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Early Oxygenation and Ventilation Measurements After Pediatric Cardiac Arrest: Lack of Association With Outcome

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Abstract

Objectives—To explore oxygenation and ventilation status early after cardiac arrest in infants and children. We hypothesize that hyperoxia is common and associated with worse outcome after pediatric cardiac arrest.

Design—Retrospective cohort study.

Setting—Fifteen hospitals within the Pediatric Emergency Care Applied Research Network.

Patients—Children who suffered a cardiac arrest event and survived for at least 6 hours after return of circulation.

Interventions—None.

Measurements and Main Results—Analysis of 195 events revealed that abnormalities in oxygenation and ventilation are common during the initial 6 hours after pediatric cardiac arrest. Hyperoxia was frequent, affecting 54% of patients. Normoxia was documented in 34% and hypoxia in 22% of patients. These percentages account for a 10% overlap of patients who had both hyperoxia and hypoxia. Ventilation status was more evenly distributed with hyperventilation observed in 38%, normoventilation in 29%, and hypoventilation in 46%, with a 13% overlap of patients who had both hyperventilation and hypoventilation. Derangements in both oxygenation and ventilation were common early after cardiac arrest such that both normoxia and normocarbia were documented in only 25 patients (13%). Neither oxygenation nor ventilation status was associated with outcome. After controlling for potential confounders, arrest location and rhythm were significantly associated with worse outcome; however, hyperoxia was not (odds ratio for good outcome, 1.02 [0.46, 2.84]; $p = 0.96$).

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Conclusions—Despite recent resuscitation guidelines that advocate maintenance of normoxia and normoventilation after pediatric cardiac arrest, this is uncommonly achieved in practice. Although we did not demonstrate an association between hyperoxia and worse outcome, the small proportion of patients kept within normal ranges limited our power. Preclinical data suggesting potential harm with hyperoxia remain compelling, and further investigation, including prospective, large studies involving robust recording of physiological derangements, is necessary to further advance our understanding of this important topic.

Keywords

hypercarbia; hyperoxia; hyperventilation; hypocarbia; hypoxia; resuscitation

Cardiac arrest (CA) affects approximately eight per 100,000 children in North America annually and causes high mortality and morbidity (1). Out-of-hospital (OH) CA has reported survival to hospital discharge of 2% to 12%, with intact neurological function in only 2% to 4% (1–3). In-hospital (IH) CA has slightly better outcomes with reported survival of approximately 25% and favorable neurological outcome in approximately 18% (4, 5). Treatment after CA is largely supportive and directed toward minimizing end-organ injury. Aside from rapid, effective cardiopulmonary resuscitation (CPR) and application of extracorporeal membrane oxygenation (ECMO) in select populations, no specific therapy has been shown to improve survival or outcomes after CA in children.

Supplemental oxygen therapy remains central to care during and after resuscitation. An association between hypoxia and poor outcome after CA has long been accepted. However, more recently, concerns about potentially detrimental effects of hyperoxia have been raised. Neonatal resuscitation reports show worse long-term outcomes with greater inspired concentrations of oxygen and higher oxygen saturations during resuscitation (6, 7). Similarly, some adult studies link hyperoxia with poorer outcomes (8, 9), although others do not (10).

The 2010 American Heart Association (AHA) Neonatal Resuscitation Guidelines recommend beginning resuscitation with room air for term infants (or a blended mixture of oxygen and air for preterm infants) with subsequent titration of inspired oxygen fraction based on pulse oximetry values (11). Similarly, the 2010 AHA Adult Advanced Cardiac Life Support Guidelines recommend initial resuscitation with 100% oxygen and then titration of inspired oxygen fraction after return of circulation (ROC) to target oxygen saturation greater than or equal to 94% (12, 13). Although investigations of hyperoxia and outcomes after CA in children are lacking, the 2010 AHA Pediatric Advanced Life Support Guidelines similarly recommend initial resuscitation with 100% oxygen and consideration of titrating oxygen delivery after ROC to achieve oxygen saturation greater than or equal to 94% (14).

Ventilatory support, another mainstay of resuscitation and supportive care, has also been linked with altered outcomes after CA. Hyperventilation reduces cerebral blood flow and is associated with hypotension during resuscitation (15). The 2010 AHA Adult Advanced Cardiac Life Support and Post-Cardiac Arrest Care Guidelines advocate avoiding hypocarbia (12, 13). Similarly, the 2010 AHA Pediatric Advanced Life Support Guidelines state that hyperventilation has no benefit and may be harmful (14). Reports of hyperventilation after CA in children are lacking; however, hyperventilation has been associated with adverse outcomes in children after stroke or traumatic brain injury (16–18) and in neonates after resuscitation from birth asphyxia (19).

In this study, we explore oxygenation and ventilation status early after CA in infants and children. Our primary hypothesis is that hyperoxia is common and associated with worse

outcome. To test this hypothesis, we conduct a retrospective cohort study involving children who suffered CA yet survived for at least 20 mins after ROC.

Methods

We queried a pediatric CA database that was generated during planning for two simultaneous multicenter, randomized controlled trials of therapeutic hypothermia after pediatric CA (the THAPCA Trials, http://www.thapca.org/) (20). The database, previously described in detail (21–23), was populated by retrospective review of pediatric CA events that occurred between July 1, 2003, and December 31, 2004, at 15 hospitals within the Pediatric Emergency Care Applied Research Network. All children 24 hours to 18 years of age (inclusive) who experienced CA with ROC for greater than or equal to 20 mins and were treated at a participating center were eligible for database inclusion. Patients cared for in a Neonatal Intensive Care Unit or who experienced planned CA as part of cardiac surgery were excluded from the initial cohort. The study populating the database was granted approval with waiver of consent at all participating studies. The current study met exemption criteria for the institutional review board, as it utilized an existing database that contained only de-identified data.

The database contains: 1) patient characteristics (e.g., demographics, chronic preexisting conditions); 2) event characteristics (e.g., location of CA, first recorded cardiac rhythm, presence and type of vascular access); 3) etiology of CA; 4) hospital course (e.g., use of ECMO, therapeutic hypothermia); 5) physiological and laboratory data; 6) Pediatric Cerebral Performance Category (PCPC) scores at baseline (pre-CA) and at hospital discharge; and 7) survival to hospital discharge. Both IH and OH CA events are included. CA is defined as receiving CPR with greater than 1 min of chest compressions. IH CA is defined as chest compressions initiated in an emergency department or other hospital setting and OH CA as chest compressions initiated prior to hospital arrival. ROC includes either spontaneous or assisted (e.g., ECMO) circulation. Duration of OH CA was poorly captured, but the number of epinephrine doses administered during CPR is available as a surrogate marker.

For the current study, we excluded: 1) subjects with a diagnosis related to cyanotic heart disease including any single ventricle physiology; 2) subjects lacking documentation of both an indwelling arterial catheter and at least one recorded Pao_2 value during the first 6 hrs post-CA; 3) subjects who died within the first 6 hours post-CA; and 4) subjects missing PCPC scores at baseline or at hospital discharge. We defined the following fields: preexisting condition as any identified chronic illness, CA etiology as respiratory or cardiac (nonexclusive), and rhythm during CA as presence of asystole, ventricular fibrillation/ventricular tachycardia, other, or missing (hierarchical). The location of CA was further refined for IH to have occurred in an ICU (IH-ICU) or another location (IH-other).

In the database, physiological and laboratory data are recorded as minimum and maximum values during select time periods. Because the risk of injury due to hyperoxia may be greatest early after reperfusion (24), our primary exposure of oxygenation status was from the period 0 to 6 hours post-CA. If there was only one value provided, it was assigned as both the minimum and the maximum values. The cutoff for hyperoxia, Pao_2 greater than 200 mm Hg (26.7 kPa), was chosen as a compromise between neonatal (6, 7) and adult (8–10) reports, which used cutoffs ranging from a pulse oximetry reading greater than 94% to a Pao_2 greater than 300 mm Hg (40 kPa); however, we also explored cutoffs of 100 mm Hg (13.3 kPa) and 300 mm Hg (40 kPa) by sensitivity analyses. The cutoff for hypoxia, Pao_2 less than 50 mm Hg (4.0 kPa), was chosen to roughly correspond to a pulse oximetry reading less than 80%. Other physiological and laboratory variables were categorized as

follows: hyperventilation as $Paco_2$ less than 30 mm Hg (4.0 kPa); hypoventilation as $Paco_2$ greater than 50 mm Hg (6.7 kPa); hypotension as systolic blood pressure less than fifth percentile for age (Pediatric Advanced Life Support criteria); hypothermia as temperature less than 35°C (excludes therapeutically induced hypothermia, which comprises a separate variable); fever as temperature greater than 38°C (25, 26); hyperglycemia as serum glucose concentration greater than 240 mg/dL (13.32 mmol/L) (27); and hypoglycemia as serum glucose concentration less than 40 mg/dL (2.22 mmol/L). Overall oxygenation status in the first 6 hrs post-CA was defined as hyperoxia (with no hypoxia), hypoxia (with no hyperoxia), both hyperoxia and hypoxia, and normoxia throughout interval. Overall ventilation status was similarly defined.

Our primary outcome variable was survival to hospital discharge with good neurological outcome. Good neurological outcome was defined by discharge PCPC score 1–2 (normal or mild disability) or no change in PCPC score between pre-CA and discharge. Conversely, poor neurological outcome was defined by discharge PCPC 3–6 (moderate or severe disability or death) or for those with an abnormal PCPC score pre-CA, any worsening. Hospital mortality was evaluated as a secondary outcome.

Statistical Analysis

For continuous variables, medians and interquartile ranges (25th–75th percentile) are reported. For categorical variables, counts and percentages are reported. The association of each variable with outcome was examined using the Wilcoxon's rank sum or Kruskal-Wallis test for continuous variables and chi-square or Fisher's exact test for categorical variables. We also described the association of oxygenation and ventilation status with key patient and CA characteristics. For the association of hyperoxia with outcome, sensitivity analyses were performed using cutoffs of Pao_2 greater than 100 (13.3 kPa) and Pao_2 greater than 300 (40.0 kPa), in addition to the defined cutoff of Pao_2 greater than 200 (26.7 kPa).

Multivariable logistic regression was used to evaluate the potential association between early (0–6 hrs) derangements in oxygenation and ventilation with outcome after pediatric CA. Factors that might confound the association between outcome and oxygenation and/or ventilation were examined. All variables with a p value less than 0.15 in univariable analysis were eligible for inclusion in the logistic regression model. Forward stepwise selection was applied, and factors were removed from the model if p values were greater than 0.05. In addition, based on reported associations with outcome after pediatric CA, we decided a priori to include the following in all models regardless of statistical significance: location of CA, patient age, number of epinephrine doses, and cardiac rhythm during CA. Similarly, based on our primary hypothesis, we decided a priori to include hyperoxia regardless of statistical significance. All analyses were performed using SAS 9.2 for Windows (SAS Institute, Cary, NC).

Results

Four hundred ninety-one pediatric CA events were available for analysis. After exclusions, a final cohort of 195 remained (Fig. 1). Hyperoxia occurred in 54% of the cohort, normoxia in 34%, and hypoxia in 22%. These percentages account for a 10% overlap of patients who had both hyperoxia and hypoxia. Ventilation status was more evenly distributed with hyperventilation observed in 38%, normoventilation in 29%, and hypoventilation in 46%. Patients with both hyperventilation and hypoventilation account for a 13% overlap. Derangements in both oxygenation and ventilation, such as hyperoxia and hypocarbia, were common. Surprisingly, both normoxia and normocarbia were maintained during the initial 6 hrs post-CA in only 25 of the 195 patients (13%).

Demographics and CA characteristics were not statistically different across oxygenation groups (Table 1). Interestingly, intubation prior to CA did not influence oxygenation status; the proportions of patients intubated prior to CA were similar across groups (hyperoxia: 45%, normoxia or hypoxia: each 41%, hyperoxia and hyperoxia: 40%, $p = 0.37$). Post-CA features did not differ by oxygenation group with the following exceptions (Table 1). Ventilation status differed in that the majority of hyperoxic patients were hyperventilated, whereas normoxic or hypoxic patients were most commonly hypoventilated. Approximately one-third of either hyperoxic or normoxic patients were normoventilated compared with less than 10% of hypoxic patients. ECMO support and spontaneous hypothermia were less frequent among normoxic than hyperoxic and/or hypoxic patients. PICU and hospital days post-CA were similar across oxygenation groups.

Demographics and CA characteristics were not statistically different across ventilation groups (data not shown). Intubation prior to CA was most common among hyperventilated patients (50%) and least common among normoventilated patients (37%), but the differences did not reach statistical significance ($p = 0.18$). Post-CA features also did not differ across ventilation groups with the exception of oxygenation status (data not shown). The associations for concurrent ventilation and oxygenation status are discussed above. PICU and hospital days post-CA did not differ by ventilation group.

About 37% of the cohort survived with good neurological outcome (Table 2). Outcome did not differ by demographics except that a preexisting condition was more common among those who survived with good outcome. Good outcomes were relatively more common among IH CA, cardiac etiology of CA, no documented asystole, and fewer epinephrine doses during resuscitation. Outcome did not differ by oxygenation or ventilation status. In contrast, poor outcomes were associated with spontaneous hypothermia and hyperglycemia.

Other physiological variables, including hypotension, ECMO support, hypoglycemia, fever, and therapeutic hypothermia, were not associated with outcome (data not shown). Interestingly, a greater documented change in body temperature during the initial 6 hrs post-CA was associated with poor outcome (median [IQR], 1.7 [0.6, 3.3] for poor vs. 1.1 [0.4, 1.8] for good outcome, $p = 0.002$). All patients who survived to PICU discharge survived to hospital discharge. Among survivors, 76% were discharged home, 17% were transferred to rehabilitation, and 7% were transferred to either another acute care facility or long-term care (data not shown).

Survival with good outcome was 34% for those with hyperoxia and 42% for those without ($p = 0.23$). This lack of association remained true in sensitivity analysis using cutoffs of 100 or 300 mm Hg (13.3 or 40 kPa). Hospital mortality was also similar between those with and without hyperoxia (56% and 52%, $p = 0.60$). Although OH CA, presence of asystole, greater number of epinephrine doses during CPR, development of spontaneous hypothermia, greater change in temperature during the first 6 hrs post-CA, and hyperglycemia were all associated with poor outcome in univariable analysis, only CA location and cardiac rhythm during CA remained significantly associated with outcome in the multivariable setting (Table 3). The adjusted odds of survival with good outcome were very similar for those with and without hyperoxia (odds ratio 1.02, 95% confidence interval 0.46, 2.27). This remained true when we excluded the 10 neonates in our cohort (odds ratio 0.925, 95% confidence interval 0.40, 2.12).

Discussion

Our most striking finding is that only 13% of pediatric patients were maintained both normoxic and normoventilated early post-CA. This is surprising, particularly given that

current AHA pediatric resuscitation guidelines advocate maintaining normal oxygenation and ventilation (14). Our study includes mainly resuscitation efforts at large, experienced pediatric centers. In addition, the location of CA (IH-ICU, IH-other, or OH) and intubation prior to CA were not associated with the frequency of normoxia and normoventilation. This suggests that although maintaining normal oxygenation and ventilation status is advocated, it remains difficult to achieve in practice throughout a range of resuscitation settings. Likely, factors associated with both resuscitation practices and patient physiology contribute to this. For example, hyperoxia likely results from administration of high FiO_2 in the absence of significant respiratory disease, whereas hypoxia likely stems from the presence of significant respiratory disease. Further, a study of resuscitation team performance during simulated pediatric CA events at a tertiary care, academic pediatric center revealed that hyperventilation was quite common during simulated resuscitation (28). A recent report of actual pediatric resuscitation events corroborated this, with hyperventilation being observed in 63% of resuscitations (29). Targeted efforts are needed to increase adherence with current oxygenation and ventilation guidelines.

Contrary to our primary hypothesis, we did not demonstrate an association between hyperoxia early post-CA and outcome. This differs from reports of worse outcomes in hyperoxic neo-nates with birth asphyxia (30–33) and from preclinical studies that suggest greater oxidative stress and worse functional and histological outcomes with hyperoxic vs. normoxic resuscitation (34–37). However, it falls in line with mixed reports of postresuscitation oxygenation status and outcomes in adults (8–10, 38).

The distribution of oxygenation status in our cohort differs from that reported in adults. Hyperoxia affected 54% of our cohort (includes isolated hyperoxia and combined hypoxia and hyperoxia) vs. 10% to 18% of adults (8, 10). Hypoxia occurred in 22% of our cohort vs. 63% to 74% of adults. Normoxia was maintained in 34% of our population vs. 16% to 19% of adults. These differences suggest that children likely have different responses to resuscitation and/or less severe hypoxic lung disease than adults. Given these differences, extrapolation of adult data regarding hyperoxia and outcomes after CA may not be appropriate.

We did not observe an association between ventilation status and outcome in our population. This differs from a study showing two-fold increased odds of poor outcome with hyperventilation (vs. normoventilation) after resuscitation from birth asphyxia (19) and from the reports of detrimental effects of hyperventilation in other types of pediatric acute cerebral injury (16–18).

Our report corroborates prior studies linking outcome after CA with arrest location (4, 39) or cardiac rhythm (5, 40, 41) and suggesting that post-CA hyperglycemia (27, 42) and spontaneous hypothermia (44) may be detrimental. Prior publications describe these associations in detail for the parent cohort of our study (21– 23). We also report a greater change in temperature during the first 6 hours post-resuscitation among those with poor outcome. Although it remains unclear whether spontaneous hypothermia is directly causal or merely associated with poorer outcome after CA, our findings support reports that the rate and degree of rewarming may be important (26, 43–45). Alternatively, our observations may simply reflect longer durations of CA, which could contribute to lower initial temperature, greater change with rewarming, and poorer outcomes. In either case, exacerbation of spontaneous hypothermia due to heat loss during transport and exacerbation of hyperglycemia should be avoided post-CA.

Strengths of our study include use of a robust, pediatric CA database that includes laboratory, physiological and outcome data from patients treated at 15 pediatric hospitals.

All included subjects had arterial blood gas data to classify oxygenation and ventilation status. This allowed more precise discrimination of oxygenation or ventilation status than use of respiratory rate or pulse oximetery readings.

Our study has several limitations that may have hindered our ability to demonstrate an association between hyperoxia and outcome after pediatric CA. First, the unanticipated small percentage of patients who were maintained normoxic and normoventilated limited our study power. Second, availability of physiological data as only maximum and minimum values during specified time periods, rather than all tested values, precluded precise temporal assessments. In addition, our focus on the earliest period postresuscitation (0–6 hrs) may not represent the most crucial period of exposure. Longer periods or different timing of exposure may be necessary to affect outcomes. Exposure to suboptimal oxygenation or ventilation prior to hospital arrival may not have been well captured for OH-CA, which would introduce heterogeneity regarding the types and durations of exposures and the time between ROC and initial blood gas measurement. Similarly, our categories of CA etiology are broad, necessarily introducing some heterogeneity and limiting our ability to address effects of suboptimal oxygenation or ventilation by more specific CA etiology. Our selection of patients who had an arterial catheter placed also likely selected for greater severity of illness, which may limit our ability to discern more subtle effects of hyperoxia. Finally, the data were collected retrospectively, adding the multiple limitations inherent to any retrospective database, including missing data for some variables.

Conclusions

Our study reveals that although recent resuscitation guidelines advocate maintenance of normoxia and normoventilation after pediatric CA, this is uncommonly achieved in practice. We did not demonstrate an association between hyperoxia and outcome after pediatric CA. However, due to the small proportion of patients kept within normal ranges, our sample size was limiting. The preclinical data suggesting potential harm with hyperoxia remain compelling, and further investigation, including prospective, large studies involving more robust recording of physiological derangements, is necessary to further advance our understanding of this important topic.

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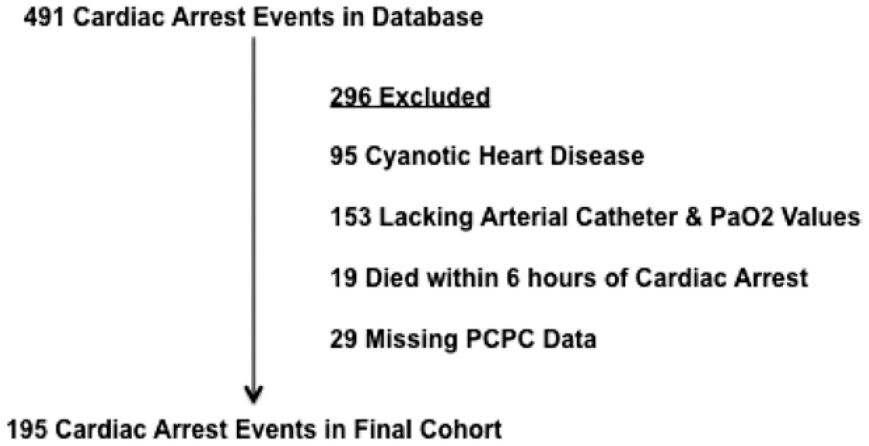

Figure 1.
Schematic of cohort selection. PCPC = Pediatric Cerebral Performance Category.

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Table 1

Cohort Characteristics by Oxygenation Status

	Hyperoxia *n* = 87 (%)	Normoxia *n* = 66 (%)	Hypoxia *n* = 22 (%)	Hyperoxia and Hypoxia *n* = 20 (%)	*p*
Demographics					
Age					
<2 yr	43 (49)	26 (39)	10 (45)	7 (35)	0.78
2–11 yr	28 (32)	24 (36)	9 (41)	8 (40)	
12–18 yr	16 (18)	16 (24)	3 (14)	5 (25)	
Gender					
Male	59 (68)	42 (64)	13 (59)	11 (55)	0.69
Female	28 (32)	24 (36)	9 (41)	9 (45)	
Preexisting condition					
Ye s	56 (64)	50 (76)	18 (82)	14 (50)	0.28
No	31 (36)	16 (24)	4 (18)	6 (30)	
Cardiac arrest details					
Location					
In-hospital in an ICU	31 (36)	34 (52)	11 (50)	10 (50)	0.34
In-hospital not in an ICU	26 (30)	15 (23)	3 (14)	5 (25)	
Out of hospital	30 (34)	15 (23)	8 (36)	5 (25)	
Etiology					
Cardiac	42 (48)	35 (53)	13 (59)	13 (65)	0.51
Respiratory	39 (45)	36 (55)	9 (41)	6 (30)	0.17
Rhythm					
Asystole	43 (49)	22 (33)	6 (27)	14 (70)	0.08
Ventricular tachycardia/ventricular fibrillation	7 (8)	5 (8)	3 (14)	1 (5)	
Other	27 (31)	27 (41)	9 (41)	3 (15)	
Epinephrine doses					
0	7 (8)	7 (11)	4 (18)	0 (0)	0.53
1	19 (22)	17 (26)	6 (27)	3 (15)	
2	14 (16)	12 (18)	4 (18)	4 (20)	
3	21 (24)	11 (17)	2 (9)	3 (15)	

	Hyperoxia *n* = 87 (%)	Normoxia *n* = 66 (%)	Hypoxia *n* = 22 (%)	Hyperoxia and Hypoxia *n* = 20 (%)	*p*
4+	23 (26)	18 (27)	4 (18)	9 (45)	
Postarrest features					
$Paco_2$					
Hyperventilation	29 (69)	10 (15)	6 (27)	3 (15)	0.001
Normoventilation	26 (30)	25 (38)	2 (9)	4 (20)	
Hypoventilation	19 (22)	29 (44)	8 (36)	8 (40)	
Hyperventilation and hypoventilation	13 (15)	2 (3)	6 (27)	5 (25)	
Extracorporeal membrane oxygenation support	11 (13)	4 (6)	4 (18)	9 (45)	<0.001
Spontaneous hypothermia	46 (53)	19 (29)	13 (59)	6 (30)	0.009
Hyperglycemia	46 (53)	31 (47)	10 (45)	9 (45)	0.99
Postarrest care					
PICU days, median (IQR)	5 (1,16)	6 (2,12.5)	11 (4,28)	6 (3,25.5)	0.33
Hospital days	9 (1,29)	7 (3,17)	16 (4,28)	7 (3,37)	0.53

Column percentages are shown. Numbers may not sum to 100 due to rounding.

Table 2
Cohort Characteristics by Outcome

	Total *n* = 195	Good Outcome *n* (%) 73 (37)	Poor Outcome *n* (%) 122 (63)	*p*
Demographics				
Age				
<2 yr	86	31 (36)	55 (64)	0.93
2–11 yr	69	27 (39)	42 (61)	
12–18 yr	40	15 (38)	25 (63)	
Gender				
Male	125	48 (38)	77 (62)	0.71
Female	70	25 (36)	45 (64)	
Baseline Pediatric Cerebral Performance Category				
1–2	161	64 (40)	97 (60)	0.15
3–5	34	9 (26)	25 (74)	
Preexisting condition				
Yes	138	59 (43)	79 (57)	0.02
No	57	14 (25)	43 (75)	
Cardiac arrest details				
Location				
In-hospital in an ICU	86	46 (53)	40 (47)	<0.0001
In-hospital not in an ICU	49	19 (39)	30 (61)	
Out of hospital	58	7 (12)	51 (88)	
Etiology				
Cardiac	103	48 (47)	55 (53)	0.003
Respiratory	90	35 (39)	55 (61)	0.57
Rhythm				
Asystole	85	14 (16)	71 (84)	<0.001
Ventricular tachycardia/ventricular fibrillation	16	9 (56)	7 (44)	
Other	66	40 (61)	26 (39)	
Epinephrine doses				
0	18	10 (56)	8 (44)	0.02
1	45	23 (51)	22 (49)	
2	34	15 (44)	19 (56)	
3	37	8 (22)	29 (78)	
4+	54	16 (30)	38 (70)	
Postarrest features				
Pao_2				
Hyperoxia	87	30 (34)	57 (66)	0.57
Normoxia	66	29 (44)	37 (56)	
Hypoxia	22	8 (36)	14 (64)	
Hyperoxia and hypoxia	20	6 (30)	14 (70)	

	Total *n* = 195	**Good Outcome *n* (%) 73 (37)**	**Poor Outcome *n* (%) 122 (63)**	***p***
$Paco_2$				
Hyperventilation	48	16 (33)	32 (67)	0.26
Normoventilation	57	27 (47)	30 (53)	
Hypoventilation	64	23 (36)	41 (64)	
Hyperventilation and hypoventilation	26	7 (27)	19 (73)	
Spontaneous hypothermia	84	20 (24)	64 (76)	<0.001
Hyperglycemia	96	25 (26)	71 (74)	0.002
Survival				
PICU discharge	89	73 (82)	16 (18)	
Hospital discharge	89	73 (82)	16 (18)	

Row percentages are shown.

Table 3
Multivariable Logistic Regression Results For Good Outcome

Variable	Adjusted Odds Ratio (95% Confidence Interval)	*p*
Hyperoxia	1.02 (0.46, 2.27)	0.96
Age category		0.85
<2 yr	Reference	
2–11 yr	1.13 (0.45, 2.84)	
12–18 yr	0.78 (0.25, 2.46)	
Arrest location		0.001
In-hospital in an ICU	12.3 (3.28, 46.1)	
In-hospital not in an ICU	9.87 (2.30, 42.4)	
Out of hospital	Reference	
Arrest rhythm		0.003
Asystole	0.23 (0.09, 0.57)	
Ventricular tachycardia/ventricular fibrillation	1.29 (0.30, 5.47)	
Other	Reference	
Epinephrine doses		0.32
0	Reference	
1	1.73 (0.39, 7.68)	
2	2.09 (0.41, 10.7)	
3	0.54 (0.10, 2.86)	
4+	1.32 (0.28, 6.19)	