Cerebral Oximetry—An Introduction

Dr Adam Carpenter[1†]

[1]Consultant Anaesthetist, Groote Schuur Hospital (University of Cape Town), Cape Town, South Africa

Edited by: Dr Clara Poon, Consultant Anaesthetist, Queen Mary Hospital, University of Hong Kong, Hong Kong

[†]Corresponding author email: adammsizi@yahoo.com

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KEY POINTS

- Cerebral oximetry uses near infrared spectrometry to assess cerebral oxygenation.
- The value derived is a mixed average of the arterial and venous components, and reflects the extraction of oxygen by the brain.
- It is a trend monitor allowing us to see the ongoing changes in cerebral oxygenation.
- Several brands are available, and clinicians should be aware of the limitations and pitfalls when interpreting their readings.
- It can be used in a wide variety of operations where there is concern about inadequate cerebral perfusion.

INTRODUCTION

"It is a trend monitor of greatest value in situations in which intracranial Hb saturation could dangerously change and in which changes in systemic haemodynamics and oxygenation would not predict that change." — Valerie Pollard and Donald S. Prough[1]

In patients under deep sedation or general anaesthesia, monitoring brain perfusion is crucial for the early detection of cerebral ischemia. Systemic blood pressure is a poor indicator of cerebral blood flow as it fails to account for interindividual anatomical variations (e.g., circle of Willis integrity) and physiological differences (e.g., robustness of pressure autoregulation). Alternative methods for measuring cerebral blood flow, such as transcranial Doppler and radiological perfusion studies, are often operator-dependent and resource-intensive. Cerebral oximetry, which utilizes near-infrared spectroscopy (NIRS) to assess tissue oxygenation, offers a noninvasive, objective, and bedside approach to directly evaluate cerebral oxygenation.

In this tutorial, we will introduce this technology, discuss common and recent trends in its clinical utilization, and acknowledge some of its limitations. Although NIRS has been used in other parts of the body, the use of somatic oximetry will not be covered in this article.

CEREBRAL OXIMETRY—THE PRINCIPLE

Coined in 1942 by physiologist Glen Milliken, the term "oximetry" refers to the measurement of haemoglobin oxygen saturation in blood or tissue. Cerebral oximetry is a non-invasive, bedside method for measuring tissue (cerebral) haemoglobin oxygen saturation (ScO2). The basic components of oximetry include a light source that emits light at specific frequencies through a tissue area, where it is partially absorbed, and light sensors that measure the scattered and unabsorbed light. Light in the near-infrared spectrum penetrates biological tissues, including the skull and brain, and exhibits distinct absorption patterns in chromophores such as oxyhaemoglobin, deoxyhaemoglobin, and cytochrome c oxidase. By emitting light in this range and measuring the differential absorption, one can determine the relative concentrations of oxyhaemoglobin and deoxyhaemoglobin, thereby

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inferring tissue oxygen saturation and blood flow. If light attenuation between a source and detector is solely due to absorption by chromophores, it follows the Beer-Lambert law. However, tissue light scattering complicates this measurement and presents significant technical challenges, which will be explored later. Wavelengths typically used range from 700 to 870 nm, where the absorption spectra of haemoglobin (Hb) and oxyhemoglobin are maximally separated and overlap with H_2O is minimal. The probe, which contains the light emitter and sensors, is usually placed on the forehead to minimize the effects of hair on light transmission.

Commercial Cerebral Oximeters

There are several commercial cerebral (tissue) oximetry monitors available, all of which operate based on the same basic principles but utilize slightly different means to address technical obstacles such as light scattering to improve their accuracies (Table 1).

The most common modification in commercial devices is multidistance (or spatially resolved) spectroscopy. This technique is based on the principle that the depth of tissue investigated is directly proportional to the distance between the light emitter and light detector. Increasing this distance can enhance the depth of tissue sampled, allowing for the measurement of oxygenation in the intracranial compartment. Other commercial modifications, such as frequency-resolved spectroscopy and time-resolved spectroscopy, are rarely used.[2]

Spatially resolved spectroscopy is employed in several widely used monitors (e.g., SenSmart, FORE-SIGHT, INVOS series, Masimo O3, NIRO series)[2,3] (see Figure 1). Various methods, some of which are proprietary, are utilized to enhance sensitivity. These include increasing the number of light wavelengths to improve the signal-to-noise ratio, using longer wavelengths to increase tissue permeation and reduce extracranial interference, and separating the source and detector by a greater distance to allow for deeper tissue penetration. Different subtraction algorithms are also employed. Consequently, direct comparison of data between commercial devices is challenging.

A cerebral oximeter measures an unknown mixture of gas-exchanging vessels (arterioles, capillaries, and venules) within the tissue beneath the sensor. Unlike pulse oximetry, which measures arterial blood oxygenation, cerebral oximetry assesses the entire returned signal, so pulsatility of tissue components is not required. It evaluates all haemoglobin in the reflectance arc (including those within the arterial, venous, and capillary compartments), resulting in a value for ScO2 that is biased toward the larger venous haemoglobin mass, or "venous-weighted." Manufacturers typically assume a fixed arterial-to-venous ratio, ranging from 25% to 30% for arterial and 70% to 75% for venous cortical blood volume in their algorithms. However, the actual ratio can vary depending on the individual, location of the measurement, and dynamic status of the cerebral vasculature.

In healthy adults, the normal cerebral oxygen extraction ratio ranges from 20% to 40%, with a commonly cited "normal value" of ScO2 between 60% and 80%. Due to significant variation in baseline values, cerebral oximetry is best used as a trend monitor. The oximetry probe should ideally be placed on the patient before the induction of anaesthesia to establish a patient-specific baseline ScO2, against which subsequent ScO2 can be compared. There is no consensus on what decrement in cerebral oxygen saturation from baseline signifies irreversible injury. Nonetheless, a widely used criterion for defining "desaturation" is a reduction of >20% from baseline or an absolute value of <50%.[4]

ScO2 is influenced by cerebral blood volume and its oxygenation, which are affected by systemic factors such as blood pressure, haemoglobin concentration, and arterial oxygen and carbon dioxide partial pressures. Since ScO2 includes arterial and venous components, it can be considered an indicator of the cerebral oxygen supply-demand balance. Low ScO2 values may indicate inadequate oxygen delivery (e.g., cerebral hypoperfusion) or increased tissue oxygen extraction due to high metabolic demand (e.g., seizures). Conversely, high ScO2 values may suggest cerebral hyperperfusion or metabolic suppression. Given the complex interplay of contributing factors, ScO2 should be interpreted alongside other available data within the appropriate clinical context (see Table 2).

Product Name	INVOS	FORE-SIGHT	SenSmart / EQUANOX	REGIONAL OXIMETRY
Latest model	INVOS 7100	ELITE	SenSmart X-100	O3
Manufacturer	Covidien (Medtronic)	CAS Medical System (Edwards Lifesciences)	Nonin	Masimo
Number of wavelengths	2 (730/810 nm)	5 (685/730/770/810/870 nm)	4 (730/760/810/880 nm)	4 (730/760/805/880 nm)
Notes	Adult, paediatric, neonatal sensors available.	Utilizing lights from 5 wavelengths lessens interference from melanin and bilirubin. Large, medium, and small sensors available for adults, paediatrics, and neonatal patients.	Dual emitter design offering more light paths for tissue interrogations. Adult and paediatric sensors available.	Adult, paediatric, and neonatal sensors available.

Table 1. Examples of Commercially Available Cerebral Oximeters

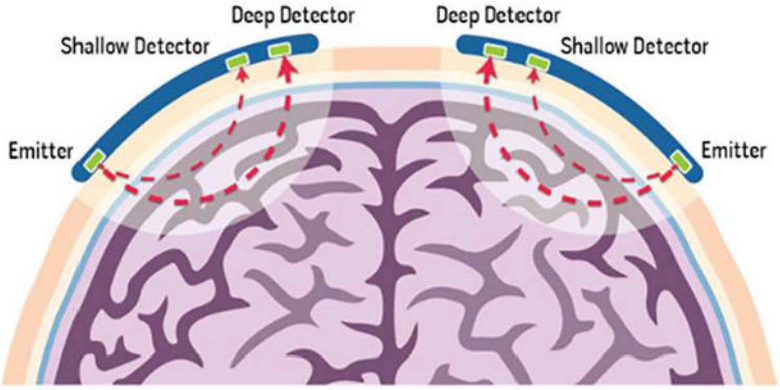

Figure 1. Diagram of cerebral oximetry with deep and shallow light detector paired with light source (Courtesy of ©2023 Masimo Corporation, used with permission).

Several pitfalls and limitations must be considered when interpreting NIRS readings:

1. **Extracranial contamination**: The NIRS signal can be affected by extracerebral tissues such as the scalp, skull, and sinuses, leading to inaccurate readings. Significant extracranial contamination has been reported with several commercially available NIRS devices.[5,6]
2. **Spatial resolution and penetration depth**: NIRS provides only a regional measure of oxygen saturation, limited to the superficial layers of the frontal cortex. It does not assess deeper brain structures or global brain tissue.
3. **Calibration and baseline variation**: There is no universally accepted gold standard for calibrating NIRS devices. Variability in individuals' baseline values and differences in device designs make it difficult to establish universal threshold values for intervention.
4. **Lack of universally accepted thresholds**: Variability in individuals' baseline values and differences in device designs make it difficult to establish universal threshold values for intervention. There is no consensus on what constitutes a significant desaturation in terms of the duration and magnitude of absolute or relative ScO2 decrement.
5. **Signal interpretation and lack of direct CBF measurement**: NIRS does not measure cerebral blood flow (CBF) directly. It provides an indirect assessment of cerebral oxygenation, which can be influenced by CBF, as well as systemic factors such as changes in arterial carbon dioxide levels, blood pressure, haemoglobin concentration, and cerebral vascular tone. The degree of venous weighting in the NIRS signal is not constant in real life, and assuming so can complicate the interpretation of ScO2 data. An example would be the observation that ScO2 often paradoxically decreases after phenylephrine boluses aimed to increase the arterial blood pressure. This phenomenon has previously caused some confusion but is now believed to be due to phenylephrine-induced reduction in extracranial blood flow and a decrease in the intracranial arterial-to-venous blood volume ratio, rather than genuine cerebral ischemia.[7]
6. **Pathological conditions**: Certain pathological conditions, such as the presence of hematoma or pneumocephaly in the measured area, may compromise the accuracy of NIRS measurements.

Factors Leading to Drop in ScO2	Possible Interventions to Increase ScO2
Cerebral hypoperfusion	Increase brain oxygen delivery • Check head position • Check central, aortic and/or superior vena cava catheter position • Improve arterial oxygen saturation (e.g. increase FiO2) • Aim to increase mean arterial pressure, improve cardiac output using combination of intravenous fluid administration and inotropes • Increase extracorporeal circulation pump flow rate • Optimize haemoglobin • Avoid hypocarbia
Increase O2 metabolic demand	Decrease brain oxygen consumption • Target temperature management – avoid hyperthermia • Exclude and treat any seizure • Increase depth of sedation/anaesthesia

Table 2. Possible Factors Contributing to Low ScO2 and Interventions to Increase ScO2. FiO2 indicates Fraction of Inspired Oxygen; ScO2, Cerebral Tissue Oxygen Saturation.

7. **Artifacts**: Other chromophores, such as bilirubin and melanin, can interfere with the measurement of tissue oxygenation. Therefore, establishing a baseline value for each patient individually is crucial. Intravascular dyes, such as indocyanine green (with a characteristic absorption peak around 805 nm) and methylene blue (with a peak around 668 nm), may also affect readings.

CLINICAL USE OF CEREBRAL OXIMETRY

The major advantages of cerebral oximetry are its noninvasive nature, ease of setup, and ability to provide real-time feedback and early warnings of cerebral hypoperfusion, particularly for patients undergoing high-risk surgeries (see Table 3). Below is a brief overview of its common clinical uses. However, it is important to note that evidence supporting its routine use is not robust. Numerous studies and meta-analyses have failed to reach definitive conclusions due to the scarcity of large studies, heterogeneity among studies, high risk of bias, poor adherence to preset protocols, and difficulties in translating data into final clinical outcomes.[8] Until more clinical evidence becomes available, current recommendations are at level III, which suggests that intraoperative ScO2 monitoring and associated management may reduce postoperative complications.

Carotid Endarterectomy

The initial application of cerebral oximetry, in conjunction with awake neurological monitoring during carotid endarterectomy, has provided valuable insights into its utility. By comparing drops in ScO2 with neurological assessments in awake patients, it has been suggested that a 20% drop from baseline ScO2 after carotid cross-clamping is associated with symptomatic cerebral hypoperfusion, prompting the insertion of a shunt. This 20% threshold, observed under local anaesthesia, resulted in pooled sensitivity and specificity estimates of 70.5% and 92.4%, respectively, compared with awake neurological monitoring.[9] A reduction in cerebral oximetry values greater than 12% from the baseline preoperative value has been identified as a reliable, sensitive, and specific indicator of brain ischemia.[4] After internal carotid artery cross-clamping, a drop in cerebral oximetry values may suggest the need for shunt placement during the procedure. Cerebral oximetry provides similar accuracy in detecting ischemia compared to transcranial Doppler and somatosensory evoked potentials monitoring.[10] However, higher-quality evidence using accurate reference standards and with low risk of bias is needed to determine the diagnostic accuracy of NIRS. It is also controversial whether intraoperative monitoring improves outcomes in carotid endarterectomies.[10]

Cardiac Surgery

Cerebral oximetry is utilized in adult and paediatric cardiac surgery, particularly in procedures involving cardiopulmonary bypass. Intraoperative and postoperative cerebral desaturation occur in up to 64% of patients undergoing cardiac surgery.[11,12] Proposed applications of cerebral oximetry in this context include preoperative risk stratification,[13] detecting malposition of aortic and venous cannula, confirming selective antegrade cerebral perfusion,[14] and early detection of hypocapnoea-induced cerebral vasoconstriction during cardiac bypass, and in close monitoring and treatment of cerebral desaturation in patients with vulnerable brains.

Importantly, Denault et al. introduced an algorithmic, goal-directed approach for treating cerebral desaturation, which includes a step-by-step process to improve physiological variables affecting cerebral perfusion (see Figure 2).[15] The “Denault algorithm” has been generally effective in reversing most desaturation events,[11,12] though not all studies have demonstrated improved clinical outcomes. Recent meta-analyses have also failed to provide strong evidence for improved postoperative outcomes.[16]

Paediatric and Neonatal Anaesthesia

Anaesthetists traditionally rely on basic monitoring parameters, such as blood pressure, heart rate, and oxygen saturation, to assess a patient’s cardiopulmonary status. However, in neonatal and paediatric patients, normal physiological parameters vary significantly across different age groups. For instance, what is considered a normal blood pressure and heart rate range differ between a 1-month-old neonate and a 3-year-old child. Cerebral oximetry enables clinicians to continuously monitor cardiac output, cerebral perfusion, and the cerebral oxygen supply-demand balance.[17] Any decrease in ScO2 from baseline should prompt an investigation and aggressive optimization of physiological variables to restore baseline levels.

Patient Factor	Carotid Stenosis Unstable Haemodynamic
Surgical Factor	Beach chair or sitting surgical position Permissive hypotension desired Surgical procedures involving aortic arch, carotid or intracranial vessels Cardiac bypass or aortic arch surgery Extracorporeal membrane oxygenation (ECMO) support

Table 3. Examples of Situations When Cerebral Ischemia is Particularly at Risk, When Monitoring Cerebral Oxygenation May Be Particularly Desirable

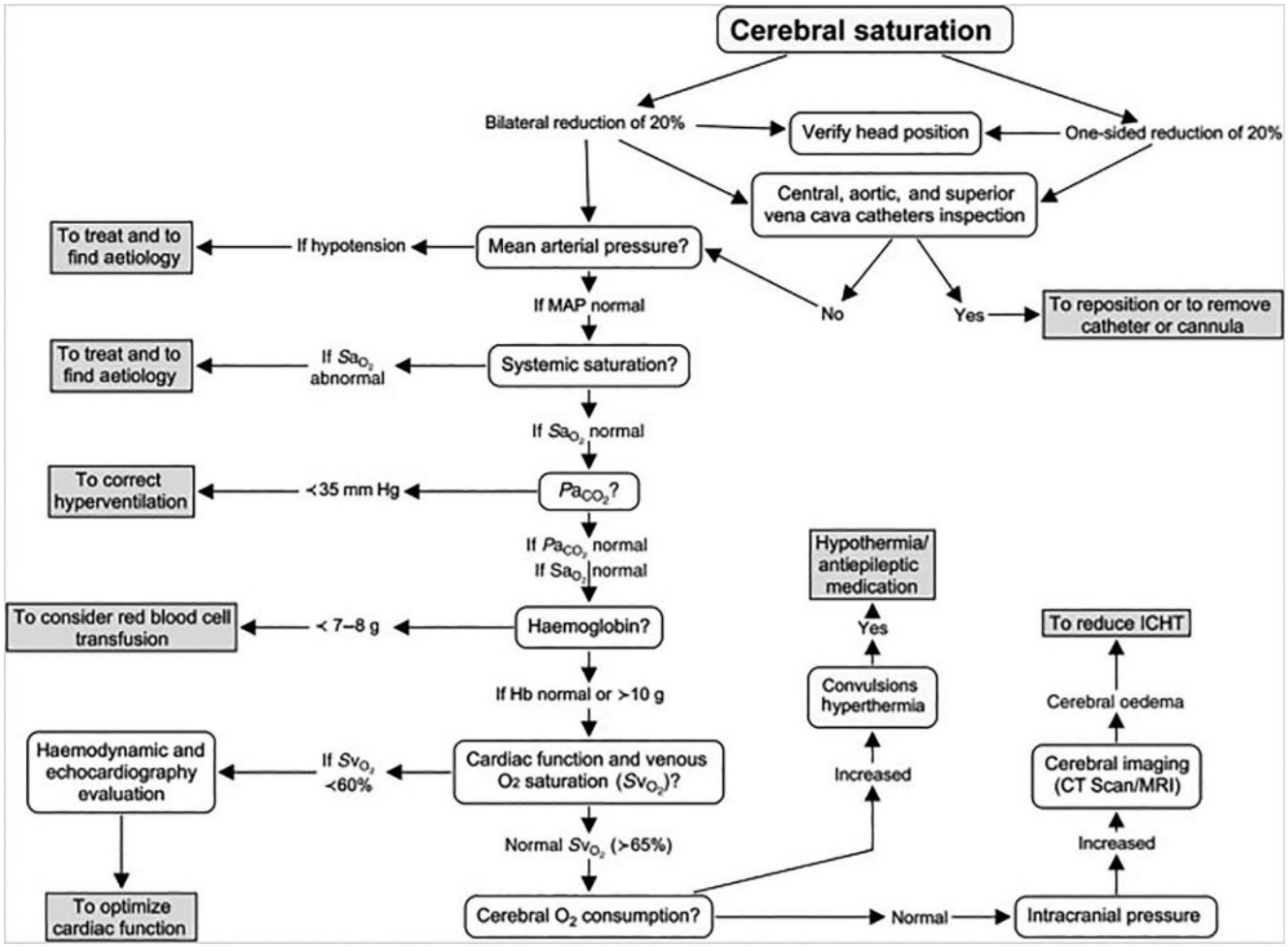

Figure 2. Algorithm proposed by Denault et al. to reverse intraoperative drop in ScO2.[15]

Hypotensive Surgery

Cerebral hypoperfusion during surgeries performed in the beach chair position is a well-documented complication. Monitoring blood pressure at the brachial artery may overestimate perfusion pressure at the level of the brain. Consequently, cerebral oximetry is becoming increasingly popular for assessing cerebral perfusion adequacy and guiding intraoperative interventions during surgeries in the beach chair position (e.g., for shoulder surgeries). Several studies have shown that significant decreases in ScO2 occur frequently, with reported incidences of up to 57% under general anaesthesia.[18] Despite the high incidence of cerebral desaturation events, the reported incidence of cerebrovascular events and neurocognitive complications after surgery in the beach chair position has been low. This unresolved association between intraoperative desaturation events and postoperative cognitive decline has led some to argue against the routine use of ScO2 in this context. However, with more sensitive neuropsychological testing and increased awareness, recent literature suggests a higher incidence of postoperative neurocognitive dysfunction, warranting closer evaluation to address this controversy.[19]

Other Uses of Cerebral Oximetry

The use of cerebral oximetry has been described in peripheral veno-arterial extracorporeal membrane oxygenation (VA ECMO). In this setup, blood oxygenated by the extracorporeal oxygenator is delivered into the femoral artery and then up the aorta to perfuse the upper body and brain. As the heart recovers and cardiac output increases, a watershed point occurs between the deoxygenated blood expelled from the heart and the oxygenated blood from the extracorporeal circuit. Depending on the location of this point, a differential perfusion phenomenon, known as “harlequin syndrome,” may occur (see Figure 3).[20] Cerebral oximetry can monitor cerebral perfusion to provide early warnings of inadequate cerebral perfusion or harlequin syndrome.[21] Additionally, when applied to the lower limb as a somatic oximeter, NIRS can be used to monitor lower limb ischemia.

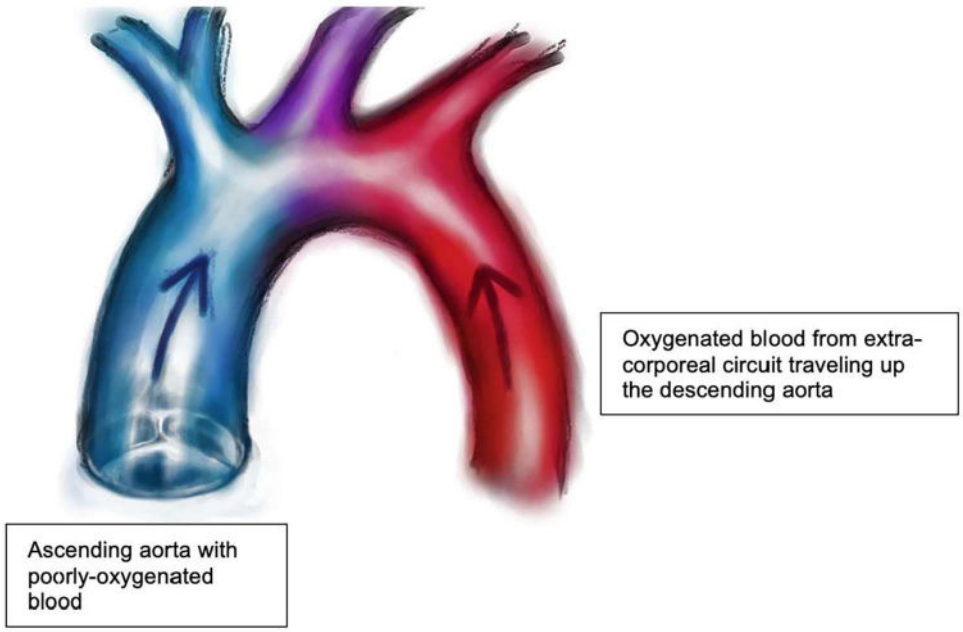

Figure 3. Harlequin's syndrome. Harlequin's syndrome can arise when relatively de-oxygenated blood, ejected by the left ventricle in case of poor pulmonary oxygenation, perfuses the aortic arch (Source: Meuwese et al.[20]).

Cerebral oximetry has also been advocated during cardiopulmonary resuscitation (CPR). The technology's ability to assess non-pulsatile flow is particularly beneficial. ScO2 is suggested to reflect the quality of CPR, predict the chance of spontaneous return of circulation (ROSC), provide early warnings of re-arrest, and assist in neurologic prognostication after ROSC.[21]

Furthermore, cerebral oximetry has been employed to evaluate brain pressure autoregulation status. The cerebral oximetry index (COx), a NIRS-based parameter, represents the correlation coefficient between mean arterial pressure and the slow waves of ScO2. COx has been validated against other autoregulation parameters, such as the mean velocity index (Mx) derived from transcranial Doppler measurements.[22,23] Positive COx values indicate impaired autoregulation, while negative values suggest preserved vasomotor response and autoregulation.[24] Individualized bedside assessment of cerebral autoregulation status, determination of optimal mean arterial pressure (MAP), and identification of the lower limit of pressure autoregulation may allow personalized physiological management in the future.

SUMMARY

Cerebral oximetry aims to measure regional brain oxygenation to inform clinicians about the adequacy of cerebral perfusion. ScO2 serves as a surrogate marker for the balance between cerebral oxygen supply and demand and is most effective when used as a trend monitor. Despite its promising role as a non-invasive neuromonitoring tool across various clinical scenarios, routine use of cerebral oximetry is not yet supported by high-quality clinical data. Furthermore, several technical challenges limit its widespread adoption.

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