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Splanchnic cerebral oxygenation ratio decreases during enteral feedings in anemic preterm infants: Observations under near infrared spectroscopy

Katherine Braski¹, Kimberlee Weaver-Lewis², Manndi Loertscher¹, Qian Ding³, Xiaoming Sheng⁴, and Mariana Baserga¹

¹Department of Pediatrics, Division of Neonatology, University of Utah School of Medicine, Salt Lake City, Utah, USA

²Women's and Newborn's Clinical Program, Intermountain Healthcare, Salt Lake City, Utah, USA

³Study Design and Biostatistics Center, University of Utah, Salt Lake City, Utah USA

⁴Department of Pediatrics, University of Utah School of Medicine, Salt Lake City, Utah, USA

Abstract

BACKGROUND—Anemia is common in premature infants. Due to risks with red blood cell transfusions, many anemic infants are not transfused. The implications of this pathophysiologic status, especially at times of increased metabolic demand (enteral feedings), is not well understood. Near Infrared Spectroscopy (NIRS) allows for non-invasive determination of regional oxygen saturations (rSO₂) in tissues such as the brain and mesentery, giving insight into the tissues oxygen sufficiency.

OBJECTIVE—We tested the hypothesis that during enteral feedings, very-low birth weight (VLBW) infants with a hematocrit < 28% will have a decrease in splanchnic rSO₂ and splanchnic cerebral oxygenation ratio (SCOR).

METHODS—This prospective, observational, two-centered study, included VLBW infants receiving full enteral feedings with a hematocrit < 28%. Cerebral and splanchnic rSO₂ were monitored via NIRS for 24 hours. Average values were calculated for periods immediately preceding, during, and after each feeding. SCOR was calculated from these values (rSO₂ splanchnic/rSO₂ cerebral), and data was analyzed using a linear mixed effect model.

RESULTS—Fifty neonates with a median gestational age of 28 weeks (range, 23–32), a birth weight of 1118±284g (mean±s.d.), and a hematocrit of 26±2% (mean±s.d.) were studied. During feedings, SCOR decreased significantly from baseline (0.72±0.17 to 0.69±0.17; p=0.043). With feedings there was a trend of decreased splanchnic rSO₂ (47±11 to 45±10; p=0.057), and no change in cerebral rSO₂ (66±8 to 66±7, p=0.597).

Address Correspondence to: Katherine Braski MD, Division of Neonatology, Department of Pediatrics, University of Utah School of Medicine; 295 Chipeta Way, Salt Lake City, UT 84108 United States, Katherine.braski@hsc.utah.edu, Phone: 801-581-7052, Fax: 801-585-7395.

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Covidien provided one near infrared spectroscopy monitor for use during this study.

CONCLUSIONS—VLBW infants with a hematocrit $\geq 28\%$ had a decrease in SCOR and a trend towards decreased splanchnic rSO_2 with enteral feedings.

Keywords

Near Infrared Spectroscopy; Anemia; VLBW; Preterm Neonate

INTRODUCTION

Anemia of prematurity is common in the neonatal intensive care unit (NICU). Packed red blood cell (PRBC) transfusions are a frequent treatment for infants who develop clinically significant anemia. However, PRBC transfusions come with significant risks including a possible temporal association with the development of necrotizing enterocolitis (NEC).[1–6] Due to the risks associated with PRBC transfusions, an increasing number of premature neonates with anemia receive few if any PRBC transfusions and can remain anemic throughout their NICU hospitalization.[7] The implications of the pathophysiological status of prolonged anemia in the preterm infant is not well understood, especially at times of increased metabolic demand, such as enteral feedings. For instance, it has been reported that anemia can be associated with increased odds of developing NEC, independent of RBC transfusion.[4]

Near-infrared spectroscopy (NIRS) has been used in premature infants to evaluate changes in perfusion and oxygenation of body tissues including the brain and mesentery.[8–10] NIRS regional oxygen saturations (rSO_2) reflect a balance of tissue oxygen supply and demand, which may be altered by pathologic states such as anemia or NEC.[11] A prior study reported that mesenteric oxygen saturations (rSO_2) and superior mesenteric artery Doppler flow velocity increased concordantly with bolus feedings.[8] In a study utilizing a piglet model, low abdominal NIRS readings identified piglets in the process of developing NEC. The authors suggested that NIRS could be a means for early detection of NEC in human neonates.[12]

The splanchnic cerebral oxygenation ratio (SCOR) has been proposed as a means of predicting mesenteric ischemia in neonates. The SCOR compares the splanchnic oxygenation to cerebral oxygenation as a reference, because typically cerebral autoregulation minimizes changes in brain oxygenation during events that affect splanchnic oxygenation.[13] SCOR was used to assess stable premature neonates on full enteral feedings, and was found to be significantly increased following enteral feedings.[9] However, it is not known whether anemia affects splanchnic rSO_2 or SCOR during enteral feedings, a time of increased metabolic demand, in VLBW premature infants. Therefore we designed the present study to test the hypothesis that VLBW neonates with a hematocrit $\geq 28\%$ would have a decrease in splanchnic rSO_2 and SCOR during enteral feedings.

MATERIALS AND METHODS

Subjects

Neonates admitted to the University of Utah Hospital and Intermountain Medical Center NICUs were screened for study eligibility between Sept 2012 and Jan 2015. The study protocol and informed consent documents were approved by the IRB of both the University of Utah and Intermountain Health Care.

Prospective study patients were deemed eligible if born ≥ 32 weeks, and were ≥ 12 weeks of age at the time of the study, and were hemodynamically stable (no mechanical ventilation, vasopressors, or sepsis at the time of monitoring), and tolerating enteral feedings without intravenous fluid support. Eligibility criteria also included a hematocrit $\geq 28\%$ prior to monitoring. At the time of this study infants were typically not transfused until they reached a hematocrit of 25–30% for those on nasal cannula and 20–25% for those on room air, based on a transfusion protocol. Infants were ineligible if they had multiple congenital anomalies, or previous NEC (Bell's stage II disease).

NIRS Monitoring

After informed parental consent was obtained infants were monitored using an INVOS 5100 C-4 (Covidien, Dublin, Ireland) near infrared spectroscopy monitor. Neonatal probes were placed according to Covidien INVOS OxyAlert™ protocols on intact skin over the right or left frontal area (cerebral rSO_2) and the infra-umbilical area of the abdomen (splanchnic rSO_2). The probe positions were not altered for the duration of the monitoring. The infants were monitored continuously for 24 hours collecting data from cerebral rSO_2 and splanchnic rSO_2 . Bedside nursing staff noted the feeding times on the INVOS monitor and on the bedside flow sheet. No changes were made to the infant's routine care during the monitoring except as clinically indicated. Other events recorded by bedside nursing staff included: repositioning of the infant and obtaining the infants weight. None of the rSO_2 values were available to the treatment team and no clinical decisions were made using the data.

Data Collection

Clinical data was collected at the time of the study and then at the time of discharge, including birth weight, gestational age, APGAR scores, hematocrit prior to monitoring, and feeding type, volume, and length. Any episodes of NEC during the infant's hospital course were also recorded.

Following the 24 hour period of observation the rSO_2 data was downloaded for analyses. Mean rSO_2 (cerebral and splanchnic) were calculated for 30 minute periods prior to each feeding, for the duration of each feeding, and for a 30 minute period after each feeding. The Splanchnic-cerebral oxygenation ratio (SCOR) was calculated (rSO_2 splanchnic/ rSO_2 cerebral) for the baseline, feeding, and post feeding time frames for each feeding instance using the mean rSO_2 .

Statistical Analysis

Descriptive statistics were used to summarize the demographics and clinical characteristics of the patients. Mean $\pm$ standard deviation (s.d.) or median and range (min-max) were used to summarize continuous variables across observed time periods (baseline, feeding, and post feeding), and count and frequency were used for categorical values. Parametric correlation between the average change in SCOR during feedings vs. the average baseline value was calculated using Pearson's correlation coefficient.

Linear mixed effect models were used to assess if there was a statistically significant association between each outcome and observed feeding time. The outcome variables were defined as average SCOR, average cerebral rSO₂, and average splanchnic rSO₂ with the time of the observation (baseline, feeding, and post feeding), hematocrit as either continuous measure or binary variable, and gestational age as the predictor. Patients were included as a random effect to account for correlation of the outcomes between the different time points.

A sample size of 50 patients was selected to facilitate a reasonable enrollment period and was based on the number of < 32 week infants typically admitted to our site NICU's and rates of anemia.

Statistical analyses were performed with SAS 9.4 (SAS Institute Inc., Cary, NC, USA). All p-values were 2-sided and were deemed significant if < 0.05.

RESULTS

Fifty two patients were enrolled in the study, of which 2 were subsequently excluded; one because the patient did not meet inclusion criteria and on who had incomplete rSO₂ data. The remaining 50 completed the study and are included in the final data analysis.

Baseline demographic data are shown in table 1. All subjects were premature neonates with a median gestational age of 28 (range 23–32) weeks, and mean birth weight of 1118 $\pm$ 284g (mean $\pm$ s.d.). All infants were on full enteral feedings, without IV fluid support, and the majority were fed breast milk fortified to 24 kcal/oz. The average hematocrit at the time of study was 26.3 $\pm$ 1.6%. Three infants (6%) developed medical NEC during their hospitalization and none required surgical intervention.

The NIRS results from mixed effect models are shown in Table 2. The mean average baseline SCOR decreased significantly during feedings (-0.03 ± 0.01 , $p=0.043$). There was no significant change noted in the cerebral rSO₂. Even though it did not reach statistical significance there was a trend (-1.70 ± 0.88 , $p=0.057$) toward decreased splanchnic rSO₂ during feedings. There was no significant difference between baseline and post feeding SCOR (-0.003 ± 0.02 , $p=0.839$), cerebral rSO₂ ($+0.57\pm0.71$), and splanchnic rSO₂ ($+0.16\pm1.1$). The mixed effect model demonstrated no significant association between SCOR and gestational age at birth ($p = 0.748$) and hematocrit as a continuous variable ($p = 0.605$) or when divided into groups of Hct < 25 and Hct > 25 ($p=0.933$).

The average SCOR ranged from 0.26 to 1.51 with an interquartile range of 0.61–0.83. The change in SCOR during feeding was inversely related to baseline value ($r = 0.31$, $p=0.028$).

The infants with the lowest SCOR at baseline had the largest decrease in SCOR with feedings.

DISCUSSION

This study is the first to utilize NIRS to evaluate the effect of bolus feedings upon SCOR in stable VLBW (GA 32 weeks) neonates with a hematocrit 28%. The most important finding is that SCOR decreased during enteral feedings in this population. The SCOR change was secondary to a decrease in splanchnic rSO₂ with feedings, while cerebral rSO₂ remained unchanged from baseline measurements. Furthermore, following feedings there was no increase in splanchnic rSO₂ or SCOR when compared to baseline values.

The subject's baseline cerebral rSO₂ values were consistent with prior published values in preterm neonates with similar hematocrit levels, and somewhat lower than published values in babies with higher hematocrits.[14,15] Cerebral rSO₂ values demonstrated no significant change with feedings. However, in contrast to earlier study results, we found both a trend in decreased splanchnic rSO₂ and a significant decrease in SCOR with feedings [9,16]. A prior study by Dave and colleagues evaluating non-anemic preterm infants with NIRS demonstrated an increased SCOR of 0.08 with feedings. We demonstrated a decreased SCOR of 0.03 with feedings in anemic VLBW infants. The total difference between the SCOR increase noted in non-anemic preterm neonates and the SCOR decrease seen in anemic preterm neonates was 0.11.[9] Importantly, the mean hematocrit in the Dave study was 37% compared to the mean hematocrit of 26% in our study. The difference noted between these 2 studies suggests that preterm infants with anemia have less ability to cope with the increased metabolic demands associated with feedings. Whether this SCOR difference is solely related to the lower hematocrit is unknown, and other factors such as the elevation of our study center (4500 feet) should be considered.

Our study was conducted at elevation while prior studies were performed at locations near sea level. We speculate that if altitude played a significant role our baseline saturations and SCOR would have been lower than those seen previously. Our average splanchnic and cerebral saturations were found to be slightly higher at baseline than in previous studies [9], suggesting that altitude did not play a major role in the lower SCOR seen with feedings.

Importantly infants with the lowest SCOR at baseline also had the largest decrease in SCOR with feedings. We postulate that babies with lower baseline SCOR values experience more pathophysiologic effects secondary to anemia. Because of this they are less able to compensate during times of increased metabolic demand, such as enteral feedings, resulting in a SCOR decrease with feedings.

There was a large amount of inter-individual variability of baseline SCOR with a range of 0.26 to 1.51. The interquartile range was 0.61 to 0.83 which was similar to the interquartile ranges reported in prior studies of preterm infants.[9,17] SCOR values, in this study, were consistent with SCOR values seen in other premature infant cohorts but were lower than values seen in term infants. [9,18] A SCOR of < 0.75 and a splanchnic rSO₂ of < 56% have been shown to predict gut ischemia in neonates.[17,19] In this study, the SCOR values of

0.72 (baseline) and 0.69 (feedings) and the splanchnic rSO_2 values of 47% (baseline) and 45% (feedings) all fall below the boundary values for the prediction of splanchnic ischemia. [17] This suggests that preterm neonates with HCT $\geq 28\%$ have a baseline level of intestinal hypo-perfusion that could then worsen during enteral feedings, potentially increasing their risk of feeding intolerance or necrotizing enterocolitis.

Another important observation is that we did not find an association between the patient's hematocrit prior to NIRS monitoring and the change in SCOR. We speculate that this could be related to the different hematocrit levels at which babies develop symptomatic anemia. Typical clinical practice for transfusion treatment utilizes hematocrit cut-offs, but not all babies with a low hematocrit level have "symptomatic" anemia or improve with RBC transfusion.[20] Although additional studies are needed future use of NIRS could involve utilizing rSO_2 and SCOR trends to guide diagnosis of "symptomatic" anemia.

Three babies (6%) in our study developed medical NEC during their NICU hospitalization. One of these patients had a hematocrit of $\geq 28\%$ when NEC occurred. The SCOR change seen in this baby with feedings was -0.05 . This is slightly larger than the average SCOR change of -0.03 . Lastly, the incidence of NEC in our study population was similar to the incidence in our NICU during the same time period.

One limitation of our study is that matched control infants without anemia were not evaluated at our centers. Also the size of the SCOR change was relatively small but when the decrease was compared with the expected increase a larger change was observed. The clinical significance of this is not known as the incidence of NEC was unchanged in our cohort. Only hemodynamically stable premature infants were included in this study because of the concern that hemodynamic instability would impact splanchnic perfusion independent of feedings. This may have decreased the presence of abdominal pathologies in our cohort [21]. Another limitation is that splanchnic rSO_2 has increased variability and changes in NIRS signal quality compared to other body sites. This is often due to the changing nature of intestinal contents i.e. peristalsis, stool, and air [14,22]. Due to these changes in signal quality some feedings were not included in analysis: for each patient studied a range of 4 to 9 feedings were included. In our study there was 22% variability in splanchnic rSO_2 and 11% variability in cerebral rSO_2 . This is comparable with a prior study that demonstrated 16–23% variability in splanchnic rSO_2 , compared to 3–4% variability in cerebral rSO_2 . [22] The increased cerebral variability seen is likely due to the long observation period utilized in this study, as the observation period encompassed times of clinical change such as enteral feedings which were avoided in the prior study. To account for the high level of variability in splanchnic rSO_2 the splanchnic cerebral oxygenation ratio was utilized to improve the reliability of the splanchnic rSO_2 by comparing it to the cerebral rSO_2 which has low variability secondary to cerebral autoregulation. We also had some infants whose splanchnic rSO_2 values were higher than their cerebral rSO_2 values. The cause of this is unknown however could be related to changes in oxygen demand.

In summary, this observational study demonstrated that preterm neonates with hematocrit $\geq 28\%$ had a significant decrease in SCOR from baseline during enteral feedings suggesting that anemic neonates have less physiologic capacity to accommodate times of increased

metabolic demand. This could lead to increased intestinal hypo-perfusion and increased risk of feeding intolerance and necrotizing enterocolitis. Based on this data, we speculate that NIRS could be used as an additional tool to better inform clinicians about splanchnic oxygenation in patients at risk for NEC. For example, an infant with either no change or a decrease in SCOR during feedings could prompt further evaluation of feeding tolerance, and potential need for transfusion and/or erythropoietic agents. Additional studies are needed to determine the efficacy of NIRS as a tool to monitor infants at risk for NEC.

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Table 1

Demographic Characteristics

	(N=50)
Birth weight, mean $\pm$ s.d. (g)	1118 $\pm$ 284
Gestation, median (range) (weeks)	28 (23–32)
Female gender, N (%)	23 (46)
Type of gestation, N (%)	
Singleton	29 (58)
Twin	18 (36)
Higher order multiple	3 (6)
APGAR score 5 at 5 min, N (%)	10 (20)
Size for age, N (%)	
Appropriate	42 (84)
Small	7 (14)
Large	1 (2)
Intraventricular hemorrhage, N (%)	
Grade I	6 (12)
Grade II	5 (10)
Grade III/IV	0 (0)
Time to full enteral feedings, median (range) (days)	10 (6–44)
Postnatal age at time of monitoring, mean $\pm$ s.d. (days)	47 $\pm$ 12
Weight at time of monitoring, mean $\pm$ s.d. (g)	1926 $\pm$ 450
Feeding volume at time of monitoring, ml/kg/day $\pm$ s.d	140.2 $\pm$ 14.8
Patent Ductus Arteriosus, N (%)	6 (12)
small	6 (12)
medium/large	0 (0)
Feeding, N (%)	
Formula	9 (18)
Breast milk	41 (82)
Caloric density of feedings, N (%)	
20 kcal/oz.	1 (2)
22 kcal/oz.	2 (4)
24 kcal/oz.	42 (84)
27 kcal/oz.	5 (10)
Feeding Delivery, N (%)	
Gavage Feeding	45 (90)
Gavage + Oral Feeding	4 (8)
Oral Feeding	1 (2)
Ventilatory support, total days	
O2 therapy prior to discharge, mean $\pm$ s.d.	57 $\pm$ 38

	(N=50)
Mechanical ventilation, mean $\pm$ s.d.	10 $\pm$ 16

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Table 2

NIRS Data

	Baseline mean $\pm$ s.d.	Feeding mean $\pm$ s.d.	Difference (Baseline – Feeding) Estimate $\pm$ std. error ^a	p – Value	Post Feeding mean $\pm$ s.d.	Difference (Baseline – Post Feeding) Estimate $\pm$ std. error ^a	p- Value
rSO ₂ – Cerebral (%)	66 $\pm$ 7	66 $\pm$ 7	+0.30 $\pm$ 0.56	0.597	67 $\pm$ 7	+0.57 $\pm$ 0.71	0.422
rSO ₂ – Splanchnic (%)	47 $\pm$ 11	45 $\pm$ 10	–1.70 $\pm$ 0.88	0.057	47 $\pm$ 11	+0.16 $\pm$ 1.1	0.887
SCOR (ratio)	0.72 $\pm$ 0.2	0.69 $\pm$ 0.2	–0.03 $\pm$ 0.1	0.043	0.71 $\pm$ 0.2	–0.003 $\pm$ 0.02	0.839

Abbreviations: SCOR, splanchnic-cerebral oxygenation ratio; rSO₂, regional oxygen saturations

^a Difference, Estimate $\pm$ std. error calculated with linear mixed effect model