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Near-infrared spectroscopy during cardiopulmonary resuscitation for pediatric cardiac arrest: A prospective, observational study

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Abstract

Aim: Cerebral oxygenation (rSO2) is not routinely measured during pediatric cardiopulmonary resuscitation (CPR). We aimed to determine whether higher intra-arrest rSO2 was associated with return of spontaneous circulation (ROSC) and survival to hospital discharge.

Methods: Prospective, single-center observational study of cerebral oximetry using near-infrared spectroscopy (NIRS) during pediatric cardiac arrest from 2016 to 2020. Eligible patients had ≥30 s of rSO2 data recorded during CPR. We compared median rSO2 and percentage of rSO2 measurements above *a priori* thresholds for the entire event and the final five minutes of the CPR event between patients with and without ROSC and survival to discharge.

Results: Twenty-one patients with 23 CPR events were analyzed. ROSC was achieved in 17/23 (73.9%) events and five/21 (23.8%) patients survived to discharge. The median rSO2 was higher for events with ROSC vs. no ROSC for the overall event (62% [56%, 70%] vs. 45% [35%, 51%], p = 0.025) and for the final 5 minutes of the event (66% [55%, 72%] vs. 43% [35%, 44%], p =

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Conflicts of Interest

The authors have no conflicts of interest to report.

0.01). Patients with ROSC had a higher percentage of measurements above 50% during the final five minutes of CPR (100% [100%, 100%] vs. 0% [0%, 29%], p = 0.01). There was no association between rSO2 and survival to discharge.

Conclusions: Higher cerebral rSO2 during CPR for pediatric cardiac arrest was associated with higher rates of ROSC but not with survival to discharge.

Keywords

Cerebral oximetry; Near-infrared spectroscopy; Pediatric cardiac arrest; Pediatric cardiopulmonary resuscitation

Introduction

Cardiac arrest occurs in up to 38,000 children per year in North America.[1–3] Return of spontaneous circulation (ROSC) occurs in 81% of patients with in-hospital cardiac arrest (IHCA) and 16–20% of patients with out of hospital cardiac arrest (OHCA) with survival to discharge rates of 40–50% and 5%, respectively.[4–7] Cardiopulmonary resuscitation (CPR) focuses on optimizing blood flow and oxygen delivery to the heart and brain with the goal of facilitating ROSC. Current guidelines recommend monitoring chest compression mechanics (e.g., depth, rate), invasive hemodynamics, and end-tidal CO2 to guide resuscitation and ensure high-quality CPR.[8] However, none of these modalities directly monitors the delivery of oxygen or blood flow to the brain. International Liaison Committee on Resuscitation and the American Heart Association have identified the need for pediatric specific data regarding whether NIRS guided CPR improves outcomes.[9]

Near-infrared spectroscopy (NIRS) non-invasively measures regional cerebral oxygen saturations (rSO2). Unlike a standard pulse oximeter, NIRS does not require pulsatile flow and thus can be used to measure rSO2 during low-flow or no-flow states such as cardiac arrest. During adult IHCA and OHCA, higher cerebral rSO2 values during the entire CPR event and specifically during the final five minutes of CPR are associated with higher rates of ROSC. Additionally, meeting a threshold rSO2 value of 50% is associated with increased odds of ROSC[10–11] and favorable neurologic outcome.[12] ROSC rarely occurred when cerebral rSO2 values were less than 30%.[13] There are limited data on whether cerebral oximetry can serve as a useful predictor of ROSC and survival to discharge in pediatric cardiac arrest.[14–16]

In this study, we aimed to evaluate whether higher rSO2 during CPR for pediatric cardiac arrest is associated with ROSC and survival to hospital discharge.

Methods

We conducted a prospective, single-center observational pilot study of cerebral oximetry using NIRS (Equanox 7600; Nonin Medical, Plymouth, MN, USA) during pediatric cardiac arrest from October 2016 to April 2020 at Children’s Hospital of Philadelphia. Patients were eligible if they suffered a cardiac arrest requiring CPR in the pediatric intensive care unit (PICU), cardiac intensive care unit (CICU) or emergency department (ED) and cerebral oximetry was recorded for at least thirty seconds during CPR. Patients were excluded if ROSC or death occurred prior to NIRS probe placement or if they received extracorporeal cardiopulmonary resuscitation (ECPR). The study was approved by our institutional review board and was exempt from informed consent for data collection.

NIRS probe placement

Our center has cardiac arrest monitoring infrastructure that includes 24/7 cardiac arrest research team notification systems, real-time CPR feedback, and standardized post-arrest care. Cardiac arrest notification in the intensive care units (ICUs) is based on a localized code blue system. Upon activation of the emergency response, a respiratory therapist not directly involved in resuscitation (e.g., ventilating or performing CPR) brought the NIRS machine to the patient’s room and placed the cerebral NIRS probe as rapidly as feasible. At least one NIRS probe placed on the forehead was adequate for data acquisition. If a NIRS probe was already in place for clinical care prior to the arrest, it was maintained but was not used to guide resuscitation as target values are not established. When newly placed intraarrest, the NIRS monitor was placed out of the resuscitation field in and not clearly visible to the resuscitation team.

Cerebral oximetry

Cerebral oximetry (rSO2) data were sampled continuously every four seconds (0.25 hertz). Data were recorded and stored on the NIRS device. Following completion of NIRS monitoring, a research assistant deidentified the NIRS data and uploaded it to our institution’s secure research server. Data were subsequently reviewed and cleaned by our research team for analysis. rSO2 data from each cardiac arrest event were manually reviewed by a study investigator (WPL) to identify artifact, periods of low signal quality, or noise. Both manual review and noise reduction filters were used to identify and classify these periods. Incomplete values were defined as data that were missing for each 4-second sampling period. If rSO2 data were recorded from bilateral cerebral NIRS probes in a single patient, they were averaged into one measurement. Using the available values for each patient, the median rSO2 was calculated for 1) the entire event and 2) the final five minutes of CPR. We calculated the percentage of rSO2 values above specific thresholds (30%, 40%, 50%, 60%) for each event for the entire monitored period and for the final five minutes of CPR.[11]

Patient characteristics

We collected demographic data (e.g., age, gender), pre-arrest characteristics (e.g., baseline pediatric cerebral performance category [PCPC] score,[17] pre-existing conditions), and cardiac arrest characteristics (e.g., cause of arrest, initial rhythm, medications administered). Intra-arrest data included continuous arterial blood pressure when available. Study data were managed using REDCap electronic data tools hosted at Children’s Hospital of Philadelphia.[18] Our primary outcome was sustained ROSC, defined as the return of a palpable pulse for at least 20 minutes. Our secondary outcome was survival to hospital discharge.

Intra-arrest blood pressure

For events in which invasive arterial blood pressure waveforms were available, waveform data underwent clinical review to identify starts and stops in CPR as previously described.[19–21] A custom code (MATLAB, MathWorks, Natick, MA, USA) was utilized to identify systolic blood pressure (SBP; peak of the arterial BP waveform) and diastolic blood pressure (DBP; sampled continuously between 60% and 70% of the distance between two consecutive SBP peaks) for each individual chest compression during CPR.[20] The event-level mean SBP and DBP were calculated for up to the first 10 minutes of CPR. The first 10 minutes were specifically evaluated to align with previously published studies on the prognostic value of DBP during pediatric CPR.[19] The average DBP for each event was characterized as being above or below established age-specific thresholds (≥25 mmHg in infants and ≥30 in older children).[19]

Statistical analysis

Continuous data are presented as mean (standard deviations) or median (interquartile ranges) if not normally distributed. Categorical data are presented as counts and percentages. Categorical values were compared using Fisher's exact or chi-squared test. T-test or Wilcoxon rank sum tests compared continuous variables.

For analyses evaluating associations between NIRS and event survival (ROSC), all CPR events were analyzed. For analyses evaluating associations between NIRS and survival to discharge, only the index event for each patient was analyzed. We compared median rSO2 and percentage of measurements above cut-offs for 1) the entire event and 2) the final five minutes of the event with the outcomes of ROSC and survival to discharge.

For events with blood pressure available, median rSO2 for the entire event and the final five minutes of the event was compared between events meeting and not meeting DBP thresholds.

Results

Twenty-one patients with 23 events were eligible. Median age was 1.67 years (0.42, 14) and 12 (57%) were female. Patient demographic and clinical characteristics are summarized in Table 1. Five patients survived to discharge.

Event-level data (n = 23) are summarized in Table 2. ROSC was achieved in 17 (73.9%) events. Two events began out of hospital with CPR continuing after arrival to the ED. Twenty events occurred in the PICU and one in the CICU. Two patients had acyanotic congenital heart disease. The most common causes of arrest were hypotension or shock in 8 (34.8%) and respiratory failure in 6 (26.1%). Patients who achieved ROSC received fewer epinephrine doses (2 [1,3] vs. 5 [4,8]; $p < 0.001$). NIRS probes were in place prior to arrest in 17 events. The median time to probe placement in events where probes were not in place prior to arrest (n = 6) was 5.5 [3.5, 14.5] minutes. The median duration of CPR with rSO2 data available was 11.2 [2.8, 17.1] minutes per event.

The median rSO2 value among all 23 events was 59% [45,68] (Table 3). The median rSO2 was higher for events with ROSC compared to no ROSC for the overall event (62% [56,70] vs. 45% [35,51], p = 0.025) and for the final five minutes of the event (66% [55,72] vs. 43% [35,44], p = 0.01). Patients who achieved ROSC had more rSO2 epochs above 50% during the final 5 minutes of the event (100% [100,100] vs. 0% [0,29], p = 0.01). There was no association between ROSC and the other thresholds evaluated (30%, 40%, 60%) for the overall event or for the final five minutes of the event.

Of the 21 patients, five (23.8%) survived to discharge (Table 4). The median rSO2 during the 21 index CPR events was 58% [45,65]. The median rSO2 in survivors was 68% [58,71] vs. 54% [42,65] in non-survivors (p = 0.09). There was no association between survival to discharge and the rSO2 cut-offs evaluated for either the overall event or the final five minutes of the event.

Arterial blood pressure and rSO2

Invasive arterial catheters were in place for 19 events. Of these 19 events, 8 had evaluable blood pressure data available. Age-based diastolic blood pressure targets during the first 10 minutes of arrest were met in 6/8 (75%) events. There was a significant difference in rSO2 between patients with DBP below target vs above target for the overall event (19% [15,23] vs 58% [44,70], p = 0.04) and during the final five minutes of the event (20% [15,24] vs 60% [51,73], p = 0.04).

Discussion

In this prospective single center study of cerebral oxygen saturation monitoring during pediatric cardiac arrest, patients who achieved ROSC had higher median intra-arrest cerebral oxygen saturations for the overall CPR event and during the final five minutes of the event than those who did not achieve ROSC. Patients who achieved ROSC also had a higher percentage of rSO2 measurements above 50% during the last 5 minutes of the event. In a small exploratory subgroup analysis, patients who achieved AHA recommended age defined DBP targets during CPR had higher rSO2 during the CPR event.

We found that the median rSO2 for the entire event and the final five minutes of the event were significantly higher in patients who achieved ROSC. These findings are similar to those of adult studies. Higher rSO2 in adults either at initiation of CPR or during resuscitation are associated with greater likelihood of ROSC.[10–11,22–25] Higher median rSO2 during both the entire and the last 5 minutes of resuscitation in adults has also been associated with ROSC.[11] Published pediatric data is more limited. In a small case series that evaluated ten pediatric patients with OHCA, Çağlar et al. found that patients who did not achieve ROSC spent more time with an rSO2 < 30%, but they did not identify an association between ROSC and rSO2 during the overall CPR event or final five minutes of CPR (16).

During CPR, higher coronary perfusion pressures are associated with increased likelihood of ROSC.[26] Higher coronary perfusion pressure values are associated with higher cerebral perfusion pressures and higher cerebral oxygen delivery.[27,28] Therefore, it is not surprising that ROSC is more common in patients with higher rSO2 during CPR as the higher rSO2 reflects superior oxygen delivery to the brain and likely to the heart as well. In a recent pre-clinical study, non-invasive diffuse optical monitoring was able to discriminate between animals with and without ROSC as early as 3 minutes into CPR.[29] The findings of this large animal study evaluating rSO2 align with our findings that non-invasively measured cerebral oxygenation is associated with ROSC. Our evaluation of the final five minutes is consistent with a large, multicenter adult study that showed that ROSC is more likely in patients with both a higher median rSO2 and more time spent above 50% during the last five minutes of CPR.[11]

We anticipated finding an association between rSO2 and survival to hospital discharge in this pilot study, because survival to discharge is likely more reflective of cerebral health than ROSC alone. We speculate that the failure to detect such an association was potentially related to low statistical power and only having five patients survive to hospital discharge in our study. While we did not find a significant association of rSO2 with survival to discharge, the median rSO2 values were 14% higher in those who survived showed a strong trend toward benefit with a p = 0.09. Furthermore, patients who survived to discharge had a median of 68% of rSO2 measurements above 60% compared to 0% of measurements reaching this threshold in non-survivors. These findings are consistent with several larger adult studies that demonstrated higher rSO2 values in patients who survived to discharge or survived with favorable neurologic outcome.[11–12,25]

We evaluated previously assessed rSO2 thresholds of 30%, 40%, 50%, and 60% and found that ROSC was associated with more time spent above an rSO2 value of 50% in the *final five minutes of the event*. This is consistent with the findings of Parnia and colleagues, who showed that meeting an rSO2 cut-off of 50% over the course of the entire CPR event and during the final five minutes of CPR discriminated between patients with and without ROSC (areas under the receiver operating characteristic curves of 0.75 and 0.74, respectively). Larger studies are necessary to more comprehensively evaluate the utility of specific rSO2 thresholds during CPR, but the potential to establish tangible rSO2 targets holds promise in making cerebral oximetry measurement a practical undertaking at the bedside.

In a small exploratory subgroup analysis evaluating the association of predefined invasive arterial blood pressure thresholds and rSO2, patients meeting these thresholds had higher rSO2 measurements over the course of the entire event and during the final five minutes of CPR. Hemodynamic-targeted resuscitation is associated with higher likelihood of ROSC, survival, and survival with favorable neurologic outcome in laboratory studies.[28,30–33] Arterial blood pressure, however, may not always be reflective of cerebral blood flow (CBF). For example, in preclinical studies, the cerebral physiologic response to vasopressors is heterogenous between animals and is even variable over the course of a single CPR event.[34–35] In a recent study in an animal model of pediatric IHCA, epinephrine increased cerebral blood flow and tissue oxygenation initially, but this effect decreased with repeated doses.[36] Thus, while cerebral blood flow and oxygenation correlate with systemic hemodynamics and CPR quality on average, systemic measurements are limited in the information they can provide regarding the brain. The ideal resuscitation method should aim to both facilitate ROSC and preserve cerebral health and thus could incorporate real-time assessment of rSO2 or other measures of cerebral physiology. Intra-arrest rSO2 monitoring to guide CPR quality and the timing of vasopressor administration should be evaluated. Currently, resuscitation guidelines advocate for CPR to be titrated to invasive blood pressure and end-tidal carbon dioxide targets, which are indirect correlates of cerebral perfusion. Understanding the association of CPR quality and cerebral rSO2 and how titration of CPR impacts cerebral oxygenation is needed to determine if targeted cerebral resuscitation will be feasible.

The study has limitations. We studied a small convenience cohort of patients who had NIRS placed during CPR and therefore included few patients with brief arrests where ROSC occurred before NIRS could be deployed. The rate of survival to hospital discharge in our study was only 24%, in contrast to the large Get With The Guidelines-Resuscitation registry where the rate of survival to hospital discharge was 35%.[5] Because deployment of the NIRS device requires time, our study disproportionately included patients with longer durations of CPR, therefore potentially biasing our study cohort to a population at risk of more severe hypoxic ischemic injury. However, patients with prolonged arrests are more likely to have neurologic morbidity and mortality, and therefore may be the most appropriate patients to target. Our study was a pilot study and did not have the sample size to evaluate outcomes such as survival to discharge and neurological outcome. Due to small sample size we were analyze the association of rSO2 trends and outcomes based on pre-arrest pathology or medications used. A larger study would be useful to explore the impact of these factors on the utility and interpretation of intra-arrest rSO2. We did not consistently collect CPR quality metrics such as chest compression depth and EtCO2 and therefore were not able to evaluate if these metrics are associated with cerebral oxygenation. Future studies should aim to include these metrics to assess the role of rSO2 during pediatric cardiac arrest.

Conclusion

In this prospective single center study, higher cerebral regional oxygen saturation during CPR for pediatric cardiac arrest was associated with higher rates of ROSC and higher rates of meeting intra-CPR blood pressure targets. Larger studies are needed to further characterize the relationship between cerebral oxygenation and survival and to evaluate whether rSO2 can be used to guide resuscitation.

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Table 1 –

Patient Demographic data (n = 21). N (%) and median (IQR).

Patient Demographics	Total N = 21
Events per patient	
1	20 (95)
2	0 (0)
3	1 (5)
Age (years)	1.67 (0.42, 14)
Sex	
Male	9 (42.9)
Female	12 (57.1)
Weight (kg)	11 (6, 42)
Race	
Black	6 (28.6)
White	6(28.6)
Other	6 (28.6)
Unknown	3 (14.3)
Ethnicity	
Hispanic	3 (14.3)
Non-Hispanic	16 (76.2)
Unknown	2 (9.5)
Pre-arrest conditions *	
None	5 (23.8)
Hypotension/hypoperfusion	4 (19)
Acute respiratory insufficiency	13 (61.9)
Chronic lung disease	4 (19)
Sepsis	3 (14.3)
Renal insufficiency	4 (19)
Myocardial dysfunction	10 (47.6)
Epilepsy / seizures	2 (8.7)
Immunosuppression	3 (14.3)
Pre-arrest PCPC	
1	14 (66.7)
2	2 (9.5)
3	4(19)
4	1 (4.8)
5	0 (0)

* Multiple selections possible

PCPC = Pediatric Cerebral Performance Category.

Table 2 –

Arrest characteristics and NIRS data for all cardiac arrest events;

Arrest Characteristics	**All events N = 23**	**ROSC N = 17**	**No ROSC N = 6**	**P-value**
Interventions in place at time of arrest*				
None	1 (4.4)	0 (0)	1 (16.7)	0.26
Mechanical ventilation	22 (95.7)	17 (100)	5 (83.3)	0.26
High frequency ventilation	5 (21.7)	4 (23.5)	1 (16.7)	1
Vasoactive infusions	8 (34.8)	6 (35.3)	2 (33.3)	1
Central venous access	16 (70)	12 (70.6)	4 (66.7)	1
Dialysis	4 (17.4)	4 (23.5)	0 (0)	0.54
Nitric oxide	4 (17.4)	4 (23.5)	0 (0)	0.54
Hospital CPR Location				0.059
PICU	20 (87)	16 (94.1)	4 (66.7)	
CICU	1 (4.3)	1 (5.9)	0 (0)	
ED	2 (8.7)	0 (0)	2 (33.3)	
Prior OHCA on this admission	5 (19.2)	4 (23.5)	1 (11.1)	0.63
Cause of arrest				0.31
Respiratory failure	6 (26.1)	5 (29.4)	1 (16.7)	
Hypotension/shock	8 (34.8)	6 (35.3)	2 (33.3)	
Ingestion/toxin	2 (8.7)	2 (11.8)	0 (0)	
Metabolic/electrolyte abnormality	1 (4.4)	1 (5.9)	0 (0)	
Seizure	2 (8.7)	2 (11.8)	0 (0)	
Pulmonary hypertensive crisis	2 (8.7)	1 (5.9)	1 (16.7)	
ALTE/SIDS	2 (8.7)	0 (0)	2 (33.3)	
Initial rhythm				0.91
Bradycardia with poor perfusion	6 (26)	5 (29.4)	1 (16.7)	
VF/pulseless VT	5 (21.7)	4 (23.5)	1 (16.7)	
Asystole	2 (8.7)	1 (5.9)	1 (16.7)	
Pulseless electrical activity	10 (43.5)	7 (41.2)	3 (50)	
CPR duration (minutes)	16 (7, 19)	10 (4, 19)	18.5 (18, 30)	0.056

Arrest Characteristics	All events N = 23	ROSC N = 17	No ROSC N = 6	P-value
Epinephrine doses				0.008
0	4 (15.4)	4 (23.5)	0 (0)	
1–2	8 (30.8)	7 (41.2)	0 (0)	
3–5	10 (38.5)	6 (35.3)	3 (50)	
>5	4 (15.4)	0 (0)	3 (50)	
Number of Epinephrine doses	3 (1, 5)	2 (1, 3)	5 (4, 8)	<0.001
Defibrillated	5 (21.7)	4 (23.5)	2 (16.7)	1
Airway during Arrest				0.39
LMA	1 (4.4)	0 (0)	1 (16.7)	
Pre-existing	19 (82.6)	14 (82.4)	5 (83.3)	
Intubated	3 (13)	3 (17.7)	0 (0)	
Arrest medications				
None	3 (13)	2 (11.8)	1 (16.7)	1
Bicarbonate	11 (47.8)	8 (47.1)	3 (50)	1
Calcium	10 (43.5)	8 (47.1)	2 (33.3)	66
Vasopressin	4 (17.4)	2 (11.8)	2 (33.3)	0.27
Lidocaine	4 (17.4)	4 (23.5)	0 (0)	0.54
Amiodarone	3 (13)	3 (17.7)	0 (0)	0.54
Steroid	1 (4.4)	1 (5.9)	0 (0)	1
Epinephrine infusion	5 (21.7)	2 (11.8)	3 (50)	0.09
Norepinephrine infusion	4 (17.4)	4 (23.5)	0 (0)	0.54
Dopamine infusion	3 (13)	3 (17.7)	0 (0)	0.54
Nitric oxide	7 (30.4)	5 (29.4)	2 (33.3)	1
Blood products	2 (8.7)	2 (11.8)	0 (0)	1
Monitoring during CPR				
Arterial line	19 (73.1)	13 (76.5)	6 (66.7)	0.66
EtCO2	18 (78.3)	13 (76.5)	5 (83.3)	1.oo
Zoll	15 (65.2)	10 (58.8)	5 (83.3)	0.37
EEG	3 (13)	3 (17.7)	0 (0)	0.53

VF: ventricular fibrillation, VT: ventricular tachycardia, ROSC: return of spontaneous circulation.

Table 3 –

Comparison of median rSO2 for all events by return of spontaneous circulation. (n = 23)

	Return of spontaneous circulation vs no ROSC (n = 23)			
Overall event (N = 23)	**All patients**	**ROSC (N = 17)**	**No ROSC (N = 6)**	**P-value**
rSO2 median (IQR)	59% (45,68)	62% (56,70)	45% (35,51)	0.025
Percent of measurements with rSO2:				
>30%	100% (99,100)	100% (99,100)	100% (89,100)	0.64
>40%	100% (85,100)	100% (93,100)	100% (4,100)	0.77
>50%	76% (24,100)	89% (68,100)	35% (0,100)	0.27
>60%	39% (0,68)	55% (0,68)	0% (0,0)	0.13
Last 5 minutes (N = 22)		**ROSC (N = 17)**	**No ROSC (N = 5)**	
rSO2 median (IQR)	64% (45,71)	66% (55,72)	43% (35,44)	0.01
Percent of measurements with rSO2:				
>30%	100% (100,100)	100% (100,100)	100% (100,100)	0.59
>40%	100% (100,100)	100% (100,100)	50% (0,100)	0.22
>50%	100% (29,100)	100% (100,100)	0% (0,29)	0.01
>60%	89% (0,100)	100% (0,100)	1% (0,0)	0.20

ROSC: return of spontaneous circulation.

Table 4 –

Comparison of median rSO2 and percent of measurements above rSO2 thresholds among index events by survival to discharge (n = 21).

Overall Event (n = 21)	All patients	Survival to hospital discharge N = 5	Non-survival N = 16	p-value
rSO2 Median (IQR)	58% (45,65)	68% (58,71)	54% (42,65)	0.09
Percent of measurements with rSO2:				
>30%	100% (99,100)	100% (99,100)	100% (95,100)	0.96
>40%	100% (85100)	100% (94,100)	100% (65,100)	0.85
>50%	72% (24,100)	89% (72,100)	68% (11,100)	0.40
>60%	31% (0,65)	68% (31,79)	0% (0,61)	0.12

Last 5 minutes of CPR event (n = 20)	All patients	Survival to hospital discharge N = 5	Non-survival N = 15	p-value
rSO2 Median	61% (45,68)	70% (55,71)	60% (43,66)	0.18
Percent of measurements with rSO2:				
>30%	100% (100,100)	100% (100,100)	100% (100,100)	0.83
>40%	100% (93,100)	100% (100,100)	100% (100,100)	0.60
>50%	100% (20,100)	100% (85,100)	100% (0,100)	0.69
>60%	42% (0,100)	83% (0,100)	1% (0,100)	0.88