Medtronic

Pediatric cardiac critical care

Cerebral oxygen saturation monitoring with the INVOS™ system

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Introduction

Congenital heart disease (CHD) is the most common birth defect, with an incidence of moderate to severe forms of 6 in 1000 live births.[1] Since the first surgical repair of congenital heart disease in 1953, the overall mortality in pediatric cardiac surgery has progressively decreased to <3%.[2] Despite this, surviving patients continue to display a relatively high incidence of neurological, developmental, and psychiatric disabilities.

During the early postoperative period following surgical repair of congenital heart defects, the vulnerable neonatal brain is subject to unstable hemodynamics with reduced systemic oxygen delivery (DO_2) at a time when oxygen demand (VO_2) is elevated.[3-5] Studies to date suggest that the first 12-24 hours after surgery represents a critical period for managing the risk of ischemic neurological injury. Reductions in cerebral tissue oxygenation during this time are associated with adverse neurodevelopmental outcomes and interventions designed to optimize hemodynamics and improve DO_2 may thus reduce neurological injury.[5-9]

In a cohort of 13 patients, Hoffman et al. demonstrated that decreased systemic oxygen delivery in the early postoperative period was significantly associated with hypoxic-ischemic brain injury and neurodevelopmental abnormalities while these adverse outcomes were not associated with either deep hypothermic circulatory arrest or cardiopulmonary bypass (CPB) time.[9] Similarly, in 22 neonates treated for the hypoplastic left heart syndrome (HLHS), 73% of patients had new or worsened ischemic lesions upon repeat MRI analysis and there was a significant association between prolonged reductions in postoperative cerebral oxygenation (<45% for >180 cumulative minutes) and the development of MRI lesions.[6] Additionally, long-term follow-up studies have indicated that reduced perioperative cerebral oxygenation is associated with impaired neurodevelopmental outcomes and brain abnormalities on MRI 4-6 years following surgery.[7,8] Together, these data indicate that perioperative reductions in cerebral oxygenation adversely impact neurodevelopmental outcomes and effectively managing the postoperative period may be critical for reducing the development or progression of neurological injury.

Key Learning Points

- Cerebral hypoperfusion is a common complication of complex thoracic aortic operations that adversely affects clinical outcome and the cost of care.
- Cerebral oxygenation and perfusion can be monitored continuously and noninvasively during thoracic aortic operations using the INVOS™ cerebral/somatic oximeter.
- Monitoring with the INVOS™ system may aid in the early detection of cerebral hypoperfusion or malperfusion.

Current clinical practice and challenges

With these data in mind, simply targeting mortality following congenital heart surgery is no longer an acceptable goal and current treatment guidelines now focus on improving neurodevelopmental outcomes. The early identification of perioperative factors that result in neurological injury allow for the rapid implementation of corrective strategies and improved outcomes. A key component to critical care monitoring of the pediatric cardiac surgery patient is the rapid and accurate assessment of cardiovascular function, cardiac output, and tissue oxygenation. Unfortunately, estimations of these variables based on routine clinical data such as the physical examination and standard hemodynamic parameters, such as heart rate, blood pressure, and central venous pressure, are often unreliable.[10-12] In children, there is a poor correlation between estimations of cardiac output and systemic vascular resistance based on peripheral pulses, capillary refill, and peripheral/core body temperatures and actual measurements of these parameters.[13,14] Blood pressure is also a poor correlate of cardiac output, as systemic vascular resistance rises to compensate for a decrease in perfusion.

The INVOS™ monitor

Near-infrared spectroscopy (NIRS) provides a non-invasive tool for continuous monitoring of regional tissue oxygen saturation (rSO_2).[15] NIRS analysis is based on the fact that hemoglobin has characteristic infrared absorption spectra that shift when bound to oxygen. This spectral shift permits the concentration and ratio of oxygenated to deoxygenated hemoglobin to be measured.

The INVOS™ cerebral/somatic oximeter employs disposable sensors with an integrated near infrared light source and photodetector that can be applied to each side of the forehead for monitoring blood in the brain or to other sites for monitoring tissue beneath the sensor. The INVOS™ oximeter uses 2 wavelengths (730 and 810 nm) of near-infrared light, which are emitted from a light emitting diode, and detected at 2 separate distances (3 and 4 cm) so that the signal arising from superficial tissues can be separated from the signal arising from the cerebral cortex (or somatic tissue of interest). More specifically, for cerebral oximetry, the 3 cm detector is designed to measure the signal generated by light passing though shallow structures such as skin, skull, and soft tissue while the 4 cm detector is designed to measure the signal generated by light passing through both these shallow structures and the frontal cerebral cortex. With this design, approximately 85% of the signal is derived from cortical tissue.[16]

In contrast to pulse oximetry, NIRS monitoring evaluates the non-pulsatile signal, reflecting the oxygen saturation of the microcirculation. Because the majority of blood in the microcirculation is venous (upwards of 75-80%), the oxygen saturation is used as a surrogate for venous oxygen saturations. The INVOS™ oximeter calculates the cerebral oxygen saturation using an algorithm that corrects the signal for extracranial noise and assumes a ratio of 25% arterial to 75% venous blood in the sampled tissue. The pediatric/neonatal INVOS™ system is designed for patients weighing <40 kg and differs from the adult system in that it incorporates a specific algorithm – taking into account the thinner skull and extracranial tissue of infants and children.[17]

Monitoring oxygen transport balance

Overall, NIRS analysis of rSO_2 reflects the balance between oxygen supply and demand and the ratio of oxyhemoglobin to total hemoglobin reflects the surplus of oxygen remaining after tissue extraction (see Figure 1 for summary). In brief, the Fick equation dictates that VO_2 is determined by the product of cardiac output (CO) and the arterial venous oxygen content difference ($CaO_2 - CvO_2$) or the volume of oxygen extracted per minute.[11] The Fick equation may be simplified to: $SaO_2 - SvO_2 / SaO_2 = VO_2 / DO_2$, where SaO_2 is the arterial oxygen saturation and SvO_2 is the venous oxygen saturation. Thus, by determining the O_2 extraction ratio ($SaO_2 - SvO_2 / SaO_2$) the relationship between DO_2 and VO_2 can be assessed.

The Fick Principle

$$VO_2 = CO \times CaO_2 - CvO_2$$

$$SaO_2 - SvO_2/SaO_2 = VO_2/DO_2 = \text{Oxygen Transport Balance}$$

$$\text{Oxygen Extraction Ratio (OER)} = SaO_2 - SvO_2/SaO_2$$

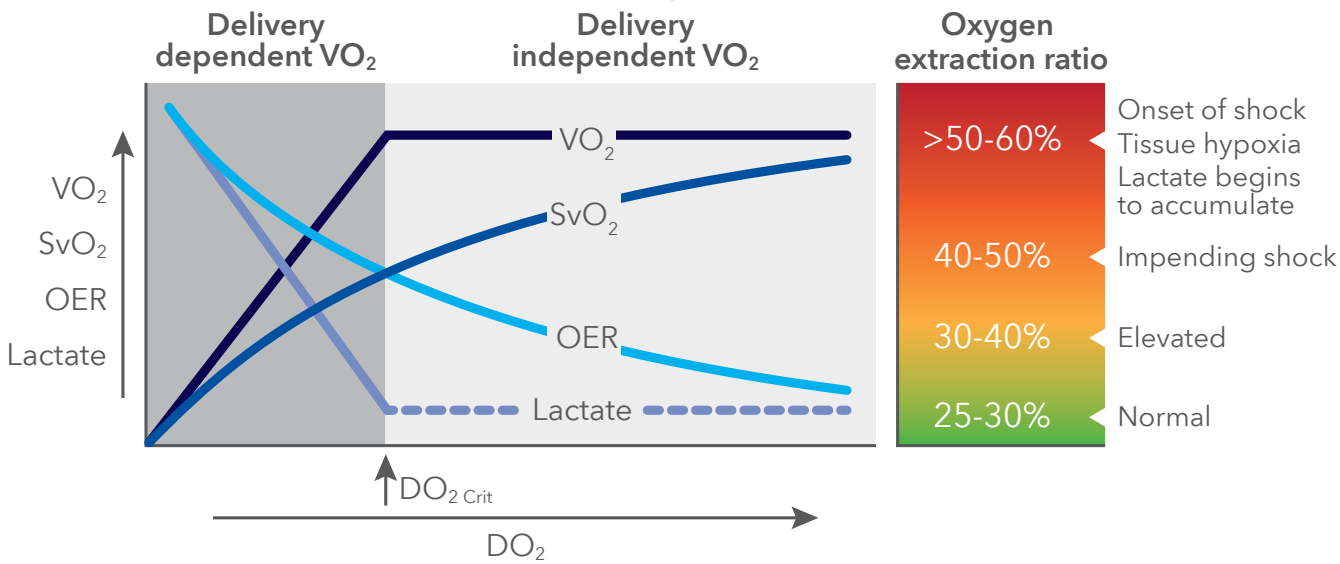

FIGURE 1. Physiological relationship between oxygen transport balance and oxygen extraction ratio. When oxygen content is decreased, either due to hypoxemia or anemia, or when O_2 demand is increased, global (increase in CO) as well as regional (vasodilation) compensatory circulatory mechanisms maintain oxygen delivery and a relatively stable regional and global oxygen extraction ratio. It is not until these compensatory mechanisms are exhausted that oxygen delivery falls and the oxygen extraction ratio begins to increase. As the oxygen extraction ratios rise above 50-60%, tissue hypoxia ensues and lactate begins to accumulate.

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Clinical value of perioperative oxygen saturation monitoring with INVOS™ technology

Over the past ~15 years, a number of studies have evaluated rSO_2 monitoring as a surrogate measure of either systemic or cerebral oxygenation in children with critical heart disease.[18-23] Overall, these studies have indicated that rSO_2 correlates well with jugular venous (SjO_2) and central venous oxygen saturations (SvO_2) (see Table 1).

Table 1. Summary of Key Studies Comparing rSO_2 with SjO_2 and/or SvO_2

Daubeney et al. (1996)	• Compared cerebral rSO_2 and SjO_2 in 40 children with diverse forms of congenital heart disease in the cardiac catheter laboratory or during cardiac surgery. • Across patients, the correlation between rSO_2 and SjO_2 was 0.69 ($p<0.0001$). • For surgery patients, correlations ranged from 0.78 to 0.96 with a median value of 0.91. • rSO_2 tended to overestimate SjO_2 at low SjO_2 values and tended to underestimate SjO_2 at high SjO_2 values.
Tortoriello et al. (2005)	• Compared cerebral rSO_2 with SvO_2 in 20 children following surgical repair of congenital heart disease. • rSO_2 was well correlated with SvO_2 across patients ($r=0.67$ and $r2=0.45$; $p<0.0001$ for both). • The overall bias for rSO_2 as a predictor of SvO_2 was 3.3% with a precision of 16.6%; rSO_2 was a more accurate predictor of SvO_2 in biventricular repair patients (bias = -0.3%, precision = 11.8%).
Li et al. (2006)	• Evaluated cerebral and somatic rSO_2, VO_2, and systemic hemodynamic and oxygen transport variables in 11 children undergoing the Norwood procedure. • Cerebral rSO_2 was correlated with SvO_2 ($r=0.43$), SaO_2 ($r=0.61$), PaO_2 ($r=0.99$), and MAP ($r=0.52$; $p<0.0001$ for all); somatic rSO_2 showed similar but weaker correlations.
Nagdyman et al. (2008)	• Compared cerebral rSO_2 with jugular bulb oxygen saturation (SjO_2) and central SvO_2 in 31 children with congenital heart defects. • Cerebral rSO_2 was well correlated with SjO_2 ($r=0.83$, $p<0.0001$) and SvO_2 ($r=0.93$, $p<0.0001$). • The mean bias was -5.2% for rSO_2 and SjO_2 and 5.6% for rSO_2 and SvO_2.
Ricci et al. (2010)	• Retrospectively compared SvO_2 and rSO_2 in 100 newborn patients between 24 hours preoperative and 72 hours postoperative for cardiac surgery. • The correlation between rSO_2 and SvO_2 was 0.37 ($p<0.001$); when analyzed by time (pre/postoperative), the correlations were 0.24 ($p=0.05$) for preoperative, 0.34 ($p<0.01$) for early postoperative, and 0.50 ($p<0.001$) for late postoperative. • During the preoperative period, rSO_2 tended to overestimate SvO_2 (bias = 1.5); during the early postoperative period, rSO_2 tended to underestimate SvO_2 (bias = -1.2). • The correlation between average rSO_2 values at consecutive time points and average SvO_2 at the same time points was 0.8 ($p<0.005$).
Moreno et al. (2013)	• Compared SvO_2 and rSO_2 (cerebral and flank) in 23 newborns and infants younger than 45 days undergoing heart surgery. • The mean difference (±SD) between SvO_2 and cerebral rSO_2 was 10.45 ± 14.12%. • The mean difference (±SD) between SvO_2 and somatic (flank) rSO_2 was 7.16 ± 16.67%. • The correlation between SvO_2 and cerebral rSO_2 was 0.48 ($p<0.001$); for SvO_2 and somatic rSO_2, the correlation was 0.35 ($p<0.001$).

Table 2. Summary of Key Cerebral INVOS™ Studies

Dent et al. (2005)	• Twenty-two neonates with HLHS were studied to compare preoperative and postoperative brain MRI findings. • Continuous cerebral oxygen saturation monitoring with INVOS™ technology was employed with a critical rSO_2 value of 45% used to indicate cerebral desaturation. • 73% of patients had new or worsened ischemic lesions upon repeat MRI analysis. • 70% of patients had prolonged (>180 minutes) cerebral desaturation (rSO_2 <45%) during the postoperative period. • There was a significant association (p=0.029) between prolonged postoperative cerebral low rSO_2 (>180 cumulative minutes <45%) and the development of MRI lesions, with a sensitivity and specificity of 82% and 75%, respectively. • The positive predictive value of rSO_2 for new MRI lesions was 90% and the negative predictive value was 60%.
Li et al. (2008)	• Sixteen neonates undergoing the Norwood procedure were studied to examine the effects of systemic hemodynamics and O_2 transport variables on rSO_2 during the first 72 hours postoperatively. • rSO_2 was 66% at baseline, 88% at the end of cooling, 90% during SACP, 64% during rewarming, 54% after separation from CPB, and 51% on arrival in ICU. • By 72 hours postoperative, rSO_2 was 56%, which was still significantly below baseline (p=0.02). • Overall ICU rSO_2 values ranged from 27% to 79% with 28% of readings lower than 48%. • rSO_2 was positively correlated with SBP, DBP, MAP, Qs, Qp, and DO_2; rSO_2 was negatively correlated with ERO_2 and SVR. • Overall, critical rSO_2 values were seen frequently in these patients and changes in rSO_2 were significantly related to changes in systemic hemodynamics and oxygen transport variables. • Hemodynamic interventions to modify systemic O_2 transport may help reduce the risk of cerebral ischemia and improve neurodevelopmental outcomes.
Horvath et al. (2010)	• Thirty-six postoperative children who had undergone delayed sternal closure after cardiac surgery were studied to determine if rSO_2 correlated with indicators of hemodynamic compromise. • Cerebral and somatic (renal) rSO_2 was reduced after delayed sternal closure suggesting mild and transient hemodynamic compromise after sternal closure.
Bronicki et al. (2013)	• Twenty-three patients were monitored for 30 minutes before and after extubation following repair of tetralogy of Fallot to determine the effects of extubation and loading of respiratory muscles on the distribution of cardiac output. • With extubation, central venous oxygen saturation increased from 65% to 70% (p=0.003) and cerebral rSO_2 increased from 67% to 72% (p=0.0001). • Before extubation, 4 patients were below or near the critical cerebral rSO_2 value of 50%. • rSO_2 data suggest that the brain was in or approaching an oxygen supply-dependent state with a subgroup of patients near or below critical rSO_2 values.
Hansen et al. (2013)	• Thirty-two patients with congenital heart disease who underwent superior cavopulmonary anastomosis were studied to assess the clinical value of NIRS monitoring. • Cerebral rSO_2 was 44% at the end of surgery and reached a minimal value of 40% at 2 hours postoperative. • Cerebral rSO_2 gradually increased during the postoperative period to 57% by the end of the 48 hours postoperative monitoring period and increased following extubation (49% before, 53% after). • Cerebral rSO_2 was moderately correlated with SvO_2 and SaO_2 (r=0.686 and r=0.547, respectively; p<0.001 for each). • Postoperative complications were observed in 7 patients and the mean cerebral rSO_2 values during the first 4 hours postoperative were lower in patients with complications compared to those without (29% vs. 45%; p<0.001). • Reduced postoperative cerebral rSO_2 may be predictive of postoperative complications.

Table 2. Summary of Key Cerebral INVOS™ Studies (cont'd.)

Hoffman et al. (2013)	• Twenty-one patients who had undergone stage 1 palliation of HLHS were studied to determine if early postoperative rSO_2 was related to later neurodevelopmental performance. • Patients with low to abnormal visual-motor integration scores had significantly reduced postoperative rSO_2 (64% vs. 68%; $p<0.05$). • The time at rSO_2 <45% and <55% was related to low visual-motor integration and neurodevelopmental index scores. • Perioperative cerebral rSO_2 monitoring can detect hypoxic-ischemic conditions associated with neurological injury and reduced neurodevelopmental performance. • Efforts to avoid cerebral hypoxia are likely to improve outcomes in this high-risk population.

Cerebral oxygen saturation monitoring with INVOS™ technology

Oxygen saturation monitoring with INVOS™ technology may aid the clinician in the detection of inadequate cerebral perfusion. Inadequate cerebral perfusion may lead to neurological injury and adverse outcomes. A number of studies have examined the clinical value of cerebral oxygen saturation monitoring with INVOS™ technology in the postoperative congenital heart patient.[5,6,8,24-27]

In general, these studies have suggested that these patients are highly susceptible to reduced cerebral oxygenation and that reductions in cerebral rSO_2 are associated with increased incidence of ischemic lesions, neurological injury, and adverse neurodevelopmental outcome. Postoperative manipulations designed to improve cerebral perfusion and thus rSO_2 may help reduce the likelihood of poor clinical outcome. Key studies describing the use of perioperative cerebral oxygen saturation monitoring with INVOS™ technology in the congenital heart patient are shown in Table 2.

Cerebral oxygen saturation monitoring with INVOS™ technology as an indicator of global / systemic tissue oxygenation

Cerebral NIRS oximetry may be used as a surrogate for mixed or central venous oxygen saturations and in doing so used to assess the global oxygen supply and demand relationship. However, the correlation between cerebral NIRS oximetry and central or mixed venous oxygen saturations may vary. Changes in $PaCO_2$ / pH "uncouple" cerebral blood flow from metabolism, altering cerebral oxygenation irrespective of changes in systemic hemodynamics. In a low cardiac output state blood flow is redistributed to maintain perfusion of the most vital organs such as the brain. Thus, cerebral rSO_2 may underestimate the degree of global disturbances in the oxygen supply and demand relationship based on an assessment of central or mixed oxygen saturations.

Somatic oxygen saturation monitoring with INVOS™ technology

In addition to cerebral monitoring, rSO_2 monitoring of peripheral tissue may provide insight into the global balance between oxygen saturation supply and demand. The rationale behind this strategy lies in the fact that as CO falls and becomes limited, blood flow is redistributed away from less vital organs (egs., renal and mesentery) to maintain perfusion of the most vital organs. As a result, renal and mesenteric oxygen saturations fall earlier and to a greater extent than cerebral oxygen saturations. A number of studies have used somatic monitoring (abdominal or splanchnic and renal) to demonstrate peripheral changes in tissue oxygenation following congenital heart surgery.[26-31] In general, these studies have indicated that perioperative monitoring of somatic oxygen saturation with INVOS™ technology in the congenital heart patient may allow for an even earlier appreciation of a falling CO. Key studies describing the use of INVOS™ technology in this setting are shown in Table 3.

Incorporating INVOS™ into clinical practice

Existing clinical experience and published studies support the benefits of incorporating oxygen saturation monitoring with INVOS™ technology into the routine perioperative management of pediatric cardiac critical care patients. The INVOS™ monitor provides an easy to use, noninvasive, real-time measurement of cerebral/somatic oxygen saturation. Overall, strategies to improve dropping INVOS™ values focus on improving oxygen balance by increasing perfusion pressure and/or arterial oxygen content and reducing oxygen demand (see Table 4).[32]

Limitations of NIRS oximetry

While NIRS oximetry appears to provide a valuable tool for real-time analysis of tissue oxygenation, there remain several unanswered questions regarding the overall clinical utility of rSO_2 monitoring. In a recent study comparing

Table 3. Summary of Key Somatic INVOS™ Studies

Kaufman et al. (2008)	• Twenty neonates and infants were studied within 48 hours of cardiac surgery to determine the correlation between abdominal NIRS oximetry and gastric tonometry. • Evaluation of anterior abdominal and renal rSO_2 revealed strong correlation between abdominal rSO_2 and intramucosal gastric pH (r=0.79; p<0.0001) as well as between abdominal rSO_2 and SvO_2 (r=0.89; p<0.0001). • There was a strong negative correlation between abdominal rSO_2 and serum lactate (r=-0.77; p<0.0001).
Chakravarti et al. (2009)	• Twenty-three children were studied to determine the relationship between rSO_2 and blood lactate levels following surgery. • Measures included cerebral, splanchnic, renal, and muscle rSO_2 monitoring for 24 hours postoperatively with blood lactate levels measured at 0, 2, 4, 6, and 24 hours. • Cerebral rSO_2 showed the strongest negative correlation with blood lactate (r=-0.74; p<0.0001) followed by splanchnic, renal, and muscle (r=-0.61, -0.57, and -0.48, respectively [all p<0.0001]). • The correlation between rSO_2 and blood lactate was further improved by averaging cerebral and renal values (r=-0.82; p<0.0001). • Averaged cerebral and renal rSO_2 ≤65% predicted a lactate level ≥3.0 mmol/L with a sensitivity of 95% and specificity of 83% (p=0.0001). • Averaged cerebral and renal rSO_2 predicts hyperlactatemia in acyanotic children after congenital heart surgery. • Noninvasive, continuous oxygen saturation monitoring may facilitate the identification of global hypoperfusion due to low cardiac output.
Owens et al. (2011)	• Forty infants undergoing biventricular repair were studied to determine if persistent low renal rSO_2 was associated with acute kidney injury. • Continuous renal rSO_2 monitoring was employed for 48 hours postoperatively. • Subjects with low renal oximetry (<50% for >2 hours) had significantly higher postoperative peak creatinine levels (0.8 vs. 0.5; p=0.003) by 48 hours along with higher incidence of acute kidney injury (50% vs. 3%; p=0.003). • Patients with reduced renal oximetry required more ventilator days and greater vasoactive support and had higher lactate levels. • Prolonged reductions in renal oxygen saturation appeared to correlate with renal dysfunction, decreased systemic oxygen delivery, and overall postoperative course.
Bronicki et al. (2013)	• Twenty-three patients were monitored for 30 minutes before and after extubation following repair of tetralogy of Fallot to determine the effects of extubation and loading of respiratory muscles on the distribution of cardiac output. • Mesenteric rSO_2 fell from 74% to 72% (p=0.04); there were no changes in renal rSO_2. • Following extubation, the presumed increase in respiratory pump perfusion, as well as the concurrent increase in cerebral perfusion, came at the expense of mesenteric perfusion.
Hansen et al. (2013)	• Thirty-two patients with congenital heart disease who underwent superior cavopulmonary anastomosis were studied to assess the clinical value of NIRS monitoring. • Somatic SO_2 (sSO_2) decreased from 77% during the early postoperative course to 68% at the end of the 48-hour monitoring period. • Postoperative complications were observed in 7 patients and the mean sSO_2 values during the first 4 hours postoperative were lower in patients with complications compared to those without (70% vs. 80%; p=0.004).
Oyaizu et al. (2013)	• Thirty-four patients with congenital heart disease were studied to evaluate the change in liver oxygen saturation following pulmonary artery banding (PAB). • PAB was considered effective (n=26) or ineffective (n=8) based on the need for secondary PAB correction following the first surgery. • Regional liver tissue oxygenation was significantly increased compared to baseline in patients with effective PAB (72% vs. 60%). • Changes in liver rSO_2 reflected the effectiveness of PAB; monitoring liver rSO_2 may be a good indicator to decide the tightness of the PAB.

NIRS oximetry devices in healthy adult volunteers, Bickler et al. showed that while the 5 NIRS monitors studied were all responsive to hypoxia-induced desaturation there was significant subject-to-subject variability and measurement bias across devices. These authors concluded that while all marketed NIRS oximeters assume a constant ratio of arterial to venous blood in the sampled region (though different devices assume different ratios); this ratio might in fact be relatively dynamic in the face of changing oxygen balance. In fact, knowledge of this dynamic physiologic environment prevents any tissue oximeter from being "absolute" in the mixed clinical environment. Given this variability, it is difficult to establish a critical cutoff for rSO_2, either in terms of absolute percent saturation or reduction from baseline. There are data to suggest, however, that the rSO_2 threshold for neurological injury may be approximately 45-50% (i.e., an oxygen extraction ratio of 50-55%). In fact, brain lactate levels begin to rise and functional disturbances on EEG begin to be seen as cerebral rSO_2 falls below 50%.[33,34] Based on these data, interventions designed to maintain rSO_2 above this critical point may lessen the incidence of ischemic neurological injury.

Conclusions

Despite advances in techniques and improved mortality, surgical repair of congenital heart defects remains associated with significant risk of poor neurodevelopmental outcomes. Studies to date suggest that one of the most important factors in determining the clinical outcome of these complex surgical procedures is the incidence and severity of cerebral hypoperfusion during the early postoperative period. The INVOS™ oximeter provides noninvasive and continuous monitoring of cerebral/somatic oxygen supply and demand and thus aids in the clinician's real-time evaluation of the adequacy of cerebral/somatic tissue perfusion. Use of oxygen saturation monitoring with INVOS™ technology during this critical period allows for the rapid identification of inadequate cerebral perfusion that might otherwise go undetected. Interventions based on rSO_2 analysis have been shown to improve cerebral rSO_2 values and may lead to improved clinical outcomes in this vulnerable patient population. Similarly, monitoring of peripheral tissue oxygen saturation with INVOS™ technology may help identify impaired systemic oxygen delivery, which can lead to organ dysfunction and contribute to adverse postoperative outcomes.

Table 4. Summary of rSO_2 and Oxygen Transport Balance

rSO_2			
• Increases with rise in oxygen delivery or fall in oxygen demand. • Decreases when oxygen delivery falls or there is an uncompensated rise in oxygen demand.			
Oxygen delivery/supply		Oxygen demand/consumption	
Influenced by:		**Increased by:**	**Decreased by:**
• Oxygen content – Hemoglobin concentration – Hemoglobin saturation	• Cardiac output – Optimize HR –Optimize preload – Intropy – Reduce afterload	• Fever, shivering • Seizures • Wakefulness, anxiety, and pain	• Normo- or hypothermia • Sedation and muscle relaxants • Mechanical ventilation

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The INVOS™ monitoring system should not be used as the sole basis for diagnosis or therapy and is intended only as an adjunct in patient assessment. Reliance on the INVOS™ system alone for detecting cerebral desaturation events is not recommended.

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