Detection and Correction of Brain Oxygen Imbalance

Surgical and Critical Care Applications of the INVOS™ Cerebral Oximeter

Harvey L. Edmonds, Jr. Ph.D.
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A Pocket Guide for Clinicians

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Learning Objectives

After reading this guide, clinicians should be able to:

» Describe the link between regional cerebral oxygen balance and the INVOS parameter, regional oxygen saturation (rSO₂)

» Integrate brain rSO₂ information with other physiologic & clinical data before, during and after surgery

» Identify special situations which can influence cerebral rSO₂ monitoring

» Discuss the clinical management and response to cerebral rSO₂ monitoring

This resource is intended for educational purposes only. It is not intended to provide comprehensive or patient-specific clinical practice recommendations for rSO₂ monitoring technology. The clinical choices discussed in this text may or may not be consistent with your own patient requirements, your clinical practice approaches, or guidelines for practice that are endorsed by your institution or practice group. It is the responsibility of each clinician to make his/her own determination regarding clinical practice decisions that are in the best interest of patients. Readers are advised to review the current product information including the Indications for Use currently provided by the manufacturer. Neither the publisher, authors, nor Covidien assumes any responsibility for any injury and or damage to persons or property resulting from information provided in this text.

Dr. Edmonds received compensation from Covidien for his professional time spent preparing this educational piece.
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Executive Overview

Regional oxygen saturation (rSO₂) monitoring systems permit the continuous noninvasive measurement of cerebral regional oxygen balance within the frontal cerebral cortex. Since cerebral rSO₂ represents an adjunct physiologic measure, local microcirculatory oxygen balance, it is important to appreciate the fundamental elements of this infrared light-based technology. This technology offers additional insights into patient clinical status; however, the novelty of the technology also makes it imperative for clinicians to review important situations and limitations that may influence rSO₂.

The non-invasive INVOS™ monitoring system is intended for use as an adjunct monitor of regional hemoglobin oxygen saturation of blood in the brain or in other tissue beneath the sensor. It is intended for use in individuals greater than 2.5 kg at risk for reduced-flow or no-flow ischemic states.

The INVOS system should not be used as the sole basis for diagnostic or therapeutic decisions however; both randomized and non-randomized controlled clinical trials have shown the positive impact of INVOS-guided patient management on patient outcomes. Compared with standard clinical practice, INVOS monitoring with a standardized interventional protocol has been shown to provide improved outcomes after surgery and resource utilization.¹⁴

For complete information about the INVOS system, refer to the full service manual/instructions for use. Additional clinical information and other educational resources can be accessed at www.covidien.com/PACE. Customer support is also available via telephone (US Toll Free) 1-800-635-5267. International contact numbers are available at http://www.covidien.com/covidien/pages.aspx?page=Contact.
Intracranial regional hemoglobin oxygen saturation (rSO$_2$) measurement by NIRS is possible because the human skull is translucent to infrared light. As with other forms of clinical oximetry, saturation determination relies on multiple wavelengths of light to discriminate between the unique absorption spectra of oxyhemoglobin and deoxyhemoglobin. Generally speaking, within the wavelength region of interest (i.e., spectrum), the only other competing infrared absorbers (i.e., chromophores) are water and the skin pigment melanin. INVOS measurements appear to be relatively unaffected by normal skin pigment variation in adult patients; however, pathologic variations in pigmentation or brain water content may influence photon absorption, scatter and the resulting rSO$_2$ measurement.5,6

**Figure 1: Photon Absorption in the Cranium**

The cranium contains four substances that absorb photons with wavelengths in the near-infrared range (i.e., 680-900 nm).
rSO2—A Clinically-Validated Measure of Regional Brain Oxygen Balance

The INVOS™ System

The INVOS system 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. This placement permits monitoring of the ischemia-susceptible cortical tissue in the border zone between the anterior and middle cerebral arteries but, may preclude detection of posterior border zone oxygen imbalance or perfusion abnormality.7,8

Figure 2 illustrates that the mean path of tissue-reflected photons is parabolic. With an INVOS optode light source-detector separation of 4 cm, infrared photons penetrate to a depth of ~2 cm [i.e., (light source - detector separation)½] below the skin surface.9 Thus, INVOS monitoring measures the outer cerebral cortical layers in adults.9 The cerebral tissue sample volume is ~1.5 cc.9 Since there is sufficient photon-absorbing hemoglobin in larger vessels to trap all incident infrared light, surface-detected infrared reflections arise exclusively from blood vessels <100 μm in diameter. Based on recent positron emission tomography (PET) studies, the arterial:venous ratio within this microcirculation appears to be ~30:70.10 Observations during retrograde cerebral perfusion demonstrated that the INVOS monitor rSO2 determination is relatively insensitive to substantial shifts in this ratio.11 In addition, rSO2 differs from other oximetric technologies such as pulse oximetry (SpO2) and jugular venous oxygen saturation (SjvO2) because it does not require actively flowing blood, pulsatile or non-pulsatile.

The INVOS system uses a proprietary process termed spatial resolution to suppress the influences of extracerebrally reflected photons. The optode contains a second infrared detector located closer to the light source. Its shallower parabolic path includes less brain and more extracerebral tissue reflections (Figure 2).12
rSO2—A Clinically-Validated Measure of Regional Brain Oxygen Balance

The shallow, deep signal ratio results in a rSO2 measure that is ~70% intracranial and independent of interpatient variation in photon scatter.9,10
Currently, rSO2 provides the only non-invasive method to continuously monitor changes in local brain oxygen balance.13

Figure 2: INVOS™ Detector Orientation

Each INVOS optode uses the proprietary orientation of two infrared detectors to measure light reflected transcranially from a neighboring source. Detector orientation ensures that the parabolic photon paths to both detectors traverse brain tissue.

Validation of rSO2 as a Measure of Brain Oxygen Balance

Compared with other oximetric technologies such as arterial (SaO2) and jugular venous (SjvO2) oxygen saturation, verification of brain rSO2 accuracy remains technically challenging. In the absence of a true reference, manufacturers and the U.S. Food & Drug Administration have adopted a proxy called field saturation (fSO2).
This metric, k1(SaO2) + k2(SjvO2), was developed by an early INVOS system clinical investigator to assess cerebral oximeter performance.14 The manufacturer-specific constants k1 & k2 are empirical estimates of the arterial and venous blood contribution to proprietary rSO2 algorithms.
rSO₂—A Clinically-Validated Measure of Regional Brain Oxygen Balance

A peer-reviewed report describes statistically significant correlations between fSO₂ and the INVOS monitor rSO₂ in healthy adults breathing room air as well as hypoxic and hypercapnic mixtures (Figure 3). Without a true reference standard however, regional cerebral oxygen saturation accuracy is indeterminate and the interpretation of device comparisons is complex and uncertain.

Clinicians should appreciate that momentary rSO₂ values and trending characteristics are machine-specific and are not interchangeable among different oximeter brands. As a result, it is unjustified to use clinical data generated from one proprietary rSO₂ system to “validate” the utility of a competing device.

**Figure 3: Correlation Between rSO₂ and fSO₂**

Cerebral oximeter performance is assessed by comparing rSO₂ values to a proxy for brain saturation termed field saturation (fSO₂). This graph derived from the study of Kim et al. (2000) shows statistically significant correlation between rSO₂ and fSO₂ in a group of adult volunteers exposed to graded levels of hypoxia and hypercapnia.
Normative Brain rSO₂ Values

Normative brain rSO₂ values are an absolute requirement for the definition of abnormality. For the INVOS systems, the normative rSO₂ is 71±6% for conscious healthy adults (n=44). Edmonds et al. preoperatively determined an rSO₂ of 67±10% in 1,000 adult cardiac surgery patients (age 21-91 years, 32% female) (Figure 4). Values <50 were thus statistically subnormal. A left vs. right hemisphere asymmetry of >10% occurred in only 5% of patients. Recently, multiple studies have confirmed both the cardiac patient normative values and asymmetry incidence. It is noteworthy that SjVO₂ asymmetry >10% occurs in a majority of patients. Thus, physiologically appropriate comparisons of rSO₂ and SjVO₂ require that both measurements invariably be made from the same side of the head.

Figure 4: Sample rSO₂ data

Edmonds et al. (2004) published the first large sample rSO₂ normative data for adult cardiac surgery patients. Since the data were normally distributed, statistical abnormality is readily defined as the upper and lower 2.5% extremes of the frequency distribution.
Brain rSO₂ Monitoring Before, During and After General Anesthesia

Pre-procedure (baseline) rSO₂

INVOS monitoring does not require establishment of a pre-procedure baseline reference. As with intraoperative blood pressure monitoring however, obtaining baseline information is sound clinical practice. Moreover, pre-procedure bilateral rSO₂ values can alert the clinician to technical difficulties in need of immediate correction or valid pre-existing symmetrical or asymmetrical subnormal values.

Collection of reliable baseline rSO₂ values is influenced by proper recording technique. Prior to optode (i.e., disposable sensor) application, patient forehead skin oil should be removed with an INVOS-provided acetone wipe. Oil removal facilitates optimal adherence of the self-adhesive optode to block interference by environmental light. If the forehead is exposed to intense light (i.e., direct forehead illumination by surgical lights) or heat sources (i.e., fluid or body warmers), the optodes should be covered with an opaque material. If practical, the sensors should be positioned above each eye with the long axis parallel to the intra-aural line and the superior optode edge adjacent to the hairline. Consistent positioning in this manner minimizes inter- and intrasubject baseline rSO₂ variation and avoids the potentially confounding effects of the frontal sinus on light scattering. Repeated optode use is not recommended because the accumulation of epidermal debris on the adhesive surface may have unpredictable effects on extracranial photon scattering.
Prior to monitoring, INVOS system recording quality should be assessed by inspection of the signal strength index (SSI) for each channel (Figure 5). The 5-unit bar scale is non-linear. Thus, the 1-bar signal strength is only 4% of that represented by 5 bars. Adequate signal strength is represented by the continuous display of >1 bar.

**Figure 5: INVOS™ Monitor**

The green vertical 5-bar Signal Strength Indicators are shown for each INVOS recording channel. Signals with a stable reading of >1 bar are sufficiently strong to permit reliable monitoring. Large font numbers are the momentary rSO2 values for each channel, which are updated every 5 seconds. Mid-size font values are the baseline values. Red numbers reflect an oxygen debt alarm and the small font values display the percentage change from baseline.

**Positioning**

A sudden symmetric or asymmetric rSO2 decrease may occur during anesthetic induction, pulmonary artery or central venous catheter insertion or final positioning (Figure 6). Without accompanying change in blood pressure or respiratory gases, precipitous rSO2 decline can help identify an otherwise unrecognized cerebral blood inflow or outflow obstruction.11,25,26
With cardiac and vascular surgery, the unexpected development of regional brain oxygen debt may be the consequence of a malpositioned heart, perfusion cannula, vascular clamp, ligature or cardiac vent.27-29

**Figure 6: INVOS™ and Patient Positioning**

INVOS monitoring detected rSO2 decline. Oxygen balance returned to normal with restoration of the supine position, and the surgery proceeded without incident.

**CO2 Influence on rSO2**

Cerebral arteries in the healthy brain are exquisitely sensitive to hydrogen ion shifts and consequently CO2 change. CO2 accumulation results in arteriolar vasodilation and attendant rSO2 increase.30,31 Of note, the CO2-mediated rSO2 rise accompanying endotracheal intubation provides a simple method to verify bihemispheric normal vascular responsiveness (Figure 7).11
Figure 7: INVOS™ and CO₂ Accumulation

The inset graph at upper left shows the normal response to pre-oxygenation and anesthetic induction. The large graph also shows large rSO₂ increase with endotracheal intubation, suggesting normal cerebral arteriole CO₂ reactivity. Multiple hypocapnic episodes consistently resulted in brain oxygen debt. Each was promptly corrected by appropriate adjustments of the respiratory rate (RR) and tidal volume (VT).

Since cerebral CO₂ reactivity is a pre-condition for autoregulation, its absence signifies increased risk of potentially injurious oxygen imbalance and hypoperfusion (Figure 8). Armed with this knowledge, rSO₂-guided blood pressure management may then be used to help avoid hypoperfusion injury. The individualized CO₂:rSO₂ relationships are also important during cardiopulmonary bypass to optimize acid-base management. With CO₂-unreactive cerebral arterioles, the risk of brain hypoperfusion is increased and the perfusionist has a diminished opportunity to improve brain oxygen balance via adjustments in acid-base management.
Brain rSO2 Monitoring Before, During and After General Anesthesia

**Figure 8: INVOS™ and Hypoperfusion**

rSO2 trends were notable initially for abnormally low and asymmetric baseline values. The trends then precisely quantified the extent of brain oxygen debt associated with three failed intubation attempts and documented the ultimately successful fourth attempt.

**Systemic & Regional Hypoxemia Influences on rSO2**

The physiologic properties of brain rSO2 make it uniquely suited for the early detection of developing hypoxemia. Inspection of the familiar oxygen dissociation curve emphasizes that venous SvO2 or venous-weighted rSO2 will change more than arterial SaO2 or SpO2 to a fixed decline in blood oxygen partial pressure (Figure 9). This fact combined with the extraordinarily high brain oxygen demand results in observation that developing hypoxemia often appears first in brain rSO2 (Figure 8). Even with the extensive physiologic monitoring used during cardiac surgery, evidence of inadequate oxygen delivery may be first observed by a declining cerebral rSO2.
The oxygen dissociation curve illustrates the differential sensitivity of arterial and venous-dominant O₂ saturation measures to small changes in oxygen partial pressure. This differential sensitivity helps explain the observation that cerebral rSO₂ routinely detects developing oxygen imbalance before pulse oximetry.

**Blood Product and Fluid Management Influences on rSO₂**

In its low-to-normal range (i.e., hematocrit <30%), hemoglobin and rSO₂ are linearly related, while at a higher hematocrit, their relationship vanishes or may become inverse. This hemoglobin-dependency explains the oft-observed transient rSO₂ decline at the onset of cardiopulmonary bypass. Initial passage of a crystalloid prime solution through the cerebral circulation momentarily lowers brain hemoglobin. It should also be appreciated that blood product administration will not invariably result in a rSO₂ increase. Naidech et al. (2008) noted a wide variation in brain rSO₂ responses to administration of packed red cells (Figure 10).
Occasional declines in rSO₂ should be expected, since stored red cells may have their oxygen-carrying capacity diminished by up to 90%.³⁸

**Figure 10: rSO₂ and Blood Product Administration**

The results of this small study illustrate the marked variation in rSO₂ response to administration of two units of packed red cells (PRC). (The graph is based on data from Naidech et al. 2008.)

**Anesthetic Influences on rSO₂**

Nearly two-thirds of cerebral oxygen is utilized to support interneuronal signal transmission. Thus, anesthetic influences on rSO₂ depend on the neuropharmacologic properties of each agent and its dose. Volatile halogenated anesthetics, barbiturate hypnotics and propofol have profound cortical suppressant activity, while opioid analgesics and benzodiazepine amnestic generally do not. Rising doses of the powerful cortical suppressant anesthetics may increase rSO₂ as oxygen consumption is decreased.³⁹ Conversely, a sudden rSO₂ decrease may signify decline in anesthetic effect (Figure 11).
Figure 11: INVOS™ and Anesthesia

At the onset of total cardiopulmonary bypass, brain responses to an initially unrecognized empty anesthetic vaporizer are shown. The increased cerebrocortical neuronal activity bilaterally increased EEG bispectral index (BIS) and decreased brain oxygen balance (i.e., rSO₂). All values normalized with vaporizer refilling.

Brain Temperature Management Influences on rSO₂

The neuroprotection afforded by hypothermia, in part, is due to reduced brain oxygen demand. However, individual patient responses to cooling vary widely. Thus, decreasing cranial temperature does not automatically ensure an adequately neuroprotective cerebral hyperoxic state. Wide variation in cooling response is due to patient-specific cerebral hemodynamics as well as mechanical perfusion strategy/tactics. For example, the enhanced cerebral blood flow and cooling efficacy afforded by temperature-corrected (i.e., pH-stat) acid-base management improve neurologic outcome in both pediatric and adult patient cohorts undergoing cardiovascular surgery with deep-hypothermic circulatory arrest. Yet, the magnitude of hypothermic neuroprotection in individual patients depends on the bi-hemispheric responsiveness of cerebral arterioles to change in hydrogen ions and CO₂.
Cerebral oximetry gives anesthesia providers and perfusionists this key information at the start of surgery to guide patient care plans and optimize hypothermia management (Figure 12). As a result, during re-warming an inverse relationship between brain temperature and rSO2 has been described in both adult and pediatric cardiac surgery patients. Prompt detection and treatment of this flow-metabolism mismatch may help avoid ischemic brain injury.

**Figure 12: INVOS™ and Brain Temperature**

The left graph shows the expected inverse relationship between cranial temperature and brain rSO2. Note that the rSO2 rise reaches an asymptote at ~23º C and that further cooling doesn’t create more regional hyperoxia. At lower right, the graph illustrates an optimal cooling response conducted with pH-stat acid-base management in a patient with CO2-reactive cerebral arterioles. Marked hyperoxia prevented oxygen debt development during later total circulatory arrest. In contrast, the upper right graph depicts suboptimal cooling with alpha-stat acid-base management. Minimal hyperoxia resulted in a large oxygen debt during total circulatory arrest.
Supplemental Cerebral Perfusion Influences on rSO₂:

During deep-hypothermic circulatory arrest, rSO₂ declines of >30% below baseline are highly associated with new neurologic deficit. Numerous studies have demonstrated that the "safe time" for systemic circulatory arrest may be extended with the use of bilateral rSO₂ monitoring to ensure adequate retrograde or selective antegrade cerebral perfusion (Figure 13).49,50

**Figure 13: Supplemental Cerebral Perfusion**

Because of the hemodynamic characteristics of this acute Type I aortic dissection, upper body cerebral perfusion was mechanically supported through a right axillary perfusion cannula. However, cerebral perfusion was not symmetrically supported. Cooling and selective antegrade cerebral perfusion were more efficacious on the right side. rSO₂ verified adequate right hemisphere perfusion throughout the emergent procedure.
The following chart (Table 1) provides a summary of pre-incision considerations for INVOS monitoring.

**Table 1: Pre-incision Bilateral rSO2 Assessment**

<table>
<thead>
<tr>
<th>Define</th>
<th>Consider</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Signal Strength Index (SSI)</strong></td>
<td>• Signal reliable with stable SSI &gt;1 bar</td>
</tr>
<tr>
<td></td>
<td>• If signal unreliable, check cable and reposition/replace optode</td>
</tr>
<tr>
<td><strong>Pre-procedure baseline</strong></td>
<td>• rSO2: &lt;50 or &gt;80 indicates normative range. Right vs. left rSO2 &gt;10%</td>
</tr>
<tr>
<td></td>
<td>• indicates asymmetry. Rule out technical cause for abnormality.</td>
</tr>
<tr>
<td></td>
<td>• Check: patient history, cardiopulmonary and hemodynamic status, hemoglobin/hematocrit.</td>
</tr>
<tr>
<td><strong>O2D Alarm Threshold</strong></td>
<td>• If rSO2 normal, set alarm @ 20% &lt; baseline</td>
</tr>
<tr>
<td></td>
<td>• If rSO2 subnormal, set alarm @ baseline</td>
</tr>
<tr>
<td><strong>Pre-oxygenation response</strong></td>
<td>• Low O2 reserve with rSO2 increase &gt;5%</td>
</tr>
<tr>
<td><strong>Endotracheal intubation response</strong></td>
<td>• Low CO2 reactivity with rSO2 increase &lt;5%</td>
</tr>
</tbody>
</table>
Special Issues Impacting Brain rSO₂ Monitoring

Hundreds of peer-reviewed studies demonstrate that despite the potential for artifact and other issues, reliable INVOS monitoring rSO₂ values can be obtained in many patient care settings.⁻⁵¹⁻⁵₃ However, in certain circumstances, momentary rSO₂ values may not accurately reflect regional brain oxygen balance. Some of the examples below emphasize the importance of rSO₂ trends in signal interpretation. As noted, INVOS monitoring is an adjunct to clinical judgment, not a substitute for it.

Extreme examples of this inherent physiologic limitation are the reports of normative rSO₂ values that were obtained from human cadavers or chromophore-containing inanimate objects like pumpkins.⁻⁵⁴⁻⁵⁵ Cadaveric rSO₂ values may be normal because post-mortem cerebral venous oxygen saturation ranges widely from 5-95% depending on the cause of death and body storage conditions.⁻⁵⁵⁻⁵⁶ Similarly, a normative rSO₂ reading may arise from pumpkins because the value depends simply on the spectrophotometric measurement of non-pulsatile reflected light.

Conversely, artifactually low rSO₂ values may be attributable to⁻⁵⁷⁻⁶₃:

1. optode positioning over an intracranial photon sink (i.e., intracranial venous sinus or hematoma),
2. excessive photon scattering (i.e., hair or hair follicles),
3. cranial bone anomaly or frontal sinus inflammation,
4. presence of infrared-absorbing intracranial or intravascular pigments or dyes, or
5. dyshemoglobinemias.
Cerebral Hyperperfusion

The vast majority of clinical rSO2 studies have focused on brain injury from hypoperfusion and oxygen debt. However, cerebral hyperperfusion manifested by hyperoxia is also potentially injurious. Under-perfused brain shifts to anaerobic metabolism for survival. Resulting lactic acidosis dilates cerebral arterioles in affected regions. Consequently, a benign transient hyperemia typically appears with restoration of normal perfusion. For example, after termination of vascular occlusion during carotid endarterectomy or carotid angioplasty and stenting, ipsilateral cerebral hyperoxia (i.e., rSO2 increase >10%) generally appears within 3 minutes and normalizes within 20 minutes (Figure 14).64 Alternatively, a pathologically persistent (i.e., >24 hr) hyperemia may produce vasogenic edema and a cerebral hyperfusion syndrome characterized by migraine symptoms, delirium, focal neurodeficit and seizures.65 The syndrome may develop with “normal” blood pressure and may be undetectable by tomographic brain imaging.66 Ogasawara et al. (2003) found the incidence of SPECT-confirmed pathologic post-endarterectomy hyperperfusion to be 12%. These authors showed cerebral oximetry to have 100% sensitivity and specificity in detecting this hyperperfusion. Other investigators have reported on the value of rSO2 in detecting hyperperfusion accompanying retrograde, or selective antegrade cerebral perfusion during aortic arch surgery.11,49,67
Seizure Activity

Cerebral vasoneural coupling ensures that local brain metabolic increases normally are met with augmented regional blood flow. With intermittent episodes of intense synaptic activation, brain rSO₂ trends recorded at the highest (i.e., 5 s) update rate may oscillate because of onset-offset variations in the hemodynamic coupling response (Figure 15). These rapidly oscillating rSO₂ trends have been used successfully to detect seizure activity in chemically paralyzed, ventilated patients and monitor patient response to anticonvulsant therapy. Clinically silent seizures occur frequently in neurocritical care patients and, if left untreated, may adversely affect outcome.
Special Issues Impacting Brain rSO₂ Monitoring

**Figure 15: Seizure Activity**

Cerebral Vasospasm

The presence of intracranial extravascular blood may trigger arterial vasospasm. Resulting local hypoperfusion may disrupt normal vasoneural coupling. As with seizure activity, the destabilized hemodynamic response can then lead to oscillation in rapidly updated rSO₂ trends.⁷² INVOS recordings from shaved scalp overlying a spastic arterial segment successfully recorded vasospasm progression and a subsequent positive therapeutic response.⁷³
Intracranial Hypertension

Cerebral rSO₂ is inversely related to intracranial pressure in critical care patients with brain tumors, head trauma or hydrocephalus. In all three pathologic conditions, intracranial hypertension is associated with a significant rSO₂ reduction, signifying developing brain O₂D. However, the presence of intracranial extravascular blood may confound this relationship because of infrared photon sequestration. It should also be appreciated that rSO₂ values obtained from dying or dead brain are typically very high because there is little or no oxygen consumption. This observation helps explain the lack of a linear relationship between cerebral blood flow and rSO₂. Furthermore, large shifts in intracranial photon scattering that accompany brain swelling may profoundly alter rSO₂ in an unpredictable manner.
Clinical Management: Responding to Brain rSO\(_2\) Changes

rSO\(_2\) fluctuations may be observed with the INVOS monitor. Variability in rSO\(_2\) values when seen during a single hemodynamic fluctuation, for example a change in blood pressure, are not necessarily clinically significant however; specific consideration should be given to a large decrease (i.e., >20%) or increase (i.e., >10%) in rSO\(_2\) from a pre-procedure or other reference point. A systematic approach is presented to guide detection and correction of noteworthy brain oxygen imbalance. It remains an evolutionary process that has emerged from earlier published algorithms.\(^2\)\(^4\)\(^7\)\(^6\)-\(^7\)\(^9\)

Table 2 presents a newly updated, objective, systematic, stepwise rSO\(_2\) assessment process.
Table 2: Assessment of Cerebral Oxygen Imbalance (Observations and Considerations with the INVOS™ System)

<table>
<thead>
<tr>
<th>Observe</th>
<th>Consider</th>
</tr>
</thead>
<tbody>
<tr>
<td>rSO₂ directly correlated with change in BP</td>
<td>• Dysautoregulation</td>
</tr>
<tr>
<td>rSO₂ and ↑BP inversely correlated</td>
<td>• Vasoconstrictor hypoperfusion</td>
</tr>
<tr>
<td>rSO₂ and ↓BP uncorrelated</td>
<td>• Airway inadequacy</td>
</tr>
<tr>
<td></td>
<td>• Ventilation abnormality (i.e., hypocapnia)</td>
</tr>
<tr>
<td></td>
<td>• Anesthetic delivery inadequacy</td>
</tr>
<tr>
<td></td>
<td>• Cardiopulmonary/CPB dysfunction</td>
</tr>
<tr>
<td></td>
<td>• Blood loss/hemodilution</td>
</tr>
<tr>
<td></td>
<td>• Non-pulsatile perfusion</td>
</tr>
<tr>
<td></td>
<td>• Brain temperature increase</td>
</tr>
<tr>
<td></td>
<td>• Intracranial hypertension</td>
</tr>
<tr>
<td>rSO₂ ↑BP uncorrelated</td>
<td>• Cerebral hyperemia</td>
</tr>
<tr>
<td></td>
<td>• Brain temperature decrease</td>
</tr>
<tr>
<td></td>
<td>• Pulsatile perfusion re-established</td>
</tr>
<tr>
<td></td>
<td>• Low O₂ reserve with rSO₂ increase &gt;5%</td>
</tr>
<tr>
<td>rSO₂ asymmetry appearance</td>
<td>• Patient malposition</td>
</tr>
<tr>
<td></td>
<td>• Heart malposition</td>
</tr>
<tr>
<td></td>
<td>• Cannula, catheter, clamp or vent malposition</td>
</tr>
<tr>
<td></td>
<td>• Low CO₂ reactivity with rSO₂ increase &lt;5%</td>
</tr>
<tr>
<td>rSO₂ trend rapid oscillation</td>
<td>• Seizure-like activity</td>
</tr>
<tr>
<td></td>
<td>• Cerebral vasospasm</td>
</tr>
</tbody>
</table>
Summary

The INVOS System Pocket Guide has discussed how rSO2 monitoring may be used most effectively in the surgical and critical care environments to detect and correct regional brain oxygen imbalance. It is important for clinicians to fully appreciate the applications, limitations and special considerations for use of INVOS monitoring.

Evidence in the literature documents patient and economic benefits resulting from the use of INVOS monitoring. These clinical investigations provide an evidence-based rationale for the incorporation of INVOS monitoring as a tool to facilitate intraoperative and critical care management.

Depending upon the specific patient characteristics and clinical situation, utilization of INVOS monitoring may be a very appropriate decision. However, the decision to use the INVOS system should be made on a case-by-case basis by the individual practitioner.

As clinical experience and investigation continue, clinicians are encouraged to stay current with available literature regarding the use, benefits and limitations of INVOS monitoring to guide patient care. Additional clinical information and other educational resources can be accessed at www.covidien.com/PACE. Customer support is also available via telephone (US Toll Free) 1-800-635-5267. International contact numbers are available at http://www.covidien.com/covidien/pages.aspx?page=Contact.
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