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Clinical paper

A feasibility study of cerebral oximetry monitoring during the post-resuscitation period in comatose patients following cardiac arrest☆

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ABSTRACT

Background: One of the major causes of death and neurological injury after cardiac arrest is delayed ischemia combined with oxygen free radical mediated reperfusion injury. Consequently determining the optimal balance between oxygen delivery and uptake in the brain using a reliable non-invasive monitoring system during the post-resuscitation period is of importance. In this observational study, we evaluated the feasibility of using cerebral oximetry during the post-resuscitation period in order to identify changes in regional cerebral oxygen saturation (rSO_2) and its association with survival to discharge.

Methods: 21 consecutive patients admitted to the intensive care units following cardiac arrest had cerebral oximetry monitoring carried out for 48 h. Mean rSO_2 values were collected during the first 24 h and then again during the subsequent 24–48 h of the post-resuscitation period.

Results: 43% ($n = 9$) patients survived to hospital discharge and 57% ($n = 12$) died. Amongst all patients the median (IQR) rSO_2% was 65.5% (62.6–68.2) in the first 24-h following ROSC and increased to 72.1% (64.6–73.7) ($p = 0.11$) in the subsequent 24–48 h. The median (IQR) rSO_2% during the first 24 h in patients who survived to discharge compared to those who did not survive were significantly higher 68.2% (66.0–71.0) vs. 62.9% (56.5–66.0), $p = 0.01$). During the subsequent 24–48 h period, while a difference in the rSO_2 between survivors and non-survivors was noted, this did not achieve statistical significance (median (IQR): 73.7 (70.2–74.0) vs. 66.5 (58.2–72.1), $p = 0.11$).

Conclusions: Our study indicates that the use of cerebral oximetry is feasible during the post resuscitation period after cardiac arrest. Further studies are needed to determine whether cerebral oximetry may be used as a novel non-invasive monitoring system to evaluate changes in the balance between cerebral oxygen delivery and uptake during the post-resuscitation period.

1. Introduction

Brain injury following cardiac arrest is a major health burden and contributes to poor overall survival rates from this condition.[1] Among survivors, long-term neurological, cognitive and functional deficits are common.[2–4] These outcomes reflect the finale to a process of anoxic and ischemic brain injury that begins with the cessation of heartbeat and reflects a state of imbalanced oxygen delivery relative to tissue oxygen requirements, which when left unrecognized and untreated leads to progressive and irreversible brain damage.[5] While anoxic and ischemic brain injury is undoubtedly a complex condition, one of the hurdles limiting the ability to improve survival and neurological outcomes from a clinical perspective has been the lack of a real-time detection system that is capable of monitoring the quality of brain oxygen delivery relative to oxygen demands.

A non-invasive technology that has emerged that may be used during cardiac arrest is cerebral oximetry using near infrared spectroscopy (NIRS). In biological tissue, the use of NIRS is made possible due to the fact that while the tissues themselves are relatively transparent to near infrared light in the 400–1000 nm range, specific *chromophores* including oxy and de-oxyhemoglobin present in tissues absorb wavelengths of light in this spectrum.[6] Therefore, the attenuation of light at a given wavelength mainly occurs as a result of the light scattering in tissue, which is related to the properties of specific chromophores. NIRS measures the fall in optical

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intensity, expressed as the change in optical density per centimeter of regional brain tissue and thus provides a read out that corresponds with regional oxygen saturation (rSO_2).[7] It is a dynamic measurement that reflects real-time changes in the balance between oxygen delivery and uptake in the frontal lobe of the brain. The normal rSO_2 range is 60–80% due to the fact that the majority of blood in the frontal lobe regions of the brain where NIRS functions is venous.[6] We have already established the feasibility and potential clinical utility of incorporating cerebral oximetry using near infra-red spectroscopy (NIRS) during cardiac arrest.[8] However, in view of the continued brain injury processes that occur following ROSC, due to a combination of secondary ischemia and reperfusion injury combined with the clinical importance of optimizing the quality of oxygen delivery during this period, we examined the feasibility of incorporating cerebral oximetry during the first 24 and 48 h of the post-resuscitation period in order to look for an association between survival to hospital discharge and rSO_2 levels.

2. Methods

The study was approved by the local institutional review board (IRB). Following an earlier successful feasibility study, cerebral oximetry monitoring (Equanox 7600, Nonin Medical, Inc., Plymouth, MN, USA) has been incorporated into the care of adult patients suffering from cardiac arrest at Stony Brook University Hospital as a marker of the quality of resuscitation since August 2011. During a 6-month period from January to August 2012, we further evaluated this technology during the post resuscitation period by performing cerebral oximetry monitoring in patients following ROSC for 48 h. The cerebral oximetry sensor, consisting of an adhesive strip with two sets of a near-infrared light source and detector, were placed on the forehead of each patient admitted to the hospital intensive care units (ICU's) following ROSC. Continuous rSO_2 measurements were collected and downloaded in accordance with the manufacturer's instructions onto a computer and analyzed by calculating the mean rSO_2 during the first 24 h and during the subsequent 24–48 h period for each patient. Results were compared between patients who survived to hospital discharge and those who died in hospital. All consecutive patients who remained in a coma and were admitted to the ICU's during this period were included in this study. Patients who were placed on comfort care protocol or who had expressed a desire not to be resuscitated as well as those who had interruptions to cerebral oximetry monitoring for more than 12 h in any 24 h period were excluded. Patients who were not in a coma were also excluded. Continuous data were expressed as the median and interquartile range (IQR) using the Mann–Whitney test. Categorical variables were compared using a Fisher's test. Statistical analyses were performed using GraphPad Prism (GraphPad Software, Inc., La Jolla, CA). The relationship between rSO_2 and hemoglobin, mean arterial pressure (MAP), and the fractional oxygen (FiO_2) was assessed through Spearman's rank correlation coefficient. The data were analyzed with the assistance of a statistician.

3. Results

A total of 21 in hospital and out of hospital cardiac arrest patients who had achieved ROSC were admitted to the ICU's during our study period and had rSO_2 monitoring. Although all 21 patients had rSO_2 measurements during the first 24 h, only 15 patients also had rSO_2 measurements carried out during the subsequent 24–48 h period. This was due to the fact that three patients died in the first 24 h, while two recovered to such an extent that they were conscious and awake and thus had monitoring discontinued.

Table 1
Patient data M = male, F = female, IH = in-hospital arrests, OH = out-of-hospital arrests.

	Survivors (n = 9)	Non-survivors (n = 12)	P
Age[a]	69 (58–74)	63.5 (56–66.5)	= 0.34
M/F (n (%))	7 (78%)/2 (22%)	9 (75%)/3 (25%)	= 1.0
IH/OH (n (%))	5 (56)/4 (44)	8 (67%)/4 (33%)	= 0.67
Hypothermia (n (%))	5 (56%)	11 (92%)	= 0.12
Days of hospitalization[a]	22 (16–34)	2 (1.75–5.75)	= 0.002

[a] Age and days of hospitalization after cardiac arrest expressed as median (interquartile range).

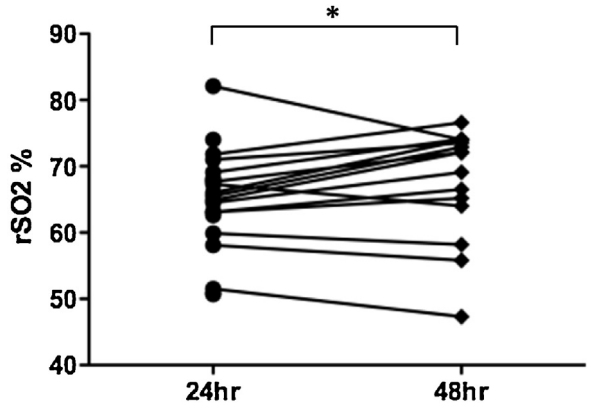

Fig. 1. The relationship between the mean regional, cerebral oxygen saturation (rSO_2) in the first 24 h, and 24–48 h after return of spontaneous circulation, (ROSC), expressed as the median and interquartile range, (IQR) of the mean rSO_2 in the first 24 h vs. 24–48 h were 65.5 (62.6–68.2) vs. 72.1 (64.6–73.7); *p = 0.11 using the Mann Whitney test.

Nursing staff also inadvertently discontinued rSO_2 monitoring in one patient. Overall, 43% (n = 9) survived to hospital discharge and 57% (n = 12) died. The mean (±SD) duration of monitoring during the first 24 h was 19.7 (±4) h and in the subsequent 24–48 h period was 18.7 (±5) h which was not statistically significant. A total of 21 sensors were utilized and there was no evidence of skin damage on any of the patients. Relatives and caregivers were fully accepting of the monitoring procedure. The mean (±SD) duration of interruptions to cerebral oximetry monitoring during the entire 48 h period was 6 (±6) h. These interruptions occurred due to a number of clinical reasons such as patients going out of the intensive care unit for radiological tests, the use of electroencephalography (EEG) monitoring or other transport issues. There were no significant differences between survivors and non-survivors with respect to age, gender, use of mild therapeutic hypothermia, or the proportion of in-hospital (IH) and out-of-hospital (OH) cardiac arrest patients (Table 1).

It was noted that while not statistically significant rSO_2 measurements trended upwards during the 24–48 h period compared with the first 24 h period following ROSC 72.1 (64.6–73.7) vs. 65.5 (62.6–68.2) (p = 0.11) (Fig. 1). There was a statistically significant difference in the median (IQR) rSO_2% measured during the first 24 h in patients who survived to hospital discharge compared to those who did not survive (68.2 (66.0–71.0) vs. 62.9 (56.5–66.0), p = 0.01) (Fig. 2A). However, while there was also a trend toward higher median (IQR) rSO_2 in survivors compared to non-survivors in the subsequent 24–48 h period, this did not reach statistical significance (73.7 (70.2–74.0) vs. 66.5 (58.2–72.1), p = 0.11)) (Fig. 2B). There was strong evidence to show a positive relationship between the first 48 h mean rSO_2 and first 48 h mean hemoglobin (rho = 0.56, p-value = 0.036). But no significant relationships existed between mean rSO_2 and mean MAP or between mean rSO_2 and mean FiO_2 (Fig. 3).