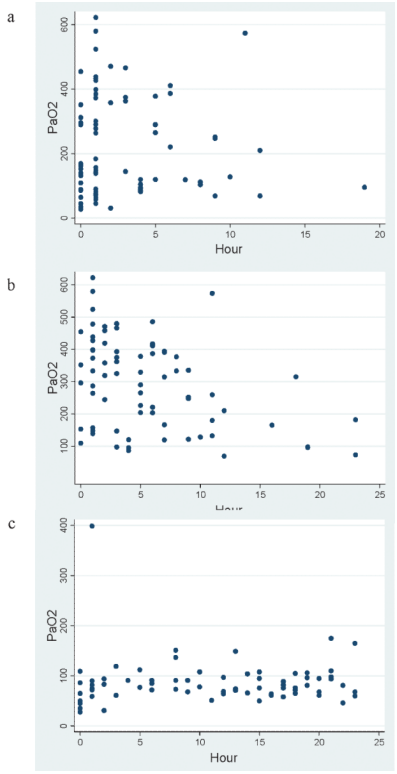

Figure 1a-c.
Graph depicts individual study patients initial PaO_2 (a) and highest (b) and lowest (c) PaO_2 and when they occurred in the first 24 hours after CA.

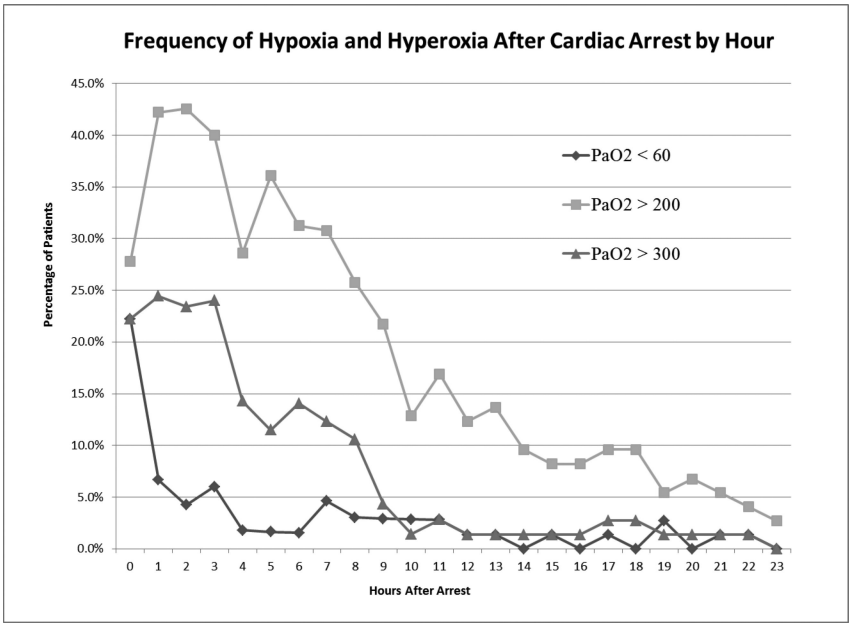

Figure 2.
Graph depicting the frequency of hypoxia and hyperoxia after pediatric cardiac arrest by hour.

Table 1

Patient and Cardiac Arrest Characteristics

Variable	No. of Patients (%)
Sex	
Male	44 (59)
Female	30 (41)
Race	
Caucasian	51 (69)
African American	16 (22)
Other/Unknown	7 (9)
Age	
Median (range) in years	1.8 (0-18)
Cause	
Respiratory	21 (28)
Shock (non-cardiogenic)	20 (27)
Drowning	11 (15)
Cardiac	9 (12)
Sudden Infant Death Syndrome	6 (8)
Non-accidental trauma	4 (6)
Neurologic	3 (4)
Location	
In-Hospital	31 (41)
Out-of-Hospital	43 (58)
Witnessed	54 (73)
Initial rhythm (n=69)	
Asystole	34 (46)
Pulseless Electrical Activity	22 (30)
Ventricular Arrhythmia	9 (12)
Sinus or Sinus Tachycardia	3 (4)
Unknown	6 (8)
Duration of Cardiac Arrest (n=66)	
Median (range), min	10 (1-45)
Survival at 6 months	53 (72)

Table 2

Logistic regression for mortality at 6 months

Variable	Coefficient	RR	p-value	95% CI
PaO2 < 60 mmHg	-0.611	0.543	0.386	0.136-2.160
PaO2 > 200 mmHg	-0.413	0.662	0.485	0.208-2.107
PaO2 > 300 mmHg	-0.058	0.944	0.911	0.343-2.596

RR, relative risk