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Review Article

Cerebral near-infrared spectroscopy: a tool for assessing cerebral oxygen saturation and oxygen extraction and identifying the need for packed red blood cell transfusion in preterm infants: a narrative review

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ABSTRACT

Anaemia is frequently observed in preterm infants and can compromise oxygen delivery to vital organs, including the brain due to reduced oxygen carrying capacity of blood. Decisions about packed red blood cell (PRBC) transfusions are usually based on haemoglobin or haematocrit levels combined with the infant's overall clinical condition. However, haemoglobin alone doesn't directly reflect tissue oxygenation or extraction. Cerebral near-infrared spectroscopy (NIRS) offers a non-invasive bedside tool to measure regional cerebral oxygen saturation ($rScO_2$), while the balance between oxygen supply and cerebral utilization is estimated by fractional tissue oxygen extraction (FTOE). These parameters could provide meaningful physiological insights in anaemic preterm infants and facilitate inform decisions about the need for blood transfusion. Clinical studies demonstrate that NIRS measurements before and after transfusion typically shows increased cerebral oxygen saturation and decreased oxygen extraction, especially in infants with more severe anaemia or higher pretransfusion extraction. Nonetheless, the clinical decision regarding transfusion threshold may vary as there is significant variability due to differences in patient populations, NIRS devices, monitoring protocols, transfusion thresholds, and outcome measures. Although some observational studies indicate haemoglobin levels around 9.5–9.7 g/dL with higher oxygen extraction or lower saturation in the preterm infants, these should not be used as fixed transfusion points. Evidence suggests NIRS could serve as a supplementary tool for an individualized assessment rather than as a sole basis for transfusion decisions. Future prospective research is needed to establish standard NIRS thresholds and to determine if NIRS-guided transfusion improves neurological and clinical outcomes.

Keywords: Cerebral oxygen saturation, Fractional tissue oxygen extraction, Cerebral near-infrared spectroscopy, Preterm infant, Packed red blood cell transfusion, Anaemia, Infant transfusion

INTRODUCTION

Anaemia of prematurity is common in preterm infants compared to term babies. It is caused by multiple factors, such as repeated blood sampling in neonatal intensive care units, decreased erythropoietin production, rapid growth after birth, and the short lifespan of neonatal red blood cells. Packed red blood cell (PRBC) transfusions are often administered when anaemia is deemed clinically significant. Current transfusion guidelines recommend blood transfusion in preterm infant typically based upon infants haemoglobin or haematocrit levels, along with respiratory and cardiovascular condition and other clinical considerations. However, haemoglobin concentration mainly indicates oxygen-carrying capacity and does not directly assess whether tissue oxygen delivery is sufficient in each infant.[1-3] The preterm infant's brain is particularly vulnerable to lower oxygen delivery, with a cerebral oxygenation has a critical role in maintaining optimal oxygenation. Cerebral oxygenation determines from the interaction among haemoglobin concentration, arterial oxygen saturation, cerebral blood

flow, and cerebral oxygen consumption. As a result, two infants with the same haemoglobin concentration can differ in cerebral oxygenation according to their cardiorespiratory and hemodynamic status.[2,4]

Near-infrared spectroscopy (NIRS) is a non-invasive, continues bedside monitoring optical technique which offers real time information regarding regional tissue oxygenation. Cerebral NIRS estimates regional cerebral oxygen saturation ($rScO_2$), which represents the balance between cerebral oxygen delivery and utilization. Fractional tissue oxygen extraction (FTOE), derived from arterial oxygen saturation and $rScO_2$, confers corroborative data regarding the proportion of delivered oxygen extracted by cerebral tissue.[4,5]

The potential utility of NIRS in neonatal transfusion medicine has receiving growing interest. Several observational studies have reported post- PRBC transfusion changes in cerebral oxygen saturation and oxygen extraction.[6-10] However, the available evidence remains heterogeneous, and NIRS has not been established as an independent transfusion trigger. A systematic review of regional oxygenation and perfusion monitoring concluded that these techniques are promising for individualized transfusion assessment although current evidence remains insufficient to justify for incorporation into routine clinical practice.[1]

This narrative review consolidates the physiological principles of cerebral NIRS, the relationship between anaemia and cerebral oxygenation and extraction, the effects of PRBC transfusion on cerebral oxygenation, and the potential advantages and limitations of NIRS in patient-specific transfusion assessment.

PRINCIPLES OF CEREBRAL NEAR-INFRARED SPECTROSCOPY

NIRS uses near-infrared light to assess tissue oxygenation. This monitoring approach relies on differential light absorption and the modified Beer-Lambert principle. NIRS devices typically emit the near infrared light within 700–900 nm wavelength. The light penetrates the scalp and skull and interacts with chromophores within the underlying tissue. Oxygenated haemoglobin (O_2Hb) and deoxygenated haemoglobin (HHb) exhibit different absorption characteristics across this wavelength range. NIRS analyses the changes in light absorption to estimate regional cerebral oxygen saturation.[4,5] In contrast to pulse oximetry, which predominantly assesses arterial oxygen saturation, cerebral NIRS provide an assessment of a mixed vascular signal from arterial, capillary, and venous blood within the interrogated tissue volume. Consequently, $rScO_2$ should be interpreted as a regional tissue oxygenation measurement instead of directly measuring arterial or pure venous saturation.[4,5]

NIRS devices differ in algorithms, wavelengths, sensor configurations, and signal-processing methods. Although comparative studies of commercially available devices have demonstrated good correlations but clinically relevant differences in absolute $rScO_2$ values. Consequently, absolute $rScO_2$ values across devices should not necessarily be considered interchangeable.[11]

REGIONAL CEREBRAL OXYGEN SATURATION

Regional cerebral oxygen saturation ($rScO_2$) is an estimated proportion of oxygenated haemoglobin relative to total haemoglobin within the tissue volume interrogated by the NIRS sensor. It can be expressed conceptually as:

$$rScO_2 = O_2Hb / (O_2Hb + HHb) \times 100$$

Where,

$Rsco_2$: regional cerebral oxygen saturation

O_2hb: oxygenated hemoglobin

Hhb: deoxygenated hemoglobin

The obtained value reflects the balance between cerebral oxygen delivery and cerebral oxygen consumption. A reduction in oxygen delivery or an increase in oxygen consumption may lead to greater oxygen extraction and, a reduction in $rScO_2$, depending on the extent of the physiological compensation.[4,5]

Cerebral $rScO_2$ should not be interpreted as a standalone measure but considered alongside other clinical parameters. Arterial oxygen saturation, cerebral blood flow, haemoglobin concentration, blood pressure, carbon dioxide concentration, cardiac output, ventilation, metabolic activity, and other physiological factors such as vasomotor immaturity, patient size and complex cardiovascular changes during postnatal adaptation can also contribute to cerebral oxygenation.[4,5,12,13]

Reference ranges for cerebral $rScO_2$ in preterm infants may differ with gestational age, postnatal age, sensor configuration, and overall clinical condition. Alderliesten et al. evaluated cerebral oxygenation in 999 preterm infants during the first 72 hours after birth and observed that $rScO_2$ changes with gestational and postnatal age. They also identified differences between neonatal and adult-sized sensors, underscoring the importance of standardized monitoring methodology.[12] Similarly, McNeill et al reported variability cerebral regional oxygen saturation during the first weeks of life in stable preterm infants, highlighting that cerebral oxygenation is dynamic physiological parameter rather than a fixed value. He also demonstrates the variably may depend on the infant's gestational age.[13]

FRACTIONAL TISSUE OXYGEN EXTRACTION

Fractional tissue oxygen extraction (FTOE) provides an estimate of the fraction of arterial oxygen extracted by tissue. Cerebral FTOE (cFTOE) can be estimated using:

$$FTOE = (SaO_2 - rScO_2) / SaO_2$$

where SaO_2 represents arterial oxygen saturation and $rScO_2$ represents regional cerebral oxygen saturation.[4,12]

Hence, FTOE offers an additional insight to $rScO_2$. When oxygen delivery reduces, cerebral tissue may initially compensate by extracting a greater proportion of the available oxygen. This may result in an increase in FTOE preceding a substantial decline in $rScO_2$ occurs.[2,4] Alderliesten et al has also observed that cerebral fractional tissue oxygen extraction showed an inverse pattern relative to $rScO_2$.[12]

A cFTOE value of approximately 0.4 has been used in numerous studies as a marker of increased cerebral oxygen extraction. Andersen et al dichotomised very preterm infants based on pretransfusion cFTOE was below or above 0.4 and found that cFTOE decreased significantly following transfusion in those with elevated pretransfusion extraction.[7] Whitehead et al similarly described raised in cFTOE as an adaptive response to anaemia and observed an increase in cFTOE at haemoglobin level of 9.6 g/dl.[8]

However, a cFTOE value of 0.4 should be considered a physiological marker identified in the various studies rather than a universally validated clinical threshold for PRBC transfusion.[7,8]

ANAEMIA AND CEREBRAL OXYGENATION

Haemoglobin concentration is a key determinant of oxygen content of an arterial blood. A decline in haemoglobin concentration reduces the oxygen-carrying capacity of blood unless accompanied by compensatory changes in arterial oxygen saturation, blood flow, or oxygen extraction.[2,3] The preterm brain may initially adapt to reduced oxygen delivery by increasing cerebral blood flow and/or oxygen extraction. Consequently, cerebral oxygen consumption may be maintained even in the presence of the reduced haemoglobin concentration. However, with progressive anaemia this compensatory mechanism may eventually become insufficient, culminating in an increased cerebral oxygen extraction and potentially reduced cerebral oxygen saturation.[2,8]

Wardle et al assessed factors affecting cerebral fractional oxygen extraction in preterm neonates and demonstrated that cerebral oxygen extraction is influenced by haemoglobin concentration and hemodynamic variables. These findings suggest that cerebral oxygen extraction reflects the interplay between multiple physiological factors rather than anaemia alone.[2] Whitehead et al prospectively assessed 144 recordings from 39 infants born at ≤30 weeks' gestation. They observed a significant negative correlation between haemoglobin and cFTOE ($p \leq .001$) and identified a substantial increase in cFTOE at a haemoglobin concentration of approximately 9.6 g/dl.[8]

In a separate cohort of 68 preterm infants, Vesoulis et al. studied the relationship between progressive anaemia and cerebral saturation. Among transfusion-naïve infants, progressive anaemia was associated with declining cerebral $rScO_2$, with a haemoglobin threshold of approximately 9.5 g/dl identified for critical cerebral desaturation.[14] These findings provide the rationale for exploring NIRS alongside conventional haemoglobin-based assessment. However, these observational thresholds should not be interpreted as indicating that all preterm infants with haemoglobin concentrations around 9.5–9.6 g/dl warrant transfusion.[1,8,14]

EFFECT OF PRBC TRANSFUSION ON CEREBRAL OXYGENATION

PRBC transfusion increases circulating haemoglobin concentration and subsequently increases arterial oxygen-carrying capacity. If arterial oxygen saturation and cerebral blood flow remain relatively stable, the increase in oxygen content may decline the proportion of oxygen extracted by cerebral tissue.[6,7] Several studies have investigated this physiological response using NIRS. Bailey et al evaluated 30 symptomatic anaemic preterm infants and observed a significant increase in cerebral and splanchnic tissue oxygen saturation following PRBC transfusion. Cerebral rSO_2 increased from approximately 63% before transfusion to approximately 68% after transfusion ($p<0.001$).[9]

Van Hoften et al studied 33 preterm infants receiving 47 transfusions. Cerebral tissue oxygen saturation and FTOE were recorded pre-transfusion and at 1 and 24 hours post-transfusion. Weighted mean $rScO_2$ increased from 61% pre-transfusion to 72% post-transfusion, while FTOE decreased from 0.34 to 0.23. Infants with lower haemoglobin concentrations (below 6.0 mmol/l ~97 g/l) exhibited the greatest decrease in FTOE.[6]

Dani et al demonstrated that red blood cell transfusion significantly improved cerebral, splanchnic, and renal tissue oxygenation in anaemic preterm infants, accompanied by a reduction in fractional oxygen extraction (all $p<0.001$). These findings suggest that NIRS-derived regional oxygenation parameters may provide useful physiological insights into the response to transfusion and help assess whether oxygen delivery is adequate in anaemic preterm infants.[15]

Seidel et al studied 76 preterm infants and measured cerebral and peripheral regional tissue oxygenation before, during, immediately after, and 24 hours after transfusion. Cerebral and peripheral rSO_2 increased significantly following transfusion ($p<0.0005$ for both),

in the infants with lower pretransfusion cerebral saturation below 55%.[10]

Andersen et al evaluated 24 very preterm neonates during the first 24 hours of life. Among neonates with elevated pretransfusion cFTOE ≥0.4 experienced a significant reduction in cFTOE following RBC transfusion, whereas the similar response was not observed by low-extraction group. It dictates that early RBC transfusion favourably modifies cerebral oxygen kinetics in infants with elevated cFTOE, potentially mitigating risk of hypoxic brain injury.[7] The latest TOP-NIRS study (Chock et al) evaluated 101 infants during 237 transfusion events. Cerebral and mesenteric tissue saturation increased after transfusion, whereas cerebral and mesenteric FTOE decreased. These

changes were observed without a significant change in peripheral oxygen saturation, indicating that NIRS may provide tissue-level oxygenation information and that's how NIRS is useful for continuous and non-invasive assessment to measure the balance between oxygen delivery and consumption.[16]

Progressive anaemia may reduce oxygen-carrying capacity and increase cerebral oxygen extraction and decline in cerebral oxygen saturation, reflected by increased FTOE (Figure 1A).

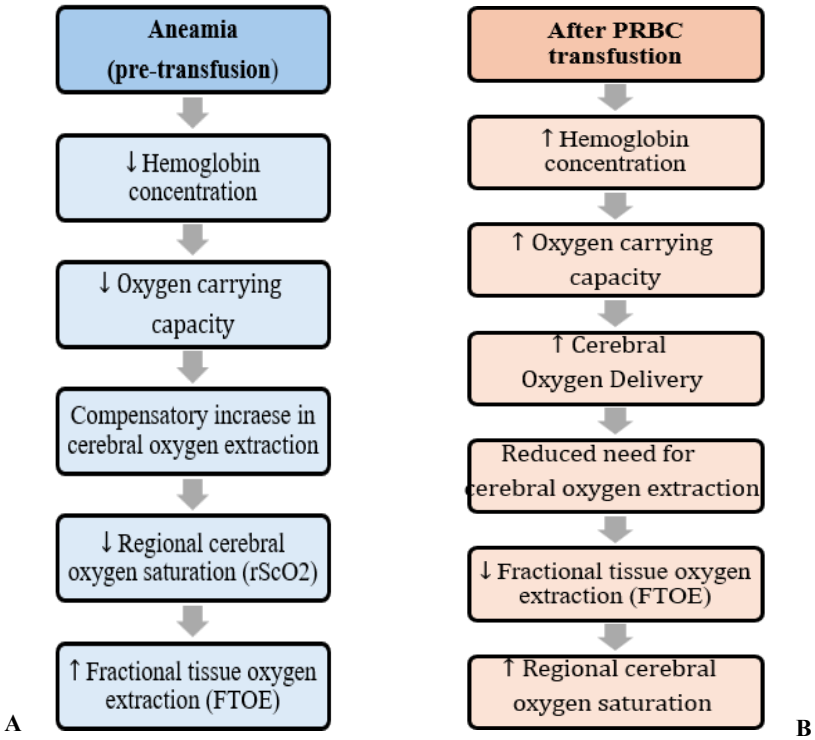


Figure 1: (A) Anaemia and cerebral physiological response at pre-transfusion (B) Anaemia and cerebral physiological response at post-transfusion.

Following PRBC transfusion, increased haemoglobin concentration may improve cerebral oxygen availability, resulting in reduced FTOE and increased $rScO_2$ (Figure 1B). This schematic represents physiological relationships and is not intended to serve as a stand-alone transfusion algorithm.

INSIGHTS FROM STUDIES OF NIRS AND PRBC TRANSFUSION

Selected studies investigating cerebral and regional oxygenation before and after PRBC transfusion are summarized in Table 1.

Table 1: Selected studies evaluating NIRS and PRBC transfusion in preterm infants.

Study	Population	NIRS parameter(s)	Transfusion/comparison	Principal findings
Wardle et al 2000[2]	Preterm neonates	Cerebral fractional oxygen extraction	Relationship with haemoglobin and hemodynamic variables	Cerebral oxygen extraction was modulated by anaemia and cardiovascular factors.
Bailey et al 2010[9]	30 symptomatic anaemic preterm infants	Cerebral and splanchnic rSO_2	Before and after PRBC transfusion	Cerebral and splanchnic oxygen saturation significantly increased after transfusion ($p<0.001$ for both)

Continued.

Study	Population	NIRS parameter(s)	Transfusion/comparison	Principal findings
van Hoften et al 2010[6]	33 preterm infants; 47 transfusions	$rScO_2$, FTOE	Before, 1 h and 24 h after transfusion	$rScO_2$ increased from 61% to 72%; FTOE decreased from 0.34 to 0.23 in pre-transfusion to post-transfusion respectively.
Dani et al 2010[15]	15 anaemic preterm infants (Hct <25%)	Cerebral, splanchnic and renal oxygen saturation and fractional oxygen extraction	Before/during RBC transfusion	RBC transfusion significantly increased cerebral, splanchnic, and renal tissue oxygenation and reduced regional oxygen extraction (all $p<0.001$)
Seidel et al 2013[10]	76 preterm infants	Cerebral and peripheral rSO_2	Before, during and after transfusion	Cranial and peripheral rSO_2 increased markedly ($p<0.0005$ for both); in infants with lower pretransfusion cerebral saturation (<55%).
Andersen et al 2015[7]	24 infants ≤29 weeks' gestation	cFTOE	Before and after early RBC transfusion	cFTOE decreased significantly after transfusion in infants with pretransfusion cFTOE ≥0.4 ($p<0.01$).
Whitehead et al 2020[8]	39 infants ≤30 weeks; 144 recordings	cFTOE	Relationship with haemoglobin	cFTOE increased as haemoglobin decreased; a significant negative correlation exists ($p\leq001$); with a critical increase in cFTOE occurred around haemoglobin declines to 9.6 g/dL.
Jani et al 2022[1]	Systematic review; 44 studies	Regional oxygenation/perfusion	Before and after transfusion	Regional oxygenation generally improved after transfusion, but substantial heterogeneity remained.
Chock et al 2023[16]	101 infants; 237 transfusion events	Cerebral/mesenteric rSO_2 and FTOE	Before and after transfusion	Cerebral and mesenteric saturation increased and FTOE decreased following transfusion.

POTENTIAL TRANSFUSION THRESHOLDS AND THE ROLE OF NIRS

Traditional neonatal transfusion management strategies are predominantly based on haemoglobin or haematocrit thresholds integrated with clinical assessment. These thresholds are based on population-level observations across studies.[1,3]

NIRS has thus been investigated as a potential complementary physiological tool to transfusion decision-making. The rationale is that an infant may maintain cerebral oxygenation despite declining haemoglobin by increasing oxygen extraction, whereas another infant with the same haemoglobin concentration may exhibit of impaired cerebral oxygenation. Thus, NIRS monitoring can identify cerebral hypoxia and hyperoxia, and hypocarbia-related changes to cerebral oxygenation.[1,2,8] Van Hoften et al observed the greatest reduction in FTOE following transfusion in infants with lower haemoglobin concentrations, with a substantial physiological response around 9.7 g/dl.[6] Whitehead et al reported a critical increase in cFTOE at approximately 9.6 g/dl, while Vesoulis et al identified approximately 9.5 g/dl as a threshold for critical cerebral desaturation in transfusion-naïve preterm infants.[8,14]

Pichler et al established reference ranges for $rScO_2$ and cFTOE during the first 15 minutes after birth in 381 clinically stable neonates using NIRS, showing a rapid increase in cerebral oxygenation and a corresponding decline in oxygen extraction during neonatal transition.[17]

A systematic review by Jani et al. included 44 studies evaluating regional oxygenation and/or perfusion monitoring following neonatal PRBC transfusion. Collectively, regional oxygenation generally improved after transfusion, with changes were observed more consistently when haemoglobin was below 9.6 g/dL or haematocrit was below 0.30. However, the review

identified substantial heterogeneity for patient characteristics, postnatal age at the time of monitoring, criteria for diagnosis of anaemia, period of monitoring and methodology for regional oxygenation monitoring.[1]

Taken together, the findings indicate that cerebral oxygenation or extraction may begin to change at haemoglobin concentrations above some conventional transfusion thresholds; however, it has not been established as haemoglobin threshold of 9.5–9.7 g/dl as a universal indication for transfusion in anaemia of prematurity.[1,8,14] Thus, NIRS should currently be considered a potential physiological adjunct, rather than standalone transfusion trigger.

DISCUSSION

The available evidence suggests a physiological basis for monitoring cerebral oxygenation in anaemic preterm infants. Haemoglobin concentration determines an important component of oxygen-carrying capacity, but tissue oxygenation is governed by an interplay among arterial oxygenation, haemoglobin, blood flow, and tissue oxygen consumption. NIRS provides an opportunity to monitor the resulting balance at the tissue level.[4,5]

Across several observational studies, it has been observed that; PRBC transfusion has generally been associated with increased cerebral oxygen saturation and decreased cerebral oxygen extraction. Findings from van Hoften et al, Bailey et al, Dani et al, Seidel et al, Andersen et al and the TOP-NIRS study are broadly consistent in demonstrating improved regional oxygenation or reduced oxygen extraction following transfusion. Most of the studies also dictate in improving post transfusion cerebral oxygenation in those infants with lower pre-transfusion cerebral saturation.[6,7,9,10,15,16] The response, however, is not uniform. The physiological effect of anaemia depends on several other parameters like gestational age, postnatal age, respiratory support, cardiovascular status, cerebral blood flow, metabolic requirements, and concurrent illness.[1,2,4]

The haemoglobin range around 9.5–9.7 g/dl is particularly interesting because several observational studies have identified changes in cerebral oxygen extraction or saturation in this range.[6,8,14] However, these observations should not be interpreted as evidence for a universal transfusion threshold. Rather, they support the framework that physiological markers may identify individual infants whose cerebral oxygen delivery is becoming compromised despite a haemoglobin concentration that may otherwise appear acceptable.[1,8] Hyttel-Sørensen et al showed the feasibility of early cerebral NIRS monitoring in extremely preterm infants, but also identified challenges in responding to prolonged hypoxia and the need for improved intervention protocols.[18] The systematic review by Jani et al highlights the current evidence gap. Although regional oxygenation and perfusion monitoring appear promising, methodological heterogeneity, physiological characteristics of infants and the predominance of observational studies, and lack of normative values in determining the validated threshold for critical oxygenation preclude firm recommendations for routine NIRS-guided transfusion.[1] Therefore, the most appropriate current role of cerebral NIRS is as a complementary physiological monitoring tool. Its future clinical value will depend on whether standardized NIRS-guided strategies can demonstrate improved patient outcomes in prospective clinical researches.

ADVANTAGES OF CEREBRAL NIRS

Cerebral NIRS offers several potential advantages in neonatal care. It is continuous, non-invasive and can be performed at the bedside without repeated blood sampling. It provides information regarding regional cerebral oxygenation by quantifying oxy and deoxy-Hb; and is adjunct to arterial oxygen saturation.[4,5] Another potential advantage is the ability to monitor trends over time. Continuous monitoring may identify changes in cerebral oxygenation associated with anaemia, respiratory instability, altered hemodynamic.[5,19] Therefore, a comprehensive assessment may consider: Haemoglobin concentration and its trend; Cerebral $rScO_2$ and FTOE and its trend with haemoglobin changes; Arterial oxygen saturation; Heart rate and blood pressure; Respiratory support and oxygen requirement; Cardiovascular and perfusion status; Clinical symptoms and overall condition; Evidence of impaired systemic or regional oxygen delivery; and Changes in NIRS measurements pre and post clinical interventions.[1,4,16]

NIRS may be particularly useful when the relationship between haemoglobin concentration and tissue oxygenation is ambiguous. For instance, an anaemic preterm neonate may maintain an apparently acceptable $rScO_2$ through increased oxygen extraction, resulting in a relatively high FTOE. Conversely, a low $rScO_2$ may indicate inadequate cerebral oxygenation even when haemoglobin concentration alone does not appear to be markedly reduced.[4,8] NIRS may also allow physiological response to PRBC transfusion to be assessed directly. Studies have generally demonstrated increased cerebral oxygen saturation and reduced oxygen extraction following transfusion, particularly in infants with greater anaemia or elevated pretransfusion oxygen extraction.[6-10,16] Serial NIRS monitoring may be more informative than a single measurement because cerebral oxygenation is dynamic and modulated by multiple physiological variables. Standardized data collection and interpretation are critical because differences in sensors, devices, signal processing, and artifact handling can affect measurements.[11,19] However, NIRS should not supersede clinical evaluation, laboratory measurements, or established neonatal transfusion guidelines. Its potential role is to provide ancillary physiological information when the clinical significance of anaemia is inconclusive.[1,16]

Limitations

Certain limitations should be considered before NIRS is incorporated into standard transfusion decision-making. First, NIRS measures regional rather than whole-brain oxygenation. A measurement from the frontal cerebral region may not accurately reflect oxygenation in all brain regions.[4,5] Second, NIRS measurements may be affected by extracerebral tissues, including the scalp and skull. Although device algorithms attempt to minimize this influence, heterogenicity in device technology and sensor design may yield clinically relevant differences in absolute $rScO_2$ values.[11]

Third, FTOE depends on both SaO_2 and $rScO_2$. Changes in either parameter can alter the calculated FTOE. FTOE should consequently be interpreted collectively with the underlying arterial saturation and holistic clinical condition.[4,12] Fourth, $rScO_2$ is influenced by multiple physiological variables. A low value may result from increased cerebral metabolic demand, reduced arterial oxygenation, significant hypoxia, reduced cerebral blood flow or combinations of these factors. Other confounding factors including presence of a hemodynamically significant patent ductus arteriosus, variability in blood pressure, partial pressure of carbon dioxide, sepsis, necrotizing enterocolitis, or intraventricular haemorrhage. Therefore, a low $rScO_2$ cannot be directly attributed to anaemia.[4,5,16]

Fifth, standardized approach to NIRS data acquisition and analysis remain challenging. Inconsistency in sensor placement, monitoring duration, artifact correction, data processing, and definitions of abnormal values contribute to heterogeneity among studies.[19] Moreover, variability in NIRS devices and sensor types, which may affect rSO_2 measurements and limit comparability across studies.[20] Finally, no universally validated $rScO_2$ or FTOE threshold independently indicates the need for PRBC transfusion. Therefore, NIRS should be used only as a supportive tool alongside clinical assessment and standard neonatal transfusion guidelines.[1,16]

CONCLUSION

Cerebral NIRS provides continuous, non-invasive bedside real-time clinical monitoring regarding regional cerebral oxygenation and may complement conventional assessment of anaemia in preterm infants. Parameters like $rScO_2$ and FTOE provide distinct yet related insights regarding the balance between cerebral oxygen delivery and oxygen utilization. Studies evaluating PRBC transfusion generally demonstrate increased cerebral oxygen saturation and reduced cerebral oxygen extraction following transfusion, particularly in infants with greater anemia or increased pretransfusion oxygen extraction. Although hemoglobin concentrations around 9.5–9.7 g/dL have been associated with changes in cerebral oxygenation or oxygen extraction in observational studies, these values should not be considered universal transfusion thresholds. Current evidence supports further investigation of NIRS as an adjunct for individualized transfusion assessment. Well-designed prospective clinical trials are needed to determine standardized NIRS thresholds and establish whether NIRS-guided transfusion strategies improve neurological and clinical outcomes.

Future research directions

Future studies should address whether NIRS-guided transfusion strategies improve clinically meaningful outcomes rather than merely demonstrating physiological changes following transfusion. Studies should also determine whether absolute $rScO_2$ values, FTOE, trends over time, or combinations of NIRS parameters with haemoglobin concentration provide the most useful information for transfusion decisions. Prospective multicentre trials should establish standardized NIRS protocols, including device selection, sensor placement, monitoring duration, artifact management, and definitions of clinically significant $rScO_2$ and FTOE abnormalities. Ultimately, standardization would improve comparability among studies and facilitate development of clinically applicable thresholds. The relationship between NIRS findings and long-term neurodevelopmental outcomes also warrants further research and clinical evidences. In the TOP-NIRS study, repeated transfusions associated with mean pretransfusion cerebral saturation below 50% were associated with death or neurodevelopmental impairment; however, this was an observational association. Future research should therefore move beyond physiological endpoints and determine whether individualized NIRS-guided transfusion strategies can safely reduce unnecessary transfusions while preventing inadequate tissue oxygen delivery.

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